Letters

Painful arm and moving fingers

Sir: A syndrome of painful legs and moving toes has been known for many years.1 We report a patient with a painful arm and moving fingers due to a lesion of the brachial plexus.

In April 1982, at the age of 54 years, our patient discovered a breast tumour on the left side. At surgical exploration an adenocarcinoma was found and one of the axillary lymph nodes showed metastasis. Six months after irradiation was started the patient noted numbness and paraesthesia in the left upper arm, gradually extending to the distal part of the arm. In November 1982 she felt a continuous deep dull pain in her left arm. Sensation was clearly diminished. In the spring of 1983 she noticed involuntary movements of the fingers of her left hand. Muscular strength was diminished especially in the hand. On admission to our department, induration was found in the supraclavicular and axillary region, with cutaneous changes due to irradiation. There were no enlarged lymph nodes. Spine function was good. Examination revealed a brachial plexus lesion with atrophy of the interosseous muscles. The left arm was generally weak. Sensory loss was indicative of a lesion of the medial cord. There were no indications of tumour metastasis; blood examination, CSF, cervical myelography and cervical CT scan were normal.

The fingers of the left hand showed continuous involuntary movements at the metacarpophalangeal joints with extension at the interphalangeal joints. These continuous movements consisted of flexion and extension combined with adduction and abduction. Each finger moved independently of the other fingers in a more or less identical rhythm, similar to athetosis or pseudo-athetosis due to sensory polyneuropathy. The movements were unlike those that originate in motoneurones (fasciculations, myokymia). Sometimes these movements were absent for a few moments. The patient was unable to imitate the movements nor could she stop or initiate them.

The electromyogram showed a markedly reduced motor conduction velocity of the brachial plexus (CV of lateral fascicle from Erb's point to axilla: 6 m/s) as well as slowed motor conduction of median nerve (33 m/s) and ulnar nerve (47 m/s), both nerves showing normal distal latencies. Distal sensory action potentials were absent in median and ulnar nerves. EMG of biceps, adductor pollicis brevis and first interosseous muscle showed signs of active and chronic denervation. At rest there were spontaneous discharges of motor unit action potentials in interosseous muscles, occurring in pseudorhythmic bursts, with a mean frequency of one burst per second. These bursts had a voltage of 200–800 μV and they lasted up to 600 ms.

This patient is suffering from a painful arm and moving fingers, caused by brachial plexus lesion due to radiotherapy. This sensorimotor disorder resembles the syndrome of painful legs and moving toes.1 Most authors suggest a peripheral generation of this syndrome:6–4 a persistent abnormality of the central nervous system may subsequently develop.3 Lance6 supposes that the origin of the disorder is in the thalamus and basal ganglia.

Our patient, patients with pseudo-athetosis due to sensory polyneuropathy and patients with painful legs and moving toes have two things in common: (1) the more or less continuous involuntary movements are associated with a dysfunction of the peripheral nervous system and (2) the absence of evidence of a central nervous system disorder, which is normally the cause of similar athetoid movements. Wilson6 and Denny-Brown7 assumed that spontaneous involuntary movements like athetosis are nothing else than a succession of cortical reflexes (Wilson) or a conflict between tonic types of automatic patterned reflex responses to contact stimulation (Denny-Brown). However, our patient, patients with painful legs and moving toes, and patients with pseudo-athetosis due to sensory polyneuropathy all demonstrate that similar movements can also be evoked by abnormal impulses from the peripheral nervous system. These abnormal impulses disturb the normal functional relationship between afferent information and motor control.10 11

The influence of afferent information on athetotic movements is well known.9 12 Furthermore, cutaneousmuscular reflexes are increased in athetosis.16 17 It is suggested6 that these reflexes are related to other sensorimotor reflexes3–15 which have a supraspinal pathway along the lines proposed by Phillips6 and Tatton.11 We suppose that in our patient (and in patients with comparable syndromes) the abnormal movements, which look so much like athetosis, are caused by a peripheral sensory disturbance of the same supraspinal sensorimotor interactions as in athetosis. Because these movements are related to a sensation of pain or paraesthesias, group II and group III afferent fibres are probably the major contributors. Strong evidence in favour of this supraspinal hypothesis is given by Marsden:7 thermocoagulation of nucleus ventralis intermedius abolished the movements and reduced the pain in a patient with a comparable sensorimotor disorder (case 1).

Segmental cutaneousmuscular reflexes15 19 20 or segmental bombardment of motoneurones may of course be held responsible for this movement disorder, but considering the more elaborate type of finger movements, in which each finger seems to move independently of the others, we believe that these movements transcend the simpler flexion-extension reflexes of spinal origin. However, Fleshman11 demonstrated subtle peripheral influences on movement. These partly cutaneous influences have a segmental pathway and exert different effects on muscle that have essentially identical mechanical actions. Therefore, a spinal pathway of the movement discussed cannot be ruled out on the
ground of the elaborate and atheothoid phenomenology alone.

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References


The arm-mouth reflex in a patient with amyotrophic lateral sclerosis

Sir: Numerous pathological or primitive reflexes may appear in a variety of diseases of the central nervous system including amyotrophic lateral sclerosis. We have observed reflex opening of the mouth upon passive flexion of the forearm in a patient with an advanced state of amyotrophic lateral sclerosis. To our knowledge, this reflex, which we have called arm-mouth reflex, has never been described previously.

A 57-year-old Japanese man noticed wasting and weakness of both hands in April 1981. During the next nine months progressive weakness of lower limbs and difficulty in swallowing and speaking developed. The patient's history was otherwise normal. He was admitted to hospital on 11 January 1982. Findings on general physical examination were normal as was his mental status. Bilateral weakness was present mildly in facial muscles and moderately in bulbar muscles with dysphagia and dysphonia. Atrophy and fasciculations in the tongue were observed. Weakness was also present mildly in neck flexors, moderately in all limb girdle and proximal limb muscles and severely in bilateral hand muscles. Deep tendon reflexes were symmetrically hyperactive in association with Babinski's reflex. Jaw jerk was exaggerated and superficial abdominal reflexes were absent. Sensation and coordination were normal. Cerebrospinal fluid examination and radiographs of the skull and spine were normal. Electromyographic examination of the right biceps brachii, first dorsal interossei, and rectus femoris showed fibrillation potentials, positive sharp waves, an increase in potential amplitudes and polyphasic potentials with a decrease in the maximum interference pattern. Pulmonary function tests showed that his vital capacity was 49%. A diagnosis of amyotrophic lateral sclerosis was made. The patient became almost aphagic and apnoeic by June 1982 and required the use of a feeding tube and a respirator. Muscle weakness also progressed rapidly. Neurological examination in April 1983 showed that the patient was alert and orientated but was totally aphagic, aphonlic, dysnomic and quadriplegic in extension. Supranuclear ophthalmoplegia and facial diplegia were noted. Forced crying was frequently seen. Deep tendon reflexes were symmetrically depressed but the sustained jaw clonus and head-retraction reflex were elicited. Tactile stimulation of the tongue resulted in reflex masticatory movement. At this clinical state, the arm-mouth reflex was observed and consisted of a wide opening of the mouth upon passive flexion of the forearm and could only be elicited by flexion of either forearm. The response movement was restricted to the perioral musculature and was constantly elicited by repetitive stimulation. The appearance, latency and duration of the response were absolutely dependent upon the strength of the stimulus (fig): faster and greater flexion of the forearm resulted in more prolonged and wider opening of the mouth at a shorter latency, whereas slower flexion of the forearm elicited the response less often. Flexions and extensions of other joints and sensory stimuli including pain and tendon tap of the triceps brachii did not elicit opening of the mouth.

The reflex opening of the mouth by the passive flexion of the forearm described here can be classified as a primitive brainstem reflex for the following reasons. Firstly, passive flexion of the forearm was the only stimulus that resulted in a stereotyped response of the perioral musculature at distinct latent periods following the stimulus. Secondly, the appearance, latency and duration of the response were dependent upon the strength of the stimulus. These physiological properties are in accordance with those of primitive brainstem reflexes. It has been demons-
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