Journal of Athletic Training 1999;34(3):246–252

www.nata.org/jat

Exercise in the Heat. I. Fundamentals of Thermal Physiology, Performance Implications, and Dehydration

Douglas J. Casa, PhD, ATC, CSCS

University of Connecticut, Storrs, CT

Objective: To present the critical issue of exercise in the heat in a format that provides physiologic foundations (Part I) and then applies the established literature to substantial, usable guidelines that athletic trainers can implement on a daily basis when working with athletes who exercise in the heat (Part II).

Data Sources: The databases MEDLINE and SPORT Discus were searched from 1980 to 1999, with the terms "hydration," "heat," "dehydration," "cardiovascular," "thermoregulatory," "physiology," and "exercise," among others. The remaining citations are knowledge base.

Data Synthesis: Part I introduces athletic trainers to some of the basic physiologic and performance responses to exercise in the heat.

Conclusions/Recommendations: The medical supervision of athletes who exercise in hot environments requires an in-depth understanding of basic physiologic responses and performance considerations. Part I of this article aims to lay the scientific foundation for efficient implementation of the guidelines for monitoring athletic performance in the heat provided in Part II.

Key Words: cardiovascular, heat stress, thermoregulatory

Exercise in the heat, as compared with a neutral environment, causes many physiologic changes in the dynamics of the human body, including alterations in the circulatory, thermoregulatory, and endocrine systems. Many interrelated physiologic processes work together to sustain central blood pressure, cool the body, maintain muscular function, and regulate fluid volume. Attempting to sustain exercise (especially if it is intense) in a hot environment can overload the body's ability to properly respond to the imposed stress, resulting in hyperthermia, dehydration, deteriorated physical and mental performance, and a potentially serious (even fatal) exertional heat illness.

CIRCULATORY RESPONSES

The circulatory responses to exercise involve 3 important components: skin and muscle vasodilation, nonactive tissue vasoconstriction, and maintenance of blood pressure[1] (Figure 1). Skin vasodilation occurs in proportion to the degree of heat load (both exogenous and endogenous),[2,3] and the amount of blood supplied to the muscles is dictated by the intensity of the exercise. Constriction of the splanchnic vascular system (supplying the kidneys, stomach, and other abdominal organs), in addition to an overall increase in the cardiac output, allows increased blood flow to the active tissues.[4–7]

However, when intense exercise occurs in the heat, the cardiovascular (CV) system simply cannot meet the maximal demands of the skin (to decrease thermal load) and the muscle simultaneously.[1,8] Ultimately, maintenance of blood pressure will take precedence over skin blood flow (ie, body cooling) and muscle blood flow (ie, performance capacity), but simultaneously increases the rate of hyperthermia and metabolic inefficiency.[1,10,11] This prioritizing can result in hyperthermia, especially in populations committed to maximal physical exertion (soldiers, athletes, etc). The metabolic changes are reflected in an increased lactate level, which results from decreased hepatic blood flow; muscle vasoconstriction (which influences waste removal, oxygen delivery, buffering capacity, etc); and an increase in muscle temperature.[11] Variations in the onset of these changes can alter the rate at which the athlete experiences fatigue.

Decreased venous return reduces the stimulation of pressure-sensitive baroreceptors in the right heart and the pulmonary circulation.[12] Messages are then sent to the medullary CV control centers, which can cause muscle or skin vasoconstriction, or both, thereby preserving blood pressure and CV function.[1,13]

Minimal decreases in cardiac output have been found in subjects exercising at submaximal intensities in the heat.[1,13,14] An increase in heart rate compensates for the decreases in stroke volume, and CV capacity is not hindered, unless

Address correspondence to Douglas J. Casa, PhD, ATC, CSCS, Department of Sport, Leisure, and Exercise Sciences, Neag School of Education, University of Connecticut, 2095 Hillside Road, Box U-110, Storrs, CT 06269-1110.

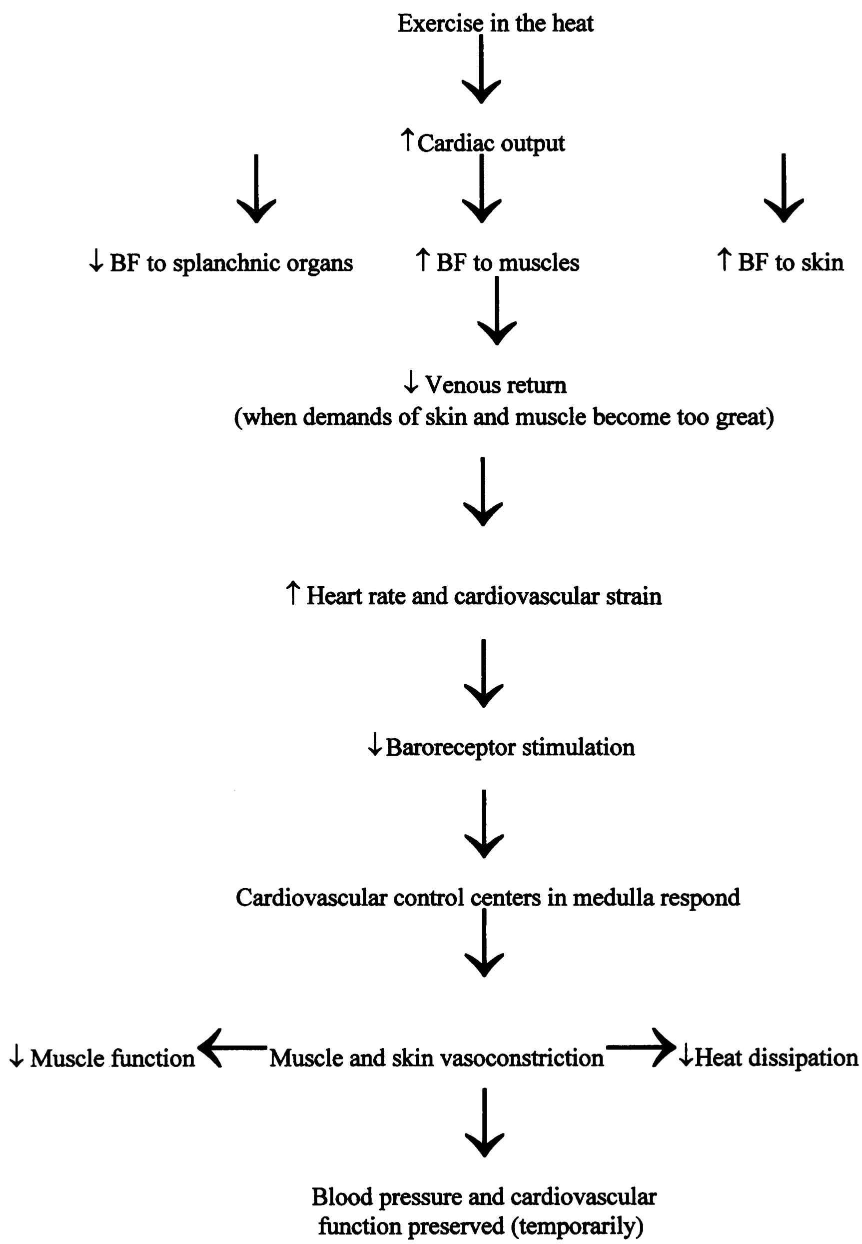


Figure 1. Potential circulatory responses to exercise in the heat.[1–7,9,12,13] BF, blood flow.

extreme sweat rates or lengthy exercise sessions, for example, induce significant dehydration. But when maximal exercise is attempted in the heat, the heart rate's finite limit does not compensate for the larger decreases in stroke volume, due mostly to shunting of blood to the skin and active muscle and to the progressive dehydration.[1,7,13] Rowell[1] concluded that the end result is decreases in both Vo_2 and performance capacity.

THERMOREGULATORY RESPONSES

The circulatory and thermoregulatory responses are interrelated, with each influencing and being influenced by the other. The degree of stress imposed by exercise in a hot environment is determined by the thermal load. Heat gain must be equaled (or closely matched) by heat dissipation if the athlete wishes to continue exercising at a consistent performance level. Exogenous factors that contribute to heat acquisition include ambient temperature, wind speed, humidity, solar radiation (direct and indirect), ground thermal radiation, and clothing.[15] Ambient temperature and humidity are the major contributors; lack of wind in the presence of high humidity and high ambient temperature can impose severe heat stress because copious sweating is not cooling the body (sweat is not evaporating from the skin), which exacerbates the hyperthermia.[16] The predominant endogenous factor is the metabolic heat from contracting muscle (capable of increasing 15 to 20 times during exercise in healthy young adults), which is profoundly influenced by the intensity of the exercise.

The body attempts to balance internal temperature by dissipating heat via conduction, convection, evaporation, and radiation.[15,17] Heat dissipation while exercising depends on the ambient temperature. As ambient temperature rises, radiation and convection decrease markedly; heat loss by conduction is insignificant at almost all times.[15,18] Convection is compromised by a temperature gradient change between the peripheral blood vessels and the skin. Heat loss from evaporation thus becomes the predominant heat-dissipating mechanism for a subject exercising in a hot environment. In a hot, dry environment, evaporation can account for as much as 98% of cooling, whereas in a hot, wet environment, evaporation is still nearly 80% (the rest is largely convection and radiation).[18] The sweating response is critical to whole-body cooling during exercise in the heat; any disturbance in this mechanism (eg, high humidity, dehydration) can have profound effects on physiologic function and athletic performance. The reader is referred to Stitt[17] and Werner[15] for in-depth analyses of the heat balance equations, but, in short, heat acquisition (from exogenous and endogenous sources) must be matched by the combined 4 heat-dissipation pathways to maintain thermal balance: heat storage = heat production minus heat dissipation or plus heat acquisition. This may be expressed as $\pm S = (M-W) \pm C \pm K \pm R - E$, where S is body heat storage, M is metabolic heat production, W is external work, and C, K, R, and E represent convection, conduction, radiation, and evaporation, respectively.[17,19]

When heat dissipation fails to equal heat acquisition, hyperthermia increases skin blood flow, and, depending on the environmental conditions, heat release via convection, radiation, and evaporation.[19] Skin blood flow changes are regulated not just by body temperature, but also by blood pressure, brain blood flow temperature, skin-core temperature gradients, muscle metabolism, etc. As discussed previously, maintenance of blood pressure takes precedence over heat dissipation.

Kenney and Johnson[2] and Sawka and Wenger[7] reported on the integration of these regulatory processes, in addition to the important role of the efferent mechanisms controlling skin blood flow (ie, passive withdrawal of constrictor tone, reflex vasoconstriction, and active vasodilation). The inherent changes in sweating rate and body cooling associated with skin blood flow changes assist in controlling hyperthermia (the primary controller of sweating rate). Nadel[10] and Sato[20] offer the best explanation of eccrine sweat secretion. Warmer air temperatures are associated with increased sweating.[21] Since only evaporation is an efficient mode of heat dissipation in this situation, physiologic strain is exacerbated by the decreased extracellular fluid volume associated with copious sweating. In the short term, the body is being cooled, but increased dehydration alters CV functional capacity, which can lead to decreased skin blood flow and sweating rate as the body attempts to maintain the central circulation and blood pressure.

In a cooler environment (with a larger temperature gradient between skin blood flow and skin temperature), the body can avoid hyperthermia while minimizing fluid losses via convection and radiation. In a warm, humid environment, all the critical variables work against the exercising individual: convection and radiation are nearly nonexistent,[10] and evaporation is thwarted by a small water vapor pressure gradient.[7] With no heat dissipation, dehydration occurs, and the core temperature rises at a potentially dangerous rate.[22] The decreased physiologic function associated with hyperthermia is well documented,[23] and the rate of onset of hyperthermia can be influenced by fitness,[15] acclimation,[24] type of exercise,[25] age,[26] and numerous other factors.

PERFORMANCE IMPLICATIONS

The additive effect of the stresses imposed by exercise in the heat will ultimately compromise athletic performance. In addition, exercise in the heat often causes dehydration (since rates of sweating are rarely matched by rates of rehydration), which further exacerbates the situation.[27,28] It is extremely difficult to separate the effects of heat and dehydration, since they often occur in parallel during prolonged exercise, but some researchers have attempted to match sweat loss with fluid intake during exercise. Rowell et al[29] found large reductions in stroke volume despite maintained central blood volume. Enhanced physical fitness and heat acclimatization increase heat tolerance independently but similarly and optimize heat tolerance when combined.[30]

Sawka et al[31] reported a 7% decrease in maximal aerobic power in the heat as compared with euhydrated subjects in cool temperatures. Febbraio et al[32] and Galloway and Maughan[33] showed the effects of increasing temperature on the capacity to exercise to exhaustion. Febbraio et al[32] found that subjects could exercise for 95 minutes at 37°F (2.78°C), 75 minutes at 68°F (20°C), and only 33 minutes at 104°F (40°C), indicating an inverse linear relationship between ambient temperature and performance capacity. The 20-minute difference in the 2 cooler environments is an important reminder that extreme heat is not necessary for potential performance decrements. Galloway and Maughan[33] concurred, reporting that subjects exercised for 92 minutes at 52°F (11.11°C), 83 minutes at 70°F (21.11°C), and 51 minutes at 86°F (30°C). These studies supported the concept of Sawka et al[34] that heat stress and dehydration can act independently to compromise physiologic function when the extreme demands for skin blood flow cause decreased cardiac output, which in turn limits the supply of oxygenated blood to the entire body. When heat stress and dehydration occur together (as they often do), this physiologic condition is exacerbated. In addition to performance decrements, the potential for an exertional heat illness increases as the environmental conditions worsen. The American College of Sports Medicine[22] provided a concise analysis of how to determine when the environmental conditions preclude physical activity and what procedures should be followed to ensure safe participation in a hot environment (to be addressed in part II).

DEHYDRATION AND EXERCISE

Each physiologic system in the human body is influenced by severe dehydration. The degree of dehydration will dictate how much these systems are compromised. Figure 2 describes similar terms used to describe water losses and gains. The work of Sawka and colleagues[34–36] is definitive in the domain of hypohydration and its impact on performance and physiologic function. Their laboratory, located within the US Army Research Institute of Environmental Medicine in Natick, MA, is one of the preeminent locations in the world for investigating the human body's capacity to perform exercise in a variety of environments.

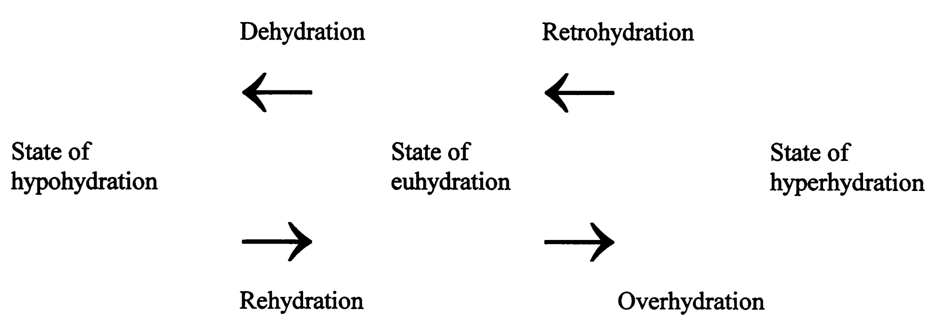
Physiologic Changes

Isolating which particular physiologic changes contribute to decrements in performance is difficult, if not impossible. The interrelation of the human body's systems means that any change in one system influences others. However, recent research has begun to uncover what occurs when an athlete becomes dehydrated during exercise. Dehydration induces changes in the thermoregulatory, cardiovascular, plasma, gastrointestinal, endocrine, muscular, and metabolic responses to exercise.[37,38]

As discussed earlier, the CV system of a hypohydrated, exercising subject attempts to maintain cardiac filling pressure while sacrificing peripheral circulation,[1] but hypohydration in combination with heat dissipation at the skin and increased muscle blood flow limits CV capacity, regardless of how much blood is shunted from the periphery to the central circulation.[1,39,40] Increased viscosity and decreased volume of blood returning to the heart decrease filling pressure, and in turn, stroke volume.[14,41,42] To counteract these changes, heart rate rises to its limit, but then cardiac output begins to fall, signaling CV system responses, which limit skin and muscle function.[34,43] The end result is a diminished ability to dissipate heat, and thus, heat production exceeds heat loss. Excess heat in combination with decreased muscle perfusion limits performance and causes thermal strain.[1,35]

Exercising while dehydrated has some effects on the thermoregulatory system[34,44–49] (Table) and may negate the physiologic advantages resulting from increased fitness[24,50] and heat acclimatization.[24,51] Sawka et al[36] noted decreased heat tolerance (by more than half) in subjects dehydrated by 8% of body weight and found that soldiers became exhausted at lower core temperatures when hypohydrated. While 8% is an extreme amount of dehydration rarely encountered in sports, the study emphasizes the decreased heat tolerance associated with dehydration.

The human body is composed of about 65% water, separated into extracellular (plasma and interstitial) and intracellular fluid.[52] At rest with normal hydration, about 45% of body weight is intracellular fluid, 15% is interstitial fluid, and 5% is plasma.[52] Exercise, heat stress, and dehydration all influence the redistribution of body fluids with changes in hydrostatic and osmotic pressure.[52,53] For instance, because sweat is hypotonic to plasma, the dehydrated athlete experiences plasma hyperosmolality, which affects the distribution of fluids.[35] Mild dehydration causes mostly extracellular space fluid losses, but, as dehydration worsens, proportionally more fluid is lost from the intracellular space.[54,55] Nose et al[56] reported that the loss of intracellular and extracellular fluid is largely from muscle and skin. This selective regulation of body fluids preserves the internal environment of the most essential organs: for instance, the brain and liver.[35] Changes in the distribution of body fluids are associated with the ability to mobilize fluids from the intracellular space, which is intimately linked with sweat sodium concentrations.[57] Thus, the de-



Euhydration: steady-state condition of normal body water
Hypohydration: steady-state condition of decreased body water
Hyperhydration: steady-state condition of increased body water
Dehydration: water loss leading to hypohydration
Retrohydration: water loss from a state of hyperhydration leading to euhydration
Rehydration: adding water from a state of hypohydration to move toward euhydration
Overhydration: fluid intake that exceeds euhydration, leading to hyperhydration

Figure 2. Clarification of terms to describe body water losses and gains during exercise. Adapted with permission from Epstein and Armstrong.[86] The term "retrohydration" is used courtesy of P. M. Meenen, July 1999.

Thermoregulatory Effects of Exercise in the Dehydrated State[34,44–49]

↓ Sweating rate at given core temperature	↓ Maximal sweating rate
↓ Skin blood flow at given core temperature	↓ Maximal skin blood flow
↑ Core temperature at which sweating begins	↑ Core temperature at given exercise intensity
↑ Core temperature at which skin blood flow increases	

creased sweat sodium concentrations noted after heat acclimatization may help to conserve plasma volume during dehydration. Ultimately, the fluid redistribution that results from dehydration causes a hypovolemic hyperosmolality,[58] which stimulates the volume and fluid receptors in the body to conserve fluid and stimulate rehydration.[52]

Plasma changes have been cited as the major cause for the thermoregulatory changes during hypohydration. Hyperosmolality[59–61] and hypovolemia[46,62] are likely responsible for the changes noted in skin blood flow and sweating rate and the resultant rises in core temperature.[9,35,40] Fortney et al[46] have argued that hypovolemia is primarily responsible for the thermoregulatory changes by reducing central blood volume, which may alter the feedback to the hypothalamus via the atrial baroreceptors. The hypothalamic thermoregulatory centers may then decrease the blood volume perfusing the skin in an attempt to reestablish a normal central blood volume. Some studies have provided support for this hypothesis,[63,64] but it is clearly not the only variable influencing thermoregulation during hypohydration.

Two primary hypotheses have been proposed to explain the role of hyperosmolality on the thermoregulatory system. The first is a strong osmotic pressure influence of the interstitium, which may limit the available fluid sources for the eccrine sweat glands.[65] While this pressure is likely to exert some influence, it seems more feasible that brain regulation, the second hypothesis, has the largest contribution. The neurons surrounding the thermoregulatory control centers in the hypothalamus are quite sensitive to osmolality.[66,67] Thus, changes in the plasma perfusing the hypothalamus can affect body water regulation and the desire for fluid consumption.[40,43] The human body is well equipped to identify small changes in the internal environment and to respond with appropriate modifications. While research may someday identify a proportional contribution to the age-old question of hyperosmolality versus hypovolemia, it is most likely that both will always be considered major contributors to the mechanisms that perturbate body fluid regulation.

Potential muscle changes associated with dehydration include an increased rate of glycogen synthesis,[11,48] compromised buffering capacity of the muscle tissue,[38] elevated muscle temperature,[68] and decreased substrate exchange.[11,38] These factors are caused by a decrease in blood flow perfusing the muscle tissue, which may alter the dynamics during the recovery between contractions.[69] These muscle changes seem to occur when exercise exceeds 30 seconds, which is reasonable from a metabolic perspective.[70] These arguments would support the notion that strength during short-term activity is not affected until dehydration becomes more pronounced, largely due to the fact that the muscle energetics of very short-term activity are, for the most part, self-contained, and thus, not as influenced by changes in blood flow.[38]

Performance Implications

Research investigating the role of dehydration on muscle strength has yielded conflicting results. Some studies have shown performance decrements,[71–74] while others have shown no changes.[14,75] However, when strength decrements were found, they usually occurred when dehydration exceeded a 5% reduction in body weight.[34,49] In addition, dehydration resulting from fluid restriction seems to be more harmful than that caused by exercise and heat stress; thus, the fluid restriction may be partially inducing a caloric deficit.[34]

The research on muscle endurance is a bit more conclusive. A sampling of the numerous studies[14,72,76–79] that have addressed the influence of dehydration on muscle endurance reveals, generally speaking, that 3% to 4% dehydration elicits a performance decrement, but some studies investigating greater levels of dehydration did not find any differences in performance.[34] Horswill[38] concluded that, in wrestlers (who are frequently hypohydrated), combined hypohydration and maximal or near-maximal muscle activity exceeding 30 seconds may combine to decrease performance. Environmental conditions may also play an important role in muscle endurance,[34,68] and, since greater hypohydration often occurs in hot conditions, more studies should investigate this relationship.

The research concerning maximal aerobic power and the physical work capacity for extended exercise is also relatively conclusive and consistent. Maximal aerobic power usually decreases when dehydration exceeds a 2% to 3% reduction in body weight, and, when performed in the heat, the decrements are exaggerated.[34] Nearly every study that has examined physical work capacity has shown some degree of performance decrement.[34] Even with only 1% to 2% hypohydration in a cool environment,[80,81] a decrement is noted. Pinchan et al[82] and Walsh et al[83] noted decreases in physical work capacity with less than 2% dehydration during intense exercise in the heat. As expected, when dehydration increased, physical work capacity decreased, sometimes by as much as 35% to 48%,[84] and physical work capacity often decreased even when maximal aerobic power did not change.[80,81,85] Buskirk and Puhl[69] suggested that some of these decrements with low to moderate levels of hypohydration may be partly due to an increased perception of fatigue. The degree of change in physiologic function will be dependent on various exercise parameters, including intensity, duration, environmental stress, and individual factors.

CONCLUSION

Exercise in the heat triggers a disturbance of the internal environment of the human body. Understanding the responses requires an astute ability to focus on many independent physiologic processes that function cooperatively. The athlete wishes for these systems to rise to any challenge, but often excessive heat, dehydration, or both cause some degree of decrement in performance. The ensuing part of this 2-part series about exercise in the heat attempts to identify ways in which athletic trainers and athletes can work toward minimizing the decrement by maximizing heat dissipation and body fluid balance.

ACKNOWLEDGMENT

I would like to dedicate this paper to the memory of my former supervisor, Dean Leo W. Anglin, Jr, PhD. I would later learn that he took his final breaths as I wrote this article. He was a visionary in the field of education, and the passion that drove him was contagious. I shall strive in his memory.

REFERENCES

1. Rowell LB. *Human Circulation Regulation During Physiological Stress.* New York, NY: Oxford University Press; 1986.
2. Kenney WL, Johnson JM. Control of skin blood flow during exercise. *Med Sci Sports Exerc.* 1992;24:303–312.
3. Nadel ER. Circulatory and thermal regulations during exercise. *Fed Proc.* 1980;39:1491–1497.
4. Laughlin MH, Korthuis RJ, Duncker DJ, Bache RJ. Control of blood flow to cardiac and skeletal muscle during exercise. In: Rowell LB, Shepherd JT, eds. *Exercise: Regulation and Integration of Multiple Systems.* New York, NY: Oxford University Press; 1996:705–769.
5. 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.
6. Rowell LB, Bregelmann GL, Blackmon JR, Twiss RD, Kusumi F. Splanchnic blood flow and metabolism in heat-stressed man. *J Appl Physiol.* 1968;24:475–484.
7. 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.* Dubuque, IA: Brown and Benchmark; 1988:97–152.
8. Holtz J. Peripheral circulation: fundamental concepts, comparative aspects of control in specific vascular sections, and lymph flow. In: Greger R, Windhorst U, eds. *Comprehensive Human Physiology: From Cellular Mechanisms to Integration.* New York, NY: Springer; 1996:1865–1915.
9. Henry JP, Gauer OH. The influence of temperature upon venous pressure in the foot. *J Clin Invest.* 1950;29:855–862.
10. Nadel ER. Control of sweating rate while exercising in the heat. *Med Sci Sports Exerc.* 1979;11:31–35.
11. Young AJ. Energy substrate utilization during exercise in extreme environments. *Exerc Sport Sci Rev.* 1990;18:65–117.
12. Coyle EF. Cardiovascular function during exercise: neural control factors. *Sports Sci Exchange.* 1991;4:34.
13. Rowell LB, O'Leary DS, Kellogg DL. Integration of cardiovascular control systems in dynamic exercise. In: Rowell LB, Shepherd JT, eds. *Exercise: Regulation and Integration of Multiple Systems.* New York, NY: Oxford University Press; 1996:770–738.
14. Saltin B. Circulatory response to submaximal and maximal exercise after thermal dehydration. *J Appl Physiol.* 1964;19:1125–1132.
15. Werner J. Temperature regulation during exercise: an overview. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Exercise, Heat, and Thermoregulation.* Dubuque, IA: Brown and Benchmark; 1993:49–77.
16. Nadel ER. Limits imposed on exercise in a hot environment. *Sports Sci Exchange.* 1990;3:27.
17. Stitt JT. Central regulation of body temperature. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Exercise, Heat, and Thermoregulation.* Dubuque, IA: Brown and Benchmark; 1993:1–39.
18. Armstrong LE, Maresh CM. The exertional heat illnesses: a risk of athletic participation. *Med Exerc Nutr Health.* 1993;2:125–134.
19. Cooper KE. Regulation of body temperature. In: Greger R, Windhorst U, eds. *Comprehensive Human Physiology: From Cellular Mechanisms to Integration.* New York, NY: Springer; 1996:2199–2206.
20. Sato K. The mechanism of eccrine sweat secretion. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Exercise, Heat, and Thermoregulation.* Dubuque, IA: Brown and Benchmark; 1993:85–110.
21. Nadel ER, Cafarelli E, Roberts MF, Wenger CB. Circulatory regulation during exercise in different ambient temperatures. *J Appl Physiol.* 1979; 46:430–437.
22. Armstrong LE, Epstein Y, Greenleaf JE, et al. American College of Sports Medicine position stand: heat and cold illnesses during distance running. *Med Sci Sports Exerc.* 1996;28(12):i–x.
23. Bergh U, Ekblom B. Physical performance and peak aerobic power at different body temperatures. *Med Sci Sports Exerc.* 1979;46:885–889.
24. Buskirk ER, Iampietro PF, Bass DE. Work performance after dehydration: effects of physical conditioning and heat acclimatization. *J Appl Physiol.* 1958;12:189–194.
25. Irion GL. Responses of distance runners and sprinters to exercise in a hot environment. *Aviat Space Environ Med.* 1987;58:948–953.
26. Pandolf KB. Aging and human heat tolerance. *Exp Aging Res.* 1997;23: 69–105.
27. 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(1):i–vii.
28. Greenleaf JE. Problem: thirst, drinking behavior, and involuntary dehydration. *Med Sci Sports Exerc.* 1992;24:645–656.
29. 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.
30. Roberts MF, Wenger CB, Stolwijk AJ, Nadel ER. Skin blood flow and sweating changes following exercise and heat acclimation. *J Appl Physiol.* 1977;43:133–137.
31. 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;59:294–298.
32. Febbraio MA, Parkin JA, Baldwin L, Zhao S, Carey MF. Metabolic indices of fatigue in prolonged exercise at different ambient temperatures. Presented at: Conference for Dehydration, Rehydration, and Exercise in the Heat; 1995; Nottingham, England. Abstract 17.
33. Galloway SDR, Maughan RJ. Effect of ambient temperature on the capacity to perform prolonged exercise in man. *J Physiol.* 1995;489:35–36.
34. Sawka MN, Montain SJ, Latzka WA. Body fluid balance during exercise-heat exposure. In: Buskirk EW, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport.* New York, NY: CRC Press; 1996:139–157.
35. Sawka MN, Pandolf KB. Effect of body water loss on physiological function and exercise performance. In: Gisolfi CV, Lamb DR, eds. *Fluid Homeostasis During Exercise.* Carmel, IN: Brown and Benchmark; 1990:1–30.
36. Sawka MN, Young AJ, Latzka WA, Neufer PD, Quigley MD, Pandolf KB. Human tolerance to heat strain during exercise: influence of hydration. *J Appl Physiol.* 1992;73:368–375.
37. Murray R. Fluid needs in hot and cold environments. *Int J Sport Nutr.* 1995;5:S62–S73.
38. Horswill CA. Applied physiology of amateur wrestling. *Sports Med.* 1992;14:114–143.
39. Coyle EF, Montain SJ. Thermal and cardiovascular responses to fluid replacement during exercise. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Exercise, Heat, and Thermoregulation.* Dubuque, IA: Brown and Benchmark; 1993:179–212.
40. Sawka MN. Physiological consequences of hypohydration: exercise performance thermoregulation. *Med Sci Sports Exerc.* 1992;24:657–670.
41. Gonzalez-Alonso J, Mora-Rodriguez R, Below PR, Coyle EF. Dehydra-

tion reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise. *J Appl Physiol.* 1995;79:1487–1496.
42. Sproles CB, Smith DP, Byrd RJ, Allen TE. Circulatory responses to submaximal exercise after dehydration and rehydration. *J Sports Med.* 1976;16:98–105.
43. Armstrong LE, Maresh CM, Gabaree CV, et al. Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. *J Appl Physiol.* 1997;82:2028–2035.
44. 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.
45. 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.
46. 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.
47. Nadel ER, Fortney SM, Wenger CB. Effect of hydration state on circulatory and thermal regulations. *J Appl Physiol.* 1980;49:715–721.
48. Murray R. Dehydration, hyperthermia, and athletes: science and practice. *J Athl Train.* 1996;31:248–252.
49. Adolph EF, ed. *Physiology of Man in the Desert.* New York, NY: Interscience; 1947.
50. 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.
51. Sawka MN, Hubbard RW, Francesconi RP, Horstman DH. Effects of acute plasma volume expansion on altering exercise-heat performance. *Eur J Appl Physiol.* 1983;51:303–312.
52. Greenleaf JE, Morimoto T. Mechanisms controlling fluid ingestion: thirst and drinking. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport.* New York, NY: CRC Press: 1996:1–17.
53. Senay LC. Effects of exercise in the heat on body fluid distribution. *Med Sci Sports.* 1979;11:42–48.
54. Costill DL, Cote R, Fink W. Muscle water and electrolytes following varied levels of dehydration in man. *J Appl Physiol.* 1976;40:6–11.
55. Durkot MJ, Martinez O, Brooks-McQuade D, Francesconi R. Simultaneous determination of fluid shifts during thermal stress in a small-animal model. *J Appl Physiol.* 1986;61:1031–1034.
56. Nose H, Morimoto T, Ogura K. Distribution of water losses among fluid compartments of tissues under thermal dehydration in the rat. *Jpn J Physiol.* 1983;33:1019–1029.
57. Nose H, Mack GW, Shi X, Nadel ER. Shift in body fluid compartments after dehydration in humans. *J. Appl. Physiol.* 1988;65:318–324.
58. Szlyk-Modrow PC, Francesconi RP, Hubbard RW. Integrated control of body fluid balance during exercise. In: Buskirk ER, Puhl SM, eds. *Body Fluid Balance: Exercise and Sport.* New York, NY: CRC Press; 1996:117–136.
59. Candas V, Libert JP, Brandenberger G, Sagot JC, Amoros C, Kahn JM. Hydration during exercise: effects on thermal and cardiovascular adjustments. *Eur J Appl Physiol.* 1986;55:113–122.
60. Harrison MH, Edwards RJ, Fennessy PA. Intravascular volume and tonicity as factors in the regulation of body temperature. *J Appl Physiol.* 1978;44:69–75.
61. Senay LC Jr. Temperature regulation and hypohydration: a singular view. *J Appl Physiol.* 1979;47:1–7.
62. Fortney SM, Vroman NB, Beckett WS, Permutt S, LaFrance ND. Effect of exercise hemoconcentration and hyperosmolality on exercise responses. *J Appl Physiol.* 1988;65:519–524.
63. Gaddis GM, Elizondo RS. Effect of central blood volume decrease upon thermoregulation responses to exercise in the heat. *Fed Proc.* 1984;43: 627.
64. Mack G, Nose H, Nadel ER. Role of cardiopulmonary baroreflexes during dynamic exercise. *J Appl Physiol.* 1988;65:1827–1832.
65. 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.
66. Nakashima T, Hori T, Kiyohara T, Shibata M. Effects of local osmolality changes on medial preoptic thermosensitive neurons in hypothalamic slices. *In Vitro Thermal Physiol.* 1984;9:133–137.
67. Silva NL, Boulant JA. Effects of osmotic pressure, glucose and temperature on neurons in preoptic tissue slices. *Am J Physiol.* 1984;247:R335–R345.
68. Edwards RHT, Harris RC, Hultman E, Kaijser L, Koh D, Nordesjo L. Effect of temperature on muscle energy metabolism and endurance during successive isometric contractions, sustained to fatigue, of the quadriceps muscle in man. *J Physiol (Lond).* 1972;220:335–352.
69. Buskirk ER, Puhl SM. Effects of acute body weight loss in weight-controlling athletes. In: Buskirk ER, Puhl SM, eds, *Body Fluid Balance: Exercise and Sport.* New York, NY: CRC Press; 1996:283–296.
70. Horswill CA. Weight loss and weight cycling in amateur wrestlers: implications for performance and resting metabolic rate. *Int J Sports Nutr.* 1993;3:245–260.
71. Bosco JS, Terjung RL, Greenleaf JE. Effects of progressive hypohydration on maximal isometric muscular strength. *J Sports Med Phys Fitness.* 1968;8:81–86.
72. Bosco JS, Greenleaf JE, Bernauer EM, Card DH. Effects of acute dehydration and starvation on muscular strength and endurance. *Acta Physiol Pol.* 1974;25:411–421.
73. Houston ME, Marrin DA, Green HJ, Thomson JA. The effect of rapid weight loss on physiological function in wrestlers. *Physician Sportsmed.* 1981;9(11):73–78.
74. 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.
75. Ahlman K, Karvonen MJ. Weight reduction by sweating in wrestlers, and its effect on physical fitness. *J Sports Med Phys Fitness.* 1961;1:58–62.
76. Bijlani RL, Sharma KN. Effect of dehydration and a few regimes of rehydration on human performance. *Indian J Physiol Pharmacol.* 1980; 24:255–266.
77. Torranin C, Smith DP, Byrd RJ. The effect of acute thermal dehydration and rapid rehydration on isometric and isotonic endurance. *J Sports Med Phys Fitness.* 1979;19:1–9.
78. Mnatzakian PA, Vaccaro P. Effects of 4% dehydration and rehydration on hematological profiles and muscular endurance of college wrestlers. *Med Sci Sports Exerc.* 1982;14:117s.
79. Serfass RC, Stull GA, Alexander JF, Ewing JL. The effects of rapid weight loss and attempted rehydration on strength and endurance of the hand-gripping muscles in college wrestlers. *Res Q Exerc Sport.* 1968;55:46–52.
80. Caldwell JE, Ahonen E, Nousiainen U. Differential effects of sauna-, diuretic- and exercise-induced hypohydration. *J Appl Physiol.* 1984;57: 1018–1023.
81. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc.* 1985;17:456–461.
82. Pinchan G, Gauttam RK, Tomar OS, Bajaj AC. Effects of primary hypohydration on physical work capacity. *Int J Biometeorol.* 1988;32: 176–180.
83. 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.
84. Craig EN, Cummings EG. Dehydration and muscular work. *J Appl Physiol.* 1966;21:670–674.
85. 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.
86. Epstein Y, Armstrong LE. Fluid-electrolyte balance during labor and exercise: concepts and misconceptions. *Int J Sport Nutr.* 1999;9:1–12.