

LETTERS TO THE EDITOR

Swelling at the site of a skull defect during migraine headache

Goltman¹ reported a patient with a cranial bone defect who had noted that the affected area became depressed before her migraine headache started and bulged during the headache phase. A patient's description of the same phenomenon prompts this brief case report.

A security patrolman aged 33 had been subject to headaches since he was 14 years old. These had recurred every six to 12 months until he reached the age of 31 when the frequency increased to twice each month. At the age of 18 he had fallen from a building site, sustaining a comminuted fracture of the skull that necessitated the removal of a bony fragment from the occiput.

His headaches were preceded by blurred vision associated with the impression of "bubbles like those in a fish tank, floating in circles" which persisted for 20-60 minutes. The headache then started as a pain in his right temple that radiated backwards to the right occiput. As the headache increased in severity, he became nauseated and sensitive to sound but not to light or smells. He then became aware that his bony defect had swollen and felt firm "like a large marble or small egg". It became larger as the headache developed and later subsided as the headache eased. He had not observed any change in the skull defect during his visual aura. On examination, the defect, roughly 3 × 2 cm, was palpated in the midoccipital region but no other relevant abnormality was detected.

It is odd that 60 years have elapsed since Goltman's paper without similar descriptions being published. Nevertheless, these findings indicate that, in some patients at least, intracranial pressure increases during migraine headache. Possible mechanisms include dilatation of intracranial arteries,² the release of vasodilator peptides such as calcitonin gene related peptide (CGRP),³ a perivascular "sterile inflammatory response",⁴ and, possibly, breakdown of the blood-brain barrier.

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- 1 Goltman AM. The mechanism of migraine. *J Allergy* 1935;36:351-5.
- 2 Thomsen LL, Iversen HK, Olesen J. Cerebral blood flow velocities are reduced during attacks of unilateral migraine without aura. *Cephalalgia* 1995;15:109-16.
- 3 Goadsby PJ, Edvinsson L. The trigeminovascular system and migraine: studies characterising cerebrovascular and neuropeptide changes seen in humans and cats. *Ann Neurol* 1993;33:48-56.
- 4 Moskowitz MA. Interpreting vessel diameter changes in vascular headaches. *Cephalalgia* 1992;12:5-7.

Thalamic stimulation for severe action tremor after lesion of the superior cerebellar peduncle

Severe postural cerebellar tremor (SPCT) generally involves cerebellar outflow tracts. Experimental lesions of the superior cerebellar peduncle produce severe SPCT in monkeys,¹ but clinical and pathological correlations are rarely available in humans. This kind of tremor is particularly disabling. Many drugs have been used without success. Stimulation of the ventral intermediate nucleus of the thalamus has been recently considered to be efficient in parkinsonian and essential distal tremors but not in proximal postural tremors.² Interest in this surgical technique has grown with the use of a multiple point electrode which offers the possibility of stimulating different areas within the ventral intermediate nucleus.³ We report a patient with severe SPCT secondary to a cavernous angioma limited to the superior cerebellar peduncle who was dramatically improved by thalamic stimulation.

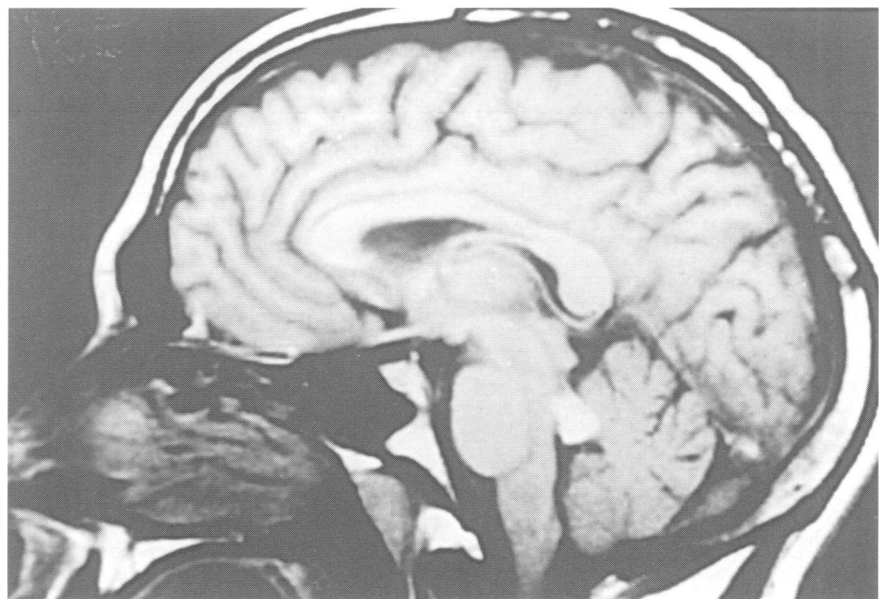
A 33 year old man had an acute paresis of the right arm with mild dysarthria, transient vertigo, and left thoracobrachiofacial paraesthesia in January 1990. Two days later, he complained of severe tremor of his right arm. Neurological examination disclosed left thermoalgic hypoesthesia, rotatory nystagmus, right Horner's syndrome, and severe action tremor of his right arm. His gait was mildly disturbed. Magnetic resonance imaging showed a small haematoma in the right superior cerebellar peduncle (figure). The patient was operated on in October 1990 and the diagnosis of cavernous angioma was confirmed histologically after neurosurgical excision. The tremor disappeared for one week only.

The patient continued to complain of a severe tremor of his right arm and was admitted to our unit in May 1991. The amplitude of the tremor was greatest (more than 10 cm) when the patient tried to keep his forefinger near his nose and was least (less than 2 cm) when he stretched out his arms. It was so disabling that the patient had no functional use of his right arm and was unable to perform daily activities such as

drinking, writing, or eating. A mild postural tremor of the right leg was present without functional consequences. No rest or head tremor was seen. Clinical examination showed a slight hypotonia of the upper and lower right limb, right patellar pendular reflex, and hypoesthesia of the left face. Somatosensory evoked potentials showed normal latencies.

The patient underwent stereotactic implantation of a brain electrode with four contact points (Medtronic®).³ Contrast ventriculography was used to identify the anterior and posterior commissures. The target was defined as the intersection of the inter-commissural line and the rostral part of the ventral intermediate nucleus located according to Guiot's geometric diagram. The electrode was introduced to place the three distal contacts in the left ventral intermediate nucleus. During the operation, the patient was awake and the abolition of the tremor confirmed the adequate placement of the electrode. On another occasion, the electrode was connected to a Ytrell II stimulator (Medtronic®). With optimal stimulation (amplitude: 2.5 V; frequency 130 Hz, pulse width 270 μs, negative plots 2, 1, and 0, positive case), the patient was able to drink from a full glass, catch an object, and write with moderate difficulties. No adverse effect was seen. Examination when the stimulator was on disclosed a right kinetic cerebellar syndrome with hypermetria, adiadococinaesia, and slight hypotonia. In 1993, the patient continued to be improved by the stimulation.

The tremor in our patient affects proximal more than distal muscles and could be considered according to classic terminology as a severe postural cerebellar tremor.⁴ This has been classically associated with lesions of the superior cerebellar peduncle. This kind of tremor was initially described in ischaemic or tumorous lesions of the brainstem, but the most common aetiologies are multiple sclerosis and severe brain trauma.⁴ In these conditions, the lesions are not usually limited to the superior cerebellar peduncle. Our finding is interesting because of the limited character of the lesion and illustrates the role of the superior cerebellar peduncle



T1 weighted (TR/TE = 600/20) MRI shows a small haematoma in the right superior cerebellar peduncle.