Pathophysiology of trigeminal neuralgia: new evidence from a trigeminal ganglion intraoperative microneurographic recording

Case report

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The origin of trigeminal neuralgia (TN) appears to be vascular compression of the trigeminal nerve at the root entry zone; however, the physiological mechanism of this disorder remains uncertain. The authors obtained intraoperative microneurographic recordings from trigeminal ganglion neurons in a patient with TN immediately before percutaneous radiofrequency-induced gangliolysis. Their findings are consistent with the idea that the pain of TN is generated, at least in part, by an abnormal discharge within the peripheral nervous system.

**KEY WORDS** • pain • trigeminal neuralgia • trigeminal ganglion • nerve action potential discharge • peripheral nervous system

Trigeminal neuralgia, or tic douloureux, is a severe, episodic, but otherwise chronic, facial pain syndrome.\(^4,5,10\) The pain is unique in its excruciating intensity, lancinating quality, and confinement to the trigeminal nervous system. The genesis of TN remains a mystery. Theories involving either alteration in synaptic processing in the central nervous system\(^3\) or abnormal sensory input from the peripheral nervous system\(^2\) have been invoked, but thus far there has been no direct neurophysiological evidence to support either explanation. To learn more about the neuronal mechanism of TN, we developed a recording technique\(^1\) that makes it possible to monitor the action potential discharge of individual trigeminal ganglion neurons in afflicted patients.

**Case Report**

*Intraoperative Microneurographic Recording.* The experimental procedure was approved by the Oregon Health & Science University Institutional Review Board. A 70-year-old man in whom TN affected the second and third trigeminal division on the right side of his face gave informed consent for an intraoperative recording session, which was undertaken before the percutaneous radiofrequency lesion procedure. After induction of anesthesia, a Tew needle with a stylet (Radionics, Inc., Burlington, MA) was introduced into the right foramen ovale by using the Hartel technique with the aid of a fluoroscopic x-ray instrument. Care was taken not to advance the tip of the needle beyond the orifice of the bony foramen. The stylet was then replaced by a custom-made, 155-mm-long tungsten microelectrode (nominal impedance 1.5 M\(\Omega\)). Neural activity was recorded by a high-input impedance isolation stage of an alternating current amplifier (model P5; Grass-Telefactor, West Warwick, RI), amplified 10,000 times, filtered (bandpass 0.3–10 kHz), and captured in digital form after it had been digitized at a 22-kHz sampling rate by a 16-bit analog–digital converter and streamed to a computer disk by using the GoldWave shareware program (version 4.02; GoldWave, Inc., St. Johns, NF, Canada). The microelectrode was advanced manually through the ganglion in small steps. The sensory search stimuli consisted of a light stroking of the face and mouth with a gloved finger.

**Findings.** In contrast to ordinary responses elsewhere in the face, the repetitive light mechanical stimulation of the patient’s known pain trigger zone, near the corner of his mouth, induced a high-frequency afterdischarge (Fig. 1). The afterdischarge lasted 10s of seconds beyond the initial period of mechanical stimulation and consisted of several bursts of activity. The afterdischarge was followed by a prolonged period of refractoriness, during which stimulation of the trigger zone could not elicit any action potential response.

**Discussion**

We observed a prolonged afterdischarge when light mechanical stimulation was applied to the trigger zone in a patient with TN. This afterdischarge was an unusual feature. No afterdischarge was produced by light mechanical stimulation outside the trigger zone. Previous trigeminal neuron
Trigeminal neuralgia microneurographic recording

![Microneurographic recording](image)

FIG. 1. Trigeminal ganglion neuron afterdischarge following gentle mechanical stimulation (mech.) of the neuralgia trigger zone. Time is given in seconds.

Recordings were made from peripheral branches of the trigeminal nerve in healthy human volunteers, with no evidence of a prolonged afterdischarge. The microneurographic recording obtained in this patient provided for the first time evidence of intrinsic hyperexcitability of trigeminal ganglion neurons in a patient with TN. There was a period of intense activity, followed by a prolonged period during which the neurons were unresponsive to mechanical stimulation of the trigger zone. Overall, the firing pattern had the profile expected of neurons involved in a typical attack of TN. The observed afterdischarge implicates trigeminal ganglion neurons as a source of abnormal activity. Another possibility is that the measured discharge resulted from excessive activation of the trigeminal dorsal root reflex. Regardless of the exact mechanism, the present finding is consistent with the hypothesis that the pain of TN is associated with abnormal discharges of action potentials within the trigeminal ganglion or the nerve itself. To affirm a strict relationship between the intense afterdischarge and a TN attack, it will be necessary in the future to obtain recordings from an awake patient who recovered from the anesthesia and can report the time at which pain occurs.

References


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