Controlled thermocoagulation of trigeminal ganglion and rootlets for differential destruction of pain fibers

Part 1: Trigeminal neuralgia

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The authors report their experience in the treatment of trigeminal neuralgia with controlled increments of radiofrequency heating from an electrode placed in the Gasserian ganglion or its posterior rootlets. Touch is preserved in some or all of a trigeminal zone rendered analgesic. The electrode tip is introduced through the foramen ovale and placed among the desired rootlets with the help of a combination of radiographs and the conscious patient's response to electrical stimulation with a square wave signal and gentle electrical heating. The degree of heat is measured by a thermister at the electrode tip. The patient's cooperation is maintained by the use of the neurolept anesthetic Innovar and the production of brief unconsciousness for the painful parts of the operation by methohexital (Brevital). Of 274 patients with facial pain so treated, 214 had trigeminal neuralgia; 91% of the latter group experienced relief of pain and 125 followed for 2 to 6 years had a recurrence rate of 22%. In a total of 353 procedures, there has been no mortality and no neurological morbidity outside the trigeminal nerve. Only six of the patients with trigeminal neuralgia have complained significantly of postoperative paresthesias. The most serious undesired result has been the production of an anesthetic cornea in 28 patients, one of whom lost the sight of one eye due to corneal scarring. Correlating findings in our patients with those in studies by other authors, we conclude that the preservation of some touch is due to resistance to heating by the heavily myelinated A-beta fibers.

KEY WORDS • trigeminal neuralgia • differential thermal rhizotomy • cutaneous sensation • nerve fiber size

IN North America various forms of open operation have been the preferred definitive treatment for patients with trigeminal neuralgia refractory to medical management. However, in Europe the feasibility, relative safety, and effectiveness of a percutaneous approach to the Gasserian ganglion and its rootlets have been convincingly demonstrated for over two-thirds of a century. The foramen ovale may be approached laterally via the sigmoid notch of the mandibular ramus as described.
by Harris or anteriorly as recommended by Taptas and by Här tel. Many thousands of patients have since then had sharp electrodes or needles inserted into the intracranial cavity with virtually no complications related to injury by the sharp tip to important vessels or neural structures. The complications have been due largely to the uncontrollable spread of injected chemicals such as alcohol or phenol or to the injury of major vessels by electrocoagulation.

Alternative Methods

Injection of Chemicals

By 1940, Harris alone had treated 2500 cases of idiopathic trigeminal neuralgia using ganglionic injections of absolute alcohol, convincing evidence that his methods were satisfactory. The details of various related chemical techniques, developed in several countries, have been summarized by White and Sweet, and need not be repeated here. A description of the complications is relevant to this paper, however. Alcohol injections into the Gasserian ganglion have been followed by an inordinate percentage of persistent anesthesia in the first division, including the cornea. This occurred when the operator's usual immediate objective was permanent anesthesia of one entire side of the face, as, for instance, Harris, and Thurel, who had treated over 3000 cases, and Henderson, in 196 such injections. Henderson's careful late follow-up revealed that only 33% of 86 patients maintained their original total trigeminal anesthesia. An astonishing pattern of returned sensation occurred in 37%, most of these involving the third division, while the first and second divisions remained anesthetic; sensation returned only in the first two divisions in 7%, while 23% had a return in all three divisions. Henderson sought the explanation for this curious recovery pattern by performing 100 postmortem dissections and injections. He came up with a convincing explanation for the frequent persistence of first and second division anesthesia. He observed a previously undescribed sinus passing from the cavernous sinus, deep to the origin of the first and second divisions from the ganglion and partially engulfing these two divisions. This anatomical peculiarity was clearly depicted by his injections of cresyl violet in absolute alcohol. Henderson's publication of this unfavorable anatomical hurdle discouraged us from continuing our efforts with less destructive chemicals such as solutions of phenol either in water or glycerine. The same complication occurred in Penman's patients even though he was seeking to produce a complete loss of touch and pain sensation only in the trigger zones; although he relieved the pain in 97 of his first 100 injections, 82 of the patients had corneal anesthesia and 15 a keratitis requiring tarsorrhaphy.

Electrocoagulative Methods

The likelihood that heat might spread in a more controllable fashion had already been investigated thoroughly beginning with Kirschner in 1931. He used a diathermy apparatus delivering 350 mA of current to a needle uninsulated over its terminal centimeter and placed in the Gasserian ganglion with the aid of radiographic control and a special apparatus to hold the needle. By 1942 he had treated 1113 cases by this method. Its speed and simplicity led to widespread use in Europe in such casual fashion that many sequelae began to occur. Tönis and Kreisell saw so many complications after Gasserian electrocoagulation carried out in other clinics that they advised against it; their patients included eight with corneal ulcers, two with blind or enucleated eyes, two with palsies of the sixth and four of the seventh cranial nerves, six with severe partial or total deafness, and one with paralysis of the twelfth nerve. White and Sweet summarized other reported complications; arterial injury has caused five of the recorded deaths; cardiac arrest under anesthesia, two; meningitis, four; and pulmonary embolism, one. Carotid thrombosis, hemiplegia, and death was the sequence in four of the arterial injuries; rupture of the coagulated wall of the internal carotid artery occurred in the fifth case. In addition, Hensell, Zenker, and Adler have each had one patient in whom stupor, hemiparesis, and/or aphasia occurred, but who recovered completely in some weeks. We mention these problems to justify the tedious but
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successful steps we have taken to eliminate the occurrence of any deficit outside the trigeminal nerve in any patient in our series.

Using the relatively crude surgical diathermy apparatus for electrocoagulation, Thiry had been able by 1962 to achieve a record of no mortality in 225 patients, an oculomotor paresis for 2 weeks to 6 months in only 2% and only one serious sequel. He passed less current into the electrode than his predecessors producing either no objective sensory loss or only a hypesthesia. The eventual complete relief in all his cases "required early recoagulation in 10% or a later recoagulation at several months to 3 years in 17%.”

Schiurman, et al., have reported 183 patients with trigeminal neuralgia treated between 1963 and 1970 by "controlled, partial coagulation under neurolept-analgesia.” These surgeons check the sensory deficit in their patients following each of the necessary number of short periods of electrocoagulation, varying the position of the electrode until the desired effect is obtained. A recurrence rate of 24% was reduced to 7% by repeat procedures yielding a final success rate of 93% with no mortality and a low rate of complications.

In general, modest injury to the trigeminal ganglion and rootlets has been shown to yield remarkably protracted relief from trigeminal neuralgia. This injury can be mechanical as in the compression operation of Sheldon, et al., thermal as in Jaeger's boiling water injections, electrothermal, alcoholic or phenolic. However, there is always the risk of paroxysmal pain if either the trigger zone or the area of initial reference of pain has not become analgesic.

Materials and Method

We have based our study on the hypothesis that some of the less myelinated small fibers evoke pain and might be selectively destroyed by heat transmitted through an electrode. Thus we have developed a technique to produce differential destruction of pain fibers. The availability of the following technical and pharmaceutical aids was important: 1) radiofrequency generators yielding a precisely controllable heat source; 2) tiny thermisters monitoring the degree of heat developed; 3) neurolept anaesthetics such as Innovar* to convert a tense anxious patient into one more casual and relaxed; and 4) the ultra-short-acting agent methohexital (Brevital) to produce brief unconsciousness. Since October, 1965, we have used heat induced by a radiofrequency current of about 2 megacycles as a consistent, controllable way to achieve a graded injury to the trigeminal ganglion or rootlets. We have done 353 such procedures on 274 patients, 214 of whom had trigeminal neuralgia.

Introduction of the Electrode

This procedure is carried out in the radiographic room after the intravenous injection of 2 cc of Innovar. A hollow 20-gauge cannula-electrode† (outside diameter 1.0 mm, inside diameter 0.6 mm) with a short-bevel and sharp tip is used to make the lesion. If analgesia in one trigeminal division is the objective, one of us uses an electrode which has its terminal 5 mm bare of insulation; 10 mm are bare if two or three divisions are involved. The other surgeon uses only the 5-mm electrode, replacing it if necessary.

The anterior approach to the foramen ovale is used (Härtel). We usually penetrate the skin 2.5 cm lateral to the labial commissure if the third division is the target, and 3 cm if the second or first divisions are included. The lateral entrance permits the point to pass more medially as it moves posteriorly in the middle cranial fossa with a better chance of avoiding the third division to enter second and/or first divisions. We have abandoned the use of holders for the electrode, having tried that devised by Kirschner as well as our own stereotaxic head frame; a free hand search for the foramen ovale is adequate and simpler. The operator places the index finger of one gloved hand in the patient’s mouth, feeling the electrode as he advances it just

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*Innovar is a mixture containing fentanyl .05 mg and droperidol 2.5 mg/cc.
†The electrode sets including thermisters, are available commercially from Eastman-Toth Associates, 333 Lee Street, Brookline, Massachusetts 02146.
medial to the mandibular ramus; thus he can be sure the electrode does not penetrate oral mucosa and carry potential infection toward the base of the skull. The electrode is directed in the coronal plane toward the lateral border of the lacrimal caruncle, more medially than has usually been recommended. Our in vivo experience accords with that of Henderson in his 40 cadaver dissections. When he pointed the needle more laterally toward the pupil it emerged from the lateral part or in front of the ganglion in 28 specimens and entered the sensory rootlet zone in only 12. We direct the electrode in the sagittal plane toward a point 3.5 to 4 cm in front of the anterior wall of the external auditory canal at the level of the lower border of the zygoma. This latter direction is likely to bring it against the floor of the middle fossa anterior to the foramen ovale. If a few minor changes in the electrode course do not bring it into the painful zone of decreased resistance indicative of the foramen ovale, a submento-vertical radiograph is taken to aid the placement and minimize the hazards of blind exploration. In general we agree with Jefferson that a needle can be placed in Meckel's cave with surprisingly little pain; but we promptly add Brevital-induced slumber for those who are unhappy at this stage. We prefer to have the electrode pass through the middle or medial third of the foramen as illustrated in Fig. 1. A painstaking attempt to secure this degree of accuracy at this stage is worth the extra trouble, since the electrode tip will then almost certainly remain within the ganglion or rootlets as it enters the intracranial cavity. With a more posterolateral placement within the foramen the electrode tends to lie outside the dural sheath of the ganglion as seen in cadavers by Henderson and in patients by Jefferson and ourselves. It may not move medially enough as it goes deeper to enter third division rootlets, much less those of the second or first division.

If the electrode is originally directed too far posteriorly it may pierce the internal carotid below its entry into the petrous canal; we experienced this complication on three occasions early in the series and stopped the procedure in each case. There were no sequelae. Other operators, after puncturing the artery, have nevertheless carried out the procedure planned. We have also demonstrated in the cadaver that it is possible to transfix this artery and go on into the intracranial cavity too far laterally when there is osseous deficiency in the petrous bone surrounding the internal carotid artery.
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arterial puncture, twice (in the same patient) because a massive hematoma developed superficially in the cheek, and six times because we were unable to pass the electrode through the foramen. Others have used fluoroscopic viewing of the base of the skull on a television screen to help find the foramen. In particular Whisler\textsuperscript{20} has demonstrated that, when the head is rotated, the foramen ovale can be seen fluoroscopically and aiming with fluoroscopic guidance is feasible. In two patients we succeeded in finding the proper portions of the trigeminal structures intracranially even though the foramen ovale was not identifiable, either because of Paget's disease or neoplastic destruction.

Figure 2 illustrates such a bone defect in a patient in whom the electrode, however, penetrated the true foramen ovale rather than the longer spurious oval foramen which marks the site of the internal carotid artery in the intrapetrous canal. Twice the electrode entered the medial part of the middle cranial fossa too far forward. In the patient shown in Fig. 3, the electrode passed to the appropriate depth painlessly and easily. We were so confident of the correct position in this patient whom we were doing a third time for his second recurrence that we did not take a radiograph but proceeded to stimulate with a 60-cycle square wave biphasic pulse. At 4.5 volts a brief generalized seizure occurred starting with unresponsiveness and a blank stare ahead. Faintly pink cerebrospinal fluid emerged from the cannula-electrode when the stilet was removed and the film showed the tip lying medial to the uncus. This type of experience has taught us to confirm the position radiographically at frequent intervals.

In addition to our 353 procedures we stopped short of penetrating the foramen ovale in 11 instances, three times because of

\textbf{Final Placement of Electrode Tip}

\textit{Positioning the Tip.} We originally used electrodes with 9 cm shafts; however, particularly for dolichocephalic skulls, 10 cm is a safer length since one can easily place a small copper clip on it just as it emerges from the unindented skin, preventing excess length from penetrating deeper. Before we used this clip the electrode occasionally slipped in a few extra millimeters in the flabby tissues of an old person so that suddenly the next lesions produced an unwanted loss of corneal sensation. Jaw or cheek movements also sometimes eased the electrode out of an ideal placement. The electrode often enters the foramen ovale at a depth of around 7 cm from the anterior skin.

The lengths of the various trigeminal structures deep to the foramen ovale are remarkably variable. Henderson\textsuperscript{8} 's measurements are the most complete. He found that the intracranial portion of the mandibular division from foramen ovale to ganglion was 0 to 10 mm long; the length of sensory rootlets from the posterior edge of ganglion to porus trigemini varied inversely with that distance being from 15 to 5 mm; the ganglion between these two was usually 5 mm long. The distance between the anterior edge of the foramen ovale and the posterior superior edge of the petrous bone at the porus trigemini varied from 16 to 26 mm. These observations plus our own experience have convinced us that radiography in two planes, plus responses both to electrical
stimulation and to electrical heating are needed if one is to obtain the best control of the site of the lesion.

Radiographic Aids. In submento-vertical radiographs we take care to have the film parallel to the Frankfurt plane joining the middle of the ear canal with the lower rim of orbit and to have the incident x-ray beam at 90° to this. In Fig. 4 left we have plotted the locus of the electrode for 87 of our first group of lesions in relation to the reference point, the posteromedial point of the foramen ovale. Only in a very general way do the lesions from lateral to medial produce third through second to first division loss. In this first series of plots analgesia in the first division occurred in only two patients whose electrode tip was less than 10 mm medial to the reference point. However, in the second series of 94 additional cases (Fig. 4 right) there were seven procedures in which first division analgesia ensued with the electrode tip less than 10 mm medial to the reference point.

A true lateral view is also required. We have made measurements in this projection locating the electrode tip anterior or posterior to the profile of the clivus in the plane parallel to the hard palate and below (rarely above) the sellar floor. Figure 5 left plots these data for the first 89 lesions. There were only three patients in this group with first division analgesia in whom the electrode tips were further forward than 3 mm behind the clivus. But in the third series of 98 more cases (Fig. 5 right), there were eight with first division analgesia produced when the electrode tip was further forward than 3 mm behind the clivus, and the same number when the electrode tip was behind that point. The most decisive measurement in the creation of first division analgesia is that in the sagittal view for the distance of the tip medial to the foramen ovale. The more medial and the further behind the
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Fig. 5. First (left) and second (right) series of plots of sensory losses correlated with positions of electrode tip related to profile of clivus and sellar floor as measured directly from lateral radiographs.

clival profile the tip, the more likely is first division sensory loss; it follows that heating must be more gradual and testing more critical if corneal anesthesia is to be avoided. Figure 6 left illustrates an electrode tip at a point 5 mm behind the clivus and Fig. 6 right its position 8 mm medial to the posteromedial point of the foramen ovale; in this patient we produced an unintentional anesthesia throughout the trigeminal distribution in skin and mucosa, by heating the 1 cm bare electrode to 70°C for 2 rain. It is obvious from Figs. 4 and 5 that the site of the lesion simply cannot be predicted accurately from radiographs alone.

Deeper insertion carries the electrode posteromedially into second and first division rootlets; withdrawal brings it more laterally into the third division. It has not been necessary to repuncture the centrally-entered foramen ovale.

Electrical Aids to Precise Localization. We have been able to predict the lesion site much better by using the results of electrical stimulation. We have used a 60-cycle square wave pulse of 1ms duration. At threshold the sensation is not painful and can hence be located and described calmly by the patient. It is felt as a touch, humming, buzzing, vibrating, pressing, tingling or sense of something moving on or under the skin. The threshold in the majority was at less than 0.4 volts for the above parameters, usually 0.2 volts. In 12 patients it was at 1.5 volts; in 12 it was from 2 to 7 volts. These higher threshold voltages did not presage difficulty in securing analgesia; in only one of the 24 patients (whose threshold was at 1.5 volts) was an inadequate lesion achieved. As an analgesic lesion in the pain fibers is developed by heating, the threshold rises only modestly for the sensation provoked in the largest fibers.

In 262 procedures the locus of the
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Fig. 6. Lateral (left) and submento-vertical (right) radiographs of an electrode position which yielded unwanted first division anesthesia.

paresthesias was an accurate guide to the division which became analgesic; however, in 40 procedures these sensations were referred to a different division from that which became analgesic upon heating at the same site. The sensations provoked by the 5 mm electrode were just as subject to this inaccuracy of sensory reference as were those from the 10 mm electrode. Hence we have added the response to gradual heating of the rootlets with immediate shutoff of the current the moment the patient reports sensation. Before any lesion is made this appears at around 50°C; it is usually a sharply localized burning pain and is usually referred to the same division, but when it is not, the ensuing lesion at higher temperature may be in either of the two divisions. Hence it is advisable to withdraw the electrode a little if either mode of stimulation causes unwanted sensation in the first division.

General Anesthesia

Each of the earlier lesions in any patient was preceded by an intravenous dose of Brevital usually in the range of 20 to 50 mg. We have used an average of 180 mg of Brevital for the total procedure. However one large man, not an alcoholic, required up to 150 mg per dose for a total of 560 mg for the four necessary anesthetic episodes. This patient had not received Innovar. Another man weighing 250 lb was given a total of 900 mg of Brevital and 2 cc of Innovar for his 15 anesthetic episodes. For periods of heating after the first few, lower doses often suffice. Usually the patient is not aware he has been unconscious and may berate us for breaking our promise to put him to sleep. It has been unnecessary to have an anesthesiologist present during any phase of the operation except in the presence of hazardous associated disease. A skilled radiographic technician and the neurosurgeon are usually the only personnel required.

Making the Lesion

Temperature of the Tip. Before the electrode is heated the bevel-ended stillet is withdrawn and replaced by tiny wires carrying a thermister at their tips. In an effort to destroy differentially the smaller fibers presumably related to pain while sparing heavily myelinated fibers for touch we have made our initial lesions only about 10°C hotter than the temperature level.
causing a burning sensation in the conscious patient. The first lesions have been made at from 47° to 65° C for 1 minute; we then gradually increase the degree of heating by 5° to 10° C increments depending on the sensory findings after each lesion. The number of lesions required has averaged 4.3 per procedure ranging from one to 15. The necessary final electrode temperatures varied through an amazing range from 47° to 108° C. In addition to recording the thermister temperatures we also note the accompanying readings of an RF milliammeter and RF voltmeter so that if the thermister should burn out, and another were not available, we could complete the procedure with a modicum of control. If sudden increases in current or voltage are required to raise or sustain the thermister record of temperature, the instruments may have become faulty or the electrode tip may have developed an insulating coagulum. This should be checked with another thermister. In general the temperature gradually rises if the current and voltage are held constant, so that one would do well to drop to 30-sec lesions if one had to rely only on current and voltage measurements. Atkinson1 who is using this general procedure tells us that he uses no Brevital, relying on very slow heating to provide adequate analgesia as it destroys.

In general any changes in sensation we have seen in the hours and first days after the procedure are in the direction of returning sensation. However, we have seen minor increases in the sensory loss a few times and Tew21 has described one real annoyance, a patient in whom simple analgesia at the end of the procedure later changed to extensive anesthesia including the cornea. Our biggest disappointments have been in patients in whom successive heatings were developing a gradually increasing zone of pure analgesia in the second and possibly a little loss in the first division; however, the very next increment of heat, perhaps only 5° C hotter, would abruptly yield complete anesthesia and analgesia throughout both of these areas. Recently we have found that when the sensory loss we wish is near to realization, it is not necessary to have the patient unconscious during the final heating episodes. We learned this from a patient who described no sensation at all upon test heating when awake at a stage well above the level for the last lesion. After a minute of such heating our retesting disclosed to our chagrin and surprise that she not only had the new desired analgesia but total anesthesia as well. Since then in the last phases of the procedure we have used very slow increases in heating plus, when necessary, doses of Brevital so low as to permit us to test sensation as the lesion is evolving. This tactic has disclosed that much less than 1 minute at a temperature a few degrees higher may yield the required increment in the lesion. We hope that this relatively minor change in the technique will reduce our incidence of corneal anesthesia.

**Autonomic Manifestations.** Flushing of an area of the face while the lesion is being made intimates extension of the destruction of pain fibers to that trigeminal division. If it occurs in forehead or eyelids and we are not seeking first division analgesia we shut off the heat at once. Ipsilateral lacrimation may occur during the lesion-making; it does not indicate spread to the first division sensory fibers. Perhaps it is due to irritation of the nearby greater superficial petrosal nerve.

**Cerebrospinal Fluid.** We prefer to have the tip of the cannula-electrode in cerebrospinal fluid (CSF), since this means it must lie behind the ganglion, and most of the lesion will be in rootlets and/or ganglion. The CSF is rarely more than faintly pink with blood and is usually clear and colorless. It was obtained in 207 out of 308 procedures for which a specific note as to its presence or absence was made. In Figs. 4 and 5, sites from which no fluid came from the properly-placed electrode are indicated by a small check; these points are randomly distributed among the majority which did yield CSF as recorded in both the lateral and sagittal projections. Curiously enough CSF was usually obtained from the most aberrant sites shown on the four graphs. There was no difference in the ease of securing analgesia related to the presence or absence of CSF in the procedure.

**Results**

**Immediate Results**

**Prompt Success and Failure.** Of the first
154 patients with trigeminal neuralgia 140 or 91% were relieved by the first procedure. The average patient was discharged the day after operation. In 25 instances (7%) the sensory loss achieved at the end of the procedure proved inadequate or receded within days, the pain recurred and a second procedure was soon needed. In 16 the same type of operation was repeated with greater success. In another five with typical trigeminal neuralgia (four of these before we used Innovar), the patient preferred an open trigeminal operation for the second attempt. In four with other forms of facial pain other operations were done.

Weakness of Masticator Muscles. Following 223 procedures a specific note was made about masseter contraction and ability to deviate the jaw to the opposite side. The muscle strength seemed normal in 103, reduced in 70, and absent in 49. Because the potential for motor recovery is great we have given special attention to conserving this function only in patients with bilateral trigeminal neuralgia. In such patients we have easily succeeded in sparing the motor root. At times stimulation at threshold provokes masticator contraction, a fact which of course dictates a more lateral placement of the electrode to yield a pure sensation.

Complications. One of the most gratifying features of the results is that no procedure has provoked in any patient any neurological deficit outside the domain of the ipsilateral trigeminal nerve. There has been no mortality.

Extra-trigeminal morbidity has been minimal, as represented by six cases. One patient had an aseptic meningeal reaction with fever to 37.6°C, severe headache and nuchal rigidity for 12 hours, a CSF red blood cell count of 35 and white blood cell count of 712 cells/mm³ with 78% granulocytes and 22% lymphocytes. No organisms grew on culture. She rapidly lost her signs and was discharged on the second postoperative day.

Two patients developed acute systemic episodes during the procedure which was promptly stopped and a brisk recovery ensued in both. One felt vaguely but acutely ill, as though he might die, and developed dysarthria and cyanosis of the nail beds. Although pulmonary function studies then showed severe obstruction and restrictive defects we were able a month later to reoperate and secure the second division trigeminal analgesia needed to control his pain. The second patient, on nitroglycerin for recognized angina, developed a typical attack of chest pain before the trigeminal lesion had been made as large as planned. It is worth noting that had such an episode occurred during general anesthesia for open trigeminal rhizotomy, the sequelae might have been serious.

In two patients pure blood emerged from the otherwise properly-placed intracranial tip of the cannula-electrode. In one the bleeding was venous; the procedure was terminated but the desired heat lesion was made a few days later. In the other patient bleeding was arterial; the procedure was terminated and we noted a new positive Babinski that lasted 1 hour. A lumbar tap yielded clear colorless CSF. No new symptoms developed, but we felt it best to carry out an open posterior fossa approach.

In one patient with preexisting right hemiplegia and severe aphasia, dysphagia appeared after a left second and third division trigeminal lesion. The patient’s wife noted some days after our operation that the problem appeared to be one of manipulating and swallowing the food in his mouth. There was no swelling in the mouth or oral pharynx and good movements of the tongue and pharynx; the disorder was described as an apraxia. Four months later the patient could still swallow only finely chopped food. We are not aware that this problem has been previously described.

Unilateral blindness from the scar following a corneal ulcer occurred in one patient in whom treatment had produced an anesthetic cornea.

Painful anesthesia or analgesia are discussed with the topic of preservation of touch.

Preservation of Some Touch Sensation. In order to minimize the unpleasant sensations which accompany total anesthesia of the skin we sought to stop short of this in all but the patients with pain due to cancer. The relatively gratifying numbers in which we succeeded are as follows. Touch was preserved throughout the analgesic skin in
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187 patients, and in part of the analgesic skin in 62. We failed to make a differential lesion in 25; in these anesthesia and analgesia coincided. In two patients, one with a periodic migrainous neuralgia and one with cancer pain, complete trigeminal anesthesia and analgesia have been followed by severe anesthesia dolorosa. Only six patients with typical trigeminal neuralgia have complained significantly of postoperative paresthesias. In one, touch and only a tiny bit of pin prick sensation were preserved in the upper gum, hard palate and tongue, to which an incessant "burning, cramping" pain was referred. This appears to be a severe "hypalgesia or analgesia dolorosa;" it cannot be properly described as "anesthesia dolorosa." Another has constant "soreness" in the anesthetic analgesic upper lip and in the lower lip which is only analgesic. All the rest of the patients with trigeminal neuralgia have been free of major paresthesias so that we think the tedious efforts to conserve touch have been worthwhile.

Incidentally, this preservation of some touch sensation makes a temporizing surgical procedure unnecessary. The alcohol blocks or peripheral neurectomies of one of the trigeminal divisions which we formerly performed, even in low risk patients, as a preliminary to trigeminal rhizotomy to give the patient a protracted trial of his tolerance to total anesthesia of a part of his face, have been obviated by a procedure that produces differential anesthesia.

These encouraging results regarding postoperative paresthesias do not apply to patients with atypical facial neuralgia. The patients in this unhappy category have often complained more of their original pain or described a new pain even in the presence of the analgesia without anesthesia that we have produced.

We produced first division analgesia intentionally in 29 patients, unintentionally in 16 for a total of 45. With proper instruction the person with analgesia but not anesthesia of his cornea learns to act at once on the warning sensation of touch of a foreign body on the eyeball. He must have it sought out and removed at once. Our patients with this type of sensory loss whether produced by a differential thermal rhizotomy or by bulbar tractotomy have never allowed a corneal injury or scar to develop. However we deprived 28 of these 45 patients of touch sensation on the cornea, with and without pain referred to the first division. One of these has become blind in that eye due to corneal scarring, as already mentioned. These unintentional degrees of sensory loss have often been due to impatience either of the person in pain or the operator. More Innovar deals with the impatient patient; as for the surgeon, this is no procedure for one who considers his time too valuable to spend the 2 or 3 hours of painstaking evaluation which may be required; a retrogasserian open rhizotomy goes much faster. Instrumental artifacts or failure are less frequent causes of impaired control over the lesion. We do not recommend competing with the surgeon who described making trigeminal electrothermal lesions in 7 patients in 1 hour. Indeed the latest feature of our technique to avoid corneal anesthesia makes it even more time-consuming.

**Delayed Improvement.** In one patient the typical second division paroxysms provoked by touching of tongue to upper jaw continued for 4 days postoperatively despite complete analgesia throughout the first and second trigeminal divisions, although touch was preserved. The paroxysms then stopped and have remained absent for the ensuing 25 months. Oddly enough, 3 years earlier, an infraorbital alcohol injection in this man produced appropriate numbness at once but did not stop the provokable paroxysms until the sixth postoperative day. The pain was then relieved for 6 months. We have had two other patients with a similar history of delayed relief from a properly placed radiofrequency lesion and a third whose paroxysms continued a few days after a retrogasserian rhizotomy with the desired sensory loss.

**Long-Term Results**

It was our initial impression that moderate to severe hypalgesia in the trigger zones should suffice to give lasting relief because of the reports of such relief from the partial injuries by alcohol, boiling water, phenol and mechanical compression. As our experi-
ence increased, two facts became apparent; 1) we could usually carry the sensory loss to full analgesia to pin prick in the desired areas without producing anesthesia to touch; 2) the less the sensory loss to pin prick the greater the likelihood of recurrence. Patients with a late recurrence sufficiently severe to request another operation have included 33 patients with trigeminal neuralgia; of these, five had more than one operation. Two patients who were especially eager to avoid anesthesia have had, in one case five procedures, and in the other six. During these no additional technical problems were apparent such as may occur when trigeminal alcohol injections are repeated into the scar evoked by the first injection. We have sought to follow each patient at 6-month intervals by personal examination or response to questionnaire. Only two were completely lost to follow-up; however, 143 of the 272 operations have been performed since January 1, 1971, and longer follow-ups from 2½ to 6 years are available in only 125. The recurrence rate in this group of 125 thus far is 22%, tending to increase with the passage of time (Table 1).

### Discussion

**Relation of Fiber Size to Conduction of Impulses for Pain and Touch**

Animal and human experiments reveal that impulses signalling noxious stimuli are conducted in the smaller fibers of the A-delta and C groups (2.5 to 4μ and less than 2μ in diameter respectively). Bessou and Perl⁵ have, however, presented evidence that about half of the unmyelinated fibers in a cat's cutaneous nerve are highly responsive to either gentle mechanical or innocuous thermal stimuli. The remaining half of these C fibers functioned at high threshold, requiring noxious stimuli for activation. Moreover it has long been known that very light stroking of the skin evokes activity in the A-delta fibers of the cat's cutaneous nerve.⁶ It is also long established that touch activates the largest fibers in the cutaneous nerves in the A-alpha-beta range from 8 to 14μ diameter in the cat.⁷ Determination of the subjective quality of touch or pain sensation, if any, which might remain after destruction of the peripheral A-delta and C fibers requires more studies in man. Our studies are the first reports of this kind of which we are aware.

The fact that we have been able with relative consistency to destroy fibers concerned with pain while many of those for touch remain intact correlates well, we think, with the excellent study of Letcher and Goldring⁸ who have photographed the compound action potential in the saphenous nerve of the cat after graded heating with radiofrequency current or saline (Fig. 7). They found that conduction in both the A-delta and C fibers was stopped before that in the A-alpha-beta group. However, at this point the alpha-beta deflection had been reduced to 10% to 20% of its original amplitude. Extrapolating these observations to our human data, we suggest that the relatively easy differential production of analgesia in trigeminal zones in man is associated with exclusive conduction of impulses for noxious stimuli in the smaller myelinated and unmyelinated fibers. In general, threshold for touch was elevated in the analgesic zones, often such that a bit of food on the lips might not be detected even though a light stroking of the skin by the examiner's finger was felt and localized. Direct questioning usually correlated this objective response with some subjective observation of numbness. This sensory status appears to be associated with a major reduction in the action potential spike of A-alpha-beta fibers.
Differential thermocoagulation of trigeminal ganglion

Fig. 7. Oscilloscopic tracings from feline saphenous nerve before and after increments of heating with radiofrequency current. Top row: a fast oscilloscope sweep showing A-beta (tall) and lower A-delta (short) peaks. In (2) the A-delta peak is greatly reduced after the first period of RF heating. In (3) after second heating the A-delta peak is gone, and A-beta is reduced. In (4) after the third heating, A-beta is still lower. Lower row of traces represents a slower sweep; the A deflections are telescoped into the first millimeter to permit full display of the later C wave which finally disappears after three periods of heat. RF = application of radiofrequency heat. (Reproduced from Letcher, F. S., Goldring, S.: The effect of radiofrequency current and heat on peripheral nerve action potential in the cat. J. Neurosurg. 29: 42-47, 1968.)

Sensation of Heat When Rootlets Are Warmed

It is interesting that heating the rootlets should cause a hot or burning sensation; no specialized sensory endings have to our knowledge been described among the trigeminal sensory rootlets. Hence this observation does not accord with the concept that special receptors are necessary to transduce heat into an axonal response signalling temperature. The relevant sensory endings, whatever their type, may be in dura. We need a neurohistological search of the areas concerned.

Summary

We have described a technique for graded radiofrequency heating which permits differential destruction of pain fibers in the trigeminal ganglion and rootlets with sparing of many fibers for touch. We have performed 353 of these procedures without mortality or any focal morbidity outside the trigeminal zone; 91% of 214 patients with trigeminal neuralgia experienced immediate relief of pain. The stress of the procedure is so small that the patient can leave the hospital the next day. In 6% the initial session gave inadequate sensory loss and early repetition was required. In 125 patients with trigeminal neuralgia followed for 2 to 6 years the late recurrence rate has been 22%. The procedure can be repeated without increased difficulty or hazard.

Correlation of cutaneous nerve action potentials in animal studies with our data indicates that pain is carried by some of the smaller myelinated and unmyelinated fibers, virtually all of which can be destroyed by carefully graded increments of heating. Left intact is a portion of the compound action potential due to the large A-beta fibers. Their heavier myelin sheaths may give them some protection from the heat. This is associated with preservation of some sense of touch, and a very low incidence of analgesia or hypalgesia dolorosa.

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