

## Exertional Heat Cramps: Recovery and Return to Play

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In contrast to muscle cramps that are brought on by muscle overload or fatigue, exertional heat cramps seem to be prompted by extensive sweating and a significant sweat-induced whole-body sodium deficit. As a result of a consequent contracted interstitial compartment, axon terminals of selected motor neurons can become hyper-excitabile and spontaneously discharge. Barely detectable muscle fasciculations or “twitches” in the affected muscles can rapidly progress to debilitating muscle cramps in just 20 to 30 minutes. To aid recovery, salt (NaCl) and water lost from sweating should be sufficiently replaced so as to restore the extracellular volume and interstitial fluid spaces. Sweat sodium, chloride, and fluid losses incurred during training and competition need to be closely matched by daily salt and fluid intake, in order to prevent an excessive sodium deficit, maintain sufficient fluid balance, and avoid exertional heat cramps. **Key Words:** athletics, hydration, sweating, muscle, injury management, prevention

In nearly all sports, athletes experience muscle cramps. For some, muscle cramps are routinely encountered; thus, these athletes often compete in fear of if and when these unexpected and painful involuntary muscle contractions will debilitate them once again. Importantly, many health care providers, coaches, and athletes do not appreciate that there are, as suggested by clinical indications and assessments, two dissimilar general classifications of exercise-related muscle cramps (when there is no underlying disease, disorder, pathology, or other abnormal condition present<sup>1</sup>) with distinct etiologies and preventive strategies. For one type of affliction, strong empirical evidence and other published theories support the perspective that insufficient conditioning (resulting in muscle overload) and muscle fatigue can lead to muscle cramping in the overworked muscle fibers.<sup>2-4</sup> In contrast, the discussion here will be on muscle cramps that are related to extensive sweat losses and a sodium deficit and are hereinafter referred to as exertional heat cramps.

### Etiology

A point of confusion with exertional heat cramps stems from the name itself. Even though in sports, these sometimes excruciating involuntary muscle spasms invariably occur during or following exertion, a hot environment is not a necessary

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prerequisite. Many athletes encounter such muscle cramps in cool conditions or even indoors. Excessive core body temperature is not an underlying determinant either. Accordingly, some authors have adopted the terms “exercise-induced” or “exercise-associated” muscle cramps (EAMC).<sup>5-7</sup> This terminology, however, makes no distinction with regard to the etiology. In contrast to a localized muscle cramp that is prompted by an overloaded and strained muscle (and can often be resolved using passive stretching, massage, and icing), exertional heat cramps are usually more widespread affecting (often bilaterally) a number of different muscle groups (eg, quadriceps and/or hamstrings in both legs) and profuse, repeated, or moderate long-term sweating appears to be the primary contributing factor.<sup>8-16</sup> Notably, water or other low-sodium fluid intake alone is often insufficient in adequately maintaining or restoring fluid and electrolyte balance<sup>17-19</sup> and averting or resolving exertional heat cramps.<sup>10, 20</sup>

Athletes who sweat extensively can lose electrolytes to a great extent as well—particularly sodium and chloride. And despite other minerals (eg, calcium, magnesium, and potassium) that are also lost via sweating<sup>21</sup> and whose purported deficiencies have been implicated for causing muscle cramps during or following exercise,<sup>8, 11, 13, 14, 16, 22-24</sup> exertional heat cramp-prone athletes usually develop a significant sweat-induced whole-body sodium deficit because of a substantially insufficient dietary intake of salt.<sup>9, 10, 20</sup> This can readily occur in certain athletes during a single long session of training or competition, or in response to multiple same-day sessions, even if the athletes are well-acclimatized to the heat,<sup>9, 10, 25</sup> so long as the combination of total sweat loss and sweat sodium concentration yields a significant deficit of this electrolyte. Alternatively, a clinically relevant sodium deficit often progressively develops over several days of repeated sweat electrolyte losses that, to a lesser extent each day, exceed daily dietary salt intake. In these cases, the athletes are often surprised to suffer exertional heat cramps, given that they encountered no problems during the previous days in similar stressful conditions.

With an increasingly large free water loss, as a result of extensive continuous or repeated sweating, the athlete’s extracellular fluid compartment can become increasingly contracted.<sup>26,27</sup> Plasma volume, osmolality, and circulating electrolyte levels, however, may be maintained for a while during exercise,<sup>27</sup> as water shifts from the interstitial compartment to help “defend” central volume,<sup>28</sup> even when fluid intake is not adequate to completely offset sweat fluid loss. As an athlete rehydrates during or following activity, plasma volume is preferentially restored and the athlete often stops drinking, while renal water clearance increases before complete restoration of the interstitial spaces. Thus, the interstitial fluid compartment remains contracted. This is particularly evident when plain water or low-sodium fluid (as is characteristic of most sports drinks) is consumed alone.<sup>18</sup>

As a result of a contracted interstitial volume, axon terminals of selected motor neurons can be mechanically deformed, and surrounding extracellular ion and neurotransmitter levels can increase.<sup>12</sup> Consequently, some of these hyper-excitable nerve terminals may spontaneously discharge and initiate action potentials in the affected muscles.<sup>12,29</sup> The progression to widespread and debilitating exertional heat cramps often begins with barely detectable muscle fasciculations or “twitches” that the athlete typically only notices during a break in activity. However, these subtle signals of what is to come alerts the experienced athlete (with a history of exertional heat cramps) that s/he may only have 20 to 30 minutes before one or more muscle

areas begin to severely contract and continued activity becomes compromised. Another distinction of exertional heat cramps compared to fatigue-related muscle cramps is that heat cramps are characteristically more widespread and the high-voltage discharges often seem to “wander” from one area to another within large muscle groups, as adjacent and nearby muscle fibers and bundles alternately relax and contract.<sup>12,30</sup>

## Recovery and Return to Play

The recovery priority from a nutrient perspective for an athlete afflicted with exertional heat cramps is to replace and retain the salt (NaCl) and water that was lost via sweating. Further rehydration with water or low-sodium fluid alone is not going to make up the sodium deficit and effectively restore body fluid volume or the interstitial fluid spaces. In order to better retain (that is, lessen urine production) and sufficiently distribute the ingested water throughout the extracellular and intracellular fluid compartments during rehydration, enough NaCl should be provided concomitantly with the appropriate amount of fluid.<sup>19</sup>

Some have argued that EAMC are not associated with a clinically significant change in serum electrolytes.<sup>7</sup> Accordingly, sodium replacement is not warranted and should not be recommended as a strategy to avert or resolve muscle cramping. Notably, others have pointed out that a whole-body sodium deficit will usually not be detectable from a serum electrolyte measurement<sup>10,31</sup> and, in fact, athletes afflicted with exertional heat cramps will predictably present with normal or slightly elevated plasma or serum sodium levels.<sup>9</sup> An athlete with fatigue-related muscle cramping would not be expected to have a sodium deficit, since that is not likely the primary contributing etiological factor. For an athlete prone to exertional heat cramps, however, a careful assessment and comparison of typical dietary sodium intake and sweat electrolyte losses usually reveals a disparity between these two factors. A 24-hour urine collection may provide much more insight to the athlete’s sodium balance versus a single measure of sodium in the blood, especially if the blood sample is collected post-exercise.

An athlete can prevent exertional heat cramps from progressing and becoming more severe, especially if an appropriate response is initiated at the first sign of muscle twitching. Consuming 16 to 20 ounces (~0.5 L) of a sport drink (such as Gatorade®) with 0.5 teaspoon (3 g) of salt added and thoroughly mixed (consumed either all at once, if tolerable, or distributed over 10 minutes or so, with a little additional water) has been shown to be effective in relieving cramping or preventing muscle twitches from developing any further. Following this, an athlete can often continue participation for up to an hour or more, so long as additional fluid and NaCl is sufficiently and appropriately consumed during the rest of the activity. After competition or training, any remaining deficit in water and electrolytes needs to be replaced (see Table 1). Salt tablets (1 g of NaCl per tablet) can work similarly; but the tablets should be taken with plenty of fluid (eg, 3 crushed and dissolved tablets to 1 liter of water). However, if the muscle cramping is severe or accompanied by other more serious clinical conditions such as exertional collapse, heat exhaustion, or hyponatremia, then intravenous rehydration and sodium replacement with normal or hypertonic saline is usually necessary.

Quinine is not recommended as a recovery strategy for exertional heat cramps, as its effectiveness is questionable, and a number of adverse effects on performance and well-being (eg, decreased neuro-motor capacity, nausea, vomiting, disturbed vision, or other toxic symptoms) have been attributed to quinine intake.<sup>32</sup> Potassium-rich foods or supplements (as well as other mineral supplements such as magnesium and calcium) are not indicated and will not typically provide any relief.

If exertional heat cramps are managed effectively (as described above) at the earliest onset of symptoms, the athlete can often continue competing or training without interruption. The longer the delay in treatment, the more severe and widespread cramping of muscles becomes, and the longer it will take to for oral rehydration to be effective. Massage of the affected musculature and application of cryotherapy may temporarily relieve discomfort while the athlete waits for the ingested fluid and salt to be sufficiently emptied from the stomach and absorbed into circulation. If intravenous fluid is warranted, return to play (following medical clearance) could be more expedient, so long as there are no other noteworthy conditions or clinical complications.

## Prevention

Prevention of exertional heat cramps is simple and straight-forward and involves maintenance of sodium and fluid balance. Sweat sodium, chloride, and fluid losses incurred during training and competition need to be closely matched by daily salt and fluid intake in order to prevent an excessive sodium deficit and maintain sufficient fluid balance. The emphasis on *daily* salt and fluid intake is important. When sweating rate and sodium loss rate are low, athletes can often comfortably ingest enough fluid and electrolytes during exercise to maintain performance and avert a significant deficit in either water or sodium. Consuming a combination of water and a sport drink with a sodium concentration up to 50 mmol (1150 mg) per liter is effective and is well tolerated by athletes who are sweating in the heat. An example of such a sport drink mixture is ¼ teaspoon of salt to 32 ounces of Gatorade.<sup>®</sup> However, for athletes who sweat excessively and lose an extensive amount of sodium, it is often impossible to completely offset sweat fluid and electrolyte losses during the actual training session or competition.<sup>33</sup> In individuals who are prone to exertional heat cramps, sweat rate is often greater than 2.5 liters per hour and the concomitant sodium loss rate sometimes exceeds 3000 mg per hour.<sup>10</sup> An attempt to match these losses during training or competition with the same rates of fluid and electrolyte intake would likely be intolerable and impede performance and can result in “bloating” and nausea or more serious clinical complications such as vomiting or diarrhea.<sup>34, 35</sup> Accordingly, it is important for cramp-prone individuals to begin an event or training session well hydrated and without a significant sodium deficit. More commonly, however, cramp-prone athletes develop a water and sodium deficit from previous training or competition bouts and begin the next event already at risk.<sup>10,20</sup> After exercise, any remaining deficit in fluid and electrolytes should be ideally replaced before the next competition or training session. Scheduling multiple same-day training sessions or competition rounds (eg, during two-a-days or a soccer or tennis tournament) can hinder accomplishing this, however. Specifically, coaches and event administrators often don’t provide

sufficient recovery time between bouts; consequently, athletes are often forced to begin the next session in a water and electrolyte (primarily sodium) deficit. When trying to restore sweat electrolyte losses, certain dietary choices (eg, canned soup, tomato juice and sauce, cheese, salted pretzels and peanut butter, pizza, etc.) can enhance the rate of sodium replenishment.<sup>36</sup> It may be necessary to also periodically consume rehydration solutions with added salt (eg, 1.5 to 3.0 g of salt to 1 liter of sport drink) after exercise (see Table 1) to fully replace and retain the needed fluid and electrolytes.<sup>19</sup>

It's difficult to know how much water and sodium needs to be consumed before and after competition or training, without having some insight to typical sweat losses of fluid and electrolytes. Sweat fluid losses can be estimated from pre- to post-exercise changes in body weight (corrected for fluid intake and urine loss); however, determining sweat-related electrolyte needs requires sophisticated assessment tools and techniques that most athletes, coaches, and even physicians do not have convenient access to or knowledge of how to obtain, and thus such evaluations are limited in availability. Excessive visible dried salt on an athlete's skin or clothing provides a certain degree of insight. Some athletes' sweat will even appear white, thick, and "milky looking" when the salt content is extremely high. With a high frequency of exertional heat cramps, obvious extensive sweating, and visible signs of salty sweat, it is appropriate to experiment with gradual additional salt intake before, during, and after training sessions or competitive events. For a precise estimation of individual sweat electrolyte losses (and thus dietary needs), athletes would need to be evaluated at a university or medical facility that is capable of collecting and analyzing sweat electrolytes using standard clinical methods.<sup>10,19,20,37</sup>

## Conclusion

Exertional heat cramps can be resolved or prevented with appropriate fluid and salt intake (orally or intravenously). Athletes can generally return to play or training fairly promptly, so long as there are no other noteworthy conditions, clinical complications, or changes in vital signs or health status that compromise performance or athlete safety.

**Table 1 Suggested Fluid Mixtures for Exertional Heat Cramp-Prone Athletes Using Gatorade® and Table Salt (NaCl).\***

Purpose	Gatorade®	Table Salt*	Frequency or Number
Emergency (resolve twitches or cramps)	16 oz. (~0.5L)	0.5 tsp. (3 g)	One time—immediately
During activity	32 oz. (~0.9L)	0.25 tsp (1.5 g)	Up to 1 per hour
Recovery from activity	32 oz. (~0.9L)	0.5 tsp (3 g)	~One per kg post-exercise body weight deficit

\***Note.** With regular use of such salted mixtures, non-iodized salt is recommended to prevent excessive iodine intake.

## References

1. Parisi L, Pierelli F, Amabile G, Valente G, Calandriello E, Fattapposta F, Rossi P, Serrao M. Muscular cramps: proposals for a new classification. *Acta Neurol Scand.* 2003;107:176-186.
2. Bentley S. Exercise-induced muscle cramp: proposed mechanisms and management. *Sports Med.* 1996;21:409-420.
3. Jung AP, Bishop PA, Al-Nawwas A, Dale RB. Influence of hydration and electrolyte supplementation on incidence and time to onset of exercise-associated muscle cramps. *J Athl Training.* 2005;40:71-75.
4. Schwellnus MP, Derman EW, Noakes TD. Aetiology of skeletal muscle 'cramps' during exercise: a novel hypothesis. *J Sport Sci.* 1997;15:277-285.
5. Maughan RJ. Exercise-induced muscle cramp: a prospective biochemical study in marathon runners. *J Sport Sci.* 1986;4:31-34.
6. Schwellnus MP, Nicol J, Laubscher R, Noakes TD. Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. *Br J Sports Med.* 2004;38:488-492.
7. Sulzer NU, Schwellnus MP, Noakes TD. Serum electrolytes in Ironman triathletes with exercise-associated muscle cramping. *Med Sci Sport Exer.* 2005;37:1081-1085.
8. Benda C. Outwitting muscle cramps - is it possible? *Physician Sportsmed.* 1989;17:173-178.
9. Bergeron MF. Heat cramps during tennis: a case report. *Int J Sport Nutr.* 1996;6:62-68.
10. Bergeron MF. Heat cramps: fluid and electrolyte challenges during tennis in the heat. *J Sci Med Sport.* 2003;6:19-27.
11. Eaton JM. Is this really a muscle cramp? *Postgrad Med.* 1989;86:227-232.
12. Layzer RB. The origin of muscle fasciculations and cramps. *Muscle Nerve.* 1994;17:1243-1249.
13. Levin S. Investigating the cause of muscle cramps. *Physician Sportsmed.* 1993;21:111-113.
14. Miles MP, Clarkson PM. Exercise-induced muscle pain, soreness, and cramps. *J Sports Med Phys Fitness.* 1994;34:203-216.
15. Simchak AC, Pascuzzi RM. Muscle cramps. *Semin Neurol.* 1991;11:281-287.
16. Stamford B. Muscle cramps: untying the knots. *Physician Sportsmed.* 1993;21:115-116.
17. Mitchell JB, Phillips MD, Mercer SP, Baylies HL, Pizza FX. Postexercise rehydration: effect of Na<sup>+</sup> and volume on restoration of fluid spaces and cardiovascular function. *J Appl Physiol.* 2000;89:1302-1309.
18. Nose H, Mack GW, Shi XR, Nadel ER. Role of osmolality and plasma volume during rehydration in humans. *J Appl Physiol.* 1988;65:325-331.
19. Shirreffs SM, Maughan RJ. Volume repletion after exercise-induced volume depletion in humans: replacement of water and sodium losses. *Am J Physiol.* 1998;274:F868-875.
20. Stofan JR, Zachwieja JJ, Horswill CA, Murray R, Anderson SA, Eichner ER. Sweat and sodium losses in NCAA football players: a precursor to heat cramps? *Int J Sport Nutr Exerc Metab.* 2005;15:641-652.
21. Costill DL. Sweating: its composition and effects on body fluids. *Ann NY Acad Sci.* 1977;301:160-174.
22. Liu L, Borowski G, Rose LI. Hypomagnesemia in a tennis player. *Physician Sportsmed.* 1983;11:79-80.
23. Weller E, Bachert P, Meinck HM, Friedmann B, Bartsch P, Mairbaur H. Lack of effect of oral Mg-supplementation on Mg in serum, blood cells, and calf muscle. *Med Sci Sport Exer.* 1998;30:1584-1591.

24. Williamson SL, Johnson RW, Hudkins PG, Strate SM. Exertion cramps: a prospective study of biochemical & anthropometric variables in bicycle riders. *Cycling Sci.* 1993;5:15-20.
25. Allan JR, Wilson CG. Influence of acclimatization on sweat sodium concentration. *J Appl Physiol.* 1971;30:708-712.
26. Nadel ER, Mack GW, Takamata A. Thermoregulation, exercise, and thirst: interrelationships in humans. In: Gisolfi CV, Lamb DR, Nadel ER, eds. *Perspectives in Exercise Science and Sports Medicine: Exercise, Heat, and Thermoregulation*, Vol. 6. Dubuque, Iowa: Brown & Benchmark; 1993:225-256.
27. 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.
28. Nose H, Mack GW, Shi XR, Nadel ER. Shift in body fluid compartments after dehydration in humans. *J Appl Physiol.* 1988;65:318-324.
29. Jansen PH, Joosten EM, Vingerhoets HM. Muscle cramp: main theories as to aetiology. *Eur Arch Psychiatry Neurol Sci.* 1990;239:337-342.
30. Hubbard RW, Armstrong LE. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes*. Indianapolis, Ind: Benchmark Press; 1988:305-359.
31. Vaamonde CA. Sodium depletion. In: Papper S, ed. *Sodium: Its Biological Significance*. Boca Raton, FL: CRC Press; 1982:207-234.
32. McGee SR. Muscle cramps. *Arch Intern Med.* 1990;150:511-518.
33. Gisolfi CV. Is the GI system built for exercise? *News Physiol Sci.* 2000;15:114-119.
34. Binder HJ. Pathophysiology of acute diarrhea. *Am J Med.* 1990;88:2S-4S.
35. Zietsman J, Hay IT, Hansen JD, Dauth J, Dreyer MJ. Comparison of the sodium contents of six commonly recommended oral rehydration solutions. *S Afr Med J.* 1989;76:478-479.
36. Bergeron MF. Sodium: the forgotten nutrient. *Sports Sci Exchange.* 2000;13:1-4.
37. Bergeron MF, Maresh CM, Armstrong LE, Signorile JF, Castellani JW, Kenefick RW, LaGasse KE, Riebe DA. Fluid-electrolyte balance associated with tennis match play in a hot environment. *Int J Sport Nutr.* 1995;5:180-193.