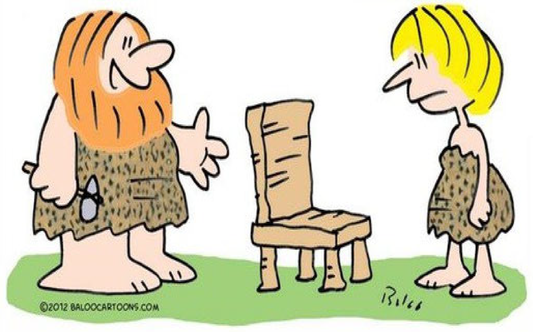


"I just invented the 'chair' —
It relieves lower back pain!"

Treat Patients with
Acquired Chronic Muscle Spasm
Causing Chronic Pain

Acquired chronic muscle spasm is a common cause of chronic pain but goes unrecognized because its characteristics had not specified until now. It can be readily diagnosed with EMG with the finding of spontaneous electrical activity. Botox® can be used for neurogenic or acquired chronic muscle spasm but is expensive and can cause atrophy. However, the technique of CMECD® using phenoxybenzamine has not been shown to cause weakness with multiple uses and is a fraction of the cost. Its use in neurogenic chronic muscle spasm has not yet been explored. Clinical research over the past 10 years has led to the identification of EMG characteristics of acquired chronic muscle spasm. What are needed are physicians with access to an EMG and the interest to treat patients with chronic pain without the use of opioid medications. CMECD.info is a web site that provides all the information and background to begin treating chronic pain caused by chronic muscle spasm. Clinical research has shown that patients with chronic pain secondary to acquired chronic muscle spasm found permanent relief with one or two injections. Many of these patients had suffered from chronic pain for more than five years and still had long term if not permanent relief. The use of this technique is not limited or restricted. The phenoxybenzamine-dexamethasone injectable is available overnight from the sterile compounding pharmacy that was involved in the initial clinical use. Order forms are available on the CMECD.info web site.

Acquired Chronic Muscle Spasm
Treatment and Recovery

Acquired chronic muscle spasm is a pathologic state of muscle that is reversible and not secondary to a neurogenic pathology. Most commonly, acquired chronic muscle spasm is an overuse injury but can be triggered by an acute injury. Denervated muscle will show spontaneous electrical activity similarly to that shown by acquired chronic muscle spasm. However, acquired chronic muscle spasm can recover with treatment of the muscle itself and does not require re-inervation for recovery.

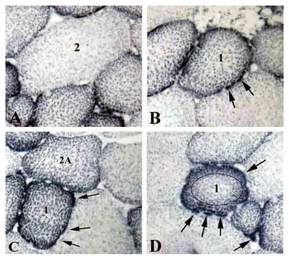
The pathologic change in acquired chronic muscle spasm is the impaired blood supply into the muscle tissue as a result of the sustained spasm of the muscle. Based upon clinical findings, it is postulated that with sustained ischemia, there are ultrastructural changes in the muscle that consist of loss of energy producing structures such as mitochondria. See study below demonstrating variability of mitochondrial density and increase in mitochondrial density with electrical 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.

It is important to keep in mind that muscle injected with phenoxybenzamine, despite the apparent initial success in relief of the muscle spasm, remains impaired. It takes a relatively small amount of overuse to put the muscle back into spasm. Consequently, post injection, all efforts should be made to prevent spasm. This can be accomplished by gradual return to normal activity and "strengthening exercises" should be avoided. If and when spasm occurs, the use of massage, heat or stretching should be used. Activity should be limited to the point of awareness of discomfort from the treated muscle. Clinical findings suggest that the muscle is capable of a full recovery and thereafter not subject to recurrent spasm with normal activity. Patients with overuse sport injuries were able to return to full competitive form.

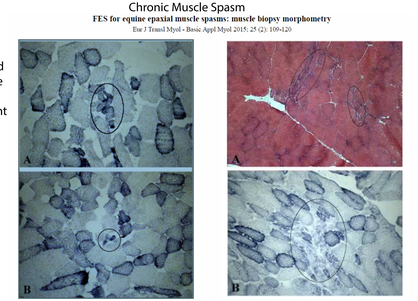
Variability of Mitochondial Density in Skeletal Muscle

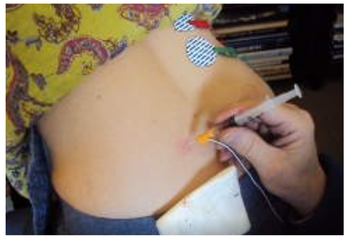
FES for equine epaxial muscle spasms: muscle biopsy morphometry
Eur J Transl Myol - Basic Appl Myol 2015; 25 (2): 109-120

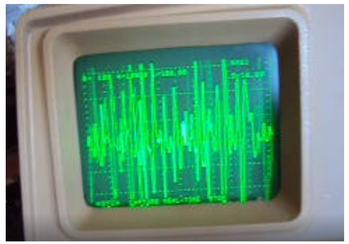


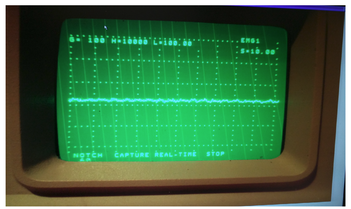
Arrows point to mitrchondria

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









PROPOSED NEW DIAGNOSTIC ENTITY OF ACQURED CHRONIC MUSCLE SPASM

INTRODUCTION: Prior work by this author has demonstrated the ability to identify chronic muscle spasm by the needle EMG finding of spontaneous electrical activity (SEA). Moreover, it was shown that the presence of SEA was not solely the result of denervation of muscle but was present in muscle that had no evidence of denervation. It was further shown that treatment of muscles in chronic spasm without a neurogenic etiology can be successful treated by elimination of the SEA to allow the muscle to return to a normal physiologic state.

OBJECTIVE/METHODS: To formulate a proper diagnosis and thereby allow for correct directed treatments for patients with clinical and needle EMG evidence of acquired chronic muscle spasm (ACMS).

RESULTS: ACMS shall be considered to be present when the following conditions are met: (1) Needle EMG tracing demonstrates chaotic SEA in muscles that are or should be at rest given proper body positioning. (2) SEA will not be abated by reciprocal inhibition by activation of competitive muscle groups. (3) No known neurogenic cause for the presence of SEA has been identified. (4) When physically accessible, muscle will demonstrate a resistance to compression and which will typically result in discomfort.

SUMMARY/CONCLUSION: The acceptance and utilization of the proposed new diagnostic entity of ACMS should allow for improved diagnosis of the etiology of chronic muscle spasm. This should allow alternative treatment of the resultant chronic pain.