Letters

Treatment of post-traumatic choreo-athetosis with sodium valproate

Sir: We report a study which establishes the efficacy of sodium valproate in the treatment of post-traumatic choreo-athetosis.

A 28-year-old male was admitted to the hospital. At the age of nine years, he fell from a three storey building sustaining severe left sided cerebral damage. He was in coma for about one month, making a gradual recovery over the next four months. During recovery he developed right sided focal seizures, a right spastic hemiparesis and choreo-athetoid movements of the right arm. On examination, the abnormal findings were all on the right side and included a homonymous hemianopia, a spastic hemiparesis, mild limb atrophy, a cortical type of sensory deficit and a Babinski sign. Choreo-athetoid movements of the right arm were present, consisting of flexion-extension of the fingers and wrist supination, pronation and flexion of the elbow, and abduction of the shoulder. These movements were absent in sleep and increased by the stress induced by rapid questioning. The EEG showed sharp waves in the left frontal and temporal areas and a mild degree of slow wave abnormality in the same area. CT of the head showed a large area of decreased density in the left hemisphere with dilatation of the left lateral ventricle.

The patient's seizures were well controlled with phenytoin, phenobarbital and methsuximide. The patient was started on sodium valproate at a dose of 250 mg by mouth three times a day. Within twelve hours of the first dose, the patient reported improvement in his involuntary movements. After one week, the dose was doubled and then increased to 500 mg four times a day with a dramatic reduction in the frequency of involuntary movements. At this point the patient entered into a controlled trial with placebo to establish the efficacy of sodium valproate in treating his involuntary movements. A single blind study design was used. The patient was not aware whether he was on sodium valproate or placebo, as the two looked identical. Since a mechanical recording device was used to count the frequency of involuntary movements in a thirty minute time period, observer bias was eliminated, thus avoiding the need for a double blind study. Simultaneous EEG and muscle recordings were made. The EEG was recorded to ensure that the involuntary movements were not part of a focal seizure. The muscle activity was recorded from surface electrodes placed on the deltoid, biceps and brachioradialis, the three muscles most involved in this patient's movement disorder. The frequency, duration and amplitude of the deflections from the resting state were used to classify the movements into major, moderate and minor. Sodium valproate levels were checked twice after administering the oral medication. Muscle activity was recorded continuously for 30 minutes each time.

The frequency of major, moderate and minor involuntary movements and the corresponding sodium valproate levels are shown in the table. As serum sodium valproate levels rose, there was a reduction in the frequency of major and moderate involuntary movements. The minor involuntary movements observed at higher levels of sodium valproate were shorter in duration and involved fewer muscle groups compared to moderate and major movements.

The progressive improvement in this patient's movement disorder as serum sodium valproate levels increased strongly suggests a cause and effect relationship. Furthermore, the "blinded" study design used adds strength to the observation. Recently there have been a growing number of reports of the use of sodium valproate in diverse movement disorders. Two groups reported its use for the treatment of hemiballism. Other movement disorders successfully treated with sodium valproate include Sydenham's chorea, dyssynergia cerebellaris myoclonica, non-epileptic myoclonus and kinesiogenic familial paroxysmal choreo-athetosis. In a previous report we emphasised the possible significance of the rapidity of clinical response to sodium valproate. A review of other studies shows that a similar early response is seen in other movement disorders. The patient responded within twelve hours of onset of therapy. However, the biological significance of this observation can only be determined once the exact mechanism of action of sodium valproate is determined.

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References


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Recurrent cerebral abscess in hereditary haemorrhagic telangiectasia

Sir: Cerebral abscess carries a high mortality, with delay in establishing the diagnosis being an important contributing factor. Recognition of any predisposing cause facilitates earlier diagnosis and improves