Muscle Cramps during Exercise — Is It Fatigue or Electrolyte Deficit?

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BERGERON, M.F. Muscle cramps during exercise — is it fatigue or electrolyte deficit? *Curr. Sports Med. Rep.*, Vol. 7, No. 4, pp. S50–S55, 2008. *Skeletal muscle cramps during exercise are a common affliction, even in highly fit athletes. And as empirical evidence grows, it is becoming increasingly clear that there are two distinct and dissimilar general categories of exercise-associated muscle cramps. Skeletal muscle overload and fatigue can prompt muscle cramping locally in the overworked muscle fibers, and these cramps can be treated effectively with passive stretching and massage or by modifying the exercise intensity and load. In contrast, extensive sweating and a consequent significant whole-body exchangeable sodium deficit caused by insufficient dietary sodium intake to offset sweat sodium losses can lead to a contracted interstitial fluid compartment and more widespread skeletal muscle cramping, even when there is minimal or no muscle overload and fatigue. Signs of hyperexcitable neuromuscular junctions may appear first as fasciculations during breaks in activity, which eventually progress to more severe and debilitating muscle spasms. Notably, affected athletes often present with normal or somewhat elevated serum electrolyte levels, even if they are "salty sweaters," because of hypotonic sweat loss and a fall in intravascular volume. However, recovery and maintenance of water and sodium balance with oral or intravenous salt solutions is the proven effective strategy for resolving and averting exercise-associated muscle cramps that are prompted by extensive sweating and a sodium deficit.*

INTRODUCTION

Skeletal muscle cramps are a common affliction in sports and numerous other physical activities. Even highly fit athletes must sometimes succumb to debilitating cramping episodes, and some often compete with concern, knowing that these painful, involuntary muscle contractions can appear seemingly without warning or apparent cause.

As empirical evidence grows regarding the etiology and effective management (treatment and prevention) of exercise-associated muscle cramps, it is becoming increasingly clear that there are two distinct and dissimilar general categories of exercise-associated muscle cramps (when there is no other underlying pathology or abnormal condition present). First, skeletal muscle overload and fatigue from overuse or insufficient conditioning can prompt muscle cramping locally in the overworked muscle fibers (4,20,50). In contrast, extensive sweating and a consequent significant whole-body exchangeable sodium deficit can lead to more widespread muscle cramping, even when there is minimal or no muscle overload and fatigue (6,7,29,57). This latter type of muscle cramping has been referred to as exertional heat cramps, which causes some confusion. Although these cramps occur during or after exertion and concomitant extensive sweat losses, which is characteristic of heavy exercise in the heat, a hot environment is not a prerequisite, and afflicted athletes are not necessarily overheated. In fact, exertional heat cramps often occur in cool environments and even indoors, although considerable sweating still is present typically. Using the terminology "exercise-induced" or "exercise-associated," when referring to muscle cramps, does not distinguish sufficiently the nature of the muscle cramping, because these terms make no distinction with regards to the separate etiologies.

The information presented here supports the contention that there are two primary categories of exercise-associated muscle cramps — those related to muscle overload and fatigue and those skeletal muscle cramps associated with a sweat-induced sodium deficit (exertional heat cramps). While the muscle fatigue hypothesis (4,50) is a reasonable and perhaps valid explanation for some exercise-associated muscle cramps, this article emphasizes the underlying proposed mechanisms and evidence that distinguish exertional heat cramps from those muscle cramps that are prompted by activity-related muscle overload and fatigue. Accordingly, it is proposed that any discussion of exercise-associated muscle cramps specifically should elucidate which category is being referred to. This is critical in helping health care providers,

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coaches, and athletes appreciate the difference and select the most appropriate and effective treatment and prevention strategies.

TWO PERSPECTIVES: FATIGUE VERSUS ELECTROLYTE DEFICIT

Muscle Overload and Fatigue

During sports competition and training or a variety of other intense physical activities, repeated or extended loading on selected muscles can lead to muscle or tendon strain and local fatigue. The muscle fatigue hypothesis suggests that such a scenario can prompt an excitatory alteration (increase) in muscle spindle afferent activity and a concomitant decrease in Golgi tendon organ inhibition leading to abnormal α motor neuron control and sustained α motor neuron activity (19,34,49,50). That is, the neural mechanisms designed to inhibit muscle contraction, in response to muscle tension detected by the Golgi tendon organ, are disrupted or depressed. At the same time, enhanced excitatory activity from the muscle spindle triggers an intense and sustained involuntary muscle contraction that is unopposed by Golgi tendon organ control. Notably, shortened muscles with sustained contraction may be particularly vulnerable to such cramping, because the neuromotor end-plate depolarization threshold may be altered (45) and Golgi tendon organ inhibitory activity is normally depressed or negligible in a shortened position and cannot respond to the muscle tension (19). Predisposing risk factors associated with overload and fatigue-related muscle cramping might include older age, poor stretching habits, insufficient conditioning, cramping history, and excessive exercise intensity and duration, and related metabolic disturbances (4,49).

Protocols to induce muscle overload, fatigue, and localized cramping effectively take advantage of increased vulnerability of the muscle in a shortened position (with or without maximal voluntary contraction) and can readily produce severe acute involuntary muscle contractions and concomitant high levels of surface electromyogram (EMG) activity and amplitude (43,44,50). Such consistent and reproducible findings in the laboratory and with certain sports activities that similarly load the muscles (*e.g.*, plantar flexion of the ankle with contraction of the calf during swimming or high-intensity running) (58) and observed muscle fiber fatigue-induced changes in muscle spindle and Golgi tendon organ activity (19,34) strongly support the muscle fatigue hypothesis and proposed etiology related to abnormal α motor neuron control and activity originating at the level of the affected muscle fibers. Distinguishably, such muscle cramping remains localized to the overloaded and fatigued muscle group (such as with the triceps surae muscle), sometimes spreading slowly across the involved muscle region, but not jumping or wandering around a muscle (43).

Electrolyte Deficit

With exertional heat cramps, an athlete typically has been sweating extensively with appreciable sweat electrolyte losses as well, particularly sodium and chloride. Whether during a single long race, match, game, or training session or consequent to multiple same- or repeated-day exercise bouts, a sizeable whole-body exchangeable sodium deficit develops when sweat sodium and chloride losses measurably exceed salt intake (6,7,57). The deficit threshold required to prompt muscle cramping is not well described; however, an estimated sweat-induced loss of 20%–30% of the exchangeable Na^+ pool has been noted with severe muscle cramping (6,29). How readily this occurs depends upon sweating rate (10), sweat sodium concentration (typically 20–80 $mmol \cdot L^{-1}$) (7,12,28), and dietary intake (27). And with continuous physical activity over an extended period of time (*e.g.*, 3–4 h or more), a high sweat sodium concentration generally stays high, even as whole-body water and sodium deficits progressively increase. This is possible because sweating rate remains fairly consistent during such long-term activity and serum sodium concentration is typically maintained or elevated, along with potential changes in sweat gland function or sympathetic nervous system activity that would tend to increase sweat sodium concentration (33). Other electrolytes also are lost in sweat to a much lesser degree, and several of these (namely calcium, magnesium, and potassium) have been implicated falsely as the cause of muscle cramping during or after exercise when purported deficiencies are suspected (3,15, 23,24,31,56,62,63). However, exertional heat cramp-prone athletes characteristically develop a sodium deficit because their sweat sodium and chloride losses are not offset promptly and sufficiently by dietary intake (6,7,57).

To compensate for the loss in plasma volume during exercise, prompted in part by extensive sweating, water from the interstitial fluid compartment shifts to the intravascular space (13,35,39,48). As sweating continues, the interstitial fluid compartment becomes increasingly contracted (13). This can persist even after exercise, as sweating continues and body temperature returns to a pre-exercise level (39). Plasma osmolality and circulating electrolyte concentrations will be maintained or somewhat elevated during and after exercise as water shifts from the extravascular space to "defend" central volume and free water loss (primarily from sweating) continues, even as considerable sodium is lost via sweating (17,35,39,47,48). However, these electrolyte changes and consequent fluid shifts would be altered, depending upon the type of fluid and amount ingested (21,35,47,48). For a given level of dehydration, higher sweat sodium concentrations could be associated with a comparatively delayed mobilization of water from the interstitial compartment and less effective maintenance of plasma volume due to a lower plasma sodium concentration and associated osmotic drive (36). This may be why some athletes with very high sweat sodium concentrations and accompanying low sweating rates develop a significant whole-body sodium deficit and exertional heat cramps only after an extended period of exercise, sweat losses, and time. In contrast, for many athletes (even some who are heat acclimatized), the combination of a high sweat sodium concentration *and* high sweating rate arguably could accelerate a plasma volume loss (17), theoretically resulting in a more rapid shift of fluid from the interstitial compartment and onset of muscle cramping. These athletes would be

considered the "salty sweaters" (6,7,16,57), and they are the ones particularly at risk for readily developing exertional heat cramps. However, athletes with much lower sweat sodium concentrations readily can develop a sweat-induced sodium deficit as well, if their sweat rate is high enough or the duration of activity is extensive (7,9).

Consequent to a contracted interstitial compartment, certain neuromuscular junctions (especially first in the quadriceps or hamstring muscles) could become hyperexcitable by mechanical deformation and exposure of the unmyelinated nerve terminals and the post-synaptic membrane to increased levels of excitatory extracellular constituents such as acetylcholine, electrolytes, and exercise-related metabolites in the surrounding extracellular spaces, which could trigger the nerve fiber to fire or independently prompt an end-plate current and excitatory postsynaptic potential (22,55). Accordingly, there would be a greater risk for spontaneous discharge and initiation of action potentials in the affected muscle fibers. For example, Sjøgaard *et al.* (55) found that submaximal and maximal exercise prompted an increase in interstitial potassium concentration to a level that they believed would be sufficient to stimulate certain nerve endings. Similarly, elevated sodium surrounding the end-plates increases the likelihood of action potentials by reducing the required depolarization threshold (45). Surface EMG analysis further confirms that action potentials during muscle cramping can be initiated from the α motor neuron axon terminals (43). As more water is shifted from the interstitial compartment to the intravascular space, adjacent and other nerve terminals and post-synaptic membranes could be similarly affected and the cramping would spread or jump around (as is often observed) with various muscle fibers and bundles alternately contracting and relaxing (18), unlike overload and fatigue-related muscle cramps that remain localized.

The evolution of exertional heat cramps typically begins with fasciculations (small localized muscle contractions visible at the skin) that are barely detectable or unnoticed by the athlete except during breaks in activity (6,7,18,22). This is usually a sign that more severe and debilitating muscle spasms may be imminent in 20–30 min or so. Fasciculations and cramps often begin in the legs (6,7), which is not surprising given that the interstitial fluid compartment in the more highly active muscle group regions is likely to be challenged more strongly by concomitant osmotic and metabolic forces that help to maintain circulatory (13,35,39,48) and intracellular (35,40,55) volumes, respectively. With rehydration, plasma volume preferentially is restored (32,47,48), prompting a reduced drive to drink and increase in renal free water clearance often before complete restoration of the interstitial spaces; thus, the interstitial fluid compartment remains contracted, even though the athlete is no longer thirsty and increased urine production (especially after activity) deceptively suggests sufficient whole-body water recovery. This particularly occurs when plain water or low-sodium fluid is consumed alone (38).

Those who do not acknowledge the relationship between a whole-body exchangeable sodium deficit and muscle cramping have argued that serum electrolyte concentrations (most notably sodium and chloride) are not associated with exercise-associated muscle cramps (51,58,59). However, others have indicated and consistently emphasized that a whole-body exchangeable sodium deficit usually is not detectable from measuring serum electrolytes (5–8,47,60), especially after extensive exercise and significant sweat losses when circulating sodium concentration is predictably normal or somewhat elevated (6,33,35,39,48). Accordingly, postexercise serum sodium concentration and osmolality are more of a reflection of free water gain or loss and fluid compartment shifts versus electrolyte losses, and thus should not be used to indicate the presence or absence of a whole-body exchangeable sodium deficit. Any determination of sodium status should be based minimally upon a suitable estimate of sweat sodium loss compared with sodium intake. Authors critical of muscle cramping prompted by a sodium deficit have not done this, and in fact, did not monitor directly their subjects' sweat sodium losses or dietary intake in these studies (51,58). It is interesting to note that it is acknowledged that a sodium deficit and changes in serum electrolytes can be associated with and result in "generalized skeletal muscle cramping" (49,50), and in separate studies, statistically significant lower postrace serum sodium concentrations have been reported in cramping athletes compared with a noncramping control group (51,58), similar to other investigators who have observed significant reductions in serum sodium and chloride concentrations in cramping subjects (25,29). These statements and findings are not consistent with the argument against exercise-associated muscle cramps being linked to a systemic abnormality of fluid balance and consequent fluid compartment volume and electrolyte changes after exercise-induced dehydration and a whole-body exchangeable sodium deficit.

RECOVERY AND PREVENTION

Muscle Overload and Fatigue

Overload and fatigue-related muscle cramps remain localized to the overworked muscle(s), and these cramps often can be resolved readily by passive stretching, massage, active contraction of the antagonist muscle group, or icing of the affected muscles. Lowering overall exercise intensity and altering the load on the distressed muscle(s) can be effective as well. Preventive measures include reducing training and competition intensity and duration, as well as improving conditioning and range of motion through appropriate and regular individualized progressive fitness and stretching programs. Adjustments to equipment configuration and selection (*e.g.*, bicycle seat and handle position, shoes), biomechanics, and relaxation techniques may also help to avert or delay fatigue-induced muscle cramping (4,23,30,42,50,54,56).

Electrolyte Deficit

At the first sign of muscle twitches or mild exertional heat cramps, a prompt oral bolus of a high-salt solution (*e.g.*, 0.5 L of a carbohydrate-electrolyte drink, with 3.0 g of salt added and thoroughly mixed, consumed all at once or over 5–10 min) has been a proven effective field strategy in

relieving cramping or preventing muscle fasciculations from developing into a more severe and debilitating condition (6). Massaging and applying ice to the affected area can assist in relaxing the muscles and relieving some of the discomfort while waiting for the ingested fluid and salt to be absorbed adequately into circulation, although the effects of an oral salt solution often can be seen in just a few minutes (6), as the ingested beverage is rapidly absorbed (14) and plasma sodium levels quickly begin to change (21). After such a high-salt solution bolus, athletes can often promptly continue and immediately resume training or competition effectively without muscle cramping or twitching symptoms for an hour or more (6), while additional lower-sodium fluid is consumed appropriately at subsequent regular intervals. Continuation of activity at the same intensity likely would not be possible if muscle overload or fatigue was the sole or primary contributing factor to the muscle cramping. After the training or competition session, any remaining body water and electrolyte deficits need to be replaced with a particular emphasis on salt intake, in order to help retain (52) and distribute the ingested fluid, so that all fluid compartments are restored sufficiently (32). Intravenous rehydration with normal or hypertonic saline may be required, if muscle cramping is severe or accompanied by a more serious clinical condition such as hyponatremia (25,37,41,53). Potassium-rich supplements or foods or other mineral supplements such as calcium or magnesium are not indicated and typically will not provide any relief of exertional heat cramp symptoms (6,16).

Maintenance of hydration *and* sodium balance is the proven effective prevention strategy for averting exertional heat cramps in athletes and workers during training, competition, and other physical activities (6,7,11,16,18, 57,61). Ideally, sweat sodium, chloride, and water losses incurred during competition or training bouts should be offset sufficiently during activity to avoid measurable deficits and more closely matched by overall daily salt and fluid intake. Although the emphasis is on *daily* salt and fluid intake, athletes who sweat considerably (*e.g.*, >2.5 L and 2500 mg of Na^+ per hour) are often not able to avoid large water and electrolyte deficits during activity and completely offset these nutrient losses between multiple same-day sessions or day-to-day with most commercial carbohydrate-electrolyte drinks and meals alone while they compete or train, especially if they are following low-salt dietary recommendations (46) or even a more typical diet (1). Particularly with a short recovery time between activity bouts (sometimes only 1 h between matches in junior tournament tennis, for example) (2) and extensive post-play electrolyte deficits (6,7), meals are often not a practical or sufficient method for rapidly replacing enough sodium. Accordingly, during activity and between games, matches, or training sessions, these athletes (especially those who are prone to exertional heat cramps) must be deliberate in consuming a high-salt solution at regular intervals (5) along with ingesting additional fluid and electrolytes (emphasizing salt intake) to make up the difference, so as to prevent progressive significant fluid and whole-body sodium deficits from developing and to ensure sufficient restoration of all fluid compartments before the athlete takes to the field or court again. Salt tablets can be effective, so long as they are consumed with plenty of water (*e.g.*, for 1 g of NaCl per tablet, three crushed and dissolved tablets to 1 L of water). Other specific dietary selections and strategies have been presented elsewhere (6–8). For the athlete attempting to reverse a pattern of exertional heat cramping, it is often not necessary to increase fluid intake; in fact, sometimes it's essential to decrease fluid intake during and after activity for those who are overdrinking (especially those who are consuming too much low- or no-sodium fluid). The key is to increase sodium intake to more closely match individual sweat sodium losses, so that the appropriate amount of ingested fluid is better retained and distributed to all fluid compartments (26,27,47,48). The result is more *complete* rehydration.

Differential Diagnosis

For the clinician or other health care provider attending to an athlete afflicted with muscle cramping during training or competition, in an effort to determine the appropriate treatment, it is important to consider the clinical signs and symptoms, as well as the surrounding setting and circumstances and time course leading up to the onset of cramps. Without the advantage of a complete physical, health history, or individual test results, and assuming there is no other underlying pathology or ischemic disorder, certain distinguishing characteristics can help in the immediate onsite diagnosis. Comparatively sudden-onset exertion-related muscle cramping that is localized (*e.g.*, affecting solely the calf), constant, asymmetric, and responsive to passive stretching and massage is highly likely to have been prompted by muscle overload and fatigue, whereas reported or observed fasciculations or slight cramping that progressively developed over a longer period of time to more severe and widespread (often bilaterally) intermittent muscle spasms suggest exertional heat cramps. Profuse sweating and a salt residue on the skin or clothing (although not always visible) and other signs and symptoms of dehydration further implicate the presence of a significant water or sodium deficit. If the athlete is treated for exertional heat cramps with an oral high-salt solution or intravenously, massage and icing can still be applied to assist in relaxing the muscles and relieving some of the spasms. It is also important to recognize that an athlete can experience both types of muscle cramping concomitantly; however, the underlying causes and effective treatments of these separate problems are different.

CONCLUSION

Definitive studies on the precise mechanisms underlying fatigue-related alterations in muscle spindle and Golgi tendon organ afferent activity and investigations to confirm the contributory presence of a whole-body exchangeable sodium deficit, contracted interstitial fluid compartment, and hypersensitive neuromuscular junctions with sweat-induced muscle cramping during exercise have not yet been performed. However, sufficient laboratory and clinical empirical evidence supports both perspectives discussed

here (a whole-body exchangeable sodium deficit *and* muscle overload and fatigue) as being valid underlying bases for exercise-associated muscle cramps with distinct and dissimilar contributing factors and mechanisms. Thus it is important to advance beyond discussions and arguments intended to favor one theory on exercise-associated muscle cramps over another. Investigators and clinicians should acknowledge the evidence supporting each of these two primary categories of exercise-associated muscle cramps that clearly seem to have separate etiologies and distinct management strategies. However, additional research is needed to further elucidate and develop a better understanding of the fatigue hypothesis and muscle cramps related to extensive sweating and an exchangeable sodium deficit to enhance treatment and prevention strategies.

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