

## THE NEURALGIAS<sup>1</sup>

VARIS ANDERSONS<sup>2</sup> AND KELLY HYSLOP<sup>3</sup>

*Halifax, Nova Scotia*

### INTRODUCTION

The purpose of this paper is to present a concise review of the more common aspects of Craniofacial Pain. Included is a Table I listing the gammit of conditions that are responsible for this symptom. It is beyond the scope of this paper to elaborate on them all in detail so the discussion will be limited to Trigeminal Neuralgia, Glossopharyngeal Neuralgia, Occipital Neuralgia and Primary Atypical Facial Pain.

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### CLASSIFICATION OF CRANIOFACIAL NEURALGIAS

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- I. MAJOR NEURALGIAS
  - A. Trigeminal Neuralgia
  - B. Glossopharyngeal Neuralgia (including tympanic plexus neuralgia)
- II. MINOR NEURALGIAS
  - A. Occipital Neuralgia
  - B. Geniculate Neuralgia (including Chorda Tympani neuralgia)
  - C. Vagus Nerve Neuralgias (superior Laryngeal, recurrent laryngeal and auriculo temporal varieties)
- III. POSTHERPETIC
  - A. Ramsy-Hunt Syndrome
  - B. Herpes Zoster Ophthalmicus with supraorbital neuralgia
- IV. SYNDROMES OF VASCULAR ORIGIN
  - A. Migrainous Neuralgia
  - B. Sphenopalatine Ganglion Neuralgia
  - C. Vidian Neuralgia
  - D. Petrosal Neuralgia
- V. PRIMARY ATYPICAL FACIAL PAIN (NEURALGIA)
- VI. SYNDROMES RESULTING FROM REFERRED OR REFLEX PAIN
  - A. Dental Diseases e.g. Aerodontalgia, Pulpitis, Periodontitis
  - B. Ocular Diseases e.g. Acute Glaucoma
  - C. ENT Diseases e.g. Otitis, Sinusitis
  - D. T.M.J. Disease (Costen's Syndrome)
  - E. Musculo-Skeletal Disorders of Head & Neck e.g. Cervical Spondylosis
  - F. Angina Pectoris (rarely)
- VII. PAINFUL SYNDROMES RESULTING FROM LOCAL PATHOLOGY (INTRA OR EXTRA-CRANIAL)
  - A. Infections e.g. Petrositis, Dental Caries
  - B. Tumors e.g. Nasopharyngeal
  - C. Vascular e.g. Aneurysms, temporal arteritis, histaminic cephalgia
  - D. Traumatic e.g. Fractures of middle fossa
  - E. Neurologic e.g. Arachnoiditis, lues, syringobulbia
- VIII. RARE AND ILL DEFINED ENTITIES
  - A. Trigeminal Ghosts
  - B. Glossodynia
  - C. Painful tic convulsif
  - D. Myalgia of Pharynx
  - E. Intermittent Claudication of Jaw Muscles
  - F. Systemic Disorders with Trigeminal Neuralgia

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Table I taken from Baker, A. B., (editor) *Clinical Neurology*, Vol. 4, page 1898.

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<sup>1</sup>References will be supplied upon request to the authors.

<sup>2</sup>4th Year Medicine, Dalhousie.

<sup>3</sup>3rd Year Medicine, Dalhousie.

### CREDITS

The authors would like to thank Dr. H. N. A. MacDonald, Dr. H. H. Tucker, and Dr. A. J. Lewis for their kind assistance in the preparation of this paper.

## TRIGEMINAL NEURALGIA

**ANATOMY:** The sensory nucleus of the V cranial nerve is a continuation of the spinal cord's substantia gelatinosa. It extends through the medulla and pons to the level of the superior colliculus in the mesencephalon. This portion of the V nerve nucleus contains proprioception primary neurons. All the other V nerve primary sensory somas are located in the gasserion ganglion. The motor nucleus is located medially to the sensory nucleus.

The fibres of the gasserion ganglion leave the brain stem at the level of the mid pons. The gasserion ganglion is positioned 4.5 to 5 cm. deep to the lateral aspect of the head, slightly anterior to the preauricular point and in the dura near the axis of the petrous temporal bone. Medially related to the gasserion ganglion is the posterior part of the cavernous sinus and the internal carotid artery. The axis cylinders of the trigeminal ganglion divide into three branches. The ophthalmic V<sub>1</sub>, the maxillary V<sub>2</sub>, and the mandibular V<sub>3</sub> branches. The sensory distribution of the trigeminal nerve may be found in any standard text.

**PHYSIOLOGY:** The cause of the pain is still not clear. For the pain to occur it is necessary to have at least two physiological mechanisms. Firstly, there must be a normal sensory input to the gasserion ganglion, because the pain is prevented with a peripheral nerve block. Secondly, it appears that it is necessary to have a malfunction of the Gasserion Ganglion or of the V nerve nucleus. It is a physiological problem in explaining the intermittent nature of the pain. Sheldon (1966) suggests there may be a central inhibition which is triggered by the pain itself which causes a cessation of the neuralgia, but whatever the physiological mechanism is, it must be remembered that there is definite pathology of the gasserion ganglion and it is therefore reasonable to expect this to be the cause of the trouble.

**AETIOLOGY:** The disease generally starts between the ages of 50 and 65, however 9% of the onsets occur before the age of 40, and

1% before 30 years of age. Males are affected slightly less frequently than females and the right side of the face is more commonly affected than the left. - (Stookey & Ransohoff 1959). Beaver *et al* (1965) described micro neuromas in 100% of the patients examined. Harris (1940) in a case study of 1433 patients showed heredity to be involved in 30 of these cases. In the same study Harris found a 3.48% association with multiple sclerosis. Stookey & Ransohoff (1959) in a study of 728 cases had an association of .68%. In all of Stookey's cases there were sclerotic plaques at the entry zone of the trigeminal root to the brain stem. Parker (1928) considered that the area may be particularly allogenetic and pointed out that the initial lesion in tabes of the C.N.S. is at the point of entrance of the posterior roots into the dorsal superior columns and this disease is also associated with lightning stabs of pain.

Herpes zoster has also been shown to be associated with facial pain in the V nerve distribution, however the occurrence of Herpes in the V cranial nerve is 95% in V<sub>1</sub> with 5% in V<sub>2</sub> and V<sub>3</sub>, whereas the ratio in trigeminal neuralgia is reversed. - (Stookey & Ransohoff 1959). This finding is hard to understand except that there is a separate phylogenetic origin of V<sub>1</sub> from the other two divisions of the V nerve. The post herpetic pain that the occasional patient does develop is not a true trigeminal neuralgia according to the definition. This post herpetic pain is constant and unremitting for days. Macroscopic trigeminal tumors may also produce pain but this again is not the general characteristic trigeminal neuralgic pain.

**PATHOLOGY:** Previous to the advent of the electron microscope no pathology of the trigeminal neuralgic patient was known. In 1965 Beaver, Moses, and Ganote demonstrated, in electronmicrographs of biopsied gasserion ganglions, irregular vacuolations of the cells, but with normal mitochondria and Golgi apparatus. The myelin sheath was extremely thin in places but also had areas of disorganized hypermyelination. The axis cylinders were often hypertrophic, tortuous and resembled plexiform micro-neuromas.

These changes occurred in all the gasserion ganglion of the neuralgic patients that Beaver *et al* examined.

**CLINICAL ASPECTS: I. Symptoms.** Pain is the only symptom of Trigeminal Neuralgia and its adequate description determines the diagnosis of the condition.

(a) Location - The ophthalmic division is least often involved, varying in different series between 2 - 5%. It is only when the pain is in the forehead directly over the eye or in the eye itself, that one may be sure it is the ophthalmic branch that is involved.

By far the commonest affliction is either in the maxillary or mandibular divisions or in both simultaneously. When the maxillary branch is affected, the pain may be located in the upper lip, ala nasi, cheek and less frequently the upper gingiva or palate. If the symptoms are those of mandibular involvement, the lower lip and less often the lower jaw and gingiva are affected.

Bilateral Trigeminal Neuralgia has been described, but even in these cases the pain on one side predominates. It is possible, however, for the pain from one division to spread and involve another; the pain never crosses the midline.

(b) Quality - All manner of adjectives are used to describe the pain, with the express purpose of portraying its severity and occurrence in paroxysms, e.g. lightning jabs, searing stabs, etc. The patient may be symptom-free one minute and the next be suffering from the most excruciating pain. The paroxysms of pain may last for less than one minute up to several minutes and they may occur at any time of day. Sleep is not commonly interrupted by the pain except late in the course of the disease. Temporal summation of stimuli is thought to occur, so that each paroxysm may be followed by a very brief refractory period.

(c) Trigger Zones - These are areas, the stimulation of which, no matter how slight, brings on the characteristic painful episodes. The pain may be brought on by facial movements, chewing, brushing teeth or even the slightest contact such as a draft of wind. These zones are generally well within the distribution of the affected branch or branches of the trigeminal nerve such as the lower or upper lip, alae nasi or eyebrow. Occasionally attacks are precipitated during periods of acute emotional upset. If present, trigger zones are highly suggestive of the diagnosis of trigeminal neuralgia.

**II. Signs** The patient may appear appre-

hensive and undernourished (for fear of provoking an attack by eating). Also because of this incessant fear, the fact may be somewhat expressionless, one side of the face may be unwashed or unshaven, and the teeth on the involved side may be uncleaned or the tongue may be coated. Another feature is that usually, when describing the area of involvement, the patient does not touch it. If seen during a paroxysm, the agonizing quality of the pain is manifest in the patient's expression.

Upon examination of the Trigeminal nerve, jaw motility, the corneal reflex and facial sensibility are all intact (unless the patient has had a previous alcohol injection).

Laboratory and X-ray examinations are all within normal limits.

**III. Diagnosis** The typical description of the pain in combination with the finding of trigger zones should exclude the other causes of craniofacial pain as listed in Table I. Clinically, other diseases may be coexistent with Trigeminal Neuralgia and although the aetiological significance of this fact may be disputed, these diseases should still be considered. In many series where the primary diagnosis has been that of Trigeminal Neuralgia, the coincidence of Multiple Sclerosis has been variously estimated at 0.68 to 3.48 per cent. Some systemic conditions that have been found to have a clinical association with Trigeminal Neuralgia are: Infections such as Typhoid, Syphilis, malaria, as well as influenza and herpes simplex viruses; Metabolic Disorders such as diabetes and gout; and Toxic Disorders such as alcohol, lead poisoning and digitalis intoxication.

**IV. Prognosis** The course of this disease is characterized by remissions and exacerbations. The symptom-free periods tend to be shorter as the disease progresses. Total remissions without treatment are rare. Although the disease is not fatal in itself, it is extremely incapacitating and may lead to a certain degree of psychological trauma and even suicide in some cases.

**MEDICAL TREATMENT:** *Trichlorethylene* was one of the first drugs used for the treatment of Trigeminal Neuralgia. It was discovered by an accident in World War I when it was used as a cleaning fluid by the Germans. Four men inhaled the fumes of the drug and after an acute episode of vomiting, vertigo and V nerve sensory loss, the men

were left with chronic sensory loss of cranial nerve V.

*Stilbamidine* was used to cause V nerve neuropathy. This drug has been used successfully by the removal of the sensitivity of the trigger zones and thereby decreasing the occurrence of the neuralgia. (Woodhall and Odom, 1955).

*Hydantoins*, Le Moyné (1951) treated 17 patients and cured eight using *Dimethyldiohydantoin*. Jensen (1954) using *Diphenylhydantoin* with 36 patients cured 16 and improved 15. *Dilantin* has also been used by Schaltenbrandt (1957). The greatest drawback to these drugs is that in many cases the required dose is higher than the toxic dose, but these drugs are very useful as a synergistic agent with other drugs e.g. Tegretol (see below).

Borsook (1940) treated 58 patients with *Vitamin B* and found a marked improvement in 37 patients, some improvement in 15, slight improvement in 3 and in the remaining 3 patients, no change at all. Fields & Hooff (1952) and Davis (1953) both consider large doses are useful in a high percentage of cases but Stookey and Ransohoff (1959) thinks that *Vitamin B*<sup>12</sup> is only useful in mild attacks of neuralgia.

*X-Ray Therapy* was used with limited success as early as Grocht in 1897. Many authors have reported the use of radiation therapy but it seldom gives permanent relief.

*Tegretol* (5-carbamylidibenz(b, f)azepine) (G. 32883) is still in its clinical investigation stage of development. The drug is an aminostilbene derivative first synthesized by Schindler in the Geigy Laboratories. It has the characteristic properties of a potent anti-convulsant and a pronounced inhibition of polysynaptic somatic reflexes, though no anaesthetic or tranquilizing effect. The drug has a low toxicity level and a high or favourable therapeutic index. Geigy Pharmaceuticals (1964) in experimental animals found there are no striking effects with a dose up to 500 mg/Kg. With increasing doses greater than 1000 mg/Kg the first disturbances noted were in coordination, then stupor and hyperextension. In larger doses abdomino-lateral positioning and increased respirations, opisthotonus spasms and clonic convulsions were noted in that order. The chief effect is anti-convulsant with a therapeutic index in the electric shock of the rat, over 150. This

exceeds the commonly used anti-epileptics. The site of action is probably in the spinal cord because its effect is greater on cord acting drugs like strychnine and subsequently less effect with liptazol, picrotoxin and amphetamine in that order. These drugs have a more central action. Mono-synaptic reflexes are affected very little, but poly-synaptic reflexes tend to be inhibited.

Clinical Experience with Tegretol shows it to be a very good anti-epileptic for the grand mal type of seizures and occasionally is successful in treating status epilepticus. The reported side effects of the drug include somnolence, headaches, giddiness, ataxia, nausea, vomiting, anorexia, rashes, ocular disturbances, and diarrhea. By 1964 two cases of aplastic anemia had been reported. Therefore routine blood counts must be taken at the start of treatment. Tic Douloureux was treated with Tegretol by Blom (1963). Forty patients were treated with G.32883. Of these 36 were relieved within 24 hours and 3 more patients responded satisfactorily when Diphenylhydantoin was added to the treatment as well. Eleven of the patients discontinued treatment with the belief that they were cured and all experienced a return of the neuralgia within twenty-four hours.

J. D. Spillane (1964) gave Tegretol to 52 patients. The effect was that 26 patients were relieved within 24 hours and 16 patients had reduced paroxysms in 48 hours. There was a slight improvement in 5 patients and 5 received no benefit from the drug at all. The subsequent effect (in one to fifteen months in 50 patients was 28 more or less free of pain. 14 patients were free of spontaneous attacks but had some residual trigger-zone pain remaining, 6 patients suffered relapses and 2 developed drug intolerance such as giddiness, nausea and vomiting. With experience in the use of the drug, treatment was started more cautiously and the side-effects were reduced.

J. O. Taylor (1963) treated 52 patients with G.32883 and 44 responded well. Ten of these patients experienced total abolition of symptoms which recurred in all when they stopped taking the drug. The remaining 34 patients had modified paroxysms described as twinges, jumps or pin pricks. Only 17 patients have been completely free of side effects. Drowsiness, Nausea, dry mouth, headache, and unsteadiness of gait are the commonest side effects, but they can be de-

creased with a careful adjustment of the dosage. Occasionally a compromise is necessary between the therapeutic effect of the drug and its side effects. There were 8 patients of the 52 who were classed as failures. Four patients got skin reactions which necessitated discontinuance of the drug. One patient got no relief at all, just side effects, and the remaining 3 were free from pain and then quite suddenly the drug became ineffective.

Campbell, F. G. et al, (1966) tested Tegretol against a placebo and found the drug to be much more effective.

It must be stressed, however, that these statistics have only been compiled over a relatively short period of time and that it will take more time to obtain a more accurate assessment of the effectiveness of this drug, Tegretol.

**SURGICAL TREATMENT:** The surgery for Trigeminal Neuralgia may be divided broadly into two main categories; those of extracranial and intracranial approach.

**I. Extracranial (1) Nerve Injection:** is effected with absolute alcohol or phenol in glycerin. This type of treatment is directed at the peripheral branches of the Trigeminal Nerve such as the supraorbital, infraorbital, maxillary, and mandibular nerves as well as the Gasserian Ganglion. The advantages are that it may be performed on a patient that is a poor surgical risk and after the relief of pain by this method the patient may be more inclined to accept surgical treatment later. The relief of pain by blocking the nerve also serves as a diagnostic tool for the physician. The disadvantages are that if performed under local anaesthesia it is an extremely trying experience for both doctor and patient (especially ganglion injection). The relief is by and large temporary (3 mos. to 3 years). Also the total numbness experienced after Gasserian Ganglion injection may be more unpleasant to the patient than the paroxysms of pain. Finally, multiple cranial nerve palsies may result from the spilling of alcohol into the subarachnoid space.

(2) *Avulsion or Section of Peripheral Branches:* If the peripheral branches of the Trigeminal Nerve appear to be involved, and if relief results from alcohol injection, more permanent relief may be afforded by avulsion or section of the supraorbital, infraorbital or mental nerves.

**II. Intracranial (1) Total or Subtotal Trigeminal Rhizotomy (Middle Fossa Approach):** This operation was first instituted by Spiller & Frazier (1901) and twenty years later modified by Frazier to what is probably the most adhered-to intracranial method used today. A subtemporal approach with partial section of the postganglionic fibers is done in Meckel's Cave. The second and third divisions are severed and the first division and motor root are spared. Thus Keratitis and paralysis of the muscles of mastication are avoided. This procedure is not without complications, however. The most common sequela is paresis and numbness over the anaesthetic area. Others include herpes zoster infection, transient facial paralysis, trophic lesions of the skin and mucous membranes, deafness and Sixth Nerve paralysis, and post-operative infection and hemorrhage. Also keratitis, corneal ulceration and paralysis of the muscles of mastication may be seen.

(2) *Subtotal Trigeminal Rhizotomy (Posterior Fossa Approach):* This procedure was first advocated by Dandy in 1925. Although technically more difficult, it has the advantage of better differential section of the sensory root at the pons and it affords the surgeon the ability to visualize and treat posterior fossa pathological lesions such as tumors or aneurysms which may give a clinical picture typical of Trigeminal Neuralgia.

(3) *Compression and Decompression Operations:* have been advocated by Taarnhøj. These operations may be put in the same category as Gangliolysis (stripping of Dura and Arachnoid mater overlying the ganglion), manipulation of the ganglion and separation of the fibers of the root. The results are not permanent and it is the opinion of most authorities that the relief from pain that may be afforded by these various procedures is the direct result of the mechanical trauma to the nerve during the operation.

(4) *Bulbar Tractotomy:* was first advocated by Sjöqvist in 1938. In this procedure the fibers of the descending part of the Trigeminal Nucleus are sectioned in the medulla. This is of course a more dangerous procedure than the previous ones because of the risk of damaging vital brain stem structures as well as the cerebellar pathways. Its appeal lies in the fact that the incidence of facial anaesthesia and paresthesias is considerably less. For example, it would be useful in a case where

a trigeminal rhizotomy was performed on one side and the patient subsequently developed pain on the other side. If this method were employed, massive facial anaesthesia would not result.

(5) *Electrical Method:* Recently, C. H. Shelden of Pasadena, California has advocated a method of electrically inducing a refractory state in a portion of the peripheral trigeminal fibers and thereby reducing the total volume of afferent impulses reaching the brain stem. A receiving unit is implanted in the skull and connected by fine platinum wires to the nerve just distal to the ganglion. During a paroxysm of pain, power is generated to the receiver from a transmitter which is at present in the form of a transistor oscillator from a transmitter which is at present in the form of a transistor oscillator in a flashlight case. This unit is held superficially over the skull. The selection for this type of procedure includes a patient in his late forties, having daily frequent severe paroxysms of pain and definite sensitive trigger areas, and the pain being limited to the third division. It is also necessary for the patient to be familiar with electrical theory.

## GLOSSOPHARYNGEAL

**AETIOLOGY:** The aetiology is unknown. So far as is known there have been no electron microscope studies of either of the IX nerve ganglia. It would be reasonable to expect to find micro-neuromas as have been found in the gasserian ganglion in Trigeminal Neuralgia. Males have a slightly greater incidence than females with an overall occurrence of 1.5% of Trigeminal Neuralgia, and 1/3 of these have both complaints. (Henderson, 1967)

**ANATOMY:** The tractus solitarius is the sensory nucleus for the IX cranial nerve and it is situated in the medulla oblongata between the level of the sensory decussation and the inferior level of the open medulla. The nucleus solitarius is positioned in the lateral aspect of the central grey matter just dorsal to the level of the central canal in the medulla, but moves gradually laterally as it ascends into the open medulla. The nucleus receives afferent fibers from the facial glosso-

pharyngeal and vagus nerves in that order from the above downwards. The nerve leaves the upper part of the medulla in three or four filaments in the line of the posterior nerve routes between the inferior cerebellar peduncle and the olive. The nerve courses anteriorly and laterally under the flocculus towards the jugular foramen through which it leaves the skull. There are two ganglia of the IX nerve situated within this foramen. The superior ganglion is generally considered to be a detached portion of the inferior ganglion and only the latter gives off branches. These branches convey taste and general sensibility from the posterior third of the tongue including the vallate papilla, sulcus terminalis and the mucus of the pharynx oropharyngeal isthmus eustachian tube and soft palate. There are also connections with the vagus nerve to its auricular branch and superior ganglion, and also to the sympathetic superior cervical ganglion of the facial nerve.

**CLINICALLY:** The character of the pain is very similar to that of Trigeminal Neuralgia - a sharp lancinating pain occurring in paroxysms. The distribution is of course different. The ear, external auditory meatus, pharynx, tonsillar area, posterior aspect of the tongue and even the angle of the mandible may be affected. Trigger zones are again a fairly common phenomenon and are located in the area of distribution of the pain. Attacks may occur spontaneously, but may also be incited by swallowing, coughing, yawning or talking.

During an attack the patient may refrain from swallowing saliva and therefore voluntarily "drool". In combination with refusal to swallow liquids, this may be a cause of dehydration. Another, but rarer, set of complications are due to central overflow of impulses into the carotid sinus regulatory system. Bradycardia, cardiac arrest, hypotension, syncope and convulsions result and this always occurs during an attack of pain. Treatment consists of i.v. atropine 0.0012 gm. Atropine has no effect on the pain, however.

**DIAGNOSIS:** One must exclude local pathology in the oropharynx - either inflammatory or neoplastic. There may also be coexistent Trigeminal Neuralgia. Sometimes there is difficulty in distinguishing Mandibular Branch Trigeminal Neuralgia, Geniculate Neuralgia

or the Vagal Neuralgias from Glossopharyngeal Neuralgia. Here one may anaesthetize the pharynx, tonsillar area and posterior aspect of the tongue with Cocaine or Pontocaine. Temporary abolition of symptoms implies a Glossopharyngeal etiology. It must be stated however, that this procedure is not without limitation and that accurate history taking should not be sacrificed for it. Finally, one must exclude intracranial conditions such as aneurysms and posterior fossa tumors.

**PROGNOSIS:** The onset is usually in middle life and the natural history is progression to a peak in severity and then self-limitation.

**SURGICAL TREATMENT:** Surgical Treatment is warranted only if the symptoms are extremely severe, which is a matter of judgment for the neurosurgeon involved. By and large an intracranial approach is used and the procedure consists of sectioning the Glossopharyngeal fibers proximal to the ganglion as well as dividing the superior 2 rootlets of the vagus. The latter part of the procedure is especially indicated if there is a trigger zone in the external auditory meatus. Extracranial approach, although more difficult may be warranted in old age, debility, or a patient with obvious cardiac disease. (Bailey 1955) Postoperatively (in the case of the intracranial approach) the sensory loss and short-term regurgitation of liquids are minor complications when one considers the facility and the permanence of the relief.

## GREATER OCCIPITAL

**ANATOMY:** The greater occipital nerve is made up predominantly from the posterior primary ramus of C<sup>2</sup> although there are also contributory branches from the posterior rami of C<sup>1</sup> and C<sup>3</sup>. The posterior ramus of C<sup>2</sup> is much larger than the anterior ramus which it leaves just distal to the dorsal spinal ganglion and emerges between the posterior arch of the atlas and the lamina of the axis under the inferior oblique muscle. The nerve curls round this muscle and goes rostrally under the semi spinalis capitis almost as far as its attachment to the skull. Here the nerve comes superficially through this muscle and ascends with the occipital artery to supply the scalp as far forward as the vertex of the skull.

**AETIOLOGY:** The aetiology is unknown.

**CLINICALLY:** The symptom is that of pain radiating from the suboccipital or cervical areas forward into the parietal, post auricular, temporal and even orbito-facial areas.

The signs may include tenderness along the course of the greater and lesser occipital nerves, some sensory changes over the posterior aspect of the scalp and limitation of neck movement.

**DIAGNOSIS:** There is still considerable dispute among authorities as to whether or not Primary Occipital Neuralgia exists as a distinct entity. It is known to exist secondarily to muscular, vascular and cervical vertebral disease. These conditions must be excluded if a diagnosis of Primary Occipital Neuralgia is to be entertained.

**TREATMENT:** Conservative measures should be given an adequate trial, e.g. rest, cervical traction, muscle relaxant drugs, application of moist heat, gentle massage and salicylates. If all of these methods fail, one may undertake a nerve block. This may be done by injecting the greater or lesser occipital nerves or the posterior root of the second Cervical Nerve - first with local anaesthetic and secondly with alcohol. As a final measure one may try avulsion of the greater occipital nerve or cervical posterior rhizotomy. The latter methods are not frequently attempted as results are likely to be temporary.

## ATYPICAL FACIAL PAIN

Although this is one of the commonest of cranio-facial pain syndromes, it remains probably one of the poorest understood. The aetiologies are as multiplex as are the synonyms for this condition (about twenty in all). - Glaser et al (1940).

**AETIOLOGY:** Due to a lack of clinically concrete neuronal distribution of pain or pathological evidence as found in the typical neuralgias, theories as to aetiology must involve speculation on diffuse neuronal mechanisms such as vascular pain afferents and psychiatric disorder theories. Secondary Atypical Facial Pain is due to the organic conditions listed in Table I - be they either local or systemic.

EXECUTIVE AND SECRETARIAL DESKS, TYPEWRITERS  
PHOTOCOPY AND DICTATING MACHINES  
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FILING EQUIPMENT  
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**CLINICALLY:** The description of the pain in this condition is characteristic. Instead of the paroxysms experienced by a patient with trigeminal neuralgia, the pain is a constant phenomenon, i.e. it lasts for days or months rather than for minutes. Exacerbations may be superimposed on this constant pain, but even then the manner of onset is a gradual one. The character of the pain is described variously as aching, burning and throbbing. Its "deepness" is almost always implied verbally or by actions. The location of the pain is not within the boundaries of distribution of any one of the cranio-facial nerves, but rather involves at least two simultaneously. For example, the pain may often be bilateral involving the face, scalp, neck, shoulders and even radiating into the arms upon occasion. Unlike the typical neuralgias, there is a distinct paucity of trigger zones. In some cases there may be signs of concomitant autonomic overactivity at the peak of the pain such as hyperlacrimation, rhinorrhea, nasal mucosa engorgement and facial flushing.

There are often additive psychiatric disorders in this condition. This does not necessarily mean that they are either cause or effect. They are seen to coexist with atypical facial pain. Anxiety, fatigue and climatic conditions are seen to aggravate the condition.

Physical examination usually shows no sensory abnormality although there are some cases that may demonstrate hyperaesthesia or hyperalgesia. The remainder of the neurological examination and laboratory studies are essentially negative.

**DIAGNOSIS:** The diagnosis in this condition is effected by exclusion of the causes of Secondary Atypical Facial Pain. Some authorities have tried to divide the Primary Atypical Facial Pain Syndrome into subdivisions - but this is not adhered to by all. Suffice it to say, one must exclude the greater majority of the conditions in Table I before the diagnosis of Primary Atypical Facial Pain may be entertained.

**PROGNOSIS:** This is extremely variable and the condition may last for weeks, months, or years.

**TREATMENT:** Medical Treatment lies along the lines of tranquilizers (such as meprobamate 0.4 gm. every six hours or chlorpromazine) or vasoconstrictors (wigraïne 2

tabs. stat). Cervical traction in the case of nuchal and shoulder radiation has also been attempted with limited success. Supportive psychotherapy has generally been found to be of value.

Surgical Treatment, be it peripheral nerve section (cranial nerves, vascular pain afferents, sympathectomy), or central operation, (bulbor tractotomy, frontal pole lobotomy, leukotomy or topectomy, stereotoxic thalamotomy) have almost all been doomed to failure.

The diversity and generally poor results of treatment in Primary Atypical Facial Pain epitomize the difficulty encountered in this syndrome.

**CONCLUSION** The present day concepts as to aetiology and treatment of the more common Craniofacial Pain problems have been presented with an eye to their magnitude and complexity. It should be emphasized that pain itself, the commonest single symptom in medicine, is a subject with which a good practising physician (no matter what the field of interest) should be thoroughly familiar.

**Harry Grant**

DIRECT AGENT

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