Exercise Associated Muscle Cramping (EAMC)

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Objectives

- Evaluate the theories on cramping
- Discuss Tx and prevention strategies
- Explain myths on cramping
- Clinical presentation & diagnosis
- Risk factors
- Case study
Definition

- “cramp” – old German/Norse root suggesting squeezing, pressing, or pinching uncomfortably
- EAMC – “painful, spasmodic and involuntary contraction of skeletal muscle that occurs during or immediately after exercise”
Theories

- Dehydration
- Electrolyte Depletion
- Environmental
- Metabolic
- Altered Neuromuscular Control
Dehydration Theory

- Cramps happen because athletes exercise in the heat, lose electrolytes in their sweat, and the depletion combined with high body temperature cause muscle cramp

- This theory states that when an individual is dehydrated, the decrease in body mass, blood volume, and plasma volume leads to muscle cramping
Dehydration Theory

- “Heat” or “Miner’s” cramp
- 100 years of case studies linking cramping to ‘Stokers’, miner’s and Hoover dam workers (no info on those who did not cramp)
- Hydration status not changed at the time of acute symptoms
- Systemic symptoms not working muscle
- Lack of sodium will cause cramping
Electrolyte Theory

The Electrolyte theory is based on observations that the involuntary muscle contractions are occurring in individuals who have a decreased concentration of electrolytes, such as sodium, potassium, magnesium, or calcium.
Electrolyte Theory

- No published studies that has shown that serum electrolyte concentrations are abnormal at the time of acute EAMC, when compared to non-cramping control group.

- When cramps subside and become asymptomatic, there is no change in serum electrolyte concentrations.
Electrolyte Theory

- Sweat sodium concentration is always hypotonic – a significant loss of sodium through sweat can therefore only occur if there is an accompanying large loss in fluid

- Study flaws – sweat concentration not collected during EAMC episode; only 23 subjects ever tested (football study done during camp when athletes are less conditioned)
Electrolyte Theory

- Systemic abnormality
- Tx for theory is rest, passive stretching and sodium intake. What does rest and stretching have to do with electrolytes?
- When you sweat you don’t actually reduce electrolyte concentration. When you sweat you lose more water than electrolytes, because sweat is hypotonic. Therefore sweating can not lead to a fall in electrolyte concentration.
- Lack of electrolytes will cause cramping
Metabolic Theory

- Abnormalities of muscle cell substrate metabolism
- Congenital or acquired diseases
- Rare
- Metabolic changes will cause cramping
Altered Neuromuscular Theory

- They postulate that cramping occurs due to an abnormality of sustained alpha motor neuron activity, which continues the stimulus to the muscle to contract. As the muscle fatigues, an excitatory effect on the muscle spindle and an inhibitory effect on the golgi tendon organ affect the activity of the muscle, thus leading to the cramp
Altered Neuromuscular Theory

- Muscle contraction is initiated by a nerve, called the alpha motor neuron. The alpha motor neuron receives input from the higher brain areas as well as from the spinal reflex.

- These reflexes are responsible for protecting the muscle against either excessive stretching or loading – they are the muscle spindles and Golgi tendon organs, respectively.
Altered Neuromuscular Theory

- There is evidence that fatigue causes increased firing from the muscle spindles, and decreased activity from the Golgi tendon organs.

- The net result of this change in the activity of these reflexes is that the alpha motor neuron activity is increased, and the muscle thus contracts involuntary.
Altered Neuromuscular Theory

- Muscle spindle reflex
  - Make sure muscle doesn’t over stretch
  - Stretch -> sends signal by Type Ia Afferents to spinal cord -> nerve impulse is passed on to the alpha motor neuron and back to muscle
  - End result is if you stretch, your muscle will contract
  - Knee-jerk reflex
Altered Neuromuscular Theory

- Golgi tendon organs
  - Role is to make sure the muscle doesn’t contract too forcefully or under too much load
  - Muscle contraction -> GTO fires -> sends signal to spinal cord along Type Ib Afferent
  - Type Ib Afferents tell the alpha motor neuron to stop firing – they are inhibitory
  - End result is the GTO is stimulated, the muscle contraction is switched off
However, if the GTO is inhibited, then the alpha motor neuron activity will increase, and the muscle will contract even more – this is called “disinhibition”
Altered Neuromuscular Theory

- So when a muscle becomes fatigued, the Type Ia Afferent fibers from the muscle spindle INCREASES (contraction) and the firing rate from the Type Ib fibers from the GTO DECREASES (contraction)
Altered Neuromuscular Theory

- Which muscles are more likely to cramp?
  - Active muscles that fatigue

- What kind of muscle cramps most often?
  - 2 joint muscle which contracts during a shortened position

- When is cramp most likely to occur?
  - Racing not training, end of competition
Altered Neuromuscular Theory

- Is there any evidence for the theory?
  - Electrical activity of muscles cramping in runners was measured after 56 km marathon, and it was found that the alpha motor neuron activity was higher than in non-cramping athletes
  - Electrolyte theory can’t explain this.
  - 20 seconds of passive stretching, the EMG activity goes down
Figure 1: The “electrolyte depletion” hypothesis for the development of Exercise Associated Muscle Cramping (EAMC)

Physical exercise with resultant sweating

Abnormally high sodium sweat concentrations (20-80 meq/L)

Sodium loss from plasma and has been suggested to cause hypotonic hyponatremia (tends to decrease osmolality)

No fluid will move from interstitial space to the intravascular space

Conflicting argument

Abnormally high sweat volume (sweat is hypotonic)

Contracting (decreasing) plasma (intravascular) volume (tends to increase plasma osmolality)

Fluid moves from interstitial space into the intravascular space

Decreased interstitial fluid compartment volume

“Certain” motor neuron axon terminals become hyper excitable by, 1) mechanical deformation, 2) exposure to increased levels of excitatory extracellular constituents such as acetylcholine, electrolytes, and exercise-related metabolites in the surrounding extracellular space

“Certain” nerve terminals are at greater risk of spontaneously discharging

Starts as localized muscle cramping

? 500ml bolus of oral fluid

Restoration of plasma volume

Treatment by fluid intake

Generalized muscle cramping
Figure 2: The “altered neuromuscular control” hypothesis for the development of Exercise Associated Muscle Cramping (EAMC)

Repetitive muscle exercise

- Increased exercise intensity
- Increased exercise duration
- Decreased muscle energy

? Muscle injury/damage

? Reflex contraction

Development of muscle fatigue

- Hot and/or humid environmental conditions
- Inadequate conditioning
- Contraction of a muscle in a shortened position (inner range)
- Altered Central Nervous System function
- Decreased inhibitory afferent activity (e.g., Golgi tendon organ)
- Increased excitatory afferent activity (e.g., muscle spindle)

? Genetic predisposition

Altered neuromuscular control (spinal)

- Increased alpha motor neuron activity (spinal)

Increased muscle cell membrane activity

Muscle cramping

Reflex inhibition

Treatment by passive stretching
Environmental Theory

- The environmental theory is based on the fact that when athletes are exposed to extreme environmental conditions, such as high heat and humidity, they are more susceptible to cramps. With this condition, athletes have lost a significant amount of fluids through sweat and thus have an electrolyte imbalance (as in the electrolyte and dehydration theories), which leads to muscle cramping.
Environmental Theory

- Passive heating alone (at rest) does not result in cramping and cooling does not relieve cramps.
- Likely that exercise in heat may result in secondary physiological changes which can cause EAMC.
Clinical Presentation

- EAMC is more likely to occur when intense prolonged exercise is performed in a competitive environment under hot and humid environmental conditions.
- Onset of EAMC is usually preceded by the development of skeletal muscle fatigue, often in athletes that are not well conditioned for the event.
Clinical Presentation

- Cramping is usually preceded by a noticeable twitching of the muscle ("cramp prone state") and is followed by spasmodic spontaneous contractions and frank muscle cramping if the activity is continued.

- Pain in the muscle, that usually develops gradually over a few minutes during intense or prolonged exercise.
Clinical Presentation

- Relief from the “cramp prone state” which occurs if the activity is stopped or if the muscle is stretched passively.
- Episodes of cramping are usually followed by periods of relief from cramping, once activity is ceased.
- Cramping can be precipitated by contraction of the muscle in a shortened position during the “cramp prone state.”
Clinical Presentation

- In the majority of cases, muscle cramping is confined to muscle groups that are very active during the athletic event – most commonly these are the calf, hamstring and quadriceps muscle group.
- In most cases, EAMC lasts for a few minutes to a few hours once activity is ceased.
Clinical Presentation

- EAMC is more common when exercise is performed in a competitive environment.
Clinical Presentation

- An athlete with EAMC typically shows obvious distress, pain, a hard contracted muscle, and visible twitching over the muscle belly.
Clinical Presentation

- In most instances the athlete is conscious, responds normally to stimuli, and is able to conduct a conversation. Vital signs and a general examination usually reveal no abnormalities. In particular, most athletes with acute cramping are not dehydrated or do not have an excessively high body temperature.
Clinical Presentation

- An athlete who has generalized severe cramping or is confused, semi-comatose, or comatose should be treated as an emergency and requires immediate hospitalization where full investigation is required.
Management

- Stop activity and rest
- Passively stretching the affected group
- Administer oral fluids containing carbohydrates / with or without electrolytes
- Return athlete to a comfortable body temperature
- Check urine color for 24 hrs
Diagnostic Approach

- Is the cramping precipitated by physical exercise of very mild intensity and duration?
- Does the cramping occur at rest?
- Is the cramping associated with any other symptoms, such as paresthesia, pain, decreased sensation, or muscle weakness?
Diagnostic Approach

- Does cramping episode occur during every exercise bout?
- Does passive stretching aggravate, rather than relieve the cramping?
- Is there a strong family history of cramping?
- Does the athlete use any drugs?
- Is cramping associated with dark urine after exercise?
Prevention

☐ Awareness that EAMC is more likely if premature muscle fatigue develops
☐ Awareness that EAMC is more common in hot and humid environmental conditions
☐ Athlete should perform their activity at a lower intensity and shorter duration if they are prone to EAMC
☐ Athlete should be well conditioned for the activity
Prevention

- Athlete should perform regular stretching for the muscle groups that are prone to cramping.
- Athlete should have adequate nutritional intake (particularly carbohydrates) to prevent premature muscle fatigue during exercise, and may need to consult a sports dietician in this regard.
Risk Factors

- Older age (w)
- Longer history of running (w)
- Higher body mass index (w)
- Shorter daily stretching routine (w)
- Irregular stretching habits (w)
- Positive family history of cramping (w)
- High intensity racing (w)
Risk Factors

- Long duration racing (after 30km) (w)
- Subjective fatigue (w)
- Hill running (w)
- Past history of EAMC (s)
- Increased exercise intensity (s)
- Increased environmental temperature and humidity [extrinsic factor] (limited evidence)
Case Study
Case Study

- Male Football player who plays off/def line and some special teams
- No family history of cramps
- Never cramped during practice
- Cramped during games as early as late 2\textsuperscript{nd} quarter
- Primarily cramped in calves, once or twice in hamstrings and only once in quads
Case Study

- Treatment strategies
  - Started with urine refractometer
  - Athlete hydrated heavily starting Thursday
  - Added lots of sodium to food and drinks (gatorlytes) = 1 tsp to 20 oz gatorade
  - Started research on cramping and learned about EAMC
Case Study

- Started to give carbohydrates to athlete before game and at half time in the form of gummy bears & swedish fish
- Started stretching program before game and at half time
- Talked to coach about resting him when possible
- Kept with hydration & gatorlytes
Thoughts

- The intense and never rest athlete (Grove)
- Pickle juice Tx 2-5 fl oz followed by beverage (50 fl oz water) 10 minutes before exercise
  - Whole 220mg  sliced 390 mg
  - Exceeds NATA guidelines
  - Gastric and palatability issues
Thoughts

- Acetylcholine (acetic acid)
  - Pickle juice, mustard and vinegar
  - Must be pure apple cider vinegar
  - Released through massage

- Quinine
  - Too risky side effects
  - Little evidence with athletes

- Heat guard, etc.,
Summary

- 5 theories of muscle cramping
- Tx and prevention strategies
- Discussed the myths behind cramping
- Show clinical signs & diagnostic approach to determine EAMC
- Stated risk factors
- If it works for you, keep doing it
Resources

Resources