Chapter 7

CLINICAL DIAGNOSIS, MANAGEMENT, AND SURVEILLANCE OF EXERTIONAL HEAT ILLNESS

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INTRODUCTION TO THE PATHOPHYSIOLOGY OF EXERTIONAL HEAT ILLNESS

Exertional heat illness (EHI) encompasses a spectrum of disorders deriving from the combined stresses of exertion and thermoregulation (Figure 7-1). These include exertional dehydration, heat cramps, heat exhaustion, heat injury, heatstroke, rhabdomyolysis, acute renal failure, and hyponatremia.[1–4] Early in the course of EHI it may be difficult or impossible to distinguish these entities and, in fact, they often overlap and are differentiated as the clinical manifestations evolve. They represent primarily a continuum of multisystem illnesses related to elevation of body core temperature and the metabolic and circulatory processes (including changes in fluid and electrolyte balance) that are brought about by exercise and the body's thermoregulatory response.[5–9]

Heat dissipation occurs primarily at the skin. The blood carries body thermal energy to the skin, where the heat is dissipated through conduction, convection, radiation, and evaporation. A large temperature differential between the skin and its surroundings provides efficient heat dissipation under cool conditions.[10,11] However, under hot conditions there is inefficient heat dissipation, and blood flow to the skin increases dramatically, even at rest, thereby requiring a large increase in cardiac output.[12–14] With strenuous exercise there is a dramatic (3- to 6-fold) increase in cardiac output owing to increased blood flow to exercising muscle (Figure 7-2). Exercise elevates body temperature because at least three fourths of energy released during exercise is converted to heat. As body temperature rises, more blood flows to the skin for heat dissipation. These circulatory demands of sustained exercise and heat stress may also encroach on visceral blood flow to the extent of producing organ dysfunction or cellular injury (eg, watery/bloody diarrhea often seen in marathon runners, and perhaps acute renal failure and encephalopathy often seen in EHI).[15,16]

Heat exposure and regular strenuous exercise produce *heat acclimatization*, which improves the body's response to heat stress within a few days. Most of the physiological improvement in heat tolerance occurs within 10 days of combined heat exposure and regular exercise.[17–22] In acclimatized individuals blood volume increases, stroke volume increases, the heart rate is lower, metabolic generation of heat decreases slightly, sweating begins ear-

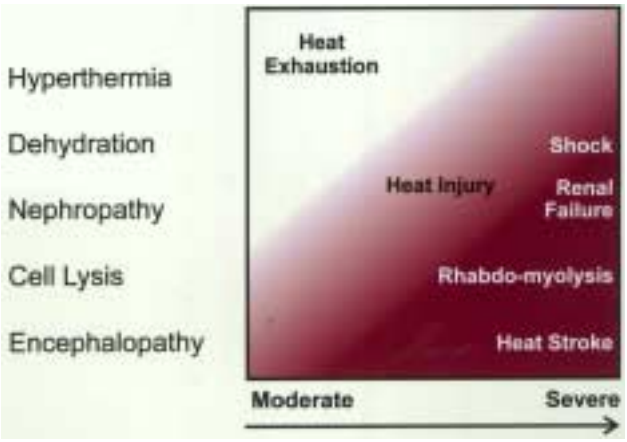

Fig. 7-1. A diagrammatic depiction of the spectrum of exertional heat illness, encompassing the continuum of mild (heat exhaustion), moderate (heat injury), and severe (heatstroke and other syndromes) in terms of severity in each area of physiological dysfunction: hyperthermia, dehydration/cardiovascular dysfunction, nephropathy, cell lysis (muscle or liver tissue damage), and encephalopathy. The horizontal arrow depicts severity of specific symptoms and signs corresponding to the categories of dysfunction on the left. The degree of shading depicts the severity of overall illness.

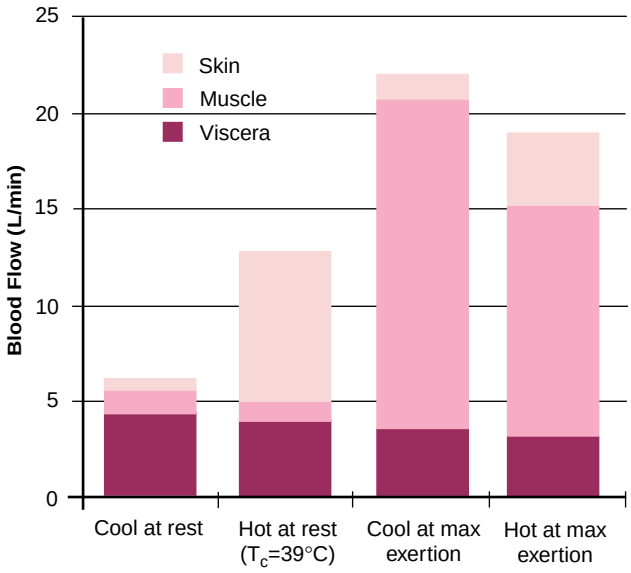

Fig. 7-2. Estimated distribution of blood flow to the skin, muscle, and viscera as fractions of cardiac output, under cool and hot conditions, at rest, and at maximum exertion. Adapted with permission from Gardner JW, Kark JA. Heat-associated illness. In: Strickland GT, ed. *Hunter's Tropical Medicine*. 8th ed. Philadelphia, Pa: WB Saunders; 2000: 141.

lier with a higher sweat rate and lower sodium content, and the threshold for cutaneous vasodilation is reduced.[11,23–32] These changes improve transfer of body heat from the core to the skin and enhance heat dissipation at the skin. Although sodium is conserved with heat acclimatization, water losses are not reduced because sweat volume increases.

Wind, solar radiation, and humidity play important roles in the efficiency of heat dissipation through convection, radiation, and evaporation. When the surrounding temperatures are at or above that of the skin (about 36°C [96°F]), then evaporation is the only mechanism for cooling. As more body heat must be dissipated, these fluid losses increase due to heavier *sweating*, which may exceed 2 L/h (up to a maximum of 12–15 L/d).[9,32] In hot and humid environments there is inefficient heat loss through convection and evaporation.[33] The resultant increased body core temperature induces heavier sweating, but dripping sweat provides minimal heat exchange, resulting in large fluid losses with minimal impact on core temperature reduction.[34]

As strenuous exercise produces more body heat than can be efficiently dissipated, core temperature continues to rise, sweating continues at maximal rates, and dehydration (ie, reduction of total body water, with some resultant reduction in plasma volume) rapidly develops, compounding the cardiovascular and metabolic stresses of exercise and thermoregulation.[35–37] These stresses may combine to produce orthostatic hypotension,[38] electrolyte imbalance,[8,39] liver and muscle cellular damage,[40] acute renal failure,[39,41] disseminated intravascular coagulation (DIC),[42–45] and neurological disturbances.[1,46] The spectrum of illness is thus quite broad, portraying the effect of these stresses on a variety of body functions.

Hyponatremia is an unusual consequence of excessive hydration in an overly aggressive attempt to prevent or treat EHI. Hyponatremia in this circumstance seems to be due to failure of the kidneys to excrete excess water. The problems of hyperhydration and hyponatremia are discussed later in this chapter and also in the chapter by Montain, Hydration, in Volume 3 of *Medical Aspects of Harsh Environments.*

CLINICAL FEATURES OF EXERTIONAL HEAT ILLNESS

We include all of the exertion-related heat illness syndromes within the term EHI. These syndromes form a continuum of multisystem illnesses, which may be divided into three main levels:

1. *mild* EHI, which includes heat exhaustion, mild dehydration, and heat cramps;
2. *intermediate* EHI, which includes exertional heat injury and mild rhabdomyolysis, renal insufficiency, orthostatic hypotension, heat-related syncope, and reversible electrolyte and metabolic disturbances; and
3. *severe* EHI, which includes heatstroke, severe rhabdomyolysis, liver necrosis, acute renal failure, cardiovascular collapse, and marked electrolyte or metabolic disturbances.

In addition, there are some minor heat-related syndromes, not part of the EHI continuum, which include heat rash (prickly heat), heat edema, parade syncope, exertion-associated collapse, and sunburn. There has been no agreement on criteria for separating EHI into levels of severity, so precise definitions are a matter of personal choice. Differentiation of heat syndromes by body core temperature has been popular, but this is unreliable because the severity of organ dysfunction correlates poorly with the maximum body temperature.

Exhibit 7-1 lists the wide variety of symptoms and signs associated with EHI. Early in the course of illness, slow mentation with impaired judgment, weakness, fatigue, headache, thirst, hyperventilation, muscle symptoms, or gastrointestinal symptoms may predominate. As the illness becomes worse, orthostatic symptoms develop such as faintness, ataxic gait, visual disturbances (eg, blurred vision, narrowed or tunnel vision, scotomata), and collapse.[1,47–50] Collapse may occur with or without loss of consciousness. Most severe symptoms are also accompanied by extreme mental status changes.[8,51] The major effects of EHI on clinical status are related to

- water and electrolyte loss,
- encephalopathy and other neurological problems,
- impaired renal function,
- muscle cell damage (rhabdomyolysis) with potential progression to the cell lysis syndrome,
- unusual complications, and
- consequences of life-threatening complications.

Rather than emphasizing distinct syndromes it is best to anticipate these manifestations, assess the appropriate level of monitoring and treatment needed for each, and derive a management plan

EXHIBIT 7-1

CLINICAL FEATURES OF EXERTIONAL HEAT ILLNESS

Nonspecific Symptoms

Thirst	Myalgia	Poor concentration	Hysteria	Vomiting
Weakness	Cramps	Impaired judgment	Headache	Diarrhea
Fatigue	Hyperventilation	Anxiety	Nausea	

Progressive Orthostatic Signs and Symptoms

Mild Symptoms

- Faintness
- Dizziness (not vertigo)
- Wobbly legs
- Stumbling gait
- Visual: blurred vision, tunnel vision, scotomata, blackout
- Collapse (without loss of consciousness)

Exertional (Heat) Syncope

- Collapse with brief loss of consciousness (< 3 min)

Orthostatic or Sustained Hypotension

- Evaluated via tilt tests

Shock or Cardiovascular Collapse

Common Presentations

Mild Exertional Heat Illness

- Nonspecific symptoms, mild orthostatic signs and symptoms
- Brief nonfocal encephalopathy, cooperative with medical care
- Hyperthermia clears immediately with cooling and rehydration
- Mild elevated creatinine without oliguria
- Mild elevated muscle enzymes

Intermediate Exertional Heat Illness

- Exertional syncope with electrolyte disturbance
- Nonfocal encephalopathy, uncooperative with medical care, amnesia
- Hyperthermia > 104°F
- Elevated creatinine with oliguria that responds to rehydration
- Elevated muscle and liver enzymes, mild acidosis

Severe Exertional Heat Illness

- Persistent hypovolemia or shock, metabolic acidosis
- Encephalopathy with delirium, obtundation, or coma
- Hyperthermia > 106°F
- Acute renal failure
- Rhabdomyolysis with life-threatening complications

Uncommon Clinical Problems

- Complications including ischemia, hypoxemia, and infection
- Hyponatremia
- Midline cerebellar speech or gait ataxia
- Seizures
- Hypoglycemia
- Gastrointestinal bleeding
- Liver failure or necrosis
- Pulmonary edema
- Cardiac arrhythmia
- Disseminated intravascular coagulation

based on the potential development of these problems.

The differential diagnosis of symptoms associated with EHI is broad and varies with locality and time. Most of the clinical findings associated with EHI are also found in other diseases. These diseases may provoke or accompany EHI, thus increasing the severity of the illness and the risk of serious complications. Infectious diseases are likely to provoke EHI by contributing to dehydration and hyperthermia. It is particularly important to consider meningitis, sepsis, pneumonia, myocarditis, viral infections, asthma, drugs and toxins, sickle cell disease, and cardiovascular or cerebrovascular disease.[1,4,50] The differential diagnosis list for patients with high body temperature also includes malaria, Rocky Mountain spotted fever, other infections, anticholinergic poisoning, neuroleptic malignant syndrome, and thyroid storm.[39]

Hyperthermia

In EHI, the elevated body temperature is not referred to as fever but as hyperthermia.[2] *Fever* regulates the body core temperature to a higher value (100°F–106°F [38°C–41°C]) as a result of change in the thermoregulatory set point (usually in response to inflammation). Heat production increases only temporarily to achieve the new set point temperature. Attempts to cool the patient (except by administering antipyretic drugs, which return the set point toward normal) are resisted by the patient's thermoregulatory responses (cutaneous vasoconstriction, shivering); and if attempts to cool the patient are successful, the body core temperature will rise again once cooling is stopped.[11]

On the other hand, *hyperthermia* refers to any significant elevation of core temperature above its normal level, and fever is only one of three ways in which hyperthermia may be produced. Hyperthermia is also a normal accompaniment of sustained exercise, and during exercise the elevation of core temperature is the chief stimulus that elicits the thermoregulatory, heat-dissipating responses that are necessary to dissipate the heat produced by the exercise. Thus, during exercise an equilibrium is achieved in a hyperthermic state. (The differences between fever and the hyperthermia of exercise are discussed in greater detail in Chapter 2, Human Adaptation to Hot Environments.) Finally, hyperthermia results if the sum of heat production and heat gain from the environment exceeds the body's level of heat dissipation to the environment. Such a situation may result from physiological factors (eg, impaired ability to sweat or to increase skin blood flow) or environmental factors (eg, extreme environments or the wearing of heavy impermeable clothing), and is a common circumstance in which EHI develops.

In EHI, hyperthermia is not the result of an elevated set point, even though there is evidence that inflammatory processes may be involved (see Chapter 5, Pathophysiology of Heatstroke). Patients with EHI usually do not shiver during cooling (unless they are overcooled), and once the heat source is eliminated and cooling is completed, a rise in body temperature is uncommon. Recurrence of hyperthermia without an added heat source implies that inflammatory processes have progressed.

While severe hyperthermia (≥ 106°F [41°C]) is sometimes considered a distinctive or defining feature of exertional heatstroke, it is more appropriate to consider it to be a common feature of the more severe forms of EHI. Survival has been reported in a patient with maximum rectal temperature of 116°F [46.5°C].[52] However, severe cases of EHI also occur at lower body core temperatures,[53–55] and many patients with high rectal temperature do not manifest the severe neurological symptoms or organ damage that define heatstroke. We have seen considerable dissociation between hyperthermia and signs of nonfocal encephalopathy that characterize exertional heatstroke. In one series of 468 cases of EHI in Marine Corps recruits, only half of the casualties with neurological symptoms consistent with heatstroke (delirium, obtundation, or coma) had maximum rectal temperatures of 106°F (41°C) or higher.[56] And half of the casualties with maximum rectal temperature of 106°F (41°C) or higher had milder forms of EHI inconsistent with heatstroke.

Dehydration and Electrolyte Imbalance

Volume depletion, with loss of water and salt, is one of the fundamental features of EHI. Both the physical signs and the laboratory abnormalities that are characteristic of volume depletion are relatively insensitive and largely nonspecific. The clinician often assumes the presence of mild-to-moderate volume depletion based on the clinical situation without confirmatory findings, and observes the patient's response to a trial of fluid and salt replacement to make the diagnosis retrospectively. Interstitial volume depletion may result in dry oral mucous membranes, loss of skin turgor (tenting), and dry tongue with longitudinal furrows. Signs of plasma volume depletion are orthostatic hypotension, syncope, recumbent hypotension, and shock; as well as lethargy, fatigue, loss of mental concentration, confusion, and obtundation. Moderate or severe

volume depletion produces oliguria. Important laboratory signs of water depletion include hypernatremia, hyperosmolality, elevated blood urea nitrogen (BUN) to creatinine ratio, elevated hemoglobin concentration and hematocrit, and high urine specific gravity.

The most common situation seen in EHI is a patient who is mildly dehydrated from prior heat stress and who then undertakes heavy exercise without adequate replacement of water losses. This usually results in hypernatremic hyperosmolar dehydration, which in its earlier stages is adequately treated with water and rest in a cool area. More severe cases (particularly those who have suffered exertional syncope, exhibit orthostatic hypotension, or have exertional heat cramps) benefit from intravenous infusion of 1 to 3 liters of half-normal saline. In our studies of EHI in Marine Corps recruits at Parris Island, South Carolina, about one third of episodes presented as exertional syncope during or immediately after strenuous exercise.[56–58]

Syncope must be distinguished from sustained obtundation, coma, or, rarely, a seizure. Guidelines for making this assessment are given in Exhibit 7-2. *Syncope* is characterized by its brief duration; frequent recall of onset; absence of abnormal tone, posture, or movement during unconsciousness; and abrupt return to consciousness. Antegrade amnesia lasting beyond 3 minutes after the collapse suggests a neurological injury other than uncomplicated syncope, while amnesia retrograde to collapse does not differentiate the cause of collapse. *Seizure* is often characterized by a stereotypic aura or prodrome, with incontinence, abnormal eye position, clonic–tonic movements, focal deficits, and a postictal stage with amnesia lasting beyond unconsciousness. *Coma* is recognized by unresponsiveness for more than 3 minutes following collapse, without the characteristic signs of seizure.

Other electrolyte problems include hypokalemia or hyperkalemia, low bicarbonate levels, and hyponatremia. Hypokalemia of uncertain etiology has been found in about one third of large case series of exertional heat stroke, and is a factor in the development of rhabdomyolysis.[43,50,59–62] Hyperkalemia results from acute renal failure, tissue necrosis, and metabolic acidosis. Lactic acidosis produces low bicarbonate. And hyponatremia usually results from extreme overhydration (discussed below).

Dehydration is most often associated with both

EXHIBIT 7-2

EVALUATION OF LOSS OF CONSCIOUSNESS

Syncope:	Nearly always occurs when upright
	Skin becomes pallid or ashen gray
	Brief prodrome (giddiness, swaying, loss of vision, nausea)
	Victim recalls onset as start of visual black out or fall
	Recovery within seconds to 3 minutes
	Abrupt return to full consciousness
	Amnesia only from time of collapse to awakening
Seizure:	Not dependent on erect posture
	Often starts with stereotypic aura or prodrome
	Often stereotypic posture or movement
	Bilateral stereotypic movements always accompanied by coma
	Incontinence (urinary more often than fecal)
	Rolling up of eyes, biting of tongue, foaming at mouth
	Often unconscious for > 5 minutes, with gradual recovery
	Postictal state with lethargy and confusion for minutes to hours
	Amnesia from prodrome through much of postictal stage
Coma:	Prolonged unresponsiveness lasting > 3 minutes
	Slow recovery without treatment (hours to days)
	If due to exertional heat illness, may reverse in minutes with cooling and hydration

water and salt depletion, so that most patients have nearly normal serum sodium levels.[63] Dehydration may be due to isotonic or hypotonic fluid loss.[1,8,30,64] Water losses are universal, but salt loss may be excessive in unacclimatized individuals, who excrete sweat with high salt concentration. This occurs primarily during the first 2 weeks of training in a warm environment. Acclimatized individuals excrete large volumes of sweat with low salt content, and thus hypotonic fluid losses predominate.[2] These patients need free water replacement as well as isotonic solutions. In predominant water depletion (usually from limited water consumption), there is characteristically intense thirst, hyperventilation with paresthesias, tetany, hypernatremia, and renal insufficiency. In predominant salt depletion (usually from limited salt intake), there are characteristically generalized muscle cramps, vomiting, and hyponatremia.[1,5,8]

Neurological Manifestations

Neurological symptoms are characteristic in EHI, particularly in exertional heatstroke.[3,65] These symptoms are not focal neurological deficits relating to specific cranial or peripheral nerves but represent a *generalized encephalopathy* affecting mental status. A precise description of this encephalopathy is not available in the literature. The symptoms range from lethargy and drowsiness, through confusion and disorientation, to delirium, obtundation, or coma. No generally accepted methodology for quantifying heat encephalopathy exists. However, we have developed and used an acute encephalopathy scale in our collection of research data on cases of EHI at the Parris Island Marine Corps Recruit Depot (Exhibit 7-3).[57,58] This scale was adapted from the Los Rancheros Scale,[66] the Reaction Level Scale,[67,68] and the Glasgow Coma Scale.[69]

Other neurological manifestations include frequent amnesia and, rarely, seizures or persistent ataxia. A *thorough neurological examination* is important. This consists of the following steps: assess pupillary size and response to light; accommodation; conjugate movement to six positions; opening and closing of eyes; drooping of lids; and response to pain in each hand, foot, the trunk, and both sides of the face. Assess ability to respond to voice and to move each limb, the neck, and the face. Ask the patient to perform simple and complex commands. Assess ability to respond with questions defining orientation to person, place, and time and, if needed, proceed to performance testing such as the Mini-Mental State Examination.[70] Assess higher function and speed of response in the history. Question the patient for amnesia, confusion, poor concentration, and judgment during the periods before and after the initial signs of EHI. Exhibit 7-4 suggests some questions for assessment of mental status.

Amnesia is present when loss of memory extends outside the period of loss of consciousness. With *syncope*, however, the patient usually recalls a prodrome (starting to black out or fall) and recovers abruptly and completely; loss of memory will exist for the period during the syncopal episode (< 3 min). If the patient does not remember passing out but others witnessed it, then he has amnesia. The duration of amnesia should be established (eg, patients may black out for less than a minute but be unable to recall the 10 minutes spent in getting into the ambulance and traveling to the clinic).[65] In our studies of Marine Corps recruits at Parris Island, about one third of EHI cases had a period of amnesia lasting at least 10 minutes.[56–58]

The *history of witnesses* is crucial to neurological assessment. An observer needs to report whether the person fell to the ground, fainted, suffered loss of consciousness (and its duration), was slow, lethargic, dazed, difficult to arouse, confused, disoriented, combative, obtunded, comatose, or had a seizure. The patient generally cannot reliably describe these manifestations. Many patients with EHI have mild deficits, especially slow mentation with a dazed affect, short periods of amnesia, or confusion and disorientation, which may only be documented by an appropriate history from witnesses.[65] It is very important to describe the chronological sequence of symptoms and signs and to explain the precise medical events for "falling out" of an activity (eg, stopping, collapsing to the ground, having a seizure, or fainting).

Seizures are usually related to hypoxia or late manifestations of advanced exertional heatstroke. Occasionally, seizures result from hyponatremia, hypoglycemia, or other disturbances.[71]

Impaired Renal Function

Acute renal insufficiency is a frequent component of EHI that is often relatively easily reversed. It is believed to be due to prerenal volume depletion, reduced perfusion of the kidney due to redirection of blood flow to the muscle and skin, and acute renal tubular injury from myoglobin and myoglobin casts. Clearly, the pathogenesis is more complex but the contributions of other factors, such as cytokines, have not been fully characterized. Acute renal failure is hard to detect in its early stage because there are

EXHIBIT 7-3

PROPOSED PARRIS ISLAND SCALE FOR NONFOCAL ENCEPHALOPATHY

The simplicity of the scores for the Parris Island Scale (Exhibit Table 1) allows rapid assessment of the global level of central nervous system function for patients with heat illness. The Glasgow Coma Scale[1] scores specific responses but does not assess global function. The Parris Island Scale is analogous to the Los Rancheros Scale,[2] which describes central nervous system function following closed head injury. The observations required for severe dysfunction are identical to those used in the Glasgow Coma Scale, but these are categorized as *functional* levels. To more precisely describe obtundation (ie, minimal conscious function), we adapted the validated definition of the Reaction Level Scale,[3] which also utilizes the observations required for the Glasgow Coma Scale. At higher levels of function we retain those features of the Los Rancheros Scale that apply to acute illness. During acute exertional heat illness, the time course may be so rapid that detailed evaluation of each level of encephalopathy may be difficult.

EXHIBIT TABLE 1: PARRIS ISLAND SCALE

Parris Island Scale	Level of Function	(For Comparison) Glasgow Coma Scale	Los Rancheros Scale
8	Normal: alert, oriented, cooperative	15	8
7	Drowsy or Lethargic, or Dazed Affect: slow mentation, poor concentration, fully arousable	13–15	7
6	Confused and Appropriate: incomplete orientation, cooperates with medical care	12–14	6
5	Confused and Inappropriate: disoriented, uncooperative, purposeful, directed	11–13	5
4	Delirious: disoriented, agitated, combative, out of touch, fragmented behavior, poorly directed	10–12	4
3	Obtunded: minimal mental response; can obey a command, ward off pain, utter a word, or make eye contact	9–12	3
2	Light Coma: unconscious; reflex response to pain	4–8	2
1	Deep Coma: unconscious; no response to pain	3	1

Patients with mild dysfunction present with slow mentation, poor concentration, and dazed affect (Level 7). Drowsiness is identified by droopy or intermittently closed eyelids.[3] Patients between Levels 6 and 4 may have a normal, reduced, or increased level of arousal. Assessment can be made quickly by determining orientation to person, place, and time; observing response to simple and complex commands; and noting spontaneous actions and cooperation with medical care. At Level 6 a patient has incomplete orientation but is cooperative and appropriate even for painful procedures. At Level 5 a patient is disoriented and uncooperative, but behavior is directed, purposeful, and appears related to real events. At Level 4 (delirium) a patient is disoriented and behavior is fragmented, poorly directed, and often appears unrelated to external stimuli. Combative behavior is common among young military personnel in delirium. Obtundation (Level 3) is defined as somnolence with minimal conscious function. The best performance an obtunded patient can accomplish is at least one of the following: (1) obeys a one-step command, (2) wards off pain, (3) utters a single word, or (4) makes sustained eye contact during movement. Patients in coma are unable to demonstrate any of these basic signs of mentation. Patients in light coma (Level 2) respond to pain, whereas those in deep coma (Level 1) do not.

We hope and expect that clinical investigations will show the predictive power of the Parris Island Scale in terms of patient outcome and the need for treatment.

(1) Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet*. 1974;2:81–84. (2) Hagen C. Language-cognitive disorganization following closed head injury: A conceptualization. In: Trexler LE, ed. *Cognitive Rehabilitation: Conceptualization and Intervention.* New York, NY: Plenum Press; 1982: 131–151. (3) Starmark JE, Stalhammar D, Holmgren E. The Reaction Level Scale (RLS85): Manual and guidelines. *Acta Neurochir (Wien)*. 1988;91:12–20.

EXHIBIT 7-4

QUESTIONS FOR ASSESSMENT OF MENTAL STATUS

- **Orientation:** (Normal: oriented to person, place, and time [3/3])
  1. Your name? Who are you? What are you doing here?
  2. What is the year? Season? Month? Day of week? Date?
  3. Where are you? What country? State? City? Building?
- Your last address and phone number before coming to this site?
- Could you concentrate normally and fully at the time of illness?
- Were you confused about anything while ill?
- **Amnesia:** Describe events from the start of activity to arrival here (especially just before and after any fainting episode). Include exertional activities and name three persons with you at the time of onset and describe one person traveling here with you.
- **Optional tests:**
  1. Calculate serial 7s (subtract 7s from 100: 93, 86, 79, 72, 65, …).
  2. Recall of three objects after 5 minutes.
  3. Recall of own Social Security number.

Examples of Patient Responses Indicating Confusion[1]:

"I just wasn't all there, thinking was fuzzy."
"I was getting dumb."
"I don't know where I was, I couldn't answer questions."
"I was having trouble doing simple math."
"I was disoriented, couldn't remember where I was."
"I couldn't think clearly, couldn't concentrate."
"I couldn't keep a thought in my head, forgot instructions."
"I don't remember if I went out or not, things became confused."

Source for patient response examples: (1) Carter BJ, Cammermeyer M. A phenomenology of heat injury: The predominance of confusion. *Mil Med*. 1988;153:118–126.

no symptoms or signs until sufficient time has passed for marked alteration in serum chemical levels regulated by the kidney. The most important early indicators of acute renal failure are inadequate urinary output, evidence of myoglobinuria, and elevated creatinine and BUN. After adequate rehydration, urinary output should exceed 50 mL/h and be approximately equal to fluid intake. While serum creatinine levels often increase during exercise, owing to release of creatine from injured muscle, elevations above 1.7 mg/dL are of concern.

Urinalysis is useful to determine whether there are signs of myoglobinuria (occult blood positive with pigmented casts but without red blood cells) or other problems contributing to impaired renal function. Mild proteinuria and modest abnormalities of the urinary sediment are frequently found in patients with heatstroke.[50] The presence of abnormal numbers of cells or cell casts in the urine is important. If urinary output remains inadequate after hydration, then urinary sodium and creatinine concentrations, fractional excretion of sodium, urine osmolality, and the urine-to-plasma creatinine concentration ratio are useful to distinguish between prerenal azotemia, which may be rapidly reversible, and acute renal failure, which is a standard indication for intensive hospital care.[72]

Although we generally think of *renal failure* as

part of rhabdomyolysis with myoglobinemia or heatstroke, acute renal failure can occur as the primary form of EHI without rhabdomyolysis, encephalopathy, or hyperthermia.[41,73–81] This was more common in the 1960s among military trainees who received salt tablets as prophylaxis or treatment for heat cramps.[41,73,74] Salt supplements cannot substitute for water replacement, and excessive salt intake increases water requirements. For this reason prophylactic use of salt tablets is inadvisable, and it is preferable to replace salt through regular meals.[8,50,82–84]

Muscle Symptoms and Rhabdomyolysis

Exertional heat cramps are painful, migratory skeletal muscle spasms. These appear most often in well-conditioned persons, at the end of the training day, or in the shower as muscles cool.[8,21,50] They are often attributed to salt depletion with resultant cellular hyponatremia and are rapidly reversed with water and salt replacement, usually by intravenous infusion of normal or half-normal saline. Oral salt solutions (0.5%) are also effective if the parenteral route is unavailable. Prevention is through adequate water replacement, a high-salt diet (salt tablets should not be used, particularly at the time of exercise), and slow progression of conditioning exercise.

Exertional rhabdomyolysis is the syndrome caused by skeletal muscle damage with release of cellular contents into the circulation, including myoglobin, potassium, phosphate, creatine kinase (CK), lactic acid, and uric acid.[85–87] Manifestations of rhabdomyolysis can vary from asymptomatic elevation of serum skeletal muscle enzymes to muscle weakness, pain, tenderness, and stiffness with associated myoglobinuria with or without acute renal failure.[77,88–91] In its most severe form obvious muscle necrosis can be demonstrated, but marked laboratory abnormalities can occur without extensive cell necrosis. Severe rhabdomyolysis may present without early muscle pain or tenderness, and muscle numbness may be the only symptom in the first few hours (documented in approximately one third of severe cases).[92,93]

Physical assessment of strength of major muscle groups and search for areas of numbness are important. Mild rhabdomyolysis with asymptomatic elevation of serum skeletal muscle enzymes is the most common laboratory abnormality in EHI. Serum levels of muscle enzymes usually peak at 24 to 48 hours after onset.[90,94] Early enzyme levels may not be helpful in making a diagnosis in the emergency department, whereas casualties with more severe rhabdomyolysis usually exhibit muscle numbness, weakness, pain, tenderness, and stiffness.

Exercising military trainees frequently develop CK values ranging from 500 to 1,000 U/L without other clinical abnormalities, but seldom above 1,200 U/L in the absence of EHI or acute muscle injury.[95] Other laboratory tests for cell damage include serum levels of uric acid, lactate dehydrogenase (LDH), aspartate aminotransferase (AST, formerly called SGOT), and alanine aminotransferase (ALT, formerly called SGPT).[8,90,96–98] LDH can also be released from erythrocytes, lung, and liver, while elevation of serum aminotransferases often reflects liver damage.

If skeletal muscle or liver necrosis is extensive, it produces the *cell lysis syndrome,* which consists of acute renal failure, hyperkalemia, hyperuricemia, a low ratio of BUN to urate, hyperphosphatemia, hypocalcemia, lactic acidosis, DIC, and hypotension.[99–102] These may progress to advanced renal failure requiring dialysis, infectious complications, pulmonary complications (eg, pneumonia, atelectasis, adult respiratory distress syndrome, pulmonary edema), shock with possible myocardial ischemia, bleeding disorders and organ damage from DIC, and death.[1,40,73,103–105] The chemical features and the management of the full-blown syndrome of cell lysis resemble the tumor lysis syndrome resulting from aggressive chemotherapy for highly responsive tumors.[106]

Exertional rhabdomyolysis may occur without elevation of body core temperature[55,107,108] or encephalopathy.[1,109] On the other hand, rhabdomyolysis occurs frequently as part of the clinical syndromes of exertional heatstroke and heat injury.[1,109] It is extremely important to closely monitor hydration, electrolytes, acid–base status, and fluid intake and output, because early aggressive parenteral correction of dehydration and metabolic disturbances is the basis of treatment. The early clinical picture may be deceptive because the patient may manifest minimal clinical symptoms in the presence of profound renal failure or metabolic abnormalities.[55]

Other Complications of Exertional Heat Illness

Hyponatremia

Several recent reports have described severe hyponatremia (Na < 130 mEq/L) associated with exercise in the heat.[110–116] This illness occurs primarily in the setting of forced water drinking (> 15 L/d) for prevention or treatment of early symptoms of EHI.[71,100,112,115–119] Casualties may present with repeated emesis or seizures due to brain swelling. These patients need urgent intensive care with management by appropriate specialists.

When hyponatremia develops slowly through sodium depletion, the brain mobilizes interstitial fluid and expels intracellular solute to avoid a dangerous degree of swelling; but if the osmolality of the extracellular fluid falls too rapidly, these protective mechanisms are not enough to protect the brain from dangerous degrees of swelling and increased intracranial pressure. Thus, the level of plasma sodium is, by itself, not a good indicator of the severity of the problem. If hyponatremia has developed slowly and there are no neurological symptoms due to increased intracranial pressure and brain swelling, the medical officer should correct the hyponatremia slowly. This is because too-rapid correction of hyponatremia can cause a dangerous demyelinating syndrome called pontine myelinosis.[120,121]

In exercise, however, hyponatremia is more commonly produced rapidly through excessive water drinking and water retention in the heat. In this circumstance the risk of demyelination is much less, and more rapid treatment is indicated in order to forestall or relieve pressure on the brain.[102,111,120,121] Owing to recent increases in military casualties with hyponatremia associated with heightened emphasis on fluid replacement during hot weather training, the US Army Research Institute of Environmental Medicine (USARIEM) has issued new fluid replacement guidelines that limit maximal water intake to 1 to 1.5 qt/h, not to exceed 12 qt/d.[122] These new guidelines appear to have reduced the rate of casualties with hyponatremia among army trainees.[123] These guidelines address only the maintenance of fluid equilibrium and body temperature (ie, prevention of EHI) and not treatment for EHI. Because early symptoms of hyponatremia may resemble mild dehydration, the duration of field treatment of casualties with even mild EHI should never exceed 1 hour, with a maximum of 2 quarts of fluid.[119] Otherwise, there is serious potential for significant exacerbation of hyponatremia through excessive fluid ingestion. Persistent, severe, or unusual symptoms or signs warrant immediate medical evaluation (including laboratory assessment).

Ataxia

While focal lesions of the central nervous system (CNS) are not generally seen in exertional heatstroke, the one exception is the rare development of midline lesions of the cerebellum, causing residual gait or speech ataxia. These unusual signs are characteristic of heatstroke when they occur (2 of 137 cases in our series at Parris Island).[56–58] These lesions often persist after complete recovery from the encephalopathy.[124,125] Presence of such ataxia following an episode of exertional rhabdomyolysis or heat injury is a strong indication that heatstroke was part of the acute heat illness syndrome, whether or not it was recognized at the time.

Seizures

Seizures usually occur late in advanced exertional heatstroke or after cardiovascular collapse, indicating a higher risk of permanent brain injury or death. This complication is common among cases of exertional heatstroke receiving delayed cooling and hydration. Several well-known early case series of exertional heatstroke have described an incidence of seizures at 50% to 70%.[39,43,53,126] In recent experience where treatment of EHI (cooling and rehydration) was initiated within minutes of collapse, early seizures have been uncommon except when associated with severe hyponatremia due to excessive water ingestion. Thus, the appearance of early seizures should engender suspicion of hyponatremia, hypoglycemia, hypoxia, cerebral hemorrhage, or other cause, in addition to heatstroke. These casualties need urgent intensive care with management by appropriate specialists.

Disseminated Intravascular Coagulation

DIC is an advanced complication of severe EHI in which there has been considerable tissue damage.[45,61,127–130] DIC usually develops between 18 and 36 hours after the onset of exertional heatstroke.[42] Rarely, it can develop in less than 1 hour.[127] DIC is an ominous sign associated with severe morbidity and high fatality rates. This complication is common among casualties of heatstroke who receive delayed cooling and hydration. However, it has not been observed in our experience with more than 200 cases of exertional heatstroke who received early cooling and rehydration.[58] Hemorrhages can occur in many organs, including the myocardium, lungs, brain, liver, muscle, and gastrointestinal tract.[42,53,127] Severe gastrointestinal bleeding most often follows shock, hypoxemia, and DIC.[15,131]

Hypoglycemia

Exertional hypoglycemia occurs only rarely but is important because immediate treatment prevents brain injury. Medical officers should consider the known causes, which include an abnormal postprandial response (seldom an issue during heavy exercise), endogenous insulin excess, severe hepatic insufficiency, alcohol intoxication, adrenocortical

insufficiency, hypothyroidism, or growth hormone deficiency. Usually the results of subsequent special studies, including tests for fasting hypoglycemia in the absence of exercise, are normal. The single documented case we have seen among more than 2,000 episodes of EHI in Marine Corps recruits and cadre presented as syncope and coma, which developed while standing in a tepid shower after correction of hyperthermia and clearing of mental confusion. The patient responded immediately to a 50-mL ampule of 50% dextrose in water, which was repeated. He then was given an infusion of 5% dextrose in half-normal saline for 24 hours. Subsequent tests for fasting hypoglycemia at rest were negative.

Liver Failure

Rhabdomyolysis is sometimes accompanied by signs of liver necrosis, especially in casualties who have life-threatening complications.[132–137] Occasionally there is little evidence of rhabdomyolysis, but severe liver necrosis produces a similar syndrome of acute cell lysis and renal failure, as discussed above. These cases are likely to be accompanied by DIC, aggravated by failure of the liver to synthesize the vitamin K–dependent factors and fibrinogen, and reduced liver clearance of fibrin degradation products.

Sudden Death, Cardiovascular Collapse, and Arrhythmia

Cardiovascular collapse (shock or a fall in blood pressure) is a relatively common complication in the early presentation of exertional heatstroke.[1,38,43,53,62] The role of cardiac arrhythmia is unclear, although spontaneous resolution of arrhythmia with cooling has been documented in several cases.[7,62,138] In nonfatal cases the blood pressure usually responds rapidly to cooling and rehydration, while failure to respond reflects a poor prognosis. Descriptions of myocardial infarction in exertional heatstroke are largely confined to cases following a period of prolonged shock or ischemia, or associated with severe hepatic or renal damage, as a late complication of heatstroke.[61,62] Several case reports have documented myocardial hemorrhage or necrosis in the absence of any coronary artery disease.[53,61,62]

There have been several published case reports of hyperthermia in exercise-related sudden death among young adults with no silent heart disease detected at autopsy or from medical history.[139] Most authors have assumed that these sudden deaths were from heatstroke, but there is controversy as to whether the mechanism of death should be considered primary cardiovascular collapse from distributive shock[38] or cardiac arrhythmia. Extreme hyperkalemia from rhabdomyolysis may also play a role.

We reviewed all EHI cases, serious cardiovascular events, and deaths among 269,000 recruits in Marine Corps basic training at Parris Island during a 12-year period. There were 7 life-threatening cardiovascular events (5%) among 137 patients during exertional heatstroke (2 sudden deaths; all 7 events were unexplained by preexisting disease from prior history or detailed autopsy) versus 4 (0.0015%) among 267,500 recruits without EHI (4 sudden deaths, with 3 explained by silent preexisting heart disease).[140] These data suggest that the risk for life-threatening cardiovascular events during exertional heatstroke is at least 3,000-fold higher than in exercise without EHI. Exertional heatstroke accounted for at least 7 of 11 fatal or serious sudden cardiovascular events in this population. This contrasts with autopsy studies of exercise-related sudden death among civilian athletes, which report that fewer than 1% of cases were due to EHI.[141,142] These preliminary data suggest that EHI should be considered in the management of exercise-related cardiac resuscitation and in the diagnosis of exercise-related sudden cardiac death.

Military recruits with sickle cell trait (hemoglobin AS) have a 10- to 30-fold greater risk for exercise-related death (unexplained by preexisting disease) than those without the trait.[139,142,143] Half of these excess deaths were due to EHI (heatstroke and rhabdomyolysis) and the other half were unexplained sudden deaths. Most exercise-related, sudden, unexplained deaths of recruits without hemoglobin S occurred in relation to heat stress. Preliminary analysis of a 10-year program to prevent EHI suggests that nearly all excess exercise-related deaths among recruits with sickle cell trait can be prevented, and that large reductions can also be obtained for exercise-related death in recruits without hemoglobin S.[144–146]

EXERTIONAL HEAT ILLNESS SYNDROMES

Exertional heatstroke is fairly common in healthy young adults undergoing strenuous physical training in warm and humid weather, but sedentary or classic heatstroke (included here, briefly, for completeness) is quite rare. *Exertional heatstroke* arises from sustained or heavy exertion, usually in hot environments.[1,147] Typically, onset is abrupt, occurring during or shortly after exertion, with orthostatic manifestations (faintness,

staggering, or visual disturbance) leading to collapse (with or without syncope), followed by confusion, combativeness, delirium, obtundation, or coma. This syndrome frequently evolves in minutes. If it is treated immediately with aggressive cooling and parenteral rehydration, then severe organ damage and mortality are almost always prevented.

Classic heatstroke, on the other hand, is generally associated with extended exposure to a hot environment in the absence of strenuous exercise and is a somewhat different illness.[1,148–150] It is seen primarily in older people (aged ≥ 45) who have diminished cardiovascular reserve and usually develops over several days due to exposure or confinement in a hot environment.[1,151–158] Classic heatstroke is minimally associated with exercise, and dehydration and obtundation are gradual.[159] Patients present with high body core temperature; hot, dry skin; and coma. This type of heatstroke has a high mortality because victims often have chronic illness and tend to present at an already advanced stage. It has been emphasized in medical textbooks because it is the predominant form seen at academic medical centers.[160–163] Classic heatstroke is frequently associated with anhydrosis (absence of sweating), but this is not a common finding in exertional heatstroke.[126,164,165]

Exertional Heatstroke

Exertional heatstroke (the most common form among military personnel) is characterized by early, severe, nonfocal encephalopathy with hyperthermia. There has been no consistent definition of this syndrome. Most case series[39,43,53,126] have described patients with exertional heatstroke who were first evaluated more than an hour after onset of obtundation or coma. This delay in medical care, largely due to medical inaccessibility at training sites, resulted in advanced life-threatening disease on presentation to the medical team. Such patients usually exhibited progressive multisystem disease with severe morbidity and high mortality, and often suffered cardiovascular collapse, rhabdomyolysis, acute renal failure, DIC, and seizures. Metabolic complications often included hyperosmolar states, hyperkalemia, hyperuricemia, hyperphosphatemia, lactic acidosis, and hypocalcemia. Mortality was high and liver necrosis, myocardial infarction, acute respiratory distress syndrome, and diffuse brain damage were often seen in survivors.

The importance of rapid access to emergency medical care is being increasingly recognized in military training. The usual clinical problem is to identify exertional heatstroke prior to onset of nonneurological complications. In this situation a reasonable definition of exertional heatstroke is EHI that causes early delirium, obtundation, or coma (see Exhibit 7-3).[2,3,151] Such cases usually occur without anhydrosis (a common characteristic in classic heatstroke) and often occur with relatively low rectal temperatures (< 106°F [< 41°C]). Many cases of exertional heatstroke with severe encephalopathy but modest hyperthermia have been reported. We do not consider hyperthermia, anhydrosis, or other complications essential to the diagnosis of exertional heatstroke.[1,4,5,50,166,167] Water and electrolyte depletion, acute renal failure, and muscle necrosis (rhabdomyolysis) are common components of heatstroke. Casualties must be closely monitored and aggressively treated to minimize morbidity and mortality.

When cooling and hydration are instituted early on the basis of the rectal temperature measurement, the encephalopathy of exertional heatstroke generally peaks within 10 to 30 minutes. Among Marine Corps recruits treated early with ice water and intravenous hydration, recovery of orientation and cooperative behavior generally occurs within 20 to 40 minutes from arrival at the emergency department, and correlates with rectal temperature lower than 102°F (39°C). Most patients can give a coherent clinical history (limited by amnesia) within 40 to 60 minutes.

Early treatment of exertional heatstroke also appears to prevent the development of life-threatening acute renal failure and cell lysis characteristic of severe rhabdomyolysis and hepatic necrosis. Among more than 200 cases of exertional heatstroke at Parris Island, from 1979 to 1994, none exhibited life-threatening complications of rhabdomyolysis involving hyperkalemia, hyperuricemia, hyperphosphatemia, altered calcium levels, lactic acidosis, progressive renal failure requiring dialysis, or DIC.[58] Minor complications in the early stages of muscle or liver injury responded quickly to cooling and hydration. The only life-threatening complications noted in this series were episodes of early cardiovascular collapse or sudden cardiac arrest.[138,140] Sudden cardiovascular collapse within the first hour was the syndrome that preceded all deaths. Exertional heatstroke was not observed in women recruits, although 18 hospitalizations for EHI were predicted from hospitalization rates for men.[58]

Exertional Heat Injury

Exertional heat injury is a progressive multisystem disorder, with hyperthermia accompanied by organ damage or severe dysfunction (usually metabolic acidosis, acute renal failure, muscle necrosis, or liver

necrosis).[49,163] We classify it as involving more-severe manifestations than exertional heat exhaustion, with less-severe complications than those of heatstroke, severe rhabdomyolysis, or acute renal failure. Casualties with exertional heat injury often have mild neurological symptoms, but periods of combativeness, delirium, obtundation, or coma define exertional heatstroke rather than heat injury.[8] Organ damage or dysfunction is frequently not manifested in early heat injury, so during the first hours of illness it may not be possible to distinguish exertional heat injury from heat exhaustion. Therefore, it is essential that all casualties with EHI be thoroughly evaluated for organ damage or severe dysfunction before release from the medical treatment facility, with reevaluation often necessary on the following day.

Exertional Heat Exhaustion

Exertional heat exhaustion is a reversible, non–life-threatening multisystem disorder reflecting the inability of the circulatory system to meet the demands of thermoregulatory, muscular, cutaneous, and visceral blood flow. It represents primarily a syndrome of dehydration without serious metabolic complications.[1,3,163] Heat exhaustion is typically thought to produce minor elevations of core temperature (< 104°F [< 40°C]), but it can be associated with the full range of rectal temperatures, from subnormal to very high. We define heat exhaustion as reversible system dysfunction, without evidence of organ damage or severe metabolic consequences.

The symptoms of heat exhaustion can be quite varied (see the mild and nonspecific symptoms listed in Exhibit 7-1) and are rapidly improved by water and salt replacement, a cool environment, and rest.[50,163] Rapid cooling to a body temperature of less than 102°F (39°C), rest, and rehydration are essential to prevent progression to more-serious levels of EHI.[2,11] Because heat exhaustion represents system dysfunction, without organ or tissue damage, rapid improvement is the rule.

Minor Heat-Related Syndromes

In addition to the disorders discussed above, which represent the primary spectrum of EHI, there are minor syndromes also associated with heat exposure. We do not recommend that the following be included as a part of an EHI reporting system, however, as they represent either very minor or unrelated syndromes.

Heat Rash

Heat rash (also known as prickly heat or miliaria rubra) is a pruritic red papular rash, located particularly in areas of restrictive clothing and heavy sweating.[49,168,169] It is caused by inflammation of the sweat glands. It may interfere with sweating and can therefore be a risk factor for more-serious heat illness. Sleeplessness due to itching and secondary infection may further aggravate thermoregulation. Treatment consists of cooling and drying the affected skin, controlling infection, and managing pruritus; the rash usually resolves over 7 to 10 days. Rare, severe cases with generalized and prolonged rash (miliaria profunda) may require evacuation to a cooler environment for several weeks to restore normal sweat gland function.

Heat Edema

Heat edema is dependent edema of the legs ("deck legs"), which may occur during acclimatization to a hot environment; it resolves spontaneously and is of no clinical significance.[21,49] Edema is due to expansion of blood volume during acclimation and does not indicate excessive water intake or cardiac, renal, or hepatic disease.[2] Management consists of loosening the clothing and elevating the legs. It is unwise to offer diuretics, as they may interfere with acclimatization and induce potassium loss.[50]

Parade Syncope

Parade syncope is fainting during prolonged standing due to inadequate venous blood return to the heart and brain.[49] It is more common in a hot environment but is unrelated to exercise. Syncope occurring during or after work in the heat should raise the suspicion of heat exhaustion or heat injury. Management of parade syncope is to restore normal blood circulation and minimize peripheral pooling through a brief period of recumbency in a cool place. Allowing individuals to move about will help prevent parade syncope, and provision of chairs and railings will decrease the risk of injury.

Exertion-Associated Collapse

Collapse *following* strenuous exercise may sometimes have a physiological etiology. During exercise, repetitive muscle pumping action assists the return of venous blood to the heart. On cessation of exercise, this muscle assistance to blood flow

ceases, and venous blood return may diminish rapidly enough to produce nausea, collapse, or syncope. Prolongation of mild exercise to allow time for adequate vasoconstriction is the rationale for a "cool-down lap" at the end of a strenuous run. Although this form of syncope may occur in the absence of EHI, clinical evaluation is warranted because these symptoms may be due to hypovolemia and other features of EHI. In contrast, collapse *during* exercise must be intensively investigated because of the greater potential for significant EHI or heart disease.

Sunburn

Sunburn reduces the thermoregulatory capacity of the skin and should be prevented through adequate sun protection. It should be managed like any other burn, and heat stress should be avoided until the burn has healed.[49]

CLINICAL MANAGEMENT OF EXERTIONAL HEAT ILLNESS

The severity of illness is often not apparent on initial presentation of EHI in the field. Severe EHI is a catastrophic event and should be treated as a medical emergency. Since milder forms of EHI may progress rapidly to more-serious illness, immediate and thorough evaluation is necessary to assess severity. Rectal temperature should be obtained immediately and rapid cooling should be initiated in the field.[170,171] Mental status assessment must always accompany measurement of rectal temperature and vital signs. Rehydration should always accompany cooling.[172] Delay in cooling and rehydration probably represents the single most important factor leading to death or residual, serious disability in those who survive.[50] In addition, there must be close monitoring of vital signs, aggressive replacement of fluid and electrolytes, and appropriate laboratory workup.[55,148,173]

In monitoring core temperature, it is important to avoid relying on measurements made at superficial sites such as the mouth, axilla, tympanum, or ear canal. Oral temperature is lowered by mouth breathing, especially in the presence of hyperventilation, a frequent response to hyperthermia. Tympanic and ear canal temperatures must be measured under carefully controlled conditions to avoid their being biased by the environment. It is not feasible to control those conditions adequately in most clinical settings, and in collapsed hyperthermic athletes, ear temperature readings have been 6 to 10 Fahrenheit degrees below rectal temperatures.[174]

Diagnostic Evaluation

Close monitoring of vital signs and serum chemistries is essential since during the first few hours, clinical symptoms may not reflect profound underlying metabolic abnormalities.[55,151,173,175] The initial laboratory evaluation for casualties, at different levels of clinical concern (mild-to-moderate and severe illness), is shown in Exhibit 7-5. The panel for mild or moderate illness screens for infection, anemia, and thrombocytopenia, and assesses dehydration. Evaluations of serum electrolytes screen for hypernatremia, hyperkalemia, and metabolic acidosis, although occasionally hyponatremia or hypokalemia may be found. The evaluation for glucose level detects exertional hypoglycemia and diabetic hyperglycemia, both of which are treatable causes of encephalopathy. The BUN and creatinine are tests for renal insufficiency, dehydration, or renal failure. Urinalysis should be performed, particularly to identify pigmented casts and a positive heme test out of proportion to red cells in the sediment (findings that are typical of rhabdomyolysis with myoglobinuria and renal tubular injury). Often the patient is unable to urinate until considerable hydration has occurred, so the urine is relatively dilute and reflects the posttreatment, rather than the pretreatment, status of the patient.

A serum enzyme panel is used to identify rhabdomyolysis and liver damage. These assays may be insensitive in early illness because of delayed release of the enzymes into the circulation. Maximum serum muscle or liver enzyme levels often occur 24 to 48 hours after the onset of illness, necessitating follow-up laboratory assessment the next day for all but the mildest cases.[50,62,154,176]

Selected cases of intermediate severity may require additional screening for the early signs of significant rhabdomyolysis and acute renal failure (see Exhibit 7-5). These tests are performed to distinguish between renal insufficiency due to poor perfusion of the kidney and that due to acute renal failure. Elevations of creatinine up to 1.7 mg/dL are often seen initially in rapidly reversible EHI, probably due more to reduced glomerular filtration rates and muscle-cell release of creatine than to tubular dysfunction. It is important to determine whether the creatinine level is improving after a few hours of

EXHIBIT 7-5

RECOMMENDED LABORATORY EVALUATION FOR EXERTIONAL HEAT ILLNESS

- **Mild-to-Moderate Illness**

  Complete Blood Count: Hemoglobin, hematocrit, white blood count and differential, platelet count

  Urinalysis: Specific gravity, pH, dipstick, and microscopic examination of sediment

  Serum Chemistries (Heat Panel): Sodium (Na), potassium (K), chloride (Cl), bicarbonate (HCO_3), glucose, blood urea nitrogen (BUN), creatinine (Cr), osmolality, creatine kinase (CK), aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), uric acid, myoglobin

- **Severe Illness, Add:**

  Examine Peripheral Blood Smear

  Serum Chemistries: Calcium, phosphorus, albumin, lactic acid, myoglobin, cardiac enzymes

  Arterial Blood Gases

  Coagulation Studies: Prothrombin time (PT), partial thromboplastin time (PTT), fibrin degradation products or D-dimer, fibrinogen level

  Consider Tests for Renal Failure (**Exhibit Table 1**)

EXHIBIT TABLE 1

LABORATORY EXAMINATION IN OLIGURIC ACUTE RENAL FAILURE

Diagnosis	U/P Cr	U_{Na}	FE_{Na} (%)	U Osmolality
Prerenal Azotemia	> 40	< 20	< 1	> 500
Oliguric ATN	< 20	> 40	> 1	< 350

U: urine; P: plasma; FE_{Na}: fractional excretion of sodium; ATN: acute tubular necrosis
Table: Reprinted with permission from Coyne DW. Acute renal failure. In: Carey CF, Lee HH, Woeltje KF, eds. *The Washington Manual of Medical Therapeutics*. Philadelphia, Pa: Lippincott-Raven; 1998: 230.

hydration. If not, then a casualty may require hospitalization or prolonged observation in the emergency treatment facility. It is also important to ensure that initial oliguria has been corrected.

If some of these screening tests are positive, casualties with severe illness may require further screening for early phases of the cell lysis syndrome, during which symptoms may be mild. Cell lysis can result in hyperkalemia, hyperuricemia, hyperphosphatemia, lactic acidosis, and hypocalcemia. Arterial blood gases should be obtained, with particular attention to metabolic acidosis. If the bicarbonate level is low, serum lactic acid levels will help determine the cause.[50,177] An evaluation of cardiac enzymes should be included to identify myocardial injury. Serum chemistries should be followed serially, with a determination of serum albumin to provide correct interpretation of calcium. Screening for DIC should include examination of the peripheral blood smear to identify fragmented erythrocytes and to confirm true thrombocytopenia rather than artifactual platelet clumping. If available, the D-dimer test is more specific and preferable to the assay for fibrin degradation products.

Stratification and Immediate Care

Immediate physical assessment must include frequent monitoring of mental status and vital signs, including rectal temperature and orthostatic changes in pulse and blood pressure (which are seen when a patient is subjected to tilt tests [see Exhibit 7-1]). For casualties with marked hyperthermia it is convenient to use a rectal probe, which provides continuous visualization of rectal temperature. Frequent assessments of mental status and muscle

symptoms are essential. Laboratory and clinical findings need to be integrated and level of care for diagnosis and management determined. There are no obvious, well-defined boundaries between the disease categories of life-threatening EHI (heatstroke, severe rhabdomyolysis, acute renal failure, shock), heat injury, and heat exhaustion. Each clinician chooses definitions that suit triaging of patients within his or her clinical system (ie, selecting patients for intensive or routine hospital care, extended emergency or clinic care, routine clinic care with follow-up, or clinic care without follow-up). Thus, each syndrome categorization along this continuum of EHI is somewhat arbitrary and has implications for treatment and prognosis in a specific healthcare setting.

A practical approach to the management of EHI is to examine each of the major areas of clinical involvement on a severity scale that reflects the usual management for each component problem. It then becomes easier to develop a sensible plan for management of the entire set of clinical problems. This is illustrated in Table 7-1 for management of a military recruit being seen at the nearest emergency care facility within 5 to 20 minutes after onset of EHI. These casualties are medically screened young adults without known chronic illness, taking few or no medications, and who were able to participate in conditioning exercise just prior to the onset of illness. The specific areas of physiological dysfunction considered are

- dehydration and electrolyte disturbances;
- nonfocal encephalopathy and other neurological signs;
- evidence of renal insufficiency;
- evidence of rhabdomyolysis (cell lysis); and
- other manifestations, including hyperthermia, muscle symptoms, hypoglycemia, and coagulopathy.

Categories 1 and 2 generally indicate clinical findings consistent with exertional heat exhaustion. A laboratory panel will be obtained for some casualties with mild illness (see Exhibit 7-5). As a rule, patients are treated with rest and rehydration and are removed from exercise overnight and perhaps throughout the following day. Laboratory evaluation is optional for Category 1. Category 2 implies that the laboratory panel for mild illness will be obtained, but follow-up is optional. Exertional heat cramps are usually treated by infusion of normal or half-normal saline (1–2 L). Oral salt solutions (0.5%) are also effective if the parenteral route is unavailable. Patients in Category 2 will often receive parenteral hydration for signs of fluid deficit or muscle cramps and will be kept at rest throughout the next day.

Patients in Category 3 and some in Category 4 would generally be considered to have exertional heat injury. The standard of care for patients in Category 3 includes the appropriate laboratory panel, rapid cooling with ice water for those whose rectal temperature is above 102°F (39°C), parenteral rehydration, and clinical and laboratory follow-up at 12 to 24 hours. The recruit will often be kept at rest for 2 to 3 days.

Patients in Categories 4 and 5 have severe EHI, and the standard of care includes a more extensive laboratory panel and dictates more vigorous intervention. Patients in Category 4 receive (*a*) rapid cooling with ice water for rectal temperature above 102°F (39°C), (*b*) rapid parenteral hydration with 2 to 3 liters of isotonic fluid, and (*c*) reevaluation of laboratory studies before discharge from emergency care (see Exhibit 7-5). At Category 4, some patients may be hospitalized, and all will be followed up at 12 to 24 hours. Indications for hospitalization are found in Exhibit 7-6. At Category 5, hospitalization is the standard of care. Most of these patients are managed in an intensive care unit. With aggressive management, most will not develop progressive rhabdomyolysis and are discharged after 2 or 3 days of hospitalization.

A frequent clinical problem is a patient with EHI who appears normal after routine management except for elevated serum CK or evidence of myoglobinuria. In this circumstance, the medical officer should maintain alkaline urine, then rule out the cell lysis syndrome and acute renal failure by laboratory evaluation (see Exhibit 7-5). If these tests are normal, the patient can be followed as an outpatient with continued hydration, observation, monitoring of urinary output, and daily laboratory follow-up until CK is returning to normal. The same applies to a patient with localized muscle breakdown due to overuse, which produces elevated CK with or without myoglobinuria and without other systemic symptoms or signs.

Usually, patients who are severely ill will declare themselves within the first few hours. Typical severe syndromes are (1) heat injury involving several systems, (2) exertional heatstroke with severe CNS dysfunction (often accompanied by rhabdomyolysis), and (3) severe rhabdomyolysis (occasionally without hyperthermia or encephalopathy). However, acute renal failure is often not apparent during the first hours of illness. Occasionally, the onset of severe rhabdomyolysis has been obscured, particularly in individuals who are highly motivated to conceal

TABLE 7-1

CLASSIFICATION, STRATIFICATION, AND CLINICAL MANAGEMENT OF EXERTIONAL HEAT ILLNESS

Category	Dehydration	Encephalopathy*	Renal Function† (mg/dL)	Cell Lysis† (U/L)	Other	Clinical Management
1	Nonspecific symptoms	Slow, drowsy, clears rapidly	Cr: 2 1.4 No oliguria	CK: < 700	Nonspecific symptoms Max temp < 100°F	Rest and rehydration. Follow-up is not usually needed.
2	Collapse or orthostatic Sx in field, normal v.s. in ER	Confused/cooperative, clears rapidly	Cr: 1.5–1.7 No oliguria	CK: 700–1,200	Cramps, myalgia resolve in ER Max temp 3 100°F	Rest, cooling, and IV rehydration. Laboratory evaluation as indicated. Clinic follow-up, laboratory follow-up, or both may be indicated.
3	Exertional syncope or positive tilt test, or abnormal electrolytes, corrected in ER	Confused/uncooperative, clears rapidly, amnesia	Cr: 1.8–1.9 High urinary SG decreases Oliguria responds rapidly	CK: 1,200–4,000 ALT/AST: 50–150 Mild rhabdomyolysis	Minor muscle Sx persist Max temp 3 104°F	Rest, rapid cooling, IV rehydration, and laboratory evaluation. Clinic and laboratory follow-up are the usual standard of care.
4	Patient requires > 3 L IV fluid, or hypotension, or electrolyte abnormality persists	Delirium, obtundation, clears rapidly, amnesia	Cr: 3 2.0 Oliguria responds slowly; mild urine abnormalities	CK: 4,000–10,000 ALT/AST: > 150 Metabolic acidosis Negative labs for cell lysis syndrome	Muscle Sx persist Max temp 3 106°F	Rest, rapid cooling, IV rehydration, and extensive laboratory evaluation. Hospitalization may be indicated.
5	Mean BP < 70, systolic < 90, tachycardia persists Hypokalemia Hyponatremia	Coma, or CNS signs clear slowly, seizure	Cr: 3 2.0 Oliguria persists Other signs of renal failure	CK: 3 10,000 Signs of cell lysis syndrome Severe metabolic acidosis	Major muscle Sx persist Hyperthermia > 40 min Hypoglycemia Thrombocytopenia Coagulopathy	Rest, rapid cooling, IV rehydration, and extensive laboratory evaluation. Close observation and life support/intensive care may be indicated. Hospitalization is the usual standard of care.

ALT: alanine aminotransferase, AST: aspartate aminotransferase, BP: blood pressure, CK: creatine kinase, Cr: creatinine, CNS: central nervous system, ER: emergency department or emergency care facility, IV: intravenous, labs: laboratory evaluations, SG: specific gravity, Sx: symptoms, U/L: units per liter, v.s.: vital signs

*See Exhibit 7-3 for explanation of terms describing encephalopathy.

†Early levels of CK and Cr have limited meaning because these values are based on maximums seen at 12–36 h. Continued muscle symptoms, other laboratory evidence of the cell lysis syndrome, and renal failure are critical determinants. The direction of change in sequential studies is essential to the early recognition of serious rhabdomyolysis. Rarely, the ALT and AST are elevated beyond the muscle enzymes (CK), implying liver necrosis as the dominant source of the cell lysis syndrome.

EXHIBIT 7-6

INDICATIONS FOR HOSPITALIZATION IN EXERTIONAL HEAT ILLNESS

- Difficulty correcting hypotension
- Persistent electrolyte abnormalities
- Seizure
- Severe encephalopathy
- Moderate encephalopathy not clearing rapidly
- Persistent creatinine above 2 mg/dL
- Persistent oliguria
- Laboratory evidence of acute renal failure (see Exhibit 7-5) or myoglobinuria
- Laboratory evidence of the cell lysis syndrome
- Persistent muscle symptoms suggesting evolving rhabdomyolysis
- Marked hyperthermia (rectal temperature 3 105°F [3 41°C]) not improving (or with slow response to aggressive cooling)
- Evidence of DIC
- Exertional hypoglycemia
- Persistent substantial diarrhea
- Significant gastrointestinal bleeding
- Hypokalemia

muscle pain, and rarely, the onset of hyperthermia is delayed in patients with exertional heatstroke.

Principles of Clinical Management

Exertional heat illness requires urgent diagnosis and treatment. Although Category 4 and Category 5 syndromes, which include heatstroke, severe muscle or liver injury, and acute renal failure, are clearly medical emergencies, patients with EHI at milder levels of severity require urgent and aggressive management to avoid progression. Rectal temperature should be obtained immediately, and if it is above 102°F (39°C), rapid cooling should be initiated in the field. Such patients should be kept at rest in a cool environment, and vital signs and mental status must be closely monitored. Rapid cooling of hyperthermic patients should proceed until the body core temperature remains below 102°F (39°C).[178,179] The degree of organ damage in EHI with hyperthermia appears to be directly related to the magnitude and duration of elevated core temperature.[53,180,181] Patients who require cooling will also require rehydration at the same time to restore adequate circulation for effective thermoregulation.[172] Frequent serial evaluation of vital signs, mental status, and clinical assessment should start in the field and continue throughout initial emergency care. After cooling and correction of fluid and electrolyte problems, it then becomes possible to assess for acute renal failure, rhabdomyolysis, hypoglycemia, and other complications. The initial clinical assessment will also permit identification and management of coexistent problems such as infection.

In controlled settings, emergency medical care for EHI should be arranged in advance. Management of EHI is always urgent, to avoid the potential for rapid deterioration. Patients need immediate access to medical support in the field, to include, at a minimum, measurement of rectal temperature, brief clinical assessment of vital signs and mental status, and effective cooling.[149,182] The field team should also be prepared to provide basic life support (BLS) and cardiopulmonary resuscitation (CPR), advanced cardiac life support (ACLS, including early automated defibrillation), and first aid for injuries. If transportation to an emergency department will require more than 5 to 10 minutes, provision should be made for administering intravenous fluids en route. The field medical team needs to provide an accurate clinical description of the immediate events, symptoms, signs, vital signs, and mental status of the patient, along with training activities, environmental conditions, clothing, and treatment given prior to arrival at the medical facility. To avoid substantial delay in treatment in settings where EHI is common, strenuous physical training should not be conducted without on-site medical capability.[183] For example, a recruit training center should require that at least one medical corpsman or medic (with equipment, ice, and a transport vehicle) be on site while strenuous training is conducted. When the vehicle leaves the training site, strenuous exercise should be stopped until medical support and transport are again available.

There is a known risk for sudden fatal cardiac arrest with exercise, which has been an important component of unexpected exercise-related mortality of recruits.[142] Furthermore, we believe that risk for sudden fatal cardiovascular events increases dramatically during the hyperthermic phase of exertional heatstroke. Although not routine in the past, we believe that cardiac monitoring should be instituted early in the treatment of heatstroke, and that ACLS must be immediately available for patients who develop exercise-related shock or arrhythmia while hyperthermic with EHI.

In this treatise we provide guidelines for field, outpatient, and emergency care of military personnel with EHI, but we leave intensive care of these patients to other specialized texts. Patients with heatstroke potentially have encephalopathy; hyperthermia; acute renal failure; rhabdomyolysis; electrolyte and acid–base imbalances, including lactic acidosis; and may develop additional complications such as DIC. Such patients obviously require intensive care following their initial presentation, but important initial treatment is given in the field and the emergency care setting before arrival in the intensive care unit.[151]

Treatment of Hyperthermia

Most important in the treatment of EHI is the early initiation of cooling and rehydration, starting in the field with an immediate rectal temperature reading. First the patient needs to lie down. An obtunded or comatose patient should be placed on one side, with the airway closely monitored to avoid aspiration of vomitus. As much clothing should be removed as is practical, and those who are hyperthermic should be doused with cold water followed by ice water sheet massage or other effective means of cooling. If lengthy transport is required, immediate parenteral access and rapid initial infusion of normal or half-normal saline are recommended.

There is debate over the best method for body cooling.[162,184–190] The American College of Sports Medicine states that "cool or cold (ice) water immersion is the most effective means of cooling a collapsed hyperthermic runner."[191] Concerns have been expressed that ice water may induce peripheral vasoconstriction or shivering and reduce skin temperature without reducing core temperature. In actual practice in Marine Corps recruit training at Parris Island, cooling with ice water was found to reduce rectal temperature by about 0.15°C (0.25°F) per minute, with no mortality from heatstroke.[184] Studies on thousands of religious pilgrims who succumbed to heat illness during their Hajj in Saudi Arabia show that a cooling rate of 0.06°C (0.1°F) per minute is obtained when a device is used that sprays tepid water and cools with fans.[192] The mortality rate for heatstroke in this setting (which included a large number of older patients) was about 20%. Because coronary arteries with atherosclerosis often undergo spasm in response to ice water exposure, patients with atherosclerosis may be at less risk from tepid water cooling.[193,194] Therefore, we recommend that ice water cooling be used for patients younger than age 35 to 45 years, but that tepid water and fans be used instead (at least initially) to cool older patients.

Body cooling should be accomplished as quickly as possible, using the most practical means available.[166] Methods for cooling include removing outer layers of clothing, soaking the skin with water, using wet sheets or spray bottles, massaging the skin, and resoaking. Cold water applications may be alternated with massage to encourage local blood flow and heat dissipation.[184] If these techniques do not rapidly reduce the rectal temperature (best monitored with a rectal probe), the patient can then be partially immersed, using a stretcher over a tub filled with ice water. The stretcher is lowered so that only the patient's buttocks and lower back are in the ice water, leaving the anterior trunk and limbs accessible for clinical evaluation, parenteral lines, and other treatments. Alternatively, ice bags or a cooling blanket—especially if placed over major arteries—can provide cooling while the patient lies on a bed board. In this situation, CPR and ACLS can safely be implemented. The cooler the water the more rapid the cooling progresses. The room should be air conditioned to maintain a low humidity and air temperature. Fanning can be accomplished manually, by electric fans, or by placing the patient under rotating helicopter blades (a well-tested military field expedient). Rapid cooling of hyperthermic patients should continue until the rectal temperature remains below 102°F (39°C), after which cooling can proceed without cold water (such as a tepid shower) until the rectal temperature remains below 100°F (38°C). The patient must be physically supported and observed during this process, and monitoring of body temperature should be continued for at least 2 to 4 hours. Regardless of the method of cooling, it appears that the degree of organ damage is directly related to the magnitude and duration of elevated body temperature.[147,172,195]

It is important to avoid using antipyretics in the treatment of hyperthermia due to EHI. This is because antipyretics work by lowering the body temperature set point in the hypothalamus. As discussed above, this is not altered during heatstroke. Disadvantages of antipyretics include risk of severe liver injury with acetaminophen, reduced potassium excretion with nonsteroidal antiinflammatory drugs, depressed platelet function and risk of subsequent DIC with aspirin, and risk of gastric irritation or bleeding. Occasionally, cooling leads to inappropriate shivering while the body temperature is still elevated; this may increase the generation of body heat and aggravate rhabdomyolysis. Chlorpromazine should not be used because of a relatively high incidence

of hypotension in patients with EHI.[4,151] Lorazepam, which is also a sedative, is probably the safest drug for this purpose, in part because of its low risk for hepatotoxicity and its rapid metabolism.

Treatment of Fluid and Electrolyte Depletion

Oral rehydration is adequate for the treatment of mild heat exhaustion and mild heat cramps. Heat cramps respond to oral salt solutions (0.5% salt, or 1 teaspoon per quart of water); otherwise patients are usually given water. Patients with more severe EHI, including those with orthostatic or supine hypotension, history of collapse or syncope, severe heat cramps, hyperthermia, or clinical findings that place them in Category 3 or higher levels of EHI (see Table 7-1) are better treated with parenteral rehydration. Young military personnel with these problems are usually first given 1 to 2 liters of normal or half-normal saline, starting at a rate of 250 to 500 mL/h, and their response is carefully monitored. Normal saline is used for hypotensive patients and those whose evaluation suggests hyponatremia or severe deficits of both salt and water.[8,166] The need for more than 2 liters of fluid is an indication for laboratory monitoring both to assess the severity of illness and to choose the best infusion to correct electrolyte abnormalities—especially alterations in sodium, potassium, and bicarbonate levels.[50,179] Most often, it is necessary to replace some free water as well as sodium. However, patients who had been drinking large volumes of water might have become hyponatremic.[115] Fluid intake and output should be measured, with frequent monitoring of vital signs, especially for those patients with Category 4 or higher levels of EHI (see Table 7-1). When supine blood pressure is adequate, tilt tests should be used to identify patients whose blood pressure control is still abnormal.[99]

A practical approach to the urgent correction of hypernatremia, hyperkalemia, hyponatremia, hypokalemia, and acidosis has been published elsewhere[196] in more complete form. Potassium depletion is best treated orally, but patients with severe EHI may not be able to take oral medications because of obtundation or nausea and vomiting. Intravenous potassium replacement should be administered in half-normal or normal saline; dextrose (which tends to move potassium into cells) should be avoided. Rates of infusion are usually limited to 20 mmol/h unless paralysis or malignant ventricular arrhythmias are present, in which case higher rates are recommended. Close electrocardiographic (ECG) and neurological monitoring are required.

The earliest ECG sign of hyperkalemia is peaked T waves. Hyperkalemia should always be suspected when young exercising individuals collapse with arrhythmias. When hyperkalemia is found by chemical analysis, the medical officer should exclude pseudohyperkalemia, primarily due to needle hemolysis (with an elevated LDH level), fist clenching during blood drawing, marked leukocytosis or megakaryocytosis, or erroneous assay. An ECG should be performed while awaiting results of the repeat assay. The acute treatment of severe hyperkalemia is a medical emergency, the management of which has been well described.[196] Treatment of either hypokalemia or hyperkalemia requires careful monitoring of response because of shifts between intracellular and extracellular compartments and changes in rate of renal loss.

Severe metabolic acidosis is often present in severe dehydration. Arterial blood gas measurements are essential to establish arterial pH.[50,177] Aggressive replacement of fluids and conservative replacement of base deficits are required until blood volume and pH are adequate.[197] This requires intensive monitoring of cardiopulmonary status and urinary output (via Foley catheter), with adjustment of intravenous fluid therapy based on the clinical response.[55,72] Severely ill patients will require invasive monitoring and management in an intensive care unit.

Treatment of Impaired Renal Function

Early management of acute renal insufficiency in EHI should focus on correction of the prerenal component due to volume depletion and hypotension. If response to initial rapid isotonic fluid infusion confirms a substantial fluid deficit, then infusion of parenteral fluids should continue, guided by monitoring of urinary output and assessment of blood volume. Persistent or severe hypotension may require the use of dopamine hydrochloride, a pressor favoring renal perfusion. Diuretics are used only after correction of hypovolemia. Probably the most common error made in emergency care is to administer diuretics too early, while the volume deficit is still substantial. Rhabdomyolysis produces myoglobinemia and lactic acidosis, which increase the risk of continued renal tubular damage from myoglobin. Forced alkaline diuresis is believed to be protective. However, we cannot administer bicarbonate safely, or increase diuresis with diuretics, unless blood volume has already been restored and urinary output is adequate (urinary output should exceed 50 mL/h).

Allopurinol should be used to treat severe levels of hyperuricemia by decreasing production of uric acid. Treatment is usually not necessary unless uric acid exceeds 15 mg/dL. The detailed management of acute renal failure requires close monitoring of renal, cardiovascular, and pulmonary function; and monitoring and correction of a number of potential electrolyte and chemical problems. This is best accomplished in an intensive care setting, with institution of dialysis if renal failure persists. An additional feature of rhabdomyolysis is the cell lysis syndrome, which causes early hyperkalemia, hyperuricemia, hyperphosphatemia, hypocalcemia, and lactic acidosis due to release of these materials from injured tissues, particularly muscle and liver. Management of this syndrome is discussed in standard texts on intensive care.

Treatment of Neurological and Other Signs and Symptoms

The encephalopathy of EHI is treated by rapid cooling of patients with hyperthermia and by rehydration. It is important to be aware of the possibility of a reversible metabolic encephalopathy from other complications of EHI, such as hypoglycemia or severe water and electrolyte disturbances involving sodium, potassium, calcium, magnesium, and acid–base abnormalities. Unrelated metabolic problems that might mimic EHI include thiamine deficiency and certain endocrinopathies (thyroid storm and adrenal crisis). Mass lesions, meningitis, brain abscess, thrombosis, and hemorrhage usually produce other signs not seen in uncomplicated EHI and fail to improve with rehydration and reduction in body temperature. Most often the duration of delirium is not sufficient to require use of sedatives, which are not recommended for EHI. Sometimes the need to protect the airway during obtundation or the development of abnormal or ineffective breathing mandates intubation and administration of oxygen via endotracheal tube. This may be followed by maintenance on a ventilator until the patient's respiratory drive and airway improve.

If the patient has a seizure, then management will include assessment for reversible metabolic causes, including hyponatremia, hypoglycemia, magnesium deficiency, infection, mass lesions, and brain injury from thrombosis or bleeding. When the seizure is due to heatstroke, then rapid cooling and rehydration are important elements of treatment. In recent years, many cases of early exertion-related seizures among military trainees have been due to hyponatremia. Regulated, but rapid, correction of hyponatremia is then urgent. Treatment may include administering antiepileptic drugs, particularly short-acting benzodiazepines followed by loading doses of phenytoin, and maintaining protection for the airway.

Management of *exertional hypoglycemia* consists of infusion of 50% dextrose in water as indicated by the level of glucose and clinical signs, followed by infusion of 5% dextrose in water or saline over the next 24 hours, with frequent reassessment.

Gastrointestinal bleeding in EHI can be due to other gastrointestinal disease aggravated by exercise-induced diarrhea. It is important to exclude DIC, to replace significant blood loss, and to stabilize the patient for early endoscopy if the bleeding is extensive.

DIC typically becomes a problem between 18 and 36 hours after onset of exertional heatstroke.[42] The most important therapy for DIC is to correct the underlying problem causing the hypercoagulable state. In EHI this is often tissue necrosis, typically of muscle and liver, but later in the course it may be due to infection. If bleeding becomes the major problem from DIC, the medical officer can replace fibrinogen and clotting factors by infusion of cryoprecipitate and fresh frozen plasma, and use transfusion of leukofiltered platelets to sustain an adequate platelet count. If thrombosis is the main problem, then the judicious use of heparin can be considered.

Disposition of Casualties With Exertional Heat Illness

The duration of increased risk for recurrent episodes of EHI is unknown, although about 10% of cases among Marine Corps recruits at Parris Island (discussed below) had more than one episode of EHI during their 12 weeks of basic training. Recurrent risk has been discussed most often with exertional heatstroke. There are numerous anecdotal reports of recurrence of EHI even years later, raising the possibility of long-term host susceptibility. Intensive physiological studies on the effects of repeated exposures to exertional heat load suggest that there may be increased susceptibility for some months following exertional heatstroke.[198] Furthermore, there are individuals who have repeated high levels of CK or symptomatic mild rhabdomyolysis with heavy exercise, some of whom have had repeated severe episodes of rhabdomyolysis. However, the data required to properly address this issue would need to be obtained from a large, controlled, follow-up study of individuals who did and did not suffer severe EHI and were reexposed at multiple, subse-

quent times to heavy exercise in hot environments. While such data are potentially available from military populations, such a study has not yet been reported.

The conventional view is that the potential for increased susceptibility is substantial and that severe EHI is grounds for permanent medical discharge from the military. In our experience, a substantial percentage of Marine Corps recruits with EHI present at a medical treatment facility with high body temperature but only mild symptoms and signs. Laboratory screening for electrolyte disturbances, renal failure, and rhabdomyolysis show only mild problems. Early aggressive cooling and hydration results in rapid reversal of hyperthermia, normal urinary output, and improvement of symptoms. We believe that these patients have not suffered severe EHI and therefore should not be considered for the military career restrictions that are placed on those who have been hospitalized for heatstroke. Preliminary data on long-term follow-up suggest that subsequent hospitalization for EHI is rare.

Many patients appear to have mental (and perhaps physical) impairment for several days following even a mild episode of EHI, including poor judgment and difficulty concentrating.[43,51,199] We recommend that patients with mild EHI be provided rest in a cool area at least overnight. Those with moderate illness should be followed up with clinical evaluation (including careful examination for slow mentation and poor concentration) and repeat laboratory studies at 24 hours, then given perhaps another 24 to 96 hours before resuming strenuous activities. In our experience, patients who suffered mild EHI with marked hyperthermia during physical fitness training have occasionally exhibited 3 to 4 days of diminished mental function, affecting their performance of tasks that require concentration, calculations, and judgment. Those with severe EHI may require several weeks or months before resuming strenuous activities.

EPIDEMIOLOGY AND PREVENTION OF EXERTIONAL HEAT ILLNESS IN THE MILITARY

EHI has been a substantial problem in military operations and training; it occurs primarily with sustained exertion, especially in a warm, humid environment.[200,201] When adequate hydration and emergency medical management are unavailable, each case is a potential death.[1,55,83] EHI has been recognized since biblical times and is mentioned in accounts of several ancient military campaigns.[202,203] Recognition of its relationship with warm environmental temperatures was solidified by 1900. During World War I there were particularly high rates of EHI in British troops in Mesopotamia, with at least 600 deaths.[204] By 1917 heatstroke stations were authorized in the British military; ice was provided daily, abundant drinking water was made available, and, as far as possible, troops were relieved of duty between 10 AM and 4 PM.[205,206]

During World War II the lessons of World War I had to be relearned[207] (not unusual in the history of warfare). British operations in the Middle East had particularly high rates of heat illness.[208,209] The biggest problem for American troops during World War II was in stateside training. During the war there were about 250 reported deaths from heatstroke, mostly at training centers in the southern United States.[53,210–215] After the war, EHI associated with US military training in hot weather continued to result in significant morbidity.[216–218] During the 1967 Six-Day War between Egypt and Israel, intelligence reports indicated that the Egyptians suffered about 20,000 deaths with no visible wounds (apparently caused by dehydration or heatstroke), while Israeli casualties were minimal.[83]

Although many other reports of EHI morbidity in military training and operations have been published,[180,215,219–221] the largest group of military trainees who have been thoroughly studied is recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina (MCRD-PI). Marine Corps recruit training was formalized at Parris Island in 1915 and consisted of 2 months of drill, physical exercise, personal combat, and intensive marksmanship qualification. Since then, recruit training has continued, varying from 4 to 12 weeks in length depending on specific wartime needs. Currently, all female and about half of all male Marine Corps recruits undergo their 12-week basic training course at MCRD-PI. The area has a semitropical climate with high temperatures and humidity, especially during the summer months. Extensive studies on EHI have been conducted in this population since the 1950s,[7,38,90,130,189,222] including those that developed the wet bulb globe temperature (WBGT) index as a guide for regulating the amount of strenuous exercise during training.[217,223–229]

Recent studies at MCRD-PI[56–58,138,144–146,230] have characterized the time patterns for occurrence of EHI during basic training. From 1982 through 1991, the average rate of occurrence of EHI in recruits was 0.7% during their 12 weeks of basic training, with

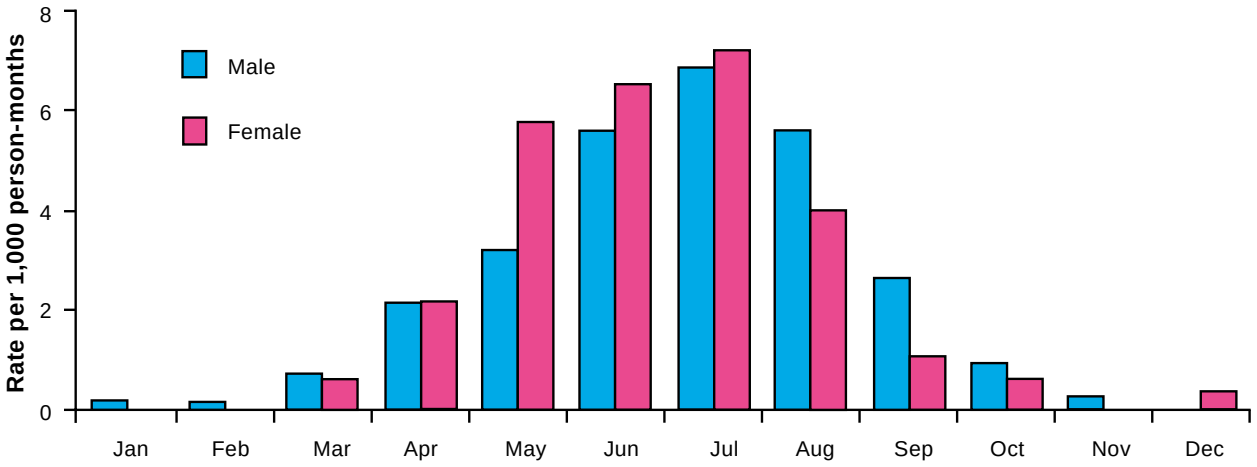

Fig. 7-3. Rates of exertional heat illness in Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by gender and month for the years 1982 through 1991. Rates are cases per 1,000 recruit person-months. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):357.

rates up to 2% during the summer months; 11% of men and none of the women were hospitalized.[58] Figure 7-3 shows the number of recruit cases per 1,000 person-months by month of year, and Figure 7-4 shows the distribution of recruit cases by hour of day. These illustrations show that case rates were highest during the summer months, and that most of the cases occurred during the 7-to-9 AM exercise period. When these early morning cases were categorized by WBGT at the hour of occurrence, we found that the case rates increased dramatically as the WBGT rose above 65°F (18°C) (Figure 7-5). When the same cases were categorized by the maximum WBGT of the previous day, we found the same pat-

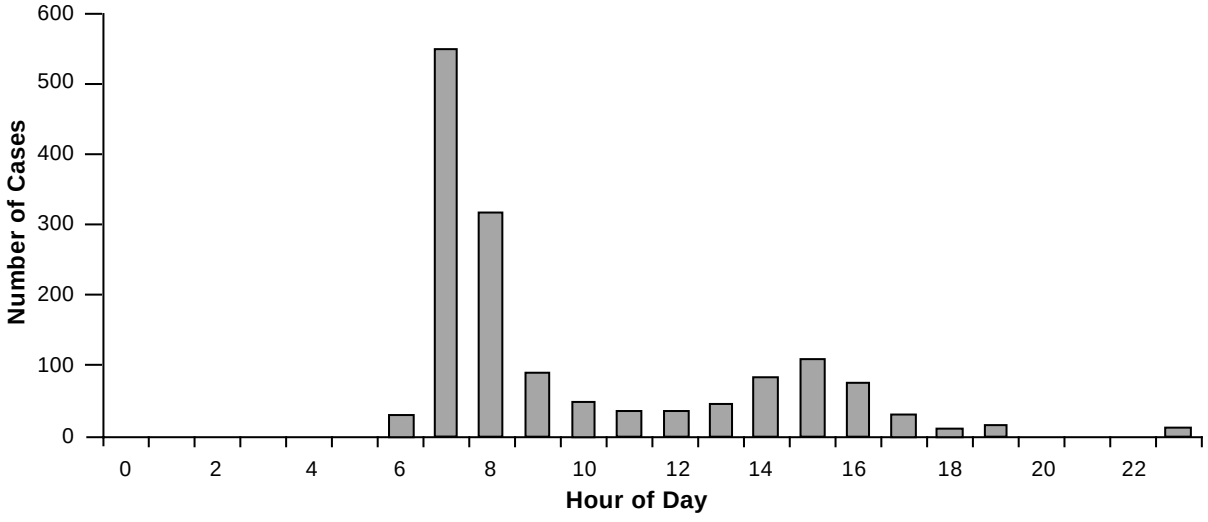

Fig. 7-4. Numbers of cases of exertional heat illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by hour of the day of illness for the years 1982 through 1991. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):357.

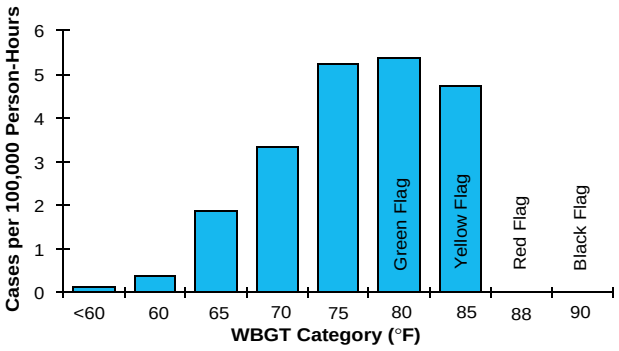

Fig. 7-5. Rates of exertional heat illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, by wet bulb globe temperature (WBGT) between 0700 and 0900 hours for the years 1982 through 1991. There were very few person-hours of exposure at WBGT of 88°F or above. Rates are cases per 100,000 recruit person-hours of physical activity. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):358.

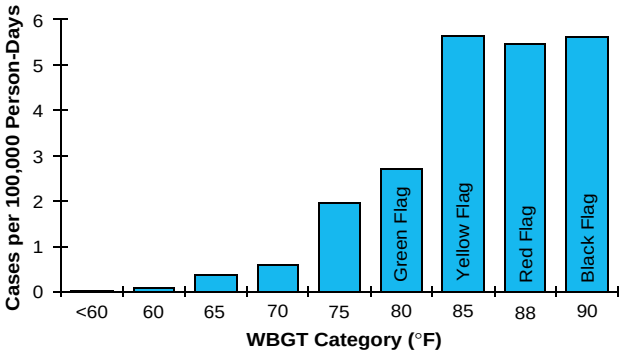

Fig. 7-6. Rates of exertional heat illness by maximum wet bulb globe temperature (WBGT) category on the day *prior* to the day of illness among Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina. The cases are only those that occurred between 0700 and 0900 hours, for the years 1982 through 1991. Rates are cases per 100,000 recruit person-days. Adapted with permission from Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med.* 1996;67(4):358.

tern, with case rates rising rapidly as the prior day's maximum WBGT rose above 65°F (18°C) (Figure 7-6). The mechanism for this carryover risk may involve persistent dehydration, fatigue, or undescribed metabolic factors, in addition to the influence of the prior day's heat on the early morning temperature. When the prior day's peak temperatures were analyzed in strata that held the early morning temperatures constant, a 4-fold gradient in EHI susceptibility persisted. These figures illustrate the effect of weather conditions on EHI rates and suggest that the effects of heat exposure may also carry over to the next day.[58]

Risk Factors for Exertional Heat Illness

Numerous risk factors have been identified for EHI, which relate primarily to (*a*) weather, discussed above, (*b*) acclimatization, (*c*) physical fitness, and (*d*) training circumstances.[200,201,215,222,226,230–235]

Acclimatization

Acclimatization reduces the risk for EHI by increasing the sweat rate, decreasing the sodium content of sweat, and initiating sweating at a lower body core temperature.[32,40,236] In addition, acclimatization increases cardiovascular capacity and blood volume.[31] Acclimatization increases the efficiency of heat dissipation, but its benefits can be decreased or nullified by sleep loss, infection, dehydration, salt depletion, or the use of drugs or alcohol.[21,237] A sensible acclimatization program is important during the first 2 to 4 weeks in a warm environment. This should include daily exercise in the heat of gradually increasing intensity and duration (< 2 h), accompanied by copious quantities of water and additional salt in the diet (in the form of high-salt foods, not salt tablets).[20,238–240]

Physical Fitness and Body Type

Poor physical fitness is an important risk factor for EHI.[73,235,240–243] For example, at MCRD-PI we documented at least a 3-fold higher risk for EHI during basic training in recruits with poor fitness (for men, 1.5-mile run-time ≥ 12 min), compared with those who were more fit (1.5-mile run-time < 10 min).[230] This experience is consistent with the above discussion of cardiovascular stresses in EHI. In addition, body type (depicted through the body mass index [BMI], calculated as weight/height^2) appears to relate to risk for EHI.[233,244–246] At MCRD-PI there was about a 3-fold higher risk for EHI during basic training in men recruits who were heavyset (BMI ≥ 26 kg/m^2), compared with those who were thin (BMI < 22 kg/m^2).[230] This relationship with BMI does not appear to hold for women recruits. The combined risk for EHI in men who were heavyset and had slow run-times was about 8-fold higher than in those who were thin and had fast run-times (Table 7-2). The

TABLE 7-2

ODDS RATIOS (95% CONFIDENCE INTERVALS) FOR DEVELOPING EXERTIONAL HEAT ILLNESS DURING MARINE CORPS BASIC TRAINING,* 1988–1992

Body Mass Index	1.5-Mile Run-Time (min)		
(kg/m^2)	< 10	10 – < 12	3 12
< 22	1.0 (referent)	1.5 (0.7–3.2)	3.5 (1.4–8.8)
22– < 26	1.6 (0.6–3.8)	2.0 (0.9–4.2)	8.5 (3.8–19)
3 26	3.7 (0.9–15)	3.3 (1.5–7.1)	8.8 (4.1–19)

Low risk (OR 2 2): BMI < 26 kg/m^2 and 1.5-mile run-time < 12 min
Medium risk (2 < OR < 4): BMI 3 26 kg/m^2 and 1.5-mile run-time < 12 min, or BMI < 22 kg/m^2 and 1.5-mile run-time 3 12 min
High risk (OR 3 4): BMI 3 22 kg/m^2 and 1.5-mile run-time 3 12 min
* Male recruits, Marine Corps Recruit Depot, Parris Island, South Carolina
BMI: body mass index (weight/height^2)
OR: odds ratios
Adapted with permission from Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc.* 1996;28:941.

18% of men recruits with these high-risk characteristics (BMI ≥ 22 kg/m^2 and 1.5-mile run-time ≥ 12 min) accounted for 47% of the cases of EHI that occurred during basic training at MCRD-PI in 1988 through 1992 (Figure 7-7).[230]

Circumstances of Training

Training circumstances provide critical risk factors for EHI.[247] The early recruit studies discussed above established that most heat illness cases at Parris Island occurred with strenuous exercise when the WBGT was 80°F (27°C) or higher.[58,225] In the 1950s, more than half of heat illness cases occurred during daytime infantry drill and training marches.[227] Subsequent studies demonstrated that as the WBGT rose above 80°F (27°C), heat illness rates were reduced by progressively limiting the level of exercise, increasing rest periods, increasing hydration, and reducing heat-retaining clothing.[223] These modifications were implemented in a standard manner for four categories of high WBGT (green flag = 82°F–84.9°F, yellow flag = 85°F–87.9°F, red flag = 88°F–89.9°F, and black flag = 90.0°F or higher). As a rough guide, exercise continued with caution during green flag, strenuous exercise (eg, "marching at standard cadence") was suspended for first-phase recruits during yellow flag, strenuous exercise was curtailed for all recruits during red flag, and all outdoor physical training was suspended during black flag conditions.[20,248,249] An important consequence of these regulations has been the scheduling of strenuous exercise in the cooler, early morning hours (as seen in Figure 7-4).

The flag conditions regulating authorized training activities by WBGT were designed for marching, rather than running, yet the focus of physical conditioning in the past 2 decades has been more on middle-distance running (1–3 miles) than marching.[49] Marching with a heavy load produces about 500 W of heat, while running at 8 to 10 mph produces 1,000 to 1,200 W.[16,149,223] This focus on higher-metabolic-rate training activities leads us to expect EHI casualties at lower WBGT levels than generally occur with marching. This has been demonstrated in studies showing increased risk for EHI at WBGT levels as low as 65°F (18°C) (see Figure 7-5).[58]

Prevention of Exertional Heat Illness

Provision of adequate rest periods during training is particularly important in preventing EHI, with

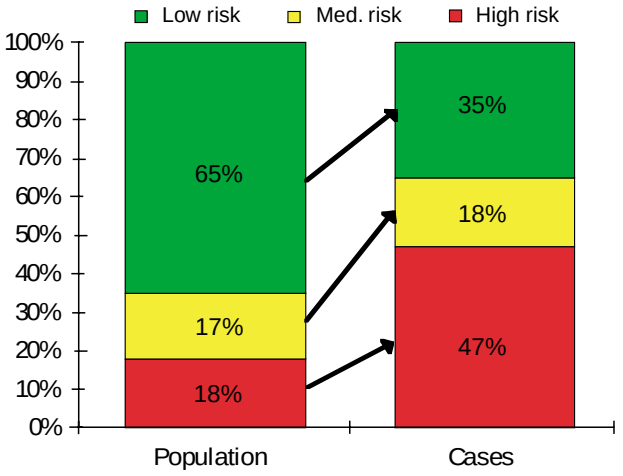

Fig. 7-7. Frequency distribution of cases and population by risk status among male Marine Corps recruits at the Marine Corps Recruit Depot, Parris Island, South Carolina, for the years 1988 through 1992. High risk: body mass index (BMI) ≥ 22 kg/m^2 and 1.5-mile run-time ≥ 12 min; medium risk: BMI ≥ 26 kg/m^2 and 1.5-mile run-time < 12 min, or BMI < 22 kg/m^2 and 1.5-mile run-time ≥ 12 min; low risk: BMI < 26 kg/m^2 and 1.5-mile run-time < 12 min.
Adapted with permission from Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc.* 1996;28(8):942.

restrictions on work activities determined by physical fitness, acclimatization, medical status, and weather conditions.[225,250,251] Strenuous activities (particularly running) should be scheduled for the coolest part of the day.[249] Work/rest cycles have been developed by USARIEM for routine military activities, and are reflected in updated regulations regarding flag conditions.[49,122,252] Exhibit 7-7 provides the current recommendations. This chart does not address exercise that exceeds hard work (more than 600 W), such as running at an 8- to 10-mph pace (1,000-1,200 W). Burr[49(Appendix D)] has provided estimates of the maximum minutes of running at a 6-mph pace, which will produce 5% EHI casualties at different levels of WBGT. For example, he estimates that at a WBGT of 90°F, 22 minutes of running will produce 5% casualties; at 82°F, 34 minutes; and at levels lower than 60°F, 50 to 60 minutes. Most trainers would consider a 5% casualty rate much too high, and Exhibit 7-7 provides guidelines (for work at < 600 W), which should be safe for at least 4 hours, given the stated assumptions. These guidelines were designed to safely maintain continuous operations for at least 4 hours, not for an intense activity that leaves one exhausted at the end and requires an extended period of rest and recuperation in com-

EXHIBIT 7-7

FLUID REPLACEMENT GUIDELINES* FOR HOT WEATHER TRAINING (AVERAGE ACCLIMATIZED SOLDIER WEARING THE BATTLE DRESS UNIFORM IN HOT WEATHER)

		Easy Work		Moderate Work		Hard Work	
WBGT Index (F°)	Heat Category (Flag Color)	Work/Rest Cycle (min)	Water Intake (qt/h)	Work/Rest Cycle (min)	Water Intake (qt/h)	Work/Rest Cycle (min)	Water Intake (qt/h)
78–81.9	1 (green)	NL	0.5	NL	0.75	40/20	0.75
82–84.9	2 (green)	NL	0.5	50/10	0.75	30/30	1.0
85–87.9	3 (yellow)	NL	0.75	40/20	0.75	30/30	1.0
88–89.9	4 (red)	NL	0.75	30/30	0.75	20/40	1.0
> 90	5 (black)	50/10	1.0	20/40	1.0	10/50	1.0

* The work/rest cycle times and fluid replacement volumes will sustain performance and hydration for at least 4 hours of work in the specified heat category. Individual water needs will vary ± 0.25 qt/h.
NL: no limit to work time per hour. Rest: minimal physical activity (sitting or standing) and should be accomplished in the shade if possible.
CAUTION: Hourly fluid intake should not exceed 1.5 qt. Daily fluid intake should not exceed 12 qt.
Mission-oriented protective posture (MOPP) gear adds 10°F to WBGT index.

Examples of work intensities:

Easy Work	Moderate Work	Hard Work
Weapon maintenance	Walking on loose sand at 2.5 mph, no load	Walking on hard surface at 3.5 mph, 3 40-lb load
Walking on hard surface at 2.5 mph, 2 30-lb load	Walking on a hard surface at 3.5 mph, 2 40-lb load	Walking on loose sand at 2.5 mph with load
Manual of arms	Calisthenics	
Marksmanship training	Patrolling	
Drill and ceremony	Individual movement techniques (ie, low crawl, high crawl)	
	Defensive position construction	
	Field assaults	

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fortable surroundings. It is clear that weather conditions determine the safety of physical activity and must be considered in scheduling the timing, intensity, and duration of training and operations. It also appears that the effects of heat and exercise stress exposure accumulate, and, as discussed above, the prior day's weather and amount of exercise should be considered in scheduling the day's activities.[58,207,253]

It is important to limit the total amount of strenuous exercise to the minimum necessary for mission accomplishment. Trainees are at particularly high risk for EHI because of their need to succeed in a competitive environment.[39,241] Trainees often work at rates higher than the highest work rates used to determine traditional work/rest guidance. In addition, these individuals are often at low levels of acclimatization and aerobic fitness. They may also have febrile responses to immunization or infection and be experiencing sleep loss and psychological stress. The motto "Train, Not Pain" needs to replace the popular phrase, "No Pain, No Gain." These principles apply to protection against musculoskeletal injury as well as EHI.[254]

Prevention of EHI requires minimizing the use of heavy or retentive clothing, particularly helmets, jackets, and vapor-impermeable materials.[255,256] Lightweight, loose-fitting clothing should be worn to protect from sunburn and allow air circulation to enhance sweat evaporation.[82,257–260] Outdoors, provision of shade and air movement at the training areas is important; indoors, provision of air conditioning, ventilation, low vapor pressure, heat insulation, and reflective shielding will minimize the effects of heat stress. Individuals should spend as much of the day as possible in cool, low-humidity surroundings with good air movement (only 2 h/d of exercise in the heat are needed for heat acclimatization).[238,261,262]

Certain medical conditions and medications can put an individual at high risk for developing heat illness.[50,139,162,170,179,242,263–269] These can make adjustment to hot environments more difficult and include acute illness (especially infections), febrile conditions, chronic illness, prior history of heatstroke, pregnancy, obesity, skin disorders, certain genetic conditions, sunburn, and poor hygiene (ie, dirt that clogs pores, impairs sweating, and insulates skin, as well as predisposes to infection). Medications, "ergogenic" nutritional supplements (with ephedra alkaloids), alcohol, caffeine, loss of sleep, and missed meals can also predispose to EHI.[50,270,271] Categories of pharmacological agents that interfere with thermoregulatory mechanisms are listed in Exhibit 6-2 of Chapter 6, Prevention of Heat Illness.

Prevention of EHI during strenuous exercise requires proper provision for water and salt requirements.[8,33,64,272–274] Leaders must force and verify hydration.[21,83,275] Palatable water must be available nearby, and water intake must be monitored.[50,276,277] Water requirements are not reduced by any form of training or acclimatization; in fact, they are increased with acclimatization owing to increased sweating. Tolerance to dehydration cannot be developed, and water supplies are as important for survival in a military environment as are food and ammunition.[83] Exhibit 7-8 illustrates the extreme hazards of strenuous exercise when overzealously conducted in a warm or humid environment. It also reminds us that in the 1950s, "water discipline" referred to restricting water intake under the false theory that one could be acclimatized to water deprivation. During the 1960s it became clear that acclimatization actually increases the need for water (due to increased sweating), and that those theories of the 1950s were fatally flawed.

Thirst is not an adequate guide to water needs because it lags behind water deficits by at least 1 pint.[18,50,273,278] Urinary volume and color, dryness of the mouth, changes in body weight, and orthostatic blood pressure (tilt tests) can be used as guides to adequacy of hydration.[277] An advantage to carbohydrate or electrolyte beverages beyond their palatability has not been established, and high-sugar solutions may impede water absorption in the gut.[279–283] Salt losses should be made up with the usual military high-salt diet, not with salt tablets.[49,82,84] In EHI, water losses usually exceed salt losses, and salt replacement should follow (not precede) water replacement. Replacement of salt without water will exacerbate cellular dehydration, increase water requirements, and may induce profound hypernatremia.[73] Thus, we strongly recommend against the use of salt tablets. The following is a good rule of thumb: *Replace water losses hour by hour, and salt losses day by day*.

Personal education is the mainstay of prevention of EHI. Each individual should be fully aware of EHI, its signs, and its prevention.[149,171,234,284,285] Personal awareness and common sense will avoid most problems.[166,191] Individuals should understand the early symptoms and signs of EHI, and increased susceptibility with use of certain medications and during and after recovery from acute (even minor) illness. It is important to avoid excessive fatigue, get adequate sleep, maintain calories and adequate salt in the diet, and avoid alcohol and caffeine. Each individual must be responsible to maintain adequate water intake, which requires drinking when not thirsty and monitoring urinary volume and color, weight changes, and so forth.[262] Individuals

EXHIBIT 7-8

HEAT DEATHS DURING OFFICER CANDIDATE SCHOOL

July 1953

The Korean War was still a threat to world peace, and young American men were being drafted into a war that was inevitable but had not been accurately anticipated by either our intelligence agency or our armed forces. Among the best fighters of this fledgling United Nations force were the US Marines, whose members were still for the most part volunteers, and whose officer corps was the product of the Naval Academy, Naval Reserve Officer Training Corps graduates, a few promoted from the enlisted ranks, or recent volunteers who were commissioned after joining the Platoon Leaders Class while in college or from the Officer's Candidate Course immediately after graduation. This last group entered the fray with no knowledge of the military, were inducted with no physical requirements other than a routine and cursory physical examination, and were given a date to report to the US Marine Corps Base at Quantico, Virginia. It was into this group that I fell.

I clearly remember one prospective fellow candidate at the induction center who seemed overweight and exhibited a notable lack of muscular attributes. I had been a college football player, varsity wrestler, and tennis player and had some misgivings about my own capacity for enduring the ordeal that lay ahead. I queried this fellow traveler about his ambitions and selection of the Marines as his choice to serve. I remember his reply, words to the effect of "Well, I had a good time at college and I thought this might be a good way to get back into shape."

The day we reported in was no small awakening to what was in store. We ran everywhere, were insulted and humiliated by the noncommissioned officers, learned what fear was, and finally learned how to get along in a totally unmanageable situation. We all struggled, but soon it became obvious that some of our contingent had chosen their branch of service unwisely and were doomed to fail from the start. The physical demands grew rapidly, and I found myself at the point of exhaustion many times. Some struggled along on pride alone, and I wondered how they had lasted as long as they did.

We were in our third week, as I remember it. We fell out in the dark of morning, did 20 or so minutes of calisthenics, swinging our 9-plus-pound rifles over our heads (the "muzzles and butts" routine), then off to the mess hall running in cadence. I lost more than the usual amount of sweat that morning—it seemed unusually warm that day. Upon returning to the barracks we underwent the usual policing of the area; "squared away" our gear; and went out into the warm, sunlit day with full packs, ammo belts, helmets, and M-1 Garand rifles. After a brief "All present and accounted for" response to the sergeant, we were off on our first "conditioning hike" along the fire line—a trail carved out in the hillsides, partially as a firebreak but also to keep the power lines free of tree limbs.

We each had one canteen of water and were expected to take salt tablets, although whether or not we took them is vague in my memory. We had been told about water discipline—not to squander in answer to thirst—and that part of our training was to acclimate to water need by disciplining ourselves and not satiating our thirsts. As we moved along at a quickstep, Company C was in the lead; we young laden-down candidates struggled to keep pace with our unladen sergeants, which kept us gasping for air. Across the plain and then the hills. Initially none faltered, but our line began to "string out" and I first became acquainted with the word "straggler." The air was unusually still and seemed heavy. As the sun climbed the heat became intense, and off we went up another hill. Finally we halted, gasping, soaked with sweat, and crumpled to the ground for a very short rest. We were ordered to take out our canteens and pour some of our water on the ground. Believe me, we did so sparingly. We were to learn that survival in the rugged clime of Korea would depend on how much more ammunition we could carry than water. Then we were off again.

We stopped about every hour and were allowed to drink, but we were all overly cautious and were never satiated. Our water had become our most precious item. By noon the heat had become oppressive and I realized how wobbly my legs were; others were stumbling, swearing silently between gasps, and longing for the next break, the next sip. Rumors were rampant: everything from "put a stone in your mouth and suck on it" (supposedly to quench thirst) to "there is a water buffalo (a mobile water tank) a few hills hence."

(**Exhibit 7-8** *continues*)

Exhibit 7-8 *continued*

In recollection the details become murky. We must have had hot chow in the field at noon and had our fill of water. The rumor came down that the temperature was over 95°F. Some had dropped out, but none in my platoon; they were apparently on their way back in trucks. Then we were off again. The rests were unrewarding; each successive time it became more difficult to get started again. The sergeant would cry out "Saddle up." Finally we were on our way back, and I remember it being a little easier; however, we could not possibly have acclimat**iz**ed so quickly. Finally, the last hill, and the plain loomed into sight, which seemed to invigorate those of us in the lead.

When standing in formation at the end of the ordeal, I found myself having to consciously maintain my balance; then finally came the cry, "Fall out." The rush for the sink faucets and water coolers was insane—no longer were we gentlemen—some were finally shoved aside to keep the line moving. Others headed to the showers to drink but realized that all had to share; some showered with their fatigues on. Within half an hour we were cleansed, replenished with water, dressed in our khakis, and back in formation.

The rumor was passed that a candidate in the opposite company had collapsed near the end of the maneuver, had been dragged across the plain by his comrades, and was no longer breathing when he was dropped in front of the company area. We were marched to the front of the mess hall where we stood in formation in the hot afternoon sun; the temperature had fallen a few degrees from 98°F. A candidate from the company in front of ours suddenly collapsed and was taken to the Naval Hospital at Quantico, where he expired in the throes of hyperthermia. A third candidate became delirious that evening and was rushed to the Naval Hospital at Bethesda, where he succumbed of "heatstroke."

Within a week some of the training command were relieved of their positions, a congressional investigation was ongoing, and the "conditioning hikes," as our maneuver had been listed on the schedule, were now entitled "terrain appreciation" and were without full gear. We all carried folding camp stools; rested every 50 minutes; and although the theory of water discipline and acclimation to water deprivation was not disavowed, we were no longer asked to pour some of our precious fluid on the ground. The water buffaloes were always in place along the trails.

I later learned that the first to die that day had been the young officer candidate I had met at the induction center who had joined to get himself into shape. The other two were unknown to me. Four years later I resigned my regular commission in the Marine Corps. By coincidence I received my discharge papers in San Francisco from the same lieutenant colonel who had commanded that ill-fated battalion. He was now a major.

Exhibit prepared for this textbook by Brigadier General John Hutton, Medical Corps, US Army (Ret), Department of Surgery, Uniformed Services University of the Health Sciences, Bethesda, Maryland 20814-4799

must be aware of the need to hydrate ahead of thirst and before and during exercise. Trainees should wear appropriate clothing, seek shade, utilize rest periods, and adjust the intensity and duration of exercise according to environmental conditions and their own sense of well-being.[20,248,249,258,275,286] Buddies can recognize the symptoms and signs in each other, assisting in early recognition and management.[49,274] Each individual must remember the motto "Train, Not Pain" to prevent serious complications of EHI.

SURVEILLANCE AND REPORTING OF EXERTIONAL HEAT ILLNESS

Surveillance for EHI provides data that are essential for maintaining a healthy population and evaluating prevention and management policies.[287] Only through data-based policy decision-making can prevention of EHI and its serious complications be minimized.[200,201] Management and prevention of EHI requires meticulous record keeping that includes both the circumstances under which the illness occurred and the time course of clinical symptoms and signs. An effective heat illness surveillance and prevention program requires active monitoring not only of EHI cases, outcomes, and all exercise-related deaths, but also of training activities, personal risk factors in the training population, and weather conditions. One must be constantly aware of the amount and timing of exercise, adherence to rest cycle and intensity guide-

lines, and training circumstances. Effective prevention requires knowledge not only of the availability of water and shade, for example, but also of the planned and actual consumption of water and use of shade. Attention must also be given to the clothing and gear involved and to the availability and capability of on-site medical personnel, equipment (including thermometers, ice, and intravenous fluids), and transportation to emergency care facilities.

Weather monitoring involves both hourly determination of the WBGT at the training sites and communication of those measurements to the trainers so they can appropriately adjust their vigilance and training activities. In the military, this is often done utilizing a flag system, which corresponds to heat categories described in the regulations (see Exhibit 7-7), with corresponding activity limitations.[20,49,248,249,275]

Monitoring of personal risk factors in the training population is more difficult, as the physical fitness, acclimatization, and medical status of each individual must be continuously assessed. Keeping track of minor illnesses, medications, hydration, diet, sleep patterns, and use of alcohol and caffeine is somewhat challenging. However, these factors may be critical in susceptibility to serious EHI. Activities need to be adapted to the age, physical capacity, and current fitness levels and medical status of each individual.

Monitoring of cases and medical outcomes involves assessment of triage and immediate care by an assigned acute care or surveillance officer. Each case must be reviewed and classified as to type and severity, with attention given to risk factors and training circumstances. Patterns of illness and relation to training activities can then be evaluated and trends analyzed. Clusters of cases can be explored to determine the specific circumstances that have produced these casualties. The discussion that follows pertains to military surveillance methodology and presupposes that the casualty has entered the military medical system.

A set of standard clinical forms is attached to the end of this chapter, which can be used both as the clinical record and as a means to collect data that are necessary for appropriate EHI surveillance. (The attached forms can be duplicated by the reader for clinical use.) The record should begin in the field (the EHI Field Form is usually completed by a corpsman or medic), with observations by the field medical team of the presenting clinical symptoms, vital signs, neurological status, and early time course of the illness. The field medical team should also provide information regarding the circumstances of EHI occurrence (eg, training events, weather conditions, clothing, and a narrative description of the clinical presentation).

In the medical clinic or emergency department (the two EHI Clinic Forms are usually completed by a nurse), assessment of vital signs and neurological status should continue every 5 minutes until the patient is stable and cooled to a temperature that remains below 102°F (39°C). The listing of symptoms and signs during the first hour of illness and of risk factors (eg, height, weight, physical fitness, amount of fluids consumed, sleep loss, coexistent illness) are important, in addition to notation of the treatments given, diagnostic tests obtained, and a medical review of systems.

The medical workup can be recorded in an efficient manner that facilitates standardization of laboratory workup, diagnoses, and dispositions (the EHI Medical Form is usually completed by the primary medical provider). This form contains blocks for recording fluid intake and output and for results of diagnostic tests, and blocks for diagnosis and disposition of the patient, as well as an open-ended section for medical history (*s*ubjective), physical examination (*o*bjective), *a*ssessment, and *p*lan (also called the SOAP notes). Summary surveillance reports can be generated from the clinical forms, and should include counts of EHI cases by severity, training event, and weather conditions.

SUMMARY

EHI represents a broad spectrum of disease associated primarily with exercise in a warm environment. Symptoms and signs of exertional heat exhaustion, heat injury, heatstroke, and rhabdomyolysis result from detrimental consequences of elevated temperature and circulatory and metabolic products of exercise and the body's thermoregulatory response. Hyperthermia, dehydration and electrolyte imbalance, neurological manifestations, impaired renal function, muscle symptoms, and rhabdomyolysis comprise the predominant clinical features of EHI. These require diligent assessment and aggressive management to prevent progression to more-severe and often life-threatening illness. Progression to severe EHI usually involves muscle- and liver-cell breakdown (cell lysis syndrome), metabolic acidosis, acute renal failure, cardiovascular collapse, DIC, coma, and death. It generally requires management in an intensive care unit.

Early clinical management of EHI requires immediate assessment of vital signs and mental status in the field, with rapid cooling and rehydration.

Moderate and severe cases need frequent reassessment and laboratory evaluation. Aggressive cooling and correction of fluid, electrolyte, and acid–base problems constitute the mainstay of treatment. With rapid and aggressive treatment, guided by meticulous clinical and laboratory reassessment, serious complications are rare. It must be remembered, however, that subtle deficits and increased susceptibility to heat stress persist for several days following even moderate EHI, so casualties should not be immediately returned to regular strenuous activities or mentally demanding tasks.

EHI has been prominent in military training and operations, representing an important cause of morbidity, as well as significant mortality. Acclimatization to hot environments provides some mitigation of EHI morbidity. However, given extreme environmental circumstances and excessive workload, as well as various individual medical and other risk factors, it is always a threat during training and operations.

The key to prevention of EHI is education both of unit leaders and individuals to maintain activities in accordance with the environmental conditions, their physical fitness and acclimatization status, and their individual medical and other circumstances that determine susceptibility. They must remember the mottoes "Train, Not Pain" and "Replace water hour by hour, and salt day by day." Personal awareness and common sense will avoid most problems. Buddies can recognize the symptoms and signs in each other, assisting in early recognition and management of EHI.

Surveillance for EHI encompasses not just tabulating EHI cases but also assessing severity and monitoring environmental conditions; training circumstances; adherence to work/rest cycle guidelines; and individual physical fitness, hydration, and medical status.

REFERENCES

1. Knochel JP. Heat stroke and related heat stress disorders. *Dis Mon*. 1989;35:301–378.

2. Yarbrough BE, Hubbard RW. Heat-related illness. In: Auerbach PS, Geehr EC, eds. *Management of Wilderness and Environmental Emergencies*. 2nd ed. St. Louis, Mo: CV Mosby; 1989: Chap 5.

3. Tek D, Olshaker JS. Heat illness. *Emerg Med Clin North Am*. 1992;10:299–310.

4. Hubbard RW, Gaffin SL, Squire DL. Heat-related illnesses. In: Auerbach PS, ed. *Wilderness Medicine: Management of Wilderness and Environmental Emergencies*. 3rd ed. St. Louis, Mo: CV Mosby; 1995: 167–212.

5. Knochel JP. Environmental heat illness. An eclectic review. *Arch Intern Med*. 1974;133:841–864.

6. O'Donnell TF Jr. The hemodynamic and metabolic alterations associated with acute heat stress injury in marathon runners. *Ann N Y Acad Sci*. 1977;301:262–269.

7. Costrini AM, Pitt HA, Gustafson AB, Uddin DE. Cardiovascular and metabolic manifestations of heat stroke and severe heat exhaustion. *Am J Med*. 1979;66:296–302.

8. Hubbard RW, Armstrong LE. The heat illnesses: Biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 8.

9. Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 3.

10. Iampietro PF. Exercise in hot environments. In: Shephard RJ, ed. *Frontiers of Fitness*. Springfield, Ill: Charles C Thomas; 1971: Chap 19.

11. Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology*. Boston, Mass: Little, Brown & Co; 1995: Chap 31.

12. Rowell LB. Cardiovascular aspects of human thermoregulation. *Circ Res*. 1983;52:367–379.

13. Rowell LB. Cardiovascular adjustments to thermal stress. In: Shepherd JT, Abboud FM, eds. *Handbook of Physiology.* Section 2, Vol 3. *The Cardiovascular System*. Bethesda, Md: American Physiological Society; 1983: Chap 27.

14. Eastman Kodak Company. Circulatory and respiratory adjustments to work. In: Eastman Kodak Co. *Ergonomic Design for People at Work*. Vol 2. New York, NY: Van Nostrand Reinhold; 1986: Vol 2, Chap 3.

15. Fogoros RN. "Runner's trots:" Gastrointestinal disturbances in runners. *JAMA*. 1980;243:1743–1744.

16. Simon HB. Exercise, health, and sports medicine. In: Rubenstein E, Federman DD, eds. *Scientific American Medicine*. New York, NY: Scientific American, Inc; 1992: 1–35.

17. Hertig BA, Belding HS, Kraning KK, Batterton DL, Smith CR, Sargent F. Artificial acclimatization of women to heat. *J Appl Physiol*. 1963;18:383–386.

18. Strydom NB, Wyndham CH, Williams CG, et al. Acclimatization to humid heat and the role of physical conditioning. *J Appl Physiol*. 1966;21:636–642.

19. Senay LC, Mitchell D, Wyndham CH. Acclimatization in a hot, humid environment: Body fluid adjustments. *J Appl Physiol*. 1976;40:786–796.

20. US Departments of the Army, the Navy, and the Air Force. *Prevention, Treatment and Control of Heat Injury*. Washington, DC: Departments of the Army, the Navy and the Air Force: 1980. TB MED 507, NAVMED P-5052-5, AFP 160-1.

21. Brooks JR, Scott A. *Prevention and Treatment of Heat and Cold Stress Injuries.* Norfolk, Va: Navy Environmental Health Center; 1992. NEHC-TM92-6.

22. Canine MK, Derion T, Heaney JH, Pozos R. An annotated bibliography of heat tolerance: Regarding gender differences. San Diego, Calif: Naval Health Research Center; 1993. TN 93-1A.

23. Robinson S, Turrell ES, Belding HS, Horvath SM. Rapid acclimatization to work in hot climates. *Am J Physiol*. 1943;140:168–176.

24. Bass DE, Kleeman CR, Quinn M, Henschel A, Hegnauer AH. Mechanisms of acclimatization to heat in man. *Medicine*. 1955;34:323–380.

25. Gilat T, Shibolet S, Sohar E. The mechanism of heatstroke. *J Trop Med*. 1963;66:204–212.

26. Shvartz E, Magazanik A, Glick Z. Thermal responses during training in a temperate climate. *J Appl Physiol*. 1974;36:572–576.

27. Shvartz E, Shibolet S, Meroz A, Magazanik A, Shapiro Y. Prediction of heat tolerance from heart rate and rectal temperature in a temperate environment. *J Appl Physiol*. 1977;43:684–688.

28. Wells CL, Constable SH, Haan AL. Training and acclimatization: Effects on responses to exercise in a desert environment. *Aviat Space Environ Med*. 1980;51:105–112.

29. Frye AJ, Kamon E. Responses to dry heat of men and women with similar aerobic capacities. *J Appl Physiol*. 1981;50:65–70.

30. Armstrong LE, Costill DL, Fink WJ. Changes in body water and electrolytes during heat acclimation: Effects of dietary sodium. *Aviat Space Environ Med*. 1987;58:143–148.

31. Nielsen B, Hales JRS, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol*. 1993;460:467–485.

32. Wenger CB. Human heat acclimatization. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 4.

33. Epstein Y, Sohar E. Fluid balance in hot climates: Sweating, water intake, and prevention of dehydration. *Public Health Rev*. 1985;13:115–137.

34. Davies CTM, Brotherhood JR, Zeidifard E. Temperature regulation during severe exercise with some observations on effects of skin wetting. *J Appl Physiol*. 1976;41:772–776.

35. Pugh LGCE, Corbett JL, Johnson RH. Rectal temperatures, weight losses, and sweat rates in marathon running. *J Appl Physiol.* 1967;23:347–352.

36. Costill DL, Fink WJ. Plasma volume changes following exercise and thermal dehydration. *J Appl Physiol*. 1974;37:521–525.

37. Nadel ER, Wenger CB, Roberts MF, Stolwijk JAJ, Cafarrelli E. Physiological defense against hyperthermia of exercise. *Ann N Y Acad Sci*. 1977;301:98–109.

38. O'Donnell TF Jr, Clowes GHA Jr. The circulatory abnormalities of heat stroke. *N Engl J Med*. 1972;287:734–737.

39. Shapiro Y, Seidman DS. Field and clinical observations of exertional heat stroke patients. *Med Sci Sports Exerc*. 1990;22:6–14.

40. Ellis FP. Heat illness, I: Epidemiology, II: Pathogenesis, III: Acclimatization. *Trans R Soc Trop Med Hyg*. 1976;70:402–425.

41. Schrier RW, Hano J, Keller HI, et al. Renal, metabolic, and circulatory responses to heat and exercise. Studies in military recruits during summer training, with implications for acute renal failure. *Ann Intern Med*. 1970;73:213–223.

42. Shibolet S, Fisher S, Gilat T, Bank H, Heller H. Fibrinolysis and hemorrhages in fatal heatstroke. *N Engl J Med*. 1962;266:169–173.

43. Shibolet S, Coll R, Gilat T, Sohar E. Heatstroke: Its clinical picture and mechanism in 36 cases. *Q J Med*. 1967;36:525–548.

44. Mustafa MKY, Khogali M, Gumaa K, Abu Al Nasr NM. Disseminated intravascular coagulation among heat stroke cases. In: Khogali M, Hales JRS, eds. *Heat Stroke and Temperature Regulation*. New York, NY: Academic Press; 1983: Chap 9.

45. Mustafa KY, Omer O, Khogali M, et al. Blood coagulation and fibrinolysis in heat stroke. *Br J Haematol*. 1985;61:517–523.

46. Shibolet S, Lancaster MC, Danon Y. Heat stroke: A review. *Aviat Space Environ Med*. 1976;47:280–301.

47. Barry ME, King BA. Heatstroke. *S Afr Med J*. 1962;36:455–461.

48. Clowes GHA Jr, O'Donnell TF Jr. Heat stroke. *N Engl J Med*. 1974;291:564–567.

49. Burr RE. *Heat Illness: A Handbook for Medical Officers.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1991. TN 91-3.

50. Knochel JP, Reed G. Disorders of heat regulation. In: Narins RG, ed. *Clinical Disorders of Fluid and Electrolyte Metabolism.* New York, NY: McGraw-Hill; 1994: 1549–1590.

51. Gutmann L. Thermal Injuries in sports: Neurologic aspects. In: Jordan BD, Tsairis P, Warren RF, eds. *Sports Neurology*. Rockville, Md: Aspen Publishers; 1989: Chap 16.

52. Slovis CM, Anderson GF, Casolaro A. Survival in a heat stroke victim with a core temperature in excess of 46.5°C. *Ann Emerg Med*. 1982;11:269–271.

53. Malamud N, Haymaker W, Custer RP. Heat stroke: A clinico-pathologic study of 125 fatal cases. *The Military Surgeon*. 1946;99:397–449.

54. Hubbard RW. Effects of exercise in the heat on predisposition to heatstroke. *Med Sci Sports*. 1979;11:66–71.

55. Gardner JW, Kark JA. Fatal rhabdomyolysis presenting as mild heat illness in military training. *Mil Med*. 1994;159:160–163.

56. Kark JA, Gardner JW, Hetzel DP, et al. Exertional heat injury in US Marine recruits. *Am J Epidemiol*. 1991;134:743.

57. Kark JA, Kark RAP, Anderson L, Salazar AM, Estella E. Neurologic diagnosis in exertional heat illness. *Clin Res.* 1991;39:412A.

58. Kark JA, Burr PQ, Wenger CB, Gastaldo E, Gardner JW. Exertional heat illness in Marine Corps recruit training. *Aviat Space Environ Med*. 1996;67:354–360.

59. Knochel JP, Caskey JH. The mechanism of hypophosphatemia in acute heat stroke. *JAMA*. 1977;238:425–426.

60. Knochel JP, Barcenas C, Cotton JR, Fuller TJ, Haller R, Carter NW. Hypophosphatemia and rhabdomyolysis. *J Clin Invest*. 1978;62:1240–1246.

61. Knochel JP, Beisel WR, Herndon EG Jr, Gerard ES, Barry KG. The renal, cardiovascular, hematologic, and serum electrolyte abnormalities of heat stroke. *Am J Med*. 1961;30:299–309.

62. Kew MC, Tucker RBK, Bersohn I, Seftel HC. The heart in heatstroke. *Am Heart J*. 1969;77:324–335.

63. Howorth PJN. The biochemistry of heat illness. *J R Army Med Corps*. 1995;141:40–41.

64. Sawka MN. Body fluid responses and hypohydration during exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 7.

65. Carter BJ, Cammermeyer M. A phenomenology of heat injury: The predominance of confusion. *Mil Med*. 1988;153:118–126.

66. Hagen C. Language-cognitive disorganization following closed head injury: A conceptualization. In: Trexler LE, ed. *Cognitive Rehabilitation: Conceptualization and Intervention*. New York, NY: Plenum Press; 1982: 131–151.

67. Starmark JE, Stalhammar D, Holmgren E. The Reaction Level Scale (RLS85): Manual and guidelines. *Acta Neurochir (Wien)*. 1988;91:12–20.

68. Starmark JE, Stalhammar D, Holmgren E, Rosander B. A comparison of the Glasgow Coma Scale and the Reaction Level Scale (RLS85). *J Neurosurg*. 1988;69:699–706.

69. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: A practical scale. *Lancet*. 1974;2:81–84.

70. Folstein MF, Folstein SE, McHugh PR. Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. *J Psychiat Res*. 1975;12:189–198.

71. US Army Center for Health Promotion and Preventive Medicine. Hyponatremia associated with heat stress and excessive water consumption: Ft Benning, GA, Ft Leonard Wood, MO, Ft Jackson, SC. *Med Surveill Monthly Rep.* 1997;3(6):2–8.

72. Coyne DW. Acute renal failure. In: Carey CF, Lee HH, Woeltje KF, eds. *The Washington Manual of Medical Therapeutics.* 29th ed. Philadelphia, Pa: Lippincott-Raven; 1998: Chap 12.

73. Schrier RW, Henderson HS, Tisher CC, Tannen RL. Nephropathy associated with heat stress and exercise. *Ann Intern Med*. 1967;67:356–376.

74. Vertel RM, Knochel JP. Acute renal failure due to heat injury. An analysis of ten cases associated with a high incidence of myoglobinuria. *Am J Med*. 1967;43:435–451.

75. Kew MC, Abrahams C, Levin NW, Seftel HC, Rubenstein AH, Bersohn I. The effects of heatstroke on the function and structure of the kidney. *Q J Med*. 1967;36:277–300.

76. Kew MC, Abrahams C, Seftel HC. Chronic interstitial nephritis as a consequence of heatstroke. *Q J Med*. 1970;39:189–199.

77. Hamilton RW, Gardner LB, Penn AS, Goldberg M. Acute tubular necrosis caused by exercise-induced myoglobinuria. *Ann Intern Med*. 1972;77:77–82.

78. Grossman RA, Hamilton RW, Morse BM, Penn AS, Goldberg M. Nontraumatic rhabdomyolysis and acute renal failure. *N Engl J Med*. 1974;291:807–811.

79. Knochel JP, Dotin LN, Hamburger RJ. Heat stress, exercise, and muscle injury: Effects on urate metabolism and renal function. *Ann Intern Med*. 1974;81:321–328.

80. Pattison ME, Logan JL, Lee SM, Ogden DA. Exertional heat stroke and acute renal failure in a young woman. *Am J Kidney Dis*. 1988;11:184–187.

81. Tietjen DP, Guzzi LM. Exertional rhabdomyolysis and acute renal failure following the Army Physical Fitness Test. *Mil Med*. 1989;154:23–25.

82. Knochel JP. Dog days and siriasis: How to kill a football player. *JAMA*. 1975;233:513–515.

83. Hubbard RW, Mager M, Kerstein M. Water as a tactical weapon: A doctrine for preventing heat casualties. In: *Army Science Conference Proceedings*. Washington, DC: Department of the Army; 15–18 June 1982: 125–139.

84. Hubbard RW, Armstrong LE, Evans PK, DeLuca PJ. Long-term water and salt deficits—A military perspective. In: *Predicting Decrements in Military Performance Due to Inadequate Nutrition*. Washington, DC: National Academy Press; 1986: 29–53.

85. Turell DJ. Primary myoglobinuria and exercise-induced secondary myoglobinuria: A report of 7 cases seen at an Army Basic Training Center. *South Med J*. 1961;54:442–448.

86. Gabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. *Medicine (Baltimore)*. 1982;61:141–152.

87. Knochel JP. Rhabdomyolysis and myoglobinuria. *Ann Rev Med*. 1982;33:435–443.

88. Greenberg J, Arneson L. Exertional rhabdomyolysis with myoglobinuria in a large group of military trainees. *Neurology*. 1967;17:216–222.

89. Demos MA, Gitin EL, Kagen LJ. Exercise myoglobinemia and acute exertional rhabdomyolysis. *Arch Intern Med*. 1974;134:669–673.

90. Demos MA, Gitin EL. Acute exertional rhabdomyolysis. *Arch Intern Med*. 1974;133:233–239.

91. Hurley JK. Severe rhabdomyolysis in well-conditioned athletes. *Mil Med*. 1989;154:244–245.

92. Howenstine JA. Exertion-induced myoglobinuria and hemoglobinuria. *JAMA*. 1960;173:493–499.

93. Rowland LP, Fahn S, Hirschberg E, Harter DH. Myoglobinuria. *Arch Neurol*. 1964;10:537–562.

94. Evans WJ, Meredith CN, Cannon JG, et al. Metabolic changes following eccentric exercise in trained and untrained men. *J Appl Physiol*. 1986;61:1864–1868.

95. Melamed I, Romem Y, Keren G, Epstein Y, Dolev E. March myoglobinemia: A hazard to renal function. *Arch Intern Med*. 1982;142:1277–1279.

96. Block P, Van Rijmenant M, Badjou R, Van Melsem AY, Vogeleer R. The effects of exhaustive effort on serum enzymes in man. *Biochem Exerc Med Sport.* 1969;3:259–267.

97. Shapiro Y, Magazanik A, Sohar E, Reich CB. Serum enzyme changes in untrained subjects following a prolonged march. *Can J Physiol Pharmacol*. 1973;51:271–276.

98. Wyndham CH, Kew MC, Kok R, Bersohn I, Strydom NB. Serum enzyme changes in unacclimatized and acclimatized men under severe heat stress. *J Appl Physiol*. 1974;37:695–698.

99. Knochel JP. Biochemical, electrolyte, and acid–base disturbances in acute renal failure. In: Brenner BM, Lazarus JM, eds. *Acute Renal Failure*. 2nd ed. New York, NY: Churchill Livingstone; 1988.

100. Knochel JP. Catastrophic medical events with exhaustive exercise: "White collar rhabdomyolysis." *Kidney Int*. 1990;38:709–719.

101. Brown J, Mitchell S. A complicated case of exertional heat stroke in a military setting with persistent elevation of creatine phosphokinase. *Mil Med*. 1992;157:101–103.

102. Knochel JP. Clinical complications of body fluid and electrolyte balance. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport*. Boca Raton, Fla: CRC Press; 1996: 297–317.

103. Saffert CA. Heat hyperpyrexia. *Minnesota Med*. 1937;77:106–108.

104. Jackson RC. Exercise-induced renal failure and muscle damage. *Proc R Soc Med*. 1970;63:566–570.

105. Barcenas C, Hoeffler HP, Lie JT. Obesity, football, dog days and siriasis: A deadly combination. *Am Heart J*. 1976;92:237–244.

106. Warrell R. Metabolic emergencies. In: Da Vita VT, Hellman S, Rosenberg SA, eds. *Cancer: Principles and Practice of Oncology*. 5th ed. Philadelphia, Pa: Lippincott-Raven; 1997: 2492–2496.

107. Gillett RL. Primary myoglobinuria. *N Engl J Med*. 1959;260:1156–1160.

108. Smith RF. Exertional rhabdomyolysis in Naval Officer Candidates. *Arch Intern Med*. 1968;121:313–319.

109. Amundson DE. The spectrum of heat related injury with compartment syndrome. *Mil Med*. 1989;154:450–452.

110. Noakes TD, Norman RJ, Buck RH, Godlonton J, Stevenson K, Pittaway D. The incidence of hyponatremia during prolonged ultraendurance exercise. *Med Sci Sports Exerc*. 1990;22:165–170.

111. Burstein R, Galun E. Exercise-induced hyponatremia—As rare as believed? *Med Sci Sports Exerc*. 1990;22:879. Letter; comment.

112. Irving RA, Noakes TD, Buck R, et al. Evaluation of renal function and fluid homeostasis during recovery from exercise-induced hyponatremia. *J Appl Physiol*. 1991;70:342–348.

113. Armstrong LE, Curtis WC, Hubbard RW, Francesconi RP, Moore RE, Askew EW. Symptomatic hyponatremia during prolonged exercise in heat. *Med Sci Sports Exerc*. 1993;25:543–549.

114. Putterman C, Levy L, Rubinger D. Transient exercise-induced water intoxication and rhabdomyolysis. *Am J Kidney Dis*. 1993;21:206–209.

115. Reynolds NC Jr, Schumaker HD, Feighery S. Complications of fluid overload in heat casualty prevention during field training. *Mil Med*. 1998;163:789–791.

116. Garigan TP, Ristedt DE. Death from hyponatremia as a result of acute water intoxication in an Army basic trainee. *Mil Med*. 1999;164:234–237.

117. Wolfson AB. Acute hyponatremia in ultra-endurance athletes. *Am J Emerg Med*. 1995;13:116–117. Letter; comment.

118. US Army Center for Health Promotion and Preventive Medicine. Hyponatremia associated with heat stress and excessive water consumption: Outbreak investigation and recommendations. *Med Surveill Monthly Rep*. 1997;3(6):9–10.

119. Craig S, Knapik J, Brundage J, et al. Overhydration with Secondary Hyponatremia, Fort Benning, Georgia. Aberdeen Proving Ground, Md: US Army Center for Health Promotion and Preventive Medicine; 1997. Report 29-HE-6781-98.

120. Berl T. Treating hyponatremia: Damned if we do and damned if we don't. *Kidney Int*. 1990;37:1006–1018. Clinical conference.

121. Cluitmans FH, Meinders AE. Management of severe hyponatremia: Rapid or slow correction? *Am J Med*. 1990;88:161–166.

122. Montain SJ, Latzka WA, Sawka MN. Fluid replacement recommendations for training in hot weather. *Mil Med*. 1999;164:502–508.

123. US Army Center for Health Promotion and Preventive Medicine. Hyponatremia associated with heat stress and excessive water consumption: The impact of education and a new Army fluid replacement policy. *Med Surveill Monthly Rep.* 1999;5(2):2–9.

124. Freeman W, Dumoff S. Cerebellar syndrome following heat stroke. *Arch Neurol Psychiatry*. 1944;51:67–72.

125. Mehta AC, Baker RN. Persistent neurological deficits in heat stroke. *Neurology*. 1970;20:336–340.

126. Kew MC. Temperature regulation in heatstroke in man. *Isr J Med Sci*. 1976;12:759–764.

127. Chao TC, Sinniah R, Pakiam JE. Acute heat stroke deaths. *Pathology*. 1981;13:145–156.

128. El-Kassimi FA, Al-Mashhadani S, Abdullah AK, Akhtar J. Adult respiratory distress syndrome and disseminated intravascular coagulation complicating heat stroke. *Chest*. 1986;90:571–574.

129. Forgan-Smith JR. Exertion-induced heatstroke. *Med J Aust*. 1987;146:154–155.

130. Jarmulowicz MR, Buchanan JD. Exertional hyperpyrexia: Case report and review of pathophysiological mechanisms. *J R Nav Med Serv*. 1988;74:33–38.

131. Hales JRS, Hubbard RW, Gaffin SL. Limitation of heat tolerance. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology. Section 4, Environmental Physiology*. New York, NY: Oxford University Press for the American Physiological Society; 1996: 285–355.

132. Herman RH, Sullivan BH Jr. Heatstroke and jaundice. *Am J Med*. 1959;27:154–166.

133. Kew M, Bersohn I, Seftel H, Kent G. Liver damage in heatstroke. *Am J Med*. 1970;49:192–202.

134. Kew MC, Minick OT, Bahu RM, Stein RJ, Kent G. Ultrastructural changes in the liver in heatstroke. *Am J Pathol*. 1978;90:609–618.

135. Sutton JR. Heatstroke from running. *JAMA*. 1980;243:1896.

136. Dickinson JG. Heat-exercise hyperpyrexia. *J R Army Med Corps*. 1989;135:27–29.

137. Hassanein T, Perper JA, Tepperman L, Starzl TE, van Theil DH. Liver failure occurring as a component of exertional heatstroke. *Gastroenterology*. 1991;100:1442–1447.

138. Kark JA, Hetzel DP, Lindgren KM, Larkin TJ, Gardner JW, Jarmulowicz MA. Life-threatening arrhythmias in exertional heat stroke. *Clinical Res.* 1993;41:341A.

139. Kark JA, Ward FT. Exercise and hemoglobin S. *Semin Hematol*. 1994;31:181–225.

140. Kark JA, Larkin TJ, Hetzel DP, Jarmulowicz MA, Lindgren KM, Gardner JW. Exertional heat illness contributing to sudden cardiac death. *Circulation*. 1997;96(Suppl 1):476.

141. Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes: Clinical, demographic, and pathological profiles. *JAMA*. 1996;276:199–204.

142. Virmani R, Burke AP, Farb A, Kark JA. Causes of sudden death in young and middle-aged competitive athletes. *Cardiol Clin*. 1997;15:439–466.

143. Kark JA, Posey DM, Schumacher HR, Ruehle CJ. Sickle-cell trait as a risk factor for sudden death in physical training. *N Engl J Med*. 1987;317:781–787.

144. Smith L, Kark J, Gardner J, Ward F. Unrecognized exertional heat illness as a risk factor for exercise-related sudden cardiac death among young adults. *J Am Coll Cardiol*. 1997;29(Suppl A):447–448.

145. Kark J, Ward F, Gardner J. Prevention of exertional heat illness eliminates unexplained exercise-related death of recruits with sickle cell trait. *Blood*. 1997;90(Suppl 1):447a.

146. Kark J, Gardner J, Ward F. Reducing exercise-related sudden cardiac death rates among recruits by prevention of exertional heat illness. *J Am Coll Cardiol*. 1998;31(2,Suppl A):133A–134A.

147. Tham MK, Cheng J, Fock KM. Heat stroke: A clinical review of 27 cases. *Singapore Med J*. 1989;30:137–140.

148. Olson KR, Benowitz NL. Environmental and drug-induced hyperthermia: Pathophysiology, recognition, and management. *Emerg Med Clin North Am*. 1984;2:459–474.

149. Sutton JR. Heat illness. In: Strauss RH, ed. *Sports Medicine*. 2nd ed. Philadelphia, Pa: WB Saunders; 1991: Chap 23.

150. Simon HB. Hyperthermia. *N Engl J Med*. 1993;329:483–487.

151. Anderson RJ, Reed G, Knochel J. Heatstroke. *Adv Intern Med*. 1983;28:115–140.

152. Austin MG, Berry JW. Observations on one hundred cases of heatstroke. *JAMA*. 1956;161:1525–1529.

153. Jones TS, Liang AP, Kilbourne EM, et al. Morbidity and mortality associated with the July 1980 heat wave in St. Louis and Kansas City, Mo. *JAMA*. 1982;247:3327–3331.

154. Tucker LE, Stanford J, Graves B, Swetnam J, Hamburger S, Anwar A. Classical heatstroke: Clinical and laboratory assessment. *South Med J*. 1985;78:20–25.

155. Centers for Disease Control. Heat-related deaths—Missouri, 1979–1988. *MMWR*. 1989;38:437–439.

156. Centers for Disease Control and Prevention. Heat-related deaths—United States, 1993. *MMWR*. 1993;42:558–560.

157. Ferris EB Jr, Blankenhorn MA, Robinson HW, Cullen GE. Heatstroke: Clinical and chemical observations on 44 cases. *J Clin Invest*. 1938;17:249–261.

158. Mao ZC, Wang YT. Analysis of 411 cases of severe heat stroke in Nanjing. *Chin Med J (Engl).* 1991;104:256–258.

159. de Galan BE, Hoekstra JBL. Extremely elevated body temperature: Case report and review of classical heat stroke. *Neth J Med*. 1995;47:281–287.

160. Gauss H, Meyer KA. Heat stroke: Report of one hundred and fifty-eight cases from Cook County Hospital, Chicago. *Am J Med Sci*. 1917;154:554–564.

161. Hart GR, Anderson RJ, Crumpler CP, Shulkin A, Reed G, Knochel JP. Epidemic classical heat stroke: Clinical characteristics and course of 28 patients. *Medicine (Baltimore)*. 1982;61:189–197.

162. Vicario SJ, Okabajue R, Haltom T. Rapid cooling in classic heatstroke: Effect on mortality rates. *Am J Emerg Med*. 1986;4:394–398.

163. Petersdorf RG. Hypothermia and hyperthermia. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, eds. *Harrison's Principles of Internal Medicine*. 13th ed. New York, NY: McGraw-Hill, Health Professions Division; 1994: 2473–2479.

164. Lim MK. Heat stroke. *Singapore Med J*. 1989;30:127–128.

165. Hubbard RW. An introduction: The role of exercise in the etiology of exertional heatstroke. *Med Sci Sports Exerc*. 1990;22:2–5.

166. Goldfrank L, Osborn H, Weisman RS. Heat stroke. *Hosp Physician*. 1980;7:30–43.

167. Birrer RB. Heat stroke: Don't wait for the classic signs. *Emerg Med*. 1988;6:9–16.

168. Morton TSC. Prickly heat. In: *The British Encyclopaedia of Medical Practice*. Vol 10. London, England: Butte; 1950: 253–255.

169. Armstrong L, Hubbard R, Epstein Y, Weien R. Nonconventional remission of miliaria rubra during heat acclimation: A case report. *Mil Med*. 1988;153:402–404.

170. Francis KT. Exertional heat illness: A review. *J Med Technol*. 1984;1:547–551.

171. Siegel AJ. Medical conditions arising during sports. In: Shangold MM, Mirkin G, eds. *Women and Exercise: Physiology and Sports Medicine*. Philadelphia, Pa: FA Davis Company; 1988: Chap 16.

172. Richards R, Richards D. Exertion-induced heat exhaustion and other medical aspects of the City-to-Surf fun runs, 1978–1984. *Med J Aust*. 1984;141:799–805.

173. Stewart CE, Dwyer BJ. Preventing progression of heat injury. *Emerg Med Reports*. 1987;8:121–128.

174. Roberts WO. Assessing core temperature in collapsed athletes: What's the best method? *The Physician and Sportsmedicine*. 1994;22:49–55.

175. Kew M, Bersohn I, Seftel H. The diagnostic and prognostic significance of the serum enzyme changes in heat stroke. *Trans R Soc Trop Med Hyg*. 1971;65:325–330.

176. Armstrong LE, Hubbard RW, Szlyk PC, Sils IV, Kraemer WJ. Heat intolerance, heat exhaustion monitored: A case report. *Aviat Space Environ Med*. 1988;59:262–266.

177. Sprung CL, Portocarrero CJ, Fernaine AV, Weinberg PF. The metabolic and respiratory alterations of heat stroke. *Arch Intern Med*. 1980;140:665–669.

178. Wyndham CH. Heat stroke and hyperthermia in marathon runners. *Ann N Y Acad Sci*. 1977;301:128–138.

179. Danzl DF. Hyperthermic syndromes. *Am Fam Physician*. 1988;37:157–162.

180. Beller GA, Boyd AE III. Heat stroke: A report of 13 consecutive cases without mortality despite severe hyperpyrexia and neurologic dysfunction. *Mil Med*. 1975;140:464–467.

181. Torre-Cisneros J, Fernandez de la Puebla Giminez RA, Jimenez Pereperez JA, et al. The early prognostic assessment of heat stroke [in Spanish]. *Rev Clin Esp*. 1992;190:439–442.

182. Jones BH, Roberts WO. Medical management of endurance events: Incidence, prevention, and care of casualties. In: Cantu RC, Micheli LJ, eds. *ACSM's Guidelines for the Team Physician*. Philadelphia, Pa: Lea & Febiger; 1991: Chap 28.

183. Surber B, Steele K. Heat illness. *US Navy Med*. 1983:25–27.

184. Costrini A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. *Med Sci Sports Exerc*. 1990;22:15–18.

185. Wheeler M. Heat stroke in the elderly. *Med Clin North Am*. 1976;60:1289–1296.

186. Barner HB, Masar M, Wettach GE, Wright DW. Field evaluation of a new simplified method for cooling of heat casualties in the desert. *Mil Med*. 1984;149:95–97.

187. Hubbard RW, Armstrong LE. Hyperthermia: New thoughts on an old problem. *The Physician and Sportsmedicine*. 1989;17:97–113.

188. Armstrong LE, Crago AE, Adams R, Roberts WO, Maresh CM. Whole-body cooling of hyperthermic runners: Comparison of two field therapies. *Am J Emerg Med*. 1996;14:355–358.

189. Harker J, Gibson P. Heat-stroke: A review of rapid cooling techniques. *Intensive Crit Care Nurs*. 1995;11:198–202.

190. Wyndham CH, Strydom NB, Cooke HM, et al. Method of cooling subjects with hyperpyrexia. *J Appl Physiol*. 1959;14:771–776.

191. American College of Sports Medicine. Heat and cold illnesses during distance running: American College of Sports Medicine Position Stand. *Med Sci Sports Exerc*. 1996;28:i–x.

192. Khogali M. The Makkah body cooling unit. In: Khogali M, Hales JRS, eds. *Heat Stroke and Temperature Regulation*. New York, NY: Academic Press; 1983: Chap 12.

193. Greene MA, Boltax AJ, Lustig GA, Rogow E. Circulatory dynamics during the cold pressor test. *Am J Cardiol*. 1965;16:54–60.

194. Neill WA, Duncan DA, Kloster F, Mahler DJ. Response of coronary circulation to cutaneous cold. *Am J Med*. 1974;56:471–476.

195. Mitchell D, Laburn HP. Pathophysiology of temperature regulation. *Physiologist*. 1985;28:507–517.

196. Singer GG. Fluid and electrolyte management. In: Carey CF, Lee HH, Woeltje KF, eds. *The Washington Manual of Medical Therapeutics*. 29th ed. Philadelphia, Pa: Lippincott-Raven; 1998: Chap 3.

197. Kaehnly WD. The patient with abnormal venous serum bicarbonate or arterial blood pH, pCO_2, and bicarbonate. In: Schrier RW, ed. *Manual of Nephrology: Diagnosis and Therapy*. 3rd ed. Boston, Mass: Little, Brown & Co; 1990: Chap 4.

198. Armstrong LE, DeLuca JP, Hubbard RW. Time course of recovery and heat acclimation ability of prior exertional heatstroke patients. *Med Sci Sports Exerc*. 1990;22:36–48.

199. Royburt M, Epstein Y, Solomon Z, Shemer J. Long-term psychological and physiological effects of heat stroke. *Physiol Behav*. 1993;54:265–267.

200. Bricknell MCM. Heat illness—A review of military experience, I. *J R Army Med Corps*. 1995;141:157–166.

201. Bricknell MCM. Heat illness—A review of military experience, II. *J R Army Med Corps*. 1996;142:34–42.

202. Wakefield EG, Hall WW. Heat injuries. A preparatory study for experimental heat-stroke. *JAMA*. 1927;89:92–95.

203. Jarcho S. A Roman experience with heat stroke in 24 BC. *Bull N Y Acad Med*. 1967;43:767–768.

204. Mitchell TJ, Smith GM. *Medical Services: Casualties and Medical Statistics of the Great War.* In: *History of the Great War Based on Official Documents*. London, England: His Majesty's Stationery Office; 1931.

205. Willcox WH. The nature, prevention, and treatment of heat hyperpyrexia: The clinical aspect. *Br Med J*. 1920;1:392–397.

206. Hill L. The nature, prevention, and treatment of heat hyperpyrexia: The physiological aspect. *Br Med J*. 1920;1:397–399.

207. Morton TC. Heat effects in British service personnel in Iraq. *Trans R Soc Trop Med Hyg*. 1944;37:347–372.

208. Crew FAE, ed. Effects of heat. In: Crew FAE, ed. *Administration*. Vol 2. In: MacNalty AS, ed. *History of the Second World War: The Army Medical Services*. London, England: Her Majesty's Stationery Office; 1955: 179–185.

209. Ladell WSS, Waterlow JC, Hudson MF. Desert climate: Physiological and clinical observations. *Lancet*. 1944;2:491–497.

210. Borden DL, Waddill JF, Grier GS III. Statistical study of 265 cases of heat disease. *JAMA*. 1945;128:1200–1205.

211. Schickele E. Environment and fatal heat stroke: An analysis of 157 cases occurring in the Army in the US during World War II. *The Military Surgeon*. 1947;100:235–256.

212. Whayne TF. *History of Heat Trauma as a War Experience.* Lecture delivered 9 June 1951. In: US Army Medical Service Graduate School. *Notes: Medical Service Company Officer Course 8-0-1 (b)*. Vol 2. Washington, DC: US Army Medical Service Graduate School; 1951. Walter Reed Army Institute of Research, Washington, DC. Library call number RC971/.U5/v.2.

213. Eichna LM. Heat casualty. In: Havens WP Jr, ed. *Infectious Diseases and General Medicine*. Vol 3. In: Heaton LD, Anderson RS, eds. *Internal Medicine in World War II.* Washington, DC: US Department of the Army, Medical Department, Office of The Surgeon General; 1968: Chap 9.

214. Garand GW, Strobridge TR. Western Pacific operations. Vol 4. In: *History of US Marine Corps Operations in WW II*. Washington, DC: US Government Printing Office; 1971: 150.

215. Cook EL. Epidemiological approach to heat trauma. *Mil Med*. 1955;116:317–322.

216. Stallones RA, Gauld RL, Dodge HJ, Lammers TFM. An epidemiological study of heat injury in Army recruits. *AMA Arch Indust Health*. 1957;15:455–465.

217. Yaglou CP, Minard D. Control of heat casualties at military training centers. *AMA Arch Indust Health*. 1957;16:302–316.

218. Minard D. Studies and recent advances in military problems of heat acclimatization. *Mil Med*. 1967;132:306–315.

219. Riddell NJ. Heat illness and the armed forces. *Lancet*. 1989;2:1042.

220. Clarke BG. Prevention and treatment of heat casualties in the Mojave Desert. *US Navy Med News Letter*. 1965;45:4–5.

221. Lovell MA, Sanders CR, Epperly TD. *Heat Injuries: Correlating Presenting Signs and Lab Findings With Discharge Diagnoses.* Fort Benning, Ga: Martin Army Community Hospital; 1990.

222. O'Donnell TF Jr. Acute heat stroke: Epidemiologic, biochemical, renal, and coagulation studies. *JAMA*. 1975;234:824–828.

223. Belding HS, Minard D, Wiebers JE, Ross DM. *Heat Stresses and Strains of Summer Training at the Marine Corps Recruit Depot, Parris Island, South Carolina.* Washington, DC: Office of Naval Research; 1956.

224. Belding HS, Hatch TF. Index for evaluating heat stress in terms of resulting physiological strains. *Heating, Piping, Air Conditioning*. 1955;(Aug):129–136.

225. Minard D, Belding HS, Kingston JR. Prevention of heat casualties. *JAMA*. 1957;165:1813–1818.

226. Hislop LR Jr. The wet bulb-globe temperature index. *US Armed Forces Med J*. 1960;11:709–712.

227. Minard D. Prevention of heat casualties in Marine Corps Recruits. *Mil Med*. 1961;126:261–272.

228. O'Donnell TF Jr. Medical problems of recruit training: A research approach. *US Navy Med*. 1971;58:28–34.

229. Ramanathan NL, Belding HS. Physiologic evaluation of the WBGT index for occupational heat stress. *Am Ind Hyg Assoc J*. 1973;34:375–383.

230. Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc*. 1996;28:939–944.

231. Khogali M, Hales JRS. *Heat Stroke and Temperature Regulation.* New York, NY: Academic Press; 1983.

232. Porter AM. Heat illness and soldiers. *Mil Med*. 1993;158:606–609.

233. Shvartz E, Saar E, Benor D. Physique and heat tolerance in hot-dry and hot-humid environments. *J Appl Physiol*. 1973;34:799–803.

234. Inbar O. Exercise in the Heat. In: Welsh RP, Shephard RJ, eds. *Current Therapy in Sports Medicine 1985–1986*. Toronto, Ont, Canada: C V Mosby Co; 1985: 45–49.

235. Armstrong LE, Pandolf KB. Physical training, cardiorespiratory physical fitness and exercise-heat tolerance. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 5.

236. Nadel ER, Pandolf KB, Roberts MF, Stolwijk JAJ. Mechanisms of thermal acclimation to exercise and heat. *J Appl Physiol*. 1974;37:515–520.

237. Sawka MN, Wenger CB, Pandolf KB. *Human Responses to Exercise-Heat Stress*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1993. TN 94-3.

238. Lind AR, Bass DE. Optimal exposure time for development of acclimatization to heat. *Fed Proc*. 1963;22:704–708.

239. Shieh SD, Lin YF, Lu KC, et al. Role of creatine phosphokinase in predicting acute renal failure in hypocalcemic exertional heat stroke. *Am J Nephrol*. 1992;12:252–258.

240. Shvartz E, Shapiro Y, Magazanik A, et al. Heat acclimation, physical fitness, and responses to exercise in temperate and hot environments. *J Appl Physiol*. 1977;43:678–683.

241. Rose RC III, Hughes RD, Yarbrough DR III, Dewees SP. Heat injuries among recreational runners. *South Med J*. 1980;73:1038–1040.

242. Kenney WL. Physiological correlates of heat intolerance. *Sports Med.* 1985;2:279–286.

243. Centers for Disease Control. Exertional rhabdomyolysis and acute renal impairment—New York City and Massachusetts, 1988. *MMWR*. 1990;39:751–756.

244. Epstein Y, Shapiro Y, Brill S. Role of surface area-to-mass ratio and work efficiency in heat intolerance. *J Appl Physiol*. 1983;54:831–836.

245. Robinson S. The effect of body size upon energy exchange in work. *Am J Physiol*. 1942;136:363–368.

246. Buskirk ER, Lundegren H, Magnusson L. Heat acclimatization patterns in obese and lean individuals. *Ann N Y Acad Sci*. 1965;131:637–653.

247. Wyndham CH. A survey of the causal factors in heat stroke and of their prevention in the gold mining industry. *Journal of the South African Institute of Mining and Metallurgy*. 1965;66:125–155.

248. Departments of the Army, the Navy, and the Air Force. *The Etiology, Prevention, Diagnosis, and Treatment of Adverse Defects of Heat.* Washington, DC: DA, DN, DAF; 1969. TB MED 175, NAVMED P-5052-5, AFP 160-1.

249. Departments of the Army, the Navy, and the Air Force. *The Etiology, Prevention, Diagnosis, and Treatment of Adverse Effects of Heat*. Washington, DC: DA, DN, DAF; 1978. TB MED 175, NAVMED P-5052-5, AFOSH STD 161-12.

250. Hubbard RW. An analysis of current doctrine in use (USA vs IDF) for the prevention and treatment of heat casualties resulting from operations in the heat. Presented at the Commanding Officers Conference 4th MAW / MARTC; 1978; New Orleans, La.

251. Murphy RJ. Heat illness in the athlete. *Am J Sports Med*. 1984;12:258–261.

252. US Army Center for Health Promotion and Preventive Medicine. Heat related injuries, July 1995. *Med Surveill Monthly Rep*. 1995;1(5):2–11.

253. Hubbard RW, Matthew CB, Durkot MJ, Francesconi RP. Novel approaches to the pathophysiology of heat-stroke: The energy depletion model. *Ann Emerg Med*. 1987;16:1066–1075.

254. Jones BH, Cowan DN, Knapik JJ. Exercise, training and injuries. *Sports Med*. 1994;18:202–214.

255. Nunneley A. Design and evaluation of clothing for protection from heat stress: An overview. In: Mekjavic IB, Banister EW, Morrison JB, eds. *Environmental Ergonomics: Sustaining Human Performance in Harsh Environments.* Philadelphia, Pa: Taylor & Francis; 1988: Chap 4.

256. Cooper JK. Preventing heat injury: Military versus civilian perspective. *Mil Med*. 1997;162:55–58.

257. Wallace AW. Heat exhaustion. *The Military Surgeon*. 1943;93:140–146.

258. Buskirk ER, Grasley WC. Heat injury and conduct of athletics. In: Johnson WR, Buskirk ER, eds. *Science and Medicine in Exercise and Sport*. 2nd ed. New York, NY: Harper & Row; 1974: Chap 16.

259. Pandolf KB, Stroschein LA, Drolet LL, Gonzalez RR, Sawka MN. Prediction modeling of physiological responses and human performance in the heat. *Comput Biol Med*. 1986;16:319–329.

260. Gonzalez RR. Biophysics of heat transfer and clothing considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988: Chap 2.

261. Leithead CS. Prevention of the disorders due to heat. *Trans R Soc Trop Med Hyg*. 1967;61:739–745.

262. Krueger GP. Environmental medicine research to sustain health and performance during military deployment: Desert, arctic, high altitude stressors. *J Therm Biol*. 1993;18:687–690.

263. Bartley JD. Heat stroke: Is total prevention possible? *Mil Med*. 1977;142:533–535.

264. Keren G, Epstein Y, Magazanik A. Temporary heat intolerance in a heatstroke patient. *Aviat Space Environ Med*. 1981;52:116–117.

265. Epstein Y. Heat intolerance: Predisposing factor or residual injury? *Med Sci Sports Exerc*. 1990;22:29–35.

266. Hopkins PM, Ellis FR, Halsall PJ. Evidence for related myopathies in exertional heat stroke and malignant hyperthermia. *Lancet*. 1991;338:1491–1492.

267. Savdie E, Prevedoros H, Irish A, et al. Heat stroke following Rugby League football. *Med J Aust*. 1991;155:636–639.

268. Dann EJ, Berkman N. Chronic idiopathic anhydrosis—A rare cause of heat stroke. *Postgrad Med J*. 1992;68:750–752.

269. Koizumi T, Nomura H, Kobayashi T, Hayano T, Kubo K, Sekiguchi M. Fatal rhabdomyolysis during mountaineering. *J Sports Med Phys Fitness*. 1996;36:72–74.

270. Sorensen JB, Ranek L. Exertional heatstroke: Survival in spite of severe hypoglycemia, liver and kidney damage. *J Sports Med Phys Fitness*. 1988;28:108–110.

271. Stephenson LA, Kolka MA. Effect of gender, circadian period and sleep loss on thermal responses during exercise. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1988; Chap 7.

272. Moroff SV, Bass DE. Effects of overhydration on man's physiological responses to work in the heat. *J Appl Physiol*. 1965;20:267–270.

273. Wyndham CH, Strydom NB. The danger of an inadequate water intake during marathon running. *S Afr Med J*. 1969;43:893–896.

274. Glenn J, Burr RE, Hubbard RW, et al. *Sustaining Health and Performance in the Desert: Environmental Medicine Guidance for Operations in Southwest Asia.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1990. TN 91–1.

275. Department of the Army. *Prevention of Heat Injury.* Washington, DC: Headquarters, DA; 1982. Cir 40-82-3.

276. Szylk PC, Hubbard RW, Matthew WT. Mechanisms of voluntary dehydration among troops in the field. *Mil Med*. 1987;152:405–407.

277. Gross TD, Shiffer SW. US Navy recruit hydration status. *Mil Med*. 1988;153:178–180.

278. Adolf EF. *Physiology of Man in the Desert.* New York, NY: Hafner; 1969. Facsimile of the 1947 edition.

279. Barr SI, Costill DL, Fink WJ. Fluid replacement during prolonged exercise: Effects of water, saline, or no fluid. *Med Sci Sports Exerc*. 1991;23:811–817.

280. Noakes TD. Hyponatremia during endurance running: A physiological and clinical interpretation. *Med Sci Sports Exerc*. 1992;24:403–405. Comment.

281. Costill DL, Kammer WF, Fisher A. Fluid ingestion during distance running. *Arch Environ Health*. 1970;21:520–525.

282. n.a. Exertional heat injury. *Med Lett Drugs Ther*. 1985;27:55–56.

283. Carter JE, Gisolfi CV. Fluid replacement during and after exercise in the heat. *Med Sci Sports Exerc*. 1989;21:532–539.

284. American College of Sports Medicine. ACSM position stand on the prevention of thermal injuries during distance running. *Med Sci Sports Exerc*. 1987;19:529–533.

285. Lee L, Chia HP, Tan EH. Prevention of heat disorders in the Singapore Armed Forces 1984–1989. *Ann Acad Med (Singapore)*. 1991;20:347–350.

286. Haymes EM. Physiological Responses of female athletes to heat stress: A review. *The Physician and Sportsmedicine*. 1984;12:45–59.

287. US Army Center for Health Promotion and Preventive Medicine. Heat injuries in active duty soldiers, 1990–1996. *Med Surveill Monthly Rep*. 1997;3(6):16–19.

Chapter 7: ATTACHMENT; Clinical and Surveillance Forms

Exertional Heat Illness FIELD FORM

Patient ID: _____

Date/Time: _____

Training Day _____

Time of onset of illness: _____ AM/PM FIELD WBGT: _____ °F

CIRCUMSTANCES before and at onset of symptoms:

Activity: ☐ Running* ☐ Marching* *distance miles _____

Training Event: _____ ☐ Table PT ☐ Other _____

In your own words, DESCRIBE THE ONSET OF ILLNESS:

CLOTHING: ☐ **PT Gear:** ☐ w/sweatshirt ☐ w/sweatpants **Shoes:** ☐ athletic ☐ boots

Utilities: ☐ w/blouse ☐ wo/blouse **Accessories:** ☐ hat ☐ helmet ☐ vest ☐ rifle

☐ pack _____ lbs ☐ MOPP _____ ☐ Other: _____

SIGNS AND SYMPTOMS:

HYPERVENTILATION: ☐ NO ☐ YES

WOBBLY/STUMBLING: ☐ NO ☐ YES VOMITING: ☐ NO ☐ YES

COLLAPSE (No LOC): ☐ NO ☐ YES SEIZURE: ☐ NO ☐ YES

LOSS OF CONSCIOUSNESS: ☐ NO ☐ YES — DURATION _____ (minutes)

PAIN/CRAMPS: ☐ NO ☐ YES — LOCATION _____

OTHER: _____

1—VS Time: _____ **T:** _____ °F **P:** _____ **R:** _____ **BP:** ___/___ **Orientation:** ☐ person ☐ place ☐ time

CNS Scale: ☐ 8 (Normal) ☐ 7 ☐ 6 ☐ 5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 (Deep Coma)

2—VS Time: _____ **T:** _____ °F **P:** _____ **R:** _____ **BP:** ___/___ **Orientation:** ☐ person ☐ place ☐ time

CNS Scale: ☐ 8 (Normal) ☐ 7 ☐ 6 ☐ 5 ☐ 4 ☐ 3 ☐ 2 ☐ 1 (Deep Coma)

TREATMENT IN FIELD: Time cooling began _____

COOLING METHOD: ☐ Remove clothing ☐ Wet down ☐ Sheets/Ice water

☐ Oral fluids: Amt: _____ (quarts) ☐ Tub ☐ Shower ☐ Ice packs

☐ MEDICATIONS/IV FLUID/OTHER:

TIME OF ARRIVAL AT CLINIC/ER: Field Watch _____ Clinic Clock _____

FIELD RECORDER (print) _____

Exertional Heat Illness CLINIC FORM 1

CONDITION ON ARRIVAL:

Patient ID: _____
Date: _____
Arrival Time: _____

EXAM (every 5–10 minutes) **Time:**						
***Rectal Temperature** (°F)**:**						
Pulse:						
Respiratory Rate:						
Blood Pressure:	/	/	/	/	/	/
Orientation (0–3/3)**:**	/3	/3	/3	/3	/3	/3
CNS Scale						
8 Normal (alert, oriented, cooperative)	☐	☐	☐	☐	☐	☐
7 Drowsy/Lethargic/Dazed (fully oriented and arousable)	☐	☐	☐	☐	☐	☐
6 Confused—appropriate (cooperates, partial or varying orientation)	☐	☐	☐	☐	☐	☐
5 Confused—inappropriate (disoriented, uncooperative, but purposeful)	☐	☐	☐	☐	☐	☐
4 Delirious (agitated, combative, fragmented behavior, out-of-touch)	☐	☐	☐	☐	☐	☐
3 Obtunded—barely responsive (wards off pain, obeys a command, utters a word, or makes eye contact)	☐	☐	☐	☐	☐	☐
2 Light Coma—w/response to pain (reflex response to pain)	☐	☐	☐	☐	☐	☐
1 Deep Coma—no response to pain	☐	☐	☐	☐	☐	☐

***When changing thermometers, record the last reading and time with the old thermometer, then the first reading and time with the new thermometer, and note the change.

NOTES:

Race _____
Height _____
Weight _____
Age _____
Sex _____

CLINIC RECORDER (print) _____

Exertional Heat Illness CLINIC FORM 2

Patient ID: _____

Date/Time: _____

Describe circumstances, onset of illness, and initial symptoms:

SYMPTOMS: (ENTIRE FIRST HOUR) Reevaluate when patient has recovered

GENERAL:
- ☐N ☐Y THIRSTY
- ☐N ☐Y SCARED/NERVOUS
- ☐N ☐Y WEAK
- ☐N ☐Y HEADACHE

EXTREMITY MUSCLE:
- ☐ N ☐ Y MYALGIA
- ☐ N ☐ Y CRAMPS
  loc: _____

ORTHOSTATIC:
- ☐ N ☐ Y FAINT/DIZZY
- ☐ N ☐ Y BLURRED VISION
- ☐ N ☐ Y TUNNEL/FADING VISION
- ☐ N ☐ Y VISUAL LIGHTS/SPOTS
- ☐ N ☐ Y WOBBLY/STUMBLING
- ☐ N ☐ Y COLLAPSE—No LOC
- ☐ N ☐ Y SYNCOPE/Brief LOC
  duration _____ (min)

PULMONARY:
- ☐ N ☐ Y HYPERVENTILATION
- ☐ N ☐ Y SHORT OF BREATH
- ☐ N ☐ Y NUMBNESS/TINGLING
  loc: _____

GASTROINTESTINAL:
- ☐ N ☐ Y NAUSEA
- ☐ N ☐ Y VOMITING
- ☐ N ☐ Y ABDOMINAL CRAMPS
- ☐ N ☐ Y DIARRHEA

SWEATING: ☐ PRESENT ☐ NONE

SLEEP last 24 hours: _____ hours

AMNESIA: ☐ N ☐ Y (List events not remembered before and after onset, and estimate duration of memory loss):

☐ **OTHER:**

WATER: (qt)	**CAFFEINE:**	**LAST MEAL:**	**SMOKING:**
Last 12 h: _____	Last 12 h (#cups/cans)	☐ Light ☐ Mod. ☐ Heavy	☐ None Last cigarette (time):
PUNCH/SODA (qt)	**Coffee** _____	Time _____ AM/PM	☐ < 1 ppd _____ AM/PM
Last 12 h: _____	**Tea** _____	**Alcohol** (# drinks)**:**	☐ 1–2 ppd
	Cola _____	Last 24 h: _____	☐ ≥ 2 ppd

REVIEW OF SYSTEMS FOR PAST 2 WEEKS:

			Date of onset:				Date of onset:
"Cold" (URI)	☐ N	☐ Y	_____	Syncope	☐ N	☐ Y	_____
Sore throat	☐ N	☐ Y	_____	Palpitations	☐ N	☐ Y	_____
Fever/Chills	☐ N	☐ Y	_____	Nausea	☐ N	☐ Y	_____
Sunburn > 20%	☐ N	☐ Y	_____	Vomiting	☐ N	☐ Y	_____
Cellulitis	☐ N	☐ Y	_____	Diarrhea	☐ N	☐ Y	_____
Vaginitis	☐ N	☐ Y	_____	Constipation	☐ N	☐ Y	_____
Dysuria	☐ N	☐ Y	_____	Bleeding	☐ N	☐ Y	_____
Hematuria	☐ N	☐ Y	_____	Fracture	☐ N	☐ Y	_____
☐ OTHER: Describe/date of onset:				Strain/Sprain	☐ N	☐ Y	_____
				(Females) LMP date _____			

Immunizations in past 2 weeks? ☐ N ☐ Y — Date and type: _____

PRIOR HEAT ILLNESS: ☐ N ☐ Y — Date and Dx: _____

TREATMENT IN CLINIC:

COOLING METHOD: ☐ Water and Fan ☐ Ice Sheets ☐ Immersion in Tub ☐ Shower ☐ None ☐ Other _____

MEDICATIONS/OTHER:

Exertional Heat Illness MEDICAL FORM

Patient ID: _____

Date/Time: _____

LAB TESTS ORDERED: ☐ TC ☐ CBC ☐ HEAT PANEL ☐ TESTS FOR ARF ☐ OTHER:

☐ U/A → ☐ before ☐ after hydration ☐ repeat in 12–24 hours U/A, CBC, & Heat Panel

IV FLUIDS				
Time start	Solution	Amt	Time finish	Amt infused

PO INTAKE		
Time	Type	Amt

OUTPUT		
Time	Type	Amt

LAB RESULTS (First set) **TIME DRAWN:** _____ **AM/PM**

Na		Glucose		CK		Hgb		Urine Specific Gravity	1.0_ _
K		Creatinine		AST		Hct		Urine pH	
Cl		Osmolality		ALT		WBC		Dipstick + 's	
HCO_3		Uric Acid		LDH		Plts		Occult Blood	
								Casts*	
BUN						Diff		WBC/hpf	
								RBC/hpf	

*especially pigmented granular casts

DIAGNOSIS:
☐ HEAT EXHAUSTION ☐ HEAT CRAMPS
☐ HEAT INJURY ☐ DEHYDRATION
☐ HEATSTROKE ☐ Parade Syncope/External Collapse
☐ RHABDOMYOLYSIS ☐ Other _____

SEVERITY (In each category, score severity level 1–5, or comment.)

Dehydration: _____

Encephalopathy: _____

Renal Function: _____

Cell Lysis: _____

Other: _____

DISPOSITION: ☐ HOSPITAL ☐ QUARTERS____days ☐ LIGHT DUTY____days ☐ Regular Duty (RTD)

EVALUATION (H/P): LIST all current medications, chronic illness, and any illness in past 2 weeks.

Patient healthy before exercising? ☐ YES ☐ NO

(Continue on additional page if needed)

MEDICAL RECORDER (print) _____