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Exercise Associated Muscle Cramps - A Current Perspective

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Abstract

Exercise-associated muscle cramps (EAMC) are a common condition experienced by recreational and competitive athletes and often require medical attention during or immediately after sports events. Despite the high prevalence of this condition, the etiology of EAMC remains poorly understood, and there is a lack of high levels of evidence to guide the management of this condition. The previous claim as to how EAMC come about is being challenged by more recent evidence suggesting a distinctive mechanism. EAMC has been long attributed to an excessive sweat sodium loss together with dehydration. However, growing evidence suggests that EAMC occurs with sustained and repetitive muscle contraction that results in fatigue. The purpose of this article is to examine the existing scientific evidence in support of various views on the etiology of EAMC and to highlight the most current understanding of this complex condition. Various strategies adopted to treat and prevent EAMC also are discussed even though most of them remain anecdotal and have yet to be substantiated by research experimentation.

Keywords

Muscle cramps, Dehydration, Electrolytes, Alpha motor neuron, Muscle spindle, Golgi tendon organ, Muscle fatigue

Introduction

Exercise-associated muscle cramping (EAMC) is a common condition that requires medical attention during sporting events. It occurs among athletes who participate in long-distance endurance events, such as the triathlon, marathon, and ultra-marathon [1,2]. EAMC also is documented in many other sports, including basketball, soccer, American football, rugby, tennis, and cycling [2]. The prevalence of EAMC has been reported for triathletes (67%), marathon runners (30-50%), rugby players (52%) and cyclists (60%) [1,2]. Despite the high prevalence of EAMC, its risk factors and underlying causes are not completely understood. This review is intended to examine the existing scientific evidence in support of various views on the etiology of EAMC and to highlight the most current understanding of this complex condition. Various strategies adopted to treat and prevent EAMC are also discussed. Muscle cramping can occur as a symptom for a variety of medical conditions including genetic disorders, muscular diseases, endocrine and metabolic diseases, hydro electrolyte disorders, and toxic and pharmacological agents [3]. This review focuses on cramps that are exercise induced and excludes muscle cramping that occur in smooth muscle or at rest and cramping that is associated with any underlying disease or drugs.

Methods of Literature Search

The literature search not restricted by publication date was conducted with PubMed and Google Scholar using key words that included muscle cramps together with exercise, muscle fatigue, dehydration, electrolyte imbalance, α-motor neuron, electromyography (EMG), and treatment. Searchers were set to accept publications that include original studies involving animals and humans as well as reviews, commentaries, position stands, and book chapters relating to both the etiology and treatment of EAMC. Articles on muscle cramping caused by factors other than exercise were not included.

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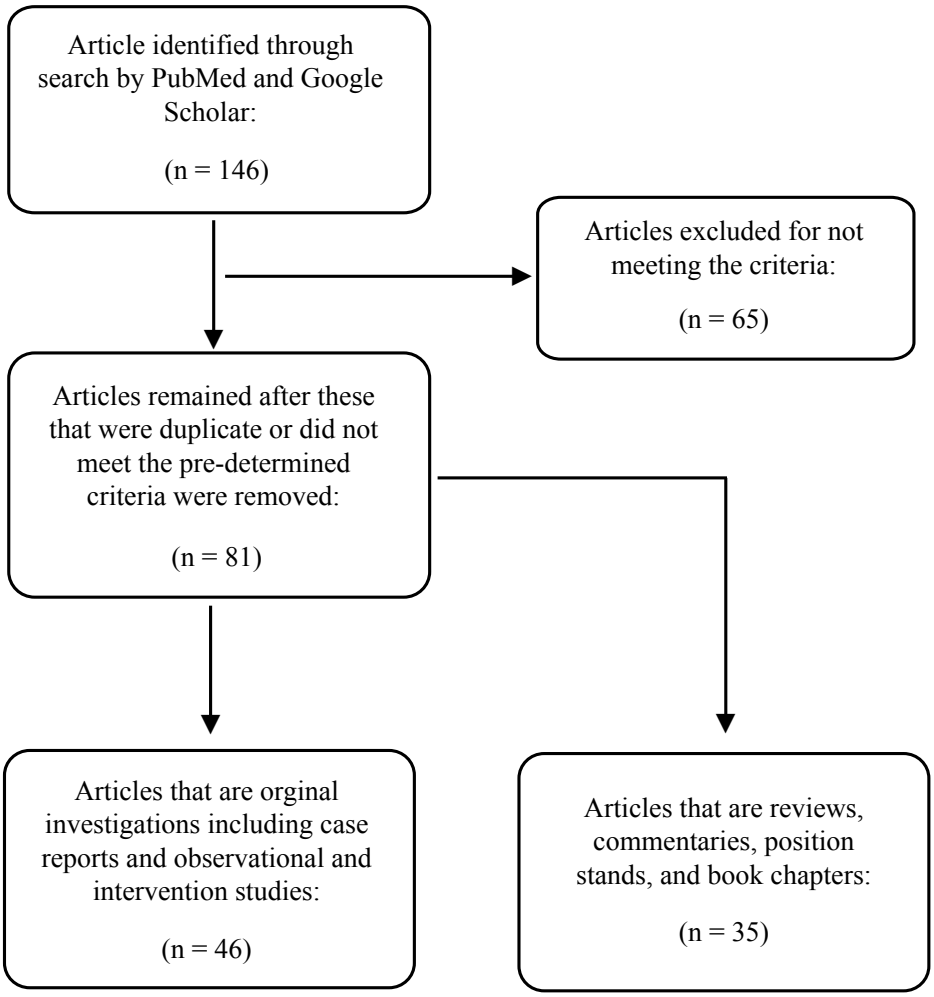

Figure 1: Selection process for the articles included in the review.

Initial searches from both search engines generated a total of 146 citations. Upon further evaluation against the per-determined criteria, 81 articles were selected that included 46 original investigations and 35 reviews, commentaries, position stands, and book chapters (Figure 1).

Overview of Exercise Associated Muscle Cramps

Defining Exercise Associated Muscle Cramps (EAMC)

EAMC is defined as a syndrome of involuntary painful skeletal muscle spasms that occur during or immediately after physical exercise [4]. It presents as localized muscle cramping that happens spasmodically in different exercising muscle groups, usually the gastrocnemius, hamstrings, or quadriceps. The gastrocnemius is the most commonly affected [4].

Signs and symptoms of EAMC

Clinically, EAMC may be recognized by acute pain, stiffness, visible bulging or knotting of the muscle, and possible soreness that can occur suddenly with no warning and last for several days [5,6]. The affected muscles often appear to be randomly involved, and as one bundle of muscle fibers relax, an adjacent bundle contracts, giving the impression that the spasms wander [7,8]. For example, twitches first may appear in the quadriceps and subsequently in another muscle group [7]. Most EAMC incidents last 1-3 min, but athletes often complain of EAMC symptoms up to 8 hours after exercise [9]. This post-exercise period of increased susceptibility to EAMC has been termed as the cramp-prone state [10]. EAMC can be completely debilitating [11,12] although in some cases EAMC do not appear to affect athletic performance [13,14].

Risk factors

EAMC seems to be more frequent in long-duration, high-intensity events. Indeed, the competitive schedule of certain athletic events may predispose to EAMC. In multi-day tennis tournaments, competitors often play more than one match a day, with only an hour between matches. This competition format induces muscle fatigue, impedes fluid and electrolyte replacement between matches, and often results in debilitating muscle cramps

[15]. Risk factors associated with EAMC in running also have been examined in a cross-sectional survey of 1300 marathon runners [16]. In this survey, the specific conditions found to be associated with EAMC included high-intensity running, long distance running (> 30 km), subjective muscle fatigue, all of which are intense and exhaustive physical efforts. Other risk factors identified in this survey were older age, a longer history of running, higher body mass index, shorter daily stretching time, irregular stretching habits, and a positive family history of cramping [16]. In a prospective study of Ironman triathletes, the only independent risk factors for EAMC were a history of the condition and competing at a higher than usual exercise intensity [17]. A review on muscle cramping in marathon also suggests that EAMC is associated with running conditions, e.g., high intensity, long duration, and hilly terrain, that can lead to premature muscle fatigue in runners who have a history of the condition [18].

History and Original Etiology of EAMC

Early reports of muscle cramps

The earliest reports of muscle cramps come from 100 years ago, when labors in hot and humid conditions of the mines and shipyards suffered from cramps [19,20]. Upon further analysis, it was noticed that the builders had a high chloride level in their sweat. In these reports, it was noted not only that muscle cramping occurred in the heat, but also that cramps were accompanied by profuse sweating [20]. More recently, by monitoring external heat illness among American football players, Cooper, et al. [21] observed that 95% of the cramping incidents occurred in hot months when the risk of developing heat illness was "high" or "extreme" [21]. It was because of these early observations that the "electrolyte imbalance and dehydration" theory was developed as an underlying cause for EAMC. In principle, this theory suggests that overly sweating and thus loss of electrolytes can cause muscles and nerves that innervate them to malfunction, thereby producing muscle cramps. It is now a common belief that EAMC happens because athletes exercise in the heat, lose electrolytes in their sweat, and the resulting electrolyte imbalance and dehydration combines with high body temperature [22,23].

The electrolyte-imbalance-and-dehydration theory

The electrolyte-imbalance-and-dehydration theory suggests that EAMC is related to the decreased concentration of serum electrolytes, particularly sodium and chloride, resulting from excessive sweating or overconsumption of water [4,8,24]. Indeed, a sizable whole-body exchangeable sodium deficit always develops following a single long race, match, game or training session or repeated exercise bouts when sweat sodium and chloride losses significantly exceed salt intake [25,26]. An estimated sweat-induced loss of 20-30% of the sodium pool has been linked to severe muscle cramping [15,27]. Other electrolytes lost in sweat to a much lesser degree, namely calcium, magnesium, and potassium, have also been implicated as the cause of muscle cramping during or after exercise when purported deficiencies are suspected [28-31]. The chief premise behind the electrolyte imbalance theory is that an increased sweat sodium concentration or "salty sweating" results in sodium depletion, which then causes EAMC [25,32]. However, the pathophysiological basis for this hypothesis remains poorly defined.

The more sensible explanation for how the electrolyte-imbalance-and-dehydration theory works seems to be more dehydration-driven. Because sweat sodium concentration is always hypotonic [33,34] a significant loss of sodium through sweat can therefore only occur if there is a large loss of bodily fluid. This would mean that dehydration would accompany any significant sodium loss in athletes experiencing EAMC. Excessive sweating will reduce plasma volume. To compensate for the loss in plasma volume, water from the interstitial fluid compartment shifts to the intravascular space to maintain central blood volume [35-37]. As sweating continues, the interstitial fluid compartment becomes increasingly contracted [35]. This can persist even after exercise as sweating continues and body temperature returns to a pre-exercise level [36]. Consequent to a contracted interstitial compartment, certain neuromuscular junctions (especially in muscles that are heavily used) could become hyper-excitable by mechanical deformation. The resulting change in mechanical pressure can then induce spontaneous discharges of the affected motor neurons, thereby causing cramps [6,38]. Figure 2 illustrates a step-by-step process of how sweat-induced dehydration may trigger EAMC.

Cramp discharges may also be attributed to the fact that terminal branches of motor axons are exposed to increased concentrations of excitatory extracellular substances such as acetylcholine or electrolytes (i.e., sodium and potassium) [39-41]. As more water is shifted away from the interstitial compartment to the intravascular space, adjacent and other nerve terminals and post-synaptic membranes could be similarly affected. This may explain why cramping is often observed in various muscle bundles alternately contracting and relaxing [42].

Evidence against the original etiology of EAMC

As mentioned earlier, support for the electrolyte-imbalance-and-dehydration theory comes mainly from case reports or observations with no actual measures

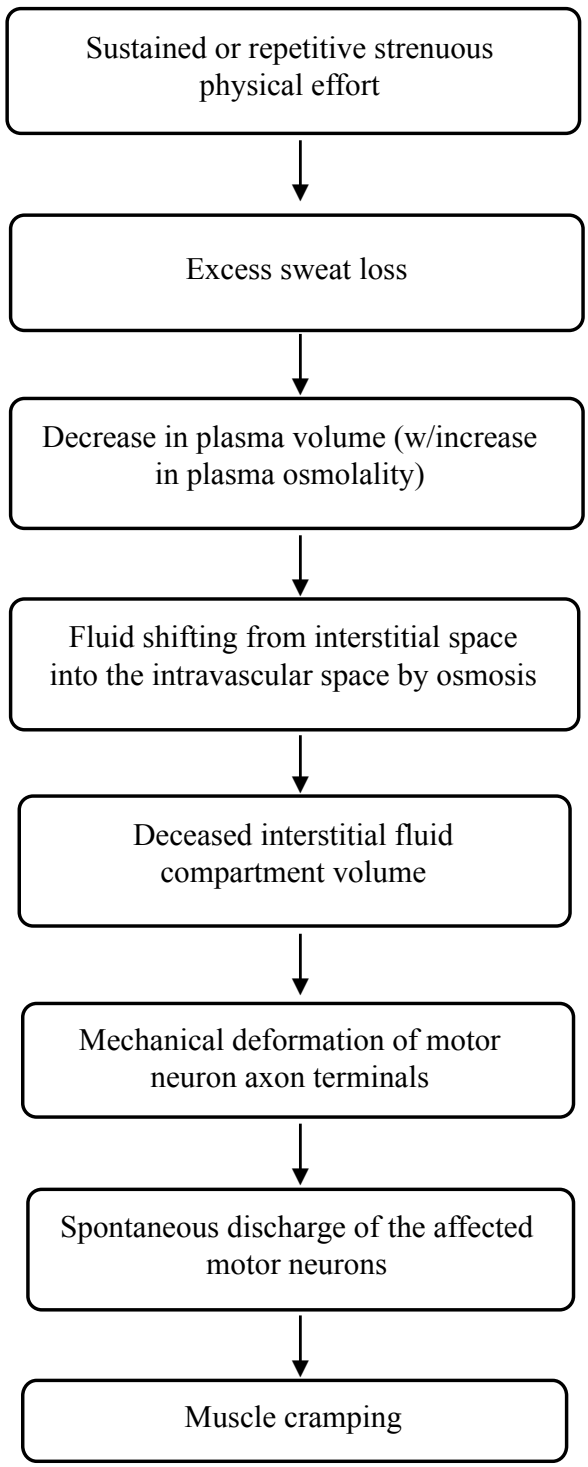

Figure 2: Schematic illustration of the electrolyte - imbalance - and - dehydration theory of exercise associated muscle cramps (EAMC).

of hydration status reported. Miners develop cramps because of their sweat losses while working in hot and humid conditions [6,20]. More recently, research on American football revealed that most cramping incidents occurred in hot months when football players trained in an environment where the risk of developing heat illness was high [21]. Other evidence for this theory comes from case studies and observational work in which large sweat losses occurred in exercising athletes [15,25,26].

The electrolyte-imbalance-and-dehydration theory seems to contradict many recent evidences. Four prospective cohort studies have shown no relationship between serum electrolyte abnormalities and EAMC in marathon runners or triathletes [13,14,17,43]. The findings have led to the suggestion that increased sweat concentration (or salty sweating) resulting in sodium depletion is the mechanism for EAMC [44,45]. However, the physiological explanation for how sodium depletion may cause EAMC remains unclear. Hyponatremia resulting from a significant loss of sodium has been associated with generalized muscle cramping at rest [46]. Nevertheless, in most athletes experiencing EAMC, cramping occurs in the localized muscle groups involved in repetitive contractions during exercise. At the present, no data is available to support the possibility that a systemic imbalance of electrolytes could result in localized muscle cramps [10].

As for dehydration, it has been argued that excessive sweating is the primary cause of EAMC [4]. However, in the four prospective cohort studies previously mentioned in which calculated body mass changes and volume of blood or plasma were used as indicators of hydration status, it was found that cramping athletes were not more dehydrated than non-cramping athletes [13,14,17,43]. Additionally, using an electrical stimulation model, Braulick, et al. [47] and Miller, et al. [48] observed that both mild (3% body mass) and severe (5% body mass) hypohydration with moderate electrolyte losses did not alter cramp susceptibility when fatigue and exercise intensity were controlled. Thus, the hypothesis of a direct relationship between dehydration and muscle cramping was not supported.

The electrolyte-imbalance-and-dehydration theory also does not stand when it is used to explain EAMC that occurs in athletes exercising in cool and temperature-controlled environments [49,13]. For example, Maughan [13] reported that some marathoners (~18%) still developed EAMC even though the ambient temperature was 10 to 12 °C. Thus, it is unlikely that a hot and humid environment is required for the development of EAMC, although EAMC may occur more frequently under conditions of elevated ambient temperatures [21]. This same study also revealed no significant differences in losses of plasma volume and body mass between runners with and without EAMC [13].

Overall, the electrolyte-imbalance-and-dehydration theory has limitations. Its supporting evidence was based

on observational studies that could not provide cause-effect conclusions. Although EAMC may appear in the presence of significant electrolyte and/or fluid losses during exercise, the underlying cause can be due to other factors such as neuromuscular fatigue, fuel deficiency, and accumulation of metabolic wastes, muscle damage, and a lack of conditioning and/or acclimatization.

"Heat cramps" - a misnomer

Case reports and anecdotal observations often related the development of cramping to physical activity performed in hot and humid conditions, and this has led to the use of "heat cramps" or "exertional heat cramps". These terms are often used synonymously with EAMC [15,42,44,50]. More substantive data to support the use of this terminology came from a study in which the term "heat cramps" was reported to be more common when American Football players trained in an environment where the heat index was "high" or "extreme" compared with "low" or "moderate" [21]. It must be noted that these hot and humid weather conditions occurred during the first 2-3 weeks of training in a season when players were also most likely less well-conditioned and/or acclimatized to the heat. EAMC is known to also occur in individuals exercising in moderate to cool temperatures [13,49] and exposure to extreme cold also has been associated with EAMC in swimmers [51]. In addition, it has been found that the development of EAMC does not correlate with an increased core temperature [13]. Clearly, heat alone is not a direct cause of muscle cramping during exercise. As such, the term "heat cramps" is inaccurate and its use should be discouraged.

Recent Discoveries on EAMC

The altered neuromuscular control theory

During sports competition, training, and a variety of other intense physical activities, repeated or extended loading on selected muscles can lead to localized muscle fatigue. The altered neuromuscular control theory suggests that muscle fatigue disrupts the normal functioning of peripheral muscle receptors, causing an increase in excitatory afferent activity within the muscle spindle and a decrease in inhibitory afferent activity within the Golgi tendon organ, both of which then lead to an increase in alpha motor neuron discharge to the muscle fibers, producing a localized muscle cramp [4,52].

Muscle spindles and the Golgi tendon organs are two important proprioceptors involved in working together reflexively to regulate muscle length and tone via alpha and gamma motor neurons. When the Golgi tendon organ is excited, it causes the muscle to relax, which is opposite of a muscle spindle that causes it to contract (Figure 3). Disturbance in the activity of these proprioceptors can occur through faulty posture, shortened muscle length, intense exercise and exercise to fatigue, thereby resulting in increased motor neuron activity and motor unit recruitment [53].

The altered neuromuscular control theory is supported by animal studies that used isolated gastrocnemius muscles derived from cats and electromyographic recordings [54,55]. In these studies, muscle fatigue was introduced via electric stimulation, and experiments were terminated when the maximum force obtainable decreased by 25%. It was found that as muscle fatigue developed, there was an increased firing rate of the muscle spindle's type Ia and II afferents concomitant with a decrease in the type Ib afferent activity from the Golgi tendon organ [54,55]. In other words, muscle cramps can be viewed as a consequence of a sustained alpha motor neuron discharge that occurs when the enhanced excitatory activity of the muscle spindle that triggers an involuntary muscle contraction is unopposed by Golgi tendon organs designed to inhibit such a muscular re-

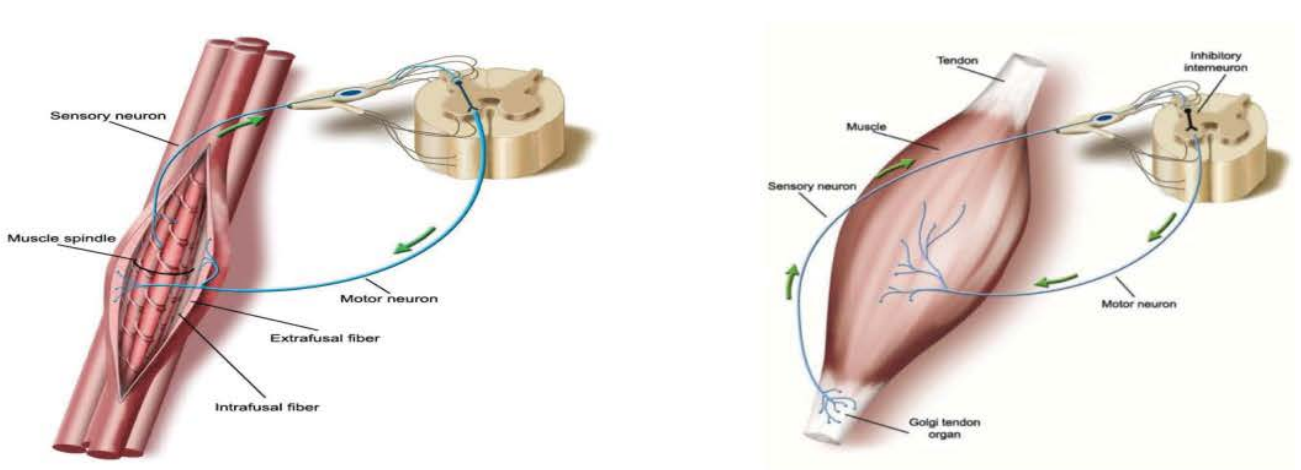

Figure 3: A muscle spindle (left) and a golgi tendon organ (right).

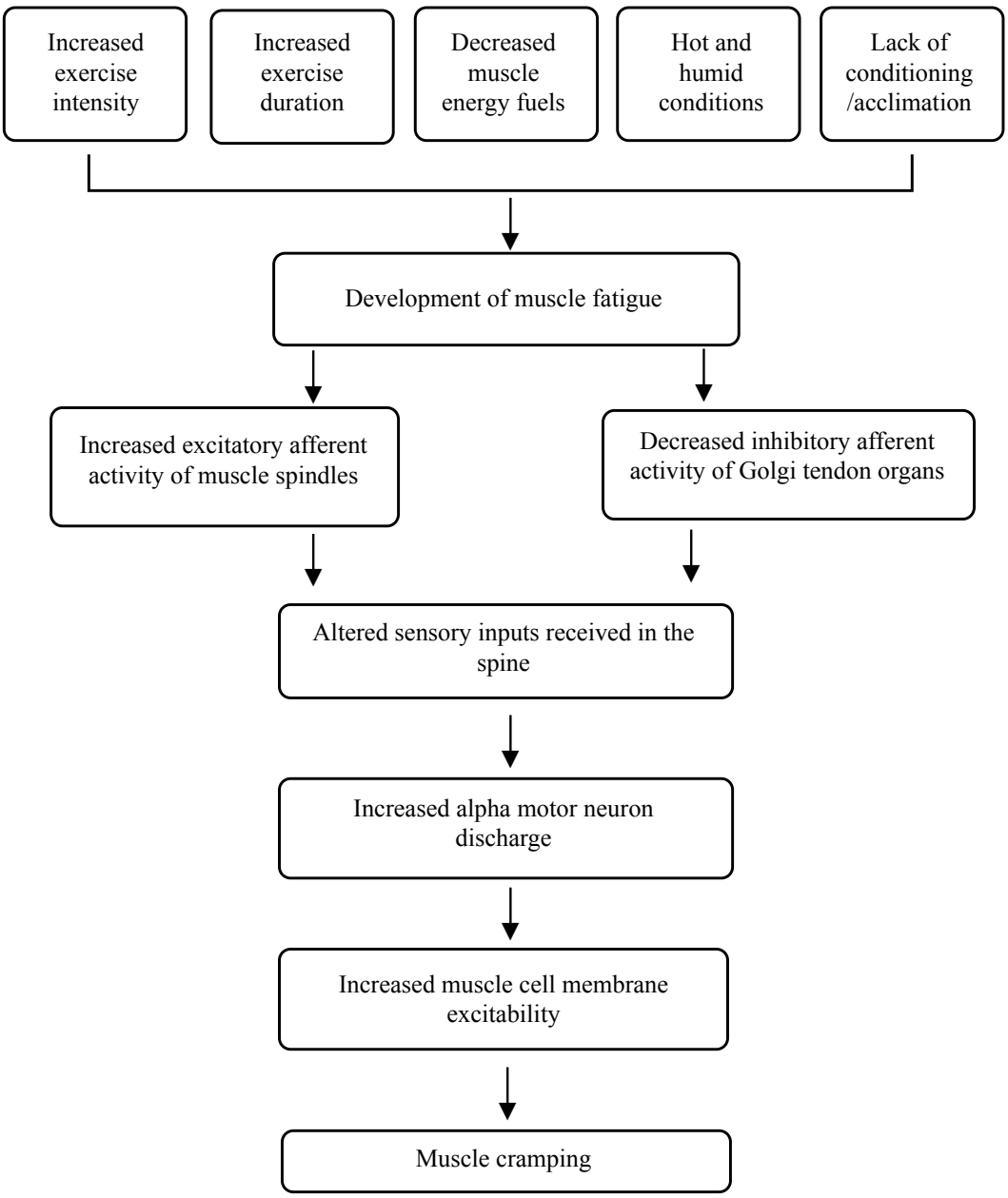

Figure 4: Schematic illustration of the altered neuromuscular control theory of exercise associated muscle cramps (EAMC).

sponse. Figure 4 illustrates more detailed explanations of how muscle cramps may come about based on the altered neuromuscular control theory.

EAMC are more likely to occur when the muscle is contracting in an already-shortened position [4,40]. This is because when muscle is in a shortened position, the inhibitory activity of the Golgi tendon organ will reduce even more than normal, causing a greater imbalance between inhibitory and excitatory drives to the alpha motor neuron [56]. The observation that the shortened muscle is more prone to cramping may explain why calf muscle cramps are prevalent in swimmers because in most swimming races a swimmer must swim with pointed toes that require the calf muscle to remain somewhat contracted. The potential risk factors associated with overload and fatigue-related muscle cramping also include older age, poor stretching habits, insufficient conditioning, cramping history, excessive exercise intensity and duration, and related metabolic disturbances (i.e., muscle glycogen depletion) [18,53].

Evidence supporting the altered neuromuscular control theory

The theory is first brought up in the early 1990s by an observational study in which 1383 marathon runners responded to a questionnaire on EAMC [16]. Of these runners, 536 (26%) reported a history of EAMC and a majority (60%) of them indicated that the onset of EAMC was

associated with muscle fatigue. This finding concurs with those of Maughan [13] who found that the occurrence of EAMC was more common in the later stages of a marathon race. Stronger evidence linking potential muscle fatigue and EAMC comes from a prospective cohort study in Ironman triathletes [17]. In this study, 210 triathletes competing in an Ironman triathlon acted as subjects, and all the subjects were surveyed for their training history, personal best performances, and cramping history prior to the race. Results of this study showed that those who developed EAMC (n = 44) exercised at a higher intensity during the race and had faster overall race time despite similar preparation and performance histories as compared to those who did not develop EAMC [17]. These findings indicate that the increased exercise intensity in the cramp group was a risk factor for the development of EAMC. In a study using an exercise protocol specifically designed to induce calf muscle fatigue, a high incidence of muscle cramping during exercise was documented [57]. This study also revealed that supplementing carbohydrate orally, compared with no fluid administration, resulted in a delay in the onset of EAMC following a calf fatiguing protocol [57]. It appears that providing athletes with more energy fuels can alleviate EAMC.

More objective evidence that supports the theory come from studies that used EMG to trace the discharge of alpha motor neurons in fatigued muscles as EAMC developed [54,55]. For example, a modest increase in EMG was noticed as muscle twitches prior to EAMC. However, as fatiguing exercise continues, there was a much greater change in EMG that coincided with a full-blown muscle cramp. By using EMG to compare ironman triathletes with and without EAMC, Sulzer, et al. [43] found that baseline EMG activity in triathletes who suffered from EAMC was significantly higher in a cramping than a non-cramping muscle. Interestingly, this study revealed no significant differences in serum electrolyte concentrations between cramping and control groups matched for race finishing time and body mass.

The theory also seems consistent with the use of passive stretching in treating EAMC. Passive stretching is the most common and effective therapy to relieve acute muscle cramping [1,58-60]. It is regarded as effective treatment by those who support the electrolyte-imbalance-and-dehydration theory [42,61]. Passive stretching increases the tension in a muscle, thereby increasing the Golgi tendon organ's inhibitory input to the alpha motor neuron [6,56]. This mechanism offers further support for the hypothesis that abnormal neuromuscular control mediates EAMC.

Other etiological factors in EAMC

Other factors have been speculated to alter neuromuscular control at the spinal cord level, thereby contributing to the development of EAMC. The first of these is the possibility that muscle injury or damage, resulting from fatiguing exercise, could cause a reflex "spasm", and thus lead to a sustained involuntary contraction. The second possibility is that changing signals from other peripheral receptors, such as chemically sensitive intramuscular afferents, pressure receptors, or pain receptors, could elicit a response from the central nervous system, which may alter neuromuscular control of the muscles [56]. In a prospective cohort study involving triathletes, one independent risk factor associated with EAMC was a previous history of EAMC [17]. In addition, studies that surveyed marathon and ultra-marathon runners and Ironman triathletes, a positive family history of cramping also has been reported as a risk factor for EAMC [16,62,63]. In this context, a genetic predisposition to EAMC cannot be ruled out. Indeed, mutation of a gene (i.e., COL5A1) that provides instruction for synthesizing collagen has been identified as a potential marker for a history of EAMC [64]. Among other theories that have been proposed for the etiology of EAMC are an inadequate intake of carbohydrate, glycogen depletion, poor biomechanics or running gait, hilly terrain and lack of adequate massage before and during a game [2].

Treatment and Prevention

Treatment

With multiple theories about the cause of EAMC, it is difficult to provide a single answer for a treatment or prevention strategy. Consequently, there are many interventions available for the treatment of muscle cramps. These treatment options include stretching of the affected muscle, decreasing exercise intensity, massage, thermotherapy, cryotherapy, sports drinks, salt and electrolytes, pickle juice, intravenous infusion, and transcutaneous electric nerve stimulation. Many of these treatment options are anecdotal or not supported by experimental research. EAMC can be viewed as the endpoint of a variety of pathways and different athletes may have different mechanisms leading to very similar-appearing EAMC. Therefore, a treatment that works for one athlete may not be effective one for others. Some of the commonly used treatment options aimed to combat against EAMC are discussed in the following sections.

The electrolyte-imbalance-and-dehydration theory suggests that ingesting fluids containing electrolytes helps normalize the interstitial or extracellular volume, thereby alleviating EAMC. However, owing to a small quantity of electrolytes in many sports drinks, it may be difficult to sufficiently replace the volume of electrolytes lost during exercise even if the athlete has modest sweat losses and sweat sodium content. Note that fluids and

electrolytes are not absorbed immediately after ingestion; that is, even hypotonic fluids require at least 13 minutes to be absorbed into the circulatory system [65]. Based on the assumption that a relationship between dehydration-electrolyte imbalance and EAMC exists, the National Athletic Trainers' Association recommends that athletes prone to muscle cramping add ~1.3 $g \cdot L^{-1}$ of salt to their drinks to avert muscle cramps [66]. Others have recommended adding higher amounts of sodium (about 3 to 6 $g \cdot L^{-1}$) to sports drinks based on the frequency of EAMC [50]. At the first sign of muscle twitches or mild cramps, a prompt oral bolus of a high-salt solution (e.g., 0.5 L of a carbohydrate-electrolyte drink with 3 g of salt added consumed all at once or over 5-10 min) has been proven effective in preventing muscle twitches from developing into a full-blown EAMC [15]. After such a high-salt solution bolus, athletes can often promptly resume training or competition effectively without muscle cramping or twitching symptoms for an hour or more [7], although additional lower-sodium fluid should be consumed at regular intervals.

Other substances often chosen to relieve EAMC are pickle juice, quinine and electrolytes such as magnesium, potassium, and calcium. A case report for using pickle juice to treat EAMC revealed that ingesting of a small volume of highly salty and acidic brine (30 to 60 mL) could relieve cramp within 35 seconds [67]. This effect was, however, attributed to the fact that pickle juice contains acetic acid that can trigger a reflex, probably in the oropharyngeal region, that acts to increase inhibitory neurotransmitter activity in cramping muscles [67]. In fact, consuming pickle juice was found to be ineffective in raising plasma electrolyte concentrations in both hydrated and dehydrated humans [68,69]. Quinine is a medication used to treat malaria caused by mosquito bites, but also often prescribed to treat cramps of all causes. A systematic review of 23 clinical trials has concluded that quinine reduces cramp frequency, intensity, and days, but not duration, compared with placebo, and that there is a significantly greater risk of thrombocytopenia for quinine compared with placebo [70]. Though magnesium supplementation has been reported to be the most treatment used to prevent recurrent cramping [71], most users report this method to be of little or no help. Additionally, potassium-rich supplements or foods or other mineral supplements such as calcium have not been proven effective in relieving symptoms associated with muscle cramps [15,32].

If the athlete has no underlying illness, then the most common treatment for EAMC is stretching [40]. In fact, moderate stretching of the affected muscle has proven to be effective for muscle cramps of all types including those that are heat related [5,7,10,72]. Therefore, moderate stretching of the affected muscle to alleviate the cramp is recommended. Passive stretching increases the tension in a muscle, thereby increasing the Golgi tendon organ's inhibitory input to the alpha motor neuron [55,56]. This will then reduce the activity of alpha motor neurons, making EAMC less likely to occur. Other methods that have been implicated for reducing motor neuron activity, thereby alleviating EAMC include massage, active contraction of the antagonist muscle group, and icing of the affected muscles. Certainly, lowering overall exercise intensity and altering the load on the distressed muscles are effective as well [38].

Recently, an effort has been devoted to evaluate a method of using food extracts like peppers, ginger, mustard, and cinnamon to resolve EAMC [68]. Consuming these food extracts does not seem to affect plasma electrolyte concentrations [68]. Instead, it is thought that these food extracts can activate transient receptor potential channels (TRP channels) that are capable of disrupting hyper excited motor neurons [73]. TRP channels are a group of ion channels located in the mouth, esophagus and stomach that regulate the flow of ions, i.e., charged particles like sodium and potassium, across cell membranes. Recent evidence suggests that oral ingestion of TRP channel agonists like cinnamon, peppers, or mustard may attenuate the intensity and/or duration of muscle cramps, presumably by dampening alpha motor neuron excitability [74,75]. These studies produced muscle cramps by electrical stimulation. Therefore, this preventive approach has yet to be examined in clinical trials where cramps can be more naturally induced by physical exercise.

Prevention

The pathophysiology causing EAMC is most likely multifactorial and complex. As such, prevention of EAMC will need a multifactorial approach [42,66]. EAMC that occur in hot conditions seems to be prevented by maintaining fluid and salt balance. Monitoring an athlete's body mass is an easy method of ensuring adequate fluid replacement. Both the National Athletic Trainers' Association and the American College of Sports Medicine recommend a volume of fluid that allows for less than a 2% body mass reduction from training or competition [42,66]. An athlete who ingests a liter of water or hypotonic sports drink at least 1 hour before competition can be confident that the majority of the fluid, electrolytes, and nutrients would be absorbed and become available in the body when the competition begins. Additionally, fluids should be available and easily accessible throughout practices and competitions. Athletes with high sweat rates and sodium loss or who have a history of EAMC may need to consume supplemental sodium during prolonged activities to maintain salt bal-

ance [7] and may need to increase daily dietary salt to 5-10 g·day^{-1} when sweat losses are large [42]. This is especially important during the heat acclimatization phase of training. Bergeron [15] demonstrated that by calculating sweat sodium losses and replacing them during and after activity, two athletes with previously debilitating EAMC were able to compete successfully in hot conditions.

As mentioned earlier, an important etiology for EAMC is muscle fatigue. As such, prevention strategy should also focus on proper conditioning of an athlete. To truly simulate race or game conditions, intense endurance training is necessary. As endurance capacity increases, muscle would be less prone to cramp at a given level of intensity. Endurance training may also serve as an effective means of preventing EAMC by expanding plasma volume and the extracellular fluid compartment and delaying neuromuscular fatigue [76,77]. The conditioning regimen should also consider resistance training of the affected muscle as well as its synergists. In a case report involving a male triathlete [78], strengthening gluteus maximus was found to be effective in preventing EAMC of the hamstrings, a finding that was attributed to a reduced relative strain placed on the hamstrings [78]. Athletes who are returning to competition after injury are particularly susceptible to EAMC as they are likely to experience early muscle fatigue, to be less acclimatized to a hot environment, and to have diminished sweating capacity [79]. Proper progression during rehabilitation will prevent overstressing the athlete while ensuring adequate sport specific conditioning before the return to competition.

Prevention exercises that target muscle spindle and Golgi tendon organs should also be implemented to delay the onset of neuromuscular fatigue and, hence, EAMC. Plyometrics may be such exercise to be considered. The explosive nature of this exercise can train neuromuscular units to operate more effectively with increasing levels of intensity. It has been reported that plyometric training can improve the efficiency of neuromuscular control by muscle spindles and Golgi tendon organs, thereby making them more resistant to fatigue [80,81].

Other preventive measures that have been taken include 1) correcting technique errors, muscle imbalance, and/or posture, 2) stretching muscle regularly, 3) having adequate warm up, 4) applying massage therapy before and during competition, 5) wearing compression garments, 5) becoming heat acclimatized, and 6) optimizing footwear and/or orthotics. These preventive measures, however, are not evidence-based.

To prevent or attenuate EAMC, the following recommendations should be used:

- Train at race-intensity or, conversely, race according to the level of ability that was attained in training.
- Know the differences in humidity, temperature, indoor versus outdoor, altitude and terrain between competition and usual training conditions.
- For those who have cramped in the past, think about all factors that could have played a role, i.e., drastic change in intensity, volume, altitude, terrain, so they can plan their training and competition accordingly.
- Learn to recognize early warning signs of EAMC and respond accordingly.
- Muscles most affected by cramping are those repetitively used and confined to a small arc of motion, so focus on form in training to avoid heavy "braking" and try to stretch out the stride with adequate hip and knee flexion and extension.
- For those who are heavy sweaters, be sure to increase salt intake and consume fluids higher in sodium content, especially in the hotter, more humid months.
- Salt tabs or pills are an easy method, but practice using them in training as they can cause upset stomach in some individuals.
- Have adequate nutritional intake, particularly carbohydrates, to prevent premature muscle fatigue during exercise.
- Consider plyometric training of key muscle groups.
- Along with regular stretching, consider corollary activities like flexing opposing muscles and massaging cramped or cramp-prone muscles.

Conclusion

EAMC are a common condition experienced by recreational and competitive athletes. As such, it is imperative for clinicians to identify underlying causes and effective strategies for treating and preventing the condition. Despite the high prevalence of EAMC, few experimental data exist on their cause, treatment, and prevention to date. EAMC has long been explained by the electrolyte imbalance-and-dehydration theory. However, its supporting evidence comes mainly from anecdotal observations and case reports. In addition, the theory does not offer plausible pathophysiological mechanisms, and it has been reported that EAMC can occur without electrolyte depletion or dehydration. More recent evidence suggests that EAMC may be mediated by muscle fatigue that altered neuromuscular control. The evidence that support this “altered neuromuscular control” theory stems from the laboratory-based experiments that used EMG to monitor spinal reflex activities in response to muscle fatigue and cramping. Although more experimental evidence is still needed, muscle fatigue and al-

tered neuromuscular control seem more plausible in explaining many of the unanswered questions with regard to the cause, treatment and prevention of this complex condition. There are numerous strategies existed for the treatment and prevention of EAMC. However, most of them are anecdotal and unsupported by experimental research. Research that involve both humoral and neurological systems and provide high levels of evidence is needed to substantiate the etiology, treatment options, and prevention strategies of EAMC.

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