A definition of Acquired Chronic Muscle Spasm as a new diagnostic entity is proposed. The key finding is the presence of Spontaneous Electrical Activity (SEA) in non-dener- vated muscle where its presence cannot be resolved by activity that would normally cause reciprocal inhibition. It is commonly acquired by overuse injury or trauma. What causes this muscle to stay in a state of chronic spasm for months and years with no known or identifiable neurologic etiology has been previously proposed to be the "Stress Relaxation Hypothesis" which is the result of ischemia from a partial or complete state of muscular contraction.

Atrial fibrillation is a good example where muscle stays in a state of chronic constriction with no neurogenic origin. There is membrane instability with ongoing electrical activity that lead to a continuous muscle contraction problem. For skeletal muscle is in the form of SEA, which is both the identifying key and the actual diagnostic tool for the condition.

The blood supply of the heart is a useful model for understanding the effect of spasm on blood supply. It has been long known that coronary blood supply is greater in dias- tole than in systole. The more plausible explanation is that the capillaries are no longer being so severely compressed to allow more blood flow to the heart during diastole than in systole. The most plausible explanation is that the capillaries are no longer being so severely compressed to allow more blood flow to the heart during diastole than in systole.

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A model for the occurrence of SEA can be seen in the heart. Studies have been done regarding the sodium, potassium and calcium channels. We also know that energy is required to maintain those channels. What happens when there is an "energy crisis" from prolonged blood shortages? What is seen are arrest of the heart in a non-systolic beat but otherwise times just like atrial fibrillation.

Some forms of arrhythmia is a front line treatment. However, there is a concern that these forms of arrhythmia and is difficult to suppress. Treatment of the chronic stomies to relieve the blood shortage typically results in resolution of the arrhythmia.

Before getting to treatment modalities for acquired skeletal chronic muscle spasm, it is useful to learn what happens to cardiac muscle that sustains chronic cardiac arrhythmias. We speak of fibrillation of muscle. This refers to muscle that is not dead but not doing much and histopathologically is not normal. It takes time for the muscle to change into normal muscle.

It has now been that chronically starved skeletal muscle loses mitochondria and it has a half life of 24 hours. Therefore, not all patients can be fully treated with a single injection. The injection readily tolerable. Also, Lidocaine was in fact an antiarrhythmic and had a functional duration of 1-2 hours. If the muscle is adequately "infused" with the medication, there should be no hot spots left to start up spasm of adjacent muscle. Different than not removing the muscle contrac- tion is not doing much and histopathologically is not normal. It takes time for the muscle to change into normal muscle.

Various treatment modalities have been tried to treat the starved muscle caused in a state of chronic atrophy. Taking up from the work of Travell, Simon and Hub- bard, this author found a clinically viable means of treating the muscle which, for all intents and purposes, was a long acting anti-arrhythmic drug. Initially Botulinum, was used but then for issues of cost and potential complications, phenoxycyanine was used. This drug was known to form a covalent bond on the muscle receptor site and has a functional duration of action of approximately 8 weeks. Also, as noted above, patients were warned that they should be warned of this and may need another treatment over the short haul if the second treatment is not successful. These newer alpha blockers appeared to be more sensitive to the muscle and less likely to lose other structural elements that can be regenerated with the normal blood supply of muscle.

As we are dealing with injured, starved and ultrastructurally damaged tissue, it is unrealistic to expect normal function immediately. Its energy capacity is limited and any form of muscle back to a normal physiological state appears to take minimally sev- eral weeks. The severity and duration of the chronic spasm appears to affect the time frame for recovery.

In short we have an overlying injury leading to a prolonged "wound-

"typical" wounded warrior" spasm which can be just enough to prevent the expected slow road to health. Secondary spasm, should they occur should be treated as the primary one. Therefore, a constant level of vigilance needs to be exercised to be aware of this and may need another treatment over the short haul if the second treatment is not successful. These newer alpha blockers appeared to be more sensitive to the muscle and less likely to lose other structural elements that can be regenerated with the normal blood supply of muscle.