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Cramping in Sports: Beyond Dehydration

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ABSTRACT

EXERCISE-ASSOCIATED MUSCLE CRAMPS (EAMC) ARE A COMMON, YET POORLY UNDERSTOOD PHENOMENON IN ATHLETIC COMPETITION. PREVAILING WISDOM SEEMS TO SUGGEST THAT DEHYDRATION AND ELECTROLYTE DISTURBANCES ARE THE MAIN CAUSE OF EAMC; HOWEVER, ON REVIEW OF THE AVAILABLE LITERATURE, THIS DOES NOT SEEM TO BE THE CASE. THIS ARTICLE PRESENTS AN EXAMINATION OF THE PAST AND PRESENT KNOWLEDGE ON THE SUBJECT OF EAMC IN THE HOPES OF FACILITATING A GREATER UNDERSTANDING SURROUNDING THE POSSIBLE CAUSES OF CRAMPS DURING EXERCISE. A VIDEO ABSTRACT OF THIS ARTICLE CAN BE FOUND IN SUPPLEMENTAL DIGITAL CONTENT 1 (SEE VIDEO, HTTP://LINKS.LWW.COM/SCJ/A151).

Exercise-associated muscle cramps (EAMC) are a painful condition that often hinder athletic performance (3,5). Despite a high prevalence of EAMC in both athletes and recreationally active individuals (5), surprisingly little research exists to explain the possible cause(s) of cramps during exercise. Scholars generally agree that cramps can be defined as painful, spasmodic, involuntary contractions of skeletal muscle during or immediately after exercise (6). Researchers similarly appear to agree that a true cramp is caused by explosive hyperactivity of motor nerves involving a combination of factors relating to spinal disinhibition and the abnormal excitability of motor nerve terminals (1–3,5,8,10). Although a high degree of consensus exists with respect to the definition of EAMC, the exact reason why cramps often occur during exercise continues to elude researchers. Dehydration and electrolyte depletion have long been considered the primary cause of EAMC (9,11); however, the body of available literature does not seem to support this position. Neurological factors relating to abnormal functioning of feedback mechanisms from within a working muscle as a result of fatigue have at present the greatest volume of empirical support. The purpose of this review is to examine current theories about EAMC with respect to the body of available literature. In the summary, practical implications for practitioners will be discussed.

A number of medical conditions are known to cause cramps; however, the majority of the conditions are rare (9). Athletes who experience EAMC are not, on the whole, believed to be afflicted by one of these conditions (9). As such, this review will focus exclusively on cramps that occur in the context of athletic endeavor.

FACTORS KNOWN TO INCREASE THE RISK OF EXPERIENCING EXERCISE-ASSOCIATED MUSCLE CRAMPS

The causes of EAMC have been proposed as the (a) fatal accumulation of contractility-impairing metabolites, (b) electrolyte depletion, (c) loss of fluid volume, and (d) stresses associated with extreme environmental conditions, particularly extreme heat and cold (10). Examination of available literature does not, however, support the possibility that these factors are primary causes of cramps during exercise. Although the cause of EAMC is still under investigation, a number of factors have been shown to increase the prevalence of EAMC. Having a history of cramping, and competing at a faster race pace than training pace, was identified in 2 studies (9,11) to positively correlate with an increased risk of developing EAMC. Cramps seem to be more prevalent in muscles that cross 2 joints (1), and individuals who cramp have been shown to have a lower threshold frequency for cramping (15 Hz) than non–cramp-prone individuals (25 Hz) (4). Threshold frequency is the minimum frequency of excitation needed to stimulate a cramp. The fact that cramp-prone individuals have lower threshold frequency for the onset of EAMC supports the validity of a neurological origin of cramps, to be explained shortly.

HISTORICAL PERSPECTIVES

The prevalence of EAMC has been shown to be as high as 37% in some populations (1). Accordingly, the

KEY WORDS:
exercise-associated muscle cramps; dehydration; muscle fatigue; cramps

possibility of cramping is a common concern to active individuals, particularly those involved in endurance events such as the marathon. Considering the prevalence of EAMC, surprisingly little research exists on the subject, although the topic was first investigated by researchers over 100 years ago (1). Numerous theories as to the cause of EAMC have passed into and out of favor with researchers over the last century. One such hypothesis is that cramps have a "psychosomatic origin" whereby the cramps are brought on by states of irritability, nervousness, or hysteria (1). A second historical perspective suggests a vascular origin of EAMC and was suggested in the late 1920s (9,11). The vascular theory of EAMC origin hypothesized that isometric contractions of a muscle that is already maximally shortened would stretch fascia to the point where blood flow to working muscles becomes completely occluded (10). Pain associated with EAMC was suggested by proponents of the vascular origin of cramping to be due to afferent signaling to the central nervous system (CNS) from pain receptors and chemoreceptors due to the inhibited blood flow. Accumulation of lactic acid was theorized to occur shortly after complete occlusion of muscle tissue, which was thought to affect contractility of muscle tissue, and would be followed shortly by a persistent contraction of the muscle. The mechanisms by which static stretching was hypothesized to alleviate cramps was by loosening fascia, which would subsequently restore blood flow to working muscles. Stretching would not only relieve the pain caused by occlusion but would also restore muscle function by clearing contractility-impairing metabolites from tissue (10).

PHYSIOLOGY OF SKELETAL MUSCLE RELAXATION

An understanding of the physiological mechanisms associated with muscle relaxation is necessary to fully grasp cramping during exercise. Cramps are generally understood to be the result of dysfunction in 1 or more of the following elements (10). The first principle to understand is that ATP is needed for 2 processes in the relaxation of skeletal muscle: uncoupling of myosin heads from actin-binding sites and the active transport of calcium into the sarcoplasmic reticulum. Dysfunction with either process will result in permanent contraction.

The second mechanism that must function properly for skeletal muscle to relax is the normal transport of free calcium ions from the sarcoplasm to the sarcoplasmic reticulum. Reduction in the rate of calcium transport back into the sarcoplasmic reticulum will result in a persistent state of contraction because actin-binding sites will be continually exposed. The third critical factor necessary for relaxation of skeletal muscle is the presence of a normal muscle resting membrane potential. Continual depolarization of the cell membrane will cause persistent signaling to muscle cells to contract. Normal motor endplate function is another critical factor necessary for muscle relaxation and is similar to the need for a normal membrane resting potential, and impaired clearance of neurotransmitters in the synaptic cleft will cause persistent depolarization of the muscle cell. The presence of normal spinal reflex activity is the most relevant criteria for muscle relaxation to EAMC; normal control of the alpha motor neuron is critical for effective relaxation (10), meaning that excitatory input from the motor cortex, extrapyramidal cells, and muscle spindle must be decreased before relaxation can occur. Prevalent theories on EAMC involve a potential dysfunction in afferent feedback from Golgi tendon organs (GTOs), leading to increased activation of the motor neuron (1,3,5,8,10).

RESEARCH

TRADITIONAL THEORIES AS TO THE CAUSE OF EXERCISE-ASSOCIATED MUSCLE CRAMP

A connection between dehydration, electrolyte depletion, and cramping was made over 100 years ago from the observation from miners that cramps almost always occurred in the presence of profuse sweating (6,9). The theory that cramping is due to dehydration and electrolyte imbalances is rooted in these early reports, and subsequent research seems to rely extensively on case reports and anecdotal evidence (8). In contrast to the limitations imposed on the dehydration and electrolyte theories of EAMC due to methodological insufficiencies, 4 prospective cohort studies have produced reliable evidence from well-controlled experimental designs to refute the theory that dehydration and electrolyte imbalances cause EAMC.

Two studies (9,11) investigated the incidences of cramping during an Ironman triathlon (n = 210) and a 56-km ultramarathon (n = 49). In both studies, competitors who experienced cramps during or immediately after the race showed no significant differences in final serum electrolyte concentration compared with participants who did not experience cramping. Similarly, the average body-weight loss for a competitor over the course of the race (an indicator of dehydration) was not significantly different between individuals who did experience EAMC from those who did not. The results of these 2 studies support the position that dehydration and electrolyte depletion are not the primary causes of EAMC.

Although sodium and hydration decreases were not observed to correlate with EAMC, Schwellnus et al. (9,11) did identify 2 potential triggers for cramping during competition. Athletes who experienced EAMC both predicted and displayed significantly faster completion times relative to the competitors who did not experience EAMC, despite both groups reporting similar preparation and performance histories. The observation that athletes who experienced EAMC both predicted and displayed significantly faster completion times relative to the competitors who did not experience EAMC led investigators to conclude that a fast race pace, relative to average training velocity, is a significant risk factor for EAMC. Second, the athletes

who experienced EAMC reported a significantly higher history of EAMC (82.9%) over the past year compared with the competitors who did not experience cramps during the race (45.5%). The finding that the athletes who experienced EAMC reported a significantly higher history of EAMC compared with the competitors who did not experience cramps led the investigators to conclude that fast race times (relative to training pace), thus indicating a higher intensity of effort and a history of EAMC are 2 major predictors of cramping during exercise. Schwellnus also concludes that electrolyte depletion and dehydration do not lead to EAMC.

Schwellnus et al. (9) investigated incidences of EAMC during a 56-km ultraendurance race and a similar study design to the researcher's investigation of cramping during an Ironman triathlon (11). In addition to concluding that a faster race pace relative to training and a history of EAMC are significant risk factors for EAMC, Schwellnus et al. (9) identified (a) history of collapse, (b) increased stretching before competition, (c) increased race duration, and (d) greater quadriceps muscle pain after race as additional risk factors for EAMC. Beginning the race with a subclinical degree of tissue damage in the quadriceps (as a result of insufficient tapering before race day) was also suggested as a risk factor for EAMC, likely because of the fact that muscle fatigue likely occurred earlier in the race compared with if the competitor's legs were "fresh." Starting an endurance race with subclinical amounts of tissue damage is a significant concern because, as will be further explained, muscle fatigue is currently understood to be a major factor involved with EAMC (1,3,5,8,10). To date, however, no study has directly investigated the possibility of subclinical damage to working muscle at the beginning of a race (due to insufficient tapering) as a primary cause for EAMC. The effect of subclinical tissue damage on the onset of EAMC has only been suggested to impact on whether or not EAMC occur, through an application of established points of physiological theory. Further research on the topic is recommended.

No study that features the conclusion electrolyte/dehydration of central mediators EAMC has proposed a mechanism to explain how electrolyte and fluid replacement may prevent cramps during exercise (8). Schwellnus (8) seems to be the first researcher to specifically identify the fact that no study featuring the conclusion that electrolyte and dehydration theories of EAMC has proposed a mechanism to explain how electrolyte and fluid replacement may prevent cramps during exercise. Schwellnus attempts to explain the sequence of events which would occur if proponents of the electrolyte depletion theory, specifically the theory that excessive sodium loss in sweat without a concomitant change in serum electrolyte concentration, are correct (Figure 1). Schwellnus states that "because the sodium concentration of sweat is always hypotonic (to blood), a significant loss of sodium through sweat can, therefore, only occur if accompanied by large loss of fluid. This would mean that dehydration would accompany significant sodium losses in EAMC" (p. 402). The relevance of the fact that dehydration would accompany significant sodium losses in EAMC is that identification of either water or electrolyte loss as the cause of EAMC is not possible.

Contraction of the extracellular fluid compartment is the only physiological mechanism to have been directly suggested to be impacted by sodium losses and dehydration; however, the hypothesis was made on the basis of theory alone, without any supporting evidence (Bergeron, 2003 as cited in Schwellnus (8)). Bergeron hypothesizes that the subsequent loss of interstitial volume due to dehydration leads to a deformation of nerve endings, which when coupled with an increased intracellular concentration of ions and neurotransmitters leads to a hyperexcited state in the muscle fiber, leading to possible spontaneous discharge. Schwellnus (8) further explains that no studies exist in which increased serum sodium concentrations (above serum sodium concentrations observed in noncramping control groups) were observed. Proponents of the electrolyte theory suggest stretching as a way to alleviate EAMC but do not suggest why stretching may be effective in alleviating cramps within the context of fluid or electrolyte balance. From a strictly analytical perspective of the mechanisms by which electrolytes and fluid replacement are thought to alleviate cramping, stretching should have no effect in alleviating cramps.

Some clinical data do exist to show that altered serum osmolality and electrolyte concentration can cause cramping at rest in individuals with specific conditions (8). However, data from 2 respected studies (9,11) did not identify any of such clinical conditions in the athletes who experienced EAMC in their investigation of cramping during completion in an ultramarathon and Ironman triathlon. Because of the fact that the clinical conditions known to cause cramps at rest are rare and obviously debilitating (e.g., amyotrophic lateral sclerosis (8)), the investigators were confident that undiagnosed clinical conditions were not responsible for incidences of EAMC observed during the endurance events. Considering the absence of an explanation to explain the mechanisms behind why dehydration and electrolyte deficiencies may lead to EAMC, in addition to findings from Schwellnus et al. (9,11) that found competitors who did experience EAMC during competition did not demonstrate significant differences in final serum sodium concentration and total fluid loss, the likelihood that dehydration and electrolyte loss are the primary causative factor for EAMC seems to be speculative.

Based on the body of available literature, the common belief that electrolyte depletion causes cramps during exercise does not seem to be valid (1–3,8,10). The results of an investigation by 1 research team (2) do, however, suggest that supplementation of a carbohydrate/electrolyte beverage before and during exercise may prolong the time until

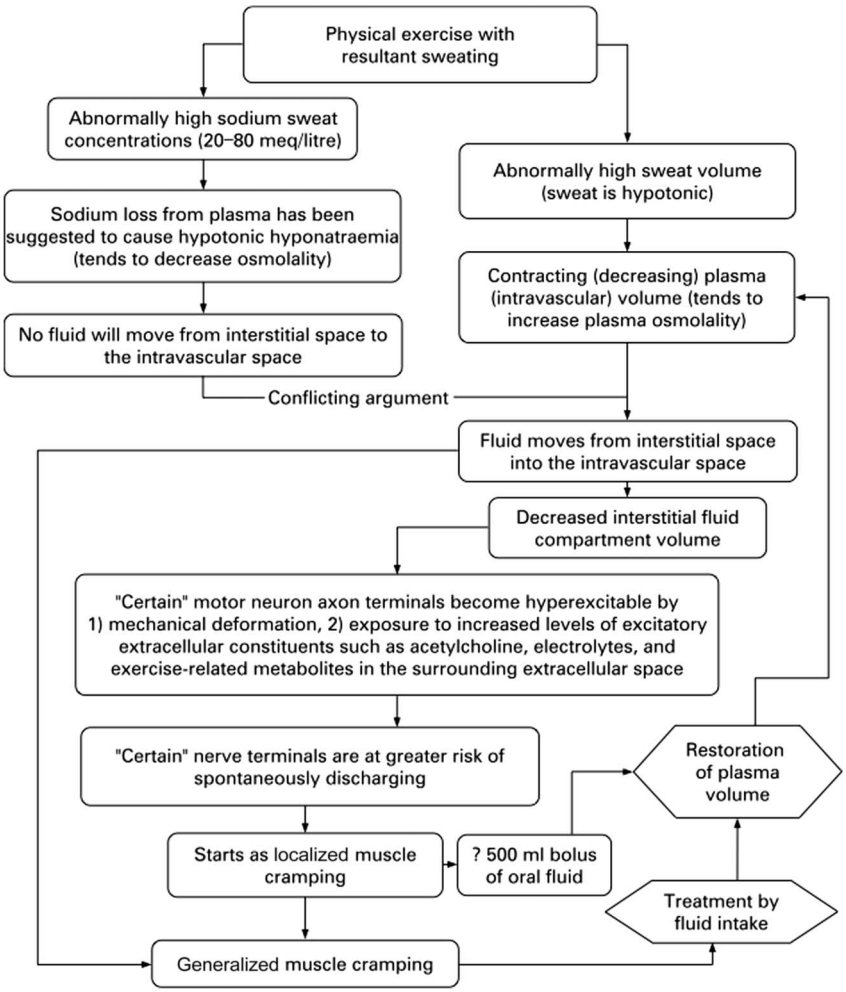

Figure 1. The "electrolyte depletion" hypothesis for the development of Exercise Associated Muscle Cramping (8).

EAMC onset. Investigators recruited 13 college-aged men to complete 2 protocols designed to induce cramps in calf muscles with a counterbalanced repeated-measure design. Participants began both trials in a euhydrated state and completed the same fatiguing protocol consisting of intervals of incline treadmill walking on the toes (heel cannot touch the treadmill), interspersed with intervals of standing calf raises, jumping rope, modified depth jumps, recumbent cycling, and kick turns. Participants were not allowed to consume any fluids during 1 trial (hypohydrated protocol) and were permitted to drink a carbohydrate/electrolyte solution (Gatorade) ad libitum during the other trial (carbohydrate/electrolyte protocol). Nine participants (69%) experienced EAMC during the carbohydrate/electrolyte protocol and 7 (54%) participants experienced EAMC during the hypohydrated protocol. An important detail to note from Jung et al. is that all subjects who experienced EAMC during the hypohydrated trial experienced EAMC during the carbohydrate/electrolyte trial. The most critical finding, however, is that the mean time to onset of EAMC was significantly greater (150%, 36.8 ± 17.3 minutes, compared with 14.6 ± 5.0 minutes, $p < 0.01$) in the carbohydrate/electrolyte solution protocol. Investigators concluded that dehydration and electrolyte depletion are not likely to be the main cause of EAMC, although supplementation of carbohydrates and electrolytes before and during exercise may prolong the time until EAMC occurs. The authors additionally suggest that because of the intensity of their exercise protocol, the carbohydrate and electrolyte supplement may not have had enough time to exert an effect and prevent EAMC. Further research using more participants and a less-intense experimental protocol was suggested. The authors additionally concluded that the factors contributing to EAMC are likely multifactorial and reducing the causes of EAMC to a single mechanism may be problematic. A limitation to the study, however, is that because carbohydrates, fluids, and electrolytes were administered in the same beverage, no inferences can be made as to whether or not it was the carbohydrates, electrolytes, fluid, or some combination of the 3 that was responsible for extending the time until onset of EAMC.

NEUROLOGICAL STIMULUS FOR EXERCISE-ASSOCIATED MUSCLE CRAMPS

Golgi tendon organs and muscle spindles. A brief review of GTOs and muscle spindle fibers is helpful to ensure readers are sufficiently versed in necessary elements of exercise physiology. Muscle spindles are sensory receptors in the belly of muscle tissue that respond to stretch in the muscle by reflexively contracting it to prevent hyperextension (7). GTOs are found at the musculotendinous junction and respond to high degrees of force in the muscle by inhibiting further contraction (7). GTO can be thought of as being similar to the emergency stop button on a treadmill; beyond a certain intensity, GTOs activate and shut down further activity in the muscle to prevent injury. With respect to theories of EAMC, it is important to note that GTO and muscle spindles have separate neural feedback pathways to the motor control unit in the spine.

NEURAL STIMULUS

In light of limitations to the theory that dehydration and electrolyte depletion cause EAMC, researchers have begun to investigate the possibility that EAMC are caused by

a dysfunction at the neurological level. Schwellnus (10) was one of the first investigators to suggest that EAMC are caused by abnormalities at the spinal level. He states that "EAMC are caused by sustained abnormal spinal reflex activity, which appears to be secondary to muscle fatigue (Figure 2). Local muscle fatigue is, therefore, responsible for increased muscle spindle afferent and decreased Golgi tendon afferent activity" (p. 407). Schwellnus furthers the hypothesis that EAMC have a neural origin from findings that show muscles that cross 2 joints (e.g., the gastrocnemius) can be more easily placed in a shortened position, resulting in decreased Golgi tendon activity due to lowered levels of tension at the musculotendinous junction. Second, prolonged abnormal functioning of reflex activity in cramping muscles would provide an explanation for both the data showing increased electromyographic (EMG) activity in cramping muscles (4,6) and the observation that stretching attenuates EAMC (likely through reciprocal reflex inhibition and stimulation of GTO afferent signaling (1,3,5,8,10)).

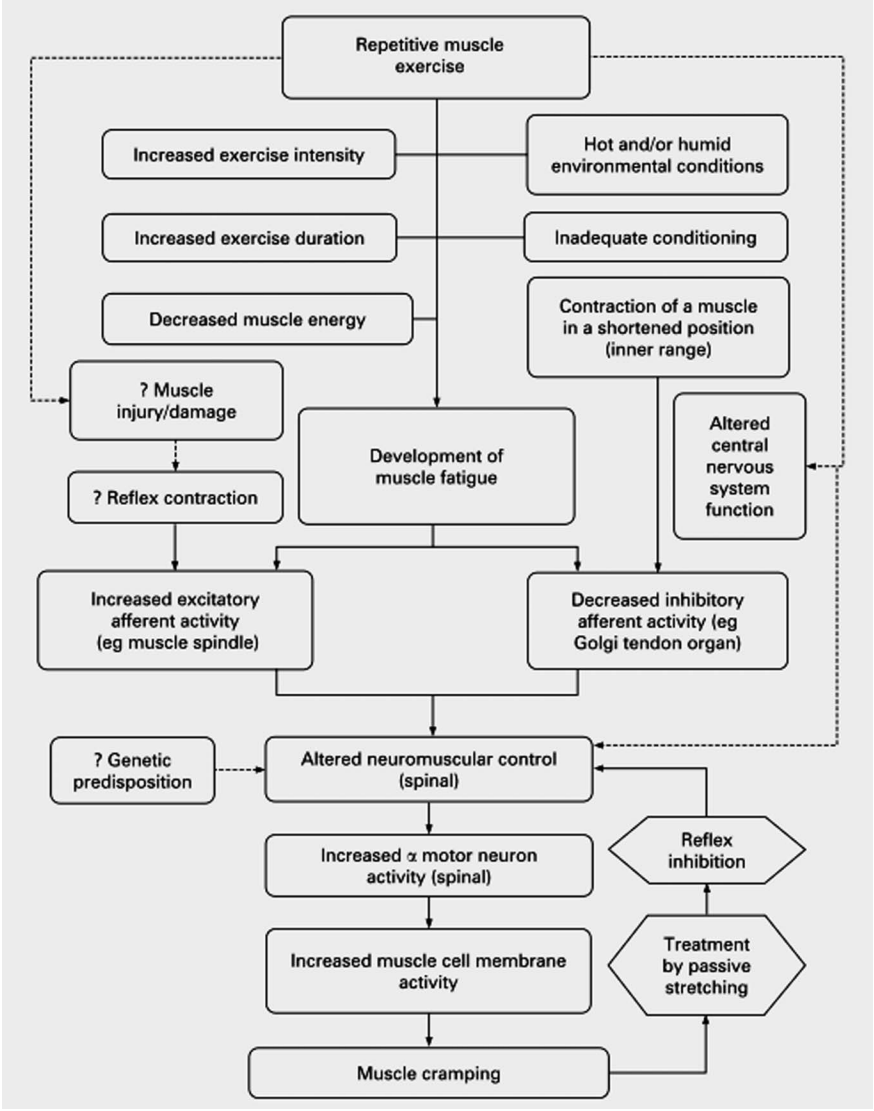

Figure 2. The "altered neuromuscular control" hypothesis for the development of Exercise Associated Muscle Cramping (8).

Schwellnus can be credited with both reviving and furthering research on the topic of the neural origins of EAMC. The concept was, however, originally suggested in 1910 by a German physician who suggested, on the basis of his medical experience treating individuals suffering from polyneuritis, that damage to peripheral motor neurons, especially motor nerve terminals, causes EAMC (4). The theory is plausible in part because of the fact that EAMC are associated with muscle fatigue in the late stages of athletic endeavor (9,11), when structural damage to working tissue is likely to have occurred. Norris et al. (1957; as cited in Schwellnus et al. (10)) added to the credibility of the theory that EAMC have a neural origin with his observation that experimentally induced cramps could be terminated by passive stretching of the muscle, possibly through activation of the reciprocal inhibition reflex of antagonist muscle groups. The observation led Norris et al. to propose excitation of motor neurons in the spinal cord as the main mechanism causing EAMC.

Schwellnus' interest in the possible neurological origins of EAMC was sparked by an investigation into cramping during either training or competition using 536 marathon runners, who reported experiencing EAMC (8). Specifically, the finding that 60% of respondents indicated that muscle fatigue was associated with, or precipitated, the onset of EAMC was recognized (8). Support for a neurological basis of EAMC comes from the fact that cramps can be reliably induced through either voluntary contractions or stimulation of the motor nerve (4,5). Observations from animal experimentation on spinal reflex activity during muscle fatigue have been used to strengthen the argument that EAMC have a neural origin occurring subsequent to fatigue (3–6,9,11).

PERIPHERAL VERSUS CENTRAL MECHANISMS

The presence of a neurogenic stimulus for EAMC is generally accepted as valid by researchers (1,3,4,8,10); however, debate exists about the origin

of the neurogenic stimulus itself. Proponents of the Central Origin Theory suggest that cramps originate from the hyperexcitability of motor neurons (4), whereas proponents of the Peripheral Origin model contend that cramps are the result of spontaneous discharges of the motor neurons due to abnormal excitation of the unmyelinated terminal branches of motor axons (1,4–6).

According to Schwellnus (11), studies by Hutton and Nelson in 1985 and 1986 present evidence that the onset of muscle fatigue can lead to altered function in the spinal neuromuscular control mechanisms responsible for contraction and relaxation. Muscle fatigue has been shown in animal models to disrupt proper functioning of peripheral muscle receptors by causing an increase in the firing rate of type Ia and II muscle spindle afferents, and a decrease in type Ib afferent activity from the GTO. Recall that afferent signals from muscle spindles (type Ia and type II) cause a reflexive contraction in the muscle (to prevent hyperextension injures) and GTOs signal the release of muscular tension (through type IIb afferent neurons). The result of increased afferent activity from muscle spindles and decreased input from Golgi tendon is that the signal to relax is not received in the motor neuron soma, meaning a positive-feedback loop of contraction, followed by afferent muscle spindle signaling, which signals further contraction, occurs. The fact that passive stretching (activation of GTOs) alleviates cramps and that cramps occur more frequently in shortened muscles and in muscles that cross 2 joints (due to GTOs being in an inactive state) supports the Central Origin hypothesis (1,3,5,8,10).

The idea that EAMC have a peripheral origin that is caused by spontaneous firing of unmyelinated axon terminals is central to the Peripheral Origin theory, which proposes EAMC are caused by dehydration and decreases in serum electrolyte level. EAMC are believed to be caused by dehydration and decreases in serum electrolyte level in the Peripheral Origin Theory because spontaneous axonal discharges (cramps) are believed to occur as a result of damage to muscle tissue and/or because of an increase in intracellular metabolite and neurotransmitter concentrations as a result of fluid loss from intense sweating (1). Acknowledgment of the fact that both the peripheral and central theories of neural cramping theorize that muscle damage plays a role in how EAMC are stimulated is necessary.

The results of a recent study by Minetto et al. (5) seem to favor a central origin of fatigue. Investigators electrically stimulated muscle contraction in the abductor hallucis muscle of healthy subjects in 2 conditions: with and without a peripheral nerve block. The peripheral nerve block was placed on the motor neuron leading to the muscle, and electrical stimulation occurred distal to this block. The "blocked" condition can be understood to mimic peripheral mechanisms leading to EAMC. In the "intact" condition, no nerve block was used. Stimulation was applied to afferent neurons from the muscle spindles, and when electrically stimulated, a cramp occurred through a positive-feedback loop from the intact motor unit pathway. The results of the study showed that the intensity and duration of cramps elicited in the intact group were of longer duration and higher intensity than the blocked condition. Motor unit activity of the blocked condition was observed to be inconsistent with known EMG profiles of muscular cramps. The authors concluded that "overall, the findings prove the importance of central mechanisms in the generation and development of ordinary muscle cramps and extend previous studies that highlighted the influence of afferent synaptic inputs to the motor neurons on cramp development" (p. 5768). Furthermore, Minetto writes:

> The greater threshold frequency observed in the blocked condition indicates that the peripheral excitation of motor nerves is not the cause for the origin of ordinary cramps, which are elicited at a lower threshold when the spinal loop is intact. It is presently unknown whether the positive-feedback loop is triggered by neural changes that occur at the receptor level or at the spinal interneuron level and what is the exact mechanism. (p. 5768).

Work by Miller and Layzer (3) strongly indicates that peripheral factors are responsible for EAMC. The authors claim the superiority of peripheral factors on the basis of a few points of physiological theory. First, the authors mention that muscle cramps are both preceded and followed by fasciculations (small twitches). Muscle fasciculations are known to originate in the peripheral nerve (Layzer, 1994 as cited in Miller and Layzer (3)), which has been taken to mean that cramps are caused by activity in peripheral axons. A second reason why Miller and Layzer suggest peripheral factors are more important than central factors in causing cramps during exercise is that central mechanisms can only discharge motor units at a rate of less than 50 Hz, whereas discharge rates in cramps are typically close to 150 Hz. However, a limitation to Miller and Layzer's argument is that they do not address the possibility that the resultant positive-feedback loop corresponding to abnormal motor neuron activity subsequent to muscle fatigue might reach supraphysiologic levels (frequencies exceeding 50 Hz), considering the fact that the mechanism is a positive-feedback loop that may continually increase in intensity, and in light of the fact that the processes that would normally interrupt the permanently contracted state of the muscle (afferent signaling from GTO) are not functioning properly.

The take-home message from the discussion of peripheral and central stimuli for EAMC is that definitive evidence for either process with respect to the neurological stimulus for muscle cramps does not exist. The possibility that both factors play a role can also not be ignored, just as how the reader should recall that the fact the origin of EAMC is neurological in nature at all has yet to be conclusively proven.

ENVIRONMENTAL THEORY

The frequency at which cramps occur during exercise in hot humid environments form the basis of the theory that environmental conditions lead to cramps (1,3,5,8,10). Currently, no evidence exists to directly link extreme heat or cold to EAMC, and the fact that passive heating alone does not induce cramps nor does cooling attenuate them leads researchers to conclude that environmental conditions are not a primary inducer of cramps. A likely role, as suggested by numerous authors (4,8,10), is that exercise in heat may lead to secondary physiological changes, such as fatigue, which are the true precipitators of EAMC.

FATIGUE

An overarching theme in available literature is that EAMC are related to muscle fatigue. Fatigue is suggested in the Central Origin theory to precipitate the abnormal afferent feedback loop that leads to cramps (4). In the peripheral model, subclinical damage to muscle tissue occurring in conjunction with muscular fatigue is thought to damage motor neurons, causing them to fire spontaneously, causing cramps. It has been suggested, but not investigated, that increased or decreased signals from peripheral receptors such as chemo, pain, and pressure receptors, as a result of muscular damage, may influence either an increase or decrease in muscle tension from the CNS (10). In studies by Schwellnus et al. (9,11), EAMC were found to occur in the last quarter of endurance events, especially in individuals who were running at a faster pace than they had trained at. The general perspective to comprehend current theories of cramping in sport is that almost all theories identify muscular fatigue as a direct cause of EAMC, and it is the specific process of how exactly muscular fatigue manifests as cramps that researchers debate (1–3,6,9,11). A helpful analogy for the current state of EAMC can be drawn to the game of cricket; every bowler will agree that hitting the wicket is an important part of winning the match, but a group of bowlers will likely debate what the best strategy is to hit the wicket.

GENETIC PREDISPOSITION TO CRAMPING DURING EXERCISE

Research into the contributing factors to cramping during exercise seems to be heading in an interesting direction. Research published in 2013 (6) has identified a variant in genetic coding for a specific type of connective tissue in tendons that is associated with a positive history of cramping during athletics. The work by O'Connell (6) highlights the fact that in recent years a number of researchers have identified a genetic predisposition to certain soft-tissue injuries, including those to tendons and ligaments. The author hypothesized that, considering the fact that muscle damage and a family history of cramping during exercise affect an individual's risk of EAMC, the possibility exists that similar genetic variation may predispose some individuals to experiencing EAMC more than others. To date, only O'Connell's study has identified a genetic linkage to history and possible risk for EAMC. Further research into how genetics impact the risk and/or onset of EAMC may lead to further clarification of issues surrounding the so-far-inconclusive etiology of cramping during exercise.

TREATMENT/PRACTICAL IMPLICATIONS OF CURRENT KNOWLEDGE

A comprehensive model of the factors leading to EAMC does not yet exist and may never be fully determined if the magnitude of investigation on the matter does not increase. However, very few comprehensive and unchallenged models exist to explain any aspect of human function, so it may be a reach to expect such a model to be developed for how EAMC occur. Accordingly, practitioners and athletes alike must go forward on the basis of available knowledge. Although current literature seems to put minimal emphasis on the role dehydration and proper electrolyte balance play in EAMC, available evidence does not yet provide a conclusive picture of why this debilitating condition occurs. Therefore, abandoning over a century of anecdotal evidence regarding the role of dehydration and electrolyte imbalance with respect to EAMC is likely premature, especially given the findings from Jung et al. (2) that suggest carbohydrate and electrolyte supplementation before and during exercise may extend the time until the onset of EAMC. Athletes would be well advised to ensure that they are both hydrated and have a sufficient amount of electrolytes and energy stores in their body before beginning a race or athletic endeavor. Even if these precautions have no impact on EAMC, the importance of proper hydration, electrolyte balance, and carbohydrate availability on performance is undeniable and therefore advisable: it is essentially a "nothing to lose if you do, possibly something to lose if you don't" situation.

In general, EAMC seem to be primarily related to muscle fatigue (1–4,8–11). Accordingly, competitors in endurance events such as marathons and extended triathlons would be wise to adhere to a conservative tapering program in the week before competition and to be cautious about pushing the pace much beyond that which was obtained in training. Additionally, judicious use of static stretching before starting a race may be a prudent practice until further research on the subject is made available. Utilization of a long-term flexibility-improvement program, possibly including static-stretching techniques used separately from training, may be advised. Considering the risks of excessive static stretching before exercise, a dynamic warm-up consisting of movements designed to actively prepare the muscle for performance may be a better idea than static stretching. In the event EAMC does occur, athletes are advised to stretch the affected muscle.

CONCLUSIONS

One possible reason why a comprehensive model of cramping during exercise has not yet been developed may have to do with the very nature of scientific inquiry. The accepted protocol for formal scientific research dictates that

investigators attempt, to the best of their ability, to isolate 1 specific variable in service of a "black or white" hypothesis and subsequent conclusion: researchers are bound to conclude that their variable under investigation has either a significant effect or it does not. Unfortunately, no "physiological fuse box," so to speak, exists in the body whereby certain processes can be "shut off" to examine 1 physiological mechanism under controlled conditions, meaning that a reductionist perspective to EAMC research may not be the best approach. All literature reviewed attempted to reach a firm conclusion that, for example, either electrolyte depletion has a significant role causing cramps or it does not have a significant role (2,9,11); the studies are limited because conventions of scientific inquiry do not typically allow for "grey" answers: answers that fall somewhere between a firm conclusion of either affirming or denying the stated hypothesis. With the exception of work by Jung et al. (2), current research does not seem to allow for the fact that multiple mechanisms may contribute to cramping, and that the relative contribution of these factors may vary between individuals and with respect to the characteristics of a given training session or competition.

The practice of causal reductionism (attempting to reduce a likely multifaceted process to a single cause) that seems to be prevalent in EAMC may slow progress toward the development of a comprehensive model to explain why cramps occur during sport. Concluding that muscle fatigue is what causes cramps during exercise is likely too simplistic of an answer: the human body is a complex machine, and rarely can any process in the body be reduced to a simple cause-and-effect relationship that is not influenced to some degree by individual and situational characteristics. When we consider the fact that electrolyte and carbohydrate supplementation have been shown to extend time until onset of EAMC (2), as well as when we consider decades of anecdotal evidence regarding the possibility proper hydration and nutrient intake help to resist the onset of cramps during exercise, dismissing the role of hydration and proper nutrition completely seems unwise, even if reliable evidence to support the practice does not exist. Additionally, considering the fact that conclusive evidence to support the neurological origins of EAMC does not exist, total dismissal of the possibility that heat and metabolic accumulation impact on EAMC seems similarly premature. One possible idea that might warrant future research is the inclusion of the idea of task dependency with regard to the causes of EAMC. Task dependency is the idea that "fatigue is not the consequence of a single omnipresent mechanism, but rather that it can be induced by a variety of mechanisms" (12). The idea of task dependency was first applied to mechanisms of fatigue during different modes of exercise and suggests that the characteristics of an individual and exercise session might affect the mechanisms that contribute to fatigue. A similar perspective may be valid with regard to cramping during exercise. In conclusion, the causes of cramps during exercise are complex and not fully understood by researchers. Traditional theories of cramping during exercise, such as dehydration, electrolyte loss, metabolic accumulation, and heat accumulation, do not seem to be the main causes of EAMC (1–6,8–11), although a secondary role cannot be excluded on the basis of available literature. Of all theories known regarding causes of EAMC, a neurological origin is supported by the most robust body of literature (1–3,5,8,10). Neural factors are theorized to impact on EAMC in 2 ways; the Central Origin theory suggests that increased afferent signals from muscle spindles, accompanied by a reduction in GTO feedback, lead to a positive-feedback loop in motor neurons, causing cramping. The fact that passive stretching (activation of GTOs) alleviates cramps by breaking up the positive-feedback loop supports the Central Origin theory. The Peripheral Origin theory contends that EAMC are caused by a combination of damage to structures in the muscle fiber and an increase in intracellular metabolites and neurotransmitters, as a result of dehydration, that cause unmyelinated axon terminals to fire spontaneously. Evidence for which theory (Central Origin or Peripheral Origin) plays a more significant role is equivocal, with a slight edge perhaps being given to the Central Origin theory. The possibility that both mechanisms play a role in EAMC does not seem to have been seriously considered by researchers. Until further research is made available, athletes should ensure that they start activity sufficiently hydrated and fueled, avoid passive stretching immediately before their race or activity, be cautious of "pushing the pace" too much beyond what was achieved in training, taper in the week before competition, and if cramps do occur, remember that passive stretching and consumption of a fluid/electrolyte beverage will likely alleviate the cramp the fastest.

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