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TRIGEMINAL NEURALGIA AND ITS CURRENT THERAPY

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OUTLINE

INTRODUCTION

Trigeminal Neuralgia and its Current Therapy

Trigeminal neuralgia is also known as Tic Douloureux. Tic is a French word meaning a convulsion motion or fit. In this case, Tic refers to convulsion of the muscles of the face. Douloureux means painful, and the two together are taken to mean spasmodic, painful contractions of the muscles of the face, unilateral or bilateral in character.

In this paper I shall attempt to present the subject of Trigeminal Neuralgia, with the major emphasis on etiology and therapy; and with a tendency to summarize other phases of this neuralgia.

HISTORY

(SUMMARY)

Although tic douloureux has been recognized as a clinical entity for centuries, its relationship to the trigeminal nerve was long unsuspected, because the course and function of the cranial nerves were unknown. In his book, DIE NEURALGIE DER TRIGEMINUS, Krause[1] said that Aricenna, 1000 A.D., was the first to give an accurate description of the disease; that Schlichtung, 1748 A.D., first cut the infra-orbital nerve for the pain that was called "facial neuralgia;" and that Nicholas Andre, A.D. 1756, first introduced the name of tic douloureux.

Dandy,[2] acting as historian, reports that, through Galen's period until the time of Meckel's careful dissection of the fifth nerve in 1748, the facial nerve was believed to supply not only the motor but also the sensory functions to the face. Experimental proof of the sensory functions of the fifth nerve and of the motor function of the seventh nerve was produced almost simultaneously by Magendie and Sir Charles Bell about 1821. It was their experimental contribution to the nerve function which permitted the first rational therapy for tic douloureux.

Other men mentioned in conjunction with tic douloureux of the last two centuries will not be mentioned here as they will be referred to when discussing the aspects of trigeminal neuralgia in which they are noted.

ANATOMY OF THE TRIGEMINAL NERVE

(SHORT SUMMARY)

Gray's[3] and Thorek's[4] anatomy texts are my sources for this summary which is as follows.

The trigeminal nerve is the thickest of all the cranial nerves and has a wide distribution. It is a large sensory root upon which the semilunar ganglion is situated, the ganglion resting in the fossa on the superior surface of the petrosus bone near its apex.

The three main divisions of the nerve are as follows: ophthalmic, maxillary, and mandibular which arise from this ganglion.

The fifth nerve resembles the spinal nerve in that it has two roots, a sensory and a motor, with a ganglion on the sensory. The motor root lies in the inferior lateral to the sensory root and does not enter the ganglion. Both roots arise from the lateral part of the inferior surface of the pons. The nerve provides the sensory supply to the face, and the anterior half of the scalp, and sends motor branches to the four muscles of mastication and to four other muscles: tensor palatini, tensor tympani, mylohyoid, and anterior belly of digastric. There are five ganglia located on the fifth nerve: the semilunar on the nerve trunk, the ciliary on the ophthalmic division, the sphenopalatine on the maxillary division, the otic on the mandibular division, and the submaxillary also on the mandibular division. All of these ganglia, with the exception of semilunar, receive motor, sensory, and sympathetic fibers. The semilunar ganglion, the sensory ganglion of the fifth

nerve, occupies a space between the outer and inner layers of the dura known as the cave of Meckel which is in reality a diverticulum of the inner layer of the dura.

The ophthalmic is the smallest division of the fifth nerve. After giving off a small twig to the dura mater, it passes forward in the lateral wall of the cavernous sinus and enters the orbit through the superior orbital fissure. In the fissure it splits into three branches: the frontal, the nasal ciliary and the lacrimal.

The maxillary division of the trigeminal nerve resembles the ophthalmic in that it is purely sensory. It leaves the middle cranial fossa through the foramen rotundum and reaches the pterygoid palatine fossa. After crossing this fossa the nerve leaves by way of the infra-orbital fissure to occupy the infra-orbital groove and canal. It appears on the face at the infra-orbital foramen and as the infra-orbital nerve; here it divides into the following terminal branches: a small meningeal branch, the dura mater, two ganglionic branches to the sphenopalatine ganglion, zygomatic branches, temporal and facial, posterior superior alveolar to the molar teeth, infra-orbital supplying pre-molars, canine and incisor teeth, and the facial which supplies the lower eyelids, the side of the nose and the upper lid.

The mandibular nerve is the largest division of the trigeminal. It consists of a sensory portion derived from the trigeminal ganglion and a motor root. These two portions are found passing separately through the foramen ovale but rejoin immediately afterward

to form the common trunk. After giving off the meningeal branch, the nervus spinosus which enters the cranium through the foramen spinosum, the common trunk furnishes a twig to the medial pterygoid and following this divides into an anterior and posterior division. The anterior division consists mainly of the motor fibers and divides into the deep temporal nerves, the nerves to the masseter, the lateral pterygoid nerves and the buccinator nerve. The posterior division gives off two roots, one of which becomes the auricular temporal, while the other divides in the lingual and inferior alveolar nerves. The only motor fibers in this division are those that form the mylohyoid branch of the inferior alveolar. The anterior division has been referred to as a lingual division and the posterior has been called the inferior dental. As both of these divisions run downward they are concealed by the external pterygoid muscle and the ramus of the mandible and are distributed to the tongue, the gums, the lower teeth and the muscles of mastication. Grant and Weinberger[5] in studies of cranial nerve V found that its fiber tracts of pain and temperature sensation are as follows:[5] the fibers conducting the pain and temperature modalities turn downward in the company with the nucleus of the spinal tract of the fifth nerve and run throughout the length of the medulla oblongata, and into the upper cervical portion of the cord. During their course they emerge from under the cover of the restiform body and take a superficial position on the lateral surface of the medulla. In this situation they form a distinct elevation

on the surface of the medulla, the tuberculum cenereum. In the closed portion of the medulla the tuberculum cenereum lies below the restiform body and above the olivary eminence. The fibers mediating touch sensation, on the other hand, turn upward at the point of entry into the brain stem, end in the main sensory nucleus of the trigeminal nerve and thence by the secondary neuron ascend to terminate in the sensory nucleus of the thalamus.

SYMPTOMS OF TRIGEMINAL NEURALGIA

Harris[6] and Frazier[7] best describe the symptoms which are as follows.

Trigeminal neuralgia may begin at any age, but is rare before middle life, and in most cases the onset occurs at about the age of 50, but may be as late as 70 or even later. Sometimes emotion, exposure to cold or a blow on the face appears to precipitate the first attack. The affection is nearly always unilateral and is, perhaps, more common on the right side. Bilateral neuralgia occurs in about 1% of the cases, but not simultaneously.

The characteristic feature of trigeminal neuralgia is the occurrence of brief, severe, paroxysms of pain, which is usually for a long time confined to the division of one nerve. The second and third divisions are the site of pain with approximately equal frequency. The first division is rarely affected and then usually only after the second division has been involved. When the pain first involves the second or third division, it usually, in the course of time, spreads to the other two divisions. In a small portion of cases it is bilateral, though rarely from the onset. In an attack the pain is usually most intense in, and may be confined to, part of the regions supplied by the affected division. Thus, it may be most marked in the cheek, the upper jaw, the lower jaw or tongue. It tends to spread, however, through the rest of the divisional area. It is usually described as burning or stabbing. The most striking features of the attacks are that

they tend to be precipitated by a chill, by touching the face as in washing, by talking, mastication and swallowing. Most patients describe trigger zones, touching of which will invariably excite an attack. The attacks are always brief and do not last longer than one or two minutes. The pain is very severe and during an attack the patient may be in agony. The pain often reflects and evokes spasm of the muscles of the face on the affected side, hence the term tic douloureux. Flushing of the skin, lacrimation and salivation may occur. In trigeminal neuralgia there is no reduction of sensibility over the distribution of the nerve. So-called trophic changes of the skin have been described, but it is probable that these are the result of the patient's rubbing the face during an attack, or remedies which have been applied in attempts to relieve the pain. The attacks may interfere with the taking of food and the recurrence of severe pain over a long period of time tends to cause loss of weight and depression. Fortunately, the attacks usually cease at night, though they sometimes awake the patient from sleep. Long periods of freedom from the pain, lasting weeks or months, are the rule in the early stages. The course of the disease is variable. It depends upon the cause, age, and condition of the patient and the treatment. Rarely, there is but one attack; usually it is repeated at gradually shortened intervals. The prognosis is fair in young healthy individuals, and also in cases following general infection or local inflammation which can be irradicated. It is poor in older and run-down patients.

ETIOLOGY AND PATHOLOGY

The cause of trigeminal neuralgia is obscure. Histological examination of the gasserian ganglion has revealed no changes which can be responsible. The association of the disorder with dental infection and occasionally with the infection of the maxillary antrum suggests that the infection plays a part in the etiology, but the fact that infection of the teeth and antrum is common, while trigeminal neuralgia is comparatively rare, appears to indicate that some additional factor exists which predisposes to the disorder.

Smolik[8] found in 54 cases with severe malocclusions and associated marked loss of vertical dimensions, that upon replacing the missing teeth, increasing the vertical dimensions, and establishing new concentric relationships 15 cases of major trigeminal neuralgia obtained relief.

There is reason to believe that certain individuals are more liable than others to all forms of neuralgic pain, and it may be that in such persons chronic infections of the endings of the trigeminal nerve sets up the attacks of pain which are perpetuated by functional changes within the central nervous system.

Females are affected more frequently than males in the proportion of three to two. Heredity plays a part in the causation of some cases. In two percent of Harris's[6] cases one of the parents had been a sufferer.

Dandy[9] states that he can almost always find the cause of Tic by the cerebellar route operation. In his series of cases he found that in 10% there is a tumor; 5% there is an aneurysm of the basilar artery on the sensory root of the nerve. He feels that tumors of gasserian ganglion cannot produce trigeminal neuralgia; tumors on peripheral branches of the nerve cannot produce tic douloureux; it is only a tumor on the sensory root of the nerve. These tic-like forms are spasmodic attacks just like epilepsy and are always due to a lesion of the cerebral hemisphere, not in the brain stem or spinal nerves. In the other 85% of his cases there is an artery that runs from the cerebellum to the brain stem, and it lies free in the cisterna, but impinges upon either the outer or inner surface. As age increases this artery, which is about as large as the lead in a pencil, hardens with age pressing upon the naked nerve and sets off these attacks.

Lewis and Grant[10] speculate on a thalmic syndrome as a possible cause of trigeminal neuralgia. They became aware that in 25% of their patients, the patients had noticed paresthesia, penalgesia, hyperesthesia, hypo-algesia and that the quantitative disturbances of sensibility plays a contributory part in this disease. Since 35% of their patients have thalmic syndromes over one side of the body and some motor signs and symptoms, the optic region of the thalmus is thought to be a possible site. In short, they feel that trigeminal neuralgia is a special form of thalamic syndrome caused by functional organic vascular insufficiency.

Summary of their impressions:

1. Qualitative disturbances of sensibility play a contributory part rather than an essential part in trigeminal neuralgia. Qualitative disturbances are pre-eminent in frequency and significance.
2. The change in pattern of sensory impulses together with motor signs and symptoms point to optic thalamus as a possible site of the lesion of trigeminal neuralgia.
3. In six cases of trigeminal neuralgia the brain showed atrophy of the ipsilateral hemisphere, widening of the lateral ventricle which could be visualized by means of an encephalogram during life. The foci of softening were located either within the lateral and medial nuclei of the optic thalamus, or in the thalmocortical tract. In the later case ipsilateral semilunar nucleus and central media of the thalamus were atrophied.
4. The lesions were observed to be angiopathic. Clinical examinations of 50 persons with trigeminal neuralgia showed signs of arteriosclerosis and renal dysfunction.
5. The frequent occurrence of heredo-familial stigmas in cases of trigeminal neuralgia led angiopathic examinations of 40 patients showing that these persons belonged to a uniform group, differing in physical habitus and mental make-up from those persons suffering from atypical facial neuralgia.

Gardener and Pinto[11] believe that the pain of tic douloureux is due to development of an artificial synapse in the sensory root

fibers where the nerve crosses the apex of the petrosal bone. Granit[12] and Lewy[13] proposed that this artificial synapse occurs as a result of the demyelization process and development of a sagging tentorium which accompanies advancing age. The sagging of the tentorium, where it merges with the roof of the dural sheath, transforms a normal oval shaped dural foramen, which transmits the nerve, into a relatively flat slit. With this change in shape of the dural foramen the filaments of the sensory root normally dispersed in the arachnoid sheath distend with cerebrospinal fluid and are held in contact with one another. Contact of various nerve elements of the sensory roots, together with the demyelization of some of the axis cylinders, permit short circuiting of this action current which accompanies the transmission of nerve impulses, with the formation of an artificial synapse. It is also felt by some that compression of the nerve by the dura may also set up an artificial synapse. Thus, this afferent impulse of the sensory root is short circuited into the naked pain fiber, and is reflected back into the brain stem as a painful impulse. Thus, they believe that tic douloureux is due to a preganglionic parasympathetic sensory synapse.

The nature of the virus as a possible cause of tic douloureux is quite open to question. It has been shown by Dolan and Bucy[14] that following complete retrogasserian section of the trigeminal that herpes zoster has occurred on the peripheral nerves of the section site.

Trigger points are areas of hypersensitivity which vary in size and location and when touched, wind blown, or chilled will excite an attack. They are found most often in the areas of the nose, lips, gums and in the skin areas over the infra-orbital and mental foramens.

DIFFERENTIAL DIAGNOSIS

Harris[6] and Frazier[7] state there is usually little difficulty in diagnosis of trigeminal neuralgia if attention is paid to the cardinal symptoms, especially the paroxysmal character of the attacks with freedom from pain in the intervals, the factors which precipitate them, and the absence of signs of organic lesions of the nerve. In rare cases, however, this syndrome may be associated with organic disease, for example, disseminated sclerosis or tumor of the eighth nerve. Other signs of these disorders, however, are usually present. It is important to distinguish trigeminal neuralgia from pain due to gross lesion of the nerve, especially compression by tumor. In such cases the pain is more persistent, and is usually associated with impairment of sensibility in the distribution of the nerve and weakness of the muscle supplied by the nerve. Trigeminal pain may follow lesions with central connections of the nerve within the brain stem, for example, thrombosis of the posterior inferior cerebellar artery. In such cases, however, other signs of brain stem lesions are present. Post herptic pain of trigeminal distribution is distinguished by the history of zoster eruption which leaves characteristic residual cutaneous scars, by the persistence of pain, and by the impairment of sensibility. Tabes dorsalis is an occasional cause of paroxysmal attacks of pain within the trigeminal area. The characteristic signs of tabes, however, render the diagnosis of the cause of the pain readily.

Neuritis of branches of the trigeminal nerve, especially of the supra-orbital and of the auricular temporal, cause pain within the distribution of the recent branch affected. In cases of neuritis there is a history of acute onset; the attacks of pain tend to last for hours, with paroxysmal exacerbations; the affected nerve is tender on pressure; and there is often hyperalgesia or more rarely relative analgesia over the cutaneous areas supplied by the nerve.

Referred pain is extremely common with trigeminal distribution, and possible causes of this must be always excluded. Frontal sinusitis and infections of the maxillary antrum tend to cause pain which is referred to areas of the first and second divisions of the nerve respectfully. In such cases there may be edema of the tissues overlying the infected ear sinuses and, in addition to the tenderness of the supra-orbital and infra-orbital nerves, the bone is also tender to palpation. Radiographic sinuses, transillumination and examination of the nose may be necessary to establish the diagnosis. Diseases of the eye may cause severe referred pain, especially glaucoma, in which the patient refers pain to the temple. Examination of the eye immediately reveals the cause of the trouble and a mistaken diagnosis is rare. The teeth are a common source of referred pain. Attention to dental caries, which are easily detected, may find the pain due to a peri-apical abscess or to an un-errupted tooth. In cases of doubt, radiograms of the teeth should be taken. Pain also may be referred to the face from lesions of the heart and lungs.

Hysterical pain in the face may lead to diagnostic difficulties. It fails to conform to the characters of either trigeminal neuralgia, or to any form of pain due to organic disease. It fails to respond to analgesic drugs, often not even to morphine. Other hysterical symptoms may be present and the patient's mental state usually affords a clue to the nature of the pain.

MEDICAL THERAPY

The current therapy of tic douloureux has two main forms, medical and surgical. The medical therapy has many different types and in this report I shall mention only some of the more frequent modern types.

Stich[15] reports a series of one case which had almost spontaneous relief of pain in tic douloureux by the use of 100 mg. banthine. The rational for this, was that the drug possibly had an inhibitory effect on the gasserian ganglion similar to its effect in various autonomic ganglia. No further evidence on this subject is present in literature at this time.

Cooper[16] relates the relief of one patient of his sharp-shooting pain, with only residual sensation of soreness of the gums on the affected side, by the oral use of 10 mg. three times a day of ethyl beta methyl choline chloride for two weeks duration. Occasional small recurrences appeared which were re-treated with choline. No other information is available in literature at this time.

Paull[17] considers tic douloureux due to typhoid invasion of the trigeminal tract or central to the gasserian ganglion as a possible etiology. He states as his evidence a case of an 80 year old man who had tic douloureux for 30 years. He treated this case with triple typhoid vaccine and by this treatment the patient became immunized. Advantages of this form of therapy while not 100%

effective are as follows:

1. It seemingly is effective in cases who get incomplete relief from surgery or alcohol.
2. The patient can be offered this when he is too old or too feeble to be considered as a surgical case.
3. It is less painful, less dangerous and has fewer complications than alcohol injections or surgical interruption of the trigeminal tract.

Cuprelone which is cuprallyl thioural sodium benzoate which contains 19% copper is advocated by Campbell.[18] He states that:

1. Intravenous injections of cuprelone have been given in 13 cases of trigeminal pain and in some of them the pain was relieved; dosage 50 milligrams each day for 30 days.
2. Toxic effects are negligible in those cases.
3. Cuprelone may possibly act directly on the trigeminal nucleus.
4. This, thus, is a possible means of palliative therapy.

Hutchinson[19] has a great deal of enthusiasm for ferrous carbonate therapy. However, Davidoff[20] reports that approximately one-third of his patients were benefited though admittedly the improvement was frequently not sustained indefinitely. The advantage of this therapy is that it should be given in a trial case in which surgical procedures is deferred for one reason or another.

Horton and Brennan[21] describe a case of typical trigeminal neuralgia in which the attacks of pain show a definite seasonal pattern, being correlated in time with the maturation of the spring

crops. These attacks of pain were relieved by the administration of pyribenzamine hydrochloric and benadryl hydrochloric.

In the pain-free periods typical attacks were provoked by the administration of histamine. After repeated injections of histamine this drug could no longer provoke an attack of trigeminal pain. Horton and Brennan propose the following theses as possible causes:

1. The attacks of trigeminal neuralgia in this patient were brought about by periodic release of some agent resembling histamine, if not histamine itself.
2. Release of the agent was provoked by exposure to some precipitating agent to which the patient was exposed seasonally.
3. The antispasmodic and sedative effect of pyribenzamine, 50 mg. and benadryl, 30 mg., were not important in precluding the trigeminal pain, because both atropine and phenobarbital were ineffective; therefore, pyribenzamine and benadryl exerted their therapeutic effect of antihistaminic activity.
4. The ineffectiveness of a test dose of histamine, after a series of subcutaneous injections were given, was due to a process of desensitivity.

Fields and Hoff[22] report good results on 13 cases in which massive doses of crystalline vitamin B_{12} (100 mg. each day for 10 days) were given. In all cases a quick relief of the severe pain occurred followed by the gradual decrease of secondary burning paresthesias.

Clark[23] relates the temporary relief of pain by means of trichlorethylene inhalation (30 drops on a handkerchief three times a day.) He states, however, that prolonged use causes toxicity in the patient and therefore discourages this form of therapy.

Wells[24] spoke of procaine injections into the trigger zones as a fair palliative procedure in trigeminal neuralgia. However, the patient must be anesthesized in order to perform the injection, and the procaine remains effective for only short periods. He has used blocking of the peripheral nerve as an aid in diagnostic procedure. The blocking agent used was procaine.

Frank[25] in his article in 1921 states that Schodosser in 1900 first demonstrated the injection of 80% alcohol into the sensory foot. It was noticed that at this time following injection there was a period of numbness and gradual return of normal sensation with pain for a period of time from 6 to 24 months until the pain returned. Each of the divisions could be injected separately although injection of the first is seldom attempted, due to its location in respect to the blood vessels, motor nerve and the optic nerve of the eye. Complications which may follow alcoholic injections are keratitis especially after the first and second divisions have been injected, paralysis of the sixth nerve, hematoma and ecchymosis, stiff jaw and occasionally injecting of the alcohol into the nasal pharynx. Neurosurgeons of recent years have injected the semilunar ganglia itself primarily while using alcohol therapy even though in 25% of their cases corneal ulcers appeared.

The x-ray has been a big aid in locating the semilunar ganglia, by means of the radiographic fluoroscopic injection techniques and x-ray films. The most common films that are taken are as follows:

1. Axial position which is used to demonstrate the sphenoid sinus. This gives a reasonable and accurate idea of the size, shape and position of foramen ovale.
2. Oblique steroscopic to show the middle cranial fossa on the affected side and thus the surgeon has valuable information as to the general contours of the region.

Clark[23] describes the procedure for the alcohol injection which is as follows: the skin of the cheek is anesthesized with novocain immediately below the tubercle on the lower margin of the molar process on the maxilla. The bevel of the needle after being introduced below the skin is directed upwards and inwards, and the needle is pushed upwards and backwards at the angle of 32° to Reid base line, and inward at an angle of 32° to the sagittal plane. At this point the tip of the needle impinges on the base of the pterygoid process of the sphenoid bone, and the bevelled surface causes the point to slip backwards towards the foramen ovale. At the site the needle is withdrawn very slightly and its proximal end is depressed about one centimeter. It is then advanced one centimeter, and the point of the needle lies well within the ganglia and parallel to the long axis of the ganglia. It is here that the alcohol is injected. The duration of effectiveness of this

therapy ranges from six to eight months. Approximately 25% of these cases have corneal ulcers.

Stilbamidine is the latest form of medical therapy used in trigeminal neuralgia. This has been used primarily by Smith and Miller[26] of John's-Hopkins. They report on 16 cases treated with stilbamidine with excellent results in 15 and good results in one. The corneal reflexes were preserved in all patients. The treatment is effective, safe and inexpensive. Procedures are:

1. One hundred fifty milligrams of stilbamidine is dissolved in 200 cc of 5% glucose solution and given slowly intravenously each day for 14 days.
2. The patient is placed on a low protein diet.

Contraindications for this form of therapy are any renal or hepatic dysfunction. The pharmacology of this drug is that is acts to produce a section of the nerve by neurotoxic means. It has also been used in the treatment of multiple myeloma, blastomycosis, and Indian Kala-azar with the apparent neurotoxic properties as side effects.

SURGICAL THERAPY

The surgical means of therapy are of five varieties:

1. Peripheral Avulsion.
2. Decompression of Semilunar Ganglia.
3. Trigeminal Rhizotomy by Transtemporal Route.
4. Suboccipital Route Section.
5. Intramedullary Tractotomy.

Grantham and Sergenberg[27] feel that peripheral avulsion is an excellent palliative procedure for the alleviation of pain in trigeminal neuralgia, and has an important place in the proper care of the aged patient in a debiliated state, and on whom a craniotomy would be haphazardous. In the literature alcohol injection has received far greater attention than nerve avulsion, which is simpler and gives more effective relief. This is particularly true if neuralgia involves the first, or the second, or both of the divisions of the trigeminal nerve. Alcohol injection should be reserved for those cases in which the pain is confined to the third division. An analysis of 109 cases in which palliative procedures were done shows that the average relief from the supra and infraorbital avulsion was 33.2 months, whereas that from alcohol injection was 15.2 months. The complications most commonly seen with peripheral avulsion are corneal ulcers in 25% of the patients who had had the ophthalmic branch incised, facial weakness, and loss of tactile sensation in a relatively high percentage.

Decompression of the semilunar ganglia as a surgical means of treatment of tic douloureux is based on the idea that compression of the ganglia causes the tic douloureux pain. Taarhoj[28] first advocated this decompression by means of a transdural approach, of dividing the dural sheath enclosing the root and ganglia, without touching the nerve itself, as relief of trigeminal neuralgia without numbness. Love[29] modified the procedure of Taarhoj to an extradural approach in which the dura was not incised until the sensory root was exposed. This approach adds to the safety of the operation, and also has a lower incidence of mild postoperative aphasia. A more complete summary of this extradural approach is given by Cleveland[30] and it is as follows: expose the lateral half of the sheath of the ganglion and then incise the dura propria lengthwise to the dura fold, at the petrosal ridge. Just lateral to this fold, the dura is incised under the temporal lobe for a direction of about one-fourth inch, and then, after careful inspection of the intradural space and the opening occupied by the posterior root, hemostats are placed on the dural fold, and the dural fold is divided between them.

The advantage of semilunar decompression is the complete relief of pain, and the complications are relatively none. The results of this new form of surgical therapy are stated by Taarhoj who reports on ten cases with excellent results in all; and by Love who has just had one case to date with his modification also with excellent results.

Trigeminal rhizotomy by the transtemporal route was first mentioned by Frazier,[31, 32, 33] who is one of the early prominent names connected with trigeminal neuralgia. The procedure for his operation, an intra-cranial transtemporal approach is as follows: this operation consists of the removal of a small portion of the squamous temporal bone through a straight temporal incision, and the elevation of the dura off the floor of the skull to expose the ganglion. The sheath of the ganglion is then opened over the posterior root and all or part of the root is divided. If the second or third divisions are alone involved, it is advised to leave the ophthalmic division intact because of the danger of keratitis.

Of great importance to neurosurgeons is the significant variations in the location and trifurcation of the semilunar ganglion. Gass and Van Wagner[34] found variations in dimensions of the semilunar ganglion and its roots, as much as 1 cm., especially in the anterio-posterior direction, in 32 fresh postmortem dissections. The points where the ganglion trifurcates into its roots, may be thus relatively far forward in one instance, or far posterior in another. In the female it appears that one might expect a more anteriorly placed ganglia trifurcation, but in the men a more posterior trifurcation would be more apt the rule. In the surgical exposure of the ganglion and its posterior root, by the temporal route, the surgeon frequently finds it difficult to obtain wide visualization of all the structures. In view of the aforementioned variations, with minimal operative exposure, the operator subjects himself to

possible errors in obtaining good differential section of the posterior root. It is suggested, therefore, that before cutting what he takes to be the posterior root, the surgeon strive to obtain visualization of these structures for not less than 1 cm. in the anterio-posterior direction.

Peet and Schneider[35] after review of their 689 cases along with Guidette[36] feel that trigeminal rhizotomy is a much more satisfactory procedure than alcohol injection in the average case of trigeminal neuralgia. There has been a complete relief of pain in all but 5.4% of the patients operated upon. Alcohol injection on the other hand in their series revealed only 15.2% of the patients with relief of pain for a period longer than one year. Other advantages are the low mortality attributed to surgery and the relatively low instance in re-occurrence.

The incidence of complaints of crawling paresthesias 56%, of burning paresthesias 30%, and of numbness 4% among the postoperative rhozotomy patients is much higher than that frequently cited, and probably approximates the percentages of those complaints after alcohol injections.

Dandy[37] in 1925 first advocated the suboccipital root section because of the ease of performance and the safeness of the operation before the major advantages became apparent. His procedure for this suboccipital root section is as follows: a crescent-shaped incision is made in the occipital region on the affected side. The trapezius muscle is divided and stripped; following this an area

of bone 4 x 4 cm. is removed. The dura is then incised in stellate fashion and at once the cisterna magna is sought and opened. The release of this fluid provides ample room for exploration. The cerebellar hemisphere is then elevated with a narrow spatula directed inward and upward. The thin membrane covering the cisterna lateralis, which extends the entire length of the posterior fossa, and lines the brain stem, is opened between the auditory nerve and the tentorium. After the collapse of the cisterna lateralis and removal of the loose arachnoid membrane between the auditory nerve and the tentorium, the sensory root of the trigeminus stands out sharply in depth. At the incisura tentorii the petrosal vein crosses from the inferior surface of the cerebellum to the petrosal sinus. It lies in and is attached to the outer lining of the cisterna lateralis. The arachnoid membrane must be therefore cautiously removed from the vein to avoid tearing.

The petrosal vein and auditory nerve are the two most important landmarks, and between them--they are approximately 1 to 1.5 cm. apart--the spatula is introduced. The sensory root of the trigeminus is then in full view throughout its course from the tentorium to the pons, a span of 1 to 1.5 cm. The sensory root lies probably 1 cm. deeper than the petrosal vein. A small blunt dissector is passed at any angle between the sensory root, and the pons, in order to free the nerve. A small angled knife also on a long flexible shank then follows up the free space between the nerve and the pons and by gentle traction on the blade of the knife

the nerve is severed. Section of the nerve is usually bloodless. Occasionally a tiny bleeding occurs and it usually is suppressed by application of moist cotton pledgets. Advantages of this operation are:

1. Ease in performance and safeness of procedure.
2. Absence of corneal keratitis because corneal reflexes remain intact.
3. Facial weakness fails to occur.
4. Paresis of the masseter muscle seldom occurs.

Complications:

1. Slight paresthesias of the face.
2. Slight loss of tactile sensation.
3. Higher mortality than in previous operations.
4. Greater frequency of recurrence.

Results of this type operation were reported by Dandy in 28 cases of total section of the sensory portion of the nerve with good results and a relatively few complications. His conclusions on partial section of the sensory nerve of the trigeminus on 23 cases are hard to evaluate due to the difference in degree of partial separation of the nerve.

Intramedullary tractotomy is one of the most recent surgical developments in the treatment of tic douloureux. The procedure was first described by Sjeqvist[38] in 1938. His procedure is as follows: a unilateral suboccipital craniectomy is performed with removal of the posterior rim of the foramen magnum and the arch of

the atlas. After the dura is opened the arachnoid membrane of the cisterna magna is torn and the cerebrospinal fluid is allowed to escape. The tonsil of the cerebellum is then gently retracted until the lateral aspect of the medulla is visualized, and thence, the identifying of the lower most vagal rootlet which is the site of the incision. This lies approximately at the level of 8-12 mm. above the opex of the fourth ventricle. The depth of the incision is 3-4 mm. on the lateral aspect. The advantages of this procedure are:

1. Touch sensation is spared and the face would not feel cold, stiff and numb as after a section of a sensory root.
2. Sparing of the motor components.
3. Elimination of a neuroparalytic keratitis since some sensation of the eye is retained.

Grant and Wineberger[39, 41] and Raney[40] differ with the Sjeqvist procedure because of the neurological disturbances encountered following surgery. These disturbances are attributed to injury of the restiform body or the cuneate nuclei. Most common complications of Sjoqvist's procedure are as follows:

1. Ataxia of the homolateral arm.
2. Numbness of the opposite body.
3. Paralysis of recurrent laryngeal nerve.

Grant's modification on 17 cases has the advantages of Sjoqvist, but none of his complications. Grant's procedure is as follows: he uses the obex of the fourth ventricle and the olive as land marks and he incises the tuberculum cinereum at a level of 4 mm. below

the obex, and about 2 mm. below the olive, and to a depth of 3 mm. This site is 12-14 mm. more caudal than Sjoqvist and no major neurological disturbances have been encountered postoperatively. The incision at this level fails to injure the restiform body or cuneate nuclei and at a depth of 3-4 mm., it will encompass all the fibers of the trigeminal tract and assures adequate facial analgesia. Occasional minor or academic disturbances may occur following this procedure and they are as follows:

1. Occasional lurching.
2. Some difficulty in walking stairs.
3. Slight rearing of gait to homolateral side.

PROGNOSIS

Trigeminal neuralgia is not spoken of as a killing disease, however, its victims may resort to suicide as a means of cure. Spontaneous recoveries from trigeminal neuralgia are extremely rare. The interval between attacks of pain may be long with remissions lasting months or even years. As a rule, however, once disorder is established, attacks tend to follow each other fairly frequently, and the interval between them tends to become shorter. Finally, there may be many attacks during the day. In a sense the prognosis of each case of trigeminal neuralgia depends upon the severity of the case, age of the patient, and the means of treatment that is undertaken in it.

SUMMARY

Trigeminal neuralgia is a disease which has afflicted people since the earliest times; and as yet the cause is unknown. Therapy does offer some hope however.

While the etiology is obscure, it is known that most cases of trigeminal neuralgia have some sort of a trigger zone, located around the nose, mouth or teeth. Some of the most frequent mentioned possibilities for the etiology are as follows:

1. Arteriosclerosis of cranial artery - applying pressure to sensory fiber of the cranial V nerve near the medulla oblongata - 80%.
2. Tumor - in medulla oblongata - 5%.
3. Aberrant artery - in medulla oblongata - small percent.
4. Artificial synapse - of nerve fibers due to: - small percent.
   A. Compression of nerves together by sagging tentorium.
   B. Compression of nerves together by dura.
5. Type of thalamic syndrome - small percent.
6. Virus (?) - small percent.

The medical treatment of this disease has had many forms, such as cuprelone, ferrous carbonate, antihistamine, procaine injections, alcohol injections, vitamin B_{12} and stilbamidine. Only alcoholic injections and stilbamidine offer a high percentage of relief. With alcoholic injections, the semilunar ganglia is blocked there giving the patient relief from his pain for a year or more - a palliative measure. Stilbamidine, a new drug of Merrill Pharamocological Co.,

offers aid by a neurotoxic tractotomy of the fifth cranial nerve. There are several aspects about the side effects of this drug which are still in the experimental stage.

Surgery seems to offer the only real cure for this disease. There are five major types of surgical procedures:

1. Peripheral avulsion - very valuable as a palliative process.
2. Decompression of semilunar ganglia - questionable results (etiology of trigeminal neuralgia must be compression on the semilunar ganglia.)
3. Trigeminal rhizotomy by Frazier transtemporal route - good procedure - however side effects undesirable.
4. Dandy suboccipital nerve section - good technique, but it has become outdated by intramedullary tractotomy which has its good effects but none of its bad ones.
5. Intramedullary tractotomy - with section of the sensory fibers of five 4 mm. below the obex of the fourth.ventricle. This gives complete permanent relief of pain in almost all cases with very minor if any side effects or complications.

This operation is the new modern advance in attempting to cure trigeminal neuralgia.

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