TRIGEMINAL NEURALGIA

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Trigeminal neuralgia or tic douloureux is a lancinating, paroxysmal pain in the distribution of one or more of the branches of the trigeminal nerve. Each paroxysm lasts only a few seconds and is frequently likened to an electric shock. It is a disease of middle and later life but occasionally appears in young subjects. In the beginning, the pain is confined to one branch of the nerve, most frequently the infra-orbital, but with the passage of time it tends to spread to the other branches. The mandibular branch is somewhat less often the site of origin and the supra-orbital branch is very seldom involved primarily. The disease affects both sexes about equally and in approximately 2 per cent of the cases the pain is bilateral.

The cause of trigeminal neuralgia is not known. By the patient, however, the teeth are always looked upon with suspicion because of the location of the pain and because it resembles that produced when a dentist's drill touches a sensitive pulp. Furthermore, the pain is frequently incited by touching a tooth or by chewing. Despite this, however, it can be said with authority that no dental condition, however neglected and aggravated, can cause tic douloureux. Within a few months of the onset, these sufferers have several or perhaps all of their teeth removed, usually at their own insistence and always to no avail. In the first complete description of this disease in 1804, Samuel Fothergill[1] suspected that "the cause of these extreme pains in the face might possibly be of a cancerous nature." This idea, of course, has long since been proved erroneous. Frazier[2] has recently advanced a theory that trigeminal neuralgia is caused by a lesion, probably of vascular origin, in the external thalamic nuclei and he has offered considerable evidence in favor of this view. Time and the opportunity to examine a sufficient amount of autopsy material will determine the correctness of this theory. In an occasional case, the pain of tic douloureux is due to pressure on the root of the nerve by a tumor or aneurysm.

The intervals between paroxysms are usually entirely pain-free but the patient lives in constant dread of the next attack. He learns that certain actions—such as talking, eating, drinking, blowing the nose, shaving, and washing the face—are especially likely to bring on a paroxysm. When certain small areas, called trigger zones, are touched, a paroxysm is apt to be incited. These trigger zones may be anywhere within the area of the trigeminal nerve but are especially frequent on the upper lip, the ala of the nose, and the upper alveolus. A light touch in these areas is more apt to induce an attack than is heavy pressure. At times there are latent intervals during which no form of stimulus will

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incite an attack. A latent interval frequently occurs while the patient is in the doctor's office so that the patient is led to remark, "Why, it is just like going to the dentist, my pain is gone!"

Spontaneous remissions are a characteristic of trigeminal neuralgia. They occur more frequently and last for a longer time in the early stages of the disease. In exceptional cases the pain may be absent for as long as three years. With the passage of years the pain seems to become more fixed, more extensive, and the remissions are shorter and fewer. As in the case of multiple sclerosis, these spontaneous remissions may lead to misconceptions regarding the efficacy of various attempts at treatment. Not infrequently the patient will report that the removal of a tooth, a change in climate, or the use of some new medicine "cured the pain for six months" when, as a matter of fact, the "cure" was a spontaneous remission. Occasionally, trigeminal neuralgia appears as a symptom of multiple sclerosis and, in England, a familial form of the disease occurs in association with multiple sclerosis[3].

In making a diagnosis of trigeminal neuralgia, the doctor is dependent entirely on the patient's description of the pain because primary objective findings of this disease are entirely absent. Usually, however, there are secondary outward signs which should be sought out. In many cases a voluntary "splinting" of the facial muscles is caused by the patient's endeavor to ward off attacks. For this reason, the facies may be mask-like and the patient will talk and sometimes drool out of the opposite corner of the mouth. Because talking is so apt to incite a paroxysm, these patients usually bring a relative to the doctor's office to do their talking for them. Touching the face is also apt to incite a paroxysm, and, for this reason, the painful side of the face may not be washed as thoroughly as the opposite side, in which case the skin is more oily, especially about the ala of the nose. During severe attacks, cleansing of the mouth is avoided, debris accumulates on the teeth, the tongue is coated, and the breath foul. The reaction to the paroxysm differs with the patient but usually follows a set pattern in each case. One patient may suddenly become immobile, "freeze" so to speak, with the onset of each pain; another may grimace; another may grab or rub the face firmly; and the more neurotic patients have been known to actually roll on the floor. Flushing of the face and lacrimation are occasionally present during paroxysms. In testing the sensation in patients with tic one finds as a rule a definite over-response to painful stimuli. These patients are apt to withdraw very quickly from a slight pin-prick even though it is not the painful area which is being tested.

There is undoubtedly a "tic constitution." Frazier[2] has stated that patients so afflicted are more or less temperamentally alike, admitting exceptions, no matter what their station in life or their occupation. He

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finds that these patients have always been apprehensive and disposed to worry about minor matters, but no matter how long the duration or how intense the pain, they do not become addicted to morphine. It seems to me that the condition is more common in the rural than in the urban population. There is a definite tendency for these patients to seek out fellow sufferers, so that the average patient knows of as many cases as the average physician will encounter in a lifetime of practice. I agree with Frazier that hypertension, arteriosclerosis, impaired renal function, and arthritis are encountered in this group of patients more frequently than in those of a corresponding age group who are unaffected. Despite this, longevity seems to be the rule. These patients do not like to go to bed because of pain, so they usually are up and around even when seriously debilitated by very frequent paroxysms and the attendant starvation.

Another disorder which frequently is confused with trigeminal neuralgia is referred to as "atypical neuralgia." This pain differs from trigeminal neuralgia in that it is constant and not paroxysmal, it is pressing and burning in character rather than sharp and lancinating. It usually extends well beyond the distribution of the fifth nerve into the occipital region, behind the ear, and down the side of the neck. The patients are very neurotic and while informing the doctor of the severe agony which they are suffering they exhibit no outward sign of pain. They are content to lie in bed and be waited upon. The pain usually originates following some manipulation, especially extraction of teeth. All efforts to relieve the pain by alcohol injections, nerve resections, or by operations upon the sinuses or teeth merely tend to aggravate the pain. I do not know what the end result is in these cases but I have never seen a patient permanently cured. These patients frequently are afraid they have tic douloureux when, as a matter of fact, they would be far better off if they did have it, at least insofar as relief of pain is concerned. These patients welcome operative procedures but are always made worse by them. Patients with true tic, on the other hand, avoid operation as long as possible but are pleased with the result.

Treatment

What is to be done for the patient with trigeminal neuralgia? In answer to this question, one may say that only two forms of treatment are worthy of the name—alcohol injection of the peripheral branches of the fifth nerve and resection of the sensory root of the nerve within the cranial cavity. Inhalations of a volatile preparation called trichlorethylene may diminish the pain in 10 per cent of the cases, but it very seldom gives satisfactory relief. Other forms of medical treatment are of no avail. Operations upon the teeth and sinuses are to be deplored.

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Peripheral resections of the infra-orbital and mandibular nerves do not give as long a period of relief as do subzygomatic alcohol injections. Resection of the supra-orbital nerve may be used instead of alcohol injection of this branch but the period of relief is short. This supra-orbital branch is so rarely involved primarily that its treatment is seldom indicated. Destruction of the gasserian ganglion by the injection of alcohol or by electrocoagulation by the closed method entails unjustifiable risks when one considers the safety of the generally accepted methods.

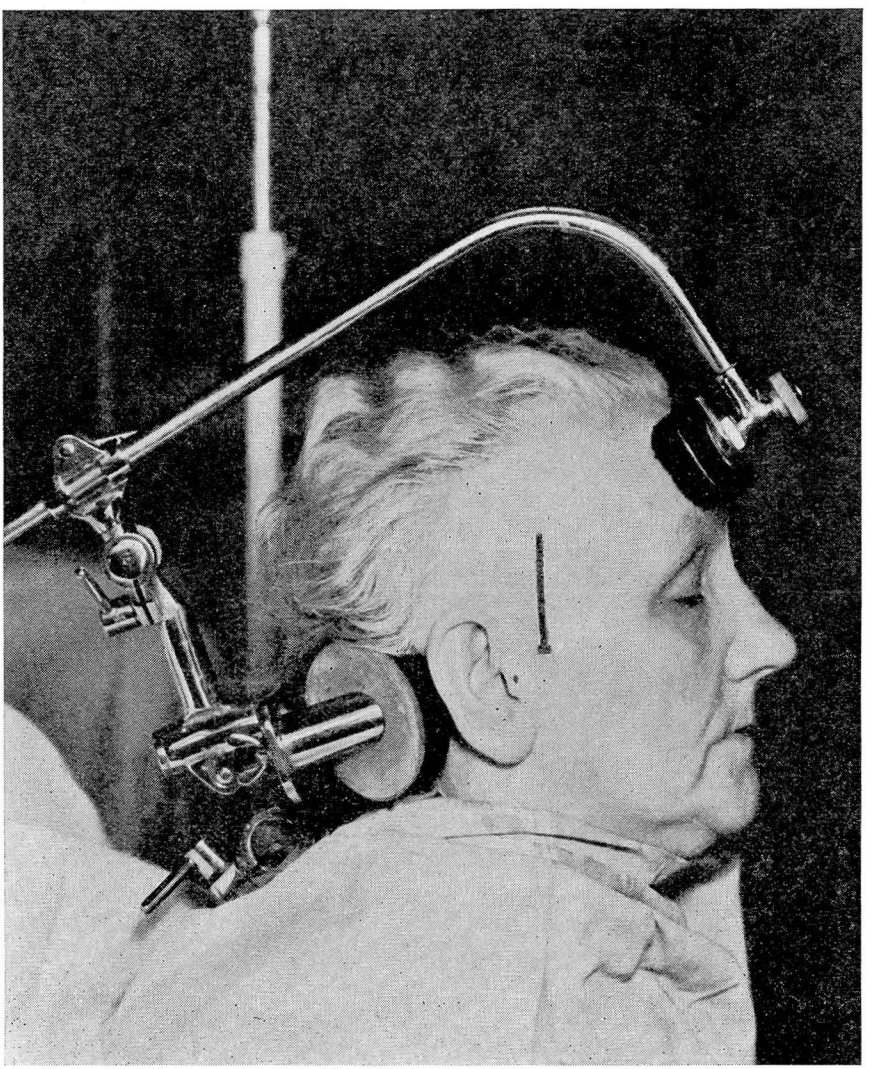

FIGURE 1: The patient is in position for operation and the skin incision is indicated.

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A good practice when confronted with a case of trigeminal neuralgia is to ask the patient if he would prefer numbness of the face to the pain. If the patient has genuine trigeminal neuralgia, he will not hesitate but will at once choose numbness. He may then be told of the two preferable methods of producing this numbness and be allowed to choose between them. As a rule the patient will choose alcohol injection if he has not had the pain very long, but if he has had previous alcohol injections, he usually wants the operation which will give him permanent relief. Of course, if there is any question about whether the patient has genuine

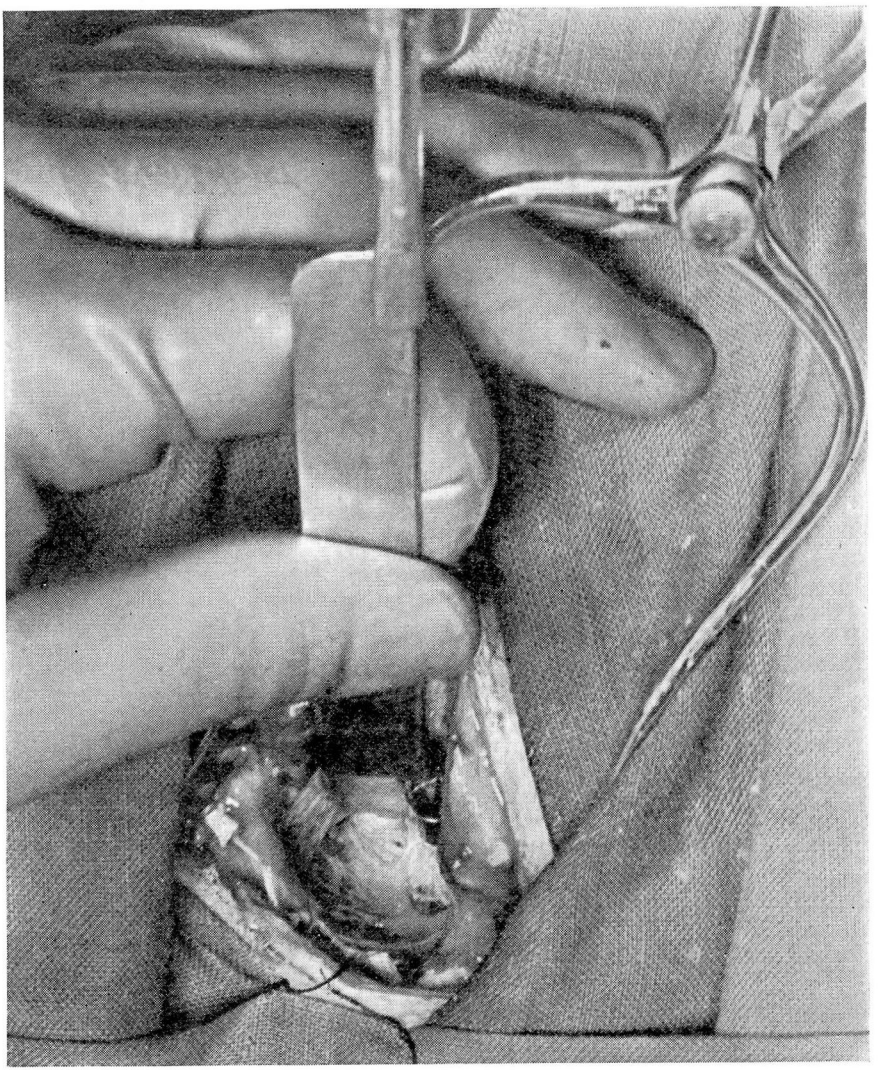

FIGURE 2: The sensory root fibers can be seen through the opening in the sheath.

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trigeminal neuralgia, the operation is never advised. In such cases the alcohol injection is a diagnostic as well as a therapeutic agent. If the patient has genuine trigeminal neuralgia and the painful area is made numb by the injection of novocain and alcohol into the nerve, the pain is immediately stopped and no stimulus will bring it on. If the neuralgia is of the atypical form, the injection will not relieve it at once or, if it does so, the pain will recur long before the resulting anesthesia wears off. An alcohol injection can be done far more accurately with local than with general anesthesia. The pain produced by the injection is comparable to a paroxysm. The disadvantage of the injection method of treatment is that when the operation is finally decided upon, the patient has suffered in the meantime and is from one to several years older and therefore may not be quite as fit for operation.

If the treatment is to be alcohol injection, the subzygomatic route is the one of choice. By this method the second or third division can be interrupted at the point of emergence from the skull. If the alcohol is introduced at some more peripheral point, the nerve regenerates more rapidly and the period of relief is correspondingly shorter. In a subzygomatic injection, the needle is introduced in front of the ear and just below the zygoma and is directed toward the foramen of exit of the branch it is desired to inject. When the needle enters the nerve, the patient will experience a sharp pain similar to one of his paroxysms. One cc. of 2 per cent solution of novocain is then injected slowly. If the point of the needle is within the nerve, the numbness will appear within two minutes, in which case 0.6 to 1.0 cc. of 95 per cent alcohol is injected slowly without moving the needle. If satisfactory anesthesia of the skin is obtained, the relief is immediate and persists for nine to eighteen months. By the end of this time, the numbness will usually have cleared up after which the pain recurs within a short period of time. The alcohol injection can then be repeated but subsequent injections become slightly more difficult.

The modern operation for trigeminal neuralgia is a resection of the sensory root. This operation was suggested by Spiller and performed by Frazier in 1901[4], and soon superseded the Hartley-Krause operation of removal of the gasserian ganglion. The Spiller-Frazier operation consists of the division of the sensory root fibers of the fifth nerve just behind the ganglion. It results in permanent numbness of the face and permanent relief of pain because these preganglionic fibers, once divided, never regenerate. The present technic is as follows:

With the patient in the sitting position and under light ether narcosis, a vertical incision five centimeters in length is made just in front of the ear and down to the zygoma (Fig. 1). The scalp and temporal muscle

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are retracted and an opening the size of a fifty cent piece is made in the squamous portion of the temporal bone. The dura is then elevated from the floor of the middle fossa of the skull, following the course of the middle meningeal artery to its point of entrance through the foramen spinosum. A bit of cotton on a dental applicator is forced into this foramen in order to occlude the middle meningeal artery which is then severed. Just anterior and mesial to the foramen spinosum the gasserian ganglion is encountered at the point where its third division leaves the skull through the foramen ovale. The dura is stripped upward from the lateral surface of the sheath of the ganglion and sensory root. As the sheath is opened posterior to the ganglion, the sensory fibers can readily be picked up on a nerve hook and divided (Fig. 2). If the patient has had no pain in the forehead, one or two small fasciculi to the first division are spared. When this is done a postoperative keratitis never occurs. This procedure of sparing the ophthalmic fibers was devised by Frazier and it marks a definite advance in the treatment of trigeminal neuralgia. If the entire sensory root is divided, a keratitis will develop in an occasional patient but this clears up promptly when the cornea is properly protected. The motor root of the fifth nerve which supplies the muscles of mastication can readily be identified and spared. It is especially necessary to spare this motor root in bilateral operations; otherwise the patient would never be able to chew. Dividing the motor root on one side causes no disability except some facial asymmetry due to atrophy of the masseter muscle.

After an operation of this sort, the course is surprisingly smooth and the patient is discharged on the seventh day. A very small amount of ether is necessary for anesthesia in these cases, presumably because of the erect posture, and there are no complications from the anesthetic. The only mortality in the author's series occurred in an elderly man who insisted on avertin anesthesia. His sudden death during the operation was ascribed to coronary disease, however, and not to the anesthetic. The risks of the operation are so slight that it is very seldom that a patient is considered unfit for it.

Hundreds of cases are on record with a mortality of less than 1 per cent, these operations having been performed by various surgeons. In view of the slight risk of the operation and the permanency of the cure, some surgeons have refused to give alcohol injections and have insisted that their patients have the operation. This is perhaps a good course to pursue, for once the natural timidity of these patients is overcome and their pain is relieved by operation, they are eternally grateful.

In the entire realm of surgery there is probably no more satisfactory operation than Frazier's subtotal resection of the sensory root of the fifth nerve. The gratitude of patients relieved by this procedure is in

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some cases really pathetic. In the well-balanced individual there are no unfavorable sequellae. The very neurotic patient may complain of the numbness or of a sensation of tickling, itching, or burning in the numb area. Occasionally there are "ghost pains" which last for some time after operation. These "ghost pains" are sudden darting sensations just like the former paroxysms except that they are painless. When the patient is convinced that they do not indicate a return of the former pain, they are disregarded.

Another method of approach has been described by Dandy[5] who advocates division of the sensory root of the fifth nerve at the pons. This method, however, will never be generally adopted. Neurological surgeons are almost universally agreed that this approach is more dangerous both to life and function than is the foregoing temporal approach, besides which it offers no practical advantages.

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