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# NON-SKELETAL ETIOLOGY OF DROP FOOT WITH THERAPEUTIC REVRSAL

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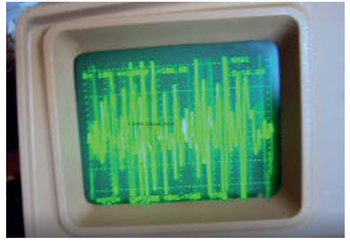
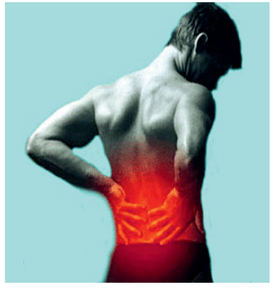


SPONTANEOUS ELECTRICAL ACTIVITY



CHRONIC MUSCLE SPASM

Drop foot has been known to occur with a variety of causes including spinal disease, stroke, neuropathies, drug toxicities and diabetes. The etiology of drop foot has been known to have two non-skeletal instances with direct physical involvement of a nerve or nerve root. Peroneal nerve injury is known to potentially cause drop foot. Piriformis syndrome has also been reported in a few cases to be responsible for foot drop. However, no clear evidence has been previously reported involving lumbar muscle groups being responsible for foot drop. Absent EMG evidence of piriformis compression of the sciatica nerve or peroneal compression, it is generally assumed that degenerative disc disease is the culprit.

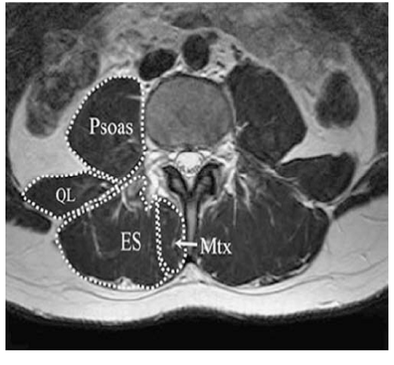
Presented here are three cases of drop foot that resolved with treatment of lumbar chronic muscle spasm as the primary and sole treatment. The variations in outcome are instructive in understanding the role chronic muscle spasm plays in this condition.

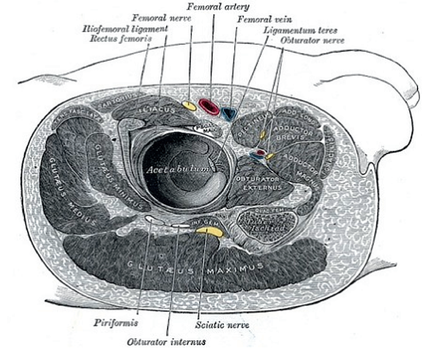
It is known that muscle in contraction increases its cross sectional size. The anatomy of the lower back as seen in the accompanying images make clear that the nerve roots are in direct contact with large muscle groups and potentially compressed by them with muscle contraction. Muscles in a state of chronic contraction can potentially cause chronic compression of nerve roots. The precise point of potential nerve compression is difficult to assess. However, the immediate relief of drop foot in one of the three cases, with injection into a lumbar muscle group, evidencing EMG evidence of chronic spasm, indicates that points of compression do exist and can be responsible for interruption of nerve function. In fact that individual had no known degenerative disc disease.

Delayed recovery noted in the other two cases appear to indicate the variation in nerve recovery dependent upon the duration or severity of nerve compression that had occurred. The one case where the foot drop had been present for several years required 6 months for full recovery. In that case the knee jerk had been absent for 4 years and had recovered by 6 months. This speaks to nerve injury with only chronic muscle spasm to blame as no other treatment was utilized.

Given the proximity of the muscle groups to the origin of the lumbar nerve roots, EMG evaluation of the exact site of nerve conduction abnormality is at best difficult if at all possible. Chronic muscle spasm identifiable with the presence of spontaneous electrical activity (SEA) at rest is readily appreciated with simple EMG interrogation of the muscle.

Based upon the cases shown it would appear that in cases of drop foot, EMG evaluation of lumbar muscles to identify chronic spasm would be warranted. When chronic muscle spasm is found in the setting of foot drop, the treatment modality of CMECD® is a relatively easy intervention. A case can be made that CMECD® treatment of chronic muscle spasm in that setting should be the preferred initial treatment. It has far less risk than epidural injections that have become the usual initial treatment modality.





Non-skeletal etiology of drop foot

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Based upon the cases shown, EMG evaluation of lumbar muscles to identify chronic spasm, in cases of drop foot, would appear warranted. The treatment modality of CMECD® is a relatively easy intervention with far less risk than epidural injections and may provide sustained relief and recovery of drop foot.