

# Chapter 3

## PHYSICAL EXERCISE IN HOT CLIMATES: PHYSIOLOGY, PERFORMANCE, AND BIOMEDICAL ISSUES

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## INTRODUCTION

US Army fighting doctrine states that “US Army forces must be prepared to fight and win on short notice anywhere in the world, from blistering deserts to frigid wastelands, in rain forests—and all types of terrain”<sup>1(p14-1)</sup> and that soldiers are the most important and most vulnerable part of the war fighting system. Troops participating in almost all military deployments (including arctic regions<sup>2</sup>) are likely to encounter heat stress that must be managed for successful mission accomplishment. Military operations conducted in oppressively hot climates (eg, the Pacific and North African campaigns in World War II, and the Vietnam and Persian Gulf wars) require troops to perform strenuous exercise for long hours and in conditions that will push them to their physiological limits. Humans are tropical animals and (if heat-acclimatized, given adequate shade and water, and able to limit their physical activity) can tolerate extended exposure to any climatic heat stress on earth.<sup>3</sup> However, some military situations, such as work in boiler rooms, firefighting, and wearing protective clothing in hot environments, involve heat stress conditions so severe they cannot be tolerated for extended periods.<sup>4-7</sup> In addition, mission requirements that demand intense physical activity and the degraded physical or nutritional status of the troops often make successful heat stress management during military operations very difficult.

Heat stress results from the interaction of climatic conditions, body heat production, and the wearing of clothing or equipment or both that impedes heat loss. Heat stress generates a need for sweating and circulatory responses to dissipate body heat, especially when the environment is warmer than skin, and may push the body’s homeostatic systems to their limits. Substantial levels of heat stress can occur even in cool climates, especially during intense exercise or while wearing clothing whose thermal properties are inappropriate to the environment and level of metabolic heat production.<sup>8</sup> The problem of heat stress due to thermally inappropriate clothing is especially pronounced in nuclear, biological, chemical (NBC) warfare protective ensembles, because their high thermal insulation and low permeability to water vapor greatly impede heat loss, and thus exacerbate heat stress.<sup>9,10</sup>

### Climatic Heat Stress

Wet bulb globe temperature (WBGT) is commonly used to quantitate environmental heat stress in military, occupational, and sports applications.<sup>11-15</sup> The

outdoor and indoor WBGT, natural wet bulb, dry bulb, and black globe temperatures are related in the following manner<sup>16</sup>:

1. Outdoor WBGT = 0.7 natural wet bulb + 0.2 black globe = 0.1 dry bulb
2. Indoor WBGT = 0.7 natural wet bulb + 0.3 black globe

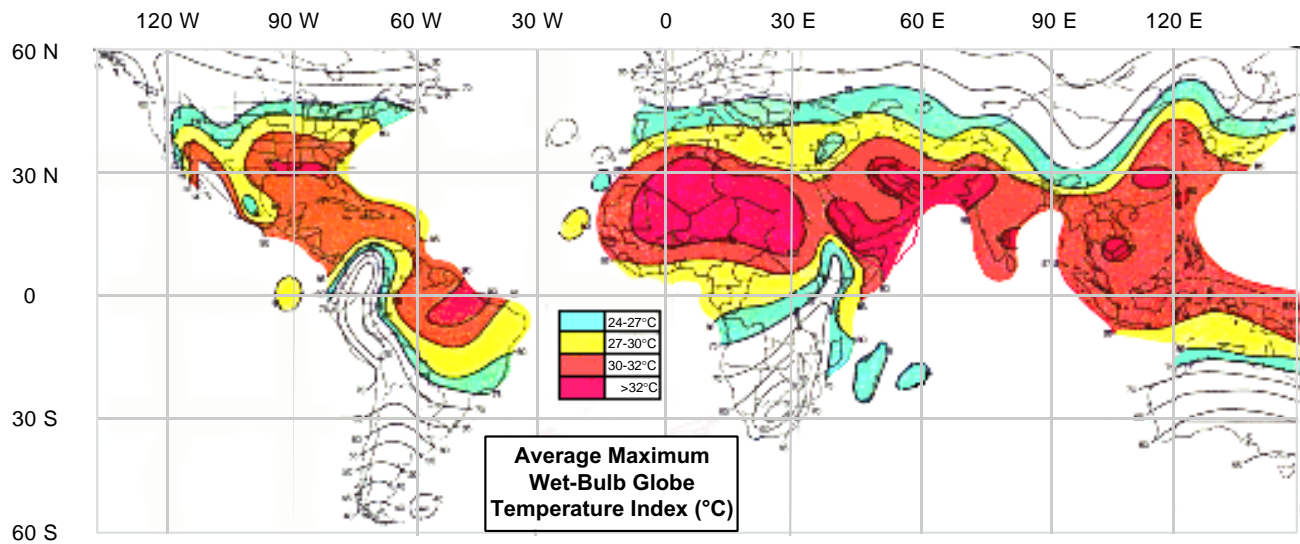
where *natural wet bulb* is defined as the wet bulb temperature under conditions of prevailing air movement; *black globe*, the temperature inside a blackened, hollow, thin copper globe, in which the thermometer is located in the center of the sphere; and *dry bulb* ( $T_{db}$ ), the temperature of the air surrounding the body. WBGT is an empirical index of climatic heat stress, and is used to determine the permitted level of physical activity and strategies to minimize the risk of heat injury. WBGT was originally developed for resting comfort and was later adapted for light-intensity physical exercise.<sup>17</sup> WBGT does not include any considerations for clothing or exercise intensity (metabolic rate), so it cannot predict heat exchange between a person and the environment.<sup>11</sup> The risk of heat injury at any given WBGT is greater in humid conditions; therefore, different guidance tables must be used in climates with low, moderate, and high humidity.<sup>11</sup>

Figure 3-1 is a global map of WBGT during July, the hottest month in the northern hemisphere.<sup>18</sup> During July, much of North America, South America, Europe, and Asia have WBGT values above 29°C (85°F). Depending on the climate, such high WBGT values can be achieved either through high humidity,<sup>19</sup> as reflected in high wet bulb temperature, or through high air (dry bulb) temperature and solar load,<sup>3</sup> as reflected in black globe temperature.

### Temperature Regulation

Body temperature is regulated through two parallel processes: behavioral temperature regulation and physiological temperature regulation (this topic is also discussed in Chapter 2, Human Adaptation to Hot Environments). Examples of behavioral temperature regulation include seeking shade, discontinuing exercise, or removing clothing or equipment, or both. Among military personnel, behavioral thermoregulatory drives are often overridden by motivation to successfully complete the mission.

Physiological temperature regulation operates through graded heat loss responses (chiefly sweating



**Fig. 3-1.** The average wet bulb globe temperature (WBGT) index in the northern hemisphere during July. Adapted from John B. Stennis Space Center. *Global Climatology for the Wet Bulb Globe Temperature (WBGT) Heat Stress Index*. Bay St. Louis, Miss: Gulf Weather; 1989: 37, 54, 71.

and skin blood flow), which are proportional to the disturbance in core temperature and modified according to information from temperature receptors in the skin. If some environmental or metabolic heat stress, such as a higher ambient temperature or increased heat production due to exercise, upsets the body's thermal balance, then the body will store heat and the temperature of the core or skin or both, will increase. In response to these temperature increases, the body will increase its heat loss responses. Unless the heat stress exceeds the capacity of the thermoregulatory system, the heat loss responses will increase until they are sufficient to restore heat bal-

ance, so that core temperature stops increasing and reaches a new steady state level, which persists as long as the conditions of exercise and environmental heat stress continue. The body temperature increase during exercise–heat stress is an example of a load “error,” which, in this case, is the core temperature increase above set point. In contrast, if environmental conditions, clothing, or equipment impose a biophysical limit for heat loss that is less than the rate of body heat production, then increases in *effector responses* (ie, increased sweating and skin blood flow) will not restore heat balance but will only result in increased physiological strain.

## BODY TEMPERATURES

Body temperatures can arbitrarily be divided into deep body (ie, core) and surface (ie, skin) measurements. Deep body and surface temperatures will vary depending on their location and the perturbation of the environment and physical work task required of the soldier.

### Core and Skin Temperatures

There is no one “true” core temperature because of temperature differences among different core sites. However, temperatures at all core sites are close to (within about one Centigrade degree) central blood temperature at thermal steady state. Core temperature is measured clinically in the rectum, mouth, tympanum, and auditory meatus. For research

and for specialized purposes, core temperature is also measured in the esophagus and gastrointestinal tract. These sites differ in the speed with which their temperatures respond to changes in central blood temperature, and their susceptibility to being biased by extraneous influences, such as environmental temperature or breathing through the mouth. Table 3-1 summarizes the advantages and disadvantages of different core temperature measurement sites.<sup>20</sup>

Esophageal temperature is measured with a sensor at the level of the left atrium, where the heart and esophagus are in contact and virtually isothermal for several centimeters.<sup>21</sup> Esophageal temperature responds rapidly (time constant  $\approx 1$  min) and quantitatively to changes in central blood tempera-

**TABLE 3-1**  
**ADVANTAGES AND DISADVANTAGES OF CORE TEMPERATURE MEASUREMENT SITES**

Anatomical Site	Advantage	Disadvantage
Esophagus	Accurate; rapid response	Affected by swallowing; can be uncomfortable
Mouth	Easy to use	Affected by drinking and mouth-breathing
Gastrointestinal Tract	Easy to use (pill form)	Affected by location of pill in GI tract (location is variable and uncertain) Affected by drinking while in stomach
Rectum	Accurate	Slow response; can be uncomfortable
Tympanum, Auditory Meatus	Easy to use; rapid response	Inaccurate; affected by ambient and skin temperatures; can be uncomfortable

Adapted from Levine L, Sawka MN, Gonzalez RR. *General Procedures for Clothing Evaluations Relative to Heat Stress*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1995. Technical Note 95-5.

ture,<sup>22-24</sup> and most thermal physiologists consider esophageal temperature to be the best noninvasive index of core temperature in humans.<sup>25</sup> Gastrointestinal tract temperature is obtained from a swallowed pill-like sensor, which telemeters a temperature signal to a receiver outside the body.<sup>26-28</sup> As the pill moves through the gastrointestinal tract its temperature will change with its location. Therefore, temperature measurements with the pill are, in general, somewhat more variable than are other core temperature measurements. However, this method is well suited to measuring core temperature in free-moving subjects because it requires no leads passing through body orifices, and the signal can be relayed to a monitoring station some distance away. A swallowed sensor therefore provides a useful means for monitoring workers in heat stress situations in which prediction of tolerance is difficult or unreliable.

Skin temperature is measured (a) to calculate the mean body temperature for heat storage determinations, (b) to calculate dry (radiative [R] plus convective [C]) heat exchange and skin conductance, and (c) to provide an index of the skin temperature input to the thermoregulatory controller. Because the skin represents the boundary between two media—tissue and the ambient air—changes in skin temperature may result from physiological adjustments (eg, cutaneous blood flow, sweat evaporation) or alterations in the environment (eg, air motion, temperature, and radiation). Skin temperature varies with ambient temperature and also according to the site where it is measured (Figure 3-2). Although a single skin temperature measurement can be useful for biophysical calculations, scientists are more often interested in the average, or mean skin tempera-

ture.<sup>25,29</sup> Mean skin temperature is calculated from individual local temperature measurements, usually weighted based on the percentage of body surface area that is represented by each body region at which temperature is measured. Early investigators<sup>30,31</sup> used 12 to 15 skin regions to calculate mean skin temperature, but, more recently, some investigators<sup>32-34</sup> have attempted to minimize the number of sites necessary for a valid estimate of mean skin temperature.

### Exercise in Heat

When performing physical work in temperate or hot climates, a soldier's core temperature will increase. The magnitude of core temperature rise will depend on the environmental conditions, exercise task, and the soldier's biomedical state.

### Influence of Metabolic Rate and Climate

Figure 3-3 illustrates that during physical exercise, core (esophageal and rectal) temperature initially increases rapidly and subsequently increases at a reduced rate until essentially steady state levels are achieved.<sup>29</sup> The core temperature increase represents the storage of metabolic heat, which is a by-product of skeletal muscle contraction. At the initiation of exercise, the metabolic rate increases immediately; however, the thermoregulatory effector responses for heat dissipation respond more slowly. Once these heat loss mechanisms increase sufficiently to balance metabolic heat production, a new steady state level of core temperature is achieved.

During muscular exercise, the magnitude of the

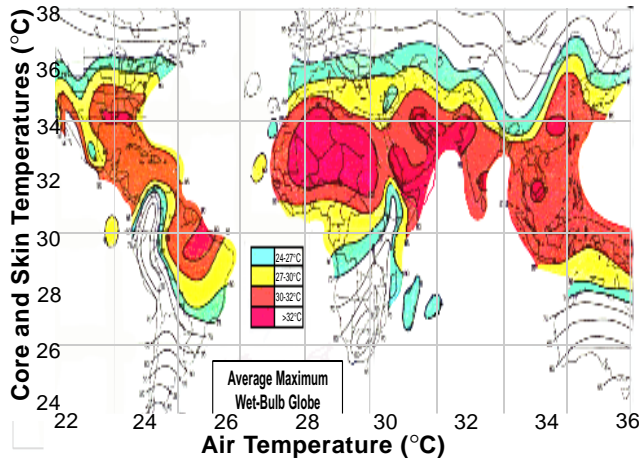


Fig. 3-2. Relationship between selected regional skin temperatures and core (rectal) temperature at rest over a range of temperate and hot climatic conditions. Adapted with permission from Hardy JD, DuBois. Basal metabolism and heat loss of young women at temperatures from 22°C to 38°C. *J Nutr.* 1941;21:383-404.

core temperature increase at steady state is independent of climatic conditions over a fairly wide range, and is proportional to the metabolic rate.<sup>35-37</sup> Figure 3-4 presents heat exchange data during physical exercise (cycle ergometer at ~ 147 W mechanical power output; metabolic rate ~ 650 W) in a broad range of environmental conditions (5°C–36°C dry bulb temperatures with low humidity).<sup>38</sup>

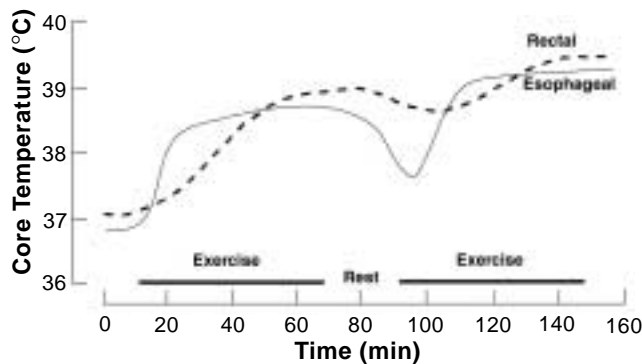


Fig. 3-3. Core (esophageal and rectal) temperature responses during exercise and recovery. Reprinted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 110.

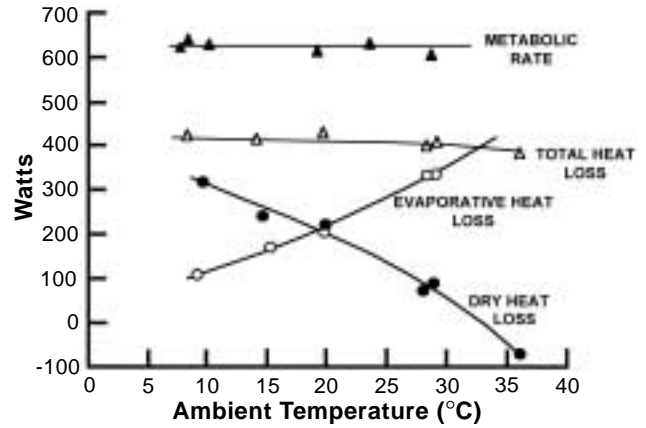
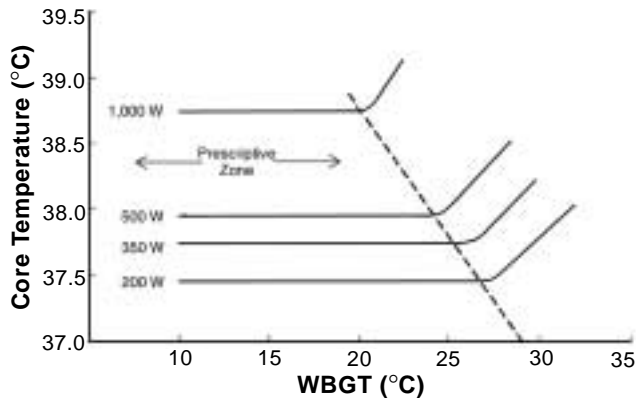


Fig. 3-4. Heat exchange during muscular exercise in a broad range of ambient temperatures. The figure shows the metabolic rate as constant at approximately 630 W. The figure also shows a fairly constant heat loss over the total temperature range. What changes is the partition between evaporative and dry heat loss. Reprinted from Nielsen M. Die Regulation der Körpertemperatur bei Muskelarbeit. *Scand Arch Physiol.* 1938;9:216.

The difference between metabolic rate and total heat loss represents the sum of energy used for mechanical work and stored as heat. Note that the total heat loss and, therefore, the heat storage and core temperature increase, are constant for each climate. The relative contributions of dry and evaporative heat exchange to the total heat loss, however, vary with climatic conditions. At 10°C, the large skin-to-ambient temperature gradient facilitates dry heat exchange, which is approximately equal to 70% of the total heat loss. As the ambient temperature increases, this gradient for dry heat exchange diminishes and there is a greater reliance on evaporative heat exchange. When the ambient temperature is equal to skin temperature, evaporative heat exchange will account for essentially all of the heat loss; and when the ambient temperature exceeds the skin temperature, there is dry heat gain to the body.

The concept that the core temperature increase during exercise is independent of the environment seems contrary to experience. Part of the reason for the discrepancy is that the feeling of being hot depends on more factors than core temperature (eg, skin temperature and cardiovascular strain). In addition, there are biophysical limits to heat exchange between the body and the environment<sup>39,40</sup> so that the core temperature increase during exercise is independent of the environment only within a range of conditions called the *prescriptive zone*.<sup>35</sup> Figure 3-5 illustrates this concept by presenting steady state



**Fig. 3-5.** Steady state core temperature responses of seminude subjects during exercise, as related to metabolic rate (expressed in watts [W]) and environmental heat stress (expressed as wet bulb globe temperature [WBGT] in Centigrade degrees). At all metabolic rates, the prescriptive zone extends from 10°C to the dotted line at right. Data source: Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. *J Appl Physiol.* 1963;18:53.

core temperature responses during muscular exercise performed at four metabolic intensities in a broad range of environmental conditions (with low humidity).<sup>35</sup> For military and other occupational tasks, metabolic rates of 250, 425, and 600 W represent light-, moderate-, and heavy-intensity exercise, respectively<sup>41</sup> (Table 3-2). (For comparison, highly trained athletes may be able to sustain exercise at metabolic rates as high as 1,000 W.) Note that as metabolic rate increases, the width of the prescriptive zone narrows. This occurs because as environmental temperature increases, skin temperature also increases (see Figure 3-2). As a result, the temperature gradient between core and skin is narrowed, and a greater skin blood flow is required to transfer metabolic heat to the skin. The upper end of the prescriptive zone is reached at the point at which further increases in skin temperature alone do not elicit a large enough increase in skin blood flow to maintain heat transfer, and core temperature must rise to allow sufficient heat transfer to achieve heat balance. At higher exercise intensities the skin blood flow required is greater, and the upper end of the prescriptive zone is reached at lower skin and ambient temperatures. Clothing and equipment that impede heat loss will further narrow the prescriptive zone because the insulation and moisture-transfer barriers provide additional biophysical limits to heat exchange.<sup>39</sup>

For any one individual, the core temperature in-

crease during exercise within the prescriptive zone is proportional to the metabolic rate.<sup>35-37,42</sup> For comparisons among individuals, the association between metabolic rate and core temperature is much more variable. However, expressing exercise intensity as relative intensity (ie, as a percentage of maximal oxygen uptake [ $\dot{V}O_2\text{max}$ ]) rather than absolute intensity, removes much of the intersubject variability<sup>43</sup> (Figure 3-6). ( $\dot{V}O_2\text{max}$  is widely used as a measure of physical fitness level.)

Studies undertaken to clarify the relationship between relative intensity and steady state core temperature response to exercise<sup>44,45</sup> show a curvilinear relationship in Figure 3-7, and steady state core temperature values during exercise, at 65% and 85%  $\dot{V}O_2\text{max}$ , in relation to the ambient dry bulb (with < 50% relative humidity [rh]) temperature.<sup>44</sup> Note that at 65%  $\dot{V}O_2\text{max}$ , core temperature is independent of dry bulb temperature from 5°C to 20°C; at 85%  $\dot{V}O_2\text{max}$ , however, core temperature is strongly influenced by dry bulb temperature, illustrating again (see Figure 3-5) that the prescriptive zone is smaller at higher metabolic rates.

Because core temperature changes are related to the relative exercise intensity, it seems logical to expect that any condition that lowers maximal oxygen uptake (and thus increases relative intensity) would also elicit a greater core temperature response at a given absolute (thus higher relative) exercise intensity. Evidence from several studies in which subjects'  $\dot{V}O_2\text{max}$  was reduced by hypobaric hypoxia<sup>46,47</sup> or carboxyhemoglobinemia<sup>48</sup> suggests that this is, in fact the case; but contrary results have also been reported.<sup>49</sup> Interpretation of such data is complicated by the fact that some experimental conditions decreased core temperature at rest, so that exercise would need to cause a greater net increase in core temperature to produce the same steady state core temperature level. Conversely, data from studies in which subjects'  $\dot{V}O_2\text{max}$  was increased by autologous erythrocyte infusion suggest that interventions that increase  $\dot{V}O_2\text{max}$  (thus lowering relative intensity) may reduce the core temperature increase elicited by exercise.<sup>50-52</sup>

### **Influence of Hydration**

Troops exercising in the heat often incur body water deficits, which typically range from 2% to 8% of their body weight.<sup>53,54</sup> Such deficits develop either because (a) adequate amounts of potable fluid are not available or (b) thirst is insufficient to make them replace all the fluid lost as sweat.<sup>55</sup> The resulting dehydration increases physiological strain,

**TABLE 3-2**  
**TYPICAL METABOLIC RATES ASSOCIATED WITH PERFORMANCE OF SELECTED MILITARY TASKS**

Work Intensity Category	Military Activity	Metabolic Rate (W)
Very Light (< 175 W)	Lying on ground	105
	Standing in foxhole	116
	Sitting in truck	116
	Guard duty	137
	Driving truck	163
Light (175–325 W)	Cleaning rifle	198
	Walking on hard surface, 1 m/s, no load	210
	Walking on hard surface, 1 m/s, 20-kg load	255
	Manual of arms	280
	Lift and carry 45-kg artillery shell, 2/min	284
	Walking on hard surface, 1 m/s, 30-kg load	292
Moderate (325–500 W)	Walking on loose sand, 1 m/s, no load	326
	Rifle fire, prone	338
	Walking on hard surface, 1.56 m/s, no load	361
	Lift and carry 45-kg artillery shell, 3/min	370
	Calisthenics	378
	Lift and carry 45-kg artillery shell, 4/min	446
	Walking on hard surface, 1.56 m/s, 20-kg load	448
	Scouting patrol	454
	Crawling, full pack	465
	Foxhole digging	465
	Field assaults	477
Heavy (> 500 W)	Walking on hard surface, 1 m/s, 30-kg load	507
	Walking on hard surface, 2 m/s, no load	525
	Emplacement digging	540
	Bayonet drill	616
	Walking on hard surface, 2 m/s, no load	637
	Walking on loose sand, 1.56 m/s, no load	642

Sources: (1) Sawka MN, Modrow HE, Kolka MA et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. Technical Note 95-1. (2) Sawka MN, Roach JM, Young AJ, et al. *Sustaining Soldier Health and Performance During Operation Support Hope: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research, Development, Acquisition and Logistic Command; 1994. Technical Note 94-3.

decreases exercise performance, and can have devastating medical consequences.<sup>56–58</sup> Dehydration magnifies the core temperature responses to exercise in both temperate<sup>59,60</sup> and hot<sup>61</sup> environments, and this effect is observed with a fluid deficit as small as 1% of body weight.<sup>62</sup> As the water deficit increases, there is a concomitant graded elevation of core temperature during exercise stress.<sup>61,63</sup> Figure 3-8 illustrates relationships between body water loss and core temperature elevations from studies that examined several water deficit levels.<sup>64</sup> The magnitude of additional core temperature elevation ranges from 0.1°C to 0.23°C for every percentage of body weight lost.<sup>53,61,63,65</sup>

Dehydration not only elevates core temperature

responses but also negates the core temperature advantages conferred by high levels of aerobic fitness and heat acclimation.<sup>59,66,67</sup> Figure 3-9 illustrates the effect of dehydration (5% body weight loss) on core temperature responses in the same persons when unacclimated and when acclimated to heat.<sup>67</sup> Heat acclimation lowered core temperature responses of euhydrated subjects; however, similar core temperature responses were observed in hypohydrated subjects regardless of acclimation state. Therefore, the core temperature penalty induced by dehydration was greater in heat-acclimated than in unacclimated persons.

Hyperhydration (greater than normal body water) has been suggested as a means to improve ther-

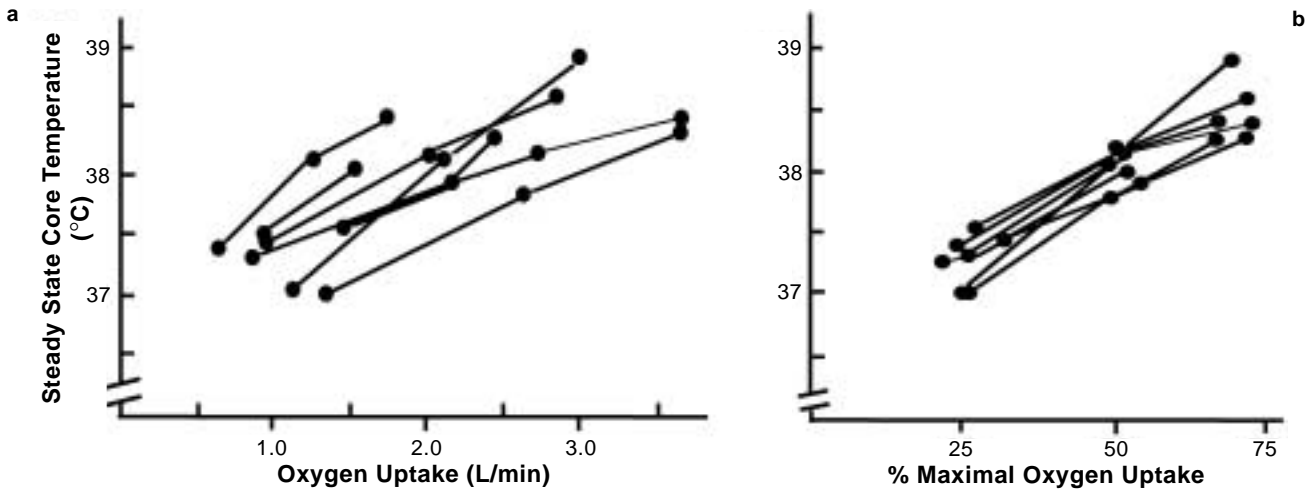


Fig. 3-6. Steady state core temperature responses during exercise as related to (a) absolute and (b) relative exercise intensities. For both categories, the dry bulb temperature ( $T_{db}$ ) was approximately 21°C and the relative humidity (rh) approximately 55%. Adapted with permission from Saltin B, Hermansen L. Esophageal, rectal, and muscle temperature during exercise. *J Appl Physiol.* 1966;21:1759.

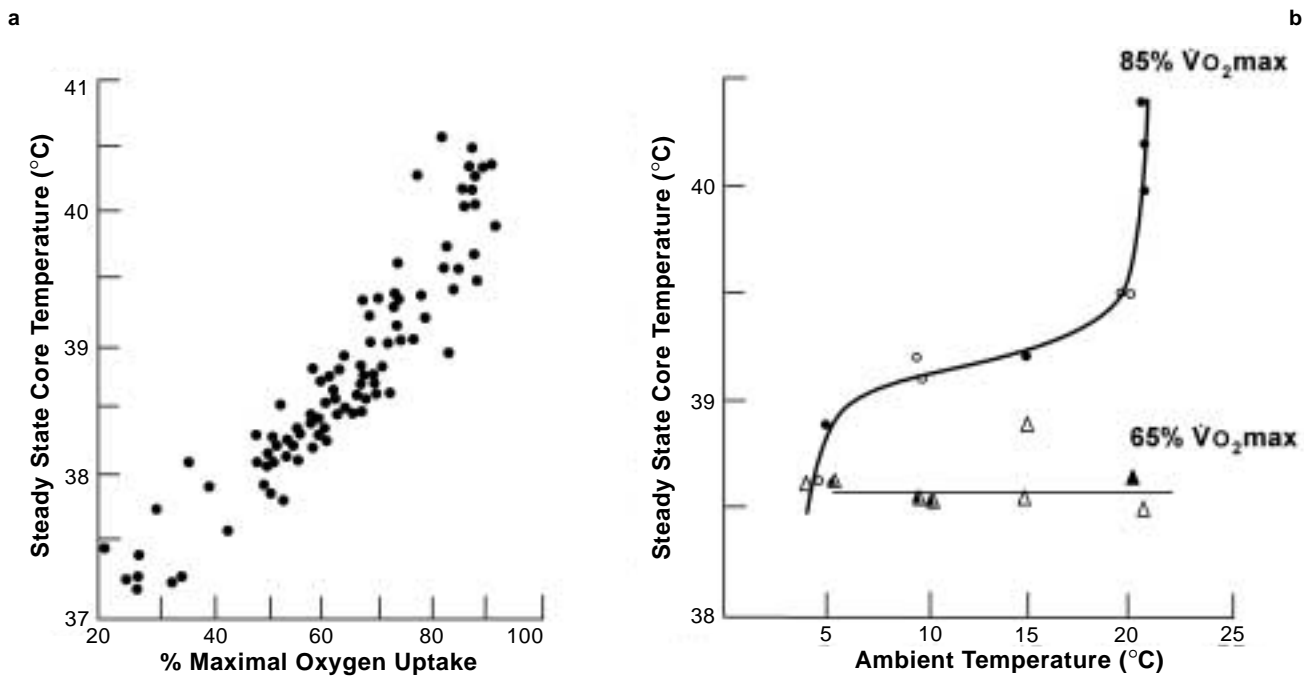
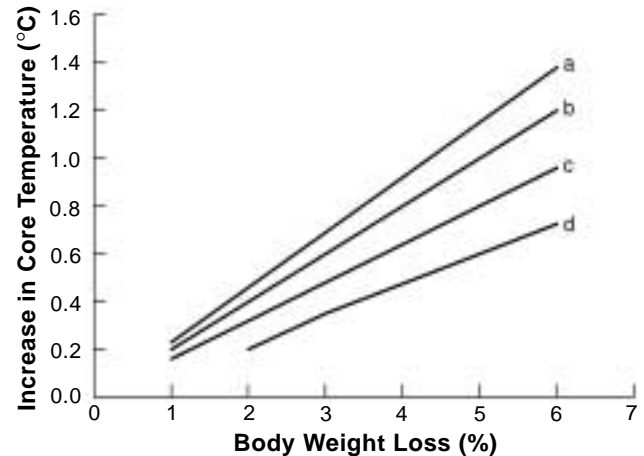


Fig. 3-7. Steady state core temperature response for given (a) relative exercise intensity (symbols represent data from four different datasets) and (b) ambient temperature (open and closed symbols in each curve represent values for two experimental subjects).  $\dot{V}O_{2max}$ : maximal oxygen uptake. (a) Adapted with permission from Davies CTM, Brotherhood JR, Zeidifard E. Temperature regulation during severe exercise with some observations on effects of skin wetting. *J Appl Physiol.* 1976;41:774. (b) Adapted with permission from Davies CTM. Influence of skin temperature on sweating and aerobic performance during severe work. *J Appl Physiol.* 1979;47:772.



**Fig. 3-8.** Relationship between body water loss and steady state core temperature elevations during exercise. Data sources for curves: (a) Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol.* 1992;73:1340–1350. (b) Adolph EF, Associates. *Physiology of Man in the Desert.* New York, NY: Intersciences, Inc; 1947. (c) Sawka MN, Young AJ, Francesconi RP, Muza SR, Pandolf KB. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol.* 1985;59:1394–1401. (d) Strydom NB, Holdsworth DL. The effects of different levels of water deficit on physiological responses during heat stress. *Int Z Angew Physiol.* 1968;26:95–102. db: dry bulb temperature,  $\dot{V}_{O_2\max}$ : maximal  $O_2$  uptake. Figure adapted with permission from Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise–heat exposure. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport.* Boca Raton, Fla: CRC Press; 1996: 148.

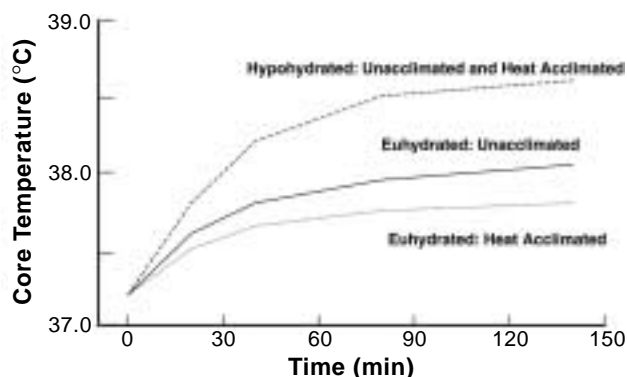


whether hyperhydration is induced with water alone or with water plus glycerol.<sup>77,78</sup>

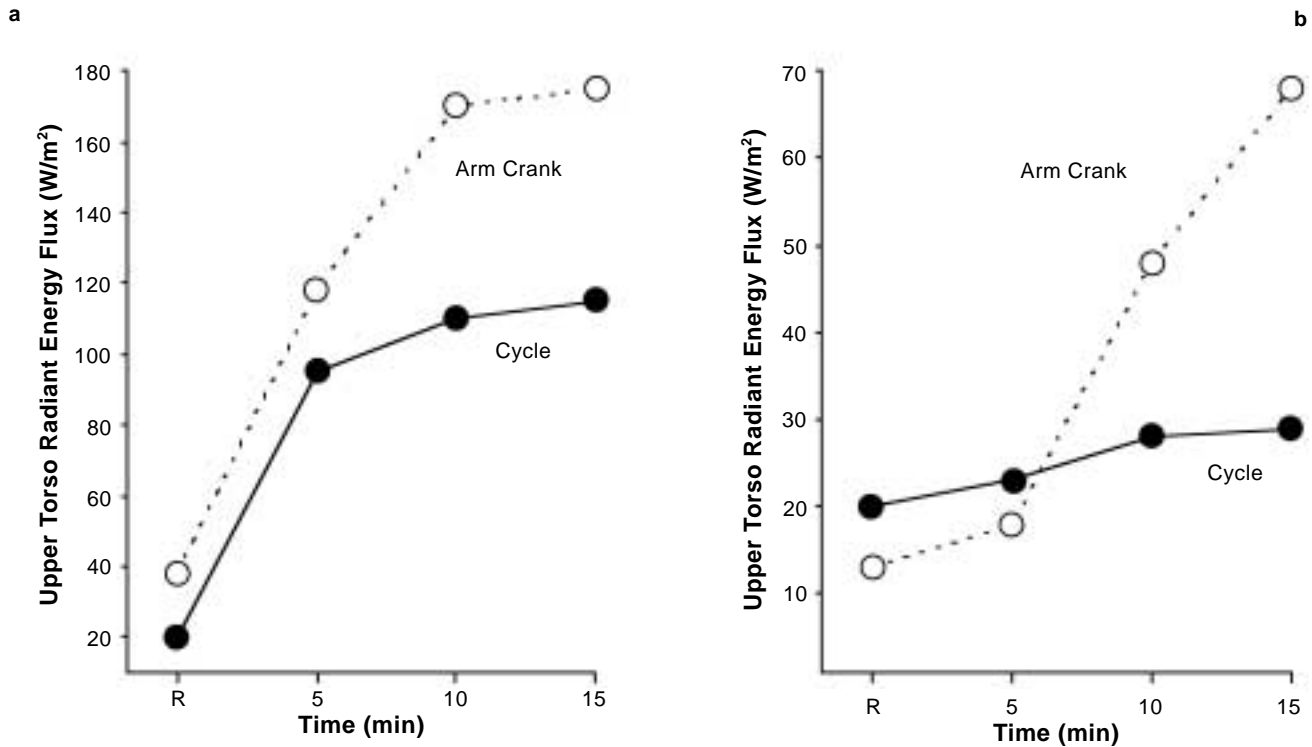
moregulation and exercise performance in the heat, compared with those that occur during euhydration. However, most studies that examined such effects of hyperhydration suffered from serious design problems (eg, control conditions that represented not euhydration but dehydration).<sup>64</sup> Some investigators<sup>68–72</sup> reported lower core temperatures during exercise after hyperhydration, while others<sup>73–75</sup> did not. One study<sup>76</sup> reported that glycerol-facilitated hyperhydration substantially lowered core temperature (0.7°C) during exercise, compared with control conditions and hyperhydration with water alone. Recent research, however, has failed to confirm that hyperhydration confers any thermoregulatory advantages during exercise–heat stress,

### Influence of Exercise Type

Military personnel perform exercise with their legs (eg, marching, climbing), arms (eg, lifting, digging), or both. All studies discussed so far have employed leg (lower body) exercise on treadmills or cycle ergometers. Maximal-effort upper body (arm-crank) exercise elicits an oxygen uptake that is typically 70% of that obtained during maximal-effort lower body exercise.<sup>79</sup> If responses during upper body exercise are compared with those of the same individual during lower body exercise, it is uncertain whether the core temperature response during exercise would be determined by the relative intensity of the exercise (ie, as a fraction of the maximum intensity for the exercising muscle group) or by the absolute metabolic rate, regardless of the exercising muscle mass.<sup>80</sup> If the core temperature responses are determined by the relative intensity (with respect to employed musculature), upper body exercise should be expected to elicit a higher core temperature for a given metabolic rate than should lower body exercise. There has been debate over this question.<sup>80</sup> In fact, several early studies<sup>81,82</sup> reported that upper body exercise elicited lower core temperature responses than lower body exercise at the same absolute metabolic rate, but experimental design and technical problems make their conclusions tenuous.<sup>80</sup> Subsequent studies<sup>83–87</sup> have reported that upper and lower body exercises performed at the same absolute metabolic rate elicit the same core temperature responses. In addition, when upper and lower body exercises are performed at the same relative intensity, upper body exercise elicits lower core temperature responses



**Fig. 3-9.** Effect of dehydration on core temperature responses during exercise–heat stress in unacclimated and acclimated persons. Data source: Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: Effects of heat acclimation, gender, and environment. *J Appl Physiol.* 1983;55:1147–1153.



**Fig. 3-10.** Torso radiative energy flux during upper and lower body exercise at (a) 18°C and 78% relative humidity (rh); and (b) 35°C and 28% rh. Reprinted with permission from Sawka MN, Gonzalez RR, Drolet LL, Pandolf KB. Heat exchange during upper- and lower-body exercise. *J Appl Physiol.* 1984;57:1051.

(by virtue of being at a lower absolute intensity) than leg exercise.<sup>84</sup> These data indicate that core temperature responses are independent of the skeletal muscle mass employed and dependent on the absolute metabolic intensity during exercise in temperate and hot climates.

The heat exchange mechanisms by which heat balance during exercise is achieved, however, do differ between the muscle groups.<sup>85</sup> Figure 3-10 illustrates the torso net radiative energy flux values (dry heat exchange, R + C) during upper body (arm-crank) and lower body (cycle) exercise in two environments<sup>85</sup>: 18°C ambient temperature with 14°C dew point,

which facilitated R + C; and 35°C ambient temperature with 14°C dew point, which facilitated evaporative heat loss ( $E_{sk}$ ). Torso R + C was greater during upper body exercise regardless of the environment. Torso  $E_{sk}$ , arm R + C, and arm  $E_{sk}$  were not different between exercise types in each environment. Leg R + C was greater during lower body than upper body exercise in the 18°C environment, whereas leg  $E_{sk}$  was greater during lower body than upper body exercise in the 35°C environment. These data indicate that to replace the greater R + C heat loss from the torso during upper body exercise, lower body exercise elicits additional R + C or  $E_{sk}$  from the legs.

### PHYSIOLOGICAL RESPONSES AND LIMITATIONS

Physiological factors that are critical to work performance and tolerance to heat stress include those related to heat production, heat loss, and the maintenance of muscular contraction. If heat production is not matched by cutaneous heat loss, then body temperatures will continue to rise until heat exhaustion occurs. In addition, the cardiovascular system is challenged to support heat loss and maintain cellular homeostasis via substrate delivery and re-

moval of metabolites.

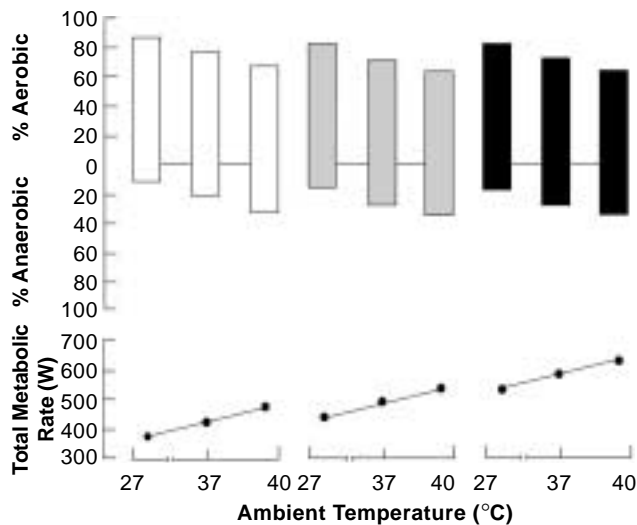
#### Metabolism

Knowledge of how heat strain (ie, the physiological change produced by a disturbance) influences metabolic rate and substrate utilization has important implications for calculating nutritional needs as well as heat balance. Acute heat stress increases

resting metabolic rate, but the effect of heat stress on the metabolic rate needed to perform a given submaximal exercise task is not so clear.<sup>88</sup>

### Metabolic Rate

Many investigators<sup>89–92</sup> report that a given submaximal exercise task elicits a greater metabolic rate in a hot than in a temperate environment. Some investigators,<sup>93–96</sup> however, report lower metabolic rates in the heat. The subjects' state of heat acclimation does not account for whether a study reported an increased or decreased metabolic rate during exercise in the heat.<sup>88</sup> Most investigators, however, calculated only the aerobic metabolic rate during submaximal exercise, ignoring the contribution of anaerobic metabolism to total metabolic rate. Figure 3-11 shows the total metabolic rate and the percentage of this metabolic rate that was contributed by aerobic and anaerobic metabolic pathways during exercise at three intensities at three ambient temperatures.<sup>91</sup> The anaerobic metabolism was calculated from the postexercise oxygen uptake in ex-



**Fig. 3-11.** Total metabolic rate and percentage of metabolic rate contributed by aerobic and anaerobic metabolism during exercise in temperate and hot climates. Exercise is performed at three different intensities (light: open; medium: gray; and heavy: solid), which is repeated at the same three temperatures (27°C, 37°C, and 40°C). The partition between anaerobic and aerobic exercise changes as functions of both the intensity of the work and the ambient temperature. Data source: Dimri GP, Malhotra MS, Gupta JS, Kumar TS, Aora BS. Alterations in aerobic-anaerobic proportions of metabolism during work in heat. *Eur J Appl Physiol.* 1980;45:43–50.

cess of resting baseline levels. The aerobic metabolic rate at a given power output decreased with increasing ambient temperature. However, the calculated anaerobic metabolic rate increased more than aerobic metabolic rate decreased, so that the total metabolic rate required to perform exercise at a given power output increases with ambient temperature. Although these calculations of anaerobic metabolic rate may be open to question, the data indicate that studies that ignore possible changes in anaerobic metabolism might underestimate the influence of heat strain on metabolic rate and nutritional needs.

Investigations that report a lower metabolic rate during exercise in the heat also report increased plasma or muscle lactate levels<sup>94–96</sup> or an increased respiratory exchange ratio,<sup>93</sup> also suggesting an increased anaerobic metabolism. However, any inference about metabolic events within skeletal muscle based on changes in plasma lactate is inconclusive<sup>97</sup> because plasma lactate concentration reflects the balance between efflux into the blood and removal from the blood. Rowell and associates<sup>98</sup> have shown that during exercise in the heat, splanchnic vasoconstriction reduced hepatic removal of plasma lactate. Therefore, the greater blood lactate accumulation during submaximal exercise in the heat can be attributed in part to a redistribution of blood flow away from the splanchnic tissues.

### Skeletal Muscle Metabolism

Several investigators have examined environmental heat stress effects on skeletal muscle metabolism during exercise.<sup>92,96,99–101</sup> When exercising in the heat (vs exercising in the cold), plasma lactate levels and muscle glycogen utilization are increased. When exercising in the heat (vs exercising in temperate conditions), plasma lactate levels are increased and skeletal muscle glycogen utilization is increased<sup>99,100</sup> or not altered.<sup>96,101</sup> Other studies, however, do not support the concept of an increased anaerobic metabolism in the heat. For example, two studies<sup>102,103</sup> that measured arterial and venous lactate concentration across the active musculature during exercise in the heat found that heat stress did not alter lactate levels, muscle blood flow, or glycogen utilization rates. In addition, when exercising in the heat, as opposed to temperate conditions, muscle creatine phosphate levels are lower but muscle creatine levels are higher, with no differences for muscle total adenine nucleotide pool, the ratio of adenosine 5'-triphosphate (ATP) to adenosine 5'-diphosphate (ADP), or inosine monophosphate (IMP).<sup>100</sup>

The question remains as to what physiological mechanism or mechanisms might be responsible for an increased anaerobic metabolism during exercise in the heat. One possibility is the redistribution of blood to the cutaneous veins for heat dissipation; this may result in reduced perfusion of the active skeletal muscles and, thus, local tissue hypoxia.<sup>104</sup> Muscle blood flow has been reported to decrease during resting heat exposure,<sup>105,106</sup> but not during exercise-heat stress.<sup>102,103</sup> A second possibility is that heat stress elevates circulating catecholamines,<sup>107</sup> which increases glycogen utilization in skeletal muscle during exercise.<sup>99,108</sup> A third possibility is that heat stress increases the recruitment of fast-twitch motor units.<sup>109</sup> Fast-twitch skeletal muscles derive a greater percentage of their total energy expenditure from anaerobic pathways, regardless of their level of perfusion, than do slow-twitch fibers.<sup>110,111</sup> A fourth possibility is that high muscle temperatures<sup>99,112</sup> alter glycogen utilization rate through a  $Q_{10}$  effect (the ratio of the reaction rates at two temperatures 10 Centigrade degrees apart).<sup>113</sup> Regardless of the mechanism or mechanisms, an increased anaerobic metabolism, increased muscle lactate, and increased glycogen utilization can all contribute to reduced physical exercise performance in the heat.

### Influence of Heat Acclimation

If heat stress alters metabolism, then does heat acclimation abate these changes? Heat acclimation probably decreases the metabolic heat load that needs to be dissipated at a given submaximal exercise power output.<sup>88,109</sup> Most reports indicate that oxygen uptake and aerobic metabolic rate during submaximal exercise are reduced by heat acclimation, although a significant effect is not always observed.<sup>88</sup> Large effects (14%–17% reductions) have been reported for stair-stepping,<sup>114–116</sup> but much of the reduction in oxygen uptake required for stair-stepping can be attributed to increased skill and improved efficiency acquired during the acclimation program. By contrast, other studies<sup>109,117–120</sup> report acclimation-induced reductions in the oxygen uptake required for treadmill and cycle-ergometer exercise, which although statistically significant, were much smaller than the large reductions reported for stair-stepping.

Heat acclimation reduces muscle lactate levels<sup>96,100</sup> and glycogen utilization<sup>96,100,121,122</sup> during submaximal exercise. The magnitude of the glycogen-sparing effect during exercise induced by heat acclimation seems to vary widely (15%–50%).<sup>99,121,122</sup>

In addition, one investigator<sup>96</sup> observed a statistically significant glycogen-sparing effect during exercise after heat acclimation, but the reduction in glycogen utilization was small (10%) in a hot climate, and more apparent (49%) in a temperate climate.

### Cutaneous Heat Loss

The skin provides a large surface area to exchange heat between the body and the environment. Dry heat loss occurs by increasing cutaneous blood volume and flow. Evaporative heat loss occurs by the evaporation of secreted sweat, which cools the blood in superficial vasculature.

### Sweating and Evaporative Heat Loss

As the ambient temperature increases, there is greater dependence on evaporative heat loss to defend core temperature during exercise (see Figure 3-4; this concept is also discussed in Chapter 2, Human Adaptation to Hot Environments). Figure 3-12 represents the pattern of sweat secretion, as measured by skin dew point hygrometry, during exercise.<sup>29</sup> Thermoregulatory sweating can begin within a few seconds to minutes after starting muscular exercise,<sup>123</sup> depending on several factors, including skin temperature, previous sweating, and body hydration status. The increase in thermoregulatory sweating closely parallels the increase in body temperature and is accomplished first by recruitment of sweat glands and then by increased sweat secretion per gland.<sup>124–126</sup> Both the density of sweat glands and local sweat rates vary among dif-

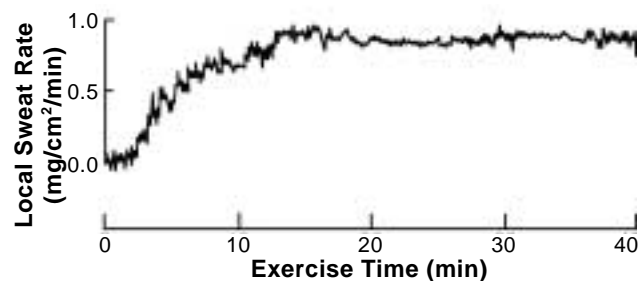


Fig. 3-12. Pattern of sweat gland secretion during exercise-heat stress, at 50% maximal oxygen consumption, 28°C, and 30% relative humidity. Reprinted with permission from Sawka MN, Wenger CB. Physiological responses to acute exercise-heat stress. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 129.

ferent regions of the skin. Figure 3-13 shows local sweating responses plotted against core temperature. Note that for a given core temperature, the back and chest have the greatest sweating rates. Conversely, the limbs will have relatively high sweating rates only after a substantial elevation (0.5°C is considered substantial) in core temperature.

The rate of sweat evaporation depends on air movement and the water vapor pressure gradient between the skin and the environment, so in still or moist air, sweat tends to collect on the skin. If sweat is secreted but not evaporated rapidly enough to maintain heat balance, core temperature will increase and physical exercise performance will be compromised. In addition, prolonged wetting of the skin surface reduces sweat secretion<sup>127-129</sup> through hidromeiosis (which is probably mediated by the stratum corneum's swelling and occluding the sweat ducts). For these reasons it is important to allow air circulation to the skin, especially on the torso, to maximize evaporative cooling.<sup>130</sup> Likewise, if clothes become soaked, not only are they uncomfortable, but the wetted skin can also inhibit sweat secretion. Clearly, military personnel deployed in hot climates should wear dry clothing next to the skin, and wear clothing systems that allow venting.

### Skin Blood Flow and Dry Heat Loss

During heat stress, the cardiovascular system acts to transfer heat from the body core to the skin.<sup>39</sup> Cutaneous vasodilation occurs with heat stress due

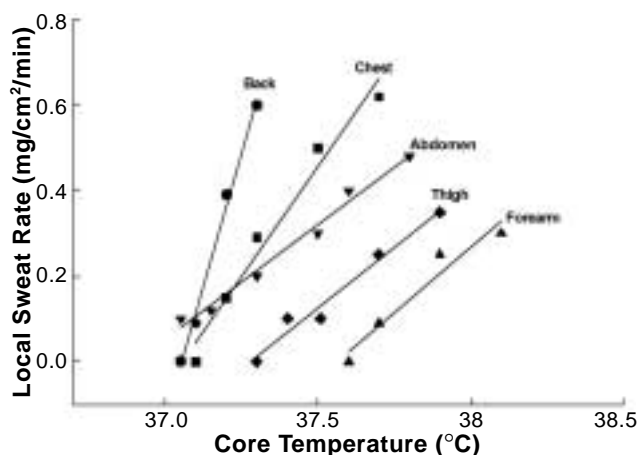


Fig. 3-13. Regional sweating responses plotted against core temperature. Adapted with permission from Nadel ER, Mitchell JW, Saltin B, Stolwijk JAJ. Peripheral modifications to the central drive for sweating. *J Appl Physiol.* 1971;31:830.

to reflex and local mechanisms,<sup>25,131,132</sup> and causes warm blood to be redirected from the core to the skin, which increases skin temperature. Skin temperature varies between body regions, but it generally increases with ambient temperature and remains below core temperature. We saw in Figure 3-2 the relationships between regional skin temperatures and core temperature over a range of temperate and hot conditions.<sup>39</sup> In any given environment, skin temperature reflects the balance between blood flow and sweating. Blood flow transfers heat by convection from the deep tissues to the skin. When core and skin temperatures are low enough that sweating does not occur, increasing skin blood flow will elevate skin temperature nearer to blood temperature, and decreasing skin blood flow will lower skin temperature nearer to ambient temperature. Thus, dry heat loss is controlled by varying skin blood flow and, thereby, skin temperature. In conditions in which sweating occurs, the tendency of skin blood flow to warm the skin is approximately balanced by the tendency of sweating to cool the skin. Therefore, after sweating has begun, a high skin blood flow acts primarily to deliver heat for evaporation of sweat.

As discussed earlier, skin temperature is higher in warmer environments, while core temperature is relatively unaffected over a wide range of ambient temperatures. Thus as ambient temperature increases, the core-to-skin thermal gradient becomes narrower, and skin blood flow increases in response to the high skin temperature, so as to achieve core-to-skin heat transfer sufficient for thermal balance. When performing military tasks, metabolic heat production may often be 3- to 8-fold higher than resting levels (see Table 3-2), and delivery of heat to the skin must increase proportionately to reestablish thermal balance. Core temperature does increase during exercise, thus widening the core-to-skin temperature gradient. However, the widening of the core-to-skin temperature gradient is proportionately less than the increase in heat production, so that only by a skin blood flow increase does core-to-skin heat transfer increase enough to match heat production and allow thermal balance to be reestablished.<sup>25,104</sup> Conversely, if increased evaporative cooling lowers skin temperature, the core-to-skin thermal gradient becomes wider, and skin blood flow requirements to achieve the same heat transfer are proportionately decreased.<sup>131</sup>

### Circulation

During maximal vasodilation elicited by heating,

skin blood flow can approach 8 L/min.<sup>132</sup> Although the skin is not maximally vasodilated during exercise in the heat,<sup>133</sup> skin blood flow can still reach levels of several liters per minute.<sup>134</sup> During exercise–heat stress, maintaining a high skin blood flow can impose a substantial burden on the cardiovascular system.<sup>39</sup> High skin blood flow promotes pooling of blood in the skin and superficial veins, thus reducing central venous pressure, cardiac filling, and stroke volume, and requiring a higher heart rate to maintain cardiac output. In addition, if fluid lost through sweating is not replaced, the resulting reduction in blood volume aggravates the hemodynamic effects of peripheral pooling.<sup>123</sup> To help maintain stroke volume despite decreased cardiac filling, cardiac contractility increases as a result of elevated sympathetic activity and direct effects of temperature on the myocardium.<sup>25,132</sup>

During exercise in the heat, the primary cardiovascular challenge is to have sufficient cardiac output to simultaneously support high skin blood flow for heat dissipation and high muscle blood flow for metabolism. Figure 3-14 provides an analysis of cardiac output responses and the redistribution of cardiac output during rest and exercise in temperate and hot environments.<sup>104</sup> This figure depicts cardiac output as unchanged at rest and elevated during exercise in the heat. In this figure, the higher skin blood flow required for thermoregulation in the heat is provided at rest almost entirely by diverting blood flow from other vascular beds (chiefly renal and splanchnic circulations); but during ex-

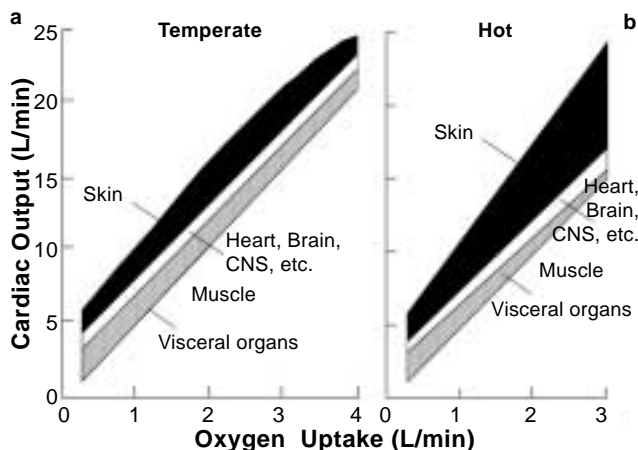
ercise, much of the additional skin blood flow in the heat is provided by increasing cardiac output. Thus maximum cardiac output is reached at a lower exercise intensity in the heat than in a temperate environment. This pattern is consistent in its outlines with other studies that investigated the relation of cardiac output responses during exercise to the intensity and duration of heat stress.<sup>135–137</sup> During mild heat stress, cardiac output is unchanged<sup>135,136</sup> or increased,<sup>137</sup> but during severe heat stress, cardiac output may even be decreased<sup>135</sup> below levels observed in temperate conditions.

Brain, spinal cord, and coronary blood flow are believed to be unaffected by exercise–heat stress.<sup>132,138</sup> However, visceral (splanchnic, renal) blood flow is reduced by both exercise and heat stress due to increased sympathetic activity mediated through thermal receptor stimulation.<sup>132,138</sup> The visceral blood flow reductions are graded to the exercise intensity, and the effects of exercise and heat seem to be additive.<sup>25,138</sup> Reduced visceral blood flow allows a corresponding diversion of cardiac output to skin and exercising muscle. Also, secondarily to reduced visceral blood flow, a substantial volume of blood can be mobilized from the compliant splanchnic beds to help maintain cardiac filling during exercise–heat stress.<sup>25</sup> If these compensatory responses are insufficient, skin and muscle blood flow may be compromised. Although Figure 3-14 indicates that muscle blood flow decreases during severe exercise–heat stress,<sup>138</sup> this conclusion is controversial.<sup>25,132</sup>

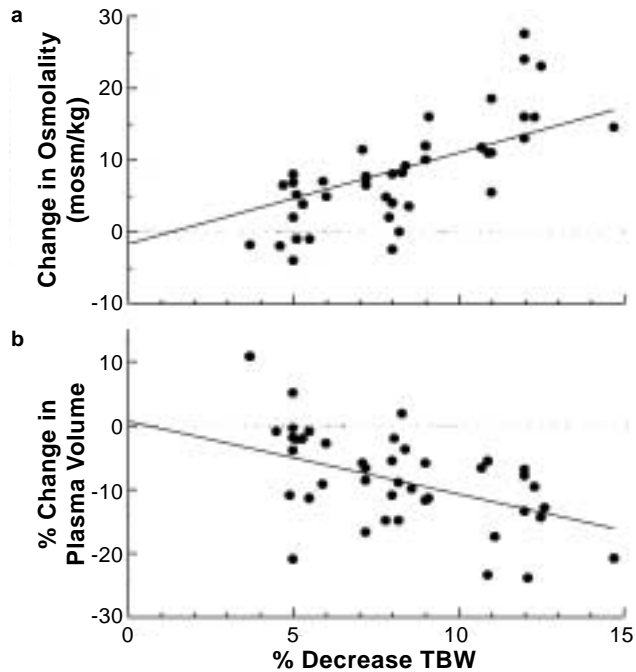
### Body Fluid Losses

If fluid losses during exercise–heat stress are not replaced, then negative fluid balance occurs, decreasing subjects' total body water and making them hypohydrated. Water is the largest component of the human body, comprising 45% to 70% of body weight.<sup>123</sup> The average 75-kg man contains about 45 L of water, or about 60% of body weight. Because adipose tissue is about 10% water and muscle tissue is about 75% water, total body water is inversely related to body fat content.<sup>123</sup> In addition, muscle water and glycogen content parallel each other, probably because of the osmotic pressure exerted by glycogen molecules within the sarcoplasm.<sup>139</sup> As a result, physically trained soldiers have relatively greater total body water than their sedentary counterparts, by virtue of a smaller percentage of body fat and a higher skeletal muscle glycogen concentration.<sup>140</sup>

Figure 3-15 presents resting plasma volume and osmolality values for heat-acclimated persons when hypohydrated at various levels.<sup>64</sup> Sweat-induced



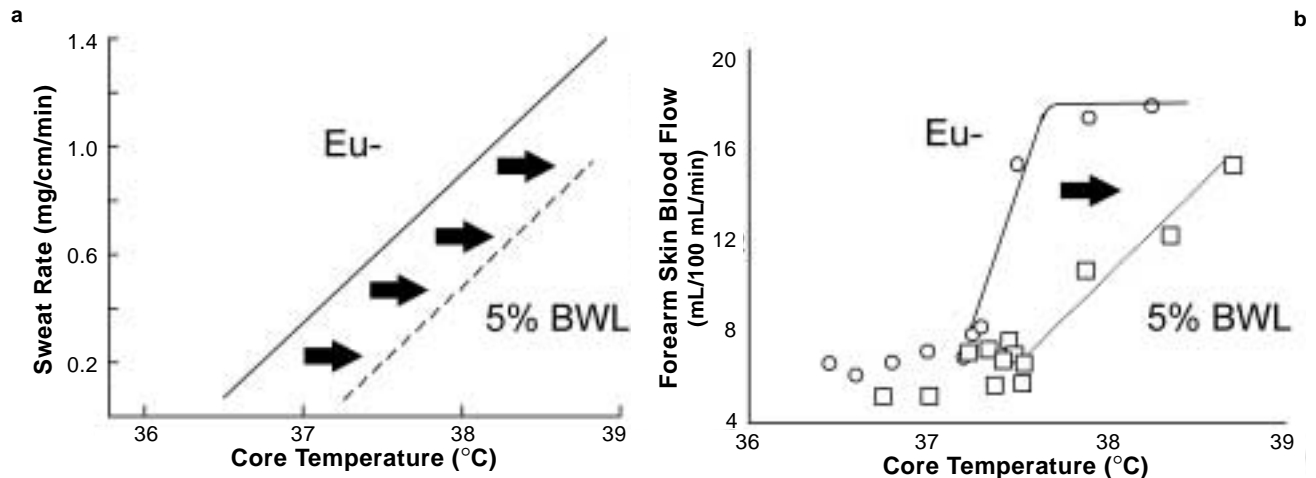
**Fig. 3-14.** Hypothetical cardiac output response and redistribution during rest (the low end of the curve) and exercise in (a) temperate ( $\leq 25^{\circ}\text{C}$ ) and (b) hot ( $> 30^{\circ}\text{C}$ ) environments. Adapted with permission from Rowell LB. *Human Circulation: Regulation During Physical Stress*. New York, NY: Oxford University Press; 1986: 375.



**Fig. 3-15.** The effect of loss of total body water (TBW) on changes in (a) plasma osmolality and (b) plasma volume (PV) in heat-acclimated persons. Reprinted with permission from Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise-heat exposure. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport*. Boca Raton, Fla: CRC Press; 1996: 142.

dehydration will decrease plasma volume and increase plasma osmotic pressure in proportion to the level of fluid loss. Plasma volume decreases because it provides the precursor fluid for sweat, and osmolality increases because sweat is ordinarily hypotonic relative to plasma. Increased sodium and chloride concentrations are primarily responsible for the elevated plasma osmolality.<sup>141-143</sup> It is the plasma hyperosmolality that mobilizes fluid from the intracellular to the extracellular space to enable plasma volume defense in hypohydrated subjects. An important consequence of this mechanism is that heat-acclimated persons, who secrete more dilute sweat than unacclimated persons, have a smaller plasma volume reduction for a given body water deficit.<sup>144</sup> The smaller loss of plasma volume occurs because the more dilute sweat of heat-acclimated persons leaves more solute remaining within the extracellular space to increase extracellular osmotic pressure and redistribute fluid from the intracellular space.

Some persons use diuretics for medical purposes or to reduce their body weight to “make weight” for an athletic competition. Commonly used diuretics include thiazides (eg, Diuril), carbonic anhydrase inhibitors (eg, Diamox), and furosemide (eg, Lasix). Diuretics increase the formation of urine and most of them cause the loss of sodium as well as water, so that diuretic-induced dehydration generally results in an isosmotic hypovolemia, with a



**Fig. 3-16.** (a) Local sweat rate and (b) forearm skin blood flow (FBF) response data for euhydrated (Eu) and dehydrated (5% body water loss [BWL]) individuals during exercise-heat stress. Open circles: euhydrated; open squares: 5% BWL. Data sources: (a) Sawka MN, Gonzalez RR, Young AJ, Dennis RC, Valeri CR, Pandolf KB. Control of thermoregulatory sweating during exercise in the heat. *Am J Physiol*. 1989;257:R311-R316. (b) Kenney WL, Tankersley CG, Newswanger DL, Hyde DE, Puhl SM, Turnera NL. Age and hypohydration independently influence the peripheral vascular response to heat stress. *J Appl Physiol*. 1990;8:1902-1908. Adapted with permission from Sawka MN. Physiological consequences of hydration: Exercise performance and thermoregulation. *Med Sci Sports Exerc*. 1992;24:664.

much greater ratio of plasma loss to total body water loss than does either exercise- or heat-induced dehydration. Correspondingly less intracellular fluid is lost after diuretic administration because there is not an increase in extracellular osmolality to promote redistribution of water from the intracellular to the extracellular space. Diuretic-induced dehydration produces the same adverse effects, including core temperature elevations, as those mediated by underdrinking.<sup>144</sup> For the same total loss of body water one might theoretically expect diuretic-induced dehydration to cause greater cardiovascular strain than thermal dehydration because of the greater loss of extracellular fluid volume in diuretic-induced dehydration. However, such a difference in cardiovascular strain has not been established.

As discussed earlier, dehydration causes an increased core temperature during rest and exercise-heat stress, by impairing both dry and evaporative

heat loss (or, if the air is warmer than the skin, by aggravating dry heat gain).<sup>144-147</sup> Figure 3-16 presents the local sweating responses<sup>148</sup> and skin blood flow responses<sup>149</sup> to dehydration (5% body weight loss) during exercise in the heat. These curves indicate that dehydration reduced both heat-loss effector responses (ie, sweat rate and skin blood flow) for a given core temperature level.<sup>144</sup> Dehydration is usually associated with either reduced or unchanged whole-body sweating rates at a given metabolic rate in the heat.<sup>150</sup> However, even when dehydration is associated with no change in sweating rate, core temperature is usually elevated, so that sweating rate for a given core temperature is lower when hypohydrated.<sup>150</sup> The physiological mechanisms mediating the reduced dry and evaporative heat loss from dehydration include both the separate and combined effects of plasma hyperosmolality<sup>144,151,152</sup> and reduced blood volume.<sup>153-155</sup>

## PHYSICAL EXERCISE PERFORMANCE

Troops deployed to hot climates are usually there to perform a mission that requires physical exercise. The amount of physical exercise they can perform is based on the climatic conditions, biomedical state (ie, acclimation state, physical fitness, hydration level, and health), and mission requirements (eg, required metabolic rate, duration, and clothing or equipment worn). Together, these factors determine the physiological strain (elevated core temperature and cardiovascular strain), which, in turn, determines exercise performance decrements. Heat stress will induce decrements in physical exercise performance; however, the exact magnitude of the decrement is difficult to predict. The effects of heat stress on exercise performance can be divided into (a) the effects on the maximum exercise intensity that an individual can achieve and (b) the effects on performance of submaximal exercise.

### Maximal Intensity Exercise

Maximal exercise intensity is achieved by performing exercise of increasing intensity until physiological criteria (ie, the determination of maximal aerobic power, or maximal oxygen uptake  $\dot{V}O_{2max}$  or volitional exhaustion (ie, physical exercise capacity) are reached. An activity that requires a low fraction of  $\dot{V}O_{2max}$  is sustained more easily than one that requires a high fraction of  $\dot{V}O_{2max}$ . In addition, the sorts of training that increase  $\dot{V}O_{2max}$  also increase the ease of performing an activity at any given fraction of

$\dot{V}O_{2max}$ . For both reasons, high  $\dot{V}O_{2max}$  is important to successfully perform tasks that require sustained high metabolic rates, so that a lower  $\dot{V}O_{2max}$  often translates into reduced physical exercise performance.  $\dot{V}O_{2max}$  is defined as the maximal rate at which oxygen can be utilized by body tissues during physical exercise. Most investigators find that  $\dot{V}O_{2max}$  is lower in hot than in temperate climates.<sup>156-159</sup> For example,  $\dot{V}O_{2max}$  was 0.25 L/min (7%) lower at 49°C than at 21°C in one study,<sup>160</sup> and the state of heat acclimation in this study did not alter the size of the  $\dot{V}O_{2max}$  decrement (Figure 3-17). However, some investigators report no effect of ambient temperature on  $\dot{V}O_{2max}$ .<sup>95,161</sup>

What physiological mechanisms might be responsible for such a reduction in  $\dot{V}O_{2max}$ ? Thermal stress, by dilating cutaneous arterioles, might divert some of the cardiac output from skeletal muscle to skin, thus leaving less blood flow to support the metabolism of exercising skeletal muscle. In addition, dilation of the cutaneous vascular bed may increase cutaneous blood volume at the expense of central blood volume, thus reducing venous return and cardiac output. In one study,<sup>135</sup> for example, heat exposure reduced cardiac output during intense (~73%  $\dot{V}O_{2max}$ ) exercise by 1.2 L/min below control levels. Such a reduction in cardiac output during heat exposure could account for a decrement of 0.25 L/min in  $\dot{V}O_{2max}$ , assuming each liter of blood delivers approximately 0.2 L of oxygen (1.34 mL of oxygen per gram of hemoglobin • 15 g hemoglobin per 100 mL of blood).



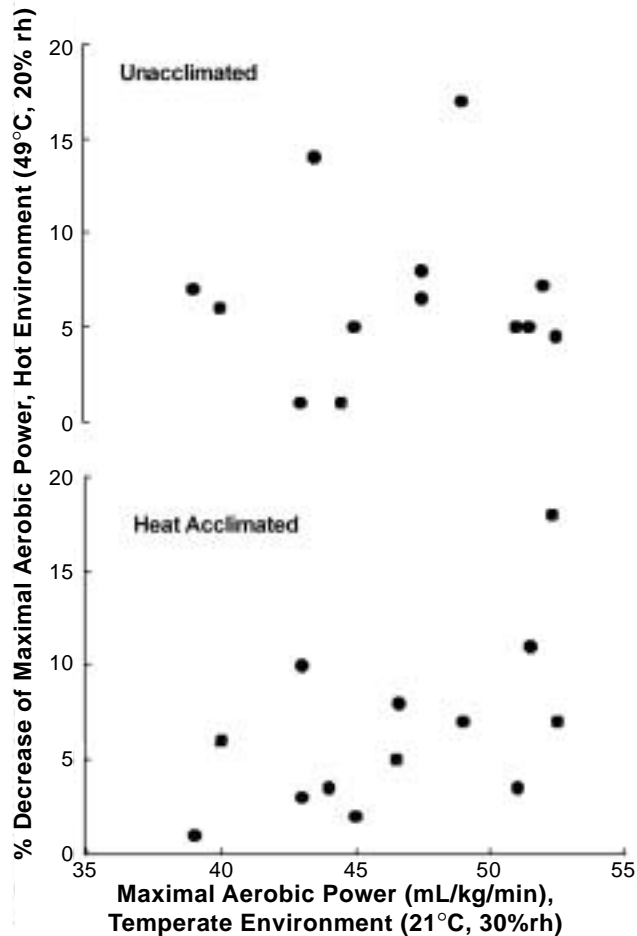


Fig. 3-17. Effects of heat exposure and heat acclimation on maximal aerobic power. The data show that neither initial fitness nor the heat acclimation state have an effect. Adapted with permission from Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol.* 1985;53:296.

### Submaximal Intensity Exercise

Numerous studies have examined the influence of dehydration on  $\dot{V}O_2\text{max}$  and physical exercise capacity (exercise to volitional exhaustion; ie, exhaustion as determined by the subject rather than by physiological criteria).<sup>64</sup> In temperate environments, a body water deficit of less than 3% body weight loss does not alter  $\dot{V}O_2\text{max}$ ,<sup>64</sup> but  $\dot{V}O_2\text{max}$  is reported to be decreased<sup>66,162,163</sup> by dehydration that equals or exceeds 3% body weight loss (Figure 3-18). Therefore, there may be a critical water deficit (3% body weight loss) that exists before dehydration reduces

$\dot{V}O_2\text{max}$  in temperate environments. In the heat, small to moderate water deficits (ie, 2%–4% body weight loss) can result in a large reduction of  $\dot{V}O_2\text{max}$ .<sup>164</sup> Thus, it seems that climatic heat stress has a potentiating effect on the reduction of  $\dot{V}O_2\text{max}$  caused by dehydration.

Physiological mechanisms for reduced submaximal intensity exercise performance in the heat include increased thermal and cardiovascular strain, more rapid glycogen depletion, increased metabolite accumulation, and diminished motivation for exercise.<sup>25,165,166</sup> Although the exact mechanism or mechanisms are unknown, they probably depend on the specific heat stress, exercise task, and biomedical state of the individual.

Figure 3-19 demonstrates the effects of air temperature and dehydration on the submaximal exercise output of soldiers.<sup>53</sup> This analysis is based on heat-acclimated soldiers marching at a metabolic rate of approximately 650 W (which represents high-intensity exercise for military or occupational tasks, or both), and an air temperature of 43°C with low humidity.<sup>41</sup> Environmental heat stress reduced submaximal exercise output at all hydration levels. Adolph and associates<sup>53</sup> suggested that for every five Centigrade degree increase in skin temperature there was a 10% decrement in endurance time during submaximal exercise. In addition, the decrements in submaximal exercise output from heat stress and from dehydration were additive.<sup>53</sup> For example, exposure to 43°C reduced submaximal exercise output by approximately 20% (compared with temperate conditions), and a 2.5% (body weight loss) dehydration reduced submaximal exercise output (compared with euhydration) by the same amount. If heat stress and dehydration were experienced together, soldiers would experience a 50% decrease in submaximal exercise output.

Studies of the effects of dehydration on soldiers' ability to tolerate heat strain during submaximal intensity exercise demonstrate that soldiers who drink water can continue to exercise in the heat for many hours, whereas those who do not drink adequate amounts of water must discontinue because of exhaustion. In the summers of 1942 and 1943, Adolph and associates<sup>53</sup> conducted experiments in the California deserts, in which heat-acclimated soldiers attempted endurance marches (4–6.5 km/h) in an ambient temperature of approximately 38°C, and either drank water ad libitum or refrained from drinking. They reported that during 3- to 8-hour desert walks, 1 of 59 (2%) and 11 of 70 soldiers (16%) suffered exhaustion from heat strain when they did

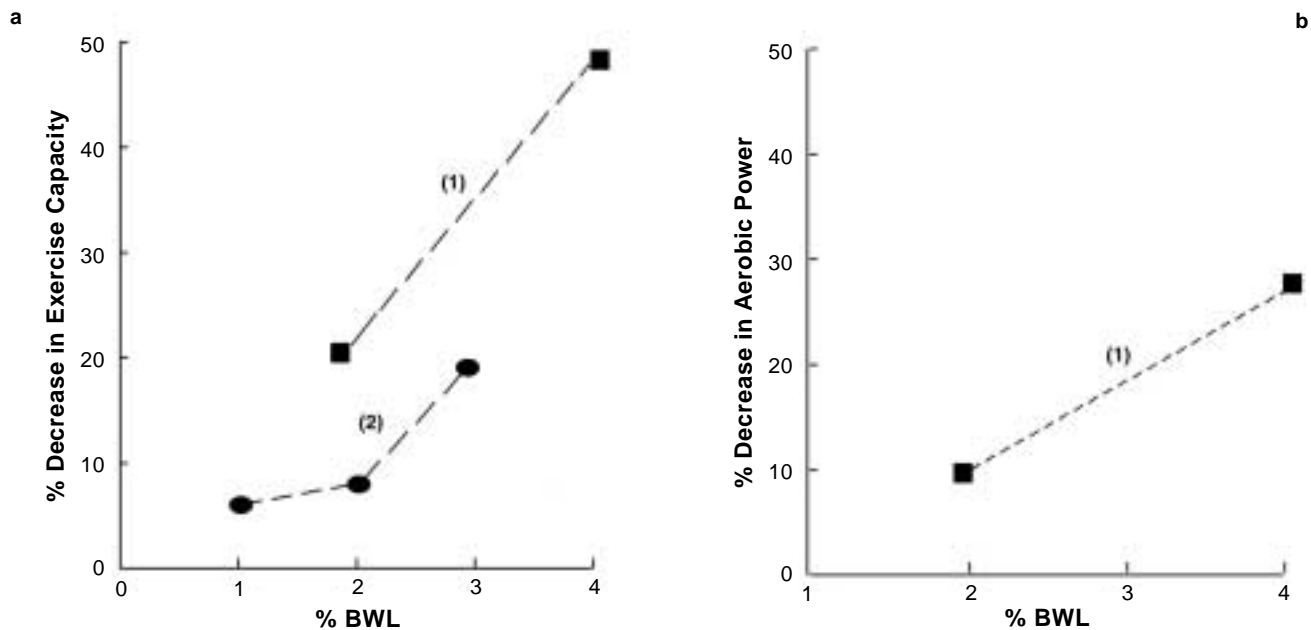


Fig. 3-18. Relationships between dehydration level and decrements in (a) physical exercise capacity and (b) maximal aerobic power during heat exposure. BWL: body water loss. Data sources: (1) Craig FN, Cummings EG. Dehydration and muscular work. *J Appl Physiol.* 1966;21:670-674. (2) Pinchan G, Gauttam RK, Tomar OS, Bajaj AC. Effects of primary hypohydration on physical work capacity. *Int J Biometeorol.* 1988;32:176-180.

or did not drink water, respectively. In other experiments, they reported that 1 of 59 soldiers (2%) and 15 of 70 soldiers (21%) suffered exhaustion from heat strain during an attempted 8-hour desert march when they did or did not drink water, respectively.

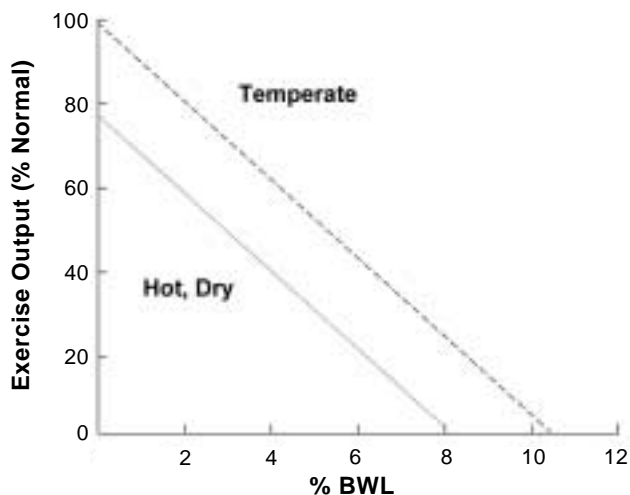


Fig. 3-19. Effects of air temperature and dehydration level on submaximal exercise output of soldiers. BWL: body water loss. Adapted from Adolph EF, Associates. *Physiology of Man in the Desert.* New York, NY: Intersciences, Inc; 1947: 207.

Ladell<sup>167</sup> had subjects attempt a 140-minute walk in a hot ( $T_a = 38^\circ\text{C}$ ) climate while ingesting different combinations of salt and water. He reported that exhaustion from heat strain occurred in 9 of 12 (75%) experiments when subjects received neither water nor salt, and 3 of 41 (7%) experiments when they received only water. Sawka and colleagues<sup>61</sup> had eight heat-acclimated soldiers attempt treadmill walks ( $\sim 25\% \dot{V}O_{2\text{max}}$  for 140 min) in a hot-dry ( $T_a = 49^\circ\text{C}$ ,  $\text{rh} = 20\%$ ) climate when euhydrated and when hypohydrated by 3%, 5%, and 7% of their body weight. All eight soldiers completed the euhydration and 3% dehydration experiments, and seven subjects completed the 5% dehydration experiments. For the 7% dehydration experiments, six soldiers discontinued after completing an average of 64 minutes. Clearly, dehydration reduces submaximal exercise performance and increases the occurrence of exhaustion from heat strain.

Either heat stress alone or dehydration alone can impair athletic endurance exercise performance. One study<sup>166</sup> found that cycle exercise endurance time was reduced by 38% in hot versus temperate conditions. Another study<sup>168</sup> examined the effects of dehydration on the performance of athletes competing in 1,500-m, 5,000-m, and 10,000-m races when euhydrated and when hypohydrated by only 2% of body weight. Dehydration impaired running

performance at all race distances, but to a greater extent in the longer races (~ 5% for the 5,000 m and 10,000 m) than in the shorter race (3% for 1,500 m). Dehydration also adversely affects rowing performance.<sup>169</sup>

Studies<sup>170,171</sup> have examined the adverse effects of dehydration on moderate to intense cycle ergometer performance. In both, high-intensity performance tests were conducted immediately after approximately 60 minutes of cycling, during which volunteers either drank nothing or drank sufficient fluid to replace sweat losses. In one of the studies,<sup>170</sup> time to fatigue when cycling at 90%  $\dot{V}O_2\text{max}$  was 51% longer (9.8 min vs 6.5 min) when subjects drank sufficient fluids to prevent dehydration. The other study<sup>171</sup> found that cyclists completed a performance ride 6.5% faster if they drank fluids during exercise. The results of these studies clearly demonstrate the detrimental effects of dehydration on submaximal exercise performance.

### Heat Tolerance

Uncompensable heat stress is a condition in which the required evaporative cooling ( $E_{\text{req}}$ ) is greater than the maximal evaporative cooling ( $E_{\text{max}}$ ) permitted by the environment. Therefore, during uncompensable heat stress, steady state core temperature cannot be achieved and body temperature continues to rise until exhaustion occurs. Ambient temperature, dew point, wind velocity, metabolic rate, and clothing are important factors that influence whether exercise-heat stress will be compensable or uncompensable. Uncompensable heat stress is associated with exhaustion from heat strain occurring at relatively low core temperatures.<sup>5,6,172</sup> Because evaporative cooling is impaired during uncompensable heat stress, skin temperature is high, causing the blood vessels in the skin to dilate. It is believed that the resulting displacement of blood to skin causes cardiovascular strain and instability, which accounts for the occurrence of exhaustion at relatively low core temperatures during uncompensable heat stress. Much higher core temperatures can be tolerated during compensable heat stress, and exhaustion is then usually associated with dehydration or substrate depletion.

Little is known about relationships between physiological indices and exhaustion from heat strain.<sup>5,6,173,174</sup> Core temperature provides the most reliable physiological index to predict the incidence of exhaustion from heat strain.<sup>5,6</sup> For heat-acclimated subjects exercising in uncompensable heat stress, Figure 3-20 presents the relationship between core temperature and the cumulative occurrence of

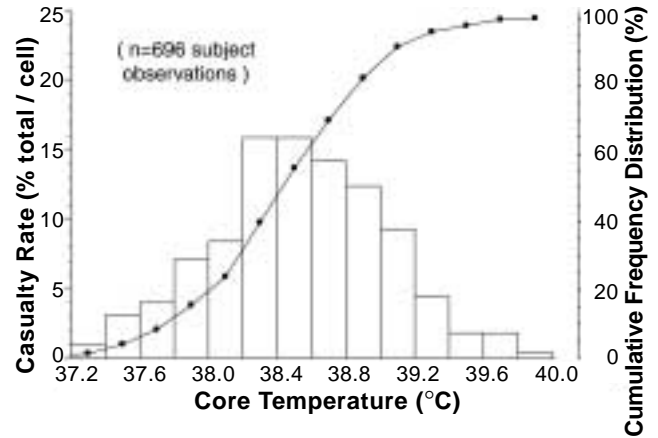


Fig. 3-20. Relationship between core temperature and cumulative occurrence of exhaustion from heat strain, and the fraction of cases of exhaustion from heat strain occurring in each 0.2 Centigrade degree interval of core temperature. Data source: Kraning KK. Analysis of data from 696 subject observations during experiments conducted at US Army Research Institute of Environmental Medicine, Natick, Mass; 1997. Unpublished.

exhaustion from heat strain, and also the fraction of cases of exhaustion from heat strain occurring in each interval of 0.2 Centigrade degrees of core temperature. Figure 3-21 presents the relationships between core temperature and the cumulative occurrence of exhaustion from heat strain for heat-acclimated persons exercising in uncompensable<sup>5</sup> and compensable<sup>175</sup> heat stress. During uncompensable heat stress, exhaustion was rarely associated with a core temperature below 38°C, but exhaustion always occurred before a temperature of 40°C was achieved.<sup>5,6</sup>

Several recent studies<sup>5,6,77,174</sup> examined the effects of different biomedical factors (eg, aerobic fitness, hydration, heat acclimation state) on physiological tolerance to uncompensable heat stress (Table 3-3). Sawka and colleagues<sup>5</sup> studied the effects of dehydration and aerobic fitness on physiological tolerance to uncompensable heat stress. Heat-acclimated soldiers walked to physical exhaustion when either euhydrated or hypohydrated (8% of total body water). A combination of environmental conditions ( $T_a = 49^\circ\text{C}$ ,  $\text{rh} = 20\%$ ) and exercise intensity (47%  $\dot{V}O_2\text{max}$ ) was used that would not allow thermal equilibrium to be reached, so that exhaustion from heat strain would eventually occur. Dehydration reduced tolerance time (from 121 min to 55 min), but more important, dehydration reduced the core temperature that a person could tolerate. Exhaustion occurred at a core temperature approximately 0.4°C lower when hypohydrated than when euhydrated. They found no influence of aerobic fitness on physi-

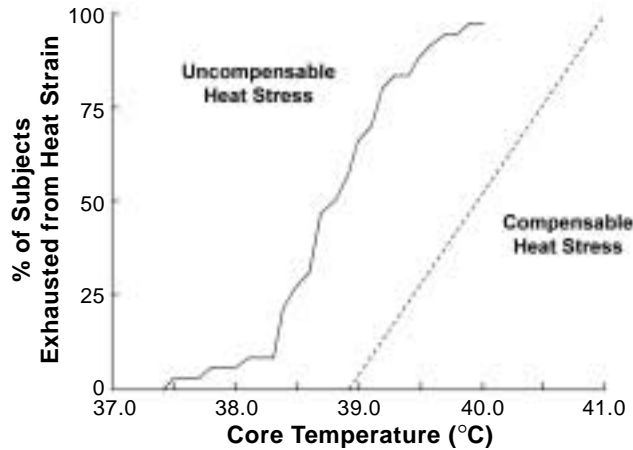


Fig. 3-21. Relationships between core temperature and occurrence of exhaustion from heat strain during uncompensable and compensable heat stress. The different shapes of the curves reflect the different experimental conditions. Data sources for uncompensable heat stress: (1) Sawka MN, Young AJ, Lutzka WA, Neuffer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. *J Appl Physiol.* 1992;73:368–375. (2) Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol.* 1994;77:216–222. (3) Lutzka WA, Sawka MN, Matott RP, Staab JE, Montain SJ, Pandolf KB. *Hyperhydration: Physiologic and Thermoregulatory Effects During Compensable and Uncompensable Exercise–Heat Stress.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1996. Technical Report TR96-6. Data source for compensable heat stress: (1) 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.

ological tolerance to uncompensable heat stress. These findings suggest that dehydration not only impairs exercise performance but also reduces tolerance to heat strain.

Montain and colleagues<sup>6</sup> studied the influence of exercise intensity, protective clothing (including NBC protective gear, armored vests, etc), and environment on physiological tolerance to uncompensable heat stress. They had heat-acclimated soldiers walk to physical exhaustion at two metabolic rates (425 W and 600 W) when wearing full or partial protective clothing ensembles in a hot-humid and a desert environment. They observed that wearing the full protective clothing ensemble reduces physiological tolerance, as core temperature at exhaustion was lower than in the partial ensemble. They also re-

ported that the exercise intensity and the environment in the laboratory did not alter the core temperature at exhaustion during uncompensable heat stress. Wearing protective clothing was believed to reduce physiological tolerance to heat strain because of higher skin temperature causing greater displacement of blood from central circulation to the skin.

Nielsen and colleagues<sup>174</sup> studied the effects of heat acclimation in an uncompensable heat stress condition. They had highly trained subjects perform exercise (60%  $\dot{V}O_2\text{max}$ ) to exhaustion for 9 to 12 days at 40°C. They reported that final core temperature was consistently 39.7°C at exhaustion and was not changed by heat acclimation; however, endurance time was increased (because the rate of core temperature increase was slowed). They also observed increased sweating rate, increased cardiac output, and reduced heart rate at exhaustion after heat acclimation. The state of heat acclimation did not affect these trained subjects' tolerance to high core temperature during uncompensable heat stress.

High core temperatures are better tolerated during compensable heat stress, presumably because the lower skin temperatures result in a lesser degree of cardiovascular strain than at the same core temperature during uncompensable heat stress. Joy and Goldman,<sup>173</sup> for example, reported that 35 of 63 (56%) elite soldiers were still performing military tasks when core temperature reached 39.5°C, their predetermined endpoint criterion. Some individuals can tolerate core temperatures greater than 40°C and continue to exercise during compensable heat stress.<sup>176–180</sup> For example, Pugh and colleagues<sup>177</sup> measured the core temperatures of 47 runners immediately after they completed a marathon race. Seven, including three of the first five finishers, had core temperatures greater than 40°C (the highest value was 41°C). None of the runners who did not finish the race achieved core temperatures as high as 40°C. In another study,<sup>180</sup> eight trained and acclimatized subjects marched 31.5 km in 8 hours on a summer day, carrying 35-kg loads. On completion, five had rectal temperatures between 41.5°C and 42.4°C. Such high core temperatures should not be considered to be safe, however. In a series of young men (aged 17–24 years) with exertional heatstroke, presenting rectal temperatures were recorded on 30 patients; and of these, 14 had presenting rectal temperatures lower than or equal to 41°C.<sup>181</sup> Tolerance to high core temperature is not well understood, and it is not possible to predict who can tolerate a high core temperature, and in what circumstances, without ill effects.

TABLE 3-3

## SUMMARY OF STUDIES EXAMINING THE INFLUENCE OF BIOMEDICAL FACTORS ON PHYSIOLOGICAL TOLERANCE TO UNCOMPENSABLE EXERCISE-HEAT STRESS

Factor	Effect		Study
	Yes	No	
Aerobic Fitness		X	Sawka MN, Young AJ, Latzka WA, Neuffer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. <i>J Appl Physiol.</i> 1992;73:368–375.
Climate		X	Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. <i>J Appl Physiol.</i> 1994;77:216–222.
Clothing Encapsulation	↓ 0.2°C		Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. <i>J Appl Physiol.</i> 1994;77:216–222.
Exercise Intensity		X	Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. <i>J Appl Physiol.</i> 1994;77:216–222.
Gender	—	—	—
Intermittent Exercise	—	—	—
Heat Acclimation		X	(1) 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. <i>J Physiol.</i> 1993;460:467–485. (2) Nielsen B, Strange S, Christensen NJ, Warberg J, Saltin B. Acute and adaptive responses in humans to exercise in a warm humid environment. <i>Pflügers Arch.</i> 1997;434:49–56.
Hyperhydration		X	Latzka WA, Sawka MN, Montain SJ, et al. Hyperhydration: Tolerance and cardiovascular effects during uncompensable exercise-heat stress. <i>J Appl Physiol.</i> 1998;84:1858–1864.
Hypohydration	↓ 0.5°C		Sawka MN, Young AJ, Latzka WA, Neuffer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. <i>J Appl Physiol.</i> 1992;73:368–375.

Encapsulation: wearing closed mission-oriented protective posture (MOPP) 4 gear

No: Effect not found; Yes: Effect found

## STRATEGIES TO SUSTAIN PHYSICAL EXERCISE CAPABILITIES

Strategies to manage heat stress and sustain physical exercise capabilities can include improving and maintaining the soldiers' heat dissipation capabilities and managing the amount of heat stress exposure. Heat dissipation capabilities can be improved by heat acclimation and physical training, whereas rehydration allows maintenance of these capabilities. Heat stress can be managed by reducing climatic exposure (seeking shade and wind), wearing less clothing or equipment, or both, and

reducing the physical exercise output. Microclimate cooling can artificially improve heat loss capabilities. Most of these heat stress management approaches either reduce the amount of work accomplished or increase the manpower requirements to complete a given task.

**Heat Acclimation and Physical Fitness**

Heat acclimation and aerobic training both im-

**EXHIBIT 3-1**

**ACTIONS OF HEAT ACCLIMATIZATION**

**Thermal Comfort: Improved**

- Core Temperature: Reduced
- Sweating: Improved
  - Earlier onset
  - Higher rate
  - Redistribution (tropic)
  - Hidromeiosis resistance (tropic)
- Skin Blood Flow: Increased
  - Earlier onset
  - Higher flow

**Exercise Performance: Improved**

- Metabolic Rate: Lowered
- Cardiovascular Stability: Improved
  - Heart rate: lowered
  - Stroke volume: increased
  - Blood pressure: better defended
- Fluid Balance: Improved
  - Thirst: improved
  - Electrolyte loss: reduced
  - Total body water: increased
  - Plasma volume: increased and better defended

Reprinted with permission from Montain SJ, Maughan RJ, Sawka MN. Heat acclimatization strategies for the 1996 Summer Olympics. *Athletic Ther Today*. 1996;1:43.

vironments). Exhibit 3-1 provides a brief description of the actions of heat acclimation.<sup>185</sup> The benefits of heat acclimation are achieved by improved sweating and skin blood flow responses, better maintenance of fluid balance and cardiovascular stability, and a lowered metabolic rate.<sup>25,184,185</sup>

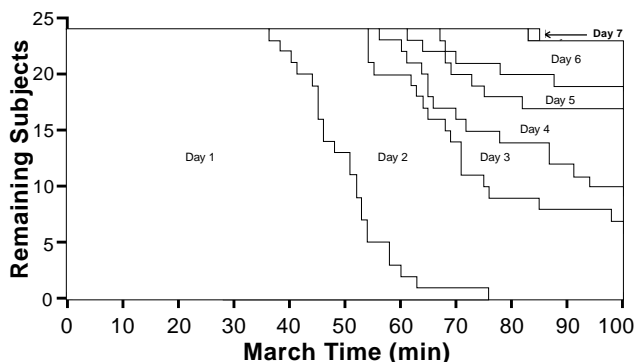
Heat acclimation can improve performance quite dramatically so that acclimated subjects can easily complete exercise in the heat, which earlier was difficult or impossible to achieve. Figure 3-22 depicts the improvement in exercise-heat tolerance time for 24 men who attempted 100 minutes of treadmill exercise at 49°C, 20% rh, for 7 days. This figure shows that no one completed the 100-minute walk on day 1; however, 40% were successful by day 3, 80% by day 5, and all but one of these men were successful by the seventh acclimation day.<sup>25</sup>

During acclimation through daily exercise in a hot climate, most of the improvement in heart rate, skin and core temperatures, and sweat rate is achieved during the first week of exposure, although there is no sharp end to the improvement.<sup>25,184</sup> Heart rate shows the most rapid reduction, most of which occurs in 4 to 5 days.<sup>25,184</sup> The thermoregulatory acclimation response,<sup>25,184</sup> improved sweating response, and greater ease of walking develop more slowly.<sup>25,184</sup> Heat acclimation is transient and gradually disappears if not maintained by repeated heat exposure, but there is considerable variability in the literature concerning its rate of decay. High levels of physical fitness<sup>186</sup>

pose heat stress on the body that induces an elevated core temperature and increased sweating rate. Repeated exposure to this heat stress will result in an improved ability to thermoregulate.

**Heat Acclimation**

Heat acclimation results in biological adaptations that reduce the negative effects of heat stress. The magnitude of the biological adaptations depends largely on the intensity, duration, frequency, and number of heat exposures. Thus daily sessions of exercise in the heat, so as to elevate both core and skin temperatures and provoke profuse sweating, are the most effective method for developing heat acclimation. However, even resting in the heat results in some acclimation, although to a lesser degree.<sup>182-185</sup> During the initial heat exposure, there are high levels of physiological strain, as manifested by elevated core temperature and heart rate; but the strain decreases progressively with each day of acclimation (see Figure 2-14 in Chapter 2, Human Adaptation to Hot En-



**Fig. 3-22.** Day-to-day improvements in exercise-heat tolerance of 24 men participating in a dry-heat acclimation program, expressed as the number able to complete a 100-minute exercise-heat exposure each day; dry bulb temperature 49°C, wet bulb temperature 27°C (relative humidity 20%). Adapted with permission from Pandolf KB, Young AJ. Environmental extremes and performance. In: Shephard RJ, Åstrand PO, eds. *Endurance in Sport*. Oxford, United Kingdom: Blackwell; 1992: 271.

and perhaps warm weather<sup>117</sup> also favor retention of acclimation, but there is conflicting evidence on the effect of the weather.<sup>187</sup> Finally, the literature suggests that dry-heat acclimation is retained longer than humid-heat acclimation.<sup>120,182,183,186,188–192</sup>

### Physical Fitness

An improvement in  $\dot{V}O_2\text{max}$  achieved through endurance training in a temperate climate reduces physiological strain and increases physical exercise capabilities in the heat,<sup>25,115,168</sup> and endurance-trained individuals exercising in the heat exhibit many of the characteristics of heat-acclimated individuals. In addition, high  $\dot{V}O_2\text{max}$  facilitates acquisition of heat acclimation,<sup>186</sup> and may reduce susceptibility to heat injury or heat illness.<sup>193</sup>

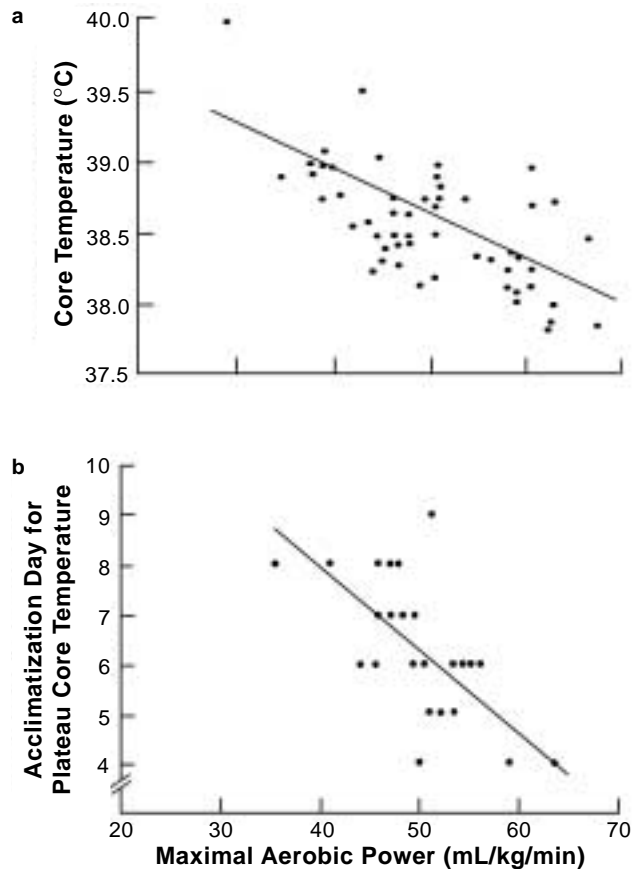
From data collected in both hot-humid and hot-dry environments, it has been estimated that a person's  $\dot{V}O_2\text{max}$  accounts for approximately 44% of the variability in core temperature after 3 hours of exercise in the heat, or the number of days of heat acclimation required to achieve a steady state in final core temperature, as illustrated in Figure 3-23.<sup>194</sup> However, endurance training alone does not entirely replace the benefits of heat acclimation produced by a program of exercise in the heat.<sup>43,195,196</sup>

High  $\dot{V}O_2\text{max}$  is not invariably associated with improved heat tolerance. It is likely that for endurance training to improve thermoregulatory responses during exercise in the heat, the training sessions must elicit substantial elevations of core temperature and sweating rate.<sup>197,198</sup> Investigators<sup>198</sup> who compared thermoregulatory responses of six skiers ( $\dot{V}O_2\text{max} = 66.5 \text{ mL/kg/min}$ ) with those of four swimmers ( $\dot{V}O_2\text{max} = 65.8 \text{ mL/kg/min}$ ) found that skiers were more heat-tolerant and better acclimated than swimmers, and attributed the difference to a smaller increase in the swimmers' core temperatures produced by training in cold water. In support of this interpretation, other investigators<sup>197</sup> found that 4 weeks of training by cycle exercise in 20°C water increased  $\dot{V}O_2\text{max}$  by about 15% but did not improve thermoregulation during exercise-heat stress.

To achieve optimal thermoregulatory results from endurance training in temperate climates, either strenuous interval training or continuous training at an intensity greater than 50%  $\dot{V}O_2\text{max}$  should be employed.<sup>25,185,194</sup> Lesser training intensities produce questionable effects on performance during exercise-heat stress.<sup>199</sup> The endurance training must last at least 1 week,<sup>200,201</sup> and some authors<sup>194</sup> show that the best improvements require 8 to 12 weeks of training.

### Rehydration

Major battles have been decided by water availability to the opposing forces, but Adolph and associates<sup>53</sup> were the first to study in a systematic way the importance of fluid replacement on the ability to perform military tasks in the heat. The importance of hydration during military operations was demonstrated more recently: dehydration-induced heatstroke was believed responsible for 20,000 deaths among Egyptian troops during the 1967 Arab-Israeli Six-Day War.<sup>56</sup> Physical exercise and

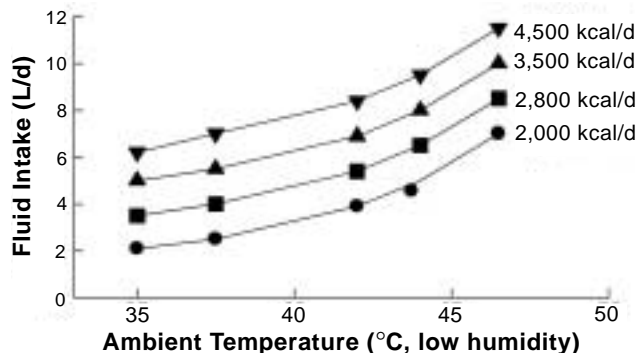


**Fig. 3-23.** Relationship between (a) maximal aerobic power and core (rectal) temperature in a hot-humid environment ( $n = 26$ ) and (b) maximal aerobic power and plateau core (rectal) temperature during dry-heat exposures was first achieved ( $n = 24$ ,  $r = -0.68$ ). The equation describing the relation between the variables in (a) is  $40.15 - 0.03 \dot{V}O_2\text{max}$ ; the correlation coefficient is  $-0.65$ . The equation describing the relation between the variables in (b) is  $14.49 - 0.165 \dot{V}O_2\text{max}$ ; the correlation coefficient is  $-0.68$ . Adapted with permission from Pandolf KB. Effects of physical training and cardiorespiratory physical fitness on exercise-heat tolerance: Recent observations. *Med Sci Sports*. 1979;11:60–65.

heat stress will cause both fluid and electrolyte imbalances that need to be corrected.<sup>146,202–204</sup> The goal of drinking is to replace fluid (and sometimes electrolytes also) lost by sweating. The requirements for fluid replacement depend on losses through sweating, which, in turn, are determined by environmental conditions, physical activity level, and the clothing and equipment worn, as well as by an individual's own characteristics.

Military operations in hot climates may require high sweating rates over many days.<sup>202,203</sup> For example, soldiers might have sweating rates of 0.3 to 1.2 L/h while performing routine military activities,<sup>53,205–207</sup> and those wearing protective clothing might have sweating rates of 1 to 2 L/h while performing light-intensity exercise.<sup>6,208,209</sup> These high sweating rates will increase daily fluid needs. Figure 3-24 presents the influence of climate and physical activity level on daily fluid requirements.<sup>210,211</sup> Daily fluid requirements range (for sedentary to very active soldiers) from 2 to 4 L/d in temperate climates and from 4 to 12 L/d in hot climates.

Electrolytes—primarily sodium chloride and to a lesser extent potassium, calcium, and magnesium—are contained in sweat. Sweat sodium concentration averages approximately 35 mEq/L (range < 10–70 mEq/L) and varies depending on diet, sweating rate, hydration, and heat acclimation level.<sup>212,213</sup> Sweat glands reabsorb sodium by active transport, and because the ability to reabsorb sweat sodium does not increase in proportion to the sweating rate, the concentration of sodium increases at high sweating rates.<sup>25</sup> Heat acclimation improves the ability to reabsorb sodium, so acclimated persons have lower sweat sodium concentrations (> 50% reduction) for any sweating rate.<sup>213</sup> Average concentrations of other electrolytes in sweat are [K<sup>+</sup>], 5 mEq/L; [Ca<sup>2+</sup>], 1 mEq/L; and [Mg<sup>2+</sup>], 0.8 mEq/L,<sup>212</sup> and neither gender nor age appears to have a marked effect on these concentrations.<sup>214,215</sup> Sweat also contain vitamins, but in very low concentrations.<sup>216</sup> Except for sodium, losses of solute in sweat are so low that supplementation to replace the losses is unnecessary. However, if sweating rates are high, sodium losses are also high, and providing some extra salt with meals or in beverages may be beneficial to unacclimatized troops. If troops are eating normally, supplemental salt is not necessary,<sup>217</sup> except for their first several days of heat exposure,<sup>202,204</sup> as normal dietary sodium intake will cover the sweat losses<sup>202–204</sup> once troops become acclimatized. Salt pills have been overused in the past and may be harmful in the presence of a water deficit. Therefore, troops should not take salt pills un-

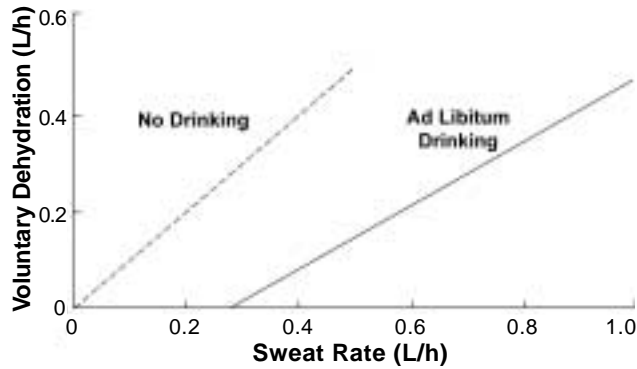


**Fig. 3-24.** Daily fluid intake plotted as a function of ambient temperature and metabolic rate. On the curves, top to bottom: 4,500 kcal/d represents heavy work; 3,500 kcal/d, moderate work; 2,800 kcal/d, light work; and 2,000 kcal/d, resting. (1) Data source: Nelson N, Eichna LW, Bean WB. *Determination of Water and Salt Requirements for Desert Operations*. Fort Knox, Ky: Armored Force Medical Research Laboratory; 1943. Report 2-6. (2) Adapted with permission from Greenleaf JE. Environmental issues that influence intake of replacement beverages. In: Marriott BM, ed. *Fluid Replacement and Heat Stress*. Washington, DC: National Academy Press; 1994: 205.

less advised to do so by a physician.

The problem of fluid replacement is to match fluid intakes to evaporative and renal losses in order to avoid dehydration. Thirst is probably not perceived until a water deficit of 2% of body weight is incurred,<sup>53,218,219</sup> and thirst provides a poor index of body water needs.<sup>53,218,220</sup> Therefore, ad libitum water intake during exercise in the heat results in incomplete replacement of body water losses,<sup>53,218</sup> (Figure 3-25) and physically active troops will typically dehydrate by 2% to as much as 8% of body weight (dehydration is measured by change in body weight) in situations of heat stress and prolonged high sweat loss in the field. Note that at a sweat rate of 1.0 L/h, the soldiers are underconsuming fluids by about 0.5 L.<sup>53</sup> As a result, some dehydration is likely to occur during exercise in the heat, unless troops make a conscious effort to drink, even when not thirsty. Flavoring and cooling fluids will increase palatability and help minimize voluntary dehydration.<sup>218,221–223</sup> On the other hand, drinking carbonated beverages may give a false feeling of stomach fullness and reduce fluid consumption.<sup>224</sup> People exercising in the heat usually fully rehydrate only at mealtime, when fluid intake is stimulated by consuming food,<sup>53,203</sup> which increases thirst and fluid intake above what would occur if only fluids were consumed. Meals should be scheduled at specific times and provide enough time for troops to





**Fig. 3-25.** Relationship between sweating rate and rate of dehydration (water deficit, based on change in body weight) without drinking (broken line) and during ad libitum drinking (solid line). Voluntary dehydration is dehydration that occurs during ad libitum drinking. Data are for heat-acclimated soldiers walking in the desert. Adapted from Adolph EF, Associates: *Physiology of Man in the Desert*. New York, NY: Intersciences, Inc; 1947: 259.

drink to satiation. If they do not eat regular meals, their existing water deficits will persist, and performance will suffer. Therefore, the importance of meals for promoting full rehydration cannot be overstressed.

Troops should be fully hydrated when they begin exercise or heat exposure. A good habit is to drink 400 to 600 mL approximately 1 hour before beginning activity to offset any preexisting water deficit.<sup>146,204</sup> If this volume does not induce urination prior to exercise, it is likely that the person is already dehydrated. Preexisting dehydration can be a problem during early morning activities because water loss occurs during sleep. Adequate fluids should be available during physical activity, and troops should be encouraged to drink whenever they get the opportunity. The rate at which fluids can be replaced by mouth is limited by the rate of gastric emptying, and the rate of fluid absorption by the small intestine. Gastric emptying rate is believed to be the most limiting factor, and, for most persons, maximal emptying rates approximate 1 to 1.2 L/h.<sup>146,225,226</sup> However, gastric emptying and fluid absorption rates vary among individuals, so each person must determine his or her own best drinking pattern. If fluid losses during exercise and heat stress exceed the gastric emptying rate, the remaining deficits should be replaced later. Because gastric emptying rate is greatest when the stomach is full,<sup>225</sup> it can be facilitated by drinking enough to keep the stomach full.<sup>226</sup> To maximize fluid replacement during exercise, it is recommended that troops drink a large volume (500–1,000 mL) initially to fill

the stomach, followed by regular drinking of smaller volumes (200–400 mL) to keep the stomach as full as is comfortable. (The actual volumes will vary depending on expected sweating rates, body size, tolerance, and other individual factors.)

Dehydration reduces the gastric emptying rate of ingested fluids during exercise in the heat.<sup>227,228</sup> In one study, for example, gastric emptying rate was reduced approximately 20% to 25% when subjects were hypohydrated by 5% of body weight,<sup>227</sup> and the reduction was related to increased core temperature. Therefore, it is important to begin fluid intake during the early stages of exercise–heat stress, not only to minimize dehydration but also to maximize absorption of the ingested fluids.

### Exercise/Rest Cycles

In some conditions of high exercise–heat stress (especially uncompensable heat stress), the ability to tolerate the exercise–heat stress is determined largely by biophysical limits on heat dissipation, which are determined by the environment and the clothing and equipment that are worn. In such conditions (eg, wearing protective clothing), physiological adaptations obtained from heat acclimation or aerobic training can do little to improve exercise performance.<sup>229–231</sup> Even if little can be done to reduce the effects of clothing and climate, the metabolic heat load can often be managed. The options are to exercise at a given metabolic rate until exhaustion from heat strain occurs, or to decrease the metabolic rate and therefore dissipate the heat load.<sup>232,233</sup> Metabolic rate can be decreased by reducing the exercise intensity or using rest periods (exercise/rest cycles) or both to lower the time-averaged metabolic rate. Decreasing the metabolic rate will extend tolerance time, although it may result in less total work being accomplished in a given period of time.

Exercise/rest cycles have been used in both military<sup>13</sup> and civilian<sup>12,234</sup> occupational settings to extend exercise tolerance time in high heat stress conditions. (During military operations, however, this approach is not always acceptable because it increases the time or personnel requirements to complete a task, and so may jeopardize the mission.) Figure 3-26 presents the relationship between exercise tolerance time and the average metabolic rate for troops working in the heat (30°C, 50% rh) while wearing chemical protective clothing either open (solid curve) or closed (dotted curve).<sup>235</sup> When 1:1 exercise/rest cycles are employed to reduce the average metabolic rate, the tolerance times are increased in a hyperbolic manner. Therefore, tolerance

times are closely related to the average metabolic rate when wearing protective clothing in climatic heat stress conditions.

A critical problem for managing heat stress during military operations is determining the effectiveness of exercise/rest cycles and calculating the optimal ratios.<sup>41</sup> The US Army Research Institute of Environmental Medicine (USARIEM), Natick, Massachusetts, has developed a family of mathematical models<sup>207,235-239</sup> that enable predictions that are customized to the climatic conditions, clothing and equipment worn, and the soldier's state of hydration and acclimatization. Tables 3-4 and 3-5 present examples of exercise/rest cycles for a matrix of conditions in desert<sup>13</sup> and warm, humid<sup>240</sup> climates. Calculations are for four exercise intensity levels (very light = 150 W, light = 250 W, moderate = 425 W, high = 600 W), six levels of WBGT, and two clothing configurations. The calculation represents the recommended number of minutes per hour for exercise, with the remaining time spent resting. For example, "34" means that the exercise/rest ratio is 34 minutes of exercise to 26 minutes of rest. These tables provide a margin of safety because the model used to generate these tables is somewhat conservative.

### Microclimate Cooling

Because protective clothing reduces both evaporative and dry heat exchange, it impairs a person's ability to dissipate body heat and can result in ex-

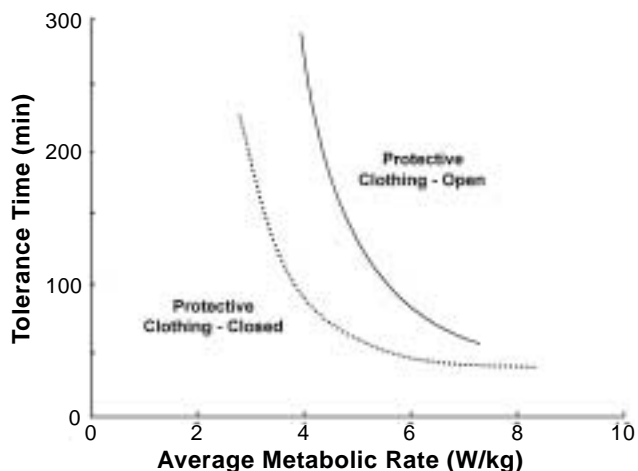


Fig. 3-26. Relationship between exercise tolerance time and the average metabolic rate while subjects wear protective clothing in the heat (35°C, 50% rh). Adapted with permission from Mclellan TM, Jacobs I, Bain JB. Continuous vs intermittent work with Canadian Forces NBC clothing. *Aviat Space Environ Med.* 1993;64:596.

treme elevations in body temperatures during exercise in moderate climatic conditions.<sup>4,6,173,209,241-243</sup> Because the clothing has high insulation and low permeability to water vapor, heat and moisture from the body are retained within the clothing, where they create a warm, humid microenvironment that seriously impairs heat loss from the body. To alleviate heat stress under these conditions, microclimate cooling systems have been developed<sup>208,244-246</sup> that cool the microenvironment immediately surrounding the person, rather than the macroenvironment (ie, the working area).<sup>247</sup>

Microclimate cooling systems use circulating cooled air, liquid in tubes next to the skin, or ice-pack vest to remove body heat.<sup>208,245</sup> In addition, microclimate cooling facilitates heat loss by maintaining the temperature gradient between the body core and the cooled skin. The amount of heat transferred from the body to any microclimate system depends on several factors, including the size and location of skin regions being cooled, coolant temperature and flow rate, and insulation from the ambient heat.<sup>87,248-250</sup>

Air-cooled garments are lighter to wear and rely on sweat evaporation to cool a person. Although air is not as effective as water in removing heat by convection due to the difference in specific heat, air-cooled systems promote evaporation of sweat and are effective in reducing heat strain; and in some environments they are believed to be as effective as water-cooled devices.<sup>245</sup> In addition, air-cooled vests keep the skin drier than do liquid-cooled systems, thereby improving thermal comfort. If the environment is not contaminated by biological or chemical agents, untreated ambient air can be circulated through an air-cooled system. However, warm, humid air will reduce cooling due to evaporation of sweat. On the other hand, air that is too hot and dry may cause local skin irritation.<sup>246</sup>

Microclimate cooling systems that utilize ice as the cooling medium are not as effective as either liquid- or air-cooled systems.<sup>208,245</sup> Furthermore, the cooling lasts only until the ice has completely melted, so the ice must periodically be replenished. The logistical problems associated with ice-cooled systems make them impractical for use as cooling devices in all but very short-duration situations.

Microclimate cooling systems are effective in alleviating heat stress and extending exercise capabilities in soldiers who are wearing protective clothing,<sup>246,251-256</sup> as illustrated in Figures 3-27 and 3-28. These curves, which are based on a modeling analysis, illustrate the effectiveness of microclimate cooling to extend physical exercise to 5 hours in hot,

TABLE 3-4

## RECOMMENDED EXERCISE/REST CYCLES (MINUTES OF EXERCISE PER HOUR) IN HOT, DRY CLIMATES

WBGT	T <sub>a</sub> (°F)	rh (%)	DBDU				DBDU and Flak Vest			
			VL	L	M	H	VL	L	M	H
82°F (28°C)	87	20	NL	NL	33	21	NL	NL	36	23
86°F (30°C)	91	20	NL	NL	30	20	NL	NL	34	22
88°F (31°C)	94	20	NL	NL	28	18	NL	NL	31	20
90°F (32°C)	96	20	NL	NL	26	17	NL	NL	30	19
98°F (37°C)	120	20	NL	NF	NF	NF	NL	9	NF	NF
115°F (46°C)	120	60	NF	NF	NF	NF	NL	NF	NF	NF

DBDU: desert battle dress uniform; H: heavy work intensity; L: light work intensity; M: moderate work intensity; NF: not feasible (exercise/rest cycle not feasible); NL: no limit (continuous exercise possible); rh: relative humidity; T<sub>a</sub>: ambient temperature (°F); VL: very light work intensity; WBGT: wet bulb globe temperature

NOTE: This table provides, for four levels of work intensity, the number of minutes of exercise per hour in exercise/rest schedules tailored to the conditions specified. Spend the remainder of the hour at rest. This model was prepared using the USARIEM Heat Strain Model. Assumptions used in this model include: (1) troops fully hydrated, rested, and acclimated; (2) windspeed = 2 m/s; (3) clear skies (full solar load); and (4) heat casualties < 5%. This guidance should not be used as a substitute for common sense or experience. Individual requirements vary greatly. Appearance of heat casualties is evidence that the selected exercise/rest cycle is inappropriate for the conditions.

Adapted from Sawka MN, Modrow HE, Kolka MA, et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. Technical Note 95-1.

humid (35°C T<sub>a</sub>, 70% rh) and desert (49°C T<sub>a</sub>, 20% rh) climates, respectively.<sup>245</sup> The cooling extraction rates on the abscissa represent heat actually removed from the body. These values may be only 30% to 80% of the heat taken up by the cooling sys-

tems because of heat gained by the systems from the environment. In each environment, curves are calculated for soldiers wearing chemical protective clothing while exercising at five different metabolic rates, from 250 to 600 W, which represent the range

TABLE 3-5

## RECOMMENDED EXERCISE/REST CYCLES (MINUTES OF EXERCISE PER HOUR) IN HOT, WET CLIMATES

WBGT	T <sub>a</sub> (°F)	rh (%)	DBDU				DBDU and Flak Vest			
			VL	L	M	H	VL	L	M	H
82°F (28°C)	82.9	75	NL	NL	NL	25	NL	NL	NL	29
86°F (30°C)	87.1	75	NL	NL	33	21	NL	NL	NL	27
88°F (31°C)	89.2	75	NL	NL	29	18	NL	NL	37	23
90°F (32°C)	91.3	75	NL	NL	25	16	NL	NL	32	20
98°F (36°C)	99.7	75	NL	NF	NF	NF	NL	16	6	NF
110°F (43°C)	109.0	50	NL	NF	NF	NF	NL	23	10	5

DBDU: desert battle dress uniform; H: heavy work intensity; L: light work intensity; M: moderate work intensity; NF: not feasible (exercise/rest cycle not feasible); NL: no limit (continuous exercise possible); rh: relative humidity; T<sub>a</sub>: ambient temperature (°F); VL: very light work intensity; WBGT: wet bulb globe temperature

Adapted from Burr RE, Modrow HE, King N, et al. *Sustaining Health and Performance in Haiti: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. USARIEM Technical Note 94-4.

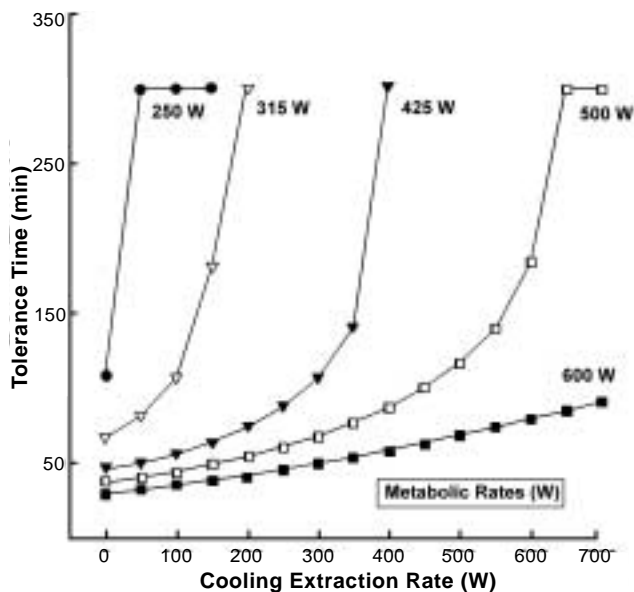


Fig. 3-27. The relationship between microclimate cooling and endurance times at selected metabolic rates (W) when subjects are wearing chemical protective clothing in a hot, humid climate (35°C, 70% rh). Reprinted from Pandolf KB, Gonzalez JA, Sawka MN. *An Updated Review: Microclimate Cooling of Protective Overgarments in the Heat*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1995: 69. USARIEM Technical Report T95-7.

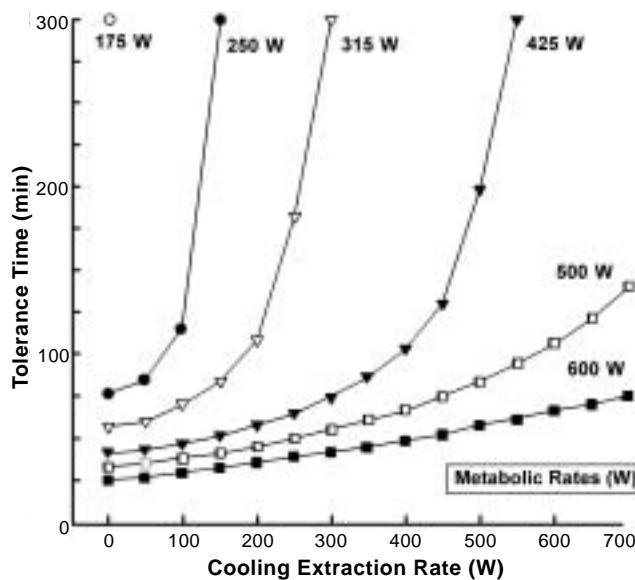


Fig. 3-28. The relationship between microclimate cooling and endurance times at selected metabolic rates (W) when subjects are wearing chemical protective clothing in a desert climate (49°C, 20% rh). The value 175 W is not a control; it demonstrates that at this lower rate, 300 min of tolerance time is possible. Reprinted from Pandolf KB, Gonzalez JA, Sawka MN. *An Updated Review: Microclimate Cooling of Protective Overgarments in the Heat*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1995: 69. USARIEM Technical Report T95-7.

of tasks performed by soldiers for extended periods during military operations.<sup>41</sup> These curves indicate that if heat extraction rates of 300 to 400 W are achieved, then microclimate cooling can substantially improve tolerance time (although microclimate

cooling alone is not sufficient to alleviate the problem). Note that at higher metabolic rates, microclimate cooling will need to be combined with exercise/rest cycles (which lower the metabolic rate) to markedly extend performance.<sup>251</sup>

### BIOMEDICAL ISSUES

The biomedical status of a soldier is an important modifier of environmental and mission-related factors to influence the physical work capacity and tolerance to heat stress. Biomedical factors include the soldier's gender, race, age, biological rhythms, health, and medications. An increased reliance on reserve forces, who are older and may use medications for health reasons, pose deployment considerations that were not present a decade ago.

#### Gender and Race

Thermoregulatory responses to heat exposure at rest appear to be similar for both genders.<sup>257</sup> Although it was once believed that women are less heat tolerant than men, it now seems that the studies on which

that belief was based compared relatively unfit women to more fit men.<sup>258,259</sup> Recent research demonstrated that if populations of men and women are matched for aerobic fitness or compared at the same relative exercise intensity, they have similar tolerance and body temperature responses to exercise in both dry and humid heat,<sup>260-263</sup> and respond similarly to heat acclimation<sup>260,261</sup> and dehydration.<sup>67</sup> Core temperature changes with the phase of the menstrual cycle in a cyclic fashion,<sup>258,259</sup> reaching its lowest point just before ovulation, and then rising 0.4 to 0.6 Centigrade degrees over the next few days to a plateau that persists throughout the luteal phase. This pattern results from a similar variation in the thermoregulatory set point, and thus is accompanied by corresponding changes in the thresholds for all the thermoregula-

tory responses.<sup>264</sup> There is some indication, as discussed in the chapter by Kolka and Stephenson, Women's Health in Deployment, in the third volume of *Medical Aspects of Harsh Environments*, of changes in heat tolerance with the phase of the menstrual cycle.

There has long been a perception that descendants of ethnic groups who are indigenous to hot climates are more heat tolerant than are descendants of ethnic groups who are indigenous to cool climates. There is some support in the literature for this perception, mostly based on comparison of white Americans, Britons, or South Africans with persons of tropical African descent. The reported differences apply mostly to hot, humid conditions,<sup>265-269</sup> with the advantage in humid heat being enjoyed by tropical Africans and their descendants. (This topic is explored more fully elsewhere.<sup>184</sup>) These results are consistent with the notion that populations gradually acquire certain biological adaptations in the course of living many generations in a particular environment. In interpreting these studies, however, it should be remembered that the subject populations were not matched for factors such as aerobic fitness and body composition. In fact, Robinson<sup>270</sup> said that such differences in responses (including those that he had reported 11 years earlier<sup>266</sup>) were

probably due largely to differences in nutritional status, acclimatization, and training and not dependent on racial characteristics or length of residence in the tropics.<sup>270(p73)</sup>

## Age

For the general population, physiological heat strain in middle-aged men and women during exercise is greater than in younger adults, particularly in the heat,<sup>198,271-276</sup> as manifested by higher core and mean skin temperatures, heart rate, and skin blood flow, as well as reductions in sweating. These differences between young and middle-aged adults appear to be greater during continuous exercise in the heat than during intermittent exercise or heat exposure at rest.<sup>275,276</sup> In addition, the differences in heat strain between young and middle-aged individuals were accentuated during prolonged exposures<sup>273,277</sup> and at higher levels of environmental heat stress, particularly those exceeding the prescriptive zone.<sup>274,276</sup>

It is not clear from the foregoing reports how much of this lesser tolerance was the result of age per se, and how much could be explained by other factors such as poorer health and decreased physical activity or low aerobic fitness. However, if middle-aged and younger men and women are matched for aerobic fitness or habitual physical activity levels, and for cer-

tain anthropometric factors (such as body surface area, surface area-to-weight ratio, and percentage of body fat), the heat-strain differences between age groups tends to disappear.<sup>278-282</sup> Two of these reports<sup>280,281</sup> emphasized the importance of maintaining physical fitness and body weight in preserving exercise-heat tolerance as one ages.

Middle-aged men and women can acclimate to exercise in the heat; but whether they acclimate as rapidly as younger persons, or achieve the same degree of acclimation, is not yet established.<sup>283</sup> Some studies<sup>280,281</sup> report that during heat acclimation, the thermoregulatory responses of physically fit middle-aged men improved as rapidly and to the same degree as those of younger men. Other studies,<sup>275,278</sup> however, report that although middle-aged men and women acclimate to exercise-heat stress, the degree of heat acclimation achieved was less than that for younger persons.

## Circadian Patterns and Sleep Loss

Core temperature at rest varies with time of day in a sinusoidal fashion, with the minimum at night, and the maximum, which is 0.5°C to 1°C higher, occurring in the late afternoon or evening. This pattern is an example of a *circadian* rhythm (ie, a rhythmic pattern in a physiological function with a period of about 1 day). The circadian rhythm in core temperature results from a similar circadian rhythm in the thermoregulatory set point, and is accompanied by corresponding changes in the thresholds for all the thermoregulatory responses that have been studied.<sup>258,284</sup> Sleep deprivation disrupts the circadian rhythm, delaying the time of minimum core temperature at rest, and altering thermoregulatory responses during exercise.<sup>285,286</sup> In addition, sleep deprivation is reported<sup>287,288</sup> to impair heat tolerance and negate the effects of heat acclimation, but it is not known whether the effects on heat tolerance are mediated through the thermoregulatory changes or through other mechanisms.

## Skin Disorders

Certain skin disorders impair the ability to dissipate body heat, and thus increase thermal strain.<sup>25</sup> Heat rash and sunburn may have substantial thermoregulatory effects, and all too often their adverse effects on exercise-heat tolerance fail to get the attention they deserve.

Artificially induced miliaria rubra (heat rash) over as little as 20% of the body surface causes an observable reduction in exercise-heat tolerance<sup>289</sup>;

and involvement of 40% or more of the body surface markedly reduces tolerance time during exercise in the heat (49°C, 20% rh) and causes greater heat storage compared with responses in a nonrash state.<sup>289,290</sup> These effects may persist for up to 3 weeks after the rash has resolved clinically.<sup>290</sup> The degree of heat intolerance that occurs depends both on the total area of skin affected and on the specific region of the body, and that region's normal sweating responses. Thus, smaller rashed areas of the trunk may, because of the greater sweating capacity of normal trunk skin, affect responses to dry-heat stress as much as larger rashed areas of the limbs. Observable sweating was absent in the rashed areas, perhaps because of physical occlusion of the affected sweat glands by keratotic plugs.<sup>290</sup>

Mild artificial sunburn impairs sweat gland activity during exercise in the heat.<sup>291</sup> Both the local sweating sensitivity and steady state sweating rate from the burned areas are reduced 24 hours after the sunburn compared with values before the sunburn, but they return to normal within 1 week. Mild sunburn thus appears to have a locally mediated effect on both the responsiveness of the sweat gland and its capacity to deliver sweat to the skin surface. These effects perhaps result from damage to the cuboidal epithelial cells composing the sweat duct, which blocks the egress of sweat from the affected duct. More severe levels of sunburn may have more profound thermoregulatory consequences during exercise-heat stress.

## Medications

For protection against organophosphate (anticholinesterase) nerve agents, military personnel are issued nerve agent antidote kits containing atropine sulfate autoinjectors, and nerve agent pretreatment kits containing pyridostigmine bromide pills. Atropine binds with muscarinic receptors for acetylcholine, where atropine acts as a competitive an-

tagonist to acetylcholine. Atropine thus reduces sweating and impairs tolerance to exercise-heat stress.<sup>292-298</sup> Pyridostigmine bromide, a reversible anticholinesterase, increases sweating and inhibits cutaneous vasodilation during exercise-heat stress.<sup>299,300</sup> At the standard pretreatment dosage (30 mg, 3 times daily), pyridostigmine administration has little effect on tolerance to exercise-heat stress.<sup>301</sup> Several other drugs that have some anticholinergic action, plus a number of others,<sup>57,302,303</sup> impair tolerance to exercise-heat stress and have been associated with heatstroke.

Many senior enlisted personnel, officers, and reservists take medications (including diuretics,  $\beta$ -adrenergic blockers, and angiotensin-converting enzyme inhibitors) for treatment of hypertension and arrhythmias. These drugs have cardiovascular effects via modifying heart rate, blood pressure, and vascular resistance. During exercise-heat stress,  $\beta$ -adrenergic blockers reduce active cutaneous vasodilation<sup>304-306</sup> and sweating<sup>306,307</sup> responses, and these actions combine to increase core temperature.<sup>304,306,307</sup> Nonselective  $\beta$ -adrenergic blockers, such as propranolol, have greater adverse thermoregulatory effects than selective blockers, such as metoprolol<sup>306</sup> and atenolol.<sup>305,308</sup> Angiotensin-converting enzyme inhibitors, such as enalapril, do not impair thermoregulatory responses to acute exercise-heat stress<sup>309</sup>; however, this drug might reduce thirst.<sup>310</sup> During short-term administration, diuretics reduce extracellular fluid volume, plasma volume, and tolerance to exercise-heat stress, as discussed earlier. With longer-term administration, plasma and extracellular fluid volumes return toward normal, but some authors report persistent reductions in plasma or blood volume,<sup>311-313</sup> suggesting the possibility of long-term impairment in tolerance to exercise-heat stress. Little is known about the effects of chronic administration of antihypertensive drugs on the ability to acclimatize to heat.

## SUMMARY

Troops participating in all military deployments may encounter heat stress. The magnitude of heat stress encountered is dependent on complex interactions among climatic conditions, exercise intensity, health and nutrition status, and clothing and equipment worn. Body temperature is regulated by two parallel processes: behavioral regulation and physiological regulation. In combat, behavioral thermoregulatory drives are overridden by motivation to successfully complete the mission. Physiological thermoregulation relies on increased skin blood flow and

sweating to dissipate body heat to the climate. The hotter the climate the greater the dependence on sweating and evaporative heat loss. Therefore, in hot climates, sweating rates will be high and drinking must be emphasized to avoid dehydration. Heat stress decreases the capacity for both maximal and submaximal intensity exercise, and dehydration magnifies these decrements in performance capacity. Strategies to manage heat stress and sustain exercise capacity include improving the troops' ability to dissipate body heat (by heat acclimation and physical

training programs), managing the amount of heat stress (reducing climatic exposure, wearing less clothing, reducing physical exercise, providing microclimate cooling), and maintaining hydration and health (avoiding certain medications, sunburn, and sleep loss). Mathematical models such as the USARIEM Heat Strain Model<sup>41</sup> can provide specific guidance to manage heat stress during military operations.

## REFERENCES

1. US Army Training and Doctrine Command. *Operations*. Washington, DC: DA; 1993. Field Manual 100-5.
2. Freund BJ, Sawka MN. Influence of cold stress on human fluid balance. In: Marriott BM, Carlson SJ, eds. *Nutritional Needs in Cold and in High-Altitude Environments*. Washington, DC: National Academy Press; 1996: 161–179.
3. Lee DHK. Terrestrial animals in dry heat: Man in the desert. In: Dill DB, Adolph EF, Wilber CG, eds. *Handbook of Physiology, Section 4: Adaptation to the Environment*. Washington, DC: American Physiological Society; 1964: 551–582.
4. Kraning KK II, Gonzalez RR. Physiological consequences of intermittent exercise during compensable and uncompensable heat stress. *J Appl Physiol*. 1991;71:2138–2145.
5. Sawka MN, Young AJ, Latzka WA, Neuffer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: Influence of hydration. *J Appl Physiol*. 1992;73:368–375.
6. Montain SJ, Sawka MN, Cadarette BS, Quigley MD, McKay JM. Physiological tolerance to uncompensable heat stress: Effects of exercise intensity, protective clothing, and climate. *J Appl Physiol*. 1994;77:216–222.
7. Kolka MA, Stephenson LA, Gonzalez RR. Thermoregulation in women during uncompensable heat stress. *J Therm Biol*. 1994;19:325–320.
8. Research Study Group 7 on Biomedical Research Aspects of Military Protective Clothing. *Handbook on Clothing: Biomedical Effects of Military Clothing and Equipment Systems*. Brussels, Belgium: North Atlantic Treaty Organization; 1988.
9. US Army Chemical School. *NBC Protection*. Washington, DC: DA; 1992. Field Manual 3-4.
10. Cornum K. Deployment operations in the heat: A Desert Shield experience. In: AGARD Advisory Group for Aerospace Research and Development, eds. *The Support of Air Operations Under Extreme Hot and Cold Weather Conditions*. Brussels, Belgium: North Atlantic Treaty Organization; 1993: 27-1–27-5.
11. Gonzalez RR. Biophysics of heat exchange and clothing: Applications to sports physiology. *Med Exerc Nutr Health*. 1995;4:290–305.
12. National Institute of Occupational Safety and Health. *Occupational Exposure to Hot Environments*. Washington, DC: US Department of Health and Human Services; 1986. DHHS (NIOSH) 86-113.
13. Sawka MN, Modrow HE, Kolka MA, et al. *Sustaining Soldier Health and Performance in Southwest Asia: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. USARIEM Technical Note 95-1.
14. 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.
15. Sawka MN, Roach JM, Young AJ, et al. *Sustaining Soldier Health and Performance During Operation Support Hope: Guidance for Small Unit Leaders*. Fort Detrick, Frederick, Md: US Army Medical Research, Development, Acquisition and Logistic Command; 1994. Technical Note 94-3.

16. Onkaram B, Stroschein LA, Goldman RF. *A Comparison of Four Instruments for Measuring WBGT Index Correlations of Botsball With WBGT*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1978. Technical Report T4-78.
17. Yaglou CP, Minard D. Control of heat casualties at military training centers. *A M A Arch Industr Health*. 1957;16:302–316.
18. John B. Stennis Space Center. *Global Climatology for the Wet Bulb Globe Temperature (WBGT) Heat Stress Index*. Bay St. Louis, Miss: Stennis Space Center, Gulf Weather; 1989.
19. Ladell WSS. Terrestrial animals in humid heat: Man. In: Dill DB, Adolph EF, Wilber CG, eds. *Handbook of Physiology, Section 4: Adaptation to the Environment*. Washington, DC: American Physiological Society; 1964: 625–659.
20. Levine L, Sawka MN, Gonzalez RR. *General Procedures for Clothing Evaluations Relative to Heat Stress*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1995. Technical Note 95-5.
21. Brengelmann GL. Dilemma of body temperature measurement. In: Shiraki K, Yousef MK, eds. *Man in a Stressful Environment; Thermal and Work Physiology*. Springfield, Ill: Charles C Thomas; 1987: 5–22.
22. Cooper KE, Kenyon JR. A comparison of temperature measured in the rectum, oesophagus and on the surface of the aorta during hypothermia in man. *Br J Surg*. 1957;44:616–619.
23. Piironen P. Sinusoidal signals in the analysis of heat transfer in the body. In: Hardy JD, Gagge AP, Stolwijk JAJ, eds. *Physiological and Behavioral Temperature Regulation*. Springfield, Ill: Charles C Thomas; 1970: 358–366.
24. Shiraki K, Konda N, Sagawa S. Esophageal and tympanic temperature responses to core blood temperature changes during hyperthermia. *J Appl Physiol*. 1986;61:98–102.
25. Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise–heat stress and heat acclimation. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology, Section 4: Environmental Physiology*. New York, NY: Oxford University Press; 1996: 157–185.
26. Fox RH, Goldsmith R, Wolff HS. The use of a radio pill to measure deep body temperature. *J Physiol (Lond)*. 1961;160:22–23.
27. Kolka MA, Quigley MD, Blanchard LA, Toyota DA, Stephenson LA. Validation of a temperature telemetry system during moderate and strenuous exercise. *J Therm Biol*. 1993;18:203–210.
28. Sparling PB, Snow TK, Millard-Stafford ML. Monitoring core temperature during exercise: Ingestible sensor vs. rectal thermistor. *Aviat Space Environ Med*. 1993;64:760–763.
29. 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*. Carmel, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 97–151.
30. Hardy JD, DuBois EF. The technique of measuring radiation and convection. *J Nutr*. 1938;15:461–475.
31. Winslow C-EA, Herrington LP, Gagge AP. A new method of partitioned calorimetry. *Am J Physiol*. 1936;116:641–655.
32. Ramanathan NL. A new weighting system for mean surface temperature of the human body. *J Appl Physiol*. 1964;19:531–533.
33. Teichner WH. Assessment of mean body surface temperature. *J Appl Physiol*. 1958;12:169–176.
34. Mitchell D, Wyndham CH. Comparison of weighting formulas for calculating mean skin temperature. *J Appl Physiol*. 1969;26:616–622.



35. Lind AR. A physiological criterion for setting thermal environmental limits for everyday work. *J Appl Physiol.* 1963;18:51–56.
36. Nielsen B, Nielsen M. Body temperature during work at different environmental temperatures. *Acta Physiol Scand.* 1962;56:120–129.
37. Nielsen M. Heat production and body temperature during rest and work. In: Hardy JD, Gagge AP, Stolwijk JAJ, eds. *Physiological and Behavioral Temperature Regulation.* Springfield, Ill: Charles C Thomas; 1970: 205–214.
38. Nielsen M. Die Regulation der Körpertemperatur bei Muskelarbeit. *Scand Arch Physiol.* 1938;9:193–230.
39. Gagge AP, Gonzalez RR. Mechanisms of heat exchange: Biophysics and physiology. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology, Section 4: Environmental Physiology.* New York, NY: Oxford University Press; 1996: 45–84.
40. Eichna LW, Ashe WF, Bean WB, Shelley WB. The upper limits of environmental heat and humidity tolerated by acclimatized men working in hot environments. *J Ind Hyg Toxicol.* 1945;27:59–84.
41. 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.
42. Saltin B, Hermansen L. Esophageal, rectal, and muscle temperature during exercise. *J Appl Physiol.* 1966;21:1757–1762.
43. Åstrand I. Aerobic work capacity in men and women. *Acta Physiol Scand.* 1960;49:64–73.
44. Davies CTM. Influence of skin temperature on sweating and aerobic performance during severe work. *J Appl Physiol.* 1979;47:770–777.
45. 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.
46. Greenleaf JE, Greenleaf CJ, Card DH, Saltin B. Exercise-temperature regulation in man during acute exposure to simulated altitude. *J Appl Physiol.* 1969;26:290–296.
47. Kolka MA, Stephenson LA, Rock PB, Gonzalez RR. Local sweating and cutaneous blood flow during exercise in hypobaric environments. *J Appl Physiol.* 1987;62:2224–2229.
48. Nielsen B. Thermoregulation during work in carbon monoxide poisoning. *Acta Physiol Scand Suppl.* 1971;82:98–106.
49. Rowell LB, Freund PR, Brengelmann GL. Cutaneous vascular response to exercise and acute hypoxia. *J Appl Physiol.* 1982;53:920–924.
50. Sawka MN, Dennis RC, Gonzalez RR, et al. Influence of polycythemia on blood volume and thermoregulation during exercise–heat stress. *J Appl Physiol.* 1987;62:912–918.
51. Sawka MN, Gonzalez RR, Young AJ, et al. Polycythemia and hydration: Effects on thermoregulation and blood volume during exercise–heat stress. *Am J Physiol.* 1988;255:R456–R463.
52. Patterson MJ, Cotter JD, Taylor NA. Thermal tolerance following artificially induced polycythemia. *Eur J Appl Physiol.* 1995;71:416–423.
53. Adolph EF, Associates. *Physiology of Man in the Desert.* New York, NY: Intersciences, Inc; 1947.
54. Draper ES, Lombardi JJ. *Combined Arms in a Nuclear/Chemical Environment: Force Development, Testing and Experimentation, Summary Evaluation Report, Phase I.* Fort McClellan, Ala: US Army Chemical School; 1986.
55. Greenleaf JE. Problem: Thirst, drinking behavior, and involuntary dehydration. *Med Sci Sports Exerc.* 1992;24:645–656.

56. Hubbard RW. Water as a tactical weapon: A doctrine for preventing heat casualties. *Army Sci Conf Proc.* 1982;2:125–139.
57. Hales JRS, Hubbard RW, Gaffin SL. Limitations of heat tolerance. In: Fregly MJ, Blatteis CM, eds. *Handbook of Physiology, Section 4: Environmental Physiology.* New York, NY: Oxford University Press; 1996: 285–355.
58. Chung NK. Obesity and the occurrence of heat disorders. *Mil Med.* 1996;161:739–742.
59. Cadarette BS, Sawka MN, Toner MM, Pandolf KB. Aerobic fitness and the hypohydration response to exercise-heat stress. *Aviat Space Environ Med.* 1984;55:507–512.
60. Grande F, Monagle JE, Buskirk ER, Taylor HL. Body temperature responses to exercise in man on restricted food and water intake. *J Appl Physiol.* 1959;14:194–198.
61. Sawka MN, Young AJ, Francesconi RP, Muza SR, Pandolf KB. Thermoregulatory and blood responses during exercise at graded hypohydration levels. *J Appl Physiol.* 1985;59:1394–1401.
62. Ekblom B, Greenleaf CJ, Greenleaf JE, Hermansen L. Temperature regulation during exercise dehydration in man. *Acta Physiol Scand.* 1970;79:475–483.
63. Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol.* 1992;73:1340–1350.
64. Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise-heat exposure. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport.* Boca Raton, Fla: CRC Press; 1996: 143–161.
65. Strydom NB, Holdsworth DL. The effects of different levels of water deficit on physiological responses during heat stress. *Int Z Angew Physiol.* 1968;26:95–102.
66. Buskirk ER, Iampietro PF, Bass DE. Work performance after dehydration: Effects of physical conditioning and heat acclimatization. *J Appl Physiol.* 1958;12:189–194.
67. Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: Effects of heat acclimation, gender, and environment. *J Appl Physiol.* 1983;55:1147–1153.
68. Moroff SV, Bass DE. Effects of overhydration on man's physiological responses to work in the heat. *J Appl Physiol.* 1965;20:267–270.
69. Nielsen B, Hansen G, Jorgensen SO, Nielsen E. Thermoregulation in exercising man during dehydration and hyperhydration with water and saline. *Int J Biometeorol.* 1971;15:195–200.
70. Nielsen B. Effects of changes in plasma volume and osmolarity on thermoregulation during exercise. *Acta Physiol Scand.* 1974;90:725–730.
71. Gisolfi CV, Copping JR. Thermal effects of prolonged treadmill exercise in the heat. *Med Sci Sports.* 1974;6:108–113.
72. Grucza R, Szczypaczewska M, Kozłowski S. Thermoregulation in hyperhydrated men during physical exercise. *Eur J Appl Physiol.* 1987;56:603–607.
73. Greenleaf JE, Castle BL. Exercise temperature regulation in man during hypohydration and hyperhydration. *J Appl Physiol.* 1971;30:847–853.
74. Candas V, Libert JP, Brandenberger G, Sagot JC, Kahn JM. Thermal and circulatory responses during prolonged exercise at different levels of hydration. *J Physiol (Paris).* 1988;83:11–18.
75. Nadel ER, Fortney SM, Wenger CB. Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol.* 1980;49:715–721.

76. Lyons TP, Riedesel ML, Meuli LE, Chick TW. Effects of glycerol-induced hyperhydration prior to exercise in the heat on sweating and core temperature. *Med Sci Sports Exerc.* 1990;22:477–483.
77. Latzka WA, Sawka MN, Matott RP, Staab JE, Montain SJ, Pandolf KB. *Hyperhydration: Physiologic and Thermoregulatory Effects During Compensable and Uncompensable Exercise–Heat Stress.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1996. USARIEM Technical Report TR96-6.
78. Latzka WA, Sawka MN, Montain S, et al. Hyperhydration: Thermoregulatory effects during compensable exercise–heat stress. *J Appl Physiol.* 1997;83:860–866.
79. Sawka MN. Physiology of upper body exercise. *Excerc Sport Sci Rev.* 1986;14:175–211.
80. Sawka MN, Latzka WA, Pandolf KB. Temperature regulation during upper body exercise: Able-bodied and spinal cord injured. *Med Sci Sports Exerc.* 1989;21:S132–S140.
81. Asmussen E, Nielsen M. The regulation of the body-temperature during work performed with the arms and with the legs. *Acta Physiol Scand.* 1947;14:73–382.
82. Nielsen B. Thermoregulatory responses to arm work, leg work and intermittent leg work. *Acta Physiol Scand.* 1968;72:25–32.
83. Davies CTM, Barnes C, Sargeant AJ. Body temperature in exercise: Effects of acclimatization to heat and habituation to work. *Int Z Angew Physiol.* 1971;30:10–19.
84. Sawka MN, Pimental NA, Pandolf KB. Thermoregulatory responses to upper body exercise. *Eur J Appl Physiol.* 1984;52:230–234.
85. Sawka MN, Gonzalez RR, Drolet LL, Pandolf KB. Heat exchange during upper- and lower-body exercise. *J Appl Physiol.* 1984;57:1050–1054.
86. Pivarnik JM, Grafner TR, Elkins ES. Metabolic, thermoregulatory, and psychophysiological responses during arm and leg exercise. *Med Sci Sports Exerc.* 1988;20:1–5.
87. Young AJ, Sawka MN, Epstein Y, Decristofano B, Pandolf KB. Cooling different body surfaces during upper and lower body exercise. *J Appl Physiol.* 1987;63:1218–1223.
88. Sawka MN, Wenger CB, Young AJ, Pandolf KB. Physiological responses to exercise in heat. In: Mariott BM, ed. *Nutritional Needs in Hot Environments.* Washington, DC: National Academy Press; 1993: 55–74.
89. Consolazio CF, Matoush LO, Nelson RA, Torres JA, Isaac GJ. Environmental temperature and energy expenditures. *J Appl Physiol.* 1963;18:65–68.
90. Consolazio CF, Shapiro R, Masterson JE, McKinzie PSL. Energy requirements of men in extreme heat. *J Nutr.* 1961;73:126–134.
91. Dimri GP, Malhotra MS, Gupta JS, Kumar TS, Aora BS. Alterations in aerobic–anaerobic proportions of metabolism during work in heat. *Eur J Appl Physiol.* 1980;45:43–50.
92. Fink WJ, Costill DL, Handel WJV. Leg muscle metabolism during exercise in the heat and cold. *Eur J Appl Physiol.* 1975;34:183–190.
93. Brouha L, Smith PE Jr, De Lanne R, Maxfield ME. Physiological reactions of men and women during muscular activity and recovery in various environments. *J Appl Physiol.* 1960;16:133–140.
94. Petersen ES, Vejby-Christensen H. Effect of body temperature on steady state ventilation and metabolism in exercise. *Acta Physiol Scand.* 1973;89:342–351.

95. Cerretelli P, Marconi C, Pendergast DR, Meyer M, Heisler N, Piiper J. Blood flow in exercising muscles by xenon clearance and by microsphere trapping. *J Appl Physiol.* 1984;56:24–30.
96. Young AJ, Sawka MN, Levine L, Cadarette BS, Pandolf KB. Skeletal muscle metabolism during exercise is influenced by heat acclimation. *J Appl Physiol.* 1985;59:1929.
97. Oyono-Enguelle S, Heitz A, Ott JMC, Pape A, Freund H. Heat stress does not modify lactate exchange and removal abilities during recovery from short exercise. *J Appl Physiol.* 1993;74:1248–1255.
98. Rowell LB, Brengelmann GL, Blackmon JB, Twiss RD, Kusumi F. Splanchnic blood flow and metabolism in heat-stressed man. *J Appl Physiol.* 1968;24:475–484.
99. Febbraio MA, Snow RJ, Hargreaves M, Stathis CG, Martin IK, Carey MF. Muscle metabolism during exercise and heat stress in trained men: Effect of acclimation. *J Appl Physiol.* 1994;76:589–597.
100. Febbraio MA, Snow RJ, Stathis CG, Hargreaves M, Carey MF. Effect of heat stress on muscle energy metabolism during exercise. *J Appl Physiol.* 1994;77:2827–2831.
101. Yaspelkis BB, Scroop GC, Wilmore KM, Ivy JL. Carbohydrate metabolism during exercise in hot and thermoneutral environments. *Int J Sports Med.* 1993;14:13–19.
102. Savard GK, Nielsen B, Laszczynska J, Larsen BE, Saltin B. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J Appl Physiol.* 1988;64:649–657.
103. Nielsen B, Savard G, Richter EA, Hargreaves M, Saltin B. Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol.* 1990;69:1040–1046.
104. Rowell LB. *Human Circulation: Regulation During Physical Stress.* New York, NY: Oxford University Press; 1986.
105. Barcroft H, Bock KD, Hensel H, Kitchin AH. Die Muskeldurchblutung des Menschen beim indirekter Erwärmung und Abkühlung. *Pflügers Arch.* 1955;261:199–210.
106. Detry J-MR, Brengelmann GL, Rowell LB, Wyss C. Skin and muscle components of forearm blood flow in directly heated resting man. *J Appl Physiol.* 1972;32:506–511.
107. Dolny DG, Lemon PWR. Effect of ambient temperature on protein breakdown during prolonged exercise. *J Appl Physiol.* 1988;64:550–555.
108. Hargreaves M, Dillo P, Angus D, Febbraio M. Effect of fluid ingestion on muscle metabolism during prolonged exercise. *J Appl Physiol.* 1996;80:363–366.
109. Sawka MN, Pandolf KB, Avellini BA, Shapiro Y. Does heat acclimation lower the rate of metabolism elicited by muscular exercise? *Aviat Space Environ Med.* 1983;4:27–31.
110. Sawka MN, Petrofsky JS, Phillips CA. Energy cost of submaximal isometric contractions in cat fast and slow twitch muscles. *Pflügers Arch.* 1981;390:164–168.
111. Wendt IR, Gibbs CL. Energy production of rat extensor digitorum longus muscle. *Am J Physiol.* 1973;224:1081–1086.
112. Young AJ, Sawka MN, Levine L, et al. Metabolic and thermal adaptations from endurance training in hot or cold water. *J Appl Physiol.* 1995;78:793–801.
113. Young AJ. Energy substrate utilization during exercise in extreme environments. *Exerc Sport Sci Rev.* 1990;18:65–117.
114. Senay LC, Kok R. Effects of training and heat acclimatization on blood plasma contents of exercising men. *J Appl Physiol.* 1977;43:591–599.

115. 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.
116. Strydom NB, Williams CG. Effect of physical conditioning on state of heat acclimatization of Bantu laborers. *J Appl Physiol.* 1969;27:262–265.
117. Eichna LW, Park CR, Nelson N, Horvath SM, Palmes ED. Thermal regulation during acclimatization in a hot, dry (desert type) environment. *Am J Physiol.* 1950;163:585–597.
118. Gisolfi CV. Work-heat tolerance derived from interval training. *J Appl Physiol.* 1973;35:349–354.
119. Jooste PL, Strydom NB. Improved mechanical efficiency derived from heat acclimatization. *S Afr J Res Sports Phys Ed Recreation.* 1979;2:45–53.
120. Robinson S, Turrell ES, Belding HS, Horvath SM. Rapid acclimatization to work in hot climates. *Am J Physiol.* 1943;140:168–176.
121. King DS, Costill DL, Fink WJ, Hargreaves M, Fielding RA. Muscle metabolism during exercise in the heat in unacclimatized and acclimatized humans. *J Appl Physiol.* 1985;59:1350–1354.
122. Kirwan JP, Costill DL, Kuipers H, et al. Substrate utilization in leg muscle of men after heat acclimation. *J Appl Physiol.* 1987;63:31–35.
123. 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.* Carmel, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 227–266.
124. Hertzman AB, Randall WC, Peiss CN, Seckendorf R. Regional rates of evaporation from the skin at various environmental temperatures. *Jpn J Physiol.* 1952;5:153–161.
125. Randall WC. Quantification and regional distribution of sweat glands in man. *J Clin Invest.* 1946;25:761–767.
126. Sato K, Dobson RL. Regional and individual variations in the function of the human eccrine sweat gland. *J Invest Dermatol.* 1970;54:443–449.
127. Candas V, Liber P, Vogt JJ. Sweating and sweat decline of resting men in hot humid environments. *Eur J Appl Physiol.* 1983;50:223–234.
128. Gonzalez RR, Pandolf KB, Gagge AP. Heat acclimation and decline in sweating during humidity transients. *J Appl Physiol.* 1974;36:419–425.
129. Nadel ER, Stolwijk JAJ. Effect of skin wettedness on sweat gland response. *J Appl Physiol.* 1973;35:689–694.
130. Desruelle AV, Bothorel B, Hoefft A, Candas V. Effects of local restriction of evaporation and moderate local ventilation on thermoregulatory responses in exercising humans. *Eur J Appl Physiol.* 1996;73:231–236.
131. Johnson JM, Brengelmann GL, Hales JRS, Vanhoutte PM, Wenger CB. Regulation of the cutaneous circulation. *Fed Proc.* 1986;45:2841–2850.
132. Johnson JM, Proppe DW. Cardiovascular adjustments to heat stress. In: Fregley MJ, Blatteis CM, eds. *Handbook of Physiology, Section 4: Environmental Physiology.* New York, NY: Oxford University Press; 1996: 215–243.
133. Rowell LB. Cardiovascular adjustments to hyperthermia and exercise. In: Shiraki K, Yousef MK, eds. *Man in Stressful Environments: Thermal and Work Physiology.* Springfield, Ill: Charles C Thomas; 1987: 99–113.
134. Rowell LB. Cardiovascular aspects of human thermoregulation. *Circ Res.* 1983;52:367–379.

135. Rowell LB, Marx HJ, Bruce RA, Conn RD, Kusumi F. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest.* 1966;45:1801–1816.
136. Nadel ER, Cafarelli E, Roberts MF, Wenger CB. Circulatory regulation during exercise in different ambient temperatures. *J Appl Physiol.* 1979;46:430–437.
137. Koroxenidis GT, Shepherd JT, Marshall RJ. Cardiovascular response to acute heat stress. *J Appl Physiol.* 1961;16:869–872.
138. Allen TE, Smith DP, Miller DK. Hemodynamic response to submaximal exercise after dehydration and rehydration in high school wrestlers. *Med Sci Sports.* 1977;9:159–163.
139. Neuffer PD, Sawka MN, Young A, Quigley M, Latzka WA, Levine L. Hypohydration does not impair skeletal muscle glycogen resynthesis after exercise. *J Appl Physiol.* 1991;70:1490–1494.
140. Maw GJ, Mackenzie IL, Comer DAM, Taylor NAS. Whole-body hyperhydration in endurance-trained males determined using radionuclide dilution. *Med Sci Sports Exerc.* 1996;28:1038–1044.
141. Senay LC. Relationship of evaporative rates to serum  $[Na^+]$ ,  $[K^+]$ , and osmolality in acute heat stress. *J Appl Physiol.* 1968;25:149–152.
142. Kubica R, Nielsen B, Bonnesen A, Rasmussen IB, Stoklosa J, Wilk B. Relationship between plasma volume reduction and plasma electrolyte changes after prolonged bicycle exercise, passive heating and diuretic dehydration. *Acta Physiol Pol.* 1983;34:569–579.
143. Shearer S. Dehydration and serum electrolyte changes in South African gold miners with heat disorders. *Am J Ind Med.* 1997;17:225–239.
144. Sawka MN. Physiological consequences of hydration: Exercise performance and thermoregulation. *Med Sci Sports Exerc.* 1992;24:657–670.
145. Sawka MN, Pandolf KB. Effects of body water loss on physiological function and exercise performance. In: Gisolfi CV, Lamb DR, eds. *Perspectives in Exercise Science and Sports Medicine, Vol 3: Fluid Homeostasis During Exercise.* Carmel, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1990: 1–38.
146. Montain SJ, Maughan RJ, Sawka MN. Fluid replacement strategies for exercise in hot weather. *Athletic Ther Today.* 1996;1:24–27.
147. Montain SJ, Latzka WA, Sawka MN. Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *J Appl Physiol.* 1995;79:1434–1439.
148. Sawka MN, Gonzalez RR, Young AJ, Dennis RC, Valeri CR, Pandolf KB. Control of thermoregulatory sweating during exercise in the heat. *Am J Physiol.* 1989;257:R311–R316.
149. Kenney WL, Tankersley CG, Newswanger DL, Hyde DE, Puhl SM, Turnera NL. Age and hypohydration independently influence the peripheral vascular response to heat stress. *J Appl Physiol.* 1990;8:1902–1908.
150. Sawka MN, Francesconi RP, Young AJ, Pandolf KB. Influence of hydration level and body fluids on exercise performance in the heat. *JAMA.* 1984;252:1165–1169.
151. Senay LC. Temperature regulation and hypohydration: A singular view. *J Appl Physiol.* 1979;47:1–7.
152. Fortney SM, Wenger CB, Bove JR, Nadel ER. Effect of hyperosmolality on control of blood flow and sweating. *J Appl Physiol.* 1984;57:1688–1695.
153. Fortney SM, Nadel ER, Wenger CB, Bove JR. Effect of blood volume on sweating rate and body fluids in exercising humans. *J Appl Physiol.* 1981;51:1594–1600.

154. Mack G, Nose H, Nadel ER. Role of cardiopulmonary baroreflexes during dynamic exercise. *J Appl Physiol.* 1988;65:1827–1832.
155. Mack G, Nishiyasu T, Shi X. Baroreceptor modulation of cutaneous vasodilator and sudomotor responses to thermal stress in humans. *J Physiol (Lond).* 1995;483:537–547.
156. Klausen K, Dill DB, Phillips EE, McGregor D. Metabolic reactions to work in the desert. *J Appl Physiol.* 1967;22:292–296.
157. Rowell LB, Brengelmann GL, Murray JA, Kraning KK, Kusumi F. Human metabolic responses to hyperthermia during mild to maximal exercise. *J Appl Physiol.* 1969;26:395–402.
158. Saltin B, Gagge AP, Bergh U, Stolwijk JAJ. Body temperatures and sweating during exhaustive exercise. *J Appl Physiol.* 1972;32:635–643.
159. Sen Gupta J, Dimri P, Malhotra MS. Metabolic responses of Indians during sub-maximal and maximal work in dry and humid heat. *Ergonomics.* 1977;20:33–40.
160. Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol.* 1985;53:294–298.
161. Rowell LB, Blackmon JR, Martin RH, Mazzarella JA, Bruce RA. Hepatic clearance of indocyanine green in man under thermal and exercise stresses. *J Appl Physiol.* 1965;20:384–394.
162. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-, diuretic-, and exercise-induced hypohydration. *J Appl Physiol.* 1984;57:1018–1023.
163. Webster S, Rutt R, Weltman A. Physiological effects of a weight loss regimen practiced by college wrestlers. *Med Sci Sports Exerc.* 1990;22:229–234.
164. Craig FN, Cummings EG. Dehydration and muscular work. *J Appl Physiol.* 1966;21:670–674.
165. Bruck K, Olschewski H. Body temperature related factors diminishing the drive to exercise. *Can J Physiol Pharmacol.* 1987;65:1274–1280.
166. Galloway SDR, Maughan RJ. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med Sci Sports Exerc.* 1997;29:1240–1249.
167. Ladell WSS, Shephard RJ. Aldosterone inhibition and acclimatization to heat. *Proc Phys Soc.* 1962;160:19P–20P.
168. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc.* 1985;17:456–461.
169. Burge CM, Carey MF, Payne WR. Rowing performance, fluid balance, and metabolic function following dehydration and rehydration. *Med Sci Sports Exerc.* 1993;25:1358–1364.
170. Walsh RM, Noakes TD, Hawley JA, Dennis SC. Impaired high-intensity cycling performance time at low levels of dehydration. *Int J Sports Med.* 1994;15:392–398.
171. Below PR, Mora-Rodríguez R, González-Alonso J, Coyle EF. Fluid and carbohydrate ingestion independently improve performance during 1h of exercise. *Med Sci Sports Exerc.* 1995;27:200–210.
172. Reneau PD, Bishop PA. Validation of a personal heat stress monitor. *Am Ind Hyg Assoc J.* 1996;57:650–657.
173. Joy RJT, Goldman RF. A method of relating physiology and military performance: A study of some effects of vapor barrier clothing in a hot climate. *Mil Med.* 1968;133:458–470.

174. 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.
175. Kraning KK. Analysis of data from 696 subject observations during experiments conducted at US Army Research Institute of Environmental Medicine. Natick, Mass: USARIEM; 1997. Unpublished.
176. Adams WC, Fox RH, Fry AJ, MacDonald IC. Thermoregulation during marathon running in cool, moderate, and hot environments. *J Appl Physiol.* 1975;38:1030–1037.
177. Pugh LGCE, Corbett JL, Johnson RH. Rectal temperatures, weight losses, and sweat rates in marathon running. *J Appl Physiol.* 1967;23:347–352.
178. Robinson S. Temperature regulation in exercise. *Pediatrics.* 1963;32:691–702.
179. Sawka MN, Knowlton RG, Critz JB. Thermal and circulatory responses to repeated bouts of prolonged running. *Med Sci Sports.* 1979;11:177–180.
180. Gilat T, Shibolet S, Sohar E. The mechanism of heatstroke. *J Trop Med Hyg.* 1963;66:204–212.
181. Shibolet S, Coll R, Gilat T, Sohar E. Heatstroke: Its clinical picture and mechanism in 36 cases. *Q J Med.* 1967;36:525–548.
182. Bean WB, Eichna LW. Performance in relation to environmental temperature: Reactions of normal young men to simulated desert environment. *Fed Proc.* 1943;2:144–158.
183. Eichna LW, Bean WB, Ashe WF, Nelson N. Performance in relation to environmental temperature: Reactions of normal young men to hot, humid (simulated jungle) environment. *Bull Johns Hopkins Hosp.* 1945;76:25–58.
184. Wenger CB. Human heat acclimatization. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes.* Carmel, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 153–197.
185. Montain SJ, Maughan RJ, Sawka MN. Heat acclimatization strategies for the 1996 Summer Olympics. *Athletic Ther Today.* 1996;1:42–46.
186. Pandolf KB, Burse RL, Goldman RF. Role of physical fitness in heat acclimatization, decay and reinduction. *Ergonomics.* 1977;20:399–408.
187. Williams GG, Wyndham CH, Morrison JF. Rate of loss of acclimatization in summer and winter. *J Appl Physiol.* 1967;22:21–26.
188. Adam JM, Fox RH, Grimby G, Kidd DJ, Wolff HS. Acclimatization to heat and its rate of decay in man. *J Physiol (Lond).* 1960;152:26P–27P.
189. Henschel A, Taylor HL, Keys A. The persistence of heat acclimatization in man. *Am J Physiol.* 1943;140:321–325.
190. Lind AR, Bass DE. Optimal exposure time for development of acclimatization to heat. *Fed Proc.* 1963;22:704–708.
191. Stein HJ, Eliot JW, Bader RA. Physiological reactions to cold and their effects on the retention of acclimatization to heat. *J Appl Physiol.* 1949;1:575–585.
192. Wyndham CH, Jacobs GE. Loss of acclimatization after six days of work in cool conditions on the surface of a mine. *J Appl Physiol.* 1957;11:197–198.
193. 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.



194. 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*. Carmel, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 199–226.
195. Pandolf KB. Effects of physical training and cardiorespiratory physical fitness on exercise–heat tolerance: Recent observations. *Med Sci Sports*. 1979;11:60–65.
196. 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.
197. Avellini BA, Shapiro Y, Fortney SM, Wenger CB, Pandolf KB. Effects on heat tolerance of physical training in water and on land. *J Appl Physiol*. 1982;53:1291–1298.
198. Henane R, Flandrois R, Charbonnier JP. Increase in sweating sensitivity by endurance conditioning in man. *J Appl Physiol*. 1977;43:822–828.
199. Shvartz E, Saar E, Meyerstein N, Benor D. A comparison of three methods of acclimatization to dry heat. *J Appl Physiol*. 1973;34:214–219.
200. Nadel ER, Pandolf KB, Roberts MF, Stolwijk JAJ. Mechanisms of thermal acclimation to exercise and heat. *J Appl Physiol*. 1974;37:515–520.
201. Roberts MF, Wenger CB, Stolwijk JAJ, Nadel ER. Skin blood flow and sweating changes following exercise training and heat acclimation. *J Appl Physiol*. 1977;43:133–137.
202. Marriott BM. *Fluid Replacement and Heat Stress*. Washington, DC: National Academy Press; 1994.
203. Marriott BM. *Nutritional Needs in Hot Environments. Application for Military Personnel in Field Operations*. Washington, DC: National Academy Press; 1993.
204. Convertino VA, Armstrong LE, Coyle EF, et al. American College of Sports Medicine Position Stand: Exercise and fluid replacement. *Med Sci Sports Exerc*. 1996;28:i–vii.
205. Molnar GW, Towbin EJ, Gosselin RE, Brown AH, Adolph EF. A comparative study of water, salt and heat exchanges of men in tropical and desert environments. *Am J Hyg*. 1946;44:411–433.
206. Shapiro Y, Pandolf KB, Goldman RF. Predicting sweat loss response to exercise, environment and clothing. *Eur J Appl Physiol*. 1982;48:83–96.
207. Shapiro Y, Moran D, Epstein Y. Validation and adjustment of the mathematical prediction model for human sweat rate responses to outdoor environmental conditions. *Ergonomics*. 1995;38:981–986.
208. Speckman KL, Allan AE, Sawka MN, Young AJ, Muza SR, Pandolf KB. Perspectives in microclimate cooling involving protective clothing in hot environments. *Int J Ind Ergonomics*. 1988;3:121–147.
209. Levine LL, Quigley MD, Cadarette BS, Sawka MN, Pandolf KB. Physiologic strain associated with wearing toxic-environment protective systems during exercise in the heat. In: Biman Das, ed. *Advances in Industrial Ergonomics and Safety*. London, England: Taylor & Francis; 1990: 897–904.
210. Nelson N, Eichna LW, Bean WB. *Determination of Water and Salt Requirements for Desert Operations*. Fort Knox, Ky: Armored Force Medical Research Laboratory; 1943. Report 2-6.
211. Greenleaf JE. Environmental issues that influence intake of replacement beverages. In Marriott BM, ed. *Fluid Replacement and Heat Stress*. Washington, DC: National Academy Press; 1994: 195–214.
212. Brouns F. Heat-sweat-dehydration-rehydration: A praxis oriented approach. *J Sports Sci*. 1991;9:143–152.

213. Allan JR, Wilson CG. Influence of acclimatization on sweat sodium concentration. *J Appl Physiol*. 1971;30:708–712.
214. Meyer F, Bar-Or O, MacDougal D, Heigenhauser GJF. Sweat electrolyte loss during exercise in the heat: Effects of gender and maturation. *Med Sci Sports Exerc*. 1992;24:776–781.
215. Morimoto T, Slabochova Z, Naman RK, Sargent F. Sex differences in physiological reactions to thermal stress. *J Appl Physiol*. 1967;22:526–532.
216. Clarkson PM. The effects of exercise and heat on vitamin requirements. In: Marriott BM, ed. *Nutritional Needs in Hot Environments*. Washington, DC: National Academy Press; 1993: 137–171.
217. Armstrong LE, Hubbard RW, Askew EW, et al. Responses to moderate and low sodium diets during exercise–heat acclimation. *Int J Sport Nutr*. 1993;3:207–221.
218. Hubbard RW, Sandick BL, Matthew WT, et al. Voluntary dehydration and alliesthesia for water. *J Appl Physiol*. 1984;57:868–875.
219. Armstrong LE, Hubbard RW, Szlyk PC, Matthew WT, Sils IV. Voluntary dehydration and electrolyte losses during prolonged exercise in the heat. *Aviat Space Environ Med*. 1985;56:765–770.
220. Engell DB, Maller O, Sawka MN, Francesconi RP, Drolet LA, Young AJ. Thirst and fluid intake following graded hypohydration levels in humans. *Physiol Behav*. 1987;40:229–236.
221. Szlyk PC, Sils IV, Francesconi RP, Hubbard RW, Armstrong LE. Effects of water temperature and flavoring on voluntary dehydration in man. *Physiol Behav*. 1989;45:639–647.
222. Szlyk PC, Sils IV, Francesconi RP, Hubbard RW. Patterns of human drinking: Effects of exercise, water temperature, and food consumption. *Aviat Space Environ Med*. 1990;61:43–48.
223. Szlyk PC, Sils IV, Francesconi RP, Hubbard RW, Matthew WT. Variability in intake and dehydration in young men during a simulated desert walk. *Aviat Space Environ Med*. 1989;60:422–427.
224. Hickey MS, Costill DL, Trappe SW. Drinking behavior and exercise-thermal stress: Role of drink carbonation. *Int J Sport Nutr*. 1994;4:8–21.
225. Costill DL. Gastric emptying of fluids during exercise. In: Gisolfi CV, Lamb DR, eds. *Fluid Homeostasis During Exercise*. Carmel, Ind: Benchmark Press (now Traverse City, Mich: Cooper Publishing Group); 1990: 97–125.
226. Duchman SM, Ryan AJ, Schedl HP, Summers RW, Bleiler TL, Gisolfi CV. Upper limit for intestinal absorption of a dilute glucose solution in men at rest. *Med Sci Sports Exerc*. 1997;29:482–488.
227. Neuffer PD, Young AJ, Sawka MN. Gastric emptying during exercise: Effects of heat stress and hypohydration. *Eur J Appl Physiol*. 1989;58:433–439.
228. Owen MD, Kregel KC, Wall PT, Gisolfi CV. Effects of ingesting carbohydrate beverages during exercise in the heat. *Med Sci Sports Exerc*. 1986;18:568–575.
229. Aoyagi Y, Mcllellan TM, Shephard RJ. Effects of training and acclimation on heat tolerance in exercising men wearing protective clothing. *Eur J Appl Physiol*. 1994;68:234–245.
230. Aoyagi Y, Mcllellan TM, Shephard RJ. Effects of 6 versus 12 days of heat acclimation on heat tolerance in lightly exercising men wearing protective clothing. *Eur J Appl Physiol*. 1995;71:187–196.
231. Chang SKW, Gonzalez RR. *Limited Effectiveness of Heat Acclimation to Soldiers Wearing U.S. Army and U.S. Air Force Chemical Protective Clothing*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1996. Technical Report TR96-6.

232. Belding HS, Hertig BA, Kranning KK. Comparison of man's responses to pulsed and unpulsed environmental heat and exercise. *J Appl Physiol.* 1966;21:138-142.
233. Lind AR. Physiological effects of continuous or intermittent work in the heat. *J Appl Physiol.* 1963;18:57-60.
234. Graveling RA, Morris LA. Influence of intermittency and static components of work on heat stress. *Ergonomics.* 1995;38:101-114.
235. Mclellan TM, Jacobs I, Bain JB. Continuous vs. intermittent work with Canadian forces NBC clothing. *Aviat Space Environ Med.* 1993;64:595-598.
236. Cadarette BS, Montain SJ, Kolka MA, Stroschein LA, Matthew WT, Sawka MN. *Evaluation of USARIEM Heat Strain Model: MOPP Level, Exercise Intensity in Desert and Tropic Climates.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1996. Technical Report T96-4.
237. Givoni B, Goldman RF. Predicting effects of heat acclimatization on heart rate and rectal temperature. *J Appl Physiol.* 1973;35:875-879.
238. Givoni B, Goldman RF. Predicting rectal temperature response to work, environment, and clothing. *J Appl Physiol.* 1972;32:812-822.
239. Gonzalez RR, Mclellan TM, Withey WR, Chang SK, Pandolf KB. Heat strain models applicable for protective clothing systems: Comparison of core temperature response. *J Appl Physiol.* 1997;83:1017-1032.
240. Burr RE, Modrow HE, King N, et al. *Sustaining Health and Performance in Haiti: Guidance for Small Unit Leaders.* Fort Detrick, Frederick, Md: US Army Medical Research and Materiel Command; 1994. TN 94-4.
241. Mclellan TM, Jacobs I, Bain JB. Influence of temperature and metabolic rate on work performance with Canadian forces NBC clothing. *Aviat Space Environ Med.* 1993;64:587-594.
242. Goldman RF. Tolerance time for work in the heat when wearing CBR protective clothing. *Mil Med.* 1963;128:776-786.
243. Bishop PA, Pieroni RE, Smith JF, Constable SH. Limitations to heavy work at 21°C of personnel wearing the U.S. military chemical defense ensemble. *Aviat Space Environ Med.* 1991;62:216-220.
244. Cadarette BS, DeCristofano BS, Speckman KL, Sawka MN. Evaluation of three commercial microclimate cooling systems. *Aviat Space Environ Med.* 1990;61:71-78.
245. Pandolf KB, Gonzalez JA, Sawka MN. *An Updated Review: Microclimate Cooling of Protective Overgarments in the Heat.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1995. Technical Report T95-7.
246. Shapiro Y, Pandolf KB, Sawka MN, Toner MM, Winsmann FR, Goldman RF. Auxiliary cooling: Comparison of air-cooled vs water-cooled vests in hot-dry and hot-wet environments. *Aviat Space Environ Med.* 1982;53:785-789.
247. Toner MM, Drolet L, Levell CA, et al. *Comparison of Air Shower and Vest Auxiliary Cooling During Simulated Tank Operations in the Heat.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1983. Technical Report TR2-83.
248. Muza SR, Pimental NA, Cosimini HM. *Effectiveness of an Air Cooled Vest Using Selected Air Temperature, Humidity and Air Flow Rate Combinations.* Natick, Mass: US Army Institute of Environmental Medicine; 1987. Technical Report T22-87.
249. Pimental NA, Cosimini HM, Sawka MN, Wenger CB. Effectiveness of air-cooled vest using selected air temperature and humidity combinations. *Aviat Space Environ Med.* 1987;58:119-124.
250. Cadarette BS, Latzka WL, Levine L, Sawka MN. *A Physiological Evaluation of a Prototype Air-Vest Microclimate Cooling System.* Natick, Mass: US Army Research Institute of Environmental Medicine; 1991. Technical Report TR14-91.

251. Constable SH, Bishop PA, Nunneley SA, Chen T. Intermittent microclimate cooling during rest increases work capacity and reduces heat stress. *Ergonomics*. 1994;37:277–285.
252. Cadarette BS, Young AJ, DeCristofano BS, Speckman KL, Sawka MN. *Physiological Responses to a Prototype Air-Liquid Microclimate Cooling System During Exercise in the Heat*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1988. Technical Report TR12-88.
253. Cadarette BS, Pimental NA, Levell CA, Bogart JE, Sawka MN. *Thermal Responses of Tank Crewman Operating With Microclimate Cooling Under Simulated NBC Conditions in the Desert and Tropics*. Natick, Mass: US Army Research Institute of Environmental Medicine; 1986. Technical Report TR7-86.
254. Muza SR, Pimental NA, Cosimini HM, Sawka MN. Portable, ambient air microclimate cooling in simulated desert and tropic conditions. *Aviat Space Environ Med*. 1988;59:533–538.
255. Piehl K. Time course for refilling of glycogen stores in human muscle fibres following exercise-induced glycogen depletion. *Acta Physiol Scand*. 1974;90:297–302.
256. Bomalaski SH, Chen YT, Constable SH. Continuous and intermittent personal microclimate cooling strategies. *Aviat Space Environ Med*. 1995;66:745–750.
257. Cunningham DJ, Stolwijk JAJ, Wenger CB. Comparative thermoregulatory responses of resting men and women. *J Appl Physiol*. 1978;45:908–915.
258. Stephenson LA, Kolka MA. Thermoregulation in women. *Exerc Sport Sci Rev*. 1993;21:231–262.
259. 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*. Carmel, Ind (now in Traverse City, Mich): Cooper Publishing Group; 1988: 267–304.
260. Avellini BA, Kamon E, Krajewski JT. Physiological responses of physically fit men and women to acclimation to humid heat. *J Appl Physiol*. 1980;49:254–261.
261. Avellini BA, Shapiro Y, Pandolf KB, Pimental NA, Goldman RF. Physiological responses of men and women to prolonged dry heat exposure. *Aviat Space Environ Med*. 1980;51:1081–1085.
262. Frye AJ, Kamon E. Responses to dry heat of men and women with similar aerobic capacities. *J Appl Physiol*. 1981;50:65–70.
263. Shapiro Y, Pandolf KB, Avellini BA, Pimental NA, Goldman RF. Physiological responses of men and women to humid and dry heat. *J Appl Physiol*. 1980;49:1–8.
264. Hessemer V, Brück K. Influence of menstrual cycle on shivering, skin blood flow, and sweating responses measured at night. *J Appl Physiol*. 1985;59:1902–1910.
265. Baker PT. Racial differences in heat tolerance. *Am J Phys Anthropol*. 1958;16:287–305.
266. Robinson S, Dill DB, Wilson JW, Nielsen M. Adaptations of white men and Negroes to prolonged work in humid heat. *Am J Trop Med*. 1941;21:261–287.
267. Wyndham CH, Bower WM, Devine MG, Patterson HE. Physiological responses of African laborers at various saturated air temperatures, wind velocities and rates of energy expenditure. *J Appl Physiol*. 1952;5:290–298.
268. Ladell WSS. Inherent acclimatization of indigenous West Africans: Assessment of group acclimatization to heat and humidity. *J Physiol (Lond)*. 1951;115:296–312.
269. Wyndham CH, Strydom NB, Morrison JF, et al. Heat reactions of Caucasians and Bantu in South Africa. *J Appl Physiol*. 1964;19:598–606.

270. Robinson S. Physiological effects of heat and cold. *Annu Rev Physiol.* 1952;14:73–96.
271. Drinkwater BL, Bedi JF, Loucks AB, Roch S, Horvath S. Sweating sensitivity and capacity of women in relation to age. *J Appl Physiol.* 1982;53:671–676.
272. Hellon RF, Lind AR. The influence of age on peripheral vasodilatation in a hot environment. *J Physiol (Lond).* 1958;141:262–272.
273. Hellon RF, Lind AR, Weiner JS. The physiological reactions of men of two age groups to a hot environment. *J Physiol (Lond).* 1956;133:118–131.
274. Lind AR, Humphreys PW, Collins KJ, Foster K, Sweetland KF. Influence of age and daily duration of exposure on responses of men to work in the heat. *J Appl Physiol.* 1970;28:50–56.
275. Wagner JA, Robinson S, Tzankoff SP, Marino RP. Heat tolerance and acclimatization to work in the heat in relation to age. *J Appl Physiol.* 1972;33:616–622.
276. Drinkwater BL, Horvath SM. Heat tolerance and aging. *Med Sci Sports.* 1979;11:49–55.
277. Hellon RF, Lind AR. Observations on the activity of sweat glands with special reference to the influence of aging. *J Physiol (Lond).* 1956;133:132–144.
278. Anderson RK, Kenney WL. Effect of age on heat-activated sweat gland density and flow during exercise in dry heat. *J Appl Physiol.* 1987;63:1089–1094.
279. Kenney WL. Control of heat-induced cutaneous vasodilatation in relation to age. *Eur J Appl Physiol.* 1988;57:120–125.
280. Pandolf KB, Cadarette BS, Sawka MN, Young AJ, Francesconi RP, Gonzalez RR. Thermoregulatory responses of middle-aged and young men during dry-heat acclimation. *J Appl Physiol.* 1988;65:65–71.
281. Robinson SH, Belding HS, Consolazio FC, Horvath SM, Turrell ES. Acclimatization of older men to work in heat. *J Appl Physiol.* 1965;20:583–586.
282. Tankersley CG, Smolander J, Kenney WL, Fortney SM. Sweating and skin blood flow during exercise: Effects of age and maximal oxygen uptake. *J Appl Physiol.* 1991;71:236–242.
283. Pandolf KB. Aging and heat tolerance at rest or during work. *Exp Aging Res.* 1991;17:189–204.
284. Stephenson LA, Wenger CB, O'Donovan BH, Nadel ER. Circadian rhythm in sweating and cutaneous blood flow. *Am J Physiol.* 1984;246:R321–R324.
285. Sawka MN, Gonzalez RR, Pandolf KB. Effects of sleep deprivation on thermoregulation during exercise. *Am J Physiol.* 1984;246:R72–R77.
286. Kolka MA, Stephenson LA. Exercise thermoregulation after prolonged wakefulness. *J Appl Physiol.* 1988;64:1575–1579.
287. Machle W, Hatch TF. Heat: Man's exchanges and physiological responses. *Physiol Rev.* 1947;27:200–227.
288. Bass DE. Thermoregulatory and circulatory adjustments during acclimatization to heat in man. In: Hardy JD, ed. *Temperature, Its Measurement and Control in Science and Industry, Biology and Medicine.* New York, NY: Van Nostrand Reinhold; 1963: 299–305.
289. Pandolf KB, Griffin TB, Munro EH, Goldman RF. Heat intolerance as a function of percent body surface involved with miliaria rubra. *Am J Physiol.* 1980;239:R233–R240.
290. Pandolf KB, Griffin TB, Munro EH, Goldman RF. Persistence of impaired heat tolerance from artificially induced miliaria rubra. *Am J Physiol.* 1980;239:R226–R232.

291. Pandolf KB, Gange RW, Latzka WA, Blank IH, Kraning KKI, Gonzalez RR. Human thermoregulatory responses during heat exposure after artificially induced sunburn. *Am J Physiol.* 1992;262:R610–R616.
292. Kolka MA, Levine L, Cadarette BS, Rock PB, Sawka MN, Pandolf KB. Effects of heat acclimation on atropine-impaired thermoregulation. *Aviat Space Environ Med.* 1984;5:1107–1110.
293. Kolka MA, Stephenson LA. Cutaneous blood flow and local sweating after systemic atropine administration. *Pflügers Arch.* 1987;410:524–529.
294. Kolka MA, Stephenson LA, Brutig SP, Cadarette BS, Gonzalez RR. Human thermoregulation after atropine and/or pralidoxime administration. *Aviat Space Environ Med.* 1987;58:545–549.
295. Kolka MA, Holden WL, Gonzalez RR. Heat exchange following atropine injection before and after heat acclimation. *J Appl Physiol.* 1984;56:896–899.
296. Levine L, Sawka MN, Joyce BE, Cadarette BS, Pandolf KB. Varied and repeated atropine dosages and exercise-heat stress. *Eur J Appl Physiol.* 1984;53:12–16.
297. Craig FN. Effects of atropine, work and heat on heart rate and sweat production in man. *J Appl Physiol.* 1952;4:826–833.
298. Robinson S. *The Physiological Effects of Atropine and Potential Atropine Substitutes.* Army Chemical Center, Md: Medical Laboratories, Chemical Corps; 1953. Contract Report 15.
299. Kolka MA, Stephenson LA. Human temperature regulation during exercise after oral pyridostigmine administration. *Aviat Space Environ Med.* 1990;61:220–224.
300. Stephenson LA, Kolka MA. Acetylcholinesterase inhibitor, pyridostigmine bromide, reduces skin blood flow in humans. *Am J Physiol.* 1990;258:R951–R957.
301. Cook JE, Kolka MA, Wenger CB. Chronic pyridostigmine bromide administration: Side effects among soldiers working in a desert environment. *Mil Med.* 1992;157:250–254.
302. Clark WG, Lipton JM. Drug-related heatstroke. *Pharmacol Ther.* 1984;26:345–388.
303. Wenger CB. The regulation of body temperature. In: Rhoades RA, Tanner GA, eds. *Medical Physiology.* New York, NY: Little, Brown; 1995: 587–613.
304. Pescatello LS, Mack GW, Leach CN, Nadel ER. The effect of beta-blockade on thermoregulation during exercise. *J Appl Physiol.* 1986;62:1448–1452.
305. Freund BJ, Joyner MJ, Jilka SM, et al. Thermoregulation during prolonged exercise in heat: Alterations with  $\beta$ -adrenergic blockade. *J Appl Physiol.* 1987;63:930–936.
306. Pescatello LS, Mack GW, Leach CN, Nadel ER. Thermoregulation in mildly hypertensive men during  $\beta$ -adrenergic blockade. *Med Sci Sports Exerc.* 1990;22:222–228.
307. Mack GW, Shannon LM, Nadel ER. Influence of beta-adrenergic blockade on the control of sweating in humans. *J Appl Physiol.* 1986;61:1701–1705.
308. Gordon NF, Kruger PE, van Rensburg JP, Van Der Linde A, Kielblock AJ, Cilliers JF. Effect of beta-adrenoceptor blockade on thermoregulation during prolonged exercise. *J Appl Physiol.* 1985;58:899–906.
309. Mittleman KD. Influence of angiotensin II blockade during exercise in the heat. *Eur J Appl Physiol.* 1996;72:542–547.
310. Oldenburg B, MacDonald GJ, Shelley S. Controlled trial of enalapril in patients with chronic fluid overload undergoing dialysis. *Br Med J.* 1988;296:1089–1091.
311. Hansen J. Hydrochlorothiazide in the treatment of hypertension. *Acta Med Scand.* 1968;83:317–321.

312. Leth A. Changes in plasma and extracellular fluid volumes in patients with essential hypertension during long-term treatment with hydrochlorothiazide. *Circulation*. 1970;42:479–485.
313. Tarazi RC, Dustan HP, Frohlich EP. Long-term thiazide therapy in essential hypertension: Evidence for persistent alteration in plasma volume and renin activity. *Circulation*. 1970;41:709–717.

