Refer to: Hankinson HL, Wilson CB: Microsurgical treatment of hemifacial spasm. West J Med 124:191-193, Mar 1976

Microsurgical Treatment of Hemifacial Spasm

H. L. HANKINSON, MD, *and* CHARLES B. WILSON, MD, *San Francisco*

Five cases of idiopathic hemifacial spasm have been successfully treated by operative manipulation of arterial branches compressing the VIIth nerve in the posterior fossa. Terminology, clinical presentation, pathology and therapeutic approaches to hemifacial spasm are discussed. Hearing loss due to operatively induced vascular impairment of the inner ear, a complication in our first case, should be avoidable.

Our experience indicates that hemifacial spasm reflects mild chronic compression of the facial nerve. The proposed mechanism is transaxonal excitation between afferent and efferent fibers.

HEMIFACIAL SPASM (HS) is an uncommon affliction characterized by embarrassing and fatiguing facial twitching, usually cryptogenic but occasionally associated with subtentorial lesions, predominantly extra-axial vascular and neoplastic.[1,2] Because the cause is poorly understood, therapy has been empiric and unsatisfactory, and relief has been at the price of significant weakness of facial muscles.[3-5] Jannetta[6] has reported a series of eight patients in whom, with the aid of an operating microscope, he separated the facial nerve from a large branch of the anterior inferior cerebellar artery (AICA) in the cerebellopontine angle (CPA). His results in this initial study as well as in his subsequent[7-9] experience have been excellent.

Our series of five cases treated by Jannetta's microsurgical method confirms the efficacy of the operation and gives weight to the hypothesis that HS is often caused by mild chronic compression of the facial nerve.

From the Department of Neurological Surgery, University of California, San Francisco.

This work was supported in part by NINDS Training Grant No. 5593.

Submitted August 4, 1975.

Reprint requests to: Charles B. Wilson, MD, Department of Neurological Surgery, University of California School of Medicine, San Francisco, CA 94143.

Method and Case Reports

Since 1970 microsurgical vascular decompression of the intracranial facial nerve has been carried out in five of our patients with HS. All were women. Ages ranged from 44 to 66 years. The length of time from onset of symptoms to operation was 3 to 15 years. The patients have been followed up from three months to five years.

Surgical Technique

The operation is carried out under general anesthesia. Although Jannetta prefers to have the patient sitting, we have used a semiprone position. Through a short vertical incision medial to the mastoid process, a 3 cm craniectomy is used to expose the lateral and inferior angle of the posterior fossa. Under the operating microscope the responsible artery, usually a branch of the

ABBREVIATIONS USED IN TEXT
AICA = anterior inferior cerebellar artery
CPA = cerebellopontine angle
HS = hemifacial spasm

posterior inferior cerebellar artery, is identified at the point where the facial nerve exits from the brainstem. The artery is teased away from the facial nerve, and a Teflon® sponge, fashioned to held the artery away from the junction of facial nerve and brainstem, is interposed.

Results

In the first patient (Case 1), an operation was carried out unsuccessfully one year before the procedure described in this report. At the original operation an arterial loop was found lying across the facial nerve midway between the brainstem and the internal auditory canal. At that time (six years ago) we did not recognize that the responsible artery compressed the facial nerve at its origin from the brainstem, and this *proximal* segment of the nerve was not exposed. Interposing a sponge between the arterial loop and the *distal* facial nerve failed to stop the patient's hemifacial spasm, and because of traumatic spasm induced in the internal auditory artery the patient became deaf. One year later the correct procedure was carried out with immediate disappearance of hemifacial spasm.

In all five patients there has been complete absence of hemifacial spasm for the duration of follow-up (three months to five years), and in all there has been normal function of the affected facial nerve. Except in Case 1, preoperative function of the VIIIth nerve has been retained in all patients. With no exceptions the postoperative course has been uncomplicated. Our results are summarized in Table 1.

Discussion

Hemifacial spasm has been called reflex facial spasm, facial hemispasm, chronic faciospasm, nictitating spasm and facial tic. Cushing coined the term "tic convulsif" to denote HS associated with tic douloureux. Nosik[10] classified isolated facial spasm into categories characterized by (a) cortical irritation, (b) facial nucleus involvement and (c) psychogenic origin. Wartenberg[11] further classified HS caused by nuclear firing as (a) cryptogenic, resulting from infectious or degenerative changes, (b) lesions of the peripheral facial nerve and (c) postparalytic following Bell's palsy. In addition HS may be classified as cryptogenic or idiopathic, and secondary. For the purposes of this paper only the former is considered to be true HS.

The clinical features are characteristic. The spasm usually begins in the periorbital muscles and is of spontaneous onset. Although mild at onset, insidious progression to involve most of the musculature of that side of the face is the rule. Spontaneous remission has been reported,[12] but is unusual. The affected side is equally likely to be the right or the left, although occasionally bilateral movements are observed. Should bilateral movements occur, one side does not move in synchrony with the other. A mixed tonic-clonic spasm is characteristic. Women are affected more often than men; HS is exceedingly rare in childhood. In adults the onset may occur at any age, although it begins most commonly between 30 and 50 years.

Paroxysms of facial spasm are precipitated by numerous factors including nervousness, fatigue and volitional facial movement. Movement often persists during sleep. Volitional effort fails to terminate the spasm. Findings on neurological examination are normal with the exception of the VIIth and VIIIth nerves unless the HS is associated with another pathologic condition. In 106 cases of cryptogenic HS reported from the Mayo Clinic by Ehni and Woltman,[13] facial weakness was present in 16 patients and hearing impairment on the side of the lesion in 14.

Contrast studies are not helpful except for the occasional discovery of a vascular abnormality in the region of the CPA[14] and assurance that the spasm is not secondary to one of the numerous pathological entities capable of producing sec-

TABLE 1.—*Results of Treatment for Hemifacial Spasm*

Case No.	*Age*	*Sex*	*Duration*	*Follow-up: No Recurrence*	*Complications*
1	47	Female	5 years	5 years	Deafness in left ear from previous operation
2	44	Female	3 years	1½ years	.....
3	61	Female	6 years	1 year	Mild hearing loss postoperatively; normal hearing restored
4	66	Female	4 years	6 months	.....
5	67	Female	15 years	3 months	.....

ondary facial spasm. Pathologic entities associated with HS include neoplasm and aneurysm in the CPA, basilar arachnoiditis,[1] intrinsic brainstem neoplasm,[2] purulent meningitis, tetanus, basal ganglion lesions, trigeminal neuralgia[15] and Paget's disease.[16] Virtually all of these entities cause additional neurologic abnormalities. Morbid pathology in HS is scarce, however, since the condition is benign.

Based on his observations in dealing with arterial loops compressing the Vth nerve in trigeminal neuralgia, Jannetta carried out operations and reported findings in eight cases of HS. He was successful in relieving facial spasm in all eight patients although in three hearing was lost in the ipsilateral ear and in one there was postoperative facial palsy. His series now numbers about 68 cases and he continues to advocate this approach.[9] There have been no additional cases of postoperative hearing loss since those that he described in his earlier series.

It appears certain that the basic pathologic lesion lies either at the nuclear level or distally.[17] Return of facial spasm due to regeneration of the interrupted VIIth nerve at the stylomastoid foramen may be prevented by anastomosis of the distal end with XI or XII. This would favor placement of the responsible lesion between the nucleus and stylomastoid foramen.[12] Gardner's attractive theory[12,18] that squeezing together nerve fibers results in reducing the thickness of myelin sheaths, leading to transaxonal short circuiting between afferent and efferent fibers, is supported by findings from both experimental[19,20] and clinical[21] observations.

Various nonspecific procedures have been applied to the treatment of HS. All of these are indirect approaches designed to differentially damage all or part of the facial nerve.[5] Although efficacious at times, alcohol injection, crushing or surgical interruption of the facial nerve results in cosmetically undesirable weakness of facial muscles. In addition, recurrence of the spasm is common.[3]

We believe that microdissection of arterial structures from the VIIth cranial nerve in the posterior fossa is a safe and effective method of dealing with cryptogenic HS and consider it the method of choice in dealing with this entity. Hearing loss due to operatively induced vascular impairment of the inner ear is a risk that the patient must accept, although this risk has been reduced with added experience.

REFERENCES

1. Hemifacial spasm [Editorial review article (unsigned)]. Br Med J 4:624-625, 1972

2. Sogg RL, Hoyt WF, Boldrey EB: Spastic paretic facial contracture—A rare sign of brainstem tumor. Neurology 13:607-612, 1963

3. Greenwood J: The surgical treatment of hemifacial spasm. J Neurosurg 3:506-510, 1946

4. Scoville WB: Hearing loss following exploration of cerebellopontine angle in treatment of hemifacial spasm. J Neurosurg 31:47-49, 1969

5. Scoville WB: Partial extracranial section of seventh nerve for hemifacial spasm. J Neurosurg 31:106-108, 1969

6. Jannetta PJ: Microsurgical exploration and decompression of the facial nerve in hemifacial spasm. Curr Top Surg Res 2:217-220, 1970

7. Jannetta PJ: Taking pressure off facial nerves. Med World News 13:44, 1972

8. Jannetta PJ: The cause of hemifacial spasm—Definitive microsurgical treatment at the brainstem in 31 patients. Trans Am Acad Ophthal Otolaryn, 1974, pp 319-322

9. Jannetta PJ: Personal communication.

10. Nosik WA, Weil AA: Selective partial neurectomy in hemifacial spasm and the electrophysiologic selection of patients. J Neurosurg 13:596-602, 1956

11. Wartenberg R: Hemifacial Spasm—A Clinical and Pathophysiological Study. New York, Oxford University Press, 1952, p 86

12. Gardner WJ, Sava GA: Hemifacial spasm—A reversible pathophysiologic state. J Neurosurg 19:240-247, 1962

13. Ehni G, Woltman HW: Hemifacial spasm—Review of one hundred and six cases. Arch Neurol Psychiat 53:205-211, 1945

14. Carella A, Caruso G, Lamberti P: Hemifacial spasm due to elongation and ectasia of the distal segment of the vertebral artery—Report of two cases. Neuroradiology 6:233-236, 1973

15. Weil AA, Nosik WA: Electrophysiologic and clinical observations in hemifacial spasm. Neurology 6:381-389, 1956

16. Gardner WJ, Dohn DD: Trigeminal neuralgia—hemifacial spasm—Paget's disease: Significance of this association. Brain 89: 555-562, 1966

17. Campbell E, Keedy C: Hemifacial spasm: A note on the etiology in two cases. J Neurosurg 4:342-347, 1947

18. Gardner WJ: Concerning the mechanism of trigeminal neuralgia and hemifacial spasm. J Neurosurg 19:947-958, 1962

19. Granit R, Leksell L, Skoglund CR: Fiber interaction in injured or compressed region of nerve. Brain 67:125-140, 1944

20. Manozzi AS, Lorente de No R: Interaction of neighboring fibres in myelinated nerve. J Neurophysiol 7:83-101, 1944

21. Neagory DR, Dohn DF: Hemifacial spasm secondary to vascular compression of the facial nerve. Cleve Clin Quart 41: 205-214, 1974