

FIVE-YEAR CURE OF HEMIFACIAL SPASM

Report of a Case

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HEMIFACIAL spasm (*Fig. 1*) is a condition in which paroxysms of contractions affect the muscles supplied by the facial nerve. In a review of 106 cases, Ehni and Woltman¹ point out that the twitchings resemble those resulting from intermittent faradization of the nerve. The eyelids are almost always involved. The condition is usually unilateral and may occur during sleep. The patient feels no compulsion to make the movement and is unable to stop it. He cannot produce the twitchings voluntarily with anything like the speed with which they spontaneously occur. Voluntary movements may precipitate the spasms. The condition affects women more commonly than men and does not occur in children. Fourteen of Ehni and Woltman's¹ patients had impaired hearing on the affected side and three had tic douloureux. Medical treatment was of no help. These authors reported no pathologic study in their own cases, but stated that such study by other investigators either disclosed nothing to account for the spasm or revealed gross progressive lesions such as tumors or aneurysms.

The analogy between hemifacial spasm and trigeminal neuralgia is obvious from the above account. Since intracranial neurolysis of the fifth nerve relieves the painful paroxysms of trigeminal neuralgia,² it seemed worthwhile to try a similar operation on the seventh nerve for the motor paroxysms of hemifacial spasm.

Report of a Case

A 36-year-old woman was examined in June, 1953, because of twitching of the left side of the face, which had increased steadily in severity for two years. The twitching consisted of bursts of clonic muscular spasms that were intermittent and limited strictly to the left side. Each paroxysm lasted from 10 to 20 seconds, usually starting at the angle of the mouth and spreading to the orbicularis oculi and all of the muscles supplied by the facial nerve. Each paroxysm was followed by a refractory period of 10 seconds or longer. The twitching of the facial muscles was precipitated by movements of the face particularly in talking and in eating, but also occurred at rest. She was free of the twitching for as long as four hours. There had been progressive impairment of hearing in the left ear for 12 years (since 1941). There was no associated tinnitus. For the preceding year she was subject to attacks of vertigo and nausea which were apt to occur when she started to eat.

On examination the patient exhibited typical paroxysms of left hemifacial spasm involving all muscles supplied by the facial nerve. Between attacks there appeared to be slight weakness of the lower facial muscles. There was no impairment of sense of taste. Audiometric studies disclosed total deafness in the left ear and mild mixed deafness in the right ear. Caloric stimulation with 1 ml. of ice water elicited no vestibular response on either side. Roentgenograms of the skull were negative. The cerebrospinal fluid pressure was normal. The clinical impression was a lesion in the left cerebello-

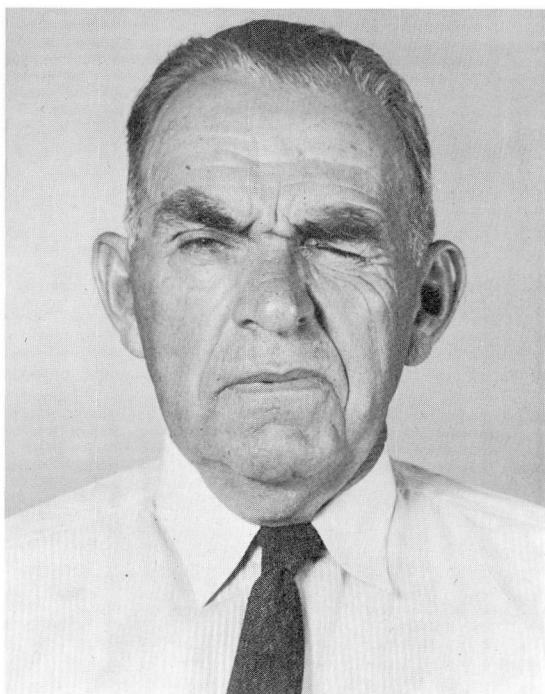


Fig. 1. Photograph of a patient with typical hemifacial spasm.

pontine angle causing hemifacial spasm and deafness. *Banthine** taken orally and vitamin B₁₂ administered intramuscularly afforded her no relief.

The patient was re-examined two years later, at which time the condition was unchanged except that caloric stimulation produced slight but definite responses in each ear. Because of increasing experience with the effectiveness of neurolysis in the treatment of trigeminal neuralgia, a surgical exposure of the seventh nerve was advised.

On July 25, 1955, with the patient in the sitting position, a right suboccipital craniotomy was performed and the cerebellar lobe was retracted from the posterior surface of the petrous bone. A loop of the internal auditory artery lay against the posterior surface of the eighth nerve. The arachnoid of the pontine cistern was opened and the artery was dissected free of the nerve. The portion of the seventh nerve that could be seen appeared normal. The seventh and eighth nerves were gently manipulated with a nerve hook introduced into the porus acusticus internus. A piece of Gelfoam then was placed beneath the artery to separate it from the eighth nerve, and the operation was concluded.

For 24 hours after operation facial twitching was absent and there was no weakness of the facial muscles. Hemifacial spasm then recurred, but it was much milder and less frequent than before operation. Six months postoperatively the hemifacial spasm con-

**Banthine* Bromide, *methantheline bromide*, U. S. P. (β -diethylaminoethyl xanthene-9-carboxylate methobromide), G. D. Searle & Co.

sisted of occasional, slight, fibrillary tremors of the orbicularis oculi and occasionally a slight twitching of the angle of the mouth. There had been no further attacks of vertigo. The hearing loss was unchanged. Five years postoperatively (1960), the patient reported only the rare occurrence of a barely detectable tremor of the lower eyelid.

Summary

In a patient with hemifacial spasm the seventh nerve was treated by an intracranial neurolysis similar to that employed on the fifth nerve for the relief of trigeminal neuralgia. The spasm rapidly diminished after operation, and five years later the patient was without symptoms except for a rare slight tremor of the lower eyelid.

References

1. Ehni, G., and Woltman, H. W.: Hemifacial spasm; review of 106 cases. *Arch. Neurol. & Psychiat.* 53: 205-211, 1945.
2. Gardner, W. J., and Miklos, M. V.: Response of trigeminal neuralgia to "decompression" of sensory root; discussion of cause of trigeminal neuralgia. *J. A. M. A.* 170: 1773-1776, 1959.