HEMIFACIAL SPASM—A REVERSIBLE
PATHOPHYSIOLOGIC STATE*

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In 19 patients having hemifacial spasm, surgical exposure and neurolysis of the
7th nerve was performed in the cerebello-
pontine angle. In 7 cases the nerve was found
to be compressed by a definite pathologic
process, and in an additional 7 by a redu-
dant loop of the anterior inferior cerebellar or
internal auditory artery. In 5 there was no
apparent compression of the nerve. Gentle
manipulation of the exposed nerve repro-
duced the hemifacial spasm. The operation
was followed by complete relief of spasm in
12 patients, by satisfactory relief in 3, by no
relief in 2 and by recurrence in 2. This article,
however, concerns mechanism rather than treatment.

REVIEW OF LITERATURE
Wilson23 introduced the subject of hemi-
facial spasm as follows:

"Facial spasm may be cryptogenic or sympto-
matic, non- or postparalytic, uni- or bilateral,
partial or total, tonic, clonic, tonicoclonic, or
fibrillary. Common though it is, both causation
and pathogenesis are obscure, while pathological
data are scanty and ambiguous."

In 1945 Ehni and Woltman5 analyzed 106
cases of cryptogenic hemifacial spasm se-
lected from the records of 663 patients with
various "unwonted" movements of the face.
They pointed out that the spasm was limited
to the muscles supplied by the 7th nerve and
characterized by contractions resembling a
response to intermittent faradization of the
nerve. They found that the spasm usually
originated in the orbicularis oculi which was
the muscle involved most frequently, that
the patient felt no compulsion to make the
movement and was unable to stop it by
exercise of the will. It occurred predomin-
antly in women, only in adults, and with
equal frequency on either side. Psychic up-
sets, fatigue, and voluntary movements made
the spasms worse, but in 12 patients spasms
also were observed during sleep. In 6 of their
cases the spasms were bilateral but were
neither synchronous nor symmetric. Nine of
their patients had experienced a spontaneous
remission. There was trigeminal neuralgia
on the side of the spasm in 3 patients. In 2
the hemifacial spasm had been preceded by
facial paralysis although in neither was the
twitching of an associated-movement type.

Ehni and Woltman5 confirmed the previ-
ous observations of others, that the spasm
never spreads beyond the distribution of the
7th nerve, is not affected by a stroke, and
therefore cannot be the result of a cortical
discharge. They found that regeneration fol-
lowing interruption of the facial nerve at the
stylomastoid foramen almost always was
accompanied by recurrence of the spasm,
whereas, as had been shown previously,5,12 if
the facial nerve was divided at this foramen
and anastomosed with the 11th or 12th
cranial nerve, the reinnervation of the face
was not accompanied by recurrence of the
spasm. These demonstrations, they pointed
out, proved that the lesion causing this
condition must lie in the portion of the nerve
between the stylomastoid foramen and the
nucleus. They found that little or no benefit
was to be expected from the use of drugs
including Dilantin Sodium.

Although Ehni and Woltman5 concluded that
gross lesions of the facial nerve can and
do cause symptomatic twitching that is very
like if not indistinguishable from true or cryptogenic hemifacial spasm, they nevertheless excluded from their series every case in which there was evidence of a gross pathologic lesion in the posterior cranial fossa. They apparently did not consider the associated neurologic abnormalities in 41 patients as evidence of such a lesion.

Wartenberg\(^2\) in 1952 stressed the fact that in hemifacial spasm there frequently is definite though mild weakness of the affected facial muscles as well as the intrafacial associated movements (synkinesis) that sometimes develop after recovery from Bell's palsy. He pointed out the many similarities between cryptogenic and postparalytic facial spasm and concluded that both are caused by a lesion in the facial nucleus. A study of the illustrations of facial asymmetry that he interpreted as weakness, however, shows it more likely to be the result of the associated contraction (synkinesis) of antagonistic muscles. He agrees with previous authors that:

"Gross organic lesions directly affecting the nucleus or trunk of the facial nerve can certainly produce the clinical picture of hemifacial spasm. . . . But practically speaking, in the genesis of hemifacial spasm such gross organic lesions are so rare as to be negligible."

In 1917 Cushing\(^4\) reported 4 instances of facial twitching in 30 patients having tumors of the 8th nerve: an illustration of 1 of these patients discloses classic hemifacial spasm. In 1920 he\(^5\) described the association of hemifacial spasm with tic douloureux in 3 patients and applied the term tic convulsif to this combination. Revilla, reporting Dandy's experience with tumors in the cerebellopontine angle, described hemifacial spasm in 4 of 160 patients with neurinomas,\(^16\) in 1 of 13 with meningiomas,\(^17\) and in 1 of 13 with cholesteatomas. Laine\(^14\) described hemifacial spasm in a patient having cirsoid aneurysm of the basilar artery, while Campbell and Keedy\(^2\) found the same lesion in 2 patients with the combination of hemifacial spasm and trigeminal neuralgia.

Woltman et al.\(^24\) carried out a neurolysis by uncovering the facial nerve in its bony canal but abandoned the procedure\(^25\) when analysis of the results showed that only 2 of 10 patients had lasting benefit. They were surprised to find that their patients also were relieved of their synkinesis, since this form of associated movement generally is attributed to anatomic misdirection of regenerating nerve fibers. As a possible explanation for hemifacial spasm and its accompanying synkinesis they suggested that local irritation of the nerve causes spontaneous activity and facilitates the initiation of impulses in inactive fibers by impulses traveling over adjacent fibers.

Varying degrees of relief of hemifacial spasm have been described following various types of partial interruption of the extraocular portion of the facial nerve\(^3,11,15,18\) but perhaps the first planned attempt to relieve cryptogenic hemifacial spasm by an intraocular approach was by Bradgon.\(^1\) He crushed the nerve with a hemostat in the cerebellopontine angle in 6 patients, producing complete facial paralysis that subsequently recovered with no return of the hemifacial spasm. The first of his patients so treated has been relieved for 11 years. We believe it significant that while this procedure produced temporary paralysis of efferent fibers, it probably resulted in permanent interruption of afferent fibers. Bradgon's results thus lend support to Hunt's\(^18\) concept that hemifacial spasm may result from irritation of the sensory portion of the 7th nerve "conveyed directly to its motor nucleus."

**ANALYSIS OF CASES**

In our 19 patients the ages at operation varied from 38 to 75 years, and the duration of the hemifacial spasm from 5 months to 18 years (Table 1). Fifteen of the patients were women and 4 were men. In 9 cases the right side was involved, in 8 the left. Only 1 of these patients (Case 3B) had experienced a spontaneous remission. The spasm was entirely unilateral with the exception of Case 6B in whom there were observed occasional mild fasciculations in the orbicularis oculi of the opposite side. In most, if not all cases, the spasm had appeared first in the orbicularis oculi and this muscle was involved in the spasm in every instance at the time of the