Structural Changes in the Trigeminal System Following Compression Procedures*

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TRIGEMINAL neuralgia has been recognized as a definite clinical entity since the middle of the eighteenth century. Various authors have given credit to a Frenchman, André,\(^1\) for his early description of this disease, but writings of the Greeks and Romans indicated that they too were well aware of this painful affection of the face. Fothergill,\(^2\) in his analysis of the various types of pain in the face, presented many accurate and careful observations on what we recognize today as trigeminal neuralgia, but wrote: "What therefore I have to offer upon the nature of this disease is rather submitted to your consideration as matter of farther inquiry, than as opinions sufficiently established."

To this day, the etiology in the great majority of instances remains unknown and not all of the answers as to the best form of therapy are at hand. Pioneers of the surgical approach for the control of facial pain in the distribution of the 5th cranial nerve were Horsley (1891), Hartley (1892), Krause (1892), Tiffany (1894), Cushing (1900), Keen, Spiller and Frazier (1901), and numerous others. Many surgical refinements of the early procedures have been offered more recently, but all of these resulted in numbness in the anatomic distribution of the severed posterior sensory root.

Taarnhøj’s\(^8\) paper in 1952 presented a new solution to the problem of pain: merely decompressing the posterior part of the ganglion and the posterior sensory root. This treatment seemed to control the pain and at the same time preserve the sensation in the face. Immediately this simple neurosurgical procedure was applied in many cases of trigeminal neuralgia. However, Svien and Love,\(^9\) reporting from the Mayo Clinic on the results of this operation in 100 patients after more than 4 years, wrote that in cases in which recheck examination revealed that sensation in the face was normal, the recurrence of pain was as high as 84.6 per cent. Interestingly, among cases in which an objective sensory loss could be demonstrated after surgery, pain recurred in only 36.4 per cent. These data gave support to the prediction of Shelden and co-workers\(^4\) that to obtain lasting satisfactory results in trigeminal neuralgia it would be necessary to apply enough trauma to the trigeminal ganglion and root to produce some degree of sensory loss in the face.

In the past 4 or 5 years, our experience with compression of the gasserian ganglion has been so favorable that it has become our procedure of choice for most patients less than 65 years of age suffering from trigeminal neuralgia. Few recurrences of pain have been noted during this time. The majority of patients have been most grateful that the degree of subjective or objective sensory change in the face is so minor in comparison with that which follows subtotal or total section of the posterior sensory root.

This brief study was undertaken in an attempt to determine to what extent the trigeminal system degenerates following compression of the gasserian ganglion as recommended by Shelden and co-workers.\(^5\)

**Materials and Methods**

Five cats weighing between 4 and 8 pounds were used. Under deep pentobarbital (Nembutal) anesthesia, left temporoparietal craniectomy and partial temporal lobectomy were performed in order to expose the floor of the middle fossa and the gasserian ganglion. The compression procedure was carried out as described by Shelden and

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co-workers, and the wound then was closed in layers. One animal died but the other 4 survived in good condition. At intervals of 4, 5, 7, and 9 days, these were anesthetized again and perfused with saline, followed by 10 per cent aqueous formalin.

Subsequently the brain stems were cut into blocks sectioned serially, and stained according to the Nauta-Gygax technic. The corresponding gasserian ganglia were sectioned serially and stained by the hematoxylin and eosin, thionine, Nauta-Gygax and Winkelman silver technics.

Results

Degeneration of the afferent trigeminal fibers occurred in all animals. It varied from very mild degeneration in 1 case to moderate partial degeneration in another, and was of intermediate degree in the remaining 2.

The microscopic changes in the ganglion were remarkably mild. An occasional minute hemorrhage was found, and there was a mild increase in small round cells located between the neurones. The ganglion cells were slightly swollen, the satellite cells being, in general, well preserved.

When the compression had been more vigorous, a decrease in the number of cells was noted; but even here, many cells in the area of compression appeared normal in terms of cytoplasmic and nuclear structure.

Fig. 1 shows sections from a normal and a compressed ganglion for comparison. In the compressed ganglion the glomeruli are well preserved and the axons are moderately swollen and irregular.

The intra-axial course of the trigeminal nerve at all levels was examined, and the degenerative changes found in the main sensory and spinal nuclei were in accord with the findings noted for the ganglia.

Thus the degeneration observed was never extreme, like that following section of the root, but consisted rather of a mild to moderate scattering of degenerated subterminal endings throughout the trigeminal nuclei, amounting to considerably less than 50 per cent of complete degeneration even in the case in which compression had been quite vigorous. The changes could be traced down into the cervical prolongation of the spinal