

Total peroxidase activity was 16% higher in the cerebellar tissue than in cerebral cortex ($p < .005$) in both experimental and control groups. Measured peroxidase activity includes both intravascular and extravascular compartments. Since tissue samples taken for peroxidase measurements include the rich vasculature of the pia, and since the amount of pia per unit brain tissue is much higher in the cerebellum, it is possible that the higher total peroxidase activity in cerebellum merely represents its larger pial compartment. However, histological measurement of leakage sites did not include leaks that were associated with the pial vasculature, but only intraparenchymatous sites. The number of leakage sites was very low in all samples, in keeping with the normal status of the BBB, but was consistently 50% higher in cerebellum compared with cerebrum ($p < 0.001$). This difference was seen in both experimental and control groups. This apparent leakiness of cerebellar vasculature, compared to cerebral cortex, should be further investigated. It is intriguing to speculate that an increase in BBB leakiness might be associated with the well-known vulnerability of the cerebellum to blood-borne toxins.

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TREMOR IN PATIENTS WITH COMPLICATED MIGRAINE

To the Editor:

Ischemic events in the territory of the posterior cerebral artery have been implicated in the generation of various movement disorders such as choreoathetosis, tremor, etc.¹ C.M. Fisher has proposed the term "catastrophic migraine" to describe permanent neurological deficits following migrainous attacks. Recently, we have seen three patients who had vascular headaches accompanied by a sudden onset of tremor and in whom an intermittent tremor was the only sequela. One of the case histories is described below.

A 32-year-old woman, with a personal and family history of migraine, came to medical attention because of a tremor of the left upper extremity. Four weeks prior to consulting, she noticed a sudden onset of burning pain in the left shoulder followed by an intermittent tremor of the left arm which occurred twice a week and lasted 1-2 days. Two days after the onset of tremor, she started complaining of severe and tenacious headaches associated with sonophobia and photophobia. On two occasions, the headaches preceded the onset of the tremor by one hour. She

also felt that the left side of her body was "like swollen" and this was made worse by headaches and tremor attacks. During the past 2 years, she had one episode of bilateral loss of vision lasting less than 10 seconds. On neurological examination, she had a decreased finger tapping on the left side and a coarse 3-5 Hz resting tremor of the left arm which was equally present on goal-directed movements and attempts to distract the patient but was slightly reduced in frequency during sleep. On 3 occasions, the tremor completely ceased 30 to 90 minutes following an IV injection of dihydroergotamine. Treatment with nifedipine 10 mg QID was ineffective. However, she had no recurrence of the tremor while treated with both nifedipine 10 mg QID and propranolol 40 mg QID.

Extensive laboratory tests including CBC, sedimentation rate, RA test, ANA, serum complement level, cryoglobulinemia, serum copper and ceruloplasmin, thyroid function tests, ECG, echocardiogram, CSF studies, EEG, and multi-modality evoked potentials were unremarkable. Surface EMG recording revealed a 3-5 Hz alternating tremor of the left arm muscles.

Tremor has rarely been described with migrainous headaches. In an article dealing with unusual vascular events in the territory of the posterior cerebral artery, C.M. Fisher¹ reported 10 patients in whom a sensory, motor, or visual deficit occurred with severe vascular headaches. In 4 of them, a resting tremor of the paretic extremity appeared 1 to 9 months after the initial event. These vascular events seemed to be of migrainous origin since no embolic source could be found. As compared to Fisher's patients, the tremor in our 3 patients was not associated with a severe neurological deficit, its onset was not delayed, and the fluctuating character of the tremor was striking. The tremor could be present for several days at a time and was on a few occasions temporally related to headaches. Two of our patients described a pain sensation in the trembling extremity and one of them complained of a "swollen" feeling on one side of the body. These descriptions suggest sensory symptoms of thalamic origin although the actual involvement of the latter remains unclear.

The cause of this unusual movement disorder is unknown. However, there is some evidence suggesting that it may be due to a vascular process such as seen in migraine. The laboratory investigation eliminated most known disease processes associated with tremor, namely chorea, systemic lupus erythematosus, Wilson's disease, multiple sclerosis, and emboli to the basal ganglia. Atherosclerosis seems highly unlikely in young women aged 23 to 32, and recurrence of the movement disorder in the same limb argues against an embolic process. A psychogenic origin also seems unlikely since the tremor persisted during sleep and upon distracting the patient. The presence of intermittent sensory and visual symptoms in young women with a personal and family history of vascular headaches suggests that migraine could be a plausible process. Therefore, we postulate that the tremor seen in our patients is most probably caused by migraine and represents a migraine equivalent.

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