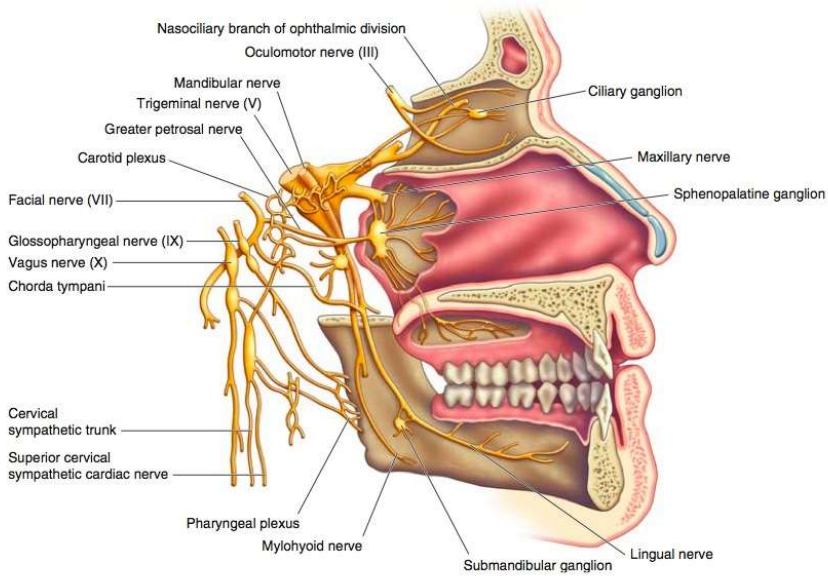
TRIGEMINAL NEURALGIA

CONTENTS

INTRODUCTION



- Largest of cranial nerve
- Nerve of 1st branchial arch
- Contains both sensory and motor fibers
- Attached to the lateral part of the pons by sensory and motor root
- In the middle cranial fossa, sensory root expands to form trigeminal /semilunar / gasserian ganglion on the apex of petrous temporal bone in trigeminal cave.

Function

Sensory (GSA)

- Exteroceptive (touch, pain & thermal)
    - Face
    - Oral and nasal mucous membranes
    - Nasal sinus
    - Teeth
    - Anterior 2/3 of tongue &

- Portion of dura.

- Proprioceptive (deep pr. & kinesthesis)
  - Teeth, periodontium,
  - Hard palate &
  - Tmj receptors
  - Stretch receptors in muscles of mastication

Function

Motor fibers(SVE) innervate muscles–

- Muscles of mastication
- Tensor tympani
- Tensor veli palatini
- Ant belly of Digastric
- Mylohyoid

Trigeminal nerve

4 nuclei

- Mesencephalic
- Main sensory
- Spinal
- Motor
- **Mesencephalic**
  - Column of unipolar neurons
  - Gray matter lateral to cerebral aqueduct
  - Extends into the pons – till main sensory nucleus
- **Main sensory**

- Posterior part of pons
- Continuous with spinal nucleus

- **Spinal**
  - Continuous with main sensory nucleus in the pons
  - Extends through medulla oblongata into the spinal cord upto 2nd cervical segment
- **Motor**
  - Pons
  - Medial to main sensory

Innervation

- Corticonuclear fibres from both cerebral hemispheres
- Also fibres from
  - Reticular formation
  - Red nucleus
  - Tectum
  - Median longitudinal fasciculus
- Monosyaptic reflex arch – fibres from mesencephalic nucleus

Motor root

- Arises in motor nucleus
- Separate from sensory root

Course

- The sensory and motor roots of the trigeminal nerve are attached to the ventral surface of the pons at its junction with the middle cerebellar peduncle

- Course of the trigeminal nerve can be divided into an
    - Intra cranial
    - Extra cranial

Intracranial Course

- Starts from the anterior part of pons
    - Small motor root
    - Larger sensory root
- Runs forward out of posterior cranial fossa
- Rests on the apex of petrous part of temporal bone in the middle cranial fossa

Sensory Root

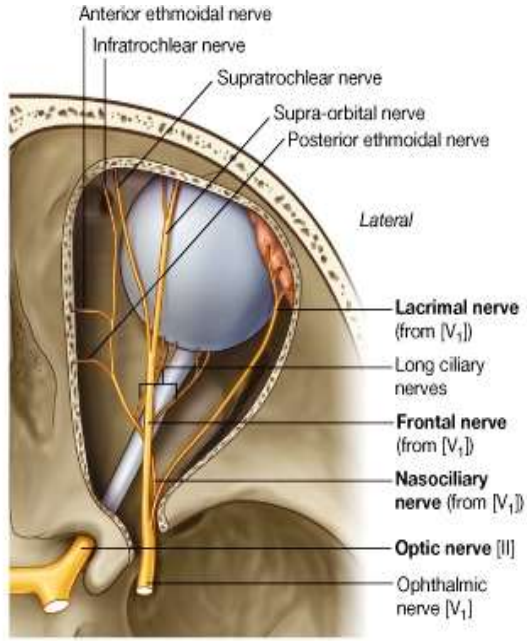
- Expands → trigeminal ganglion
- Lies in Meckel's Cave

Trigeminal Ganglion

- Flat, crescent shaped, measuring 1.0 X 2.0 cm
- Convexity faces anteriorly and downwards
- Concave area is the entrance for the sensory root fibres

Intracranial Course

- Convex area gives rise to three sensory divisions
    1. Ophthalmic (V_1)
    2. Maxillary (V_2)
    3. Mandibular (V_3)



Ophthalmic Nerve (V1)

- First branch of the trigeminal nerve.
- Exclusively sensory and smallest of the three divisions.
- Arises from the anteromedial part of the trigeminal ganglion
- Enters the orbit through the superior orbital fissure.
- In the middle cranial fossa, the nervus tentori branches – supply the dura

Lacrimal nerve

- Superior orbital fissure → above the lateral rectus
- Runs in the lateral wall with the lacrimal artery

- Lateral part of the upper eyelid & a small adjacent area of skin next to the eyelid laterally
- Zygomaticotemporal br.→ secretory fibres→Lacrimal gland

Frontal Nerve

- Superior orbital fissure → above LR

- Passes forward → above levator palpebrae → roof of orbit → 2 branches
- Supraorbital nerve
    - Larger branch → supraorbital foramen
    - Upper eyelid
    - Scalp(as far back as the vertex of the skull)
- supratrochlear nerve
    - Smaller branch
    - Conjunctiva & skin of the medial aspect of the upper eyelid
    - Skin over the lower & mesial aspects of the forehead
    - Descending branch joins the infratrochlear branch of nasociliary

Nasocillary Nerve

- Long root of ciliary ganglion
    - Few filaments to the ciliary ganglion (ramus communicans)
- Posterior ethmoidal
    - Absent in many cases
    - Enter the foramen
    - Posterior ethmoid cells and sphenoid sinus
- **long ciliary nerve**
    - Generally 2 in no – either side of optic nerve
    - Pierce the sclera to supply the eyeball
    - Largely sensory

Nasocillary Nerve

- Anterior Ethmoidal Nerve
    - Enters the foramen → cranial cavity

- Runs fwd on the cribriform plate → nasal cavity → 2 branches

Internal nasal

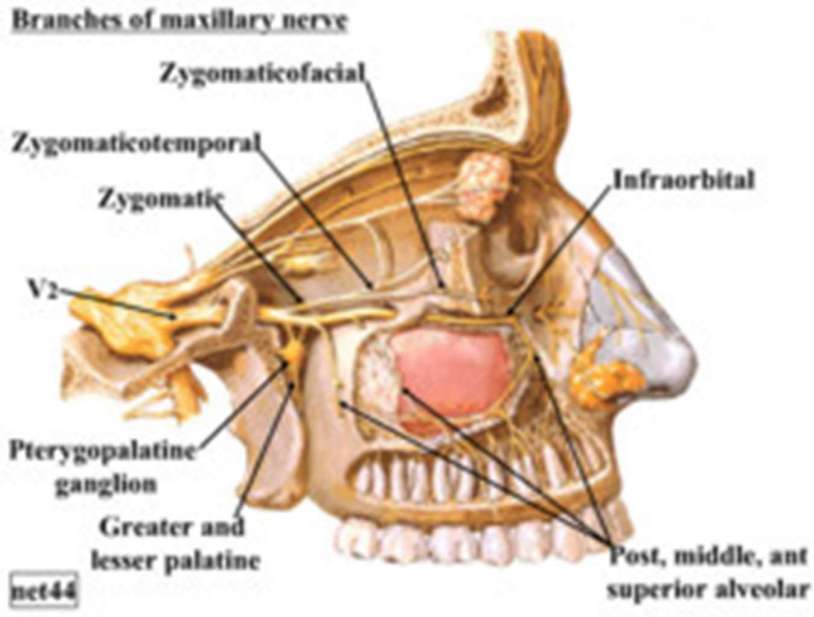
- Mucous membrane of the anterior part of the nasal septum
- Lateral wall of the nasal cavity

External Nasal Nerve

- skin of the nasal ala, apex & vestibule

- Infratrochlear Nerve
  - Passes below the pulley of superior oblique
  - Skin of the lacrimal sac, eyelids & nose
  - Lacrimal caruncle

Maxillary nerve (V2)



MAXILLARY DIVISION

Entirely sensory in function

Originates at middle of the trigeminal ganglion

Continues forward in lower part of cavernous sinus

Enters pteryogopalatine fossa

Orbital cavity

Moves forward and emerges on ant. surface of maxilla through infraorbital foramen

In its course from the semilunar ganglion, the maxillary division gives off branches in four regions:

In the middle cranial fossa

In the pterygopalatine fossa

In the infraorbital groove and canal

On the face (terminal branches)

IN THE MIDDLE CRANIAL FOSSA

Middle meningeal branch- provide sensory innervation to dura mater

IN THE PTERYGOPALATINE FOSSA

zygomatic nerve

pterygopalatine nerve

posterior superior alveolar nerve

IN THE INFRAORBITAL CANAL

1) Middle superior alveolar nerve:

Runs along lat wall of maxilla

Participates in superor dental plexus

Supplies premolars.

2) Anterior superior alveolar nerve:

Runs in canal in ant wall of maxilla - canalis sinosus

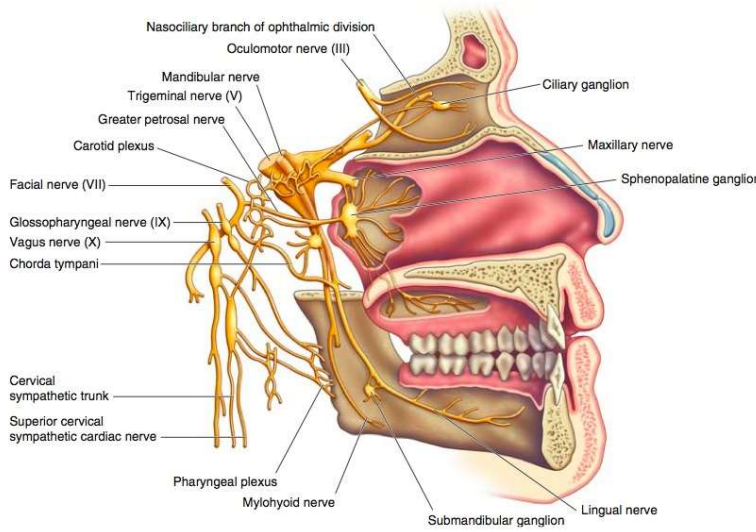
Dental branches nasal branches

Joins superior dental plexus anterior part of nasal cavity to supply incisors & canines

Branches On The Face

- ❖ Inferior Palpebral Branch - supplies skin of lower eyelid with sensory innervation to both surfaces of the conjunctiva
- ❖ External/Lateral Nasal - sensory innervation on lateral aspect of the nose
- ❖ Superior Labial Branch - supplies the skin & mucous membrane of whole of upper lip

Mandibular Nerve



- ❖ Largest division of trigeminal nerve
- ❖ Consists of:

Sensory root(large)

Motor root(small)

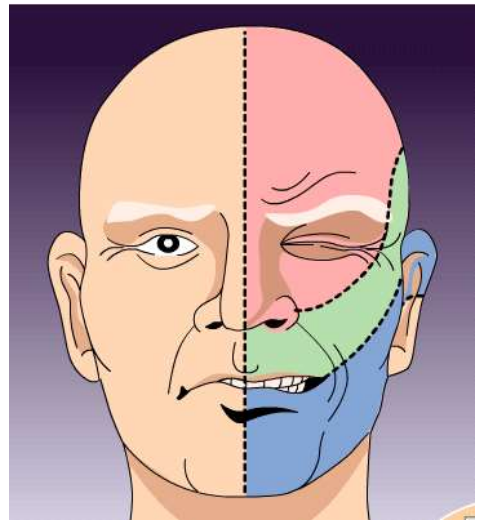
The motor fibres become incorporated with the sensory fibres in the mandibular trunk, while leaving the middle cranial fossa through the foramen ovale

Ganglia

4 small ganglia.

- ❖ Ciliary ganglion is connected with the ophthalmic nerve
- ❖ Sphenopalatine ganglion with the maxillary nerve
- ❖ Otic and submaxillary ganglia with the mandibular nerve.
- ❖ All four receive sensory filaments from the trigeminal, and motor and sympathetic filaments from various sources
- ❖ These filaments are called the roots of the ganglia

TRIGEMINAL NEURALGIA



It is a truly agonizing condition, in which patient may clutch the hand over the face and experience severe, lancinating pain associated with spasmodic

contractions of the facial muscles during attacks—a feature that led to the use of the term (its archaic name) ‘Tic Douloureux’ (painful jerking).

DEFINITION

Trigeminal neuralgia (TN) is defined as sudden, usually unilateral, severe, brief, stabbing, lancinating, recurring pain in the distribution of one or more branches of 5th cranial nerve.

- Sudden, usually unilateral, severe, brief, stabbing and recurrent pain in the distribution of one or more branches of fifth cranial nerve.

  “International association for the study of pain.”

- Unilateral disorder characterised by brief electric shock like pain, abrupt in onset and termination, limited to distribution of one or more divisions of trigeminal nerve.

  “International Headache society”

• John Locke in 1677 gave the first full description with its treatment.

• Nicholaus Andre in 1756 coined the term ‘Tic Douloureux.’

• John Fothergill in 1773 published detailed description oil TN, since then, it has been referred to as ‘Fothergill’s disease’.

In spite of the condition being known since centuries, still continues to baffle the clinician and its pathogenesis remains as enigma to the medical profession. Multiple views have been hypothecated regarding its etiology generating nothing, but confusion and simultaneously opting for many different therapies in an effort to treat this ongoing condition.

Epidemiology

- Age - 50-70 years
- Sex predilection – Female (60%)
- Affliction for side : Predilection for right side
- Multiple sclerosis – 1%
- Division of Nerve affected* -

Maxillary(V^2)- 35%

Mandibular (V^3)- 29%

Ophthalmic (V^1)- 4%

Maxillary & Mandibular- 19%

All Branches- 1%

CLASSIFICATION

- Based on etiology
  - Idiopathic TN
  - Secondary TN
- Based on symptoms
  - Typical TN
  - Atypical TN

ETIOLOGY

The cause of this disease process is unknown. It is usually idiopathic.

Vascular factors such as transient ischemia and auto- immune hypersensitivity responses have been proposed as causes of the demyelination of the nerve.

Mechanical factors have also been postulated, such as the pressure of aneurysms of the intrapetrous portion of the internal carotid artery that may erode through the floor of the intracranial fossa to exert a pulsatile irritation on the ventral side of the trigeminal ganglion.

Anomaly of the superior cerebellar artery has been more recently blamed for causing TN. The artery lies in contact with the sensory root of the trigeminal nerve and it anomaly has been implicated as a cause of demyelination. Surgical elevation of the artery or decompression of the sensory root has been highly successful in relieving paroxysmal pain in cases of idiopathic TN.

The probable etiologic factors are listed below:

i. Dental etiology According to Westrum and Black (1976) differentiation from loss of teeth and degeneration of nerve is not restricted to peripheral parts of the ganglia, but proceeds proximally to involve areas of spinal nucleus. This can partly explain the affliction in the maxillary and mandibular divisions of 5th cranial nerve.

ii. Infections Various granulomatous and nongranulomatous infections involving the 5th cranial nerve can bring about neuralgic pain.

iii. Ratner's jaw bone cavities (1979) Cavities found in the alveolar and jaw bones are the causative factor: Patients with neuralgia inducing cavitational osteonecrosis also can be candidates.

iv. Mu1tip sclerosis Olfson (1966) suggested the presence of sclerotic plaque located at the root entry zone of the trigeminal nerve. Usually patients will have an established diagnosis of multiple sclerosis with demyelinating disease.

Petrous ridge (Basilar) Compression Lee (1937) suggested that trigeminal neuralgia may be caused by compression of the nerve at the dural foramen or over the petrous tip and advocated decompression by performing removal of the bony rim of petrous bone.

vi. Post-traumatic neuralgia The most common types of traumatic neuromas involving the trigeminal branch are those following trauma and those resulting from some dental procedures.These may lead towards neuralgic pain.

vii. Intracranial tumours Many lesions such as epidermoid tumours, meningiomas of cerebellopontine angle and Meckel's cave, arteriovenous malformations, aneurysms and vascular compression have been suggested as the causes. Trigeminal neuromas in the middle cranial or the posterior fossa may be also the causative factor. These intracranial tumours or vascular malformations may impinge on the nerve.

viii. Intracranial vascular abnormalities Compression — distortion of the root entry zone of the trigeminal nerve at the pons by an arterial loop, usually of the superior cerebellar artery, or by venous compression by arteriovenous malformations, etc. Compression of the intracranial retrogasserian portion of the 5th cranial nerve by a displaced vein or artery may be also a cause. Aneurysm of the internal carotid artery may cause TN

ix. Viral etiology Postherpetic neuralgia is seen in elderly patients. History of a previous episode of infection by varicella zoster virus may be present in these patients. Viral lesions of the ganglion can be the etiological factor.

GENERAL CHARACTERISTICS

• Incidence: It is a rare affliction, seen in about 4 in 100,000 persons.

• Age of occurrence Late middle age or later in life (5th or 6th decade)

• Sex predilection With female predisposition (58%).

• Affliction for sides Predilection for the right side is noted (60%).

• Division of trigeminal nerve involvement V3 is more commonly involved than V3 division. Very rarely V3 ophthalmic division is involved in about 5 per cent of cases (Only sensory division is affected).

CLINICAL CHARACTERISTICS

Trigeminal neuralgic pain typically arises in the persons, who have no abnormal neurologic deficit such as loss of corneal reflexes, anaesthesia, paraesthesia, muscular atrophy or weakness, etc.

• TN typically manifests as a sudden, unilateral, intermittent paroxysmal, sharp, shooting, lancinating, shock like pain, elicited by slight touching superficial 'trigger points' which radiates from that point, across the distribution of one or more branches of the trigeminal nerve

• Pain is usually confined to one part of one division of trigeminal nerve-mandibular or maxillary, but may occasionally spread to an adjacent division or rarely involve all three divisions

• Pain rarely crosses the midline

• The pain is of short duration and lasts for a few seconds, but may recur with variable frequency Even though there is a refractory period (complete lack of pain) between the attacks; some patients report a dull ache in between the attacks

• During an attack, the patient grimaces with pain, clutches his hands over the affected side of the face, stopping all the activities and holds or rubs his face, which may redden or the eyes water until the attack subsides. Male patients avoid shaving. The oral hygiene is poor as patient avoids brushing of teeth

• The paroxysms occur in cycles, each cycle lasting for weeks or months and with time, the cycle appears closer and closer. With each attack, the pain seems to become more intense and unbearable.

• In extreme cases, the patient will have a motionless face—the 'frozen or mask like face'

• Presence of an intraoral or extraoral trigger points provocable by obvious stimuli is seen in TN. It may be brought on by touching face at a particular site or by chewing or even by speaking or smiling, brushing, shaving or even washing the face, etc.

• The location of the trigger points depends on which division of trigeminal nerve is involved.

i. In V2 – points are located on the skin of the upper lip, ala nasi or cheek or on the upper gums.

ii. In V3 - this is the most frequently involved branch. Trigger points are seen over the lower lip, teeth or gums of the lower jaw. Tongue is rarely involved.

iii. In V1 – the trigger zone usually lies over the supraorbital ridge of the affected side.

• It is characteristic of the disorder, that attacks do not occur during sleep.

• Many patients will lead a very poor quality of life, because of excruciating pain.

• It is very common for these patients to undergo indiscriminate dental extractions on the affected side without any relief from pain, because the pain of the trigger zone and pain fibre distributions often mimic pain of odontogenic origin.

• More than 50 per cent of patients experience early remissions of greater than 6 months before return of active pain.

WHITE AND SWEET DIAGNOSTIC CRITERIA

1. The pain is paroxysmal
2. The pain may be provoked by light touch to the face (trigger zone)
3. The pain is confined to the trigeminal distribution
4. The pain is unilateral
5. The clinical sensory examination is normal

ICHD CRITERIA FOR CLASSICAL TN

- Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B & C.
- Pain has at least one of the following characteristics:
    - Intense, sharp, superficial or stabbing.
    - Precipitated from trigger areas of trigger factors.
- Attacks are stereotyped in the individual patient.
- There is no clinically evident neurological deficit.
- Not attributed to another disorder.

ICHD CRITERIA FOR SYMPTOMATIC TN

A. Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B & C.

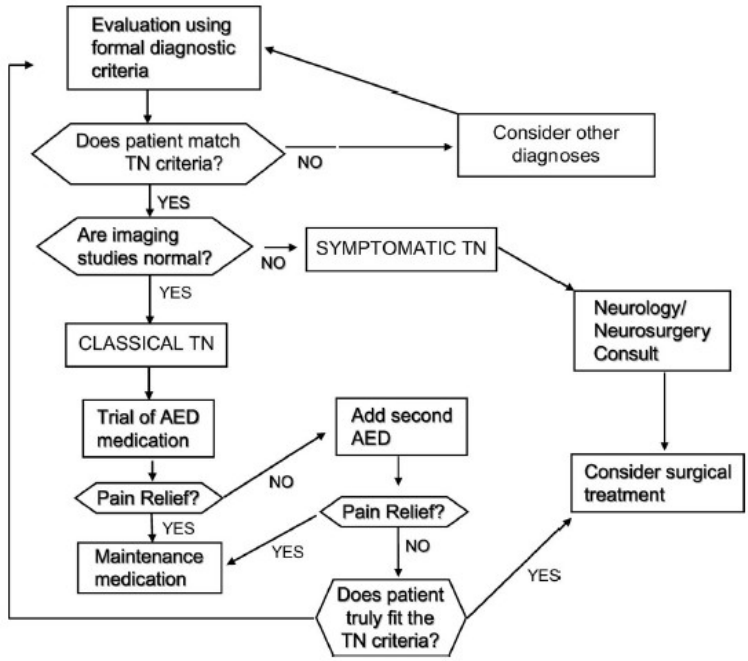
B. Pain has at least one of the following characteristics:

   a) Intense, sharp, superficial or stabbing.

   b) Precipitated from trigger areas of trigger factors.

C. Attacks are stereotyped in the individual patient.

D. A causative lesion, other than vascular compression has been demonstrated by special investigation and/or posterior fossa exploration.



DIAGNOSIS

Diagnosis is made from a well-taken history. The classic clinical pattern will lead towards the diagnosis. Some times, if symptoms may be less classic and may mimic toothache, sinusitis, stomatitis or other inflammatory conditions.

Proper clinical examination along with history is mandatory. The neuralgic symptoms in younger group of patients (<35 years of age) should alert the clinician to a possible intracranial space occupying lesion or intracranial arteriovenous anomalies. Other differential diagnosis should include acaustic neurilemoma, multiple sclerosis, postherpetic neuroma or post-traumatic neuralgias. All patients should ideally have MRI scanning or at least a CT scan. Preoperative localization of compressive vessels at the root entry zone is done by MRI scanning.

Response to treatment with tablet carbamazepine is universal in trigeminal neuralgia, as in other types of facial pain it is not useful. Many clinicians use this response as a step in definitive diagnosis of the condition. Failure to obtain any improvement with this treatment should bring the diagnosis into question.

Diagnostic injections of a local anaesthetic agent into the patient's trigger zone should temporarily eliminate all pain.

Protocol for Diagnostic Nerve Blocks

Material required:

3 — 1 cc syringes

3 — 25 gauge needles

Sterile normal saline

Two per cent lignocaine without adrenaline.

Several alcohol wipes

• Always begin injections at surface site of pain and then move proximally. For example, if the pain is perceived in the lower lip, then inject lower lip, then mental nerve and then inferior alveolar nerve.

• Inject 0.5 cc of normal saline at test site. Wait for 5 minutes. If pain is relieved, then psychogenic pain is likely.

• If the pain persists, then inject 0.5 ml of 2 per cent lignocaine without adrenaline at surface site and wait for 5 minutes. If pain is relieved, then direct therapy at small nociceptor fibres

• If the pain persists — inject little deeper and wait for 5 minutes. If pain is relieved, then consider musculo skeletal origin of pain.

• If pain is not relieved, inject at more proximal portion of nerve—If pain is relieved, direct therapy at site, when relief occurred

Thus selective inferior alveolar, lingual, buccal, infra orbital, posterior superior alveolar blocks can be given to know the involvement of the branch of the trigeminal nerve.

TREATMENT

Once the diagnosis of trigeniinal neuralgia is established, then the treatment regime is started. First medicinal management is advocated. If the patient does not respond to it, then only surgical management is opted.

Medicinal Management

This is the first line approach for most of the patients. TN does not respond to analgesics including opiates.

Blom (1962) showed a response to anticonvulsants. Carbamazepine is highly specific in only relieving pain of TN and not any other type of facial pain. It has, therefore, suggested that its response can be used as a diagnostic indicator.

Carbamazepine (Tegretol) and phenytoin (Dilantin) are the traditional anticonvulsants used primarily, as soon as the diagnosis is done. This therapy consists of titration and maintenance with anticonvulsant drugs.

Carbamazapine 100 mg three times a day is introduced and titrated over 1 to 5 weeks period until either remission is achieved or side effects or toxicity are unacceptable. (Commercially, tegretol 100 mg 200 mg or 400 mg tabs are available. Controlled or slow release tablets are available.

The dosage of the drug used initially should be kept small to minimum especially in elderly patients to avoid nausea, vomiting and gastric irritation. More of daily drug dosage should be taken at night, so that adequate serum concentration can be present in early morning, when pain most occurs. Complete blood count with platelet count, liver function screening should be done prior to treatment, a month after treatment and at 3 to 4 months intervals, particularly, if patient continues to receive a high dose (1000 to 1500 mg/day).

Side effects: Visual blurring, dizziness, sonmolence, skin rashes and ataxia and in rare cases hepatic dysfunction, leukopenia, thrombocytopenia—aplastic anaemia (It is known to suppress the bone marrow. Patients should be monitored to avoid agranulocytosis). Whenever the side effects appear, a reduction of 200 mg of drug will often eliminate them. Once the pain remission has been achieved, the drug dose should be kept at maintenance level or withdrawn and restarted if symptoms appear.

If carbamazepine does not control symptoms adequately, then another anticonvulsarit like sodium vaiproate 600 mg/day can be added or amitriptyline can be added. Co-administration of phenytoin or baclofen is also advocated.

When carbamazepine is contraindicated clonazepam 1.5 mg/day can be used.

Side effecfs Drowsiness, fatigue, lethargy.

• Tab. Phenytoin: Dose—100 mg three times a day.

Side effects—slurred speech, abnormal movements, swelling of lymph glands, gingival hypertrophy, hirsutism, folate deficiency.

• Tab.Oxcarbazepine — 1200 mg/day

Side effects— hyponatraemia, double vision.

• Valproic acid—600 mg/day.

Side effects—irritability, tremours, confusion, hepatoxicity, weight gain.

• Mephenesin Carbamate (Tolceram)—5 to 15 ml/5 times a day to every 3 hours.

• Other less toxic agents:

- Baclofen (Lioresal) - 10 mg tds

Side effects—fatigue, vomiting.

- Gabapentin (Neurontin) recently introduced drug.

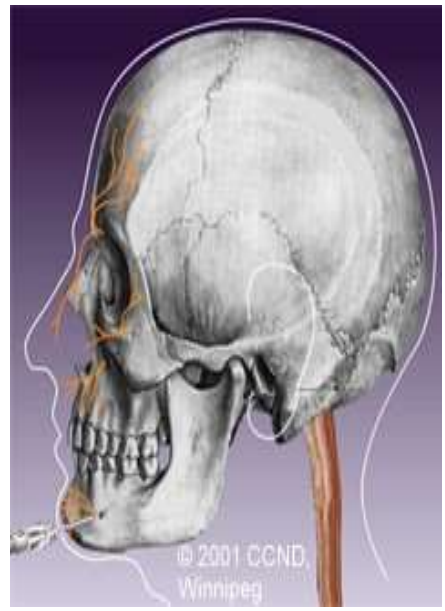
- Lamotrigine

- Felbamate

- Topiramate

- Vigabtrin

Peripheral Injections



For several years, it has been known that injections of destructive substances into peripheral branches of trigeminal nerve, produce anaesthesia in the trigger zones or in areas of distribution of spontaneous pain, and this procedure can be effective in relieving this syndrome, usually as long as the anaesthesia persists. Care should be taken to ensure that IV injections are avoided. This method is successful, when there is well-localized trigger area.

a. Long-acting anaesthetic agents—without adrenaline such as bupivacaine with or without corticosteroids may be injected at the most proximal possible nerve site. The selective nerve blocks can be given as an emergency measure, where the patient is suffering quite a lot, but the pain free period will be very short lived. The injection can be repeated, when the pain recurs.

b. Alcohol injections —peripheral branches of trigeminal nerve can be blocked by the intraoral injection of 95 percent absolute alcohol in small quantities (0.5 to 2 ml). This produces anaesthesia of the region, supplied by the branch. Repeated alcohol injections should be avoided, as it causes local tissue toxicity inflammation and fibrosis. It can also cause a complication of burning

alcohol neuritis. The results are variable. Sometimes it provides relief for a period of 6 to 12 months or some times patient comes back with pain immediately within short time span. Extraoral injections into maxillary and mandibular division of the trigeminal nerve at the level of the base of the skull also can be given. Peripheral injections—infraorbital, mental, inferior alveolar nerve blocks can be given depending on the involvement.

Peripheral Neurectomy (Nerve Avulsion)

• Oldest and most effective peripheral nerve destructive technique

• Simple procedure can be repeated and relatively reliable technique

• It acts by interrupting the flow of a significant number of afferent impulses to central trigeminal apparatus

• Indicated in patients, in whom craniotomy, a more extensive procedure is contraindicated, because of age debility or significant systemic diseases, limited life expectancy

• Performed most commonly on infraorbital, inferior alveolar-mental and rarely lingual nerves

• It has a disadvantage of producing full anaesthesia or deep hypoesthesia related dysfunction

• There is also the expected eventual return of pain with proliferation of amputed nerve stump neuromas

• To achieve better results, the peripheral nerve is always avulsed both from the bone as well as from the soft tissues

• The duration of pain remission after neurectomy may be lengthened, if the cut nerve end is cauterized or redirected and sutured into viable muscle, periosteum or bone tissue to prevent active neuroma formation

• The bony foramen may be plugged with nonabsorbable material or by the bone piece itself

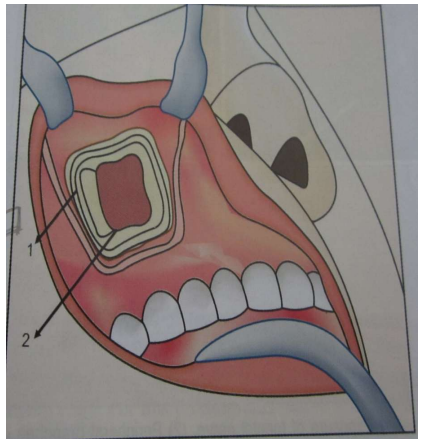
• The procedure is carried out under general anaesthesia to ensure successful avulsion.

lnfraorbital neurectomy: It can be performed through (i) conventional intraoral approach or (ii) through Braun's transantral approach.

i. Intraoral conventional approach –

U- shaped Caldwell-Luc incision is made in the upper buccal vestibule in the canine fossa region. Muco periosteal flap is reflected superiorly to locate the infraorbital foramen. Once the nerve is exposed, all the peripheral branches are held with the hemostat and avulsed from the skin surface intraorally. Then the entire trunk is separated from the skin surface is held with the hemostat at the exit point from the foramen and is removed by winding it around a hemostat and pulling it out from the foramen. The infraorbital foramen may be plugged with polyethylene plug and wound is closed with interrupted sutures.

ii. Braun's transantral approach (1977)—



ig. 44.6: Surgical approach to pterygomaxillary fossa
1) Anterior antral wall, (2) Posterior antral wall

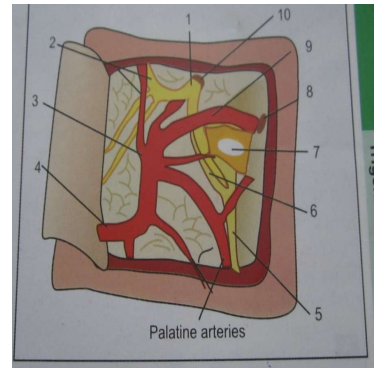


Fig. 44.7: View of the contents of pterygomaxillary fossa
(1) Maxillary nerve, (2) Infraorbital nerve and artery,
(3) Maxillary artery, (4) Posterior superior alveolar artery,
(5) Palatine nerves, (6) Vidian nerve, (7) Sphenopalatine
ganglion, (8) Sphenopalatine foramen, (9) Sphenopalatine
artery, (10) Foramen rotundum

It has got the potential to have sound treatment for intractable V2 neuralgia, because of the direct access and visualization it provides. The key to this approach is a thorough knowledge of anatomy. With sectioning of the maxillary nerve, anaesthesia is created over its entire distribution.

An intraoral incision is made from the maxillary tuberosity to the midline in the maxillary vestibule. The mucoperiosteal flap is reflected to expose the anterior and lateral maxillary antral wall, the zygoma and the infraorbital nerve. A 3 cm window is made in the antero lateral wall of the maxillary sinus. The operating microscope is usually required for the remainder of the procedure. The lining in the posterosuperior portion of the antrum is carefully excised and bone is removed to create a posterior window. Careful dissection is now performed to expose the descending palatine branches of V which are then traced superiorly to the sphenopalatine ganglion. In order to provide anatomical verification, the infraorbital nerve is identified in the roof of the maxillary sinus and is carefully followed posteriorly to the trunk of V2 near the sphenopalatine ganglion. Dissection is then completed by isolating and identifying the trunk of

V2 superiorly and posteriorly to the sphenopalatine ganglion. The trunk of the maxillary nerve (V2) is then sectioned posterior near the foramen rotundum to the inferior orbital fissure. The antral mucoperiosteal flap in the vestibule is repositioned and sutured back.

Complications

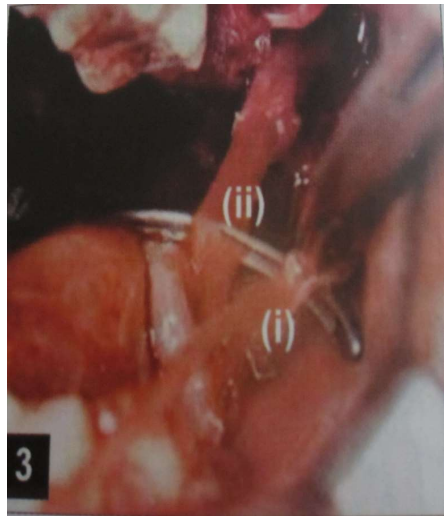
(i) Inadvertent section of the vessels in the pterygopalatine fossa

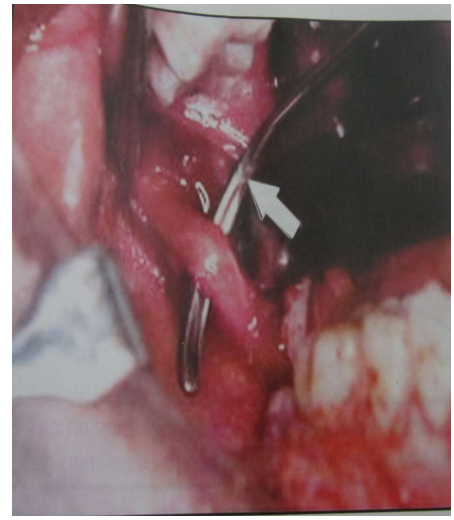
(ii) Inadvertent sectioning branches of the sphenopalatine ganglion or the vidian nerve, entering the posterior aspect of the ganglion.

Inferior alveolar neurectomy. It can be performed via intraoral or extraoral approach. The intraoral approach is preferred, as it is simple and more cosmetic.

1. The extraoral approach—is through Risdon's incision, where after reflection of masseter, a bony window is drilled in outer cortex and nerve is lifted with nerve hook and avulsed from its superior attachment and mental nerve is avulsed anteriorly through the same approach.

2. Intraoral approach —Via Dr Ginwalla's incision —





It is mainly used in dentulous cases. Incision is made along the anterior border of ascending ramus, extending lingually and buccally and ending in a

fork like an inverted Y (Dr Ginwalla's incision). Such incision provides better exposure of the field. The incision is then deepened on the medial aspect of the ascending ramus by means of blunt and sharp dissection. The temporalis and medial pterygoid muscles are split, rather than divided at their insertion and the inferior alveolar nerve is located. Two heavy black linen threads are then looped around the nerve using nerve hook and then divided between the 2 threads. This is done as high as possible and the upper end is cauterized while dividing and lower end is held with the hemostat. Another linear incision is made in the buccal vestibule overlying the mental foramen. A mucoperiosteal flap is reflected to expose the mental nerve. It is then tied with heavy black linen just little away from the foramen. The nerve is then caught with the hemostat distal to the knot and is divided between the two. The distal part held between the hemostat and is wound around it and the peripheral branches entering the mucosa are avulsed out. There is puckering of the skin surface seen during this procedure.

Now after the mental nerve is freed, then at the mandibular foramen, the distal part of the nerve which is held with the hemostat is pulled until the entire nerve length of the canal is avulsed out. If any obstruction is encountered a window may be made in the buccal cortex posterior to the mental foramen along the level of the inferior alveolar canal and the nerve is lifted out of the canal through the window. The wound is closed with interrupted sutures.

Lingual neurectomy:

A vertical incision is made at the inner border of the ascending ramus, extending from the coronoid process down the level of the floor of the mouth. Keeping the two sides of the incision retracted, the dissection is continued

downwards until the lingual nerve comes into view at the border of the medial pterygoid muscle. In the region of the floor of the mouth, the nerve lies even more superficially and it can be easily found between the anterior pillar of the fauces at the root of the tongue. After dissection, the nerve is grasped with a hemostat and is then either avulsed or cauterized and cut. The wound is closed with interrupted sutures.

Cryotherapy or Cryoneurolysis for Peripheral Nerves

Direct applications of cryotherapy probe at temperatures colder than — 60°C are known to produce Wallerian degeneration without destroying the nerve sheath itself. For this procedure the nerve is exposed as described in peripheral neurectomy procedure and is frozen with a cryoprobe (Nitrous oxide probe) for a period of 1-2 minutes followed by 3 minutes thaw, to be repeated three times. The pain remission follows the procedure. But regeneration of axons is expected. No large series are available for comparison with peripheral cryotherapy techniques. But the procedure is relatively simple.

Peripheral Radiofrequency Neurolysis (Thermocoagulation)

Gregg and Small in 1986, reported surgical management of trigeminal pain with radiofrequency lesions of the peripheral nerves. A radiofrequency electrode that has the capacity to definitely destroy the pain fibers is used in this procedure. The use of radiofrequency (RF) lesioning of trigeminal nerve has been used sparingly, but with success. RF neurolysis has been shown to induce pain remission in 80 per cent of cases with a 20 percent/year recurrence rate.

Procedure

Topical anaesthesia with mild sedation is used. The patient is grounded in an electronic circuit and the 22 gauge lesion probe is positioned adjacent to nerve to be lesioned. Paraesthesias are elicited to ensure proximity to the nerve and

tissue temperature is measured with the probe tip through the probe thermocouple. Lesioning is then carried out at 65 to 75°C for 1 to 2 minutes. Repositioning may be required to ensure adequate RF wave effect on the nerve fibers.

Advantages : Low morbidity in high risk—elderly patients.

Disadvantages : Needs specific electronic armamentarium and reasonable patient cooperation. In case of inaccessibility of some pain triggering nerve trunks, the technique will fail to achieve pain relief.

Gasserian Ganglion Procedures

• Around 1900—first open surgeries were performed on the gasserian ganglion for TN. These were recorded as hazardous procedures

• 1910—Harris, Tapatas and Hartel separately introduced percutaneous approaches to the ganglion via the foramen ovale. Absolute alcohol or phenolglycerol mixture was used as the neurolytic agent.

- 1931—Kirschner introduced percutaneous electro coagulation of the gasserian ganglion.

Since then, three main percutaneous gasserian ganglion procedures are being used with variable success rate:

(i) Glycerol injection,
(ii) Thermocoagulation,
(iii) Balloon compression.

The technique of needle placement is common to all of these. Initial pioneering procedures were performed free hand, but now hard-copy X-ray or image intensification on monitors is used first for visualization of position of the

foramen ovale and then for confirmation of the depth of penetration of the needle and the position of any contrast medium used.

Technique for Percutaneous Approach to the Gasserian Ganglion: Anaesthesia Protocol

Patient is admitted on the day of surgery and kept nil by mouth for at least 4 hours prior to surgery Injection atropine (0.6 mg TM) is given one hour prior to surgery to reduce oral secretions and to prevent intraoperative bradycardia.

Injection methahexitone — ultrashort-acting barbiturate (Brevital) is administered intravenously in dose of 1.5 to 2 mg/kg body weight in increments, so as to maintain an adequate level of unconsciousness during lesion production and during painful part of the procedure, like when needle/electrode comes in contact with the base of the skull, when it penetrates foramen ovale. Pulse oximeter, oxygen saturation, vital signs including systolic and diastolic blood pressure, respiratory rate, pulse rates as well as ECGs are monitored throughout the procedure. The duration of procedure is usually of one hour. Patient should be given intranasal oxygen and intravenous fluids

Procedure

The patient is made to lie on a table with neck well-extended. The foramen ovale is best visualized with the X-ray tube placed for a submentovertex position Infiltration of the skin and cheek is done with local anaesthetic agent on the affected side. Three points of Hartel are marked on the side of the face using marking ink.

• First point—is marked at lateral orbital rim and a perpendicular line is drawn till the inferior border of the mandible.

• Second point—is marked at about 15 mm (3 inch) lateral to the angle of the mouth on the perpendic first line. This is a point of penetration of needle/ electrode.

• Third point—is marked at the level of TMJ. 2.5 cm from the centre of external auditory meatus. This point is joined with the 2nd point of Hartel. This line will form a plane, which is the plane of elevation. When the patient is positioned in supine position with head extended, the plane of elevation is perpendicular to the floor (a flat pillow is kept under the shoulder). The needle/electrode is passed through the cheek from the point of penetration (2nd point). The finger of gloved hand is placed inside the oral cavity to prevent inadvertent penetration of needle/electrode in the oral cavity.

Pass the needle/electrode along the plane of elevation till it reaches the anterior border of ramus of mandible, then turn the needle medial to ramus, pass it upwards to the base of the skull in pupillary plane.

During this phase, the needle/electrode lies below the orbit, medial to ramus of the mandible and lateral to maxilla. Engagement of the needle/electrode in the foramen ovale is best confirmed by biplanar radiology or image intensifier.

The final position of the needle/electrode is then pushed for another half a centimeter.

Glycerol injection

Glycerol or absolute alcohol can be used for percutaneous ganglion neurolysis. The agent is injected into Meckel's cave or in the ganglion. The agent then diffuses throughout the ventral ganglion, producing low grade damage to nerve cells, presumably through dehydration. This technique induces pain relief in around 80 per cent of cases, it can be repeated if required and it prevents gross facial sensation with lower levels of anaesthesia. It also spares the important ophthalmic division and the motor root.

For this injection a 16 gauge spinal needle is used and inserted through the foramen ovale into the ganglion till CSF is obtained on withdrawal of the stylet. Contrast medium is injected to check the position of the needle. The contrast is evacuated and replaced by 0.5 to 0.75 ml of pure glycerol or 0.5 ml of absolute alcohol. The patient is sent to ward and kept there with head extended for 2 hours. The relief obtained varies from six months to two years. In this procedure patient cooperation is needed, if it is done under local anaesthesia.

Controlled radiofrequency thermocoagulation

A radiofrequency electrode that has the capacity to definitely destroy pain fibres is now used.

Electrocoagulation of gasserian ganglion was introduced first by Kirschner (1931) and later modified by Sweet (1970).

Advantages

1. Avoidance of denervation of cornea.
2. Comparative lower rate of recurrence.
3. Procedure can be repeated in case of recurrence.
4. Zero mortality
5. Well-tolerated by elderly and medically compromised patient.
6. Patient will prefer thermocoagulation rather than opting for neurectomy, which leads to facial numbness.
7. Where facilities permit, it can be performed on outpatient basis.

8. Thermocoagulation preserves the motor function of trigeminal nerve.

9. Simple, accurate procedure, less time-consuming and less expensive, and comfortable for the patient.

Indications

• Toxicity of drugs

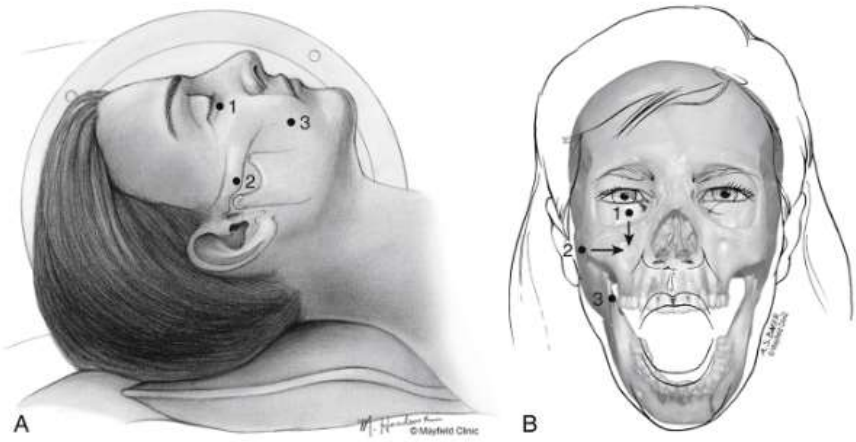
• Failure of response to other modalities

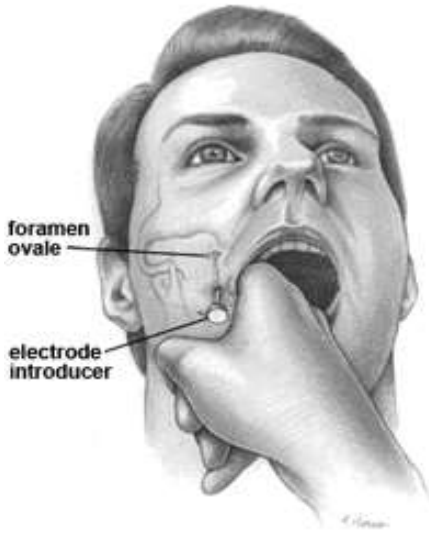
• Dependence on drug for lifetime

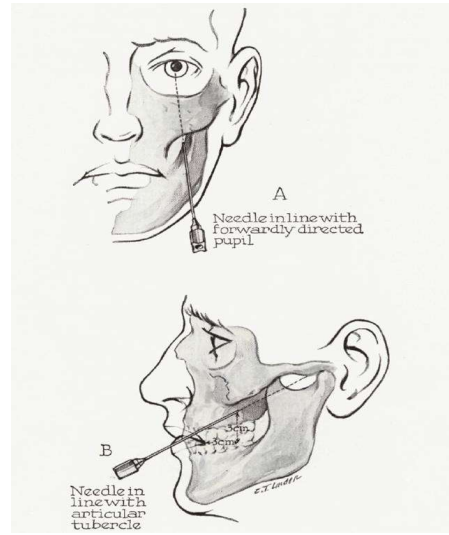
• Elderly patients

• Medically compromised patients

• Recurrence cases—previously treated by intracranial surgery, neurectomies, etc.







Procedure

The trigeminal ganglion may be approached through the foramen ovale by percutaneously placing a 22 gauge probe under fluoroscopic guidance. The image intensifier is used and middle cranial fossa is focussed in the centre of screen, the contrast is adjusted to sharpen the bony landmarks. Clear visualization of foramen ovale is obtained by adjusting the position. The probe used should have an insulated shaft and a bore sufficient for the passage of a radiofrequency electrode. When the probe is placed correctly in the foramen ovale and advanced into the trigeminal gang lion, CSF should emerge on removal of the stylet as the ganglion contains CSF (in patients who have undergone previous surgeries or chemical injections you will not get CSF flow). The electrode is inserted then just beyond the tip of the probe and low amplitude current is applied using a lesion generator.

Patient at this stage should be awake and cooperative, is asked whether on the face the stimulation is felt (electric like impulse). The position of the probe is adjusted until stimulation is experienced by the patient in the area where trigeminal neuralgia pain was present. Stimulation is initiated utilizing square wave pulses at 50 cycles per second slowly raising the voltage until full areas of pain covered.

Lesion production Patient at this stage should be given neurolept anaesthesia. Thermal lesions of 30 to 90 second duration are then made at 65 to 75°C using RF generator of microwave energies.

Power 25 watts, voltage 40-45 volts. Current 120-140 mA. With these parameters a temperature range of 65- 75°C is achieved. A 5 mm bare tip electrode with 2 mm diameter will produce a lesion of 10 x 6 mm within the trigeminal root at 75°C.

A radiofrequency current when an alternating current of high frequency is passed through the electrode, it produces ionization in biological tissues. Heat results from ionic friction, which leads to coagulation of tissues. Great care is exercised to avoid over shooting to preserve the sense of touch. After partial result is produced, it is mostly possible to complete the lesion without additional short-acting anaesthetic agent. A facial blush usually appears at this point and helps to localize the region of nerve root undergoing thermal destruction. This is due to the vasodilator system emerging from the brainstem and passing to the facial vasculature with trigeminal nerve.

At the end of the procedure, patient is asked to perform those manoeuvres that characteristically trigger the TN. Once the patient and operator is satisfied with the desired RF lesion production, the electrode is removed and patient is sent to recovery room.

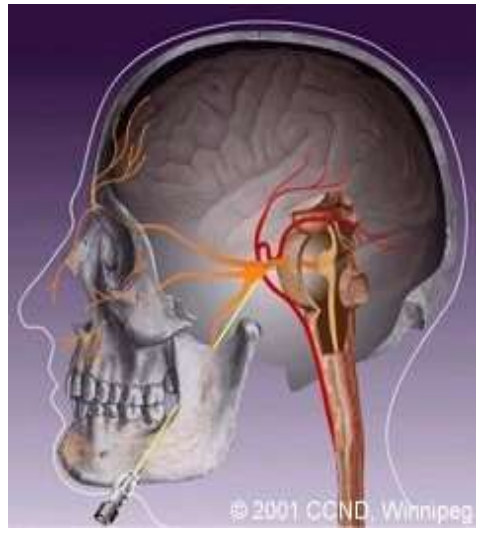
Balloon compression

It is done under general anaesthesia. It is a mechanical technique to destroy root fibres partially by advancing 4FG Fogarty catheter 1 to 2 cm within Meckel's cave and inflating the balloon at the ventral aspect of the ganglion root.

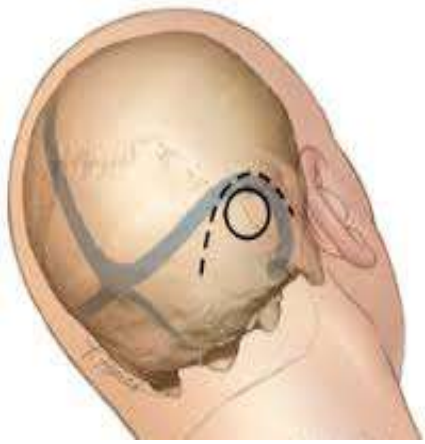
A 12 gauge spinal needle is passed first to the foramen ovale and the balloon catheter is passed through it. Once it is in position the balloon is inflated with

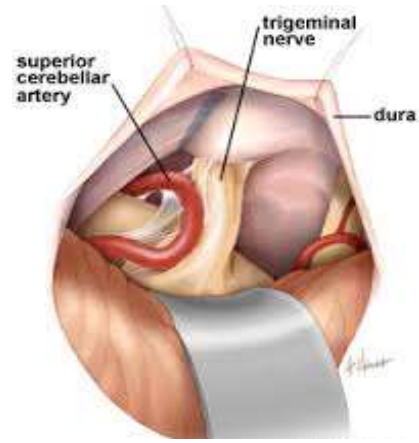
X-ray contrast medium upto 0.75 ml. When inflated, the balloon should take up the pear shape of Meckel's cave and it should remain inflated for 1 minute.

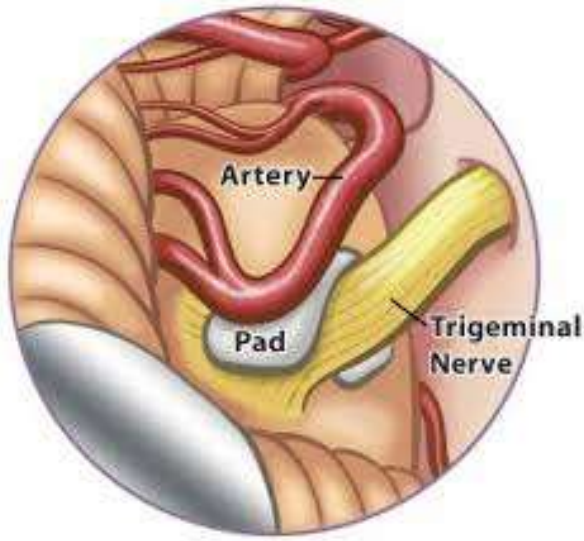
Open Procedures (Intracranial Procedures)



Microvascular decompression of the sensory root (Posterior Fossa Decompression – PFD)







• Procedure popularized in 1967-1976 by Jannetta

• Most commonly performed intracranial open procedure

• Open craniotomy approach is used to gain access to the trigeminal root entry zone and adjacent brainstem

• The root is examined under the microscope. A com pressing branch of the superior cerebellar artery will be seen medial to the nerve at the root entry zone

• The artery is carefully separated from the nerve and interpositioned by using sponge or Teflon wool

• Patient usually retains good facial sensation without anaesthetic dysfunction.

• 75 to 80 per cent of patients are pain free for at least years

• Overall mortality rate is 2 per cent. Can be associated with infrequent hearing loss, vertigo, cranial nerve VII weakness

• Contraindicated in elderly patients and medically compromised patients.

Trigeminal root section

a. Extradural sensory root section: Frazier's approach (1901)

The subtemporal extradural route retrogasserian. rhizotomy (root sectioning).

Here sensory root is divided, sparing the motor root as close to the brainstem as possible.

This procedure is rarely used now and it is of historical value, because of the profound sensory loss and high incidence of anaesthesia dolorosa. Sensory loss involving ophthalmic division leads to keratitis.

b.Intradural root section Described by Wilkins (1966)

is superior to extradural approach as it has less chances of damage to superior petrosal nerve and facial nerve, less chance of bleeding.

In 1932 Dandy recommended posterior fossa surge but technique is more difficult than Frazier's approach and it can damage 5th, 7th or 8th cranial nerve because of excessive retraction or manipulation, vascular damage, etc.

Advantages of posterior fossa approach is unsuspected tumours or vascular malformation are more readily delineated and handled.

c. Trigeminal tractotomy (Medullary Tractotomy) Incision of descending trigeminal tract near the cervicomedullary junction will reliably cause the loss of pain temperature sensation in the ipsilateral face and pharynx and usually will relieve the pain of TN. It is helpful in patients with pain perceived in both glossopharyngeal and trigeminal distribution. It can be also used for intractable facial pain and should not be routinely used.

Streptomycin-lidocaine injections for the treatment of postherpetic neuralgia: Report of three cases with literature review. Eur J Dent 2013;7:105-10.

The sudden, stabbing, paroxysmal pain of neuralgia is the fiercest agony that a patient may experience in his life. Many varied medical treatments and

surgical procedures have been suggested in the literature for neuralgic pain. Most of the patients fail to respond to medical treatments or succumb to complications of total anesthesia owing to surgical procedures. Herein, we tried a new treatment modality in patients suffering from postherpetic neuralgia with appreciable success in all the three cases that are presented in this paper. Streptomycin sulfate dissolved in 2% lidocaine solution was deposited at the peripheral branches on the involved nerves targeting the trigger zones, given weekly once for a maximum of 6 week period and continued once in 2 weeks if symptoms persisted. All patients were followed‐up for 1 year and there was a marked improvement on follow‐up.

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