

Heat Stroke

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Abstract

When psychological and physiologic adaptation mechanisms become dysfunction to environment induced a period of elevated heat stress, heat-related emergencies occur. Heat-related illness may be trivial to life threatening. They represent a continuum of disorders such as heat edema, prickly heat, heat cramps, heat tetany, heat syncope, and heat exhaustion to critical disease well known as heat stroke. It can be easily found in higher temperature situation such as the hot summer in Taiwan or those who have chronic medical diseases, older than 75 years, younger than 4 years, mental illness, alcoholics, and vigorous exertion without proper training and acclimatization. Due to the high mortality is from 10 to 75 % in heat stroke, delay treatment after 2 hours onset of severe symptoms may result in poor outcome. Early recognition and prevention become more important as early actually treatment. For the above reasons, the articles about heat stroke were reviewed to help emergency physicians to be familiar with the heat related illnesses and cooling techniques and provide optimal management for such victims. (*Ann Disaster Med.* 2004;2 Suppl 2:S97-S109)

Key words: Heat Stroke; Environmental Accident; Disaster Medicine

Introduction

The increasing numbers of participants in many wilderness activities or adventure travelers in recent years result in more environmental accidents. Studies of morbidity and mortality in National Parks demonstrated an overall illness and injury rate of 9.2/100,000 visits. Heat-related illnesses are a spectrum of conditions ranging from mild wilderness to life threatening heat stroke. It was also the most important and frequent cause of wilderness-related morbidity and mortality in the United States, responsible for 7 % of wilderness-related deaths. Many military campaigns have been lost because of training

poor acclimatized troops with forced heavy physical exercises. The U.S. Army reported at least 125 deaths from heat stroke between 1941 and 1944.¹ Besides, 46 American football players died of heat stroke between 1961 and 1971.² It is also the third leading cause of deaths among American athletes. Heat-induced illnesses occur when body is unable to maintain appropriate homeostasis at the elevated ambient temperature situation. This rise in body temperature may produce heat edema, heat rash, heat cramps, heat tetany, heat syncope, heat exhaustion, or heat stroke. Due to the high mortality of heat stroke, we review articles to

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help emergency physicians recognize and learn how to manage the illness. Especially in most situations, heat illnesses may be preventable through many preventive methods and well public education with alarm for predictable heat waves.

Epidemiology

Heat-induced illness is the most frequent cause of environment-related death in the U.S. Heat-related death rates average 1/1000000 for those 5 to 44 years of age but increase to five times in the population older than 85 years. From 1979 to 1999, 8015 heat-induced fatalities were reported to the C.D.C in the United States, and 3829 among them died of heat waves (adverse weather condition). Analysis of the 3764 weather-related deaths revealed 142 deaths and 1068 deaths for those younger than 4 and older than 75, respectively. Heat-induced death rate varies significantly according to the weather condition. In definition, heat waves are 3 or more consecutive days of sustained temperature above 32.2 °C. In the heat wave years 1952 to 1955 and 1966, an average of 820 annual heat stroke deaths were reported in the United States compared with 179 deaths per non-heat wave year. Despite advances in prevention and treatment, more than 700 deaths were induced by the heat during the 1995 heat wave in Chicago.³⁻⁴ Besides, 15 high school students died in heat stroke during football competition between 1995 and 2001. We found that athletes have higher risk to heat-related illnesses in vigorous exertion without proper training, acclimatization, and fluid replacement. One California study about occupational heat injuries indicated 1128 cases seeking medical treatment in a year. There were 7 deaths, 15 %

hospital admission rate, and 40 % workers off work for several days after initial medical management. Populations at risk include: elderly (age > 75 y/o), younger (age < 4 years) with congenital CNS disease or diarrhea illness , chronic illness or take some medications that interfere with heat loss or production such as antipsychotic drugs, anticholinergics, antiparkinsonian drugs, B-blockers, calcium channel blockers, diuretics, vasodilators, alcohol, amphetamine, cocaine, and ketamine. Other infrequent situations include congenital absence of sweat glands, scleroderma, hyperthyroidism, and pheochromocytoma. In addition, individuals with history of heat stroke are at greater risk for another episode. Other risk factors are obesity, dehydration , and vigorous exertion without proper training and acclimatization. The environmental risk factors are high humidity, high temperature, and those without air-condition breaks. Mortality of heat stroke ranges from 10 to 75 %. For those with severe underlying disease or delayed treatment for more than 2 hours after the onset of severe symptoms, the rate would be higher.

Pathophysiology

Body temperature is regulated through a dynamic balance between heat production (and absorption) and heat loss.⁵ Heat is a byproduct of cellular metabolism and the mechanical work of skeletal muscle. It is gained by direct contact with hot objects and radiation from the sun. The body heat is lost in four ways:

(1) Conduction: represent direct heat transfer into cooler surrounding environment (eg. air, water, sweat, or clothes). When the surrounding environment reaches the same temperature as body skin surface, an insulator zone occurs

and conduction stops. Since conduction of heat in water is thousand times than air, the insulator zone will not be developed in the existence of water. So water or sweat is the primary determinant factor for heat loss.

(2) Convection: is an adjunct to conduction, accounting for about 15% of total heat loss. Convection fail to work when humidity and atmosphere temperature above 35% and 32.2°C, respectively.

(3) Radiation: is the primary method when surrounding temperature lower than body temperature and responsible for 60% body heat loss.

(4) Evaporation of sweat into the surrounding air: is the primary way of heat loss when environmental temperature higher than body. Even without sweating, respiratory and skin evaporation are responsible for 600 ml of water loss and 12 to 16 Kcal/h of heat loss per day. It accounts for about 25% of heat loss in cooler situations and reaches nearly 100% at high temperature weather. The limitations are environmental humidity and whether adequate sweat amounts secretion is achieved. High humidity will decrease the evaporation efficiency. For each 1% body weight dehydration, core temperature increases 0.1 to 0.3°C. Furthermore dehydration, with fluid loss as high as 6-10% of body weight, appears to be the most common risk factors for heat illness in exercising individuals in the heat.⁶ Dehydration also compromises cardiovascular and thermoregulatory adaptation by decreasing cutaneous blood flow and sweating rate. The Olympic athletes study showed that well acclimatized individuals get sweat secreting rate exceeding the ability of water absorption via gastrointestinal tract. Then, dehydration may occur. It is well known that sweating rate may be greater than 1 L/h.

Oral rehydration alone may be not enough.

Convection and evaporation are more important than other methods of losing heat because they are regulated primarily by the body itself. Wind velocity is also related to heat removal. Heat loss is proportional to the square root of the wind velocity. But fan alone doesn't lower the rate of heat stroke. According to the above reason, lower socioeconomic status populations without home air conditioning are at higher risk of heat-related illnesses. Furthermore, taking a heat break as short as 2 hours a day will reduce the risk of heat stroke if no air-condition available.

The body have a diurnal temperature change, ranging from about 36°C in the morning to 37.5°C in the afternoon.⁵ We tend to keep core temperature between 36°C and 38°C. The regulatory mechanisms fail to work when body temperature is above 40°C or below 35°C. We can stay at the temperature between 40°C to 42°C for a short periods without severe complications. Body temperature is regulated by four primary methods including skin vasodilation, heat gain and production decreased (inhibit shivering), behavior heat control, and increased sweat secretion. The anterior hypothalamus, responsible for heat loss, receives afferent information from cutaneous (surface) receptors and internal (core) temperatures and gives efferent signal to the skin by sympathetic nerves to stimulate skin vasodilatation and sweating.⁷

Cutaneous vasodilatation increases convective heat loss.⁸ Sweating increase heat dissipation because of cooling the skin through evaporation. When the hypothalamic set point is normal and heat is produced or gained abnormally or cannot be dissipated, the condition

is known as hyperthermia. In contrast, when the hypothalamic set point elevate, it is well known as fever.⁵ Exposure to high temperatures and an elevation of body temperature will induce a number of changes in the cutaneous circulation. Skin blood flow is increased from a baseline of approximately 250 mL/minute to approximately 6 to 8 L/minute through increased cardiac output and concomitant vasoconstriction of the renal and splanchnic circulation. Cardiac output may increase nearly 3 L/min for each 1°C of temperature elevation. As the above statements, such victims may worsen the cardiovascular situation with underlying diseases at this kind of condition. Arrhythmia, worsening CHF, myocardial ischemia to infarction may be developed. This change results in an increase in heat loss above the resting state of 80 to 90 kcal/hour. Evaporation of sweat cools the skin, further enhancing heat transfer. Cutaneous dilation and sweating increase as body temperature rises until the heat dynamic balance regained and body temperature elevation stopped. Endurance athletes and heat-environment workers might reach and maintain body temperatures of 40°C (104°F) without substantial morbidity.⁹

The human body's response at hot climate to increase efficiency of heat loss is well known as acclimatization, providing maximal heat losing. Under the physiologic and biochemical adjustments, individuals will adapt heat stress that may result in others substantial morbidity or even death.¹⁰ It may take 7 to 10 days to target the adaptation. The methods are moderate exercise in a hot and dry temperature situation for 60 to 100 min per day or simple movement in the same situation for 1 to 4 hours per day. It can reach acclimatization within 2 weeks. When return to cold temperature, use the same

time for adaptation. Primary routes are:

- (1) Changing the amount and rate of sweating.
- (2) Increasing skin blood flow and cardiovascular function.
- (3) Changing the thermoregulatory set point: lower the thermal set point within the hypothalamus.

Acclimatization results in plasma volume expansion, increased renal blood flow, and increased ability to shunt blood away from non-critical circulatory areas (including the splanchnic circulation), thus improving the renal resistance to exertional rhabdomyolysis. Acclimatization will also fasten activation of the renin-angiotension-aldosterone system, enhance the kidneys and sweat glands to retain sodium and prevent volume depletion. Despite this enhanced ability to retain sodium, acclimated sweat glands can actually secrete a greater volume of sweat by secreting sweat with significantly less tonicity, making volume depletion less likely and putting the heat-stressed individuals at less risk for volume depletion or dilutional hyponatremia if fluid replacement is maintained solely with free water or other non-electrolyte-containing solution. The acclimatization plans may be useful for specific individuals such as soldier, athletes, or occupational exposure workers. Those will prevent much heat induced illness and mortality.

Different mechanisms or pathologies between exertional and non-exertional (classic) heat stroke will not make the final medical management different. Emergency physicians must search underlying drug history or illnesses to interfere heat loss or production. Obesity guys have less effective surface area and much adipose tissue (less water content amount), making heat loss ineffectively. Febrile diseases that reset thermal point also worsen the heat release

from our skin. Antipyretics may be helpful in the situation but play no role in truly environmental heat-related illness. Alcohol inhibits ADH. Confinement hyperpyrexia is a subtype of non-exertional hyperpyrexia usually encountered when children left inside the cars. It is found that temperature reach up to 54°C to 60°C within a locked car without ventilation in less than 10 minutes in hot weather. Illegal immigrants within a closed box are also the major problem in U.S.A.

Usually, heat causes damage to the body by three mechanisms: First, heat is directly damage to cells. Increasing cell temperature results in protein denaturation and interrupts critical cellular processes, leading to apoptosis and cell death. Temperatures above 41.6°C to 42°C are considered critical thermal maximum for human beings and can be expected to produce injury even in a few hours. Extreme temperatures, above 49°C, result in nearly immediate cell death and tissue necrosis.

Second, heat induces release of inflammatory cytokines including tumor necrosis factor- α , interleukin-1 (β) and interferon γ , and the anti-inflammatory cytokines IL-6, IL-10, and TNF receptors p55 and p75. The elevated temperatures seem to result in vascular endothelium injury, increased vascular permeability, activation of the coagulation cascades, and even disseminated intravascular coagulation (DIC).

Finally, combination of direct cytotoxicity and severe systemic inflammatory responses in which encephalopathy predominates early in the course of the disease.¹⁰ If left unchecked, renal failure, coagulopathy, hepatic dysfunction, and ultimately multiple organ dysfunction systems will result.

Clinical Presentation

Heat edema

Heat edema occurs when cutaneous vasodilatation and pooling of interstitial fluid in dependent extremities result in swelling of the hands and feet. It is a self-limited process for the first few days in hot situation and rarely lasts more than a few weeks. Increased secretion of antidiuretic hormone and aldosterone in response to the heat stress also contribute to some edema formation. It is easily found in non-acclimatized and elderly human or during traveling in airplanes or cars from a colder climate to a hotter environment. Pitting edema over ankles may be seen but pre-tibia area is never involved. Treatments includes elevation and application of compressive stockings or support hose of the extremities. Diuretics should avoided because of potential volume depletion, and electrolyte imbalance.

Prickly Heat

It is a maculopapular, pruritic, and erythematous rash over the clothes covered areas, which is well known as heat rash or miliaria rubra. The etiology is that sweat gland duct pores are blocked. In acute phase, it can be easily treated by antihistamines. Other preventive maneuvers are wearing clear, light, and bigger clothes than usual to facilitate heat loss. Besides, baby powder is of no value in the situation and may interfere sweating and heat loss. Chlorhexidine lotion or light cream can be the alternative treatment choice. In severely cases, damage involves deep into the dermis causing white and non-pruritic papules, which may progress into chronic dermatitis and associate with *Staphylococcus aureus* infection. Antibiotics like oxacillin or erythromycin may be useful in this kind

situation. In addition, using 1% salicylic acid three times a day can cause desquamation of the involved skin.

Heat Cramp

The illnesses are characterized by painful, involuntary, and self-limited muscle spasms, especially involved the large muscle groups such as the calves, thighs, and shoulders. It's often occurred several hours after vigorous exercise in a hot environment and during a rest period of showering. Common for non-acclimatized individuals just beginning to receive training. Hypokalemia combined with hyperventilation is also considered a predisposing factor. Dilutional hyponatremia will be developed because individuals replace sweat loss with free water without salt contents.¹¹⁻¹³ Core body temperature might be normal or elevated. Managements include resting in a cool environment and salt replacement. Oral electrolyte solution with commercially available balanced sports drinks or 650 mg sodium chloride tablet in 500 ml water (about 0.1 to 0.2% saline solution) are the choices. Rarely, intravenous normal saline is required. In addition, the pain will not be relieved by opiates alone, and occurred in the absence of other symptoms including rhabdomyolysis. Nevertheless, they can be accompanied by symptoms and signs of heat exhaustion.

Heat Tetany

It is the condition that hyperventilation syndrome occurred under the heat stress, like the typical hyperventilation presenting with paresthesia of extremities and circumoral area, respiratory alkalosis, and carpopedal spasm. It results in little pain and cramps than heat cramps.

Otherwise, more paresthesia over specific areas are noted than heat cramps. Managements include lowering the respiratory rate and resting in a cool environment.

Heat Syncope

Heat syncope results from volume depletion, peripheral vasodilatation, pooling of bloods and decreased vasomotor tone while exercising in a hot environment, with subsequent transient loss of consciousness. It affects most commonly in poorly acclimatized and elderly individuals. Recovery after supining and usually normal body temperature separate heat syncope from heat stroke. Postural hypotension might or might not be noted on presentation to the emergency department. The key points should be thorough evaluation for injuries induced by a fall, and exclude all cardiac, neurologic, or other potentially serious causes of syncope. Treatments consist of rest and oral or intravenous fluid supply in a cool environment. Education of victims to prevent damage from fall is important as well.

Heat Exhaustion

Heat exhaustion is the most common heat-related illness¹⁴ and characterized by significant volume depletion.¹⁵ Classically, heat exhaustion is classified as water depletion and salt depletion. Water depletion heat exhaustion occurs in individuals working in a hot environment without adequate water replacement. Nevertheless, salt depletion heat exhaustion takes longer time to develop and occurs during exercise in a hot environment because of heavy sweating and hypotonic solutions replacement. But most victims presenting to ED always combine with both water and salt loss. Those who have heat exhaustion will present with nonspe-

cific systemic symptoms and signs such as fatigue, headache, nausea, vomiting, weakness, malaise, dizziness, myalgia, irritability, diaphoresis and muscle cramps. Patients can also experience orthostatic hypotension or heat syncope. Physical examination usually shows core temperatures of less than 40°C and normal mental status. As physical response for heat stress, victims may present to ED with tachycardia, orthostatic hypotension, and clinical evidence of dehydration. Patients who have heat exhaustion will usually have profuse sweating almost.

Mild heat exhaustion and heat stroke are at opposite ends of the heat illness disease spectrum. We sometimes have difficulties to differentiate from them. Anhidrosis, CNS dysfunction, and body core temperature above 40°C are clues. Exertional heat stroke may initially present to ED without anhidrosis. Besides, heat stroke may occur at any core temperature especially when the patients with heat stroke had been treated before arriving to ED. If any doubt in differentiating from the two kinds of situations, the patients should be treated for heat stroke as soon as fast. In fact, heat stroke may happen if heat exhaustion left untreated. Always keep in mind that heat exhaustion is a diagnosis of exclusion. The only objective and useful method is the level of hepatic enzymes. It is found elevated in heat stroke and may contribute to the delayed complications of heat stroke.

Laboratory abnormalities, including hemoconcentration, hyponatremia or hypernatremia can also be measured. In heat exhaustion, altered mental status or seizures without combination with severe hyperpyrexia, hypoglycemia, or trauma may alert the physicians that hyponatremia maybe the primary etiology, especially if symptoms progress after

exercises stop. Therapies consist of increased heat loss by fan evaporation, rest in a cool situation, salt with fluid repletion. Oral sports drinks is acceptable in those without unstable vital signs or have severe vomiting. On the other hand, electrolytes measurement and intravenous normal saline may achieve the better efficiency if the victims are hemodynamically unstable or severe GI upset. Furthermore, avoid exercise for 2 to 3 additional days needs to be informed. Hospital admission is rarely acquired except victims of CHF or severe electrolyte imbalance.

Heat Stroke

Classic heat stroke. Classic heat stroke is typically seen in debilitated patients after several days of high environmental temperatures and humidity exposure. This condition is seen most commonly during heat waves. Individuals especially at higher risks include those who are chronic ill, the elderly, alcoholic abuse, those without air conditioning. Others taking specific drugs as previously mentioned are also at particular risks.¹⁶ Chronic and debilitating diseases such as cardiac conditions, dementia, and chronic obstructive pulmonary disease have also been found at risk.¹⁷ In addition, infants and ill, febrile children are at higher risk.¹⁸ One animal study streptozotocin-induced diabetic rats to demo that chronic illness such as DM is more easily induced to elevated striatal glutamate (represent more serious heat-shock syndrome) by high temperature exposure. So that, insulin treatment seems helpful in the prevention of heatstroke in STZ-diabetic rats.¹⁹ Two major diagnostic findings are core temperature more than 40°C and CNS dysfunction.¹⁰ Typically, victims may be anhidrosis, but absence of sweat is not the absolute diagnostic

criterion. Follow the rule might lead to a delay in the diagnosis and treatment of heat stroke with serious complication.²⁰ CNS dysfunction is usually manifested by delirium, seizures, or coma. Other clinical findings such as hallucinations, ataxia, or bizarre behavior can also be noted. Patients can also present with systemic symptoms like nausea and vomiting similar to heat exhaustion but with altered mental status. Besides, most will present with tachycardia and hypotension when arrival to ED.^{17,21} Hyperventilation, with concomitant metabolic acidosis, is also common.^{5,22} Unfortunately, more than 50% may be combined with infection at presentation. This might be explained by that increased leukocyte count with decreased circulating neutrophil phagocytic capacity and increased expression of lymphocyte adhesion molecules increase susceptibility to infections in exertional heat stroke.²³ Laboratory findings might include hemoconcentration, acute renal failure (ARF), abnormal liver function tests, hypernatremia or hyponatremia, and hypokalemia according to the degree of dehydration.^{17,20}

Exertional heat stroke. Exertional heat stroke is found more commonly in poorly acclimatized young persons involved in vigorous physical exercise under a hot environment. It can be typically found in healthy military soldiers, miners, and athletes²⁰ particularly if they lack water supply. In retrospective analyses, most of the cases can be preventable.²⁴ It presents similar symptoms as classic heat stroke, except profuse sweating,^{14,25} account of that 50% cases are still sweating at presentation. Vomiting and diarrhea are relative more common, occurring in up to two thirds of patients.²⁶ 25 % of patients will develop ARF.¹⁴ Rhabdomyolysis, ARF, and DIC are more common in victims of

exertional heat stroke. Laboratory abnormalities are similar to classic heat stroke. Hypokalemia is developed in early stages, with hyperkalemia manifesting later. Sodium levels can be normal or slightly elevated depending on the hydration status of the patients. Elevated creatinine phosphokinase (CPK) secondary to rhabdomyolysis may be seen. Those who have peak CPK levels above 10,000 IU/L are at significant risk to develop ARF.²⁷ However, low levels of CPK on initial measurement do not rule out the development of ARF.²⁸ Besides, marked lactic acidosis, hypocalcemia, and hypoglycemia can also be seen. Differ from classic heat stroke from another form have no clinical value due to result in the same treatment. The determine factor affect mortality is the underlying diseases other than the height of core temperature.^{10,29-30} Heat stroke is the diagnosis of exclusion. It include such as drug withdrawal syndrome, ethanol withdrawal, neuroleptic malignant syndrome, systemic infection, CNS infection, DKA, thyroid storm, Intracerebral hemorrhage, status epilepticus, serotonin syndrome, drug toxicity(anticholinergics , amphetamines, etc).So, clinical laboratory survey must be ordered to detect end organ damage and to rule out other etiology that may mimic heat stroke.³¹

Treatment

Remove the victim immediately from hot condition to a cool shaded area. Take off all clothing and evaluate the adequacy of airway, breathing, and circulation following as ACLS guideline suggestion. Rapid cooling the core temperature to below 40°C is the only goal and should not be delayed if any method is available. Cold water or ice water immersion should be

instituted if readily available and lower temperature more rapidly although disadvantages such as shivering, monitor lead disconnection, equipment less available, primary in young and health victims without comorbid diseases. In a study of 252 exertional heat stroke cases, ice water immersion lowered core temperature to below 39°C within 10 to 40 minutes without fatalities in this group.³² In another study, ice water immersion cooled twice as fast as evaporative cooling.³³ Alternatively, evaporative cooling by spraying water over the patient's skin and fanning the patient maybe useful in pre-hospital setting and receiving facility.³⁴ Ice packs are also frequently used in ED and placed over the neck, axillae, and groin. However, several studies have not shown a significant reduction of cooling time with their use.³⁵⁻³⁶ Finally, a core temperature should be checked with serial monitoring by an electronic rectal thermistor probe or temperature probe-equipped urinary drainage catheter during the cooling process to ensure adequate cooling and to prevent iatrogenic hypothermia and rebound of hyperthermia requiring further treatment. If patient is intubated, esophageal thermometer is another choice. If equipment is limited, use tympanic thermometer as frequent as possible every 5 to 10 minutes. Seizures and excessive shivering can be managed by using the benzodiazepine

Intravenous LR or NS at a rate of 250ml/hr is advised for most patients. Fluid challenge must be adjusted by underlying conditions, urine output, serial BP monitor, and PCWP or CVP. An animal study result demonstrates that the neuroprotective effect of hypervolaemic haemodilution by NS supply decrease cerebral ischaemia and hypoxia injury in heat stroke rats.³⁷ Another study using 3% hypertonic saline in

heat stroke rats shows a better heat stroke syndrome preventive effect than NS.³⁸ Randomized and larger clinical trials are pivotal before widely use. Dopamine causes peripheral vasoconstriction at higher doses and impaired cutaneous perfusion and heat loss. Glucose level must be checked at bedside since hypoglycemia can be noted in exertional heat stroke. Hematocrit, electrolytes, BUN, creatinine, liver function tests, CPK, coagulation studies, and urinalysis should be measured. Electrolytes and metabolic abnormalities should be corrected appropriately. Other complications such as rhabdomyolysis, ARF, DIC, or liver damage need to be evaluated and treated. The majority of patients having exertional heat stroke will recover without sequelae.²⁵ Long-term effects for heat stroke survivors are rare with adequate treatment. In a case-control study of 21 young patients suffering exertional heat stroke followed up for 6 months and tested for heat tolerance and psychological sequelae, none was found to have any abnormal findings.³⁹ Moderate to severe residual neurologic defects in classic heat stroke survivors have been reported in up to 33% of cases.²¹ Neurologic deficits are paraplegia, paresis, dysarthria, memory loss, concentration difficulty, and ataxia.²⁵ With appropriate rehabilitation, significant motor and cognitive function can be recovered over time.⁴⁰ A randomized, controlled, prospective rat model demonstrated that direct retrograde hypothermic perfusion via the external jugular vein protected the brain after heatstroke. This technique cooled the brain without significantly interfering body temperature.⁴¹ Another study showed that although pretreatment with alphatocopherol and mannitol did not prevent the heat stroke syndrome entirely, an attenuation of the

syndrome is observed.⁴² But it is still controversial and may induce osmotic diuresis and compromise dehydration.

Disposition

All heat-related illness may be discharged for outpatient follow-up except

- (1) Heat exhaustion combined with extremity age, significant electrolyte or fluid disturbance, severe underlying diseases, or end organ damage.
- (2) Heat stroke.

Prevention

Prevention remains the most important method for heat-induced illnesses. Always keep hydration, wear adequate clothing, aware of risk factors and weather prediction especially the heat wave from TV or newspaper can prevent most heat injuries. Elderly patients at risk for classic heat stroke should be evaluated frequently. Avoid strenuous physical activities during extreme hot weather with frequent heat break and adequate fluid and salt supply is essential. Overhydration should be avoided since severe hyponatremia have been reported in marathon runners, especially in women and slow runners, due to excessive fluid consumption.⁴³ White or light-colored clothing reflects radiant energy is also helpful.

Summary

The majority of heat-related illnesses can be prevented by adequate fluid and electrolytes supply and awareness of potential risk factors. Familiarity of emergency physicians and primary physicians is fundamental for early diagnosis and treatment. Public education is essential as well.

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熱休克

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摘要

當心理和生理適應機制對環境熱壓力升高失去功能時，熱急症即發生。熱引發的疾病可以從輕微至危及生命。此疾病是一個連續的過程，從熱水腫，痲子，熱痙攣，熱抽搐，熱昏厥，熱衰竭，到危急的情況，熱休克。此疾患好發於台灣炎熱夏天的高溫時或有慢性疾病者，大於75歲，小於四歲，精神疾病，酗酒者，和沒有經過良好訓練和適應之過度勞動者。因為熱休克之高死亡率可以從10%到75%，且於嚴重症狀發生之後兩小時才延遲治療將導致不好的結果。早期的認知和預防變的比確實的治療還重要。基於以上原因，本文回顧有關於熱疾患的文章已幫助急診醫師熟悉熱疾患和各種降溫的技巧，以致於能提供病患適切的處置。(Ann Disaster Med. 2004;2 Suppl 2:S97-S109)

關鍵詞：熱休克；環境意外；災難醫學

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