

Pediatric Heat-Related Illnesses

Bryan Greenfield, MD; Joel M. Clingenpeel, MD, MPH, MS.MEdL



Children are more susceptible than adults to heat-related illnesses. While heat-related conditions often are minor and self-limited, heatstroke can be fatal without early recognition and prompt treatment.

Heat-related illnesses in children encompass a wide range of disease processes—from minor conditions such as heat rash to life-threatening thermoregulatory emergencies such as heatstroke. Physiological differences in children compared to adults make them particularly susceptible to illnesses caused by heat exposure.

Pediatric heat-related illnesses can usually be prevented if appropriate precautions are taken (see “Taking Steps to Prevent Heat-Related Illnesses” box on page 250). In lieu of prevention, early recognition and treatment of heatstroke in children may drastically reduce life-threatening complications related to multisystem organ dysfunction. Management of heatstroke rests primarily on prompt initiation of rapid cooling measures and evaluation for organ dysfunction.

Case Scenarios

Case 1

An obese 10-year-old boy was brought to the ED by emergency medical services (EMS) during the first week of youth football tryouts. It was a hot day in late August, with 100% humidity and temperatures over 95°F. The patient, who weighed approximately 240 lb, was trying out for football but had no previous athletic-conditioning experience. Despite his obesity, he had been generally healthy and only took a

stimulant medication for attention-deficit/hyperactivity disorder (ADHD).

At approximately noon, the boy collapsed on the field and had a seizure. When the EMS technicians arrived, they administered a dose of intramuscular (IM) midazolam. Although his seizure ceased, he remained obtunded and was intubated. A rectal temperature revealed a temperature of 105.8°F and paramedics noted that while the patient felt hot, he was no longer sweating. While en route to the ED, EMS technicians removed the patient’s football uniform; placed a fan in front of him; and sprayed cool water on him in an effort to lower his body temperature. At the time of arrival to the ED, his rectal temperature was 104.9°F.

Case 2

A previously healthy 3-month-old female infant was brought to the ED by EMS after she was accidentally left in a car on a summer day with a temperature of 90°F and 100% humidity. The infant’s father said that while running errands, he had forgotten his daughter was in the car and had left her in the rear facing backseat car carrier for approximately 10 minutes. When he returned to the car, he found his daughter awake but crying inconsolably. She had sweated through her clothes, vomited, and felt very hot, so he called 911. Her initial rectal temperature was 102.2°F, and her

Dr Greenfield is a resident, department of pediatrics, Eastern Virginia Medical School/Children’s Hospital of The King’s Daughters, Norfolk. **Dr Clingenpeel** is the fellowship director of pediatric emergency medicine; and associate professor of pediatrics, Eastern Virginia Medical School/Children’s Hospital of The King’s Daughters, Norfolk.

DOI: 10.12788/emed.2016.0033

Taking Steps to Prevent Heat-Related Illnesses

Acclimatization is important in preventing most forms of heat-related illness. Children should be given ample time to acclimate to elevated temperatures and humidity, a process that for many children may require 10 to 14 days of repeated, limited heat exposure.¹⁰ Another important consideration for both pediatric athletes and infants is the provision of loose-fitting and single-layered clothing during times of extreme heat. Infants should of course never be left in an unattended vehicle.¹¹ Hydration with water and/or electrolyte-containing fluids (not soda or any other carbonated beverages) should be reinforced before, during, and after exercise on hot days.²⁸

Exercise may need to be restricted on days that are excessively hot or humid. While the wet-bulb globe temperature measurement is considered a more predictive measurement of potential risk for heat illness, the heat index is generally more accessible to coaches and pediatric caregivers. Standard guidelines recommend cancelling outdoor activities if the heat index is above 126°F.²⁹ Heat indices between 104°F to 125°F are considered dangerous for children who have not yet acclimated to these conditions, and activities should be rescheduled to a cooler time of the day if possible. Heat indices between 91°F to 104°F warrant extreme caution and close monitoring of all pediatric athletes for heat-related illnesses. Other helpful adaptations include providing shade for rest and allowing more frequent hydration breaks.³⁰

clothes were removed as she was being transported in an air-conditioned ambulance to the ED for further evaluation. Once undressed, she was noted to have an erythematous rash with multiple papules and pustules on her trunk.

Epidemiology

From 2006 to 2010, an average of 668 heat-related deaths per year occurred among people of all ages in the United States. Of these deaths, approximately 7% occurred in children younger than age 4 years (2.5% in those younger than age 1 year and 4.5% in those age 1-4 years). These figures have remained relatively stable over the last 10 years.^{1,2} Adolescents are particularly at risk for overexertion, and heatstroke is the third leading cause of death in young athletes, after traumatic and cardiac causes.³ As may be expected, most heat-related deaths (76%) occur in the southern and western regions of the United States.

Pathophysiology of Heat-Related Illnesses

The hypothalamus is the main control center for temperature homeostasis. As the core temperature rises due to either meta-

bolic or environmental causes of heat, the hypothalamus primarily acts on the autonomic nervous system to engage mechanisms of heat dissipation.⁴ Evaporation of sweat is believed to be the most important mechanism of heat dissipation in humans; however, this method becomes less effective when humidity levels are above 75%.⁵ Radiation allows heat to transfer from the skin to the air, but is reliant on a temperature gradient. Conduction can allow heat to transfer to a cooler object through physical contact (as seen with cold-water immersion), while convection utilizes air movement to transfer heat (as illustrated by fanning).⁶

Thermoregulation is disrupted when the body is unable to balance metabolic heat production and heat dissipation. Heat dissipation mechanisms are easily overwhelmed when a person is exposed to excessive heat from the environment. The resulting stress from hyperthermia can directly injure cells, leading to a cytokine storm and endothelial injury. Heat can cause proteins to denature and cells to undergo apoptosis, which, if severe, can result in multisystem organ dysfunction.⁷

Physiological Differences in Children

Several physiological differences in children compared to adults compromise their ability to manage heat exposure. Thermoregulation in infants is less developed secondary to an immature hypothalamus; therefore, they are less able to utilize compensatory mechanisms to dissipate heat.⁸ In addition, infants and young children have a decreased sweating capacity, which makes evaporative cooling less effective.⁹ Children also produce more endogenous heat per kilogram than adults, which is believed to be secondary to a higher basal metabolic rate. They have less blood volume than adults, which decreases their ability to transfer warm blood into the periphery in order to cool the central core. Lastly, children have a higher surface area-to-body mass ratio, which causes in-

creased heat absorption. All of these factors ultimately result in a slower rate of acclimatization in children compared to adults.¹⁰

Environmental Factors

Several environmental risk factors predispose children to heat-related illnesses. Infants are completely dependent on their caregivers for hydration and environmental protection from the heat. Infants who are over-bundled or left in a hot car are particularly at risk for heat-related illnesses.¹¹ Older children are at risk for sports-related overexertion and typically must depend on permission from a coach or supervising adult to hydrate or take a break from exercise. Lastly, medications such as stimulants frequently prescribed for ADHD or medications with anticholinergic properties (secondary to decreased sweating) can predispose children to heat intolerance.¹²

Minor Heat-Related Illnesses

Heat-related illnesses range from benign conditions (eg, heat rash) to life-threatening processes (eg, heatstroke).

Miliaria Rubra

There are several forms of miliaria. Miliaria rubra, also known as heat rash or prickly heat, is a common, benign manifestation of heat exposure in infants and young children. A combination of heat exposure and obstructed sweat glands results in a pruritic, erythematous rash with papules and pustules (**Figure**). This is often seen in areas of friction from skin rubbing against skin or clothing.¹³

Heat Edema/Heat Cramps

Heat edema is another benign process related to heat exposure that generally occurs in older adults but can also occur in children. It is the result of peripheral vasodilation as the body attempts to shunt warm blood to the periphery.¹⁴ Heat cramps are a common manifestation in young athletes exercising in hot, summer conditions. Al-

though benign, the cramps are very painful spasms that often affect large muscle groups, particularly in the legs, such as the calves, quadriceps, and hamstrings. There is conflicting data regarding the underlying cause of heat cramps. Many believe there is a significant component related to dehydration, while others attribute the cramps to fatigue or a combination of the two.¹⁵

Heat Syncope

Heat syncope secondary to peripheral vasodilation, and venous pooling occurs as the body attempts to dissipate heat by transferring warm blood to the periphery. Relative dehydration plays a role in heat syncope, which is often precipitated by a rapid change in positioning during exercise, such as moving from a sitting to standing position. Heat syncope usually improves after the patient is supine, and children with heat syncope do not have an elevation in core body temperature.¹⁴ Some



Figure. A child with miliaria (also known as miliaria rubra, sweat rash, heat rash, or prickly heat).

© Science Source

patients who experience heat syncope, however, may also have heat exhaustion.

Heat Exhaustion

Heat exhaustion occurs in patients with a known heat exposure. As opposed to the previously described processes, heat exhaustion is characterized by a body temperature elevated up to 104°F. Heat exhaustion is often accompanied by diffuse, nonspecific symptoms such as tachycardia, sweating, nausea, vomiting, weakness, fatigue, headache, and mild confusion. Dehydration often plays a significant role in heat exhaustion, but in contrast to heatstroke (described in the following section), mentation is normal, or there is a transient, mild confusion.¹⁶

Heatstroke

Heatstroke is observed in patients with a known heat exposure who have a temperature greater than 104°F accompanied by central nervous system (CNS) dysfunction.¹⁴ The CNS dysfunction involves an alteration in mental status manifested by slurred speech, ataxia, delirium, hallucinations, or seizure activity. In severe cases, obtundation or coma may result in airway compromise.¹⁷ Vital signs are unstable, and tachycardia and hypotension are often present. Patients with heatstroke may stop sweating, although the absence of sweating is not required for the diagnosis. Other nonspecific findings such as vomiting and diarrhea are common.⁶

The hallmark of heatstroke is multisystem organ dysfunction, which is caused by heat-induced tissue damage resulting in a systemic inflammatory response.¹⁸ Since the pediatric brain is particularly sensitive to temperature extremes, cerebral edema and herniation are potential complications of heatstroke.¹⁷ Damage to myocardial tissue, coupled with dehydration and systemic vasodilation, results in hypotension and poor systemic perfusion.¹⁹ Muscle breakdown causes rhabdomyolysis that can lead to kidney failure and hepatic injury. Degradation

of clotting factors disrupts the clotting system and can cause disseminated intravascular coagulation (DIC).²⁰ Damage to the mucosal lining of the intestines may result in ischemia and massive hematochezia.²¹

Heatstroke is classified as either nonexertional or exertional. Nonexertional heatstroke occurs most frequently in younger children who are exposed to a hot environment, such as an infant left in a car on a warm day. Exertional heatstroke occurs primarily in children exercising on a hot day, such as young athletes.⁶

Due to its significant morbidity and mortality, heatstroke is the most concerning manifestation of excessive heat exposure. The mortality rate for children with heatstroke is significantly lower than for adults; however, approximately 10% of children with heatstroke will not survive,²² and 20% will have long-term neurological disabilities, including permanent impairment in vision, speech, memory, behavior, and coordination.²³

Management of Minor Heat-Related Illnesses

For most minor heat-related illnesses, supportive care is the mainstay of treatment (Table).

Miliaria Rubra

Infants with miliaria rubra typically improve once they are placed in a cool environment and their clothing is removed. In infants, lotions may contribute to sweat gland obstruction and should be used sparingly.¹³

Heat Edema/Heat Cramps

Similarly, heat edema generally improves once the child is removed from the hot environment and the extremities are elevated.¹⁴ Heat cramps are likely the result of fatigue and dehydration; therefore, these painful contractions often improve with rest, stretching, oral hydration, and removal from the hot environment. If cramps persist despite these measures, parenteral

Table. Heat-Related Pediatric Illnesses: Signs, Symptoms, and Treatment

Condition	Signs/Symptoms	Treatment
Miliaria rubra	Pruritic, erythematous rash with papules and pustules often seen in areas of skin friction.	Place patient in a cool environment, remove excess clothing, and avoid lotions.
Heat edema	Swelling that often affects the lower extremities due to peripheral vasodilatation.	Remove patient from hot environment and elevate the extremities.
Heat cramps	Painful spasms that often affect large muscle groups.	Patients usually improve with rest, stretching, oral hydration, and removal from the hot environment. If cramps persist, parenteral rehydration (20 mL/kg of normal saline) may be beneficial.
Heat syncope	Transient loss of consciousness, often related to dehydration and rapid changes in positioning during exercise (eg, moving from a sitting to standing position).	Remove patient from hot environment. Oral rehydration with salt-containing fluids is safe; patients may require IV rehydration with normal saline if orthostatic hypotension does not improve with oral rehydration alone.
Heat exhaustion	Body temperature elevated up to 104°F and usually accompanied by tachycardia, sweating, nausea, vomiting, weakness, fatigue, headache, and mild confusion. Dehydration often plays a significant role. Mentation is normal (in contrast to heatstroke).	Cessation of exercise with removal from hot environment. Oral rehydration with salt-containing fluids is important; most patients improve with these measures alone. Patients who do not improve require emergent evaluation. Check electrolytes; patients typically warrant a 20 mL/kg IV bolus of normal saline. Obtain complete neurological examination and a rectal temperature at initial presentation.
Heatstroke	Occurs in patients with a known heat exposure who have a temperature >104°F accompanied by central nervous system dysfunction (eg, slurred speech, ataxia, delirium, hallucinations, seizure activity). In severe cases, obtundation or coma results in airway compromise. Vital signs are unstable with tachycardia and hypotension. Patients may stop sweating (this is not required for the diagnosis). Vomiting and diarrhea are common. Multisystem organ dysfunction is hallmark of heatstroke.	Support the airway, breathing, and circulation with intubation, if necessary, and initiate fluid resuscitation. Initiate rapid cooling measures immediately with cool water spray, air conditioning, and ice packs (placed along the neck and axilla). Avoid medications such as acetaminophen, dantrolene, and ibuprofen. Benzodiazepine (eg, lorazepam 0.1 mg/kg IV) can be given for seizure activity. Evaluate for multisystem organ dysfunction with imaging (chest X-ray and head CT) and laboratory assessment (CBC, PT/PTT/INR, fibrinogen, CMP, UA, CPK, ABG). Admit to ICU for further management of multisystem organ dysfunction.

Abbreviations: ABG, arterial blood gas; CBC, complete blood count; CMP, comprehensive metabolic profile; CPK, creatine phosphokinase; CT, computed tomography; ICU, intensive care unit; INR, international normalized ratio; IV, intravenous; PT, prothrombin time; PTT, partial thromboplastin time; UA, urinalysis.

rehydration (20 mL/kg of normal saline) may be beneficial.¹⁵

Heat Syncope

Patients with orthostatic hypotension from heat syncope usually improve once they are resting in a cool environment and have been rehydrated. Pediatric oral rehydration with salt-containing fluids, such as commercial sports drinks, is safe; nonetheless, these patients may require intravenous (IV) rehydration with normal saline if orthostatic hypotension does not improve with oral rehydration alone.¹⁴

Heat Exhaustion

Differentiating heat exhaustion from heatstroke is of utmost importance because the treatment courses vary greatly. The difference in neurological status is the most effective way of differentiating the two diseases. All patients with slurred speech, ataxia, delirium, hallucinations, or seizure activity should be treated for presumptive heatstroke until proven otherwise (see “Management of Heatstroke” section).

Although children with heat exhaustion may have mild confusion, this tends to be transient and resolves with supportive care. Patients with heat exhaustion should stop exercising and be placed in a cool environment without excess clothing. Oral rehydration with salt-containing fluids is important, and most patients improve with these measures alone.

Children with apparent heat exhaustion who do not improve should be evaluated in the hospital setting, and laboratory studies should be obtained to evaluate for electrolyte abnormalities. Such patients typically warrant a 20 mL/kg IV bolus of normal saline. A complete neurological examination and a rectal temperature should be obtained on initial presentation.¹⁶

The evaluation of an overbundled infant with hyperthermia may be particularly challenging. Studies have demonstrated that it is possible for an infant to develop core temperature elevation if overbundled

and placed in a warm environment.²⁴ Nonetheless, it is important to address these patients with a broad differential diagnosis in mind, and always consider the possibility of sepsis. If the history and examination are consistent with hyperthermia secondary to heat exposure, a period of observation with supportive care may be a reasonable option. Infants should have a rectal temperature assessed every 15 to 30 minutes to monitor for improvement; if they improve with supportive care alone, a septic evaluation can be potentially avoided. Antipyretics will confuse the clinical picture and should be avoided in this situation.²⁴

Management of Heatstroke

Significant morbidity and mortality are associated with heatstroke, and prompt recognition and initiation of therapy are required to prevent or minimize serious complications.²² As in any other life-threatening condition, the initial treatment of heatstroke requires support of the airway, breathing, and circulation. Patients are often neurologically unstable and cannot protect their airway, which should prompt endotracheal intubation. Children who are tachycardic and hypotensive should be resuscitated with normal saline prior to intubation if oxygenation and ventilation are maintained with supplemental oxygen alone. Most patients require at least 20 mL/kg of IV normal saline but many ultimately need up to 60 mL/kg.¹⁴ If blood pressure (BP) does not respond adequately to fluid resuscitation alone, vasopressors may be necessary. Seizure activity can be managed with IV benzodiazepines, such as lorazepam (0.1 mg/kg with maximum 4 mg per dose).¹⁴

Rapid cooling therapy is the mainstay of treatment for heatstroke and should be initiated as soon as the diagnosis is suspected, since morbidity and mortality correlates directly with the duration of hyperthermia. These measures are ideally started prior to arrival at the hospital. Evaporative cooling can be achieved in the field or ambulance with a cool water spray and air condition-

ing. Additionally, ice packs can be placed along the neck and axilla to augment rapid cooling measures and can be continued in the ED until the patient's core temperature decreases to 101.4°F.²⁵

Medications have a limited role in the treatment of heatstroke. Antipyretics such as acetaminophen and ibuprofen have no proven benefit and may exacerbate hepatic, gastrointestinal, clotting, and renal dysfunction.²⁶ Benzodiazepines are helpful for seizure activity and may have a role in seizure prophylaxis. Dantrolene is not recommended for treating heatstroke as studies have not demonstrated a statistical improvement in cooling time, complications, or mortality.¹⁴ The use of chilled IV fluids instead of room-temperature fluids is not definitively supported in the literature.²⁷

Further diagnostic evaluation is directed at determining the degree of multisystem organ dysfunction that results from heatstroke. A head computed tomography (CT) scan can evaluate for cerebral edema, whereas a comprehensive metabolic profile (CMP) will screen for electrolyte abnormalities such as hyponatremia (salt loss), hypernatremia (volume depletion), and possible transaminase elevation, which may indicate hepatic injury. Prolonged coagulation studies may reveal DIC and an arterial blood gas (ABG) analysis often may reveal metabolic acidosis. A serum creatine phosphokinase (CPK) and urinalysis (UA) can help to identify rhabdomyolysis or the presence of an acute kidney injury (AKI).¹⁶

After their condition is stabilized, children with heatstroke should be monitored in the pediatric intensive care unit (PICU) to effectively address complications of multisystem organ dysfunction.

Case Scenarios Continued

Case 1

[The 10-year-old boy who collapsed during football tryouts.]

The initial evaluation revealed an obese child who was intubated and obtunded.

His vital signs included the following: rectal temperature, 104.9°F; heart rate (HR), 149 beats/minute; and BP, 82/36 mm Hg. Heatstroke was diagnosed and rapid cooling measures were initiated.

Evaporative heat loss was maintained with a fan and water spray, and ice packs were placed along the patient's groin and axillae. Laboratory evaluation included a complete blood count (CBC), CMP, CPK, UA, coagulation panel, and ABG. A normal saline IV bolus at room temperature was given and a postintubation chest X-ray confirmed appropriate position of the endotracheal tube, without any evidence of acute respiratory distress syndrome (ARDS). A head CT scan did not reveal cerebral edema. Since the child's BP and HR did not improve after the first normal saline bolus, he was given a total of 40 mL/kg of IV normal saline in the ED. The patient's laboratory results were concerning for an AKI, with elevated CPK, hepatic injury, coagulopathy, and severe metabolic acidosis. He was subsequently admitted to the PICU for further care.

The child's PICU course was complicated by multisystem organ failure, which ultimately included DIC, ARDS, acute renal failure requiring hemodialysis, and hypotension requiring vasopressors. A repeat head CT scan 3 days after admission revealed marked cerebral edema. The patient subsequently died within a week of presentation.

Case 2

[The 3-month-old girl who was left in a hot vehicle.]

The initial evaluation revealed a fussy infant with dry mucous membranes, elevated HR, and sunken fontanelle. Her rectal temperature on arrival to the ED was 100.7°F after conservative measures were taken (ie, removing her from the hot environment and removing her clothing). A peripheral IV was placed due to her clinical dehydration and she received a 20 mL/kg bolus of normal saline at room temperature. A glu-

Antipyretics such as acetaminophen and ibuprofen have no proven benefit and may exacerbate hepatic, gastrointestinal, clotting, and renal dysfunction.

cose level was obtained and was normal. The patient's rectal temperature was monitored every 30 minutes over the next 4 hours, and her temperature and HR gradually normalized.

The patient's rash appeared consistent with miliaria rubra and improved as her temperature decreased. The infant underwent a brief period of observation in the ED where she continued to look well and tolerated oral fluids without vomiting. Nei-

ther a septic work-up nor empiric antibiotics were initiated, since heat exposure was felt to be the likely source of her core temperature elevation. Child Protective Services (CPS) was notified and opened a case for further evaluation of possible child neglect. The patient ultimately returned to her baseline in the ED and was discharged home with a family member, according to the safety plan outlined by CPS, and close follow-up with her pediatrician.

References

1. Berko J, Ingram DD, Saha S, Parker JD. Deaths attributed to heat, cold, and other weather events in the United States, 2006-2010. *National health statistics reports; no 76*. Hyattsville, MD: National Center for Health Statistics; 2014. <http://www.cdc.gov/nchs/data/nhsr/nhsr076.pdf>. Accessed May 22, 2016.
2. Centers for Disease Control and Prevention(CDC). Heat-related deaths--United States, 1999-2003. *MMWR Morb Mortal Wkly Rep*. 2006;55(29):796-798.
3. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. *Circulation*. 2009;119(8):1085-1092.
4. Romanovsky AA. Thermoregulation: some concepts have changed. Functional architecture of the thermoregulatory system. *Am J Physiol Regul Integr Comp Physiol*. 2007;292(1):R37-R46.
5. Smith CJ, Johnson, JM. Responses to hyperthermia. Optimizing heat dissipation by convection and evaporation: Neural control of skin blood flow and sweating in humans. *Auton Neurosci*. 2016;196:25-36.
6. Becker JA, Stewart LK. Heat-related illness. *Am Fam Physician*. 2011;83(11):1325-1330.
7. Aggarwal Y, Karan BM, Das BN, Sinha RK. Prediction of heat-illness symptoms with the prediction of human vascular response in hot environment under resting condition. *J Med Syst*. 2008;32(2):167-176.
8. Charkoudian N. Human thermoregulation from the autonomic perspective. *Auton Neurosci*. 2016;196:1-2.
9. Wendt D, van Loon LJ, Lichtenbelt WD. Thermoregulation during exercise in the heat: strategies for maintaining health and performance. *Sports Med*. 2007;37(8):669-682.
10. Falk B, Dotan R. Children's thermoregulation during exercise in the heat: a revisit. *Appl Physiol Nutr Metab*. 2008;33(2):420-427.
11. Booth JN 3rd, Davis GG, Waterbor J, McGwin G Jr. Hyperthermia deaths among children in parked vehicles: an analysis of 231 fatalities in the United States, 1999-2007. *Forensic Sci Med Pathol*. 2010;6(2):99-105.
12. Levine M, LoVecchio F, Ruha AM, Chu G, Roque P. Influence of drug use on morbidity and mortality in heatstroke. *J Med Toxicol*. 2012;8(3):252-257.
13. O'Connor NR, McLaughlin MR, Ham P. Newborn skin: part I. Common rashes. *Am Fam Physician*. 2008;77(1):47-52.
14. Howe AS, Boden BP. Heat-related illness in athletes. *Am J Sports Med*. 2007;35(8):1384-1395.
15. Bergeron MF. Muscle cramps during exercise – Is it fatigue or electrolyte deficit? *Curr Sports Med Rep*. 2008;7(4):S50-S55.
16. Glazer JL. Management of heatstroke and heat exhaustion. *Am Fam Physician*. 2005;71(11):2133-2140.
17. Sharma HS. Methods to produce hyperthermia-induced brain dysfunction. *Prog Brain Res*. 2007;162:173-199.
18. Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. *J Appl Physiol*. 2010;109(6):1980-1988.
19. Wilson TE, Crandall CG. Effect of thermal stress on cardiac function. *Exerc Sport Sci Rev*. 2011;39(1):12-17.
20. Chapin JC, Hajjar KA. Fibrinolysis and the control of blood coagulation. *Blood Rev*. 2015;29(1):17-24.
21. Lambert GP. Intestinal barrier dysfunction, endotoxemia, and gastrointestinal symptoms: the 'canary in the coal mine' during exercise-heat stress? *Med Sport Sci*. 2008;53:61-73.
22. Jardine DS. Heat illness and heat stroke. *Pediatr Rev*. 2007;28(7):249-258
23. Argaud L, Ferry T, Le QH, et al. Short- and long-term outcomes of heatstroke following the 2003 heat wave in Lyon, France. *Arch Intern Med*. 2007;167(20):2177-2183.
24. Cheng TL, Partridge JC. Effect of bundling and high environmental temperature on neonatal body temperature. *Pediatrics*. 1993;92(2):238-240.
25. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. *Crit Care*. 2007;11(3):R54.
26. Walker JS, Hogan DE. Heat emergencies. In: Tintinalli JE, Kelen GD, Stapczynski S. *The American College of Emergency Physicians, eds. Emergency Medicine: A Comprehensive Study Guide, Section 15*. China: The McGraw-Hill Companies, Inc; 2004:1183-1189.
27. Smith JE. Cooling methods used in the treatment of exertional heat illness. *Br J Sports Med*. 2005;39(8):503-507.
28. Rowland T. Fluid replacement requirements for child athletes. *Sports Med*. 2011;41(4):279-288.
29. National Weather Service, National Oceanic and Atmospheric Administration: NWS Heat Index. http://www.nws.noaa.gov/om/heat/heat_index.shtml. Accessed May 19, 2016.
30. Council on Sports Medicine and Fitness and Council on School Health; Bergeron MF, Devore C, Rice SG; American Academy of Pediatrics. Policy statement—Climatic heat stress and exercising children and adolescents. *Pediatrics*. 2011;128(3):e741-e777.