Effect of trigeminal tractotomy on dental sensation in humans

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Two patients with intractable pain of the head and neck due to cancer underwent trigeminal tractotomy and rhizotomy of the ninth and 10th cranial nerves, as well as an upper cervical dorsal rhizotomy. Postoperatively, cutaneous analgesia was present ipsilaterally in peripheral portions of the face and the neck, but paramedian facial regions and the oral mucosa were hypalgesic. The procedures had no significant effect on dental pain perception in these patients, as tested electrically and thermally. These results are similar to those in recent reports of dental sensation after trigeminal tractotomy in animals. The results raise questions about the validity of the commonly espoused hypothesis that trigeminal nucleus caudalis is the exclusive locus for processing of orofacial nociceptive information. The findings are consistent with the idea that the entire spinal trigeminal nucleus serves as a site for integration of pain-related information from orofacial structures that are supplied by afferent fibers in the fifth, seventh, ninth, and 10th cranial nerves and the upper cervical nerve roots.

KEY WORDS
- trigeminal nerve
- trigeminal tractotomy
- dental pain
- cervical rhizotomy
- facial sensation

A variety of clinical reports have described changes in facial sensation after trigeminal tractotomy. The procedure is most often described as producing facial analgesia and dermatanalgesia with preservation of tactile sensation. The dental pulp is a unique orofacial structure involved almost exclusively with a nociceptive function. A variety of clinical studies of dental sensation have been reported in normal individuals; however, no study of human dental sensation has apparently been carried out after trigeminal tractotomy. Results of such a study are of interest, since recent work in our laboratory with primates and cats suggests that dental analgesia does not result from trigeminal tractotomy. Such results have been interpreted as showing that the neurophysiological mechanisms involved in dental nociception may differ from those involved in cutaneous facial nociception or that past conceptualizations of the function of the spinal trigeminal nucleus in orofacial and dental nociception have been overly restrictive.

Few patients who have undergone trigeminal tractotomy are available for sensory studies, and many of these have had their dental sensation altered or abolished by malignant disease or radiotherapy. This report describes the results of electrical and thermal testing of dental sensation in two patients who underwent trigeminal tractotomy. In both cases, dental sensation was not significantly affected by tractotomy.

Case Reports

Case 1

This 65-year-old woman was seen because of intractable pain in the left side of the neck, tonsillar fossa, and face, and about the left ear. She had previously undergone two surgical procedures and radiotherapy for treatment of a carcinoma of the left tonsillar fossa. Radiation therapy was completed 18 months before referral. The patient had taken a variety of narcotic analgesics, including morphine, meperidine, and Dilaudid, without satisfactory pain relief. She had also begun to drink alcohol heavily for pain relief. She had no complaints of sensory changes on the face or oral cavity. Her voice had become hoarse over the few months before admission.
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Examination. There was an ulcerated lesion, about 1.5 cm in diameter, in the left tonsillar fossa. There were minimal radiation changes in the skin of the neck and tenderness to palpation anterior to the sternocleidomastoid muscle and overlying the muscle belly. Neurological examination was normal except for the following findings. Hyperalgesia to pinprick was noted in the distribution of the left trigeminal nerve, particularly the maxillary and mandibular divisions. Touch and thermal perception and the corneal reflex were normal. Motor trigeminal function was normal. The gag reflex was decreased on the left. The left vocal cord was paralyzed. Examination of dental sensation in response to both thermal and electrical stimuli was carried out bilaterally on maxillary and mandibular teeth, using methods described by Grossman and Trowbridge. For examination of thermal sensation, ethylchloride was applied to a small cotton pledget and placed against the tooth surface for cold testing. Warm testing utilized a guttapercha probe heated to 112° to 115°. In each case the time period between application of the stimulus and the first sensory perception by the patient (the sensory response time (SRT) of Trowbridge) was recorded. The SRT was less than 2 seconds for all teeth tested and was not significantly different on the two sides. Electrical testing of dental-pulp sensation utilized a monopolar dental-pulp test instrument, which is commonly used in clinical testing of dental-pulp viability. The instrument provided a positive pulse of about 60-μsec duration at a frequency of 500 Hz. Stimulus intensity could be varied in 10 steps from 1 to 13 volts. In this patient, dental pain was perceived consistently at 1 volt when mandibular or maxillary teeth were stimulated bilaterally.

Operation. Suboccipital craniectomy and laminectomy of C1–3 were carried out with the patient in the sitting position. Trigeminal tractotomy was accomplished at a point just rostral to the obex in order to avoid a small crossing artery. Using microsurgical technique, an incision was made to a depth of 3.5 mm, with the emerging spinal accessory rootlets as the ventral limit of the incision and the apparent junction of the tuberculum cinereum and accessory cuneate tubercle as the dorsal limit. The incision was purposely carried slightly into the fasciculus cuneatus in order to section the afferent fibers of the seventh, ninth, and 10th cranial nerves which run in the dorsal portion of the spinal trigeminal tract. Likewise, the incision was carried well ventrally, so as to include a portion of the subjacent spinothalamic tract and insure section of the most ventrally located fibers in the trigeminal tract. The depth of 3.5 mm was selected to section the descending tract, the subjacent spinal nucleus, and the internuclear ascending connections.

* Dental-pulp test instrument (Dentotester) manufactured by Parkill, Farmingdale, New York.

Subsequently, the posterior roots of C-2 and C-3 were sectioned. No C-1 posterior root could be identified despite a careful search. Rhizotomies of the left glossopharyngeal nerve and several vagal nerve rootlets were also carried out.

Postoperative Course. The patient experienced immediate relief of her preoperative pain. By the time of her hospital discharge, she complained only of occasional pain deep in the left ear canal.

Sensory examination immediately postoperatively (Fig. 1) revealed analgesia to pinprick and thermal-analgesia in the cutaneous distribution of the left trigeminal nerve. She was markedly hypalgesic, but not analgesic over the left side of the tongue, or buccal and alveolar mucosa. She was anesthetic on the pinna and tragus of the left ear, but not deep in the ear canal nor on the tympanic membrane. The corneal reflex could be elicited on the left but was reduced, and touch sensation was preserved over the entire trigeminal distribution on the left although the threshold was elevated compared to the right side. Anesthesia was present on the posterior scalp and lateral and anterior aspect of the neck on the left. There was analgesia of the right leg as well. Examination of dental sensation by means of both thermal and elec-
trical stimuli showed no significant changes compared to preoperative results. The patient continued to experience dental pain at the lowest setting (1 volt) of the electrical test instrument when the test was performed on both maxillary and mandibular teeth bilaterally. In addition, the SRT in response to both heat and cold testing remained at 2 seconds or less bilaterally.

Delayed sensory examination 5 weeks and 4 months after tractotomy revealed certain changes. Additionally, the patient noted some pain in the left ear canal, particularly with swallowing. There was also some mild pain above the left eye. Hypalgesia rather than analgesia was now noted on pinprick testing near the midline of the left upper lip, nose, periorbital region, and introraorally. There was dense hypalgesia throughout the remainder of the cutaneous trigeminal distribution on the left. In these zones, a single, double, or occasionally triple pinprick was perceived as touch only. Repeated pricks were occasionally perceived as mildly painful. The left corneal reflex remained decreased, and the patient noted a subjective decrease in sensation on the left cornea. Repeat testing of both thermal and electrical sensation of the dental pulp revealed no significant changes from the immediate postoperative results.

**Case 2**

This 65-year-old man was diagnosed 2 years prior to neurological evaluation as having squamous cell carcinoma of the floor of the mouth. Initial treatment with radiotherapy was followed by an attempted radical neck dissection which was incomplete due to extensive involvement of the internal carotid artery with tumor, from the skull base to well below the carotid bifurcation. Four months before evaluation, the patient had onset of left facial pain primarily in the distribution of the mandibular division of the trigeminal nerve; he also noted pain at the lateral aspect of the neck and in the region of the left ear canal. Narcotics provided only partial pain relief. On the day of admission, the patient suffered an attack of sharp knife-like pain deep in the left ear canal, tonsillar fossa, and the mandibular fossa on the left. This was associated with syncope. During hospitalization, several further attacks of severe pain ensued, associated with documented bradycardia (30 beats/min) and severe hypotension (40/0 mm Hg).

**Examination.** The patient was cachectic. The left side of the neck was immovable, with a stony-hard nodular mass. A loud left carotid bruit was audible. Neurological examination disclosed a normal mental state. A left Horner’s syndrome, mild left facial weakness, and atrophy of the left trapezius muscle and the left side of the tongue were present. Sensory examination (Fig. 2 left) revealed hypalgesia to pinprick testing on the left side of the neck, supraclavicular region, and posterior scalp. Tactile sensation was reduced in the same region. The remainder of the neurological examination was normal. Preoperative testing of dental sensation was not carried out for fear of triggering an attack of bradycardia, hypotension, or asystole.

**Operation.** A left suboccipital craniectomy and laminectomy of C1–3 were performed. Trigeminal tractotomy was carried out on the left, at the level of the obex, using a technique similar to that in Case 1. Rhizotomies of the glossopharyngeal root, the upper four vagal rootlets, and dorsal roots C1–4 were also accomplished. Rhizotomy of the nervus intermedius was also planned, but a definite intermediate fascicle could not be identified.

**Postoperative Course.** Upon awakening from anesthesia, the patient was free of the pain he had experienced preoperatively. No further attacks of bradycardia or hypotension occurred. Immediately postoperatively, analgesia to pinprick included the entire left side of the face to the midline (Fig. 2 center). Marked hypalgesia, but not analgesia, was present over the left buccal and alveolar mucosa and tongue. A portion of the left auditory canal and the tympanic membrane were also hypalgesic. Anesthesia was present over the posterior scalp and neck and shoulder on the left side. The left corneal reflex was markedly decreased. Tactile sensation was preserved over the face on the left, but the threshold was elevated and two-point discrimination was decreased. The right leg and right side of the trunk were analgesic below the T-12 dermatome. Dental sensation could not be assessed immediately postoperatively because respiratory complications required prolonged endotracheal intubation, tracheostomy, and respiratory support, and resulted in an altered state of consciousness for a few weeks.

Four weeks postoperatively (Fig. 2 right), the central facial regions showed marked hypalgesia but were no longer completely analgesic to pinprick, particularly multiple pricks. Introral hypalgesia was reduced compared to that in the immediate postoperative period. Electrical testing of dental sensation revealed no significant difference in pain thresholds for either maxillary or mandibular teeth on the same side as the tractotomy compared to that on the opposite side. In this patient, dental pain was perceived consistently at 4 volts on the dental test instrument. Likewise, no significant differences were noted in dental sensitivity to hot and cold stimulation on the two sides. The SRT was 2 seconds or less bilaterally for both hot and cold stimuli.

**Discussion**

The brain-stem trigeminal complex involved in somatosensory information processing from the face and oral cavity is subdivided cytoarchitectonically into the rostrally located nucleus principalis (main sensory nucleus) and the more caudally located spinal
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The changes in pinprick, thermal, and tactile sensation seen in the patients described in this paper are similar to those reported by others after trigeminal tractotomy and upper cervical rhizotomy. A dense analgesia to pinprick in the peripheral portions of all three trigeminal divisions was noted. Intraorally and near the midline, hypalgesia, rather than analgesia, was present. These findings are in agreement with past concepts that nucleus caudalis is the major component of the trigeminal brain-stem complex involved in the processing of orofacial pain information.

The difficulty in producing satisfactory analgesia intraorally and in the midline of the face with trigeminal tractotomy is well known. The failure to produce such analgesia is often ascribed to incomplete tract section, although sparing of analgesia in these regions has been described in humans and monkeys with pathologically proven complete tract and nuclear destruction at the level of the obex. White and Sweet described a patient with pathologically documented complete tract degeneration at the level of the obex after tractotomy. In spite of this, the patient exhibited sparing of analgesia of the mucosa in the region supplied by the maxillary and mandibular trigeminal divisions and also sparing of cutaneous analgesia in the mandibular division.

The results of the dental sensory examinations reported here are unique. They reveal that trigeminal tractotomy does not produce dental analgesia, even when combined with upper cervical rhizotomy. These findings are similar to those in reports of dental pain sensation in cats and monkeys after tractotomy. The use of natural thermal stimulation of the teeth in the present report supplements past animal studies that have relied exclusively on electrical stimulation. The animal studies are also limited because they rely on the assumption that certain behavioral avoidance responses, elicited by electrical stimulation of dental pulp, are provoked by the sensation of pain. In these two patients, who were able to describe their pain sensibility, neither the pain threshold for electrical stimulation nor the sensory response latencies to ther-

Fig. 2. Case 2. Left: Preoperative sensory examination findings. Center: Sensory changes 1 week after trigeminal tractotomy and rhizotomy of the ninth and 10th cranial nerves and cervical dorsal roots C1–4 on the left side. Right: Sensory changes 4 weeks after surgery.
nal stimulation were significantly affected by tractotomy and rhizotomy.

The effect of trigeminal tractotomy on cutaneous pain sensation had been interpreted to indicate that the nucleus caudalis was the exclusive locus within the brain-stem trigeminal nuclear complex for the processing of orofacial nociceptive information.\textsuperscript{1,10,20} Although the rostral end of the nucleus caudalis usually terminates at the level of the obex, the latter is only an approximate gross anatomical landmark of the subdivision between the nucleus caudalis and the rostrally located nucleus interpolaris. Consequently, the preservation of dental sensation in the patients reported here could be due to processing of dental nociceptive information by portions of the nucleus caudalis rostral to the point of tractotomy. The lack of neuropathological confirmation of lesion location in these patients leaves this question open to speculation. In our animal experiments, pathological confirmation was available of lesion location as high as 3.1 mm above the obex, and well above the rostral end of the nucleus caudalis in several animals; however, dental pain sensation was unaltered.\textsuperscript{30} At least in nonhuman primates, therefore, it seems clear that dental pain sensation may be processed in the absence of trigeminal input into the nucleus caudalis.

Physiological studies have shown that second-order trigeminal neurons responsive to dental-pulp stimulation are located widely in the trigeminal brain-stem complex, from the rostral main sensory nucleus in the midpons, through the nuclei oralis and interpolaris,\textsuperscript{6,12,17,18} and at least a few millimeters below the obex into the nucleus caudalis.\textsuperscript{6,30,32} Interestingly, although the nucleus caudalis extends from about the obex level caudally to the C-2 or C-3 spinal segments, dental nociceptive neurons are mainly located in a very narrow zone within a few millimeters of the obex. In fact, until our demonstration of dental nociceptors in the nucleus caudalis, their presence in that locus was denied.\textsuperscript{8} The cases presented here, in conjunction with the previously reported animal studies, suggest that dental nociceptors located rostral to the nucleus caudalis are capable of processing nociceptive information from the dental pulp and producing the sensation of dental pain. This occurs in the absence of afferent input to dental pulp neurons in the nucleus caudalis and in the absence of any modulating influence of the nucleus caudalis on more rostral portions of the spinal trigeminal complex.\textsuperscript{12,33}

These findings are consistent with our recent physiological studies,\textsuperscript{16} which show little change in the firing patterns of rostrally located pulpal neurons after tractotomy.

These clinical and laboratory observations invite a number of interpretations. First, dental pain mechanisms may represent one aspect of this specialization. It is well to point out, however, that cutaneous nociceptive neurons, although considerably less numerous than dental nociceptive neurons, have also been identified rostral to the nucleus caudalis,\textsuperscript{3} making the apparent dental specialization less convincing. Second, the failure of tractotomy to alter dental pain sensation may imply that past concepts of trigeminal nociceptive mechanisms have been overly restrictive. Denny-Brown and Yanagisawa\textsuperscript{1,2} suggested that the entire spinal trigeminal nucleus is involved in orofacial nociception. The ability of certain pharmacological agents, such as strychnine and L-dopa, to reverse the facial analgesia resulting from tractotomy and rhizotomy in both man\textsuperscript{10} and nonhuman primates\textsuperscript{1,5,34,35} suggests that facial nociception may depend on spatial and temporal summation via multiple neuronal circuits that converge in the spinal trigeminal complex, rather than on an exclusive nociceptive pathway relayed via the nucleus caudalis.

The effect of upper cervical rhizotomy and rhizotomy of the ninth and 10th cranial nerves on dental versus cutaneous sensation is also of interest. Apparent overlapping of cutaneous facial innervation via the seventh, ninth, and 10th cranial nerves, as well as the upper cervical roots, in addition to the trigeminal nerve, may be one explanation for residual cutaneous sensation or failure of pain relief after trigeminal rhizotomy or tractotomy alone. Thus, when rhizotomy of the seventh, ninth, and 10th cranial nerves and of the C1–4 nerve roots is added to trigeminal rhizotomy, a dense cutaneous facial analgesia results, which is irreversible by pharmacological agents.\textsuperscript{1,2} The present cases indicate that, contrary to the situation with cutaneous facial pain sensation, overlapping afferent supply to the teeth and oral mucosa via other nontrigeminal nerve roots is of minimal significance to pain perception from these structures. Thus, in the patients described here, dental pain perception was unaffected even with the addition of rhizotomy of the ninth and 10th cranial nerves and upper cervical roots (C1–4) to tractotomy. The situation for the oral mucosa and the midline region of the face may represent an intermediate position between that of the teeth and of the majority of facial skin. That is, the midline and intraoral structures are rendered very hypalgesic but not analgesic by the combination of tractotomy and rhizotomy of lower cranial nerves and upper cervical nerve roots. This suggests that nociceptive information from these zones is processed primarily, but not exclusively, caudal to the obex. The failure to section the seventh cranial nerve (nervus intermedius) in the patients described prevents a categorical statement that no overlapping afferent supply to the dental pulp exists. It appears unlikely, however, that this small afferent pathway, with its zone of supply usually thought to be confined to the ear and ear canal, could account for normal dental sensory function after tractotomy. Thus, there is support for the idea that dental,
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and perhaps intraoral, nociceptive pathways may differ somewhat from those for cutaneous facial nociception, the former being represented more rostrally and the latter more caudally in the spinal trigeminal nucleus. The difference is a quantitative rather than a qualitative one, however.

It seems clear that the concept of orofacial nociceptive mechanisms as being exclusively dependent on trigeminal relays via the nucleus caudalis, with little or no dependence on other portions of the spinal trigeminal nuclear complex, is inadequate to explain the results of trigeminal tractotomy. Orofacial nociception appears to depend upon afferent supply via the fifth, seventh, ninth, and 10th cranial nerves and cervical roots C1–4, and upon central processing throughout the spinal trigeminal nucleus.

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