CONCERNING THE MECHANISM OF TRIGEMINAL NEURALGIA AND HEMIFACIAL SPASM*

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“Whatever the cause of trigeminal neuralgia may be, it must be located in the sensory root.” — Walter E. Dandy.1

Despite the fact that one is a sensory and the other a motor phenomenon, trigeminal neuralgia and hemifacial spasm have many features in common; each occurs in spontaneous paroxysms that resemble the effect of electrical stimulation of the nerve; each is limited to the distribution of the nerve involved; occurs only in adults, predominantly in women; is subject to remissions; and although there seldom is demonstrable impairment of function of the nerve, each has been attributed to pathology in the nucleus. Each begins in one branch of the nerve and tends to spread to all. Both types of paroxysms are precipitated by facial movements on some occasions but not on others. They also occur during sleep. There is a tendency for the opposite side to become involved, in which case the attacks are neither synchronous nor symmetric. In each condition, permanent relief can be promised only by total and permanent interruption of the nerve.

All of the above features, so well recognized in trigeminal neuralgia, have been described in hemifacial spasm by Ehni and Woltman.12 In addition, Gardner and Sava19 showed that the paroxysms of hemifacial spasm, like trigeminal neuralgia, may be stopped immediately, and with no impairment of function, by a nontraumatic manipulation of the nerve root. Both trigeminal neuralgia and hemifacial spasm may occur in the same patient, in which case the paroxysms, though asynchronous, always are on the same side and relief of one by alcohol injection will not affect the other.27 Another important point of similarity is the fact that although each is considered a primary or cryptogenic disease, occasional cases are known to occur as a symptom of a tumor, aneurysm or arteriovenous anomaly in the cerebellopontine angle,11,12,19,49,50 and operation invariably has disclosed such a lesion when both conditions coexist.19

Trigeminal neuralgia differs from hemifacial spasm in the following respects: It may occur as a symptom of a tumor14 or aneurysm16 in the middle fossa; as a symptom of multiple sclerosis,25,26 Paget’s disease and basilar impression.11,20 In addition to facial movement, it also is triggered by light touch, is more common on the right side, is helped by Dilantin,29 and the paroxysms are followed by a refractory period.34,38

In 50 cooperative patients with typical trigeminal neuralgia, Kugelberg and Lindblom34 studied this refractory period and also made careful observations regarding the stimulus necessary to elicit a paroxysm (Fig. 1). One of these patients incidentally had platybasia and another an intracranial aneurysm. They found that spatial summation of the stimulus triggered the pain at a lower strength of stimulus and at a shorter latency than if a small area or a single hair were stimulated. In addition to stroking with the finger, they employed a vibrator consisting of a round head of a pin attached to the membrane of a loud speaker. Increasing the frequency or the amplitude of the vibrations shortened the time of summation. They found that a paroxysm is followed by a refractory phase, relative or absolute, the duration of which is a function of the duration as well as of the severity of the preceding

attack. The refractory period lasts from 20 seconds to 3 minutes. They concluded that a short-latency jab of pain possibly may be explained by simple interaction or short-circuiting between touch and pain fibers, but that the lesion responsible for "the trigeminal neuralgic syndrome" most likely is located in the brain stem as was thought also by Crue and Sutin7 and by List and Williams.28 They found the observations of King et al.21–22 in animal experiments difficult to apply to the phenomena that they had studied since the potential changes observed in the cat proceed on a time scale in milliseconds as compared to seconds and minutes in the human subject.

Impressed by the many similarities between hemifacial spasm and trigeminal neuralgia, and since trigeminal neuralgia is relieved by a nontraumatic manipulation of the trigeminal root,17 we carried out a similar procedure on the 7th nerve in 19 cases of hemifacial spasm.19 On exposure of the cerebellopontine angle, the nerve root was found to be compressed by a cirsoid aneurysm of the basilar artery in 3 patients; by an arteriovenous malformation in 3 (2 of whom also had trigeminal neuralgia); by a dislocation of the pons attributed to a tumor of the posterior fossa of the opposite side in 1; by an anomalous loop of the anterior inferior cerebellar artery in 7; while in only 5 cases did the nerve root appear to be uninvolved. During operation it was observed that paroxysms of hemifacial spasm were produced by the retraction of the cerebellum and by the slightest touching of the nerve, but with continued gentle manipulation, this response subsided. The operation was followed by immediate relief of hemifacial spasm in 12; by delayed relief in 5; by no relief in 2; by demonstrable weakness in 5; and by subsequent recurrence in 2.

As a result of these observations, it was concluded that hemifacial spasm is the expression of a reversible pathophysiologic state commonly produced by mild chronic compression of the 7th nerve in the cerebellopontine angle; that it results from a transaxonal "short-circuiting" of the action current because of pressure atrophy of the insulating myelin sheaths; that the associated movement or synkinesis which constitutes a feature of hemifacial spasm (similar to that seen in the post-Bell's phenomenon) can be explained by interaction between efferent fibers, while the repetitive clonic twichings are compatible with a reverberating circuit set up between afferent and efferent fibers of muscle.

The results of these operations for hemifacial spasm are comparable to those of Taarnhøj's operation for trigeminal neuralgia. In an analysis of 200 cases of the latter followed for 3 to 6 years Gardner and Miklos17 found that 62 per cent had immediate, complete and lasting relief of their pain. Contrary to the observations of others,60 they found the incidence of sensory impairment no higher in the patients with lasting relief than in those with recurrences. They found that a most effective method of manipulating the nerve root without damaging it was to spray it with a forceful stream of Ringer's solution delivered by a 10 cc. syringe through a 21-gauge needle. This method, also em-