# Proposed New Diagnostic Entity of Acquired Chronic Muscle Spasm

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## Pathophysiology of Acquired Chronic Muscle Spasm Revisited

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-denervated 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 the muscle to stay in a state of chronic contraction for months and years with no known or identifiable neurologic etiology has been previously proposed to be the SEA resulting from the membrane instability, which is the result of ischemia from an initial state of prolonged muscle contraction.

Atrial fibrillation is a good example where muscle stays in a state of chronic contraction with no neurogenic origin. There is membrane instability with ongoing electrical activity and continuous muscle contraction. Arrhythmia is the core of the problem. For skeletal muscle it is in the form of SEA, which is both the identifying key and the actual cause of the chronicity of the spasm.

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 diastole than in systole despite the higher pressures in systole. The answer lies in the compression of the capillaries during systole. Unpublished work by this author demonstrated that if the force of contraction is lowered, at a certain point, there is greater coronary blood flow during systole than in diastole. The most plausible explanation is that the capillaries are no longer being so severely compressed to allow more blood to flow with the higher blood pressure in systole.

It would not be unlikely that skeletal muscle in a state of severe contraction would also limit blood flow. The muscle does survive, as enough nutrients seem to make it to the muscle. Research noted below on this poster, found that muscle in chronic spasm has limited numbers of mitochondria and treatment of the spasm leads to recovery of the mitochondrial numbers for the oxidative muscle fibers. Research needs to be done to better understand what happens to the glycolitic fibers with prolonged ischemia and recovery.

Contraction and relaxation of muscle fibers is somewhat paradoxical to what would initially seem to make sense. Energy is required for muscle to relax. Contraction is preordained and occurs on command using stored energy. Just like a mouse trap, energy is required to set the trap and minimal force is required to set it off. So when the muscle runs out of energy it remains in a contracted state. Normally enough nutrients reach the muscle to allow it to eventually relax. However, the occurrence of SEA resulting in self exiting spasm may continue to exhaust the energy supplies and prevent the muscle from ever having the needed energy to relax.

A model for the occurrence of SEA can been seen in the heart. Studies have been done regarding the sodium, potassium and calcium membrane 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 arrhythmias which are sometimes fatal but other times chronic just like atrial fibrillation. Some form of anti-arrhythmic is a front line treatment. However, if there is a coronary artery stenosis that causes coronary ischemia, the arrhythmias are more difficult to suppress. Treatment of the coronary stenosis 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 explore what happens to cardiac muscle that sustains chronic ischemia. We speak of hibernating 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 recover not only its function but also the structural elements before it can normalize. It has now been that chronically starved skeletal muscle looses mitochondria and likely looses other structural elements that can be regenerated with the return of normal blood supply.

Various treatment modalities have been tried to treat the starved muscle caught in a state of chronic arrhythmia. Taking up from the work of Travell, Simon and Hubbard, this author found a clinically viable means of treating the muscle which, for all purposes, was a long acting anti-arrhythmic drug. Initially Botox, was used but then for issues of cost and potential complications, phenoxybenzamine was used. This drug was known to form a covalent bond on the muscle receptor site and had a functional duration of action of 2-3 months as the muscle had to grow new receptors.

This drug, however, was not in any way a perfect candidate. It was FDA approved and therefore could be used "off label" following the FDA guidelines. However, it was fairly insoluble and required a pH of 3 which was not well tolerated by tissue. Fortuitously, Lidocaine also had a pH of 3 and could be mixed with phenoxybenzamine to make the injection readily tolerable. Also, Lidocaine was in fact an antiarrhythmic and had an almost immediate effect on the SEA that was being targeted. This allowed the clinician to know if all the tissue to be treated had in fact received an adequate dose of the mixture. Ultimately, a medium acting steroid, dexamethasone, was added to improve patient comfort over the first few days following injection as the low pH still had an irritant effect on the tissue.

In most cases a single injection would resolve the spasm if all of the affected tissue could be fully injected at one time. It need to be remembered that phenoxybenzamine is an alpha blocker which was approved for control of high blood pressure and it has a half life of 24 hours. Therefore, not all patients can be fully treated with a single injection. The injection nearly immediately stops the SEA. The Lidocaine wears off in about 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 typical steroid or Botox injections, every bit of SEA has to be eliminated requiring a different injection technique using very small boluses of injectate, typically 1/10 cc at a time. Repeat needle penetration of the muscle, generally through a single skin puncture, is strongly recommended over the entire area being treated to look for pockets of residual SEA.

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 "flight to health" use of the muscle following the injection will likely put it back in a typical "weekend warrior" muscle spasm. This may be just enough to prevent the expected slow road to health. Secondary spasms, should they occur should be treated with classic treatments of massage, heat and stretching. Patients do need to be warned of this and may need another treatment over the short haul if the secondary spasm lasts for several weeks.. Also, not uncommonly seen are other segments of muscle that had not been called upon because the chronic pain of the treated muscle is now absent and new activities end up overpowering the reserve of other muscles. It is critically important to take note of the exact location of the treated spasm. Once a site of pain is resolved, the principle of "Hierarchy of Pain" takes effect which states that the brain then takes note of the next most significant site of pain, previously ignored due to a more severe site of pain. A clear geographic map of pain for any treatment is therefore required and follow up surveys must also be specific regarding sites of treatment and resolution of pain.

Once treated, repeat EMG evaluation is not generally needed but exploration of residual pain should typically wait two weeks. By that time any local irritation from the phenoxybenzamine should be fully resolved. Pockets of SEA not adequately treated can then be identified and treated with a second injection. Recovery of muscle back to a normal physiological state appears to take minimally several weeks. The severity and duration of the chronic spasm appears to affect the time frame for recovery.

In short then we have an overuse injury leading to a prolonged "weekend warrior" spasm that by virtue of the marked limitation of blood supply lasts long enough to exhaust the muscle cells energy storage and disrupt the membrane channels causing membrane instability seen as SEA. This then perpetuates the spasm by further exhausting the energy supply and maintains the starvation level of blood supply. This is the black hole of skeletal muscle physiology.

Starting from an understanding of the above, there should be great potential for further clinical and basic research. The published treatment with its results will hopefully be the jumping off point for a better solution for the very high proportion of chronic pain that is caused by chronic muscle spasm.