The Pathophysiology and Care of Exercise Related Muscle Cramps

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ABSTRACT

Cramps are major concerns to competing athletes occurring during or after exercise, are common yet, poorly understood phenomena. Pain alone is not object of treatment as serious musculophysiologic and metabolic disturbance of fluid and electrolyte deserve correction. Acute muscle pain and stiffness may cause soreness for longer time. Based on observations, two etiological theories are construed, i.e. the muscle fatigue theory and sodium-water deficit theory. Either has supporting and contradicting facts, but these are relevant to guide prevention and management interventions. Cramps may be different in kind based on different local and/or general causes. Occurrence of cramps in varied situations, environmental conditions and populations suggests of pleural causal determinants. These include neuromuscular and fluid-electrolyte disturbance factors directly responsible under specific circumstances of individual sports person. Degree of conditioning to particular kind of physical exercise appears most significant factor. Prevention exercises target theorised physiology of muscle tendon and golgi organ receptors, toward delaying fatigability and cramp risk. Occurrence of cramps mostly in hot environments emphasizes support to dehydration-electrolyte imbalance theory. Maintenance of hydration and adequate electrolyte levels in cramp-prone individuals thus makes sense. Worth of variety of measures empirically employed for cramp relief can be judged by scientific understanding. Drugs found useful may not be the best match to pathophysiologic proprieties and thus irrational. The pathophysiological details and relevant clinical information is presented and discussed as first hand understanding for the sports persons and their care givers.

Keywords: exercise cramps, heat exhaustion, muscle fatigue, sports medicine, sweat loss

Athletes commonly suffer muscle cramps that create fear of appearing without warning or apparent cause during competition. Exercise related muscle cramps are painful spasmodic involuntary contraction of skeletal muscle that occur during or immediately following extensive exercise. The musculoskeletal dysfunction may persist to compromise competitive performance besides leading to muscle damage [1]. Cramps may often localize in over worked, fatigued muscle due to inadequate conditioning. More generalised exertional heat cramps occur due to excessive sweating and sodium loss during or after exercise. Distinction between the two is crucial toward appropriate preventive and alleviating strategies. Prominent two of the proposed causal theories of cramping are muscle fatigue or disrupted neuro muscular control and the sodium deficit-dehydration theory that have guided prevention and treatment efforts [2].

FATIGUE CRAMPS

As a distinction, fatigue cramps are localized to over-loaded and fatigued muscle group; and sometimes spread slowly across the involved muscle region without jumping [3]. The local fatigue implies an excitatory alteration (increase) in afferent activity in muscle spindle. There is concomitant decrease in inhibition in Golgi tendon organ, resulting in uncontrolled sustained activity of α-motor neurons [2, 4-6]. Inhibitory neural mechanism of muscle contraction, when tendon organ senses increased muscle tension, is disrupted. Enhanced excitatory activity in muscle spindle triggers unopposed sustained involuntary muscle contraction, without control of tendon organ. Altered depolarization threshold at neuro-motor endplate results in vul-
nerability to cramp [7], as inhibitory activity of golgi tendon organ is low in shortened position of muscle [4]. Primary factors in development of fatigue are intensity and duration of exercise, muscle contraction under shortened state and possibly, tissue damage [8]. Old age, poor stretching habit, insufficient conditioning, history of cramping, excessive exercise duration and intensity and related metabolic disturbances, are all predisposing risk factors [6,9]. Study in humans showed higher baseline EMG activity in cramping muscle in comparison to non cramping muscle [10]. Central as well as peripheral origins of the altered neuromuscular control have been proposed [11-13].

CRAMPS DUE TO SODIUM DEFICIT

Sizeable deficit of exchangeable sodium develops when sodium and chloride loss in sweat, exceeds salt intake [14]. About 20-30% loss in exchangeable sodium associates severe muscle cramping [15]. The sweating rate, sweat sodium concentration (20-80 mmol/L) and dietary intake of salt, determine the rapidity with which cramps may develop. The sweat sodium concentration does not decepte with decrease in whole body sodium and water content. Sweating rate also remains constant over long term physical activity. Even the serum sodium concentration is not allowed to fall due to changes in sweat gland behaviour or sympathetic nervous activity, which increases sweat sodium concentration [16]. Other electrolytes like calcium, potassium and magnesium are also lost in sweating. The cramp prone athletes however, characteristically show sodium deficit due to inadequate dietary intake [14].

Water from interstitial compartment shifts to intravascular compartment to compensate loss of plasma volume during exercise due to sweating. Interstitial fluid therefore, contracts with continued exercise [17]. This continues even after exercise, due to continued sweating and cooling of body [18]. Plasma osmolality and electrolyte concentration are maintained or slightly elevated. As exercise related cramps occur during endurance or prolonged exercise, they are suspected to result from deficit of hydration and electrolyte, especially, sodium [19,20]. Body does not store enough water for exercise. Fluid and electrolyte depletion sensitizes select nerve terminals [21]. Dehydration induced contracture of interstitial space, presses on the motor nerves that may culminate in cramp.

Braulick et al. [1], induced hypo-hydration with moderate electrolyte loss in subjects, but did not detect increased susceptibility to exercise related cramps. Cramp threshold frequency remained unchanged despite hypo-hydration. Ingestion of carbohydrate-electrolyte fluid matching the losses in sweat did not appear to protect in 69% of cramp sufferers following exercise, in another study [22]. Stretching of the affected muscle offers immediate relief, while fluid electrolyte status remains same [2]. Maintenance of fluid and salt balance during exercise in hot environment however, is clearly relevant to prevention of heat cramps [14].

The type and the amount of fluid taken by the athlete however, may change electrolyte and fluid shift [23]. At a level of dehydration, lowered plasma sodium concentration and continued high sodium sweating would lower osmotic drive of plasma and delay maintenance of plasma volume through fluid shift [24]. In such state, low sweating rate will cause significant sodium deficit and risk of cramp only on extended period of exercise. High rate of sweating would accelerate loss of plasma volume and cause muscle cramping [25]. These athletes are ‘salty’ sweaters and are at high risk of rapid development of exertional heat cramps [26]. Other athletes may suffer similarly, if their sweating rate is high and exercise duration is prolonged [27].

Contraction of interstitial fluid compartment would cause mechanical distortion of some neuromuscular junction and the unmyelinated nerve terminals. The synaptic membrane would face more concentrated exercise-related metabolites, electrolytes and acetylcholine in extracellular milieu. These may cause the nerve fibers to independently mount end plate excitatory post synaptic potential, or fire [21,28]. Surface electromyographic analysis has verified occurrence of action potentials initiated from axon terminals of alpha-motor neurons during cramping [3]. Further contraction of interstitial fluid compartment would spread such exaggerated excitability among surrounding muscles resulting in jump of the cramps [29].

APPLIED PERSPECTIVES

Overload and fatigue related local cramps may resolve with passive stretching, massage, active antagonist muscle contraction or ice pack on affected muscles. Proper exercise training and conditioning as well as reduced exercise load and duration help to avoid these [30,31]. Fatigue cramps would not permit continued activity. Stretching at full length with afflicted muscle group, can restore the physiological relationship between excitatory and inhibitory impulses [32]. Decrease in EMG activity was observed after stretching in exercise related cramp victims [33]. Stretching is
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CONCLUDING REMARK

Sudden significant localized cramping is often the fatigue type. The sodium deficit cramps grow over minor fasciculation’s with wider spread, accompanied by intermittent spasms not relieved by stretching. These would follow severe sweating noticeable from garments. It is noteworthy that athletes may have both kind of cramps requiring keen detection and appropriate address. To prevent exercise related cramps, clinicians should take detailed medical history of each athlete, determine their level of conditioning and monitor their fluid and electrolyte levels. The approach to treating and preventing cramps should be multifaceted as multiple causes are implicated. As most reliable therapy measure, clinician should stretch the cramping muscle. Once the cramp is alleviated, hydration and electrolyte needs must be assessed and addressed. Plyometric exercises that target neuromuscular system may be beneficial to elicit neural adaptations in muscle spindle and golgi tendon organ receptor activity. That may enhance efficiency and sensitivity of reflexive and descending pathways used for neuromuscular control [48]. Because exercise related cramp is a temporarily debilitating condition, prevention is of great interest. It is important for athletes to be well conditioned on return to sports in order to prevent premature muscle fatigue. It is important, however, to use proper progression (intensity, duration, and frequency) during rehabilitation to avoid overstressing the athlete. In combination these factors might decrease the incidence of exercise cramps in athletes during and after functional phases of rehabilitation.

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