Exercise-associated muscle cramps

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Although exercise-associated muscle cramping is a common complaint among athletes, it remains poorly understood and there is a lack of good quality scientific evidence to guide management. This article presents the current understanding of this complex condition.

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Exercise-associated muscle cramping (EAMC) is a common condition that requires medical attention during sporting events. It is common among athletes who participate in long-distance endurance events, such as triathlon and marathon or ultra-marathon distance running, and it is documented in many other sports, including basketball, the various football codes, tennis, cricket and cycling.[1] The prevalence of EAMC has been reported for triathletes (67%),[2] marathon runners (between 30% and 50%),[2] rugby players (52%)[1] and cyclists (60%).[1] Despite the high prevalence of EAMC, its risk factors, pathophysiology, treatment and prevention are not completely understood.

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Muscle cramping can occur as a symptom of a variety of medical conditions. These include genetic causes, muscular diseases, endocrine and metabolic diseases, hydroelectrolyte disorders, and toxic and pharmacological agents.[3] This article focuses on EAMC, and excludes muscle cramping in smooth muscle, cramping at rest and cramping associated with any underlying disease or drugs.

WHAT IS EXERCISE-ASSOCIATED MUSCLE CRAMPING?

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

RISK FACTORS

The risk factors for EAMC are not well documented. However, factors associated with EAMC in running have been examined in a cross-sectional survey of 1300 marathon runners and found to include older age, a longer history of running, higher BMI, shorter daily stretching time, irregular stretching habits and a positive family history of cramping.[5] Specific sporting conditions associated

WHAT CAUSES EXERCISE-ASSOCIATED MUSCLE CRAMPING? A SUMMARY OF DIFFERENT THEORIES

The first hypotheses for the aetiology of EAMC were proposed over 50 years ago, when the condition was thought to be related to abnormal serum electrolyte concentrations, dehydration or environmental stress.[4,9,10] A new hypothesis, proposed in the late 1990s, suggested that muscle fatigue, and therefore altered neuromuscular control, was the primary factor associated with developing EAMC.[4] Muscle fatigue is now acknowledged as the principal predisposing factor in the development of EAMC.

Serum electrolyte theory

The serum electrolyte theory suggests that EAMC is related to the decreased concentration of serum electrolytes (sodium, potassium, magnesium, chloride and calcium) resulting from profuse sweating or overconsumption of water.[4,10,11] Abnormalities of serum electrolyte concentrations in patients with EAMC were first reported in the early part of the twentieth century as a case series.[12,13] Patients exposed to physical exercise in hot, humid conditions have developed hyponatraemia and hypochloraemia.[14]

The association between serum electrolyte abnormalities and skeletal muscle cramping at rest has been further documented.[15,16] Experimentally induced hyponatraemia, if accompanied by sodium loss, has been associated with generalised skeletal muscle cramping. However, it is well known that EAMC occurs in localised muscle groups that are involved in repetitive contraction, whereas serum abnormalities associated with altered serum electrolyte concentrations cause generalised skeletal muscle cramping.[4]

Four prospective cohort studies have shown no relationship between serum electrolyte abnormalities and EAMC in marathon runners or triathletes.[6,17-19] The findings have led to suggestions that increased sweat concentration ('salty sweating') resulting in sodium depletion, rather than changes in serum electrolyte concentrations, is the mechanism for EAMC.[20,21] However, the pathophysiological basis for this proposal is not clear and has not been formally outlined.

Dehydration theory

According to the dehydration theory, excessive sweating is the primary cause of EAMC.[4] This theory is propagated because of the association of heat illness with cramps. However, the dehydration theory is based on anecdotal observations, with no actual measures of hydration status reported. In the four prospective cohort studies mentioned above, in which calculated body weight changes and volume of blood or plasma were used as indicators of hydration status, the hypothesis of a direct relationship between dehydration and muscle cramping was not supported.[6,17-19]

Environmental theory

The environmental theory suggests that exercising in hot conditions and the subsequent electrolyte loss and dehydration results in EAMC.[4,11,21] However, EAMC is not directly related to an increased core temperature. At rest, passive heating does not result in skeletal muscle cramping and cooling does not relieve it, so it is unlikely that exercising in hot conditions causes secondary physiological changes that can cause EAMC.

Altered neuromuscular control theory

According to this theory, EAMC is a result of altered neuromuscular activity, and the underlying cause is muscle fatigue. Disturbances at various levels of the central and peripheral nervous systems and skeletal muscle are involved. Muscle fatigue disrupts the functioning peripheral muscle receptors and causes increased excitatory afferent activity within the muscle spindle and reduced inhibitory afferent activity within the Golgi tendon organ.[22,23] It is proposed that the combination of these events along with the developing muscle fatigue results in sustained motor neuron activity caused by abnormal motor neuron control at the spinal level, resulting in muscle cramp.

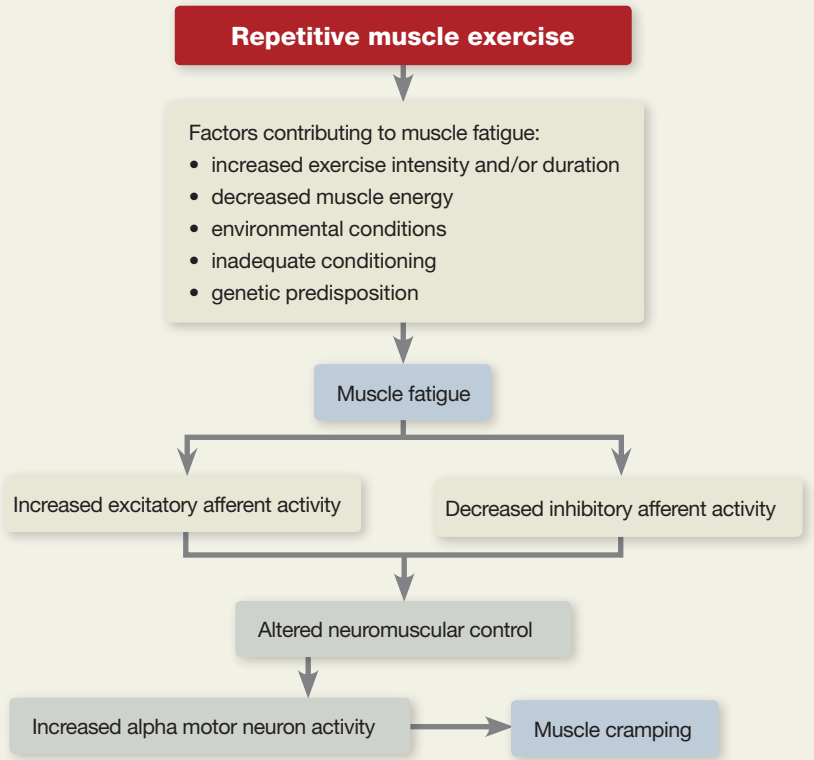
Study findings that support this theory include an increase in baseline EMG activity recorded between bouts of cramping in athletes experiencing EAMC,[17] which indicates that cramping muscles exhibit increased neuromuscular excitability. Another study has shown that athletes who exercised at a higher intensity than usual during a training session or a competition were more likely to develop EAMC,[7] and a prospective study of Ironman triathletes who developed EAMC exercised at a higher intensity during the race compared with the rest of the field.[6] A laboratory-based exercise protocol specifically designed to cause premature fatigue of the calf muscles has been shown to result in a high incidence of muscle cramping during exercise.[24] The fact that passive stretching is the most effective way to relieve acute muscle cramping supports the theory that altered neuromuscular activity is associated with EAMC because this stretching increases muscle tension and therefore increases the inhibitory activity of the Golgi tendon organ.[22]

A summary of the altered neuromuscular control theory is presented in the flowchart on page 64.

Other theories

Other theories have been proposed for the aetiology of EAMC. Potential contributing factors in these theories include genetic predisposition and family history, lack of adequate massage before and during a game, insufficient carbohydrate loading or carbohydrate inadequacy during exercise, ground conditions (ground 'hardness') and poor biomechanics or poor running gait.

AETIOLOGY OF EXERCISE-ASSOCIATED MUSCLE CRAMPING: A SUMMARY OF THE ALTERED NEUROMUSCULAR CONTROL THEORY*



* Adapted from: Schwellnus MP. Cause of exercise associated muscle cramps (EAMC) – altered neuromuscular control, dehydration or electrolyte depletion? Br J Sports Med 2009; 43: 401-408 (reference 8).

with EAMC included high-intensity running, long distance running (>30 km), subjective muscle fatigue and hill running.[5] In a prospective study of Ironman triathletes, the only independent risk factors for EAMC were a past history of the condition and competing at a higher than usual exercise intensity.[6] Importantly, the available data suggest that EAMC is associated with running conditions that can lead to premature muscle fatigue in runners who have a history of the condition.[7]

PATHOPHYSIOLOGY

EAMC may be caused by a combination of factors, but muscle fatigue is likely to be the principle factor. As muscle fatigue develops, there is an association with increased excitatory and decreased inhibitory signals to the alpha motor neurons; if muscle contraction continues then muscle cramping results. Effective immediate treatment is to increase inhibitory input to the muscle, either by stretching or by electrical stimulation of the tendon.[8] However, science has not emphatically disproven earlier theories that EAMC is related to abnormal serum electrolyte concentrations, dehydration or environmental stress, and there is a paucity of rigorous scientific research addressing these theories. The aetiology is most likely multifactorial, and some athletes are more susceptible to EAMC than others, given their genetic endowment and physiological response to exercise.

Different theories for the aetiology of EAMC are discussed in the box on page 63.[4,9-24]

TREATMENT

There are many interventions available for the prevention or treatment of muscle cramps – most notably, stretching of an acute cramp. Much of the available scientific data for treatment is aimed at nighttime calf cramps. However, no drug therapy has demonstrated adequate efficacy for nocturnal cramping.

Quinine has been used to treat cramps of all causes. A Cochrane review of 23 clinical trials has concluded that there is moderate quality evidence that quinine reduces cramp frequency, intensity and cramp days, but not duration, compared with placebo, and that there is a significantly greater risk of minor adverse events for quinine compared with placebo.[25] In 2004, the TGA withdrew approval of quinine for nocturnal muscle cramps because of the risk of thrombocytopenia.[26]

The most commonly reported treatment used to prevent recurrent cramping is magnesium supplementation.[27] However, most users report these supplements to be of little or no help. The efficacy of magnesium for muscle cramps has never been evaluated by systemic review.

Salt tablets are widely used in the athletic population to treat EAMC because they are thought to target abnormal serum electrolytes and dehydration. However, the scientific evidence suggests that salt tablets do not target the principal cause of cramps and are therefore not beneficial.

There is one case report and anecdotal evidence (level 4 evidence-based medicine) for use of pickle juice to treat EAMC. The ingestion of a small volume of this highly salty and acidic brine (30 to 60 mL) is claimed to relieve cramp within 35 seconds.[28] It is unlikely that the effects of pickle juice on muscle cramp duration are due to changes in plasma electrolytes or body fluid chemistry, and the rapidity with which pickle juice relieves electrically induced muscle cramps cannot be attributed to spontaneous cramp cessation, weakness of the induced muscle cramps, a placebo effect, or lack of fluid and electrolyte losses. It is speculated that pickle juice triggers a reflex, probably in the oropharyngeal region, that acts to increase inhibitory neurotransmitter activity in cramping muscles.[28] The

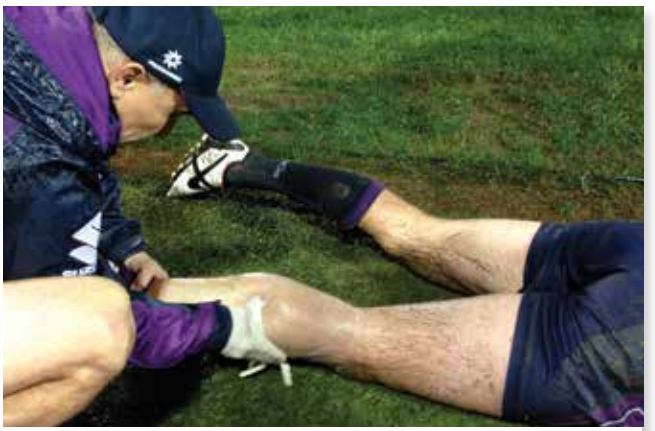


Figure 1. A rugby league football player with a history of EAMC receives soft tissue therapy/massage for treatment of EAMC during the second half of a game.

Figure 2. Two AFL players receiving soft tissue massage as a preventive measure for EAMC.

proposed ingredient that elicits the decrease in cramp duration is acetic acid.

PREVENTION

The pathophysiology causing EAMC is most likely multifactorial and complex and, in turn, prevention of EAMC will need a multifactorial approach. It has been found that athletes who are returning to competition or beginning the functional return to sport phase of rehabilitation after injury are particularly susceptible to EAMC. These athletes are likely to experience early muscle fatigue, to be less acclimatised to a hot environment and to have diminished sweating efficiency, thereby increasing the potential to develop EAMC.[29] From the aforementioned review of the available literature, it is muscle fatigue that is the most likely principal cause. Proper progression during rehabilitation will prevent overstressing the athlete while ensuring adequate sport-specific conditioning before the return to competition.

Unfortunately, there are no proven strategies for the prevention of EAMC. However, regular muscle stretching using post-isometric relaxation techniques, correction of muscle imbalance and posture, adequate conditioning for the activity, mental preparation for competition and avoidance of provocative drugs may be beneficial. Other strategies, such as including plyometric or eccentric muscle strengthening in training programs, maintaining adequate carbohydrate reserves during competition or treating myofascial trigger points, are speculative and require investigation.[30]

OTHER MANAGEMENT STRATEGIES

At the present time, level 1 evidence-based medicine does not exist for the treatment or prevention of EAMC. We surveyed 30 Sports and Exercise Medicine physicians currently practising in Australia and New Zealand for their opinions (unpublished data). Muscular fatigue was thought to be the most likely risk factor and cause of EAMC. Survey respondents believed that useful treatments for EAMC, in addition to those discussed above, may include:

- withdrawal from athletic activity after the onset of first cramp, as this is a sign of fatigue
- active and passive stretching
- active contraction of the antagonist muscle (e.g. dorsiflexors of the ankle for calf cramp)
- heat packs in cold weather
- massage therapy (Figure 1).

Preventive measures identified by the Sports and Exercise Medicine physicians include identifying at-risk athletes and biomechanical and/or gait disturbances or technique errors. Other preventive measures included:

- massage therapy before and during competition (a strategy particularly used in AFL football – Figure 2)
- compression garments
- neural stretching
- sport-specific training
- adequate warm up
- heat acclimatisation
- optimisation of footwear and/or orthotics.

Readers should note that the treatments and preventative measures in this section are not evidence-based.

CONCLUDING COMMENTS

Unfortunately, EAMC remains poorly understood and there is a lack of high level evidence-based medicine to guide management. The pathophysiology is most likely multifactorial, but muscle fatigue and altered neuromuscular control are thought to be central to better understanding, treatment and prevention of this complex condition. MT

REFERENCES

A list of references is included in the website version (www.medicinetoday.com.au) and the iPad app version of this article.

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REFERENCES

1. Schwellnus MP, Drew N, Collins M. Muscle cramping in athletes – risk factors, clinical assessment and management. Clin Sports Med 2008; 1: 183-194.
2. Kantarowski PG, Hiller WD, Garrett WE. Cramping studies in 2600 endurance athletes. Med Sci Sports Exerc 1990; 22: S104.
3. Parisi L, Pierelli F, Amabile G, et al. Muscular cramps: proposals for a new classification. Acta Neurol Scand 2003; 107: 176-186.
4. Schwellnus MP, Derman EW, Noakes TD. Aetiology of skeletal muscle 'cramps' during exercise: a novel hypothesis. J Sports Sci 1997; 15: 277-285.
5. Manjra SI, Schwellnus MP, Noakes TD. Risk factors for exercise associated muscle cramping (EAMC) in marathon runners. Med Sci Sports Exerc 1996; 28(Suppl 5): S167.
6. Drew N. Exercise-associated muscle cramping (EAMC) in Ironman triathletes [dissertation]. Cape Town: University of Cape Town, 2006.
7. Schwellnus MP. Muscle cramping in the marathon: aetiology and risk factors. Sports Med 2007; 37: 364-367.
8. Schwellnus MP. Cause of exercise associated muscle cramps (EAMC) – altered neuromuscular control, dehydration or electrolyte depletion? Br J Sports Med 2009; 43: 401-408.
9. Talbot JH. Heat cramps. Medicine 1935; 14: 323-376.
10. Ladell WSS. Heat cramps. Lancet 1949; 2: 836-839.
11. Armstrong LE, Maresh CM. The exertional heat illness: a risk of athletic participation. Med Exerc Nutr Health 1993; 2: 125-134.
12. Edsall DL. New disorder from heat: a disorder due to exposure to intense heat. JAMA 1908; 51: 1969-1971.
13. Oswald RJW. Saline drink in industrial fatigue. Lancet 1925; 1: 1369-1370.
14. Derrick EH. Heat cramps and uraemic cramps, with special reference to their treatment with sodium chloride. Med J Aust 1934; 2: 612-616.
15. McCance RA. Experimental sodium chloride deficiency in man. Proc R Soc Lond B 1936; 119: 254-268.
16. McCance RA. Sodium deficiencies in clinical medicine. Lancet 1936; 1: 765-768, 823-830.
17. Sulzer NU, Schwellnus MP, Noakes TD. Serum electrolytes in Ironman triathletes with exercise-associated muscle cramping. Med Sci Sports Exerc 2005; 37: 1081-1085.
18. Schwellnus MP, Nicol J, Laubscher R, Noakes TD. Serum electrolyte concentration and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. Br J Sports Med 2004; 38: 488-492.
19. Maughan RJ. Exercise-induced muscle cramp: a prospective biochemical study in marathon runners. J Sports Sci 1986; 4: 31-34.
20. Eichner ER. The role of sodium in 'heat cramping'. Sports Med 2007; 37: 368-370.
21. Bergeron MF. Heat cramps: fluid and electrolyte challenges during tennis in the heat. J Sci Med Sport 2003; 6: 19-27.
22. Nelson LD, Hutton RS. Dynamic and static stretch response in muscle spindle receptors in fatigued muscle. Med Sci Sports Exerc 1986; 18: 69-74.
23. Hutton RS, Nelson LD. Stretch sensitivity of Golgi tendon organs in fatigued gastrocnemius muscle. Med Sci Sports Exerc 1986; 18: 69-74.
24. Jung AP, Bishop PA, Al-Nawwas A, Dale RB. Influence of hydration and electrolyte supplementation on incidence and time to onset of exercise-associated muscle cramps. J Athl Train 2005; 40: 71-75.
25. El-Tawil S, Musa AI, Valli H, Lunn MP, El-Tawil T, Weber M. Quinine for muscle cramps. Cochrane Database Syst Rev 2010; 8: CD005044.
26. Quinine indications – cramps deleted. Aust Adv Drug Reactions Bull 2004; 23: 20.
27. Blyton F, Chuter V, Burns J. Unknotting night-time muscle cramp: a survey of patient experience, help-seeking behaviour and perceived treatment effectiveness. J Foot Ankle Res 2012; 5: 7.
28. Williams RB, Conway DP. Treatment of acute muscle cramps with pickle juice: a case report. J Athl Train 2000; 36: S106.
29. Jung AP. Exercise-associated muscle cramps and functional return to sport. Athl Ther Today 2006; 11: 48-50.
30. Bentley S. Exercise-induced muscle cramp. Proposed mechanisms and management. Sports Med 1996; 216: 409-420.