Evidence for a Peripheral Etiology of Trigeminal Neuralgia

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Because the paroxysmal nature of trigeminal neuralgia suggests an epileptic discharge, Kinnier Wilson proposed that trigeminal neuralgia was due to a paroxysmal discharge of centrally-located trigeminal neurons. This concept has met with considerable favor and is an acceptable working hypothesis, provided it is not interpreted as an argument for a central etiology. As a matter of fact the organization of the central nuclei of the fifth cranial nerve provides one of the important lines of evidence in favor of a peripheral etiology for trigeminal neuralgia.

The portion of the fifth spinal nucleus extending several millimeters below the level of the obex is primarily concerned with pain and temperature. The evidence for this in man stems from Sjöqvist’s section of the spinal tract of the trigeminus; it has further been shown by Hamby that an incision as far as 10 mm below the obex level gives satisfactory trigeminal analgesia. Thus only a small caudal portion of the nucleus, located mainly in the C-1 and C-2 segments of the spinal cord, is the major relay for pain in this system. Brodal demonstrated thermal analgesia of the glossopharyngeal and vagal nerves in Sjöqvist’s patients and concluded that the corresponding sensory relay must reach the spinal nucleus of the trigeminus below the obex level. Cajal demonstrated fibers of the ninth and tenth cranial nerves joining the spinal tract of the fifth nerve, but Ingram and Dawkins could not trace them below C-1 or follow them to their termination in the nucleus.

The manner of termination of the three divisions of the trigeminal nerve and of nerves 7, 9, and 10 in the subnucleus caudalis (pars spinalis)* of the fifth nerve has recently been demonstrated in considerable detail. Whereas all of the divisions of the trigeminal nerve are laminated throughout their course in the spinal tract and nucleus, the endings of nerves 7, 9, and 10 are diffusely distributed throughout the nucleus, intermingling with each other and with those of the trigeminal (Figs. 58 and 59). Moreover, the sensory input from the three upper cervical dorsal roots enters this nucleus and distributes terminals widely and irregularly among those of the preceding root systems. Thus, an area of dense anatomical convergence of sensory input from all the exteroceptive cranial nerves and all sensory modalities of the upper cervical roots is located in the subnucleus caudalis. Whether such anatomical convergence is associated with physiological convergence, and if so with what root systems, has been determined only in part. Kerr and Olafson demonstrated that a proportion of the single units in the spinal nucleus of the trigeminus responded both to trigeminal and to cervical (C-1 and C-2) volleys in the cat. Whether this input was from primary terminals or via interneurons was not determined.

Our current interest has concerned central pathways for the spread of atypical face pain. Although our data do suggest a possible route for the bizarre spread of this type of pain, they also provide a contradictory type of evidence with regard to the location of the primary lesion in trigeminal neuralgia. We have summarized below the symptomatology and natural history of trigeminal neuralgia.

Characteristic Features of Trigeminal Neuralgia
1. Onset usually after age 40
2. Females affected twice as often as males
3. Right-sided involvement predominates by a ratio of 3 to 2
4. Trigger points present at some time in the course of the disease
5. Quality of pain superficial, intense, brief, and paroxysmal
6. Pain limited strictly to some part of the distribution of the fifth cranial nerve
7. Less than 5% start in the first division
8. Extreme rarity of combined first and third division involvement
9. 3% of cases ultimately bilateral
10. No neurological deficit, or one detectable only with von Frey hairs
11. Tendency to progression in the frequency and severity of episodes

* The term “pars spinalis” of the subnucleus caudalis is used here for that portion of the nucleus which lies in the spinal cord and is characterized by its multiple cranial and cervical root inputs and by the pronounced anatomical convergence of these systems.
12. Mechanical factors involving the root or ganglion may produce a similar syndrome.  

13. Sensory rhizotomy gives complete relief  

14. Decompression procedure provides temporary relief  

15. Compression procedure gives more lasting relief.  

Although nerves 5, 7, 9, 10, C-1, C-2, and C-3 all feed into the same general neuron pool, fifth nerve neuralgia is about 100 times more common than ninth nerve neuralgia, while seventh and tenth nerve neuralgias are rarities. Paroxysmal neuralgia of the upper cervical roots is non-existent.  

Fig. 59 indicates the density of the several root systems converging in the subnucleus caudalis at C-1 and C-2. It is difficult to conceive how a lesion or pathophysiological factor located in this area could have so specific an affinity for trigeminal interneurons. Also, this lesion would have to involve the second or third division neurons leaving the adjacent first division neurons unaffected. It would have to affect women twice as often as men and the right side more frequently than the left. Other improbable considerations of this type might include the remarkable and variable relief of pain by decompression, or compression of the ganglion and sensory root. Although the sensory input has not been markedly affected, one possible location for a central lesion has ceased to discharge paroxysmally. It is also probably significant that there is no well-documented case of syringomyelia, tumor, or infarction involving the spinal nucleus and associated with the clinical syndrome of trigeminal neuralgia.  

Experimental efforts to produce trigeminal neuralgia in animals have been made, particularly by King and Barnett, who injected alumina gel into the spinal nucleus of the fifth nerve in cats. They produced a syndrome of dysesthesia of the face consisting of overreac-