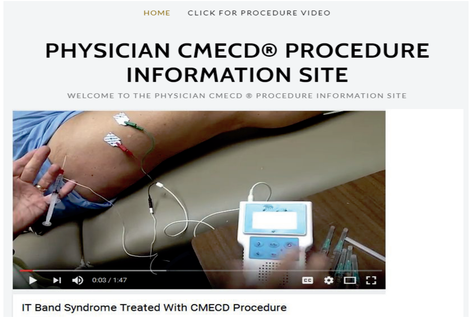
Lorem ipsum

For full information visit the CMECD.info



PATTERN OF RECOVERY OF ACQUIRED CHRONIC MUSCLE SPASM
CONSISTENT WITH ISCHEMIC INJURY MODEL

Introduction:
It is known that the blood supply of cardiac muscle is lessened by cardiac contraction. Flow is significantly less in systole than diastole even though the perfusion pressure is higher in systole. Prior unpublished work by this author demonstrated that with a weakening of the force of contraction, predominant cardiac flow occurred in systole. Microvascular supply of skeletal and cardiac muscle are similar. High heart rates that can occur with atrial fibrillation with near constant contraction can lead to cardiac dysfunction and muscle injury that slowly recovers over a one to two month period. Skeletal muscle in chronic spasm has been shown to have a depleted number of mitochondria that normalize with relief of the chronic spasm.
Objective:
To record patterns of clinical recovery of chronic muscle spasm to find potential consistency with an ischemic injury model.
Methods:
Muscles identified as acquired chronic muscle spasm by EMG according to criteria previously described, were treated with the CMECD® procedure, see CMECD.info.
Results:
Relief of pain occurred early but the capacity of the muscle to perform work, without return to spasm, gradually increased. The severity and extent of the spasm appeared to correlate with the length of time to full recovery noted to occur within a two month span.
Summary/Conclusion:
Clinical findings are supportive of an ischemic injury model of chronic muscle spasm. Expectations of full recovery and physical therapy interventions should be informed by consideration of this model. Assessment of treatment success or failure viewed within the context of this model may improve treatment outcomes.

PATTERN OF RECOVERY OF ACQUIRED CHRONIC MUSCLE SPASM CONSISTENT WITH ISCHEMIC NJURY MODEL

Roger H. Coletti, MD, FACC, FASNC, FSCAI
InterventionalHealth.com

The pathologic change in acquired chronic muscle spasm is the impaired blood supply into the muscle tissue as a result of the sustained spasm of the muscle. Based upon clinical findings, it is postulated that with sustained ischemia, there are ultrastructural changes in the muscle that consist of loss of energy producing structures such as mitochondria. See the study on the right side of this poster demonstrating variability of mitochondrial density and increase in mitochondrial density with eletrical stimulation treatment of the equine chronic muscle spasm. Also shown is the presence of atrophic muscle fibers in equine chronic muscle spasm with improvement in size with electrical stimulation treatment. The sustained spasm of the muscle requires no energy as it is the state of an energy-depleted muscle. This speaks to the question of recovery of the muscle and what must be done to best facilitate its recovery.

The energy depleted tissue lacks the ability to maintain membrane stability resulting in the spontaneous electrical activity which secondarily keeps the muscle in chronic spasm. Phenoxybenzamine functionally acts as an antiarrhythmic by blocking the neuromuscular junction receptors on the muscle with a covalent bond. This explains the 2-3 month duration of action that gives adequate time for gradual recovery of the muscle and replenishment of energy producing structures.

A study of chronic muscle spasm in retired race horses examined the ultrastructural changes that occurred with chronic muscle spasm and the normalization with recovery of spasm. The photomicrophagaphs seen on this poster are from that study. Biopsies were taken from these horses at sites where there was clinical evidence of chronic muscle spasm. Subsequent physical treatment of the chronically spastic muscles was performed with resolution of the spasm. Repeat biopsies were then performed. What was found was that in the chronically spastic muscle there was a diminished number of mitochondria and there was initial atrophy of muscle fibers that improved following treatment.

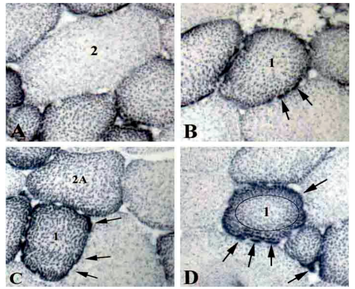
There are important clinical implications of the ischemic model. In evaluation of the individual, it must be taken into account the duration and severity of the chronic spasm. This will be an indication of the expected time required for recovery. Obviously, the longer the duration of the spasm, the greater likelihood of significant ultrastructural change and the longer time needed for recovery toward normal. This model also speaks to potential treatments of chronic muscle spasm. Any treatment modality that can lead to improved blood supply to the chronically spastic muscle should be beneficial. It should be kept in mind that this model speaks to acquired chronic muscle spasm that is not of neurogenic origin. Various types of massage, heat or rhythmic stim/release may be successful in improving blood supply temporarily. However, such therapies will need to be repeated on a frequent, if not daily basis, to provide adequate blood supply for muscle recovery.

The CMECD® procedure takes advantage of the covalent bond formed by phenoxybenzamine onto the neurotransmitter receptor on the muscle. The effect is then for 2-3 months while the muscle then gradually re-supplies the surface receptors. This abates the spontaneous electrical activity (SEA) and prevents the membrane surface activity from constantly stimulating the muscle. In that setting initial partial return of blood supply may occur with then progressive relaxation and recovery.

Based upon the ischemic model, clinical expectations of recovery need to be altered. Muscles treated with the CMECD® procedure or any other procedure that succeeds in getting the chronically spastic muscle to relax must be considered as damaged tissue. Consequently, expectation of activity and especially of prolonged activity need to be altered. Activity that the muscle may have been capable of prior to the onset of chronic spasm will likely overwhelm the muscle, deplete it of energy reserves and head it back into a chronic spasm state. Retraining with very gradual increase in activity should be able to prevent that from happening. However, should the muscle go back into spasm with this relative overuse injury, simple techniques such as heat or massage to relax the muscle should be used to restore the blood supply.

Variability of Mitochondial Density in Skeletal Muscle

FES for equine epaxial muscle spasms: muscle biopsy morphometry
Eur J Transl Myol - Basic Appl Myol 2015; 25 (2): 109-120



Arrows point to mitrchondria

Atrophic Equine Muscle Fibers in
Muscles Demonstrating Clinical Evidence of
Chronic Muscle Spasm

Improved
Fiber Size
Post
Treatment