



Management of Heat Injuries

SAF-MOH Clinical Practice Guidelines 1/2010



MINISTRY OF HEALTH
SINGAPORE



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Manpower,
Singapore



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701 Transit Road
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ISBN 978-981-08-6960-1

November 2010

Levels of evidence and grades of recommendation

Levels of evidence

Level	Type of Evidence
1 ⁺⁺	High quality meta-analyses, systematic reviews of randomised controlled trials (RCTs), or RCTs with a very low risk of bias.
1 ⁺	Well conducted meta-analyses, systematic reviews of RCTs, or RCTs with a low risk of bias.
1 ⁻	Meta-analyses, systematic reviews of RCTs, or RCTs with a high risk of bias
2 ⁺⁺	High quality systematic reviews of case control or cohort studies. High quality case control or cohort studies with a very low risk of confounding or bias and a high probability that the relationship is causal
2 ⁺	Well conducted case control or cohort studies with a low risk of confounding or bias and a moderate probability that the relationship is causal
2 ⁻	Case control or cohort studies with a high risk of confounding or bias and a significant risk that the relationship is not causal
3	Non-analytic studies, e.g. case reports, case series
4	Expert opinion

Grades of recommendation

Grade	Recommendation
A	At least one meta-analysis, systematic review of RCTs, or RCT rated as 1 ⁺⁺ and directly applicable to the target population; or A body of evidence consisting principally of studies rated as 1 ⁺ , directly applicable to the target population, and demonstrating overall consistency of results
B	A body of evidence including studies rated as 2 ⁺⁺ , directly applicable to the target population, and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 1 ⁺⁺ or 1 ⁺
C	A body of evidence including studies rated as 2 ⁺ , directly applicable to the target population and demonstrating overall consistency of results; or Extrapolated evidence from studies rated as 2 ⁺⁺
D	Evidence level 3 or 4; or Extrapolated evidence from studies rated as 2 ⁺
GPP (good practice points)	Recommended best practice based on the clinical experience of the guideline development group.

CLINICAL PRACTICE GUIDELINES

Management of Heat Injuries

SAF - MOH Clinical Practice Guidelines 1/2010

Published by SAF Medical Corps
701 Transit Road #03-02
Singapore 778910

Printed by Oxford Graphic Printers Pte Ltd

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ISBN: 978-981-08-6960-1

Available on the MOH website: <http://www.moh.gov.sg/cpg>

Statement of Intent

These guidelines are not intended to serve as a standard of medical care. Standards of medical care are determined on a basis of all clinical data available for an individual case and are subject to change as scientific knowledge advances and patterns of care evolve.

The contents of this publication are guidelines to clinical practice, based on the best available evidence at the time of development. Adherence to these guidelines may not ensure a successful outcome in every case. These guidelines should neither be construed as including all proper methods of care, nor exclude other acceptable methods of care. Each physician is ultimately responsible for the management of his/her unique patient, in the light of the clinical data presented by the patient and the diagnostic and treatment options available.

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Foreword

The tragedy of exertional heat stroke is that it frequently strikes highly motivated young individuals under the discipline of work, military training and sporting endeavours. Indeed, heat stroke is reported to be the third leading cause of death in athletes in the contemporary setting. The equatorial climate in Singapore poses a significant risk of heat injuries for individuals participating in strenuous physical activities, particularly in endurance sports like marathons and triathlons, which have become increasingly popular locally.

Over the years, the Singapore Armed Forces (SAF) has developed guidelines on the prevention and management of heat injuries. Through its research, programmes for acclimatisation, hydration and managing work-rest cycles have been implemented, which coupled with soldier education, have substantially reduced the incidence of exertional heat injuries by more than ten-fold. In addition, the SAF has implemented an in-house evaporative body cooling unit which has been shown to effectively treat hyperthermia.

This set of guidelines incorporates the best available evidence from the scientific literature and expert consensus to assist medical practitioners in the prevention and clinical management of exertional heat injuries. Through its publication, we seek to promulgate the evidence-based lessons learnt from various expert agencies to benefit the wider medical community.

We trust that you will find this set of guidelines useful in your practice.

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SAF Medical Corps

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DSO National Laboratories

Executive summary of recommendations

Details of recommendations can be found in the main text at the pages indicated.

Definition and diagnosis of heat injuries

C Core body temperature should be measured using rectal temperature (pg 15).

Grade C, Level 2+

C Aural, oral, skin, temporal and axillary temperature measurements are not reliable and should not be used for the diagnosis of exertional heat stroke and exertional heat exhaustion (pg 15).

Grade C, Level 2+

GPP A normal or lower presenting temperature should not exclude the diagnosis of heat stroke. If there is uncertainty differentiating between heat exhaustion and heat stroke, the patient should be promptly managed as for heat stroke (pg 15).

GPP

Risk factors for heat injuries

GPP Be vigilant about heat injury even when exercising in cooler conditions, especially if relative humidity is high (pg 16).

GPP

GPP The use of wet-bulb globe temperature to assess environmental heat stress should be carefully calibrated, measured and interpreted in the local context (pg 17).

GPP

Prevention of heat injuries

C Individuals who suffer from or who have recently recovered from acute illness or exertional heat injury can be gradually conditioned to participate in intense training following full recovery (pg 20).

Grade C, Level 2+

D Sporting event organisers, coaches, athletes and soldiers should receive information on the prevention, recognition and treatment of heat injury and the risks associated with exercising in hot, humid conditions (pg 20).

Grade D, Level 4

B Individuals involved in working in a high heat stress environment should undergo a heat acclimatisation regime over 10 to 14 days to improve body temperature regulation during heat exposure (pg 21).

Grade B, Level 2++

C Individuals should maintain proper hydration during the heat acclimatisation process. Fluid replacement improves induction and the effects of heat acclimatisation (pg 21).

Grade C, Level 2+

D Individuals undergoing acclimatisation should progressively increase the intensity and duration of work in the heat for up to 2 hours continuously (pg 21).

Grade D, Level 4

GPP Individuals should drink sufficient water to maintain a clear urine colour before exercise. Fluid intake should start the night before and in the hours leading to the event to maintain clear urine colour. Quenching of thirst together with maintenance of body weight can also be used as an indicator of euhydration if urine colour cannot be used (pg 22).

GPP

C Individuals should drink to replace water loss of about 2% body weight and to quench thirst (pg 22).

Grade C, Level 2+

D Where possible, a customised personal fluid replacement programme should be developed by measuring fluid requirements to prevent dehydration over a few training sessions (pg 22).

Grade D, Level 4

C For rapid and complete recovery from dehydration, drink 1.5L of fluids for each kilogram of body weight loss after exercise (pg 23).

Grade C, Level 2+

D After exercise, continue to rehydrate at regular intervals until clear urine colour is achieved (pg 23).

Grade D, Level 4

C Consume beverages and snacks with sodium to expedite recovery by stimulating thirst and fluid retention (pg 23).

Grade C, Level 2+

C Wear clothing that is light-coloured and lightweight to facilitate body cooling (pg 23).

Grade C, Level 2+

D Plan physical activities and rest breaks in accordance with the intensity of activity and environmental conditions (pg 24).

Grade D, Level 4

Medical and safety coverage for sports and exercise mass participation events

GPP Ensure adequate and appropriate medical and safety coverage according to accepted guidelines when organising a mass participation sporting event (pg 25).

GPP

Treatment of exertional heat injuries

D To relieve muscle spasms, the individual should stop the activity and initiate mild stretching and massage the muscle. A recumbent position may allow more rapid distribution of blood flow to cramping leg muscles (pg 26).

Grade D, Level 3

D A sodium-containing sports beverage may prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat (pg 26).

Grade D, Level 4

D Intravenous hydration with 0.9% normal saline may be required in severe or refractory cases when the symptoms continue to rebound (pg 26).

Grade D, Level 3

GPP To treat heat syncope, rest in a cool place and in a supine position with both legs and hip elevated to increase venous return. Other causes of syncope need to be ruled out (pg 26).

GPP

The following general measures should be instituted in the management of heat exhaustion and heat stroke:

1. **GPP** Transfer the patient to a cooler and shaded environment (pg 27).

GPP

2. **D** Assess the patient's airway, breathing and circulation (ABCs) and institute basic resuscitative support as appropriate (pg 27).

Grade D, Level 4

3. **B** Measure core body temperature with a rectal thermometer if available (pg 27).

Grade B, Level 2++

4. **D** Institute immediate and aggressive cooling efforts to reduce core body temperature (pg 27).

Grade D, Level 3

5. **D** Maintain hemodynamic stability (pg 27).

Grade D, Level 3

D Individuals who do not improve rapidly should be transported to an emergency department (pg 27).

Grade D, Level 4

D Progressive worsening of consciousness should trigger a detailed evaluation for hyperthermia, hyponatraemia, hypoglycaemia and other medical problems (pg 27).

Grade D, Level 3

C Rapid cooling is desirable as decreasing body temperature to below 39°C within 30 minutes of presentation has been shown to improve survival (pg 28).

Grade C, Level 2+

D Cooling by tap water and the application of ice packs can be used as the initial cooling methods prior to evacuating a heat injury patient to a medical facility (pg 28).

Grade D, Level 3

B Immersion in ice water may be done to manage exertional heat stroke occurring in young people, military personnel, and athletes (pg 28).

Grade B, Level 2++

GPP Body cooling should be initiated immediately and the selection of method should depend on availability of equipment and the staff's familiarity with the selected technique (pg 29).

GPP

B The use of iced peritoneal lavage and gastric lavage has yielded inconsistent results and is therefore not recommended (pg 30).

Grade B, Level 2 ++

B The use of pharmacologic agents has not been shown to accelerate body cooling in the treatment of heat stroke and is therefore not recommended (pg 30).

Grade B, Level 2 ++

D The initial management of heatstroke should include adequate fluid replacement to restore blood pressure and tissue perfusion (pg 31).

Grade D, Level 3

D Intravenous fluid replacement should be carefully titrated to clinical endpoints to avoid fluid overload and iatrogenic pulmonary oedema (pg 31).

Grade D, Level 3

D Large amounts of intravenous infusions should be guided by invasive hemodynamic monitoring (e.g. using central venous line or pulmonary artery flotation catheter) (pg 31).

Grade D, Level 3

D For heat stroke patients, short acting benzodiazepines are generally effective in controlling seizures (pg 31).

Grade D, Level 4

D In patients with hypotension that is refractive to intravenous therapy, vasopressors may be indicated, but should be used with caution because catecholamines can lead to increased heat production. In such patients, invasive hydrodynamic monitoring (e.g. central venous pressure) is indicated (pg 32).

Grade D, Level 4

GPP Intravenous fluids and diuretics (e.g. mannitol at 0.25g/kg) help to maintain renal blood flow and may prevent renal destruction in heat stroke. Alkalinisation has been recommended for rhabdomyolysis and hemofiltration should be considered for severe cases (pg 32).

GPP

D Disseminated intra-vascular coagulation is an indication of poor prognosis and should be managed with blood products for bleeding accordingly (pg 32).

Grade D, Level 4

D Non Steroidal Anti-Inflammatory Drugs (NSAIDs) and paracetamol should be avoided in the treatment of exertional heat stroke as these may precipitate hepatic damage (pg 32).

Grade D, Level 4

GPP Cardiac monitoring should be used in a patient with heat stroke (pg 33).

GPP

Prognosis and return to physical activity

D Rehabilitation of a heat stroke patient follows a slow course. The patient must be asymptomatic and all laboratory tests and body weight should have normalised before he can be considered to have recovered (pg 34).

Grade D, Level 3

GPP After heat stroke, a gradual and monitored return to physical activity is recommended with progressive exposure to heat to increase tolerance. The specific regime will be determined based on the severity of injury (pg 35).

GPP

1 Introduction

1.1 Guideline development

These guidelines were developed by a multidisciplinary workgroup appointed by the Singapore Armed Forces and the Ministry of Health. The workgroup comprised experts in Sports Medicine, Exercise and Sports Science, Emergency Medicine, Family Medicine, Military Medicine and Public Health. They were developed by adapting existing guidelines, reviewing relevant scientific literature and expert clinical consensus.

1.2 Objectives

The aim of these guidelines is to provide an evidence-based guide for the diagnosis, prevention and management of heat injuries in individuals at highest risk of exertional heat injury, i.e. those involved in strenuous physical activities.

1.3 Target group

These guidelines are intended for use by medical practitioners for preventive and clinical management and for consultation to event organisers. It may also be useful for athletes, sports officials, military and non-military personnel organising or participating in physically strenuous activities and endurance sports.

1.4 Review of guidelines

Evidence-based clinical practice guidelines are only as current as the evidence that supports them. Users must keep in mind that new evidence could supersede recommendations in these guidelines. The workgroup advises that these guidelines be scheduled for review 5 years after publication, or if new evidence appears that would require substantive changes to the recommendations.

2 Heat injury and its relevance

2.1 Introduction

Historically, heat stroke has been a health threat for centuries. From anecdotal reports, hundreds of fatalities were sustained during heat waves and thousands of casualties reported among Muslim pilgrims in Mecca.¹ Heat stroke also contributed to the defeat of King Richard's forces in their battle for the Holy Land in the 12th century² and of the Egyptian forces (~20,000 cases) during the Six-Day War against Israel in 1967.³

The tragedy of heat stroke is that it frequently strikes highly motivated young individuals under the discipline of work, military training and sporting endeavours. The actual incidence of heat injuries may be underestimated as some cases may not be diagnosed. Exertional heat stroke is reported to be the third leading cause of death in athletes in the contemporary setting.⁴ With the increasing popularity of large-scale endurance activities like marathons and triathlons, the incidence of heat stroke is likely to rise. In events such as The Falmouth Road Race, an 11.5-km race, staged in hot and humid summer conditions (wet-bulb globe temperature 21-27°C) averages 10 to 20 exertional heat stroke cases per 10,000 entrants.⁵

In the Singapore context, the equatorial climate poses significant risk of heat injuries for all individuals participating in strenuous physical activities. Those involved in endurance sports, military training and working outdoors (eg. construction workers) are particularly vulnerable. A retrospective review of exertional heat injury in the Singapore Armed Forces (SAF) from 1990 to 2008 revealed that heat injury cases had decreased from 50 cases per 10,000 training population to about 5 cases per 10,000, following the introduction of a system for risk management during training, soldier education, hydration regime and acclimatisation and periodisation training.⁶ In 2009, there were 6 construction workers who suffered from heat stroke including 2 deaths and 1 with permanent brain injury. Following the introduction of preventive measures to the industry such as risk management, acclimatisation, hydration and worker education, there was only 1 case of heat stroke reported to the Ministry of Manpower in the first half of 2010.

2.2 Singapore as a sports hub and the increased relevance of heat injuries

Singapore is gradually becoming an international sports hub and is the venue for major sporting events like the Youth Olympic Games in 2010. Large-scale local sporting events have also consistently garnered substantial participation. 70,000 people participated in the Singapore Bay Run / Army Half Marathon 2008 and approximately 48,000 participated in the Standard Chartered Singapore Marathon 2008. There is significant risk of heat injuries to both professional as well as recreational athletes for such events, especially in our hot and humid climate.

There is a need for an increased awareness of heat injuries among medical practitioners, event organisers and participants, so as to enhance preventive measures, to enable timely and accurate diagnosis and to institute rapid and appropriate treatment. These clinical practice guidelines serve to standardise evidence-based case definitions and clinical management across institutions and serve to reduce the morbidity and mortality of heat-related illness.

3 Models of heat stroke

3.1 Pathogenesis of Heat Stroke

The classical models of heat stroke attributed the pathogenesis of heat stroke to the direct cytotoxic effect of hyperthermia coupled with circulatory shock resulting from excessive dehydration.^{7,8} Hyperthermia results in internal organ tissue temperatures rising above critical levels causing damage to the cell membranes and energy systems.⁹ Dehydration and salt depletion impairs thermoregulation and the compromise in blood circulation reduces blood flow to the major organs, causing them to fail eventually. However, these factors cannot explain fully the observation of systemic coagulation, massive haemorrhages and other sepsis-like clinical symptoms observed in heat stroke patients. Clinical and autopsy report findings in heat stroke patients also show similarity with sepsis patients from other causes.⁹⁻¹⁴

Since the 1990s, researchers introduced the idea that heat stroke is triggered by heat, but its progression to multiorgan-dysfunction syndrome is due to a complex interplay between the acute physiological alterations associated with hyperthermia (circulatory dysfunction, hypoxia and increased metabolic demand), the direct cytotoxicity of heat, and the inflammatory and coagulation responses of the host. This constellation of events leads to alterations in blood flow in the microcirculation and results in injury to the vascular endothelium and tissues.^{15,16}

Recent models of heat stroke suggest that heat stroke may be triggered by the coupling of hyperthermia and endotoxemia.¹⁷

Endotoxins or lipopolysaccharides (LPS) play an important role in fuelling the inflammatory response. Under resting conditions, the epithelium of the intestinal wall forms the barrier that prevents LPS from infiltrating the sterile environment of the circulating blood. However, the integrity of the gut epithelium is compromised during exercise and heat stress, resulting in the translocation of LPS into the blood circulation system.¹⁸⁻²⁰ Mild endotoxemia has been reported in endurance athletes during races²¹ and in laboratory settings.²² This is also observed at moderate heat stress levels.²³

The body, under normal circumstances, clears circulating LPS through the reticuloendothelial system of the liver, high density lipoproteins and LPS antibodies.²⁴ A period of intense exercise may compromise the anti-LPS system, thus impairing the removal of LPS and hence reduce heat tolerance. Similarly, in immune-compromised individuals, where LPS clearance is suppressed, endotoxemia can develop and LPS can accumulate to a concentration that triggers sepsis, which in turn, triggers the systemic inflammatory response.¹¹ Sepsis and systemic inflammation results in disseminated intravascular coagulation, central nervous system dysfunction and multi-organ failure.^{8,12} In animal experiments, inhibiting endotoxemia through pharmacological agents protected monkeys,^{25,26} dogs,²⁷ rabbits²⁸ and rodents²⁹ from lethal heat load, whereas the placebo animals died from the effects of endotoxemia.

4 Definition and diagnosis of heat injuries

4.1 Types of heat injuries

Heat injuries can be viewed as a continuum of illnesses relating to the body's inability to cope with an increase in heat load.³⁰ This can occur when intrinsic or extrinsic heat generation overwhelms homeostatic thermoregulation.³¹ The consequential dysfunction at cellular and organ level results in a spectrum of disease ranging from minor heat cramps through moderate symptoms of heat exhaustion to life threatening heatstroke.

There is no evidence that mild heat injuries (heat cramps, heat syncope) will progress to severe disease if untreated. However, the development of heat exhaustion is significant. Without treatment, heat exhaustion has the potential to progress to heat stroke.

4.2 Mild heat-related illness

Heat Syncope occurs with orthostatic hypotension resulting from volume depletion, peripheral vasodilatation (physiologic response to heat production) and venous pooling. Prolonged standing after significant exertion and rapid change in body position after exertion, such as from sitting to standing, may lead to heat syncope.^{32,33}

Heat Cramps are acute, painful and involuntary muscle contractions that occur during or after intense exercise sessions in the heat. Muscles involved are usually the calf, quadriceps and abdominal muscles. Heat cramps are associated with fluid deficiencies (dehydration), electrolyte imbalances and neuromuscular fatigue.³⁴⁻³⁶ They are one of the earliest indications of heat injuries and may occur independently or with other symptoms of heat exhaustion.

4.3 Heat exhaustion

Exhaustion is a physiologic response to work defined as the inability to continue exercise.

Heat exhaustion is characterised as follows:

1. Inability to continue strenuous physical exertion due to fatigue from heat stress.
2. Normal mental state and stable neurological status.

Heat exhaustion may be associated with mild central nervous system (CNS) or other non-specific symptoms such as profuse sweating, nausea, vomiting, headache, dizziness, light-headedness, intestinal cramps, weakness, hyperventilation and cool and clammy skin. The core temperature is usually in the range of 37.7°C to 40°C. Critical to the diagnosis is a normal mental state and stable neurological status. If heat exhaustion is not treated appropriately, it can progress to heat stroke.^{30,33,37}

4.4 Heat stroke

Heat stroke is a serious and potentially fatal systemic condition that occurs when the thermoregulation system is overwhelmed. It is a form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates. Heat stroke can be categorized into exertional and classical heat stroke.^{16,32,37-39}

Exertional heat stroke occurs during physical exertion, when hyperthermia is due largely to the inability to remove endogenous metabolic heat.^{40,41} Athletes, outdoor adventure enthusiasts, military personnel and manual workers who work under hot and humid conditions are exposed to the risk of exertional heat stroke.

Classical heat stroke occurs during passive exposure to environmental heat stress.³¹ It is most frequently observed during heat waves. Young children, the aged sick, schizophrenic patients⁴² and patients who are bed-ridden are particularly vulnerable to classical heat stroke during heat waves.^{13,43,44} In aged patients, existing chronic illnesses (e.g. heart disease) may contribute to the higher morbidity and mortality from heat stroke in extremely hot weather.^{45,46} Classical heat stroke has been attributed to impairment of homeostatic mechanisms under conditions of high ambient temperature.^{47,48}

Although there is general agreement on the key features of heat stroke, a universal definition of heat stroke has yet to be stated. This condition is generally described based on the characteristics of heat stroke from forensic and clinical reports. The clinical presentation of heat stroke includes hyperthermia (core temperature $>40^{\circ}\text{C}$), systemic inflammation, central nervous system dysfunction (coma, delirium and convulsion), disseminated intravascular coagulation, and multi-organ dysfunction. Hot and dry skin and cardiovascular deterioration may be observed at times.^{9,49}

Heat stroke is characterised as a systemic condition that includes:^{8,43}

1. Elevated core body temperature, usually above 40.0°C .^{17,34,37,50-52} A delay in core temperature measurement may delay diagnosis, as this may result in a lower presenting temperature.
2. Central nervous system dysfunction. Neurological changes due to central nervous system dysfunction are often the first marker of heat stroke.⁵³ Neurological changes may take many forms and are non-specific. These include dizziness, drowsiness, confusion, irritability, aggressiveness, apathy, disorientation, seizures and even coma.^{50,54}
3. Systemic inflammatory response with multi-organ failure. Multi-organ-dysfunction syndrome includes conditions such as encephalopathy, rhabdomyolysis, acute renal failure, acute respiratory distress syndrome, myocardial injury, hepatocellular injury, intestinal ischemia or infarction, pancreatic injury, and hemorrhagic complications, especially disseminated intravascular coagulation, with pronounced thrombocytopenia.^{16,17,49,55,56}

Rectal temperature reading is a reliable measure of core body temperature as this is stable and not influenced by ambient conditions.⁵⁷⁻⁵⁹ It is also relatively easy to obtain, including in a neurologically disturbed patient.

C Core body temperature should be measured using rectal temperature.^{41,53,60-62}

Grade C, Level 2+

C Aural, oral, skin, temporal and axillary temperature measurements are not reliable and should not be used for the diagnosis of exertional heat stroke and exertional heat exhaustion.^{34,58,60,62,63}

Grade C, Level 2+

GPP A normal or lower presenting temperature should not exclude the diagnosis of heat stroke. If there is uncertainty differentiating between heat exhaustion and heat stroke, the patient should be promptly managed as for heat stroke.

GPP

5 Risk factors for heat injuries

5.1 Environmental risk factors

Environmental risk factors that influence the risk of heat illness include the ambient air temperature, relative humidity, air movement and the amount of radiant heat from the sun and other sources.^{38,64,65}

When the environmental temperature is above skin temperature, individuals begin to absorb heat from the environment and depend entirely on evaporation for heat loss.^{66,67}

The ability to sweat is very important for thermoregulation and for sustaining exercise over long duration. However the propensity for sweat to be evaporated is inversely related to the amount of water vapour in the air. A high relative humidity inhibits evaporative heat loss whereas a low relative humidity promotes evaporative heat loss.⁶⁸ Exercising under warm and humid conditions causes the body to lose fluid through sweating with minimal heat loss. Conversely, the cooler temperatures at night facilitate convective and radiative heat dissipation but may impede evaporative heat loss as relative humidity is higher. Since evaporative heat loss accounts for > 80% of heat dissipation during exercise, the net result can be an increase in heat storage in the body.

GPP Be vigilant about heat injury even when exercising in cooler conditions, especially if relative humidity is high.

GPP

5.2 Wet-bulb globe temperature (WBGT) and heat stress and strain

The wet-bulb globe temperature (WBGT) was developed by the U.S. Military in 1957 and remains the most widely used index of heat stress. WBGT is a composite temperature calculated using the weighted averages of natural wet-bulb (Tw), dry-bulb (Td), and the black-globe (Tg) temperatures. The natural wet-bulb (Tw) temperature represents the integrated effect of humidity, air

movement and radiant heat. The black-globe (T_g) temperature represents the integrated effect of air movement and radiant heat.⁶⁹ The dry-bulb temperature represents the air temperature. The relative risk of heat illness can be estimated using the WBGT and the use of this index to modify activities has been demonstrated to decrease morbidity and mortality from heat injuries.⁷⁰

However, the WBGT has several limitations. In tropical climates, the high environmental temperatures and high relative humidity result in a WBGT that exceeds the recommended upper limits of existing standards and guidelines used by most organisations. Measurement errors are common because of non-standard instrumentation and calibration procedures. Moreover, the interpretation of WBGT values requires careful evaluation as other confounding internal factors may also affect the heat injury risk.⁷¹

GPP The use of wet-bulb globe temperature to assess environmental heat stress should be carefully calibrated, measured and interpreted in the local context.

GPP

5.3 Non-environmental risk factors

While the external environment plays an important role in causing heat injuries, various internal environmental factors also contribute to an individual's risk of developing heat injury.

Dehydration

Dehydration reduces performance of strenuous physical activities, decreases time to exhaustion and increases heat storage.⁷² Dehydration increases the risk of heat exhaustion.⁷³⁻⁷⁶

Physical barriers to heat loss

Clothes or equipment (e.g. plastic or rubber) that impair or do not allow heat dissipation by evaporation, convection or radiation increase the risk of heat injury.^{77,78}

Previous history and predisposing medical conditions

Some individuals with a history of heat injury and malignant hyperthermia are at a greater risk of recurrent heat illness.^{79,80}

The following medical conditions are also associated with higher risk of heat injury: neuroleptic malignant syndrome, arteriosclerotic vascular disease, scleroderma, cystic fibrosis and sickle cell trait.⁸¹⁻⁸⁶

Increased body mass index

Obese individuals are at increased risk for heat injuries because the fat layer decreases heat loss. Obese persons are also less efficient and have greater metabolic heat production during exercise.^{87,88} Muscular individuals have increased metabolic heat production and a lower surface area to mass ratio, contributing to a decreased ability to dissipate heat.^{76,89}

Poor physical condition

Individuals who are unfit are more susceptible to heat injury than trained athletes. As the physical fitness of an individual improves, the ability to tolerate heat stress improves independent of acclimatisation and heat adaptation.⁹⁰

Lack of acclimatisation to heat

An individual with no or minimal physiological acclimatisation to hot conditions is at increased risk of heat injury.^{68,91}

Concurrent illnesses

Individuals who are currently or were recently ill, particularly from febrile illness or dehydration, may be at an increased risk of heat injury.

Overzealousness

Individuals who are overzealous in performing strenuous physical activities may be at a higher risk of heat injury because they tend to ignore the early symptoms of heat injuries.⁶⁷

Medications and drugs

Medications associated with heat injury can be divided into two broad groups: drugs that impair heat loss and drugs that increase heat production.^{4,31,92}

Drugs that impair heat loss include neuroleptics and anticholinergics. The phenothiazine class of neuroleptics has been most implicated in heat-related illness, attributed primarily to dopamine blockade. This inhibits afferent neuronal input to the hypothalamus, which decreases the hypothalamus's normal compensatory effect of increasing cutaneous blood flow to aid heat dissipation.⁹⁶ The dibenzapine derivatives, including clozapine, olanzapine and quetiapine are implicated in neuroleptic malignant syndrome and increased temperature.^{94,95}

Anticholinergic agents such as atropine and benztropine impair thermoregulation through the inhibition of sweating and reduction in heat elimination.⁹⁶⁻⁹⁸

Stimulants like amphetamines, thyroid agonists and, alpha-adrenergic agents can cause an increase in heat production. Over-the-counter drugs and nutritional supplements containing ephedrine, synephrine and other sympathomimetic compounds may increase heat production.^{99,100}

Other medications that affect the cardiovascular system like anti-hypertensives and diuretics may inhibit natural cardioprotective responses to dehydration and heat illness.¹⁰¹ Alcohol, caffeine and theophylline at certain doses are mild diuretics.^{102,103}

6 Prevention of heat injuries

6.1 Screening

There are currently no tests to effectively screen individuals for their risk of heat injury.

C Individuals who suffer from or who have recently recovered from acute illness or exertional heat injury can be gradually conditioned to participate in intense training following full recovery.^{79,80,104,105}

Grade C, Level 2+

6.2 Education

D Sporting event organisers, coaches, athletes and soldiers should receive information on the prevention, recognition and treatment of heat injury and the risks associated with exercising in hot, humid conditions.^{5,37,38,53,63,106-109}

Grade D, Level 4

Individuals should be educated to balance their fluid intake with sweat and urine losses to maintain adequate hydration status.^{110,111} Adequate rest¹⁰⁹ and a balanced diet^{112,113} prior to strenuous physical activity may be beneficial.

6.3 Heat acclimatisation

Heat acclimatisation is a progressive and systemic process of conditioning the thermoregulatory system to operate optimally during physical exertion in the heat. The heat acclimatisation process involves 10 to 14 days of gradual increase in duration of exposure to performing moderate work under hot weather conditions. Physiological adaptation can be observed from the 4th day and are usually optimised by 10 to 14 days.^{114,115} The key physiological adaptations to heat acclimatisation include earlier onset of sweating, higher sweat rate and cardiac output, lower resting core temperature and rate of increase in core temperature during exercise in the heat.^{92,116,117} The rate of acclimatisation is related to aerobic conditioning and fitness; with better

conditioned athletes acclimatising more quickly.¹¹⁸ The heat acclimatisation adaptations may vanish after a period of inactivity.¹¹⁹

B Individuals involved in working in a high heat stress environment should undergo a heat acclimatisation regime over 10 to 14 days to improve body temperature regulation during heat exposure.^{37,53,80,104,119-123}

Grade B, Level 2++

C Individuals should maintain proper hydration during the heat acclimatisation process. Fluid replacement improves induction and the effects of heat acclimatisation.^{124,125}

Grade C, Level 2+

D Individuals undergoing acclimatisation should progressively increase the intensity and duration of work in the heat for up to 2 hours continuously.¹²⁶⁻¹²⁸

Grade D, Level 4

6.4 Hydration

Athletes should maintain proper hydration before, during and after physical exertion. Dehydration increases the risk of heat exhaustion¹²⁹ and is a risk factor for heat stroke.¹³⁰⁻¹³² Appropriate hydration is important to minimize health risks. One should start the exercise activity in a euhydrated state. During the activity, it is normal to be slightly dehydrated, but the degree of dehydration should not exceed 2% of body weight. After the activity the athlete should continue drinking fluid at regular intervals to correct the fluid deficit incurred during the activity. If time permits, consuming normal meals and beverages will restore euhydration. Drinking beverages with sodium and or small amounts of salted snacks or sodium-containing foods at meals will help to stimulate thirst and retain the consumed fluids.^{133,134} Hydration status can be ascertained by urine colour status and body weight measurement.^{135,136} As there is considerable inter-individual variation in sweat response, recommending a fixed volume for water intake during exercise may be inappropriate. Drinking excessive volumes of fluids may not be tolerated and over-drinking may contribute to exercise-associated

hyponatraemia. The following are recommendations for a hydration regime before, during and after an activity.^{122,137-143}

Before activity

GPP Individuals should drink sufficient water to maintain a clear urine colour before exercise. Fluid intake should start the night before and in the hours leading to the event to maintain clear urine colour. Quenching of thirst together with maintenance of body weight can also be used as an indicator of euhydration if urine colour cannot be used.

GPP

During activity

C Individuals should drink to replace water loss of about 2% body weight and to quench thirst.^{137,140,143-145}

Grade C, Level 2+

It is inappropriate to use a single fluid replacement rate for all runners. An estimate proposed for marathon runners is to drink ad libitum from 400 to 800mls/h, with the higher rates for faster, heavier individuals competing in warm environments and the lower rates for slower, lighter persons competing in cooler environments.¹⁴⁰ Body weight loss during exercise can be established by measuring body weight before and after exercise during routine training. Measurements taken over several sessions can be used as the norm of body weight loss for the similar type of exercise. For example, 2% body weight for a 70kg man is 1.4kg, which translates into 1.4 L of fluid. If this individual ascertains that he loses about 1.8kg in body weight after a 1h run, he would need to drink more than 400mls of fluid during the exercise to prevent excessive dehydration.

D Where possible, a customised personal fluid replacement programme should be developed by measuring fluid requirements to prevent dehydration over a few training sessions.^{137,139,143}

Grade D, Level 4

After activity

C For rapid and complete recovery from dehydration, drink 1.5L of fluids for each kilogram of body weight loss after exercise.^{137,143,146}

Grade C, Level 2+

This form of rehydration is useful for athletes who have multiple bouts of training within a short span of time.

D After exercise, continue to rehydrate at regular intervals until clear urine colour is achieved.^{137,143}

Grade D, Level 4

C Consume beverages and snacks with sodium to expedite recovery by stimulating thirst and fluid retention.^{121,122,133,135}

Grade C, Level 2+

6.5 Minimize barriers to evaporation

While exercising in hot and humid conditions, minimise the amount of clothing and equipment as these may retard heat loss from evaporation.

C Wear clothing that is light-coloured and lightweight to facilitate body cooling.^{38,67,80,109,121,147}

Grade C, Level 2+

6.6 Work-rest cycle

Exercise intensity and environmental conditions should be the main determinants in deciding the duration of physical activity and frequency of rest breaks. Where possible, the exercise session should be adjusted in accordance with the environmental conditions, for example, avoiding the hottest part of the day or avoiding radiant heat from direct sunlight.^{65,148} Adequate rest breaks should be planned to match the intensity of activity and environmental conditions.^{5,41} Where possible, rest breaks should be in the shade and planned during mealtimes to allow 2 to 3 hours for fluids, nutrients and electrolytes to move into the small intestine and bloodstream before the next activity.⁵³ Hydration should be encouraged during the rest breaks.^{5,149}

D Plan physical activities and rest breaks in accordance with the intensity of activity and environmental conditions.^{37,41,53,150}

Grade D, Level 4

7 Medical and safety coverage for sports and exercise mass participation events

7.1 Medical and safety coverage

Organising a mass participation sporting event includes planning for adequate and appropriate medical and safety coverage for the participants. Such planning should take into consideration the total number of participants, the intensity and duration of physical activity, as well as the estimated time for a casualty to reach medical attention. Planning encompasses first aid, on-site medical posts and casualty evacuation, as well as notifying the emergency ambulance services and nearby hospitals of the event taking place.¹⁴⁹⁻¹⁵¹ The organisers should ensure that there is sufficient supply of cold water and/or sports drinks to meet the participants' needs before, during and after the activity, as well as the availability of ice for active cooling of heat injury casualties.^{152,153} All rescue and medical personnel have to be familiar with the recognition and treatment of heat injuries.

GPP Ensure adequate and appropriate medical and safety coverage according to accepted guidelines when organising a mass participation sporting event.

GPP

8 Treatment of exertional heat injuries

8.1 Treatment of heat injuries

Morbidity and mortality are directly related to the duration and intensity of elevated core body temperature.^{30,41,51,154-159} Treatment protocols for heat injuries thus aims to lower the core body temperature to an acceptable level as rapidly as possible. Early diagnosis and proper treatment are critical for the patient's survival.⁵²

8.2 Treatment of heat cramps

D To relieve muscle spasms, the individual should stop the activity and initiate mild stretching and massage the muscle. A recumbent position may allow more rapid distribution of blood flow to cramping leg muscles.^{36,53}

Grade D, Level 3

D A sodium-containing sports beverage may prevent or relieve cramping in athletes who lose large amounts of sodium in their sweat.^{34,36,53,65}

Grade D, Level 4

D Intravenous hydration with 0.9% normal saline may be required in severe or refractory cases when the symptoms continue to rebound.^{36,101,160-162}

Grade D, Level 3

8.3 Treatment of heat syncope

GPP To treat heat syncope, rest in a cool place and in a supine position with both legs and hip elevated to increase venous return. Other causes of syncope need to be ruled out.

GPP

8.4 Treatment of heat exhaustion and heat stroke

General measures

The following general measures should be instituted in the management of heat exhaustion and heat stroke:

1. **GPP** Transfer the patient to a cooler and shaded environment.

GPP

2. **D** Assess the patient's airway, breathing and circulation (ABCs) and institute basic resuscitative support as appropriate.^{30,37,53}

Grade D, Level 4

3. **B** Measure core body temperature with a rectal thermometer if available.⁵⁷⁻⁵⁹

Grade B, Level 2++

4. **D** Institute immediate and aggressive cooling efforts to reduce core body temperature.^{30,52,163-165}

Grade D, Level 3

5. **D** Maintain hemodynamic stability.^{8,166,167}

Grade D, Level 3

D Individuals who do not improve rapidly should be transported to an emergency department.⁵³

Grade D, Level 4

D Progressive worsening of consciousness should trigger a detailed evaluation for hyperthermia, hyponatraemia, hypoglycaemia and other medical problems.^{53,168,169}

Grade D, Level 3

8.5 Lowering the core body temperature

C Rapid cooling is desirable as decreasing body temperature to below 39°C within 30 minutes of presentation has been shown to improve survival.^{30,52,141,164-166,170-172}

Grade C, Level 2+

Ice packs and tap water

A simple and efficient method to effect body cooling is to spray copious volumes of tap water directly on the skin and to facilitate evaporation using a fan.^{50,173,174} This does not require medical expertise and can be applied by sports events organisers.

Placing ice packs in the axilla, groin and neck has also been recommended to promote body cooling.¹⁷⁵⁻¹⁷⁷

D Cooling by tap water and the application of ice packs can be used as the initial cooling methods prior to evacuating a heat injury patient to a medical facility.^{30,41,51,106,153,178,179}

Grade D, Level 3

Cooling by immersion

Immersion of a heat stroke patient in ice water provides effective whole body cooling and reduces morbidity and mortality in exertional heat stroke. The rate of cooling has been reported to be in the range of 0.15 to 0.24°C/min.^{13,180,181}

B Immersion in ice water may be done to manage exertional heat stroke occurring in young people, military personnel, and athletes.^{163,164,171,181}

Grade B, Level 2++

However, this technique was found to be poorly tolerated among the elderly and was associated with increased morbidity and mortality.¹⁶⁴ Early studies reported severe shivering, agitation, and combativeness in this group, which often required restraining and sedation of the patient.⁹ Other drawbacks reported included poor hygiene (heatstroke is often associated with vomiting and diarrhoea) and difficulty achieving optimal monitoring and

resuscitation of unconscious and hemodynamically unstable patients in an ice bath.

Cooling by evaporation

Evaporative cooling is based on the physical principle that the conversion of 1.7ml of water to gaseous phase consumes 1kcal of heat.^{164,171} Evaporation-based cooling can be achieved by continuous spraying of water over the skin combined with forced-air equipment (ventilator/fan) which creates a warm, dry microclimate around the skin and promotes water evaporation.^{181,182}

Several methods have been described to achieve cooling by evaporation.

One report found a cooling rate of 0.14°C/min by simply splashing copious amounts of water (20-40L) over the heat stroke patient and fanning continuously.¹⁸⁰

The body cooling unit (BCU) developed by Khogali and Weiner was reported to be successful in treating heat stroke victims during the annual pilgrimage to Mecca, achieving a cooling rate of 0.05°C/min.^{182,183}

The Singapore Armed Forces (SAF) has developed a body cooling unit that has been reported to achieve a cooling rate of between 0.09 to 0.18°C/min in heat injury patients, which is comparable to the ice bath.¹⁸⁴

While there are reports that cooling by evaporation appear to be less efficient than immersion in ice water to dissipate heat^{163,164,171,181}, these methods are well tolerated and facilitate continuous monitoring and resuscitation. No difference in morbidity and mortality outcomes has been reported.

GPP Body cooling should be initiated immediately and the selection of method should depend on availability of equipment and the staff's familiarity with the selected technique.

GPP

Invasive cooling methods

Iced peritoneal lavage and gastric lavage are two invasive methods reported for body cooling.¹⁸⁷⁻¹⁸⁷ The rationale of invasive therapies is to bypass the body shell and achieve direct cooling of internal body organs. The results of such invasive techniques have been inconsistent, with the majority of studies performed on animal models.^{188,189}

B The use of iced peritoneal lavage and gastric lavage has yielded inconsistent results and is therefore not recommended.^{188,189}

Grade B, Level 2 ++

Pharmacologic methods

Dantrolene impairs calcium release from the sarcoplasmic reticulum and by doing so reduces muscle excitation and contraction. It is used to treat malignant hyperthermia and neuroleptic malignant syndrome and is proposed to have a role in reducing core body temperature in exertional heat stroke.¹⁹⁰⁻¹⁹⁴ However, studies have shown that there is no additional benefit in using dantrolene in the treatment of heat stroke.^{137,195-197}

Anti-pyretic, anti-inflammatory agents like paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs) and corticosteroids, as well as antibiotics have also been shown to have little or no benefits in managing heat injury.^{32,198}

Aspirin and paracetamol should be avoided because they may trigger fulminant hepatitis in patients with heat stroke and heat-induced hepatic damage.^{32,199} Antibiotics are not routinely administered in heat stroke unless the patient develops an infection or has suspected meningitis.

B The use of pharmacologic agents has not been shown to accelerate body cooling in the treatment of heat stroke and is therefore not recommended.^{137,195-197}

Grade B, Level 2 ++

8.6 Hydration and hemodynamic management

Although rapid and effective cooling is the cornerstone of treating heat stroke, the management of circulatory failure is also important.^{8,165,166} Hypotension is associated with a mortality rate of 33% compared with 10% in patients without hypotension. While hydration should be administered, it is not without complications.

Fluid resuscitation should be titrated to clinical endpoints of optimal heart rate, urine output and blood pressure. Patients who remain hypotensive after initial fluid and cooling therapy should be considered for invasive hemodynamic monitoring.¹⁸² However, invasive hemodynamic monitoring should be avoided in patients with complications of DIVC.

D The initial management of heatstroke should include adequate fluid replacement to restore blood pressure and tissue perfusion.^{8,165,166,182,200}

Grade D, Level 3

D Intravenous fluid replacement should be carefully titrated to clinical endpoints to avoid fluid overload and iatrogenic pulmonary oedema.^{201,202}

Grade D, Level 3

D Large amounts of intravenous infusions should be guided by invasive hemodynamic monitoring (e.g. using central venous line or pulmonary artery flotation catheter).²⁰¹⁻³

Grade D, Level 3

8.7 Treatment of complications of heat stroke

Heat stroke may be complicated by seizure^{204,205}, hypotension, rhabdomyolysis, hepatic injury²⁰⁶ and/or arrhythmias.^{16,41,51,207,208}

Seizure

D For heat stroke patients, short acting benzodiazepines are generally effective in controlling seizures.^{16,32,53}

Grade D, Level 4

Hypotension

D In patients with hypotension that is refractive to intravenous therapy, vasopressors may be indicated, but should be used with caution because catecholamines can lead to increased heat production. In such patients, invasive hydrodynamic monitoring (e.g. central venous pressure) is indicated.^{32,209}

Grade D, Level 4

Rhabdomyolysis

Rhabdomyolysis is a serious complication of exertional heat stroke and the monitoring of urine output is crucial.

GPP Intravenous fluids and diuretics (e.g. mannitol at 0.25g/kg) help to maintain renal blood flow and may prevent renal destruction in heat stroke.^{32,210,211} Alkalinisation has been recommended for rhabdomyolysis and hemofiltration should be considered for severe cases.^{38,209,211,212}

GPP

Disseminated intra-vascular coagulation

D Disseminated intra-vascular coagulation is an indication of poor prognosis and should be managed with blood products for bleeding accordingly.^{38,41,213,214}

Grade D, Level 4

Hepatic injury

Hepatic injury can result in coagulopathy and hepatitis.²¹⁵ Hypoglycemia should be detected and treated.

D Non Steroidal Anti-Inflammatory Drugs (NSAIDs) and paracetamol should be avoided in the treatment of exertional heat stroke as these may precipitate hepatic damage.³²

Grade D, Level 4

Arrhythmia

Hypotension and cell death can lead to myocardial damage and arrhythmia.^{53,163} Many of the arrhythmias will resolve with cooling.^{32,41}

GPP Cardiac monitoring should be used in a patient with heat stroke.

GPP

9 Prognosis and return to activity

9.1 Prognosis

Poor prognostic factors in heat stroke include hypotension,^{216,217} the need for endotracheal intubation, altered coagulation profile, advanced age, core body temperature $>41^{\circ}\text{C}$, duration of hyperthermia, prolonged coma, hyperkalemia and oliguric renal failure.^{9,41,218}

An elevation in serum levels of aspartate transaminase (AST), lactate dehydrogenase (LDH) and creatine kinase (CK) in the first 24 hours is reported to be associated with poorer prognosis as well.^{80,219-221}

9.2 Recovery and rehabilitation

After an episode of heat stroke, the individual may experience impaired thermoregulation, persistent central nervous system dysfunction,^{202,222} hepatic insufficiency, and renal insufficiency.²²³

The following laboratory tests should be monitored following heat stroke: haematological counts, blood chemistry, liver function test, urine biochemistry and creatine kinase.²²⁴ It is also prudent to monitor body weight until these normalises.

Transient or persistent heat intolerance may affect 15 to 20% of persons after an episode of heat stroke,^{225,226} which may persist up to 5 years.²²⁷ In some individuals, a history of exertional heat stroke increases the chance of recurrence and it is important to educate the patient on this risk.⁵ Such individuals may also have compromised heat tolerance and heat acclimatisation.^{228,229}

D Rehabilitation of a heat stroke patient follows a slow course. The patient must be asymptomatic and all laboratory tests and body weight should have normalised before he can be considered to have recovered.^{63,80}

Grade D, Level 3

9.3 Heat intolerance testing after an episode of heat stroke

Heat intolerance is defined as an inability to acclimatise to heat and is usually assessed by a patient's ability to complete a full day of labour in a hot environment.^{80,230} This occurs in a small percentage of patients with a prior history of heatstroke, and can be temporary or permanent.²³¹ However the benefits of heat intolerance testing are still uncertain.

9.4 Return to physical activity

For patients with mild forms of heat injury, proper hydration allows return to activity within 24-hours. In contrast, patients with heat stroke require further monitoring and evaluation.⁵¹

GPP After heat stroke, a gradual and monitored return to physical activity is recommended with progressive exposure to heat to increase tolerance. The specific regime will be determined based on the severity of injury.

GPP

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Self-assessment MCQs

After reading the Clinical Practice Guidelines, you can claim one CME point under Category 3A (Self-Study) of the SMC Online CME System. Alternatively, you can claim one CME point under Category 3B (Distance Learning - Verifiable Self Assessment) if you answer at least 60% of the following MCQs correctly. You can submit your answers through the SMJ website at this link: <http://smj.sma.org.sg/cme/smj/index.html> (*the link will only be available once the October 2010 issue of the SMJ becomes available*). The answers will be published in the SMJ December 2010 issue and at the SAF webpage for these guidelines after the period for submitting the answers is over.

Instruction: Indicate whether each statement is true or false.

- | | True | False |
|---|--------------------------|--------------------------|
| 1. Core body temperature should be measured using: | | |
| A) Oral thermometer | <input type="checkbox"/> | <input type="checkbox"/> |
| B) Tympanic/ Aural thermometer | <input type="checkbox"/> | <input type="checkbox"/> |
| C) Skin temperature | <input type="checkbox"/> | <input type="checkbox"/> |
| D) Rectal thermometer | <input type="checkbox"/> | <input type="checkbox"/> |
| 2. The following are risk factors of exertional heat injuries: | | |
| A) Dehydration | <input type="checkbox"/> | <input type="checkbox"/> |
| B) Schizophrenia and other mental illnesses | <input type="checkbox"/> | <input type="checkbox"/> |
| C) Previous history of heat illness | <input type="checkbox"/> | <input type="checkbox"/> |
| D) Concurrent upper respiratory tract infections | <input type="checkbox"/> | <input type="checkbox"/> |
| 3. Please state if the following statements are true or false: | | |
| A) Heat injuries can only occur when there is a large amount of heat stress. | <input type="checkbox"/> | <input type="checkbox"/> |
| B) The diagnosis of heat exhaustion and heat stroke is differentiated solely by the core body temperature. | <input type="checkbox"/> | <input type="checkbox"/> |
| C) Heat cramps and heat syncope frequently progress to heat stroke. | <input type="checkbox"/> | <input type="checkbox"/> |
| D) A patient with heat syncope usually recovers consciousness spontaneously when laid supine with both legs and hip elevated. | <input type="checkbox"/> | <input type="checkbox"/> |

	True	False
4. The following are effective cooling modalities for heat injuries:		
A) Placing ice packs in the axilla, groin and neck in the pre-hospital setting.	<input type="checkbox"/>	<input type="checkbox"/>
B) Iced peritoneal and gastric lavage.	<input type="checkbox"/>	<input type="checkbox"/>
C) Evaporative cooling with a body cooling unit.	<input type="checkbox"/>	<input type="checkbox"/>
D) Cold water immersion.	<input type="checkbox"/>	<input type="checkbox"/>
5. Please state if the following statements are true or false:		
A) Anti-hypertensive medications may inhibit natural physiological responses to heat stress.	<input type="checkbox"/>	<input type="checkbox"/>
B) Anti-pyretic agents like paracetamol have been shown to be effective in the management of heat injury.	<input type="checkbox"/>	<input type="checkbox"/>
C) Rapid and effective intravenous fluid administration is the cornerstone of treating heat stroke.	<input type="checkbox"/>	<input type="checkbox"/>
D) A patient with previous heat stroke can return to full exercise once he is asymptomatic and all his laboratory tests and body weight have normalised	<input type="checkbox"/>	<input type="checkbox"/>

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Acknowledgements

The workgroup would like to acknowledge the following people for their contributions to the development of these guidelines.

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CPT(DR)(NS) Tang Dilin	Nazirul Hannan Bin Abdul Aziz
CPT(DR) Tan Zhong Wei, Mark	

The following organisations have endorsed these guidelines:

SAF Medical Corps

Ministry of Health, Singapore

Ministry of Manpower, Singapore

Academy of Medicine, Singapore

DSO National Laboratories

Army Medical Services

Singapore Sports Council

Sport Medicine Association, Singapore

College of Family Physicians, Singapore

Society for Emergency Medicine in Singapore

