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*J Neurol Neurosurg Psychiatry* 1985 48: 582-584

doi: 10.1136/jnnp.48.6.582

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## Short report

# Brachial plexus myoclonus

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**SUMMARY** Rhythmic myoclonus in an arm began abruptly following an injury and persisted continuously for six years. Topographical EMG showed abnormal activity confined to muscles innervated by the axillary and radial nerves from the posterior cord of the brachial plexus. Abduction of the arm above horizontal level stopped myoclonus and EMG discharges. EEG was normal. It is suggested that the myoclonus was caused by mechanical irritation of the posterior cord of the brachial plexus.

Myoclonus is usually of central origin. Impulses may be generated in the cerebral cortex or brainstem, or in the spinal cord with synchronous contractions in muscles innervated by one spinal segment (segmental myoclonus).<sup>1-5</sup> Recently, myoclonic-like movements have also been attributed to lesions in *peripheral nerves*.<sup>6-8</sup> We present another patient, in whom rhythmic myoclonus clinically and electromyographically seemed to arise from a lesion in the posterior cord of the brachial plexus.

### Case report

A 62-year-old woman presented with continuous rhythmic abducting movements of her right arm, unchanged for about 6 years. Twelve years ago, she had developed a "frozen" shoulder following a fracture of the right humerus with persistent stiffness, but without abnormal movements. Six years later she underwent mastectomy for right breast carcinoma followed by irradiation (5000 rads) to the breast and mediastinum; the radiation field excluded the brachial plexus and spinal cord. Regional lymph nodes showed no metastases. Subsequently, she underwent physical therapy for shoulder stiffness and to prevent scar contracture.

During one session, her right arm was forcibly abducted, resulting in sudden pain in the shoulder and arm and onset of jerking movements of the arm. These have been present since, rhythmic in character and also observed in sleep. The patient was unaware of triggering mechanisms, but

could minimise jerking by holding the arm in an abducted position. Treatment with haloperidol and diazepam was ineffective, but infiltration of the brachial plexus with local anaesthetic abolished movements for about 10 minutes, indicating that the generator was located at or above that site.

When first seen by us the mastectomy scar was indurated. There were no other skin changes and no palpable regional lymph nodes. Neurological examination was normal except for myoclonus of the right arm at a rate of about 5 Hz, conspicuous at rest and during volitional arm movements. Jerking involved the right finger extensors, triceps, and deltoid muscles, and was capable of causing a 45° abduction of the arm. It could be stopped by active or passive abduction of the arm above the horizontal level, but resumed when the arm was lowered again. It was unaffected by other arm positions, sensory stimuli, or voluntary manoeuvres. In hospital, jerking was observed in sleep.

Routine laboratory tests, haematological studies, Ca<sup>++</sup>, Mg<sup>++</sup>, Cu, and ceruloplasmin concentrations were normal. Bone scans were negative at the time of onset of movements, but recent examinations now reveal multiple bony metastases involving the right anterior ribs, right iliac crest, and the right humeral head. Soft tissue lesions in the vicinity of the right brachial plexus have never been observed. Radiographs of the cervical spine showed no lytic lesions and only mild retrolithosis of C3 and C4 with normal neural foramina. Myelography with spinal CT was not performed because of lack of progression in symptoms and other evidence of spinal involvement over six years. CT of the head with contrast enhancement and repeated EEG's, recorded awake, were normal. Specifically, a 16-channel EEG including a record of right arm activity showed no accompanying synchronous scalp activity.

*Electromyography*, using concentric needle electrodes, was performed twice. Synchronously with jerks, rhythmic activity was recorded at a rate of 4.8 to 5.4 Hz in muscles

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Received 14 August 1984 and in revised form 20 October 1984.  
Accepted 3 November 1984

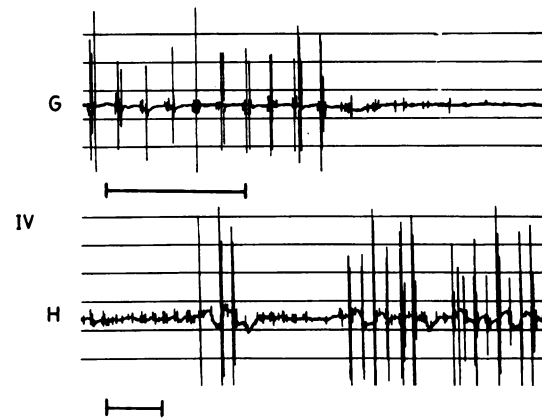
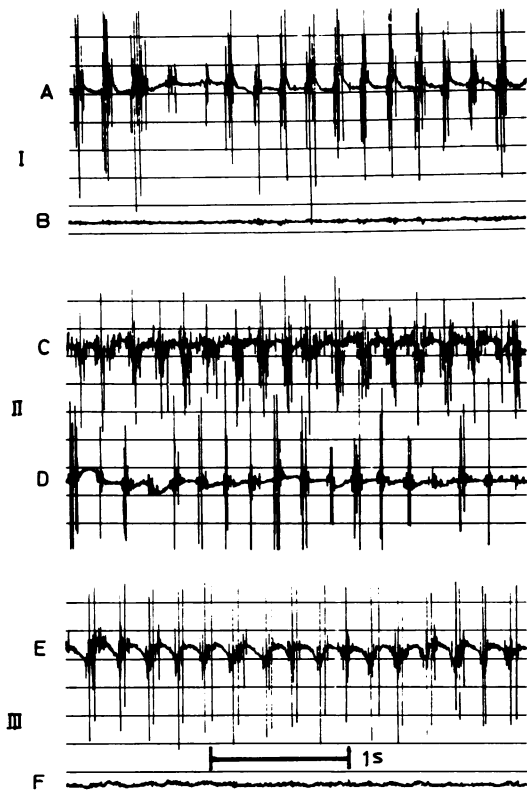


Fig EMG recordings of rhythmic activity. 1. Simultaneous recording from deltoid (A) and infraspinatus (B) muscles (500  $\mu\text{V}/\text{div}$ ). 2. Simultaneous recording from extensor digitorum communis (C) and deltoid (D) muscles (500  $\mu\text{V}/\text{div}$ ). 3. Simultaneous recording from extensor digitorum communis (E) (500  $\mu\text{V}/\text{Div}$ ) and flexor digitorum superficialis (F) muscles (100  $\mu\text{V}/\text{div}$ ). 4. Passive elevation of arm stops rhythmic activity (G), and lowering of arm starts activity in deltoid muscle (H) (500  $\mu\text{V}/\text{div}$ ). Active elevation of the arm was avoided in order to prevent volitional muscle activity from masking the rhythmic activity. Amplification (in parenthesis) = distance between horizontal lines. Recordings were made with a concentric needle electrode.

innervated by the right axillary and radial nerves, which arise from the posterior cord of the brachial plexus and include fibres from roots C5 through C8. Notably, however, rhythmic activity was absent in muscles innervated by the suprascapular nerve originating just proximal to the cord (fig. A–B), and in muscles innervated by fibres in the lateral and medial cords, representing the same spinal segments as the axillary and radial nerves (table). Simultaneous recording from a proximal and distal muscle innervated by the posterior cord showed rhythmic activity occurring in time-locked synchrony (fig. C–D), whereas recording from antagonistic extensor and flexor muscles of the forearm only showed rhythmic activity in the extensor muscle (fig. E–F). Continuous EMG recording from the deltoid muscle during passive abduction of the arm showed cessation of rhythmic activity, which reappeared when the arm was lowered again (fig. G–H), concomitant with disappearance and reappearance of visible jerking. Other spontaneous activity at rest (fibrillations, positive sharp-waves, fasciculations) was absent, and motor unit potentials and contraction pattern at maximal effort were normal. Motor nerve conduction velocities and F-wave latencies were normal on both sides.

## Discussion

There is direct and indirect evidence to suggest that

myoclonus in our patient was due to a *peripheral* nerve lesion. Evidently, the onset of myoclonus was associated with a traumatic event, a forceful and painful abduction of a “stiff” shoulder. The topography of EMG findings (table) suggests that the generator is located in the short segment of the posterior cord of the brachial plexus, above the departure of the axillary nerve. The proximal limit for its location is defined by the absence of abnormal activity in muscles innervated by the suprascapular nerve.

Unlike irregular, epileptiform myoclonus of central origin, jerking in our patient was unenhanced by sensory stimuli, present in sleep, and unaccompanied by EEG abnormalities or other evidence of cortical or brainstem involvement. A spinal cord generator seems unlikely, because of the absence of abnormal activity in muscles with the same segmental innervation as the involved muscles, or radiological evidence of a lesion in the cervical spine or cord, and of clinical symptoms or signs of myelopathy. The long duration of myoclonus without progression is incompatible with intraspinal metastasis. In fact, there were no radiological signs of metastases when the myoclonus started.

Table EMG findings

Muscle	Nerve	Cord	Segment
<b>Myoclonus present</b> (Frequency = 4.8–5.4 Hz)			
Deltoid	Axillary	Posterior	C5–6
Triceps	Radial	Posterior	C7–8, T1
Brachioradialis	Radial	Posterior	C5–6
Ext. Dig. Comm.	Radial	Posterior	C7–8
Ext. Carpi Rad.	Radial	Posterior	C6–7
<b>Monoclonus absent</b>			
Supraspinatus	Suprascapular	—	C5–6
Infraspinatus	Suprascapular	—	C5–6
Biceps Brachii	Musculocutaneous	Lateral	C5–6
Flex. Dig. Superf.	Median	Lateral	C7–8
Abd. Poll. Brev.	Median	Lateral	C8–T1
Hypothenar	Ulnar	Medial	C8–T1

The history suggests that the posterior cord was subject to external pressure, possibly due to fibrous scar tissue. This may cause ectopic excitation, which is probably the mechanism in hemifacial spasm.<sup>9,10</sup> Accordingly, abduction of the arm may have released a mechanical pressure on the cord with immediate cessation of jerking. The postulated site of a lesion would be verified, if myoclonus persisted after blocking of the spinal roots proximal to the plexus with local anaesthetic, or if surgical release of the posterior cord resulted in cessation of jerking. Unfortunately, neither option was possible in the present case.

Involuntary movements following peripheral nerve injury have been reported sporadically.<sup>5–8,11–13</sup> Analogous to our case, in one patient with a schwann-cell sarcoma of the femoral nerve,<sup>6</sup> quadriceps myoclonus could be abolished by knee flexion. Patients with involuntary movements may show electrophysiological evidence of peripheral nerve injury.<sup>8</sup> Hence, a peripheral nerve lesion should be considered in the differential diagnosis of myoclonus. Topographic EMG examination may establish the location, which has therapeutic implications as peripheral nerve lesions are surgically accessible.

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