SUCCESSFUL TREATMENT OF LONGSTANDING CHRONIC MUSCLE SPASM WITH EMG GUIDED CHEMODENERVATION

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Pathophysiology of Acquired Chronic Muscle Spasm Revisited

It's now a new day! There is now a definition of Acquired Chronic Muscle Spasm. Spontaneous Electrical Activity (SEA) in non-denervated muscle where its presence cannot be resolved by activity that would normally cause reciprocal inhibition. It is just that simple and is commonly acquired by overuse injury or trauma. So, what is happening that leaves muscle in a state of chronic contraction for months and years with no known or identifiable neurologic etiology?

Let's looks elsewhere where muscle stays in a state of chronic contraction. Atrial fibrillation is a good example. Ventricular fibrillation would be good too except that it is a terminal event. In both cases there is membrane instability with ongoing electrical activity and continuous muscle contraction. Sound familiar? 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. But where did that come from and why did it happen?

Let's look at the blood supply of the heart, mostly made of muscle, during systolic contraction. 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 Unpublished work by this author demonstrated that if the force of contraction is lowered, at a certain point, there is greater coronary blood flow during systole than in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.

So, it would not be unlikely that skeletal muscle in a state of severe contraction would also limit blood flow. But the muscle does not seem to die as enough nutrients seem to make it to the muscle for at least survival mode.

Time here to remember how muscle works. Energy is required for muscle to relax. Contraction is preordained and occurs on command. 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. But what if something else happens that prevents that from happening? Like for example, an electrical storm that keeps the muscle in a state of constant contraction just like the frogs leg experiment we all did in biology lab with the electrical charge to contract the frogs leg till it ran out of energy.

But there does seem to be an electrical storm in chronic muscle spasm. It is that SEA we were talking about. Clearly not good for the muscle to ever relax but where did it come from? In cardiology, herein the author happens to be a cardiologist, we have learned about membrane channels, sodium, potassium and calcium. We also know that energy is required to maintain those channels. What happens when there is an "energy crisis" from prolonged blood shortages. What happens to the heart when this occurs? Of course, what we see are arrhythmias which are sometimes fatal but other times chronic just like atrial fibrillation. How are they treated? Well some form of anti-arrhythmic is a front line treatment. However, if there is a coronary artery stenosis, it is not uncommon to open the artery and relieve the blood shortage. Low and behold, the arrhythmia not uncommonly goes away.

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. Little, if any research, has been done on chronically starved skeletal muscle. It would most likely be the case that chronically starved skeletal muscle it has lost structural elements that can be regenerated with the return of normal blood supply.

Getting back to our starved muscle caught in a state of chronic arrhythmia, how do we begin to normalize it? Multiple clinical attempts using every modality imaginable have tried and with varying success to relieve chronic muscle spasm. 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.

What then was happening? In most cases a single injection would do the trick if all of the affected tissue could be fully injected at one time. Not to forget that phenoxybenzamine is an alpha blocker which was approved for control of high blood pressure and it has a half life of 24 hours. So not all patients can be fully treated with a single injection. How about the muscle? 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 is strongly recommended over the entire area being treated to look for pockets of residual SEA

But what did happen and what is expected to happen? Keep in mind that we are dealing with injured, starved and ultrastructurally damaged tissue. 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. Patients do need to be warned of this and may need another treatment over the short haul should this happen. 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.

Once treated, repeat EMG evaluation at 1 day, 1 week, 1 month and for years do not exhibit SEA. Patients successful in causing a second overuse injury can cause SEA to appear, but not necessarily in the same muscle. The point being that treatment leads to what can be best understood as allowing complete recovery of the muscle tissue. Tissue research should likely show a full resolution of the "taut bands" that are the histologic presentation of trigger points representing at least part of the pathology of chronic muscle spasm.

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.