

phages in the brain stem in this case, at and beyond the margin of the area involved with CPM. No multinucleated cells were seen.

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Could midbrain "resting" tremor be caused by postural maintenance at rest?

James Parkinson described resting tremor suggesting that tremor in Parkinson's disease (PD) persists even when the patient no longer has to maintain limb posture. So-called midbrain or rubral tremor characteristically includes resting, postural, and intention tremor. Gordon Holmes¹ noticed that midbrain resting tremor ceased when the limb was completely at rest. Holmes' observation suggests that midbrain resting tremor, contrary to resting tremor in PD, is caused by postural maintenance. We describe a patient with presumed midbrain tremor showing evidence that PD resting tremor and midbrain resting tremor may have a different neurophysiological background.

At the age of 63, a 67 year old man was suddenly struck by a left-sided third nerve palsy and a right-sided hemiparesis which disappeared after a few weeks. After this period resting, postural, and intentional tremor appeared in the right arm. He developed coarse, irregular myoclonic head shaking to the right and frequent generalised shuddering tremor lasting a few seconds. The patient was unsuccessfully treated with Sinemet. With orphenadrine 50 mg four times daily the tremor diminished, as did the shuddering attacks. Four years later the patient noticed that the entire limb tremor would disappear if he pushed firmly on the upper edge of the homolateral trapezius muscle.

On physical examination the right arm showed a complex resting tremor (Webster grade 2-3) with flapping flexion-extension at the wrist and elbow, and pronation-supination of the forearm. In our patient the first finger was beating against the thumb, though the classic "pill-rolling" movement of the thumb against the first finger, was absent (According to Denny-Brown² these movements of our patient's first finger and thumb differentiate midbrain tremor from PD tremor). The tremor amplitude increased on stretching the arms out and performing the finger-nose test. With distraction, when the patient was at rest or lying on a bed the tremor sometimes disappeared. The right

arm was hypokinetic (grade 1) and rigid (grade 2).

EMG showed regular 5 Hz bursts in the trapezius, supraspinatus and splenius capitis muscles, with the trapezius muscle constantly discharging 10 to 20 ms before the supraspinatus muscle. There were alternating 5 Hz bursts in the biceps and triceps muscles. CT head scan did not reveal any focal abnormalities.

The resting and action tremors were completely abolished by local intramuscular injection of 10-20 cc bupivacaine-adrenaline solution into the supraspinatus and the adjacent part of the trapezius muscle. The effect on the tremor lasted for days to weeks, although it diminished after the first few days. The patient was treated 17 times with intervals of two weeks to three months. Unfortunately the 17th injection caused a troublesome pneumothorax, so that the patient refused further injections.

Although we do not have direct anatomical proof of a mesencephalic lesion in our patient, the clinical picture consisting of acute ipsilateral third nerve palsy and contralateral hemiparesis warrants a diagnosis of Benedikt's syndrome as a result of mesencephalic stroke. In these patients a so-called midbrain tremor, that is, combined resting, postural and intentional tremor, may develop.

Direct evidence that postural maintenance rather than rest was involved in our patient was provided by local intramuscular anaesthetic infiltration after which both action and resting tremor disappeared. Although we cannot explain why the beneficial effect was so long lasting, the effect itself is well known. According to Rondot,³ postural tremors may spread from one muscle to the other muscles of the limb. Intramuscular anaesthesia of the muscles in which the rhythmic activity originates stops the rhythmic phenomena in all muscles of the corresponding limb. This procedure was neither effective in suppressing the resting tremor in 3 of our PD patients with classic resting tremor, rigidity and hypokinesia at the injected side, nor in PD patients elsewhere, Rondot *et al*³ and Rondot (personal communication).

Sabra and Hallett⁴ argued that in cases of severe tremor appearing on postural maintenance, Holmes' term "rubral tremor" should be avoided; "severe postural cerebellar tremor" is more appropriate because it is mainly the superior cerebellar peduncle which is involved. The most typical vascular form of this postural tremor is associated with a contralateral third nerve palsy.⁵ Antagonist muscles in these patients showed Parkinson-like alternating activity. Both findings are also present in our patient. Although Sabra and Hallett mention only one of their 32 patients having a tremor at rest, our case suggests that postural tremor at rest may be part of such postural tremor.

We cannot exclude the possibility that, depending on the site and extent of the lesion, other patients with so-called midbrain tremor show the characteristic resting tremor of PD. Dopa responsive midbrain tremor^{6,7} may belong to this group. Patients with midbrain tremor will be studied carefully in an attempt to resolve this issue.

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Transcutaneous phrenic nerve stimulation

Transcutaneous phrenic nerve stimulation, with measurement of the terminal latency of the nerve, is a well recognised technique for assessing phrenic integrity. The technique used is essentially that described by Newsom-Davis in 1967,¹ with measurement of the diaphragmatic compound muscle action potential (CMAP) using surface electrodes placed over the chest wall. The exact position of the electrodes has been the subject of some discussion. Newsom-Davis originally recorded from the eighth intercostal space in the anterior axillary line. In other studies the fifth and sixth spaces, also in the anterior axillary line, the eighth space and the xiphisternum, and the seventh or eighth space near the costochondral junction have been used.² Most recent studies have used the seventh or eighth intercostal spaces just anterior to the costal margin.^{3,4}

In some papers, notably Newsom-Davis' original work,² there was slight concern over the possibility of stimulating nerves other than the phrenic, especially those supplied by the brachial plexus, and producing a CMAP that did not reflect diaphragmatic contraction. This was not borne out clinically and brachial plexus stimulation, while common (especially in children⁴), does not affect the CMAP seen. Other muscles which also may be stimulated, such as *larissimus dorsi*, lie too far away from the electrodes to affect the signal. Stimulation of the serratus anterior muscle was also suggested as a possible confounding contraction² but anterior placement of the electrodes should avoid this as the origins of the muscle are from the lateral borders of the upper 8-10 ribs.

We are involved in a prospective study of phrenic nerve function in children having cardiac surgery, and over the period of a year have successfully studied over 250 children before and after surgery. Chest electrodes are placed over the seventh intercostal space and over the eighth rib in the anterior axillary line. In a small number of children we are now recognising an artefactual trace which was initially thought to be part of the diaphragmatic CMAP; we now recognise that it is clearly not. Figure 1 shows the preoperative trace of a normal five year old boy, with latency measured at 5 ms. Post-operatively his trace was that seen in figure 2. This shows an apparent latency of 2.6 ms with a normal appearance to the CMAP.