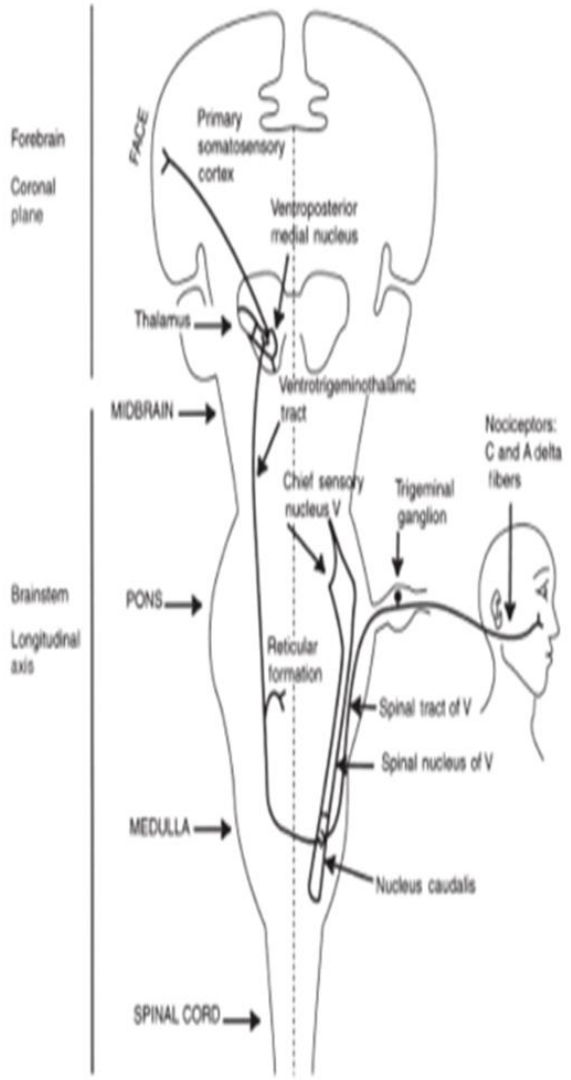
Facial pain

د.رائدة نوري

Pain, An unpleasant sensory and emotional experience associated with actual or potential tissue damage.

Cranial nerve V (CN V), the trigeminal nerve, is the dominant nerve that relays sensory impulses from the orofacial area to the central nervous system. The facial (CN VII), glossopharyngeal (CN IX), and vagus (CN X) nerves and the upper cervical nerves (C2 and C3) also relay sensory information from the face and surrounding area.



Nociceptive transmission associated with the trigeminal nerve

Primary sensory neurons associated with pain are characterized by small diameter axons with slow conduction velocities (ie, finely myelinated A delta fibers and unmyelinated C fibers). Nociceptors are the sensory receptors specialized for detecting noxious stimuli.some are unimodal and respond only to thermal or mechanical stimuli, others are polymodal and respond to mechanical ,thermal ,and chemical stimuli.

Information associated with pain is carried in three divisions of the trigeminal nerve to the trigeminal sensory ganglion then end at the somatosensory cortex.

Chronic pain:

Nociceptors,specialized recptors that signal tissue damage,terminate in a highly ordered manner in the dorsal horn of the spinal cord and its homologous subnucleus caudalis in the spinal trigeminal nucleus. Following peripheral tissue or nerve injury, a pathologic state may develop; resulting in persistent pain long after the injured tissue has healed. In this state, pain no longer represents a warning signal of potential or actual tissue damage; pain itself becomes the disorder.

Chronic pain has been defined as pain that persist past the normal time of healing ,but this may not be an easy determination.alternatively ,chronic pain has been related to duration (ie,pain that lasts longer than 6 months).recently, pain lasting longer than 3 months has been used to define chronic pain.

The proposed explination for the persistence of pain after healing related to changes (neuroplasticity) in the central system. The clinical manifestations of these changes include hyperalgesia (an increased response to a stimulus that is normally painful); allodynia (pain due to a stimulus that does not normally provoke pain); and spontaneous, radiating, and referred pain.

Drug therapy:

- ✓ **Non-opioid Analgesics:**

This group consists primarily of acetaminophen and the large group of nonsteroidal anti-inflammatory drugs (NSAIDs).

Acetaminophen generally has fewer adverse effects when compared to NSAIDs. It does not affect platelet function, rarely causes gastrointestinal (GI) disturbances, and can be given to patients who are allergic to aspirin or other NSAIDs. Caffeine has been shown to enhance the effectiveness of non-opioid drugs and is often added to the analgesic.

The mechanism of action of acetaminophen is different from that of the NSAIDs but remains unknown; there is some evidence that suggests a central action.

NSAIDs are thought to work primarily at the site of injury by inhibiting the enzyme cyclo-oxygenase (COX), which is required for the synthesis of prostaglandins, substances that sensitize peripheral sensory nerves and contribute to the experience of pain.

The maximum dose for acetaminophen in a 24 hour period is 4 grams.

Opioids:

Their most important effects are on the central nervous system and GI system.

- ✓ These drugs bind to m-opioid receptors, resulting in actions that lead to the analgesic effects. Effects at the membrane level leading to a decrease in neuronal excitability.
- ✓ Opioids increase activity in some neuronal pathways such as the descending inhibitory pathways.
- ✓ The use of opioid therapy in moderate to severe acute pain and cancer pain is well established.

- ✓ **Adjuvant Drugs**:
- ✓ This group of drugs has been approved for use in conditions other than pain. they have been found to be of value in pain management.
- ✓ Amitriptyline (a tricyclic antidepressant), the antidepressant that has been most frequently studied in clinical trials, has been proven to be effective in chronic orofacial pain treatment.
- ✓ The neurotransmitters serotonin and norepinephrine are thought to play a role in the descending inhibitory transmissions from the brain to the dorsal horn, modulating nociceptive impulses. Tricyclic antidepressants (TCAs) block the reuptake of serotonin and norepinephrine (NE), and this is thought to enhance the central inhibitory system in pain processing.
- ✓ These effects occur at doses that are lower than those required for an antidepressant effect.

Anticonvulsant drugs are effective in the treatment of trigeminal neuralgia and diabetic neuropathy and for migraine prophylaxis. These drugs frequently produce side effects (including sedation, dizziness, ataxia, and mood changes) that can limit their usefulness.

Newer anticonvulsant gabapentin is receiving attention as possible therapies for pain.gabapentin has become commonly used in pain management partly because of its relatively few side effects.movement disorders have been reported with gabapentin.the disorders resolve after administration of the drug is stopped.

Topical Medications:

Topical analgesic therapy on the skin or oral mucosa has the advantage of reduced systemic absorption and thus a reduced risk of side effects.

Capsaicin used as a topical cream. It is effective in treating postherpetic neuralgia. Topical application blocks C-fiber conduction, inactivates the release of neuropeptides from peripheral nerve endings.

Facial Neuralgias:

Group of neurologic disorders involving the cranial nerves and are characterized by:

Brief episodes of shooting, often electric shock–like pain along the course of the affected nerve branch.

(b) Trigger zones on the skin or mucosa that precipitate painful attacks when touched.

(c) Pain-free periods between attacks and refractory periods immediately after an attack, during which a new episode cannot be triggered.

These clinical characteristics differ from neuropathic pain, which tends to be;

1- Constant.

2- Burning quality.

3- Absent of trigger zones.

Trigeminal neuralgia:

- 10% of cases have detectable underlying pathology such as a tumor of the cerebellar pontine angle, a demyelinating plaque of multiple sclerosis, or a vascular malformation.
- The remainder of cases of TN are classified as idiopathic.

The most widely accepted theory is that a majority of cases of TN are caused by an atherosclerotic blood vessel pressing on the trigeminal nerve. This pressure results in focal demyelinization and hyperexcitability of nerve fibers, which will then fire in response to light touch, resulting in brief episodes of intense pain.

-evidence for this theory was obtained from a study using tomographic magneyic resonance imaging (MRI), which showed that contactbetween a blood vessel and the trigeminal nerve root was much greater on the affected side .

Clinical features:

- Episodes of intense shooting stabbing pain that lasts for a few seconds and then completely disappears. The pain characteristically has an electric shock–like quality and is unilateral.
- The maxillary branch is the branch that is most commonly affected, followed by the mandibular branch and (rarely) the ophthalmic branch, involvement of more than one branch occurs in some cases.
- Pain in TN is precipitated by light touch on a "trigger zone" present on the skin or mucosa within the distribution of the involved nerve branch.

Common sites for trigger zones include the nasolabial fold and the corner of the lip. Shaving , showering , eating ,speaking ,or even exposure to wind can trigger a painful episode.

- Just after an attack, there is a refractory period when touching the trigger zone will not precipitate pain.

The number of attacks may vary from one or two per day to several per minute.

Management

-combinations of drugs should be attempted before surgery is recommended.

-initially drugs that are effective in eliminating the painful attacks.

-anticonvulsant drugs are most frequently used and are most effective.

-Carbamazepine (Tegretol) is the most commonly used drug and is effective therapy for greater than 85%of newly diagnosed cases of TN. The drug is administered in slowly increasing doses until pain relief has been achived.

- Surgery is indicated when the drug is ineffective or the patient cannot tolerate the side effects of the drugs.

Glossopharyngeal neuralgia:

- It is a rare condition that is associated with paroxysmal pain that is similar to, but less intense than, the pain of TN.
- The location of the trigger zone and pain sensation follows the distribution of the glossopharyngeal nerve (the pharynx, posterior tongue and ear).
- Pain is triggered by stimulating the pharyngeal mucosa during chewing, talking, and swallowing.

Glossopharyngeal neuralgia may occur with TN, and when this occurs, a search for a common central lesion is essential.

- Glossopharyngeal neuralgia also may be associated with vagal symptoms, such as syncope and arrhythmia, owing to the close anatomic proximity of the two nerves.

The application of a topical anesthetic to the pharyngeal mucosa eliminates glossopharyngeal nerve pain and can aid in distinguishing it from the pain of other neuralgias.

-the most common causes of glossopharyngeal neuralgia are intracranial or extracranial tumors and vascular abnormalities that compress CN IX.

-TREATMENT is similar to that for TN, with a good response to carbamazepine and baclofen.

Geniculate neuralgia:

- It is an uncommon paroxysmal neuralgia of facial nerve, characterized by pain in the ear and (less frequently) the anterior tongue or soft palate.

There is often some degree of facial paralysis, indicating the involvement of the motor root.

- Geniculate neuralgia commonly results from herpes zoster of the geniculate ganglion, a condition referred to as Ramsay Hunt syndrome. Viral vesicles may be observed in the ear canal or on the tympanic membrane and facial paralysis.

The symptoms result from inflammatory neural degeneration, and a short course (2 to 3 weeks) of high-dose steroid therapy is beneficial. Acyclovir significantly reduces the duration of the pain. Patients with geniculate neuralgia are also treated with carbamazepine and antidepressants.if not responed then undergo surgery.

Occipital neuralgia:
- It is a rare neuralgia in the distribution of the sensory branches of the cervical plexus.
- The most common causes are trauma, neoplasms, infections, and aneurysms involving the affected nerve.
-palpation below the superior nuchal line may reveal a tender spot.

postherpetic neuralgia:
- Herpes zoster (shingles) is caused by the reactivation of latent varicella-zoster virus infection that results in both pain and vesicular lesions along the course of the affected nerve.
- Persistent pain, paresthesia, hyperesthesia, and allodynia months to years after the zoster lesions have healed.
- Approximately 15 to 20% of cases of herpes zoster involve the trigeminal nerve although the majority of these cases affect the ophthalmic division of the fifth nerve, resulting in pain and lesions in the region of the eyes and forehead.
- In a majority of cases, the pain of herpes zoster resolves within a month after the lesions heal. Pain that persists longer than a month is classified as postherpetic neuralgia (PHN) although some authors do not make the diagnosis of PHN until the pain has persisted for longer than 3 or even 6 months.
- The combination of peripheral and central injury by varicella-zoster virus results in the spontaneous discharge of neurons and an exaggerated painful response to nonpainful stimuli.

- antiviral drugs, particularly famciclovir, along with a short course of systemic corticosteroids during the acute phase of the disease.
- topical anesthetic agents, such as lidocaine, or analgesics, particularly capsaicin.
- tricyclic antidepressants such as amitriptyline and nortriptyline.

- Gabapentin, carbamazepine or phenytoin.

Post-traumatic neuropathic pain: A neuroma is an incomplete or failed attempt at nerve repair following injury to a peripheral nerve, resulting in a disorganized nerve fiber that is focally electrically excitable.
The pathologic sensitization of the injured peripheral nerve or CNS results in both peripheral and CNS hyperexcitability, which are manifest as allodynia (pain caused by a stimulus that is normally not painful), or hyperalgesia (an exaggerated response to a mildly painful stimulus).
Trigeminal nerve injuries may result from facial trauma or from surgical procedures,such as the removal of impacted third molars,the placement of dental implants ,the removal of cysts or tumors of the jaws, genioplasties, or osteotomies.
Minor nerve damage (classified as axonotmesis) results in the degeneration of neural fibers (axons) within an intact nerve shealth.these injuries cause symptoms for several months but have a good prognosis for recovery after axonal regeneration is complete.
-total nerve section (neurotmesis) frequently causes permanent nerve damage, resulting in anesthesia and / or dysthesia.
Clinical manifestations:

The pain associated with peripheral nerve injury may be persistent or may occuer only in response to a stimulus such as light touch.
Patient with nerve damage may experience anesthesia (loss in sensation),paresthesia (a feeling of pins and needles),allodynia,or hyperalgesia.

Management:
- Systemic corticosteroids are considered helpful in decreasing the incidence and severity of traumatic neuropathies when administered within the first week after a nerve injury.
- The tricyclic antidepressants (TCAs) such as amitriptyline and nortriptyline can be used alone; in severe intractable cases, they potentiate the effect of narcotic analgesics.
- Gabapentin, an anticonvulsant drug.
- Topical capsaicin may also be effective in controlling pain

Atypical Facial Pain: