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REVIEW

Exercise associated muscle cramps: Discussion on causes, prevention and treatment



Crampes musculaires associées à l'exercice : discussion sur les causes, la prévention et le traitement

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Summary

Objective. – To discuss the causes, prevention and treatment of exercise associated muscle cramps (EAMC) according to the level of evidence of the available literature, in order to present some evidence-based guidelines for athletes, coaches and health professionals.

News. – Since it appears fundamental for sports medicine physicians and sports health professionals to be able to manage and prevent EAMC, although pathophysiology and causes of EAMC are discussed, clear understanding of EAMC causes seems important in order to treat and prevent EAMC.

Perspectives and projects. – The present review evaluated the available literature on EAMC based on their level of evidence to present some evidence-based guidelines for sports professionals. Fifty articles were selected: 24 after full-text reading and 26 articles after screening selected articles references. Level of evidence was from 1 ($n=3$), 2 ($n=8$), 3 ($n=10$), 4 ($n=13$), and 5 ($n=16$).

Conclusions. – The “Altered neuromuscular control theory” seems to be the most scientifically acceptable theory, and suggests that EAMC are caused by an imbalance between increased afferent activity (e.g. muscle spindle, Ia) and decreased inhibitory afferent activity (e.g. Golgi tendon organs, Ib) which leads to increased α -motor neuron activity and muscle cramping, especially with muscle contraction in a shortened position. EAMC prevention measures should

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take into account the preparation of muscle to exercise (adapted training) and the respect of muscle fatigue during exercise (warm-up before exercise, well-controlled effort and rest during exercise). EAMC treatments should be non-pharmacological and should play a role on neuromuscular control (rest and/or stretching).

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Résumé

Objectif. – Discuter les causes, la prévention et le traitement des crampes musculaires associées à l'exercice (EAMC) selon le niveau de preuve de la littérature disponible, afin de présenter des recommandations basées sur des preuves pour les athlètes, les entraîneurs et les professionnels de santé.

Actualités. – Comme il semble fondamental pour les médecins du sport et les professionnels de santé en milieu sportif d'être en mesure de gérer et de prévenir les EAMC, bien que la physiopathologie et les causes des EAMC soient discutées, la compréhension claire des causes des EAMC semble être important afin de traiter et de prévenir les EAMC.

Perspectives et projets. – Cette revue de littérature a évalué les articles disponibles sur les EAMC en fonction de leur niveau de preuve afin de présenter des recommandations pour les professionnels du sport. Cinquante articles ont été sélectionnés : 24 après la lecture de texte intégral et 26 articles après la recherche de proche en proche à partir des articles sélectionnées. Le niveau de preuve était de 1 ($n=3$), 2 ($n=8$), 3 ($n=10$), 4 ($n=13$), et 5 ($n=16$).

Conclusions. – La « théorie du contrôle neuromusculaire altéré » semble être la théorie la plus scientifiquement acceptable, et suggère que les EAMC sont causées par un déséquilibre entre l'activité afférente augmenté (par exemple : des fuseaux neuromusculaires, Ia) et la diminution de l'activité afférente inhibiteur (par exemple : l'appareil de Golgi des organes tendineux, Ib) ce qui conduit à une augmentation de l'activité des neurones α -moteur et des crampes musculaires, en particulier avec la contraction des muscles dans une position raccourcie. Les mesures de prévention des EAMC devraient prendre en compte la préparation des muscles à exercer (entraînement adapté) et le respect de la fatigue musculaire lors de l'exercice (échauffement avant l'exercice, effort bien contrôlé et observer des récupérations pendant l'exercice). Les traitements des EAMC doivent être non pharmacologique et devraient jouer un rôle sur le contrôle neuromusculaire (repos et/ou étirement).

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1. Introduction

Exercise associated muscle cramps (EAMC) is a particular muscle cramps associated with exercise [1,2]. It represents a common health problem for athletes. Indeed, in a study following 12 years of the Twin Cities Marathon, Roberts [3] reported an incidence of EAMC of 1.2 cases per 1000 racers, and EAMC represented 6.1% of medical encounters. It represents 6% to 67% of complaints in endurance exercises (triathlon or marathon) [3–6], and 30 to 50% in team sports [7–9]. EAMC leads to pain and musculo-skeletal dysfunction that could induce a decrease in performance [10] and could also lead to muscle damage. Thus, it appears fundamental for sports medicine physicians and sports health professionals to be able to manage and prevent EAMC.

EAMC is defined as: "painful spasmodic involuntary contraction of skeletal muscle that occurs during or immediately after muscular exercise" [1]. Since muscle cramps is a symptom which can occur in many clinical conditions (metabolic dysfunction, neurologic conditions, pregnancy, or exercise) [2,11–13], diagnosis of EAMC should be performed by eliminating through medical examination of other muscle cramps aetiologies (e.g. symptomatic cramps [neurological, muscular, or cardiovascular diseases], or idiopathic cramps [familial, sporadic or others]...)

[1,2,11,14,15]. Although pathophysiology and causes of EAMC are discussed, clear understanding of EAMC causes seems important in order to treat and prevent EAMC.

In this context, the aim of this study was to discuss the causes, prevention and treatment of EAMC according the level of evidence [16] of the available literature, in order to present some evidence-based guidelines for athletes, coaches and health professionals.

2. Methods

With this aim, a systematic search was performed in May 2014 on PubMed database for articles about EAMC published using the following keywords combination: ("Muscle Cramp"[Mesh] AND "Exercise"[Mesh]) OR ("Exercise associated muscle cramps"). Only articles in English were selected. Articles were included if they deal about EAMC and/or muscle cramps occurring in relation to exercise. Articles were firstly selected on the basis of the title, then on the abstract, and then full articles were read. References of selected articles were screened for other appropriate articles, using the same inclusion criteria: articles in English dealing about EAMC and/or muscle cramps occurring in relation to exercise.

The Oxford Centre for evidence-based medicine levels of evidence taxonomy, developed by Phillips et al. [16], was used to characterize the quality, quantity, and consistency of the included articles in order to discuss their relevance for evidence-based sports medicine.

3. Results

With the combination of keywords, the literature search on PubMed database revealed 84 articles. Fifty-seven articles were excluded after reading the titles, and 3 from the abstracts. Twenty-four articles were selected after full-text reading. In addition, 26 articles were selected based on the screening of references of the selected articles. Thus, a total of 50 articles were selected for this discussion.

Among these 50 articles, 20 were literature review including 3 systematic review (with homogeneity) of randomized controlled trials (RCTs) (level of evidence [LoE]: 1) [17–19], 1 systematic review of prospective studies (LoE: 2) [20], and 16 narrative review (LoE: 5) [1,2,11–14,21–31], and 30 were original studies including 7 prospective studies (LoE: 2) [4–6,9,32–34], 10 case-control studies (LoE: 3) [10,35–43], and 13 case reports/series (LoE: 4) [7,8,44–54].

Among these 50 articles, 27 articles deal with EAMC [1,4–10,21–27,32–36,38,39,50–54], 7 articles deal with electrically induced muscle cramps [40–47], 7 deal with “heat cramps” [28–31,37,48,49], and 9 deal with muscle cramps without clear diagnosis [2,11–14,17–20].

4. Discussion

4.1. Causes

Numerous theories for the aetiology of EAMC have been proposed: “serum electrolyte and dehydration theory” based on hypothesis of imbalance in fluid and electrolyte induced by sweat during exercise, “metabolic abnormalities theory” based on the description of muscle cell abnormalities in subjects with cramps, “environmental theory” based on case reports of muscle cramps occurring during exercises in extreme environmental conditions, and “altered neuromuscular control theory” based on hypothesis of the role of muscle fatigue in the EAMC development [1,26].

Since muscle cramps in subjects with metabolic abnormalities are not EAMC [1,2,11], the “metabolic abnormalities theory” cannot be discussed as aetiology for EAMC [1].

Moreover, since EAMC can occur whether it is hot or cold, therefore, environment alone does not induce muscle cramps, and muscle cramps at heat results to secondary physiological changes [1,24,26]. In addition, Armstrong et al. [28] insisted on the fact that EAMC are different than “heat cramps”, although EAMC can occur in heat conditions. “Heat cramps” have been described as the extreme end of the EAMC spectrum [30,31]. Thus, information from studies dealing with “heat cramps” cannot support the theory of cause, prevention and treatment for EAMC, and the “environmental theory” cannot be discussed as aetiology for EAMC [1].

Thus, two theories for EAMC causes can currently be discussed: “serum electrolyte and dehydration theory” and “altered neuromuscular control theory”.

4.1.1. Serum electrolyte and dehydration theory

This theory suggested that decreases in electrolyte concentrations of sodium, potassium, magnesium, chloride, and/or calcium for “serum electrolyte theory”, and decreases in body mass, blood volume, and plasma volume for “dehydration theory”, induced by exercise (result of sweating), may be causative factors of EAMC [1,10,22,23,39]. Exercise-induced sweating causes fluid to shift from interstitium to intravascular space [22], which alters excitability on selected nerves [22,24].

This “serum electrolytes and dehydration theory” has been traditionally and initially hypothesised/extrapolated in comparison to “heat cramps” causes. Indeed, “heat cramps” seems to be a consequence of fluid and electrolyte losses induced by heavy and salty sweating in heat conditions [29,30,37,48,49]. This has been hypothesised from case reports/series (LoE: 4) [48,49], case-control study in football players (LoE: 3) [37], and an expert opinion narrative review including data without clear explanation of their measurements (LoE: 5) [29]. Given the fact that EAMC often occurs during endurance or prolonged exercise [3–6], it has been suggested that EAMC could be due to the change/decrease in hydration and serum electrolyte status induced by exercise [22,24,28]. Moreover, exercise-induced electrolyte losses were reported in subjects who benefited rehydration with intravenous saline solution for severe muscle cramps, but without comparison to control group (LoE: 4) [8].

However, these studies have a poor level of evidence (from 3 to 5). Majority of these studies deal with “heat cramps” [29,30,37,48,49], making these results not applicable for EAMC, and especially EAMC occurring in normal or cold conditions. Moreover, it seems that larger exercise-induced sweat losses are adequately and physiologically tolerated by concomitant fluid and electrolyte compartments adjustments [55]. In addition, 4 well-design prospective studies [4–6,32] (LoE: 2) and a case-control study [38] (LoE: 3) in long-duration exercises (marathon and triathlon), and two humans laboratory-based studies [39,46] (LoE: 4), reported no relationships between EAMC occurrence and loss of serum electrolyte and/or dehydration status, and/or no clinically significant alterations in serum electrolyte concentrations and/or hydration status in subjects with EAMC. Braulick et al. [10], in a well-designed case-controlled study (LoE: 3), reported no changes in EAMC susceptibility when significant and serious hypohydration with moderate electrolyte losses were induced in subjects. Moreover, cramp threshold frequency was unchanged after hypohydration with minimal muscle fatigue [46].

In this context, scientific evidence for supporting this “serum electrolytes and dehydration theory” was poor (LoE: 3–5) compared to high level of evidence of studies against (LoE: 2–4). Therefore, currently and in agreement with previous publications [1,24,26], this theory of EAMC causes cannot be scientifically promoted.

4.1.2. "Altered neuromuscular control theory"

Schweltnus et al. [1,26] postulated that muscle fatigue alters α -motor neuron control and causes EAMC through abnormal reflex activity. Indeed, an imbalance between increased afferent activity (e.g. muscle spindle, Ia) and decreased inhibitory afferent activity (e.g. Golgi tendon organs (GTOs), Ib) leads to increased α -motor neuron activity and muscle cramping, especially with muscle contraction in a shortened position [1,21,24,26]. Primary factors in the development of EAMC are: "increased exercise intensity or duration, development of muscle fatigue, muscle contraction in a shortened position, and possible tissue damage" [26,35].

Arguments in favour of this "altered neuromuscular control theory" were from 4 well-design prospective field studies [5,6,32,33] (LoE: 2) and two case-control studies [34,36] (LoE: 3), which reported that EAMC occurrence has been associated with muscle fatigue. Higher EAMC rate was reported towards the end of races [4,5,33,34,36]. In runners, Schweltnus et al. [33] reported that "higher training time within the 3 days before the event, faster running time for the first half of the race, increased exercise duration" were associated with EAMC. In triathletes, Schweltnus et al. [6] reported that "faster race time during Ironman triathlon with similar past-training and past-performances of athletes, faster speed and faster relative (to personal best) speed" were associated with EAMC. Moreover, animal experimentations [45,47] (LoE: 4) reported that "neuromuscular fatigue appeared to decrease the inhibition from the GTO and increase the excitatory stimuli from muscle spindles" [24]. Humans laboratory-based studies [42,44,56] reported that repetitive muscle contraction results in fatigue and can lead to muscle cramping. A field case-control study [38] (LoE: 3) reported higher baseline EMG activity in cramping muscle than in non-cramping control muscle.

However, all these mechanisms have not been clearly described and scientifically supported, and methodological limits on these experimental studies have been discussed [24,43]. It is important to note that animal experimentation and humans laboratory studies were not always focused on EAMC, but also analysed mechanisms of muscle cramps in general. Thus, their application to the pathophysiology/mechanisms of EAMC should be taken into account with caution. Central or peripheral origin of the "altered neuromuscular control theory" mechanisms is discussed [11,13,14,24,40,42].

Thus, in agreement with previous publications [1,24,26], this "altered neuromuscular control theory" can be scientifically promoted given the high level of scientific evidence (LoE: 2–4).

Finally, further researches should also be conducted on the "soft tissue theory", since COL5A1 genotype has been reported to be associated with EAMC past-year history in a retrospective case-control study (LoE: 4) [35].

Moreover, Miller [24] suggested that because EAMC occur in a variety of situations, environmental conditions, and populations, it is unlikely that a single factor (e.g. dehydration, electrolyte imbalance, or neuromuscular factors) is responsible for causing them directly. It is more likely that EAMC are due to a combination of factors that simultaneously occur under specific physiological circumstances in each athlete. Indeed, other parameters have been

suggested to predispose to EAMC: past-history of EAMC, history of tendon and/or ligament injury, low back pain [6,9,24,33,35,36].

4.2. Prevention of EAMC

Some factors/causes have been reported to be associated with EAMC occurrence: "higher training time within the 3 days before the event, faster running time for the first half of the race, higher sub-clinical pre-race muscle damage, higher stretching before exercise, increased exercise duration, and muscle fatigue" [6,10,26,33]. Thus, several potential prevention measures for EAMC can be suggested, although these are only hypotheses, which are not supported by scientific studies, and their effectiveness should be analysed in future prospective studies and/or randomized controlled trials (RCTs):

- to perform an adapted training to prepare competition, by increasing neuromuscular endurance and/or correcting muscle imbalances (plyometric exercises) [10,15,24];
- to decrease training volume/intensity and exercises inducing muscle damage during the days before exercise;
- to perform a warm-up before exercise;
- to do reasonable/moderate stretching before exercise [11,24];
- to start slowly exercise with a lower/reasonable speed and/or a well-controlled effort;
- to respect pause/rest during exercise and/or between exercises/competitions [15,28].

Pycnogenol® has been reported as efficient for EAMC prevention in case series (LoE: 4), but further studies should confirm this result [52].

Since "serum electrolyte and dehydration theory" does not seem to be scientifically acceptable, maintaining fluid and salt balance during exercise [28,29,31] do not appear relevant to prevent EAMC. However, for preventing EAMC occurring in heat conditions, it seems to be relevant to follow the recommendations of heat illness prevention, and especially "heat cramps" prevention recommendations [22,28,29,31]. Thus, adapting schedule of exercise/competition to avoid heat conditions, and maintaining fluid and salt balance during exercise in heat conditions, could be suggested, according to the "heat cramps" prevention recommendations [22,28,29,31].

4.3. Treatment of EAMC

Some treatments have been evaluated for EAMC, and other treatments can be hypothesised from the EAMC causes discussion.

4.3.1. Non-pharmacological treatments

- Rest: when EAMC occurs it has been suggested to stop exercise and to observe a recovery time [28].
- Stretching: when EAMC occurs, it has been suggested to do prolonged stretch with the muscle groups at full length [24,28,44]. Stretching may restore the physiological relationship between excitatory and inhibitory impulses via inhibition of the α -motor neuron caused by GTO activation

[24,26]. Case report/series (LoE: 4) reported a decrease in EMG activity after stretching in subjects with EAMC [51], and effectiveness of stretching to stop/treat EAMC [44,54]. This simple low-risk treatment should first be suggested to all patients with EAMC.

- Hyperventilation: hyperventilation (deep frequent breaths, approximately 20–30/min) has been reported as 100% efficacy without side-effects in a small series of 3 cases (LoE: 4) [53].

Other non-pharmacologic interventions have been suggested without high scientific support: ice, massage, heat, walking, leg elevation, leg jiggling, pickle juice... [22,24,41].

Based on the fact that “altered neuromuscular control theory” seems to be the most scientifically acceptable theory, all these non-pharmacological treatments, which play a role on neuromuscular control seem to be relevant for treating EAMC (rest and/or stretching), although not high quality study (RCT) scientifically supports it [19].

4.3.2. Pharmacologic treatments

- Sodium: oral (or intravenous) NaCl ingestion in fluids or foods is not supported by high quality studies (e.g. RCTs) and is supported by studies on “heat cramps”, and is based on “serum electrolyte and dehydration theory” which is not supported by high level of evidence [22,28–31,49]. In practice, in normal climatic conditions, oral NaCl ingestion does not seem to be relevant to treat EAMC, such as to prevent EAMC. However, in heat conditions, recommendations for heat illness prevention should be followed, and oral NaCl ingestion should be relevant [22,28,29,31].
- Magnesium: few evidences have been reported to support magnesium supplementation use for idiopathic cramps and pregnancy-associated leg cramps, and no RCTs have been found on magnesium supplementation for EAMC [18].
- Creatine: an open study (LoE: 4) reported lower rate of cramping in football players using creatine supplementation, however, it is not clear whether it studied EAMC [7,50].
- Quinine: although quinine sulfate remains an effective treatment for muscle cramps, the high risk/benefit ratio makes this treatment unwarranted [11,17,20]. Moreover, these results of effectiveness were not for EAMC, thus, there is insufficient data to support the use of quinine for EAMC treatment.

Other pharmacological treatments (calcium salts, sodium bicarbonate, sodium channel-blocking agents (carbamazepine, phenytoin), naftidrofuryl, calcium channel-blockers, verapamil, diltiazem, vitamin B, vitamin E, lidocaine, gabapentin, botulinic toxin, dilantin) have been proposed for treating muscle cramps. Their effectiveness were analysed in studies with a low level of evidence (grade of recommendation C) without focusing on EAMC, and reveal potential severe side-effects without legal authorization in this indication [11,20].

Based on these results, pharmacological treatments should not be promoted to treat EAMC (few evidence for

treating muscle cramps from other aetiologies and no evidence for treating EAMC).

5. Conclusions

The “altered neuromuscular control theory” seems to be the most scientifically acceptable theory based on the available scientific literature and its level of evidence. Following this theory, EAMC seem to be caused by an imbalance between increased afferent activity (e.g. muscle spindle, Ia) and decreased inhibitory afferent activity (e.g. Golgi tendon organs (GTOs), Ib) which leads to increased α -motor neuron activity and muscle cramping, especially with muscle contraction in a shortened position [1,21,24,26]. EAMC prevention measures should take into account the preparation of muscle to exercise (adapted training) and the respect of muscle fatigue during exercise (warm-up before exercise, well-controlled effort and rest during exercise). EAMC treatments should be non-pharmacological and should play a role on neuromuscular control (rest and/or stretching).

Disclosure of interest

The author declares that he has no conflicts of interest concerning this article.

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