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Title : REGIONAL NEUROMUSCULR BLOCKADE

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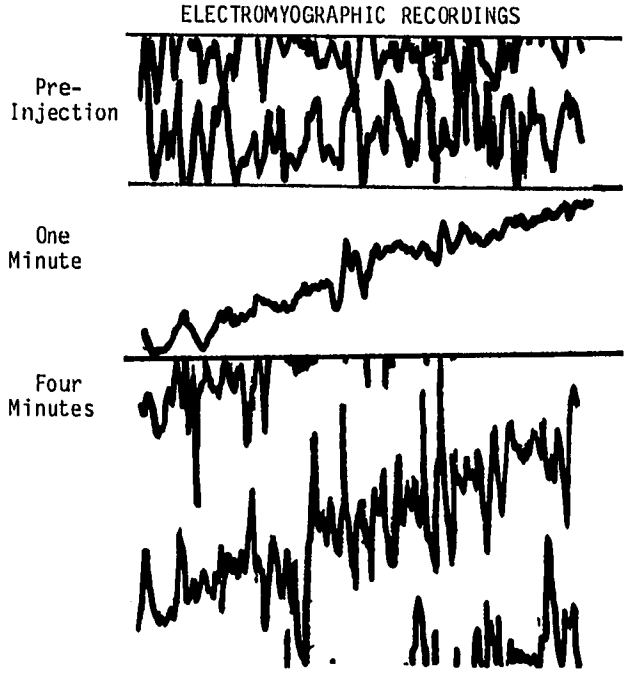
Introduction: Spasticity is a common symptom in patients with multiple sclerosis, hemiplegia, paraplegia, arachnoiditis and cerebral palsy. When pain is associated with these conditions it is often difficult to determine whether the pain arises from peripheral spasticity or from the central nervous system lesion itself. Determining the exact origin of this pain may present a major diagnostic challenge. This study evaluated the intra-arterial injection of a non-depolarizing neuromuscular blocker as a simple method of minimizing spasticity without altering cognitive function.

Methods: Six patients with CNS lesions, spasticity and pain were studied after informed consent and institutional approval.

Pt.	CNS Lesion	M. Spasm	Pain
1	Post-CVA Hemiplegia	RUE, RLE	RLE
2	Arachnoiditis	RLE	RLE
3	Post-CVA Hemiplegia	RLE	RLE
4	Post-CVA Hemiplegia	LUE	LUE
5	Spinal-Cerebellar Degen.	RLE, LLE	RLE
6	Cerebral Palsy	UE's LE's	LLE

A 19G catheter was placed percutaneously into the artery of the affected limb under local anesthesia and directed proximal to the arterial branches for the involved musculature. One mg of pancuronium in 20 ml saline was then injected over 20 seconds. The EMG was monitored with a Teca TE-4 using a Teca Tm monopolar recording needle. The recordings were made with a voltage setting of 200 mv and a sweep setting of 20 m/sec/division. Voluntary muscle contractions were elicited by verbal request. Peak to peak action potentials were measured on three samples; pre-injection, 1 minute and 4 minutes post injection. A range of peak to peak action potentials were summed and mean calculated on each sample. Each patient evaluated his pain by subjective impression and visual analog scale.

Results: The mean amplitude of the pre-injection EMG was 32 mm with an incomplete interference pattern. Maximal muscle relaxation was recorded at one minute post-injection with a mean amplitude of 15 mm and a random single unit interference pattern. Patients 1, 3, 4, and 5 did not describe any relief of their symptoms at the one minute interval, whereas patients 2 and 6 described total relief. At 4 minutes post-injection the EMG interference pattern had returned to the pre-injection level with a mean amplitude of 31.25 mm. Pain and spasticity had returned in patients 2 and 6. An EMG recording at the three time intervals is illustrated below.



Discussion: Spasticity may occur in CNS disorders where descending inhibition of the bulbo-spinal pathways is diminished.[1] Pain may arise either from the CNS disorder itself or from the resulting muscle spasticity. Removal of the spasticity by the intra-arterial injection of a non-depolarizing neuromuscular blocking agent provides ideal conditions to determine the origin of the pain. It does not impair cognitive function, as may occur with medical treatment using tranquilizers.[2] The intra-arterial route delivers the drug directly to the motor end-plate, minimizing the dose required and obviating the need for a tourniquet. The treatment of spastic pain is specific. The indiscriminate reduction in muscle tone may not be in the patient's best interest, particularly if spastic stiffness is necessary for weight bearing and mobility. Our technique allows precise determination of the origin of the pain, before more aggressive medical or surgical modalities are attempted.

References: 1. Burke DJ: An approach to the treatment of spasticity. Drugs 10:112-120, 1975
2. Rushworth G: Some aspects of the pathophysiology of spasticity and rigidity. Clin Phar Ther 5, 1965