IN THE NAME OF GOD

Facial Pain

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Facial pain

1. Trigeminal and other cranial nerve neuropathic conditions (Trigeminal neuralgia, Glossopharyngeal neuralgia, Nervus intermedius neuralgia)
2. Odontogenic and TMJ disorders

3. Chronic headache disorders causing facial pain

Trigeminal neuropathy

- Trigeminal neuropathy is a spectrum (based on pathophysiology of neuralgia):

1. Trigeminal neuralgia type 1 (**classic form**)
2. Trigeminal neuralgia type 2
3. Symptomatic Trigeminal neuralgia
4. Neuropathic Trigeminal neuralgia
5. Postherpetic Trigeminal neuralgia
6. Deafferentation Trigeminal neuralgia
7. Atypical facial pain

Trigeminal neuralgia type 1 (**classic description**)

- Idiopathic
- Sharp, shooting, electrical shock-like
- Episodic pain lasting several seconds
- Pain-free interval between attacks

➢ **Pathology**: **vascular compression near the root entry zone** (junction of the central and peripheral myelin that susceptible to the pathology)

- Vascular compression → **demyelination** → ectopic action potential generation → sharp lancinating episodes of facial pain.

Trigeminal neuralgia type 2

- A variant of classic form (idiopathic)
- ➢ **The Crucial features**: **dull aching or background pain for more than 50% of time.**
- More evidence of **sensory** neuropathy compared to type 1.
- Some of these patients do progress from type 1, and some present as such initial presentation.
- **Success of surgery**: type 1 > type 2
- Idiopathic forms → **normal bedside sensory examination** (fairly common/ regardless of the type)

Presentation of TN type 1 and 2

- Type 1: **sharp stabbing** episode pain > 50% of the time

- Type 2: sharp stabbing < 50% with **predominant component of dull aching or burning pain**

Symptomatic Trigeminal neuralgia

- Not an idiopathic form
- **Due to**:
- MS (demyelinating disorder)
- Tumor
- Vascular malformation (AVM, aneurysm)
- Structural pathology leading to compression of trigeminal root entry zone (other than idiopathic form)
- **Presentation**: either type 1 or type 2; however, **constant dull aching pain (type 2 like) is more common**.

Neuropathic Trigeminal neuralgia

- The degree of trigeminal neuropathy is **more advanced** compared to idiopathic form
- Objective sensory loss in the distribution of trigeminal nerve
- **Not limited** to allodynia, hyperalgesia, and burning pain

➢ **Caused by**:

1. **Unintentional injury**: trauma, postdental injection, postprocedural in maxillofacial surgeries, ENT surgery, poststroke
2. **Intentional injury**: neurosurgical ablation of the one or more branches of TN
3. **Spontaneous neuropathic trigeminal neuralgia**: Rare

Postherpetic & Deafferentation TN

- **Postherpetic Trigeminal neuralgia**
- **More severe** form
- Significant allodynia, hyperalgesia, burning dysesthesia
- Commonly affects **V1** of the TN
- **Deafferentation Trigeminal neuralgia**
- Known as anesthesia dolorosa
- **Most severe** form of TN
- Marked sensory loss or even corneal anesthesia. in severe cases.
- An iatrogenic condition (induced by surgical interventions to denervate the trigeminal distribution)
- **Common**: rhizotomy or alcohol denervation
- **Less common (or require repeated exposure):** RF or glycerol rhizotomy, balloon compression, or radiosurgery

Atypical facial pain

- **Refer to somatoform disorder**
- A psychological disorder can be unequivocally diagnosed.
- This is a difficult condition to treat and would be a contraindication to interventional therapies.

Treatment of TN

- The **degree** of neuropathy determine the possible **reversibility** and therefore dictates the treatment options.
- Medical treatments for most of cranial nerve neuropathy-related pain is **medical at first** (carbamazepine, gabapentin, topiramate)
- Medical treatment fail → interventional Treatment (based on the **degree** of neuropathy)
- **Early neuropathy** such as type 1→ microvascular decompression
- **Postherpetic TN** → advanced intervention such as neuromodulation of CNS

Carbamazepine

- Is considered first-line treatment for TN.
- A rapid response to this drug essentially confirms the clinical diagnosis.
- **Baseline measurements before starting the drug** : CBC, UA, blood chemistry
- **Starting dose: 100-200 mg** at bedtime for 2 nights.
- The drug is increased in 100-200 mg increments given in equally divided doses over 2 days.
- **Total dose: 1200 mg/day .**
- **Side effects**: dizziness, sedation, confusion, rash, life-threatening **blood dyscrasia** (**aplastic anemia** / **rare**)
- Careful monitoring of **laboratory** parameters is **mandatory.**
- **At the first sign of blood count abnormality or rash, this drug should be discontinued.**
- When pain relief is obtained, the patient should be kept at that dosage of carbamazepine for **at least 6 months before considering tapering** the medication.

- **Gabapentin** :
- Baseline blood tests should be obtained before starting.
- **Side effects**: dizziness, sedation, confusion, and rash.
- **The initial dose**: 300 mg at bedtime for 2 nights.
- The drug is then increased in 300-mg increments given in equally divided doses over 2 days,
- **Total dose: 2400 mg/day**
- Rarely is a dosage greater than 3600 mg/day required.
- **Baclofen**:
- In patients who **fail** to obtain relief from carbamazepine or gabapentin.
- Baseline laboratory tests should be obtained before beginning.
- **The initial dose**: 10-mg dose at bedtime for 2 nights; then, the drug is increased in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a **total dose of 100 mg/day** is reached.
- **Side effects**: hepatic and CNS (weakness and sedation).
- **Premature tapering or discontinuation of the medication may lead to the recurrence of pain, which will be more difficult to control**.

Invasive Therapy

- **Trigeminal Nerve Block**
- **Retrogasserian Injection of Glycerol**
- **Radiofrequency Destruction of the Gasserian Ganglion → CRF**
- **Balloon Compression of the Gasserian Ganglion**.
- **Microvascular Decompression of the Trigeminal Root: (**Jannetta's procedure)

• is the major neurosurgical treatment of choice for intractable TN.

- **Gamma Knife:**

• a painless, outpatient procedure

• Uses the focused emission of **gamma rays** from a cobalt source to **destroy** the area anterior to the junction of the trigeminal nerve and the pons, the trigeminal nerve entry site immediately adjacent to the pons, the midposterior portion of the trigeminal nerve, or the cisternal segment of the trigeminal nerve.

• **Complications**: Facial numbness, sensory deficit

Atypical facial pain = Atypical facial neuralgia

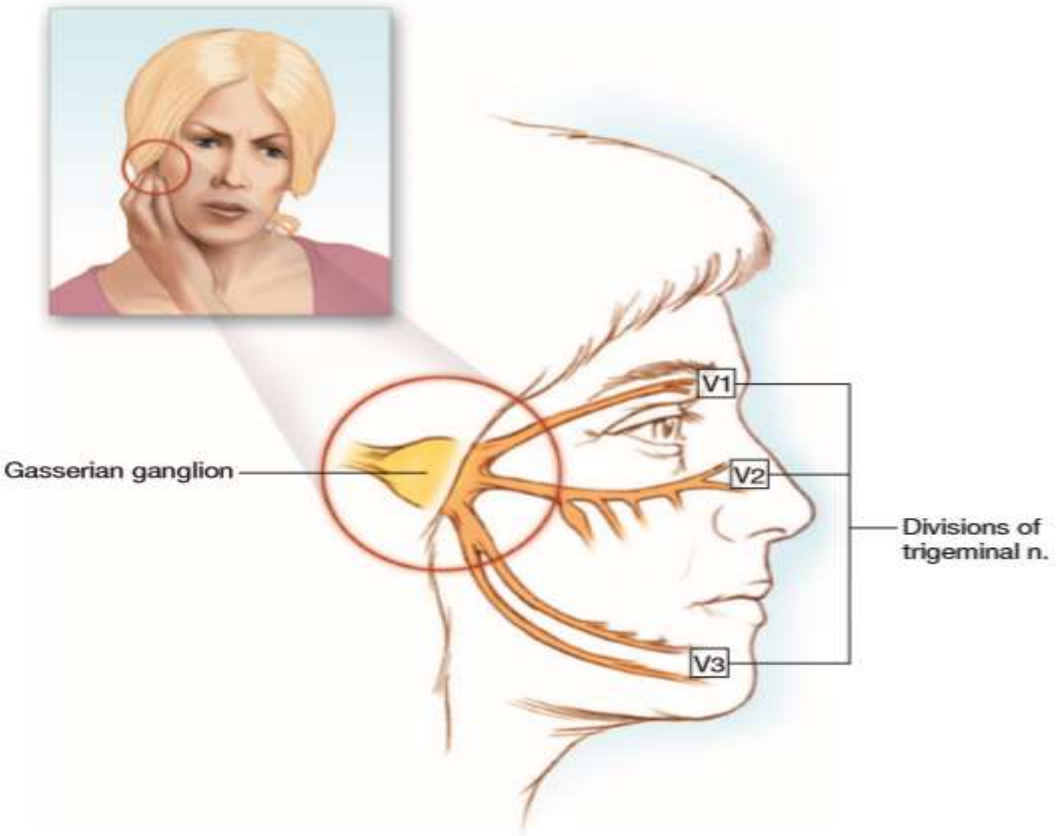
- **The pain type**: **continuous** (but may vary in intensity), **almost always unilateral**, **aching** or **cramping** (rather than the shocklike neuritic pain typical of trigeminal neuralgia).
- **Gender**: **Female** > male
- **Pain location**: in the distribution of the trigeminal nerve (but invariably **overlaps** the divisions of the nerve)
- **Headache** often accompanies it (clinically indistinguishable from TTH).
- **Precipitating factor** : **Stress** (exacerbating factor), Depression, sleep disturbance, facial trauma, infection, or tumor of the head or neck; ***BUT***:

➢ **In most cases, no precipitating event can be identified**.

Comparison of trigeminal neuralgia and atypical facial pain

	Trigeminal Neuralgia	Atypical Facial Pain
Temporal pattern of pain	Sudden and intermittent	Constant
Character of pain	Shocklike and neuritic	Dull, cramping, aching
Pain-free intervals	Usual	Rare
Distribution of pain	One division of trigeminal nerve	Overlapping divisions of trigeminal nerve
Trigger areas	Present	Absent
Underlying psychopathology	Rare	Common

Patients with **atypical facial pain** often **rub** the affected area; those with **trigeminal neuralgia** do **not**.



Atypical facial pain

TESTING

- **Radiographs of the head**:(tumor or bony abnormality),are usually within normal limits
- **MRI of the brain and sinuses**: tumor, sinus disease, and infection.
- **CBC, ESR, ANA**: if inflammatory arthritis or temporal arteritis is suspected.
- **Injection of the TMJ** (diagnostic maneuver to determine whether the TMJ is the source of the patient's pain).
- **MRI of the cervical spine**: if the patient is experiencing significant occipital or nuchal pain.

DIFFERENTIAL DIAGNOSIS

- Pain of dental or sinus origin
- Trigeminal neuralgia.
- Tumors (zygoma and mandible, posterior fossa and retropharyngeal tumors)
- Temporal arteritis
- **RSD** of the face (ill-defined facial pain after trauma, infection, or CNS injury).
  - ➢ RSD of the face causes **burning pain**, and **significant allodynia.**
  - ➢ **Stellate ganglion block** may help **distinguish** these two pain syndromes:
  - ❖ The pain of RSD of the face : **readily responds** to STG block
  - ❖ Atypical facial pain: **does not.**

Treatment of atypical facial pain

- **The mainstay of therapy**: TCA + physical modalities such as oral orthotic devices + physical therapy.
- **Trigeminal nerve block**
- **Intraarticular injection of the TMJ**
- **Sleep disturbance** and any underlying **myofascial pain syndrome**: Antidepressants such as nortriptyline / bedtime / 25 mg .
- **Orthotic devices**: avoid jaw clenching and bruxism
- Management of underlying depression and **anxiety**: **is mandatory**.
- **Topical agents**: capsaicin, lidocaine
- **Botulinum toxin A**: into the muscle of mastication
- **Case reports** suggest that a single inhalation of 25 mg of 9.4 **tetrahydrocannabinol** three times daily may alleviate **neuropathic facial pain.**
- **Opioid analgesics and benzodiazepines should be avoided in patients suffering from atypical facial pain.**

Reflex Sympathetic Dystrophy of the Face (Chronic Regional Pain Syndrome type I of the face)

- An infrequent cause of face and neck pain.
- The diagnosis is often missed.
- **The common denominator**: trauma (soft tissues, dentition, or bones of the face; infection; cancer; arthritis)
- **The hallmark: burning pain**.
- **Allodynia** (cutaneous or mucosal)
- Pain does not follow the path of either the cranial or the peripheral nerves.
- **Trigger areas** (especially in the oral mucosa): common
- **Trophic skin and mucosal changes**
- **Sudomotor and vasomotor changes** (often **less obvious** than in patients suffering from RSD of the extremities).
- **Often, patients with RSD of the face have evidence of previous dental extractions performed in an effort to achieve pain relief.**
- **significant sleep disturbance and depression**

RSD of the face

- No specific test exists for RSD.
- A presumptive diagnosis can be made if the patient experiences significant pain relief after STG block.
- MRI of the brain and the cervical spine (if significant occipital or nuchal symptoms are present)
- CBC, ESR, blood chemistry (to rule out infection or other inflammatory causes of tissue injury)

- **Differential diagnosis:**
- Pain of dental or sinus origin
- Trigeminal neuralgia
- Atypical facial pain

➢ **STG block:**

✓ **RSD readily responds**

❖ **Atypical facial pain: does not**

- Zygoma and mandible tumors
- Posterior fossa and retropharyngeal tumors (**must be excluded in any patient** with facial pain).
- Temporal arteritis.

	Trigeminal Neuralgia	Atypical Facial Pain	RSD of the Face
Temporal pattern of pain	Sudden and intermittent	Constant	Constant
Character of pain	Shocklike and neuritic	Dull, cramping, aching	Burning with allodynia
Pain-free intervals	Usual	Rare	Rare
Distribution of pain	One division of trigeminal nerve	Overlapping divisions of trigeminal nerve	Overlapping divisions of trigeminal nerve
Trigger areas	Present	Absent	Present
Underlying psychopathology	Rare	Common	Common
Trophic skin changes	Absent	Absent	Present
Sudomotor and vasomotor changes	Absent	Absent	Often present

Treatment of RSD of the face

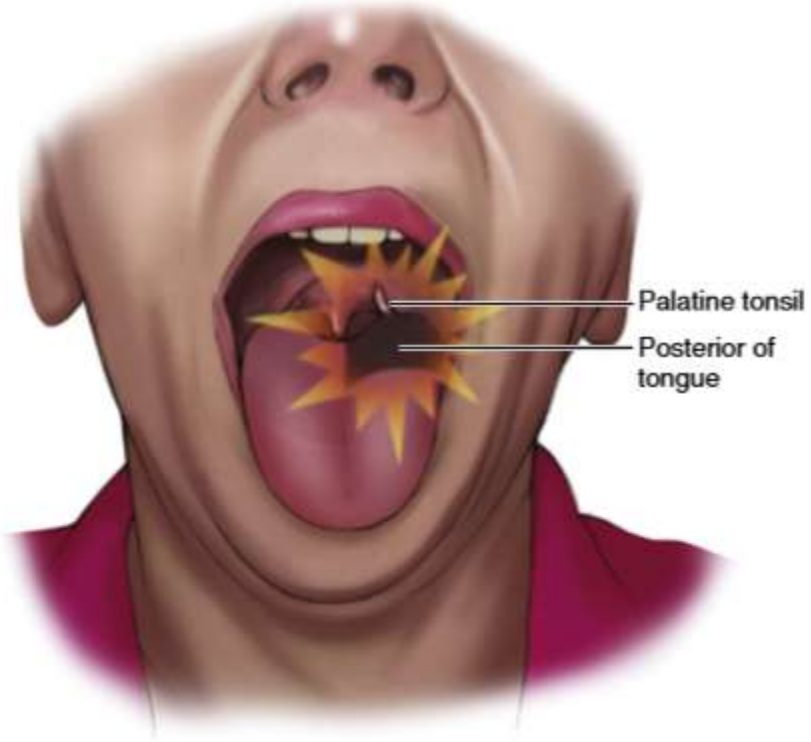
- **Two phases:**
- **First**: Any nidus of tissue trauma must be identified and removed.
- **Second**: Interruption of the sympathetic innervation of the face by means of STG block (daily STG block for a significant period).
- **Occupational therapy** (tactile desensitization of the affected skin)
- **Treatment of Underlying depression and sleep disturbance**: TCA , nortriptyline, 25-mg / at bedtime.
- **Gabapentin**: neuritic pain / 300 mg, bedtime, maximum dose of 3600 mg/day.
- **Pregabalin**: 50 mg, TDS, to 100 mg TDS. (alternative of gabapentin)
- ✓ Pregabalin is excreted primarily by the kidneys, the dosage should be decreased in patients with compromised renal function.
- **Intravenous infusion of mannitol** (to scavenge free radicals): larger studies have **failed** to demonstrate its efficacy.
- **Opioid analgesics and benzodiazepines should be avoided to prevent iatrogenic chemical dependence.**

Glossopharyngeal neuralgia (GN)

- A rare condition (incidence: 1%).
- **Presentation**: **paroxysmal** episodes of pain along the **auricular** and **pharyngeal** branches of both **glossopharyngeal** and **vagus** nerves.
- Age > 50 yr
- Unilateral (98%)
- **Severe lancinating pain** attacks along one side of the **throat**, with occasional radiation to **ear**.
- Precipitated by swallowing, chewing, coughing, or yawning.
- **Debilitating** when the **vagus nerve** is involved as patients may experience loss of sympathetic tone that can lead to bradyarrhythmias , syncope and seizures in **up to 10%** of the cases.

Glossopharyngeal neuralgia (GN)

- **Localization of pain**: tonsil, laryngeal region, posterior tongue.
- **Severe pain**
- Neurological examination should be normal.
- **Dull**, **aching** pain that persists between the paroxysms of pain is highly suggestive of a **space occupying lesion**.



Glossopharyngeal neuralgia (GN)

- Most often caused by:
- ➢ Neurovascular compression at the root entry zone of the brain stem.
- Posterior inferior cerebellar artery (**PICA**): **most often**
- Anterior inferior cerebellar artery (AICA)
- ➢ **Testing**:
- MRI for R/O any other causes such as neoplasm (cerebellopontine angle)or an elongated styloid process.
- MRI, MRA
- CT-scan
- CBC, blood chemistry, ESR (to rule out infection, temporal arteritis, malignancy)
- Endoscopy of the hypopharynx and piriform sinuses (rule out occult malignancy).
- Diagnostic glossopharyngeal nerve block

Differential diagnosis and Treatment

DDx

- Diseases of the eye, ENT and teeth
- Tumors: hypopharynx (tonsillar fossa, piriform sinuses), cerebellopontine angle
- Demyelinating disease
- Temporal arteritis
- Trigeminal neuralgia

Treatment

- ❑ **Medical**: similar most cranial neuralgias → carbamazepin, gabapentin, baclofen
- ❑ **Interventional Tx:**
- Glossopharyngeal Nerve Block
- RF Destruction
- Gamma knife ablation
- Microvascular Decompression of the Glossopharyngeal Root

Nervus Intermedius Neuralgia (NIN)

- Also known as **geniculate neuralgia**
- An **uncommon** cause of primary otalgia
- **Paroxysmal, deep ear pain.**
- Be caused by compression of the **nervus intermedius** portion of the cranial nerve VII (**facial**) by aberrant blood vessels or tumor.
- Patients describe the pain as "being stabbed in the ear with an icepick."
- Attacks can be debilitating and socially isolating and associated with suicide (**should be treated as an emergency).**
- **Cranial nerve VII (facial):** primarily motor nerve (innervate the facial muscles) + small number of sensory and parasympathetic fibers .
- **Sensory innervations** : the skin of the external auditory meatus, portions of the nasal and nasopharyngeal mucosa, and the anterior two-thirds of the tongue.
- NIN and its associated geniculate ganglion are infected with HZV: Ramsey Hunt syndrome
- **Pain type: severe**
- **Trigger zone:** The posterior wall of the auditory canal, the superficial ear drum
- **Triggers**: loud noises, cold wind, contact with the external acoustic meatus or auricle,lying on the affected side .

NIN

- Disorders of lacrimation, salivation, and taste have also been reported in these patients.
- microvascular decompression or surgical resection: ~ 50% of cases.
- Sensory innervation to the skin of the external ear and auditory canal:

CN V, IX, X, the nervus intermedius, upper cervical dorsal root

- ➢ This complex innervation leads to confusion about the culprit nerve in primary otalgia and confusion in diagnosis.

NIN

- **Unilateral, electric shock–like pain**
- **Pain duration**: several seconds to less than 2 minutes.
- Avoid any contact with trigger areas.
- Relatively pain free Between attacks
- A dull ache remaining after the intense pain subsides may indicate persistent compression of the nerve by a structural lesion.
- **Almost never seen in persons younger than 30 years**.
- Often have severe depression and anxiety

NIN

Testing

- **MRI** of the brain and brainstem, with and without gadolinium (to R/O posterior fossa or brainstem lesions and demyelinating disease)
- **MRA** (is also useful to confirm vascular compression).
- **Functional MRI** (anatomical location of the pathophysiology)
- **Imaging of the sinuses**
- **Ophthalmological evaluation** (IOP and intraocular pathology)
- CBC, ESR, Blood chemistry

Differential diagnosis

1. Diseases of the eyes, ENT, and teeth
2. Atypical facial pain (dull and aching pain)
3. TMJ disorders
4. Multiple sclerosis (MS) (**should be considered in all patients who present with NIN before the fifth decade of life**).

Drug therapy of NIN

- **Carbamazepine:**
- **first-line treatment**
- **A rapid response to this drug helps confirm the clinical diagnosis.**
- CBC, UA and blood chemistry: before starting the drug.
- **starting dose**: 100 -200 mg / at bedtime for 2 nights..
- The drug is increased in 100-200 mg increments given in equally divided doses over 2 days, as side effects allow, until pain relief is obtained or a **total dose of 1200 mg per day is reached.**
- Careful monitoring of laboratory parameters is **mandatory** to avoid the rare possibility of a life-threatening blood dyscrasia.
- At the first sign of blood count abnormality or rash, this drug should be discontinued.
- When pain relief is obtained, the patient should be **kept at that dosage of carbamazepine for at least 6 months** before tapering of the medication is considered.
- **Side effects**: dizziness, sedation, confusion, rash, blood dyscrasia(life-threatening)

Drug therapy of NIN

- **Gabapentin**:
- **The initial dose**: 300 mg at bedtime for 2 nights.
- increased in 300-mg increments given in equally divided doses over 2 days,until pain relief is obtained or a **total dose of 2400 mg/day** is reached. **Rarely** is a dosage greater than 3600 mg/day required.
- **side effects**: dizziness, sedation, confusion, and rash
- **Pregabalin**:
- **The Initial dose**: 50 mg TDS and may be titrated **upward to 100 TDS** as side effects allow.
- Excreted primarily by the **kidneys** → the dosage should be decreased in patients with compromised renal function.
- **Baclofen**:
- May be of value in some patients **who fail to obtain relief from carbamazepine, gabapentin, or pregabalin**.
- Baseline laboratory tests should be obtained before beginning.
- **The initial dose**: 10 mg, at bedtime for 2 nights, then increase the drug in 10-mg increments given in equally divided doses over 7 days, as side effects allow, until pain relief is obtained or a **total dose of 100 mg /day** .
- **Side effects**: significant hepatic and CNS side effects (weakness , sedation).

➢ **Carbamazepin, gabapentin, pregabalin, baclofen: premature tapering or discontinuation of the medication may lead to the recurrence of pain, which will be more difficult to control.**

Invasive Therapy of NIN

- ❑ Microvascular Decompression of the NIN and/or Geniculate Ganglion
- ❑ Section of the NIN
- ➢ **choice** for those patients who have failed to respond to drug therapy and microvascular decompression procedures.
- ➢ Excellent palliation of pain in 75% to 90% of cases.

❖ **NOTE**:

- ✓ The pain of NIN is **severe** and can lead to **suicide**; therefore it must be considered a **medical emergency (** hospitalizing such patients).
- ✓ **If a dull ache remains between the intense paroxysms of pain**, the clinician should have a high index of suspicion that the **nidus** of the patient's pain is **persistent** compression of the nerve by a structural lesion such as a brainstem tumor or schwannoma.

Hyoid Syndrome

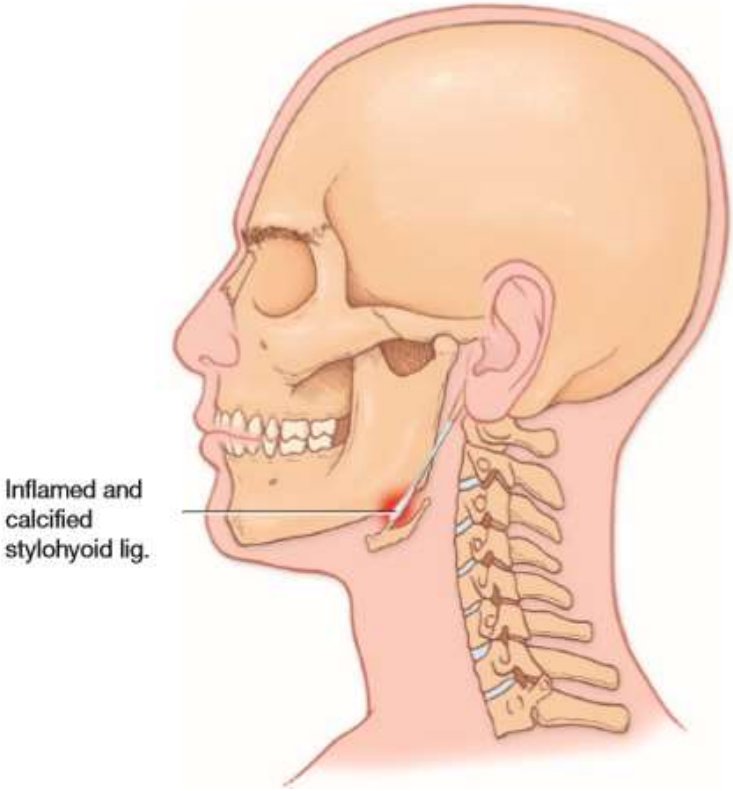
- Caused by calcification and inflammation of the attachment of the stylohyoid ligament to the hyoid bone.
- The stylohyoid ligament attachment :
  - cephalad → styloid process
  - caudad → hyoid bone

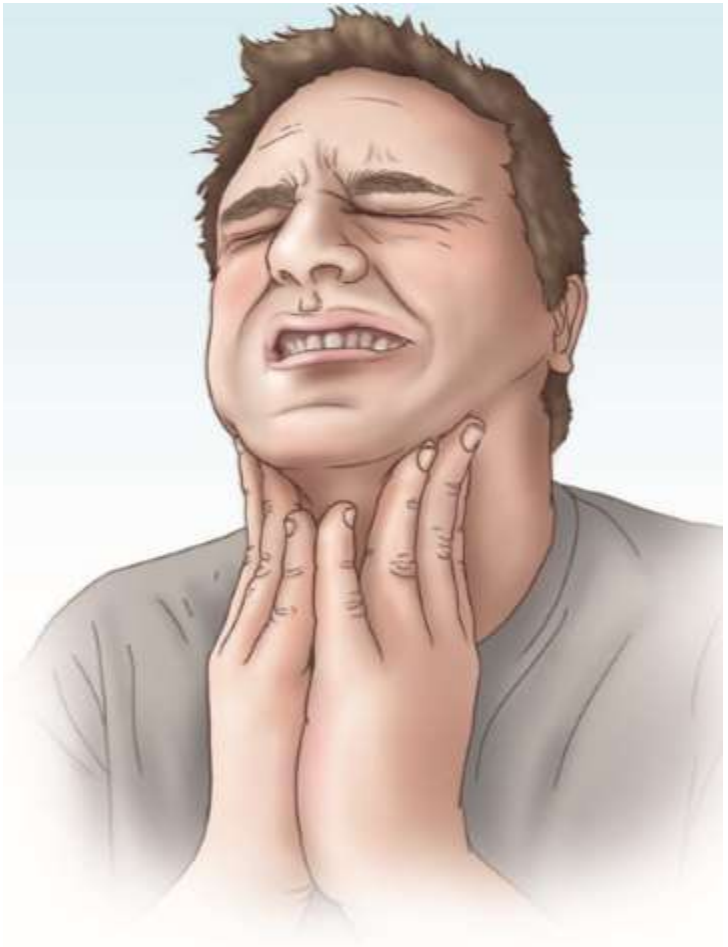
❖ Tendinitis of the other muscular attachments to the hyoid bone may contribute to this painful condition.

❖ May be seen in conjunction with Eagle's syndrome.

❖ Patients suffering from diffuse idiopathic skeletal hyperostosis (**DISH**) are thought to be susceptible to this syndrome.

Sharp and **stabbing** pain / occurs with movement **of the mandible**, **turning of the neck**, or **swallowing**. / The pain **starts below the angle of the mandible** and **radiates** to the **anterolateral neck**.





- The pain is often referred to the **ipsilateral ear**.
- Some patients complain of a **foreign body sensation in the pharynx.**
- Injection of L.A and steroid into the **attachment** of the stylohyoid ligament to the greater cornu of the hyoid bone is both a diagnostic and a therapeutic maneuver.

Testing & DDx

- **No specific test exists for hyoid syndrome.**
- Plain radiography, CT and MRI of the neck: calcification of the caudad attachment of the stylohyoid ligament at the hyoid bone.
- CBC, ESR, ANA
- Injection of L.A into the attachment of the stylohyoid ligament to the hyoid bone .
- If **difficulty swallowing** is a prominent feature → **endoscopy** of the esophagus, with special attention to the gastroesophageal junction, is **mandatory** to identify esophageal **tumors** or **strictures** resulting from gastric reflux.

DIFFERENTIAL DIAGNOSIS:

- Glossopharyngeal neuralgia
- Retropharyngeal tumor
- Retropharyngeal
- Abscess
- Osteomyelitis of the hyoid bone
- Atypical facial pain
- Mandibular tumor
- Esophageal disease
- Jaw claudication of temporal arteritis
- Sternohyoid muscle syndrome

➢ Present as a mass in the lower lateral neck that appears when the patient swallows and then disappears after swallowing is completed.

Treatment

- Is best treated with L.A and steroid injection of the attachment of the stylohyoid ligament.

➢ Owing to the **vascularity** of this area and the **proximity to neural structures**, **this technique should be performed only by those familiar with the regional anatomy .**

- NSAIDs: in mild cases
- Antidepressants (nortriptyline, 25 mg, bedtime): Sleep disturbance, underlying myofascial pain syndrome.

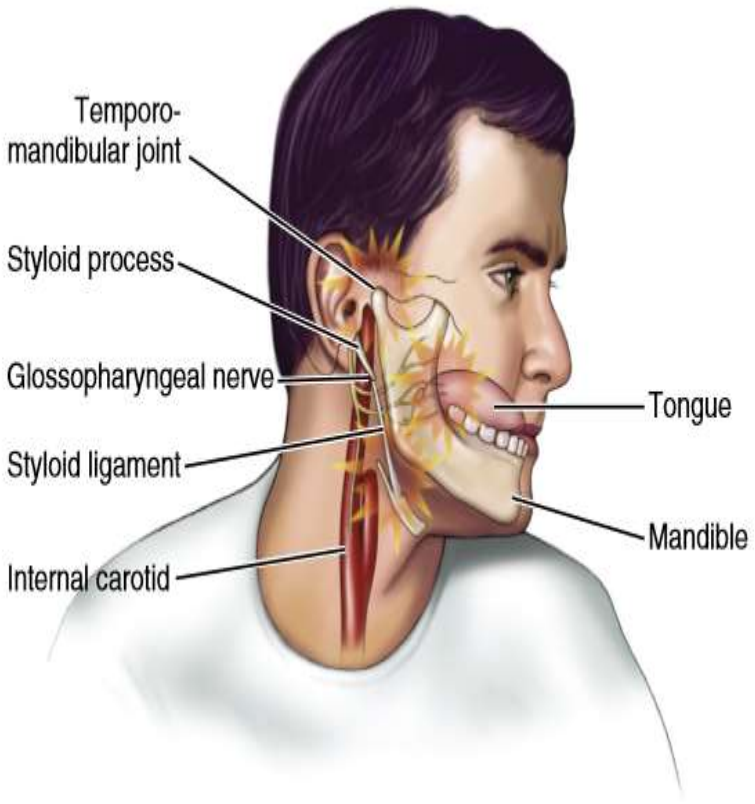
Eagle syndrome (stylohyoid syndrome)

- An **uncommon** cause of facial pain.
- Is caused by **pressure** on the **internal carotid artery and surrounding structures**, including branches of the glossopharyngeal nerve, by an abnormally **elongated styloid process**, a **calcified stylohyoid ligament, or both**.
- **The pain type**: sharp and stabbing
- **Occurs with movement of the mandible or turning of the neck**.
- The Pain **starts below the angle of the mandible**.
- Pain **radiates** into the **tonsillar fossa**, **TMJ**, and **base of the tongue**.
- A **trigger point** may be present in the **tonsillar fossa**.
- Injection of the attachment of the stylohyoid ligament **to the styloid process** with L.A and steroid serves as a diagnostic maneuver and a therapeutic maneuver.

Eagle syndrome (stylohyoid syndrome)

- **The pain is triggered by**: swallowing, movement of the mandible, or turning of the neck, palpation of the tonsillar fossa.

- **Pain intensity**: moderate to severe
- The neurological examination: normal



Testing and DDx of Eagle syndrome

testing

- Radiographs and CT scans: elongated styloid process calcified stylohyoid ligament
- Diagnostic injection: attachment of the stylohyoid ligament to the **styloid process**

❖ **Pain relief** after this injection **suggests a *local cause* for the pain** rather than a **more distant cause**, such as glossopharyngeal neuralgia or retropharyngeal tumor.

DDx

- Glossopharyngeal neuralgia

➢ **Because glossopharyngeal neuralgia may be associated with serious cardiac bradyarrhythmias and syncope, the clinician must distinguish the two syndromes**.

- occult malignancy
- Tumors of the larynx, hypopharynx, and anterior triangle of the neck
- **Eagle syndrome must be considered a diagnosis of exclusion.**

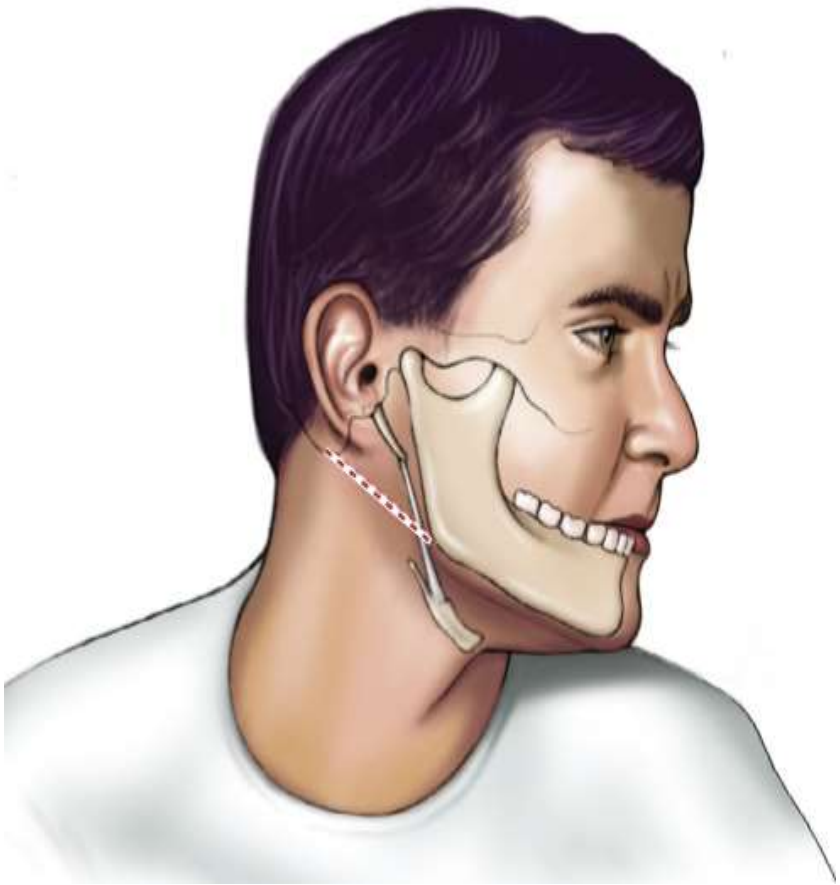
Treatment of Eagle syndrome

- Injections of the attachment of the stylohyoid ligament to the styloid process

1. An imaginary **line** is visualized running from the **mastoid process to the angle of the mandible.**
2. The **styloid process** should lie just below the **midpoint of this line**.
3. P&D of skin
4. A **22-gauge**, **1½inch** needle attached to a 14-mL syringe is advanced at this midpoint location in a plane perpendicular to the skin.
5. **The styloid process should be encountered within 3 cm.**
6. After contact is made, the needle is withdrawn slightly out of the periosteum or substance of the calcified ligament.
7. After careful aspiration reveals no blood or CSF, **5 mL** of 0.5% preservative-free lidocaine combined with 80 mg of methylprednisolone is injected in incremental doses.
8. Subsequent daily nerve blocks are performed in a similar manner, substituting 40 mg of methylprednisolone for the initial 80-mg dose.

- Ultrasoundguided: improve the accuracy of needle placement and decrease the incidence of needle-related complications.

COMPLICATIONS AND PITFALLS



- trauma to the internal jugular and carotid artery
- Hematoma
- intravascular injection of L.A and toxicity
- Inadvertent blockade of the motor portion of the glossopharyngeal nerve (dysphagia secondary to weakness of the stylopharyngeus muscle).
- Inadvertent blockade of the vagus nerve (dysphonia secondary to paralysis of the ipsilateral vocal cord ,A reflex tachycardia)
- Inadvertent block of the hypoglossal and spinal accessory nerves (weakness of the tongue and trapezius muscle)

Treatment of Eagle syndrome

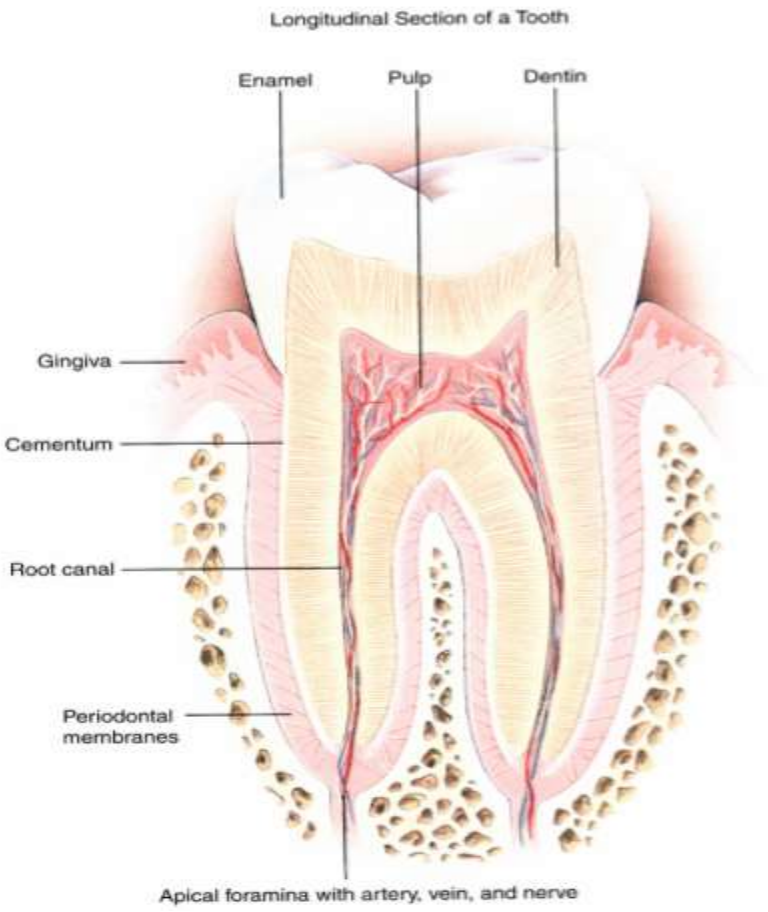
- ❑ **Surgical** excision of the styloid process and calcified stylohyoid ligament
- ❑ Drug therapy for sharp, shooting pain:
- **Gabapentin**: started: 300 mg, bedtime → titrated by 300-mg increments every 2 days in divided doses → until pain relief is achieved or a total daily dose of 3600 mg.
- ❑ **Alternative** for gabapentin:

carbamazepine, phenytoin

Odontogenic pain

- The Normal anatomy of a tooth:
  - The crown: enamel, dentin, dental pulp
  - The root: cementum, dentin, dental pulp

Dental pulp: generative part of the tooth, and is made up connective tissue and generative cells (odontoblasts) and innervated and can cause dental pain.



Sources of Odontogenic pain

1. Dental caries
2. Tissues surrounding the dentition

➢ **Dental carries**:

Enamel → dentin → pulp → infection and inflammation → acute episode of pain → abscess formation

- Once the dental pulp becomes **necrotic**, the pain may **subside**.

❖ **Radiographic examination**: radiolucencies within the tooth structure

Pain from the tissues surrounding the dentition

➢ **Periodontitis:**

- Chronic, periradicular disease
- inflammation of gingiva causing soft tissue loss and bone loss surrounding the tooth.
- In **early** stages, may be **painless**.
- If untreated, culminates in mobility of teeth and ultimately tooth loss.
- In the **absence of abscess** formation, **pain is typically mild** and **not limited to one particular area of the mouth**.
- Associated with other systemic and chronic disease such as DM.

Pericoronitis

- A type of gingival inflammation
- Typically occurs around an erupting or partially erupted third molar (**wisdom tooth**).
- Can cause **severe pain** and **trismus**.
- May lead to **severe** cellulitis and abscess spreading to the deep space of the neck and posterior oropharynx.
- **Constant aching or throbbing, moderate to severe pain.**

Pericoronitis

- **Pain characteristics**: constant aching or throbbing, moderate to severe pain
- **Localization of pain**: depending of **severity** of pain, can be localized to the posterior mandible or can be diffuse to include the entire side of the face extending to the neck
- **Factors that aggravate**: chewing/functioning
- **Examination findings**: erythematous, edematous mucosa overlying the third molar area. Purulence may be present. Patient may exhibit **trismus**. In more severe cases, the patient may exhibit malaise, fever, and abscess formation extending into the neck.
- **Radiographic findings**: submerged or impacted wisdom tooth.

Dentin sensitivity

- **Pain characteristics**: **mild to moderate**; ceases when aggravant is removed.
- **Localization of pain**: the affected teeth
- **Factors that aggravate**: **cold**
- **Examination findings**: gingival recession
- **Radiographic findings**: none

Reversible pulpitis

- **Pain characteristics**: sharp, throbbing, **moderate to severe, intermittent**
- **Localization of pain**: affected tooth
- **Factors that aggravate**: **cold/heat, sweets, chewing/percussion**
- **Examination findings**: dental caries or dental trauma (fractured tooth)
- **Radiographic findings**: caries or fracture appropriating the pulp

Irreversible pulpitis

- **Pain characteristics**: sharp, throbbing, **moderate to severe, can be constant**
- **Localization of pain**: generalized to ipsilateral side of face or jaw
- **Factors that aggravate**: **cold/heat, sweets, chewing/percussion**
- **Examination findings**: caries, may exhibit gingival or facial swelling
- **Radiographic findings**: caries appropriating or invading pulp, widened periodontal ligament or periapical radiolucency

Note:

- The decay → invasion to the pulp → pain
- Mobility of a tooth in the presence of pain may indicate a **periapical abscss** or **chronic periodontitis.**

✓ **Abscess**: typically limited to one tooth or one area of the mouth.

✓ **Chronic periodontitis**: multiple teeth usually are affected in several areas of the mouth.

Temporomandibular (TMJ) disorders

- ✓ The **most common** musculoskeletal disorder that cause orofacial pain.
- TMJ disorders can typically be separated into **2 entities**:

1. Myofacial Pain Disorders (MPD)
2. Interarticular disorders (internal derangements) [ID]

- These 2 entities can do coexist.

- ➢ **ID**: damaged intra-articular tissues leading to disturbance in the biomechanical functioning of the TMJ.
- ➢ **MPD**: regional muscle pain disorder characterized by localized muscle tenderness, limited ROM, and regional pain.

Myofacial pain disorders (MPD)

1. Trigger points in muscle bands
2. Tenderness to muscle on palpation
3. Consistent points of tenderness
4. Pain in zone of referral/reference
5. **Constant** pain
6. **Dull** ache
7. Pain fluctuates in intensity
8. Consistent patterns of referral
9. Alleviation with extinction of trigger point

Treatment of MPD

- **Aim**:

1. Decrease muscle activity
2. Increase ROM

- Accomplished by:

✓ Pharmacotherapy

✓ Occlusal splint therapy

✓ Physical therapy

Clinical characteristics of ID

1. Localized pain in the preauricular area to palpation
2. Localized pain on opening and closing
3. Deviation of mandible to affected side on opening
4. Minimal lateral excursive movement opposite of the affected side
5. Appreciation of joint noises on opening or closing
6. Decreased mouth opening (hypomobility of the mandible)

- **Common finding**: joint capsule tenderness, decreased ROM, and joint noises

Wilkes classification of ID of the TMJ according the severity of disease (used by TMJ surgeons)

Stage		Clinical	Radiographic
I.	Early	Painless clicking, no limitation on opening	Mild displacement of disk with reduction; normal disk morphology
II.	Early/ intermediate	Occasional painful clicking, intermittent locking	Anterior disk displacement with reduction
III.	Intermediate	Joint tenderness, frequent prolonged locking, restricted motion	Anterior disk displacement with or without reduction; no degenerative changes
IV.	Intermediate/ late	Chronic pain, no clicking, restricted motion	Anterior disk displacement without reduction; degenerative changes; adhesions
V.	Late	Variable pain, painful/reduced function, crepitus	Anterior disk displacement without reduction; advanced degenerative changes; advanced adhesions; gross disk deformity and/or perforation

ID

- MRI (disk displacement, disc perforation, or other soft tissue abnormalities)
- CT: degenerative process, neoplasms or bony ankylosis of the joint

➢ **Treatment aim**: decreasing inflammation, increasing ROM

❖ Nonsurgical intervention: pharmacotherapy, occlusal splint therapy, physical therapy

❖ Surgical

Chronic headache disorders causing facial pain

Migraine:

Criteria for diagnosis according to ICHD (International Classification of Headache Disorder):

- **At least 5 attacks** fulfilling the 1 to 3 criteria:

1. Attacks lasting **4 to 72 hours** whether untreated or unsuccessfully treated.
2. The headache has **at least 2 of** the following **4** characteristics:

a. unilateral

b. pulsating in nature

c. moderate to severe in intensity

d. aggravation by or causing avoidance of routine physical activity

3. Headache is associated with **at least one of the following**:

a. nausea and/or vomiting

b. photophobia and phonophobia

Migraine

- The phases of migraine:
- **Prodrome**: vague affective symptoms as long as **24 hours** before the onset of the attack.
- **Aura**: may last up to 1 hour.
- **Resolution**: after the headache resolves and usually characterized by deep sleep.
- **Migraine hangover**: malaise, fatigue, and head pain after sudden movement or coughing.
- **Pathophysiology**: hyperexcitability, hypersensitivity to stimuli, recurrent activation and sensitization of trigeminovascular pathway, reduced activation of descending inhibitory pathway, structural/functional changes in pain pathway.

Treatment of the migraine

1. **Life style modification**
2. **Acute attack treatment**:

- Paracetamol, aspirin, ibuprofen, naproxen, triptans, sTMS, nVNS

3. **Preventive treatment**:

- **First line**: beta blockers, TCA
- **Second line**: anticonvulsants, onabotolinum toxin A, flunarizine, supplements (Riboflavin, magnesium), occipital nerve blocks and stimulation, DBS

Tension type headache (TTH)

✓ **Featureless**

- Persist for minutes to weeks
- Bilateral, tightening in nature (Bandlike)
- May be associated with pohotophobia and phonophobia
- Nausea and vomiting: **usually absent**
- Relation with stress and muscular tension: **?**
- Trigger factor (stress, lack of sleep, not eating, alcohol, menstruation): **may report by patients**

❑ **Pathophysiology**: matter of debate

- Episodic form: pericranial myofascial mechanism
- Chronic form: sensitization of central pathways due to prolonged nociceptive stimulation

Treatment of TTH

- **Acute attack:**

NSAIDs (mainstays treatment) +/- acetaminophen, caffeine, codeine, sedative and tranquilizers.

- **Preventive an attack:** Amitriptyline

Relaxation therapy, biofeedback

- **Chronic TTH:**

Amitriptyline

Some studies (Modest Efficacy): citalopram, sertraline, mianserin, fluvoxamine, venlafaxine, sulpiride, mirtazapine.

Muscle relaxant: Tizanidine

Botulinum toxin A: is not recommended (conflicting data)

physical therapy, psychological therapy, nerve block

Cluster Headache (CH)

Diagnostic criteria:

At least 5 attacks fulfilling criteria 1 to 4.

1. severe unilateral orbital, supraorbital, and/or temporal pain **lasting 15 to 180 minutes** if not treated.
2. headache accompanied by at least one of the following:

A. ipsilateral conjunctival injection and/or lacrimation.

B. ipsilateral nasal congestion and/or rhinorrhea

C. ipsilteral eyelid edema

D. ipsilateral forehead and facial sweating

E. ipsilateral miosis and/or ptosis

3. a sense of **restlessness or agitation**
4. attacks have a frequency of 1 every other day to 8 per week.

Cluster Headache (CH)

- **M** > F
- **Pathophysiology:** unknown (maybe: Hypothalamus)

➢ **Acute attack treatment:**

1. Inhaling oxygen: 8-12 L/min
2. Serotonin receptor agonists
3. Triptans

➢ **Preventive therapy (in frequent attacks):**

1. CCB: Verapamil
2. Lithium
3. Corticosteroid (temporary)
4. Oral ergotamines (note to side effects)
5. Na channel blocker: topiramate

➢ **Interventions**:

➢ occipital nerve block, SPG block, neuromodulation, rhizotomy

Exertional headache

Diagnostic criteria:

1. Headache is associated with physical activities
2. Bilateral, throbbing, and may develop migrainous features
3. Can last 5 minutes to 24 hours
4. Avoiding physical exercises prevents the headache
5. No other disorders to explain those symptoms

- Uncommon, self-limited, short-lasting, is precipitated by exertion
- Mechanism of pain: most believe → vascular (cerebral arterial pressure ↑)

➢ **Treatment**: not well studied

- Indomethacin is the most frequently suggested.

Hypnic headache

Diagnostic criteria:

1. Develops only during sleep
2. Occurs > 10 times/month for > 3 months
3. **Lasts** 15 minutes and up to 4 hours after waking
4. No cranial autonomic symptoms or restlessness, not accounted for by any condition

- **Pathophysiology**: unclear (probably: posterior hypothalamus and central sensitization of trigeminal processing)

❑ **Treatment:**

➢ **Acute attack**: caffeine-containing analgesic (most effective)

➢ **Preventive (chronic hypnic headache)**:

- Lithium, caffeine, indomethacin, topiramate

Medication Overuse Headache (MOH)

Diagnostic criteria:

1. Headache on **≥ 15 days/month**
2. Preexisting headache disorder
3. **Overuse** of acute and/or symptomatic headache drugs for **> 3 months**
4. No other condition can explain those symptoms

- **Pathophysiology**: is not fully understood.
- Several theory: polymorphism (ACE, COMT, serotonin transporter, brain-derived neurotrophic factor), neurotransmitter system interaction, neuronal hyperexcitability, drug dependence.

➢ **Treatment:**

- Education to the patient
- Reducing the dose of medications used for treating primary headache
- Detoxification (sometimes)
- Preventive therapy later at lower doses

Sinus headaches

- Although commonly used, it is **inaccurate term.**

- It refers to headache or facial pain associated sinus disease.

- **Rhinogenic headache** is a **more accurate term**.

The HIS diagnostic criteria for headache attributed to rhinosinusitis:

Frontal headache associated with **pain in one or more regions of the face** and fulfilling **criteria 2 and 3.**

1. Presence of clinical, nasal endoscopic, CT and/or MRI, and/or laboratory **evidence** of acute or acute on top of chronic **rhinosinusitis**.
2. Headache and facial pain that **develops** simultaneously **with onset of rhinosinusitis**.
3. Headache and/or facial pain that **resolves within 7 days after remission or treatment** of acute rhinosinusitis.

Treatment of sinus headache

- Treatment of sinusitis
- Migraine-directed therapy
- Nasal surgery for mucosal contact point.

SUNCT/SUNA

- SUNA = SUNCT - conjunctival injection and tearing

Diagnostic criteria:

1. Unilateral, mostly described as ocular-related pain but can involve larger area of the head
2. Typically does not change sides or cross midline
3. Moderate to severe
4. Usually stabbing or pulsating in nature
5. Pain **lasts from 5 to 240 seconds**
6. The **frequency** of attack: **3 to 200 daily**
7. Ipsilateral conjunctival injection and tearing

➢ **3 pattern of attacks:**

- Classical single attack, groups of a number attacks, sawtooth pattern

SUNCT/SUNA

- Nasal stiffness, rhinorrhea: common
- Sweating: rare

➢ **Pathophysiology**: unclear (studies suggested hypothalamus)

➢ **Treatment**:

- Refractory to most treatment modalities except for the antiepileptic drug groups.
- **First line**: **Lamotrigine**
- Intravenous lidocaine
- Phenytoin

❖ **Note**: one of the main between SUNA and SUNCT: the attack duration can be extended to 10 min in SUNA .

Paroxysmal hemicrania

- Episodic (paroxysmal), continuous
- Unilateral (rarely bilateral)
- Severe orbital or periorbital pain
- May extended to a larger area of the head (temporal, periauricular, maxillary, rarely occipital region)
- Can refer to the shoulder, neck and arm: **common**
- **Lasts 2 to 30 minutes**
- Sharp in nature
- Accompanied by at least **one of these ipsilateral autonomic feature**: conjunctival injection/lacrimation, nasal congestion, eyelid edema, facial sweating, miosis/ptosis
- **Frequency of attacks: > 5/day**
- **Treatment**: Indometacin (The response to indomethacin is absolute)
- **Other alternative**: CCB, Naproxen, Carbamazepin

Head injury headache

- Develops within **7 days after** a head trauma
- There is no specific pattern

➢ **Pathophysiology**:

- peripheral or central sensitization

➢ **Treatment:**

- Multidisciplinary approach
- According to the trauma

Contact point headache

- = anterior ethmoidal neuralgia = Sluder's neuralgia = SPG neuralgia = pterygopalatine neuralgia

➢ **Presentation**: a persistent stabbing or sharp pain in a **single localized area/spot** on the face

➢ **Pathophysiology**: nerve compression related to a structural abnormality inside the nose, such as a septal spur or a deviated septum

- The nerve affected: **usually anterior ethmoid nerve** (a branch of SPG or pterygopalatine ganglion)

Contact point headache

- **Usually starts after an URI** (upper respiratory infection)
- Localized pain on one spot and one side of the face
- can be localized to the roof of the mouth and upper teeth.
- Pain is commonly localized to an area **between the eye and cheek** but can radiate to other parts of the face.

➢ **Treatment**:

- OTC decongestant (**the only single medication**)
- Surgery (the main treatment to cure and relieve the pressure on the nerve)

THANKS FOR YOUR ATTENTION