“GANGLIOLYSIS” FOR THE SURGICAL TREATMENT OF TRIGEMINAL NEURALGIA

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In 1952 Taarnhøj\(^1\) described a method of decompression of the trigeminal nerve for the treatment of tic douloureux. In this procedure the dural sheath enclosing the trigeminal nerve is divided without sectioning the nerve. The dural incision is made over the most posterior part of the ganglion, and follows the course of the trigeminal root until the superior margin of the petrous bone has been reached and the superior petrosal sinus has been divided. It is then usually extended to the tentorium. The operation leaves no sensory deficit.

In Taarnhøj’s opinion neuralgia is caused by narrowing of the porus trigemini and chronic pressure, especially in older people. All of his 10 patients were free from attacks from 1 to 8 months after operation. His findings were confirmed by Kautzky\(^4\) who had similar results in 16 cases. A purely mechanical explanation of the results of Taarnhøj’s operation, however, would seem unsatisfactory. There is no reason to assume that anatomical narrowing takes place in the trigeminal canal or in other foramina through which cranial nerves pass. Furthermore, trigeminal neuralgia occurs in attacks while a compression would be more likely to lead to continuous pain rather than to paroxysms which can be precipitated by a great variety of external factors, such as variations in temperature as well as by eating, shaving, talking, or brushing of the teeth.

In an unpublished case at Foerster’s clinic in Breslau, a Frazier operation for tic douloureux was started but could not be finished because of severe hemorrhage necessitating tamponade and closure before the root could be sectioned. This patient recovered and was surprisingly free of attacks of pain for several years without sensory deficit. With this case in mind an attempt was made to modify Taarnhøj’s method by simply exposing the gasserian ganglion without splitting the dura mater or sectioning the nerve.

TECHNIC

The procedure is started, as in Frazier’s operation, by an extradural approach after occluding the middle meningeal artery in the foramen spinosum and by blunt dissection of the dura mater from the 3rd branch. After the ganglion has been visualized the 2nd branch is similarly exposed until finally the root itself can be seen (Fig. 1). At this stage the coverings of the cavum Meckelii still overlie the ganglion. This tenuous membrane is occasionally torn by manipulation of the dura mater but usually it is necessary to remove it after an incision with a small hook knife. In re-
moving the dura mater one should be extremely careful, as traumatization of the ganglion as well as of the pars triangularis of the root may lead to a sensory deficit.

Fig. 1. Exposure of the gasserian ganglion.

**CLINICAL MATERIAL AND RESULTS**

The gasserian ganglion was exposed in 18 cases during the past 13 months, and in most instances the cavum Meckelii was opened. Of the 18 patients, 16 had typical tic douloureux, 1 sustained trigeminal pain following an inflammatory process in the maxillary sinus, and 1 presented a mixture of continuous pain with additional paroxysmal attacks of pain superimposed. In all cases of uncomplicated tic douloureux, pain was abolished for up to 13 months. In 1 of the earlier cases in which the cavum Meckelii had not been opened, some paresthesias occasionally recurred in the distribution of the 2nd and 3rd branches. A relapse of pain occurred in the patient in whom tic douloureux was complicated by continuous pain, and in this case also the cavum Meckelii had not been opened. Therefore in subsequent cases the opening of the cavum was considered to be the most important part of the procedure. In 2 additional cases there was moderate hypesthesia postoperatively in the distribution of the 2nd and 3rd branches, probably as the result of traumatization of the ganglion.

**DISCUSSION**

An attempt should be made to discuss the etiology of the paroxysmal attacks of pain in tic douloureux. Several older authors believed that the attacks were caused by a vasomotor disorder, especially angiospasms (Quincke, Kulenkampff, Haertel, Hughes). Others like Olivecrona, and Dandy were inclined to explain them on a mechanical basis, such as irritation of the root by vascular malformations in the posterior fossa. In some cases of verified cerebellopontine-angle tumors tic douloureux is a symptom. Lewey regarded trigeminal neuralgia as a partial thalamic syndrome. Schaltenbrand
recently described autopsy findings in a case of unilateral trigeminal neuralgia in which both gasserian ganglia showed severe arteriosclerotic changes. In this case no local treatment by alcohol or novocain injections had been performed. On the basis of these findings Schaltenbrand considered trigeminal neuralgia as "claudicatio intermittens" of the gasserian ganglion. This explanation, however, does not seem fully satisfactory, as trigeminal neuralgia may occur in younger or middle-aged individuals.

One might consider an old theory according to which tic douloureux is somehow related to the numerous inflammatory processes of teeth and sinuses. Almost every one has suffered some of these many times in his life. This does not mean that a given inflammatory process as such could produce trigeminal neuralgia. Another interpretation seems justifiable: In all these infectious processes, pain, i.e. irritation of a branch of the trigeminal nerve, is produced. Each attack of pain, according to the theories of Leriche and others, leads to contractions of the blood vessels of the nerve. As many patients suffer from these inflammatory processes repeatedly one could conceive that a "summation of stimuli" in certain cases leads to conditioned vascular reflexes, thus creating a new disease, namely neuralgia. Neuralgia follows its own course and is no longer immediately related to the original episodes of pain which have subsided long ago. In patients with causalgia and in others with the thalamic syndrome tactile stimuli are perceived as pain. One might be justified in speculating that on the basis of irritative stimuli in the distribution of the trigeminal nerve, repeated off and on for 10 or 20 years, a similar mechanism is produced, which leads to precipitation of attacks of pain by physiologically tactile stimuli, such as touching the skin, shaving, touching the gingiva in brushing the teeth, etc.* In this situation of lowered threshold for pain tactile stimuli lead to a paroxysm of pain associated with a "spastic vascular crisis." The single episode of tic douloureux could then be conceived as an acute state of anoxemia. This in turn leads to compensatory mechanisms, usually effective in a short time, with the well known signs of vasodilator activity, as hyperemia of the skin in the area involved, increase in secretion of the nasal mucosa and tearing.

If the paroxysm of pain is associated with a vasomotor reflex mechanism it is also conceivable that arteriosclerotic mechanisms may play a part, as in Schaltenbrand's8 case. There is a definite increase in incidence of neuralgia with increasing age.

There is one other form of true neuralgia with similarly unbearable attacks of pain: glossopharyngeal neuralgia. On the basis of what has just been discussed concerning trigeminal neuralgia it is quite conceivable that recurrent inflammatory states of the tonsils in certain patients may lead to glossopharyngeal neuralgia. Again by summation of stimuli and progressive conditioning a pathological vasomotor reflex of the 9th cranial nerve might be produced.

* The extreme rarity of tic douloureux involving the 1st branch would indirectly support the theory that neuralgia is somehow a secondary disease following repeated attacks of dental pain.
If one assumes this mechanism to be valid the operative results initially described might be understood. By exposing the ganglion, its vasomotor supply and that of the branches of the trigeminal nerve would be altered. The operation leads to hyperemia and possibly also edema. In the subsequent stage of reparation new formation of capillaries in the region of the ganglion and its coverings should lead eventually to a different vasomotor situation with improved blood supply, and the spastic vascular reflexes might conceivably cease for this reason. This in turn might lead to cessation of the neuralgia.

Time has been too short for a final evaluation of the therapeutic results of this method. It will be necessary to wait for relapses, and to compare the incidence of relapses with that after other operative procedures. The advantages of this method in comparison with Taarnhøj's procedure are: the surgical risk is smaller, the operation is less extensive, and complications such as extraocular muscle palsies are not encountered. Mainly the method does not lead to the very annoying sensory deficit that follows section of the root.

SUMMARY

Trigeminal neuralgia has been abolished in 18 cases by operative exposure of the gasserian ganglion. In most instances the cavum Meckelii was opened. This seems to be the most important step of the operation. Root section is not necessary with this method, and therefore there is no sensory deficit. One might describe the method as "gangliolysis." So far only patients with genuine tic douloureux have been benefited by the operation.

An attempt is made to explain the paroxysms of pain on the basis of a faulty conditioned vasomotor reflex, leading to paroxysmal anemia. The effect of the operation consists in a fundamental change in the blood supply and vasomotor situation of the ganglion.

REFERENCES