Headaches, classification.
Migraine
Tension type headache
Cluster headache
Trigeminal neuralgia
Giant cell arteritis (GCA)

M.Maretta

Headache

- The most common symptom in medical practice (lifetime prevalence >90%)
- Headaches in the emergency department: **1.7-4.5%** / year (US 4%)
- A frequent symptom even in acute neurological diseases
  - TIA 30%
  - Ischemic stroke 14-38%
  - Hemoragic stroke 34-57%
  - SAH 30% as the only symptom
  - CVT 70-90% as presenting symptom

I. Primary headaches

1. Migraine
2. Tension type headache
3. Trigeminal automonic headache
   - 3.1 Cluster headache
   - 3.2 Paroxysmal hemicrania
   - 3.3 Short-lasting unilateral neuralgiform HD attacks
   - 3.4 Hemicrania continua
   - 3.5 Probable trigeminal autonomic cephalalgia
4. Other primary headaches
   - 4.1 Primary cough headache
   - 4.2 Primary exercise headache
   - 4.3 Primary headache associated with sexual activity
   - 4.4 Primary thunderclap headache
   - 4.5 Cold-stimulus headache
   - 4.6 External-pressure headache
   - 4.7 Primary stabbing headache
   - 4.8 Nummular headache
   - 4.9 Hypnic headache
   - 4.10 New daily persistent headache (NDPH)

II. Secondary headaches (10%;↑ > 50 year)

5. Headache attributed to trauma or injury to the head and/or neck
6. Headache attributed to cranial or cervical vascular disorder (ischemic stroke,bleeding,vascular malformation,arteritis,cranial venous disorders)
7. Headache attributed to non-vascular intracranial disorder (↑/ ↓ CSF pressure, non-infectious inflammatory intracranial disease,intracranial neoplasia)
8. Headache attributed to a substance or its withdrawal
9. Headache attributed to infection (intracranial/systematic)
10. Headache attributed to disorder of homoeostasis (hypoxia and/or hypercapnia,dialysis,arterial hypertension,
11. Headache or facial pain attributed to disorder of the cranium, neck, eyes,ears, nose, sinuses, teeth, mouth or other facial or cervical structure
12. Headache attributed to psychiatric disorder

Secondary headaches "red flags“ - SNNOOP10

1	Systemic symptoms including fever	Headache attributed to infection or non-vascular intracranial disorders, carcinoid, or pheochromocytoma	Headache with fever is primarily alarming when accompanied by relevant symptoms (e.g., neck stiffness, decreased consciousness, and neurologic deficit)
2	Neoplasm in history	Neoplasms of the brain; metastasis	A newly developed headache in a patient with neoplasm is highly suspect for an intracranial metastasis
3	Neurological deficit or dysfunction (including decreased consciousness)	Headaches attributed to vascular, non-vascular intracranial disorders Brain abscess and other infections	Headache occurs in one-fourth of episodes of acute stroke. The severity of headache is not related to the size of the lesion
4	Onset of headache is sudden or abrupt (thunderclap headache)	Subarachnoid hemorrhage and other headaches attributed to cranial or cervical vascular disorders	Thunderclap headache can be the only initial symptom of subarachnoid hemorrhage
5	Older age (after 50 years)	Giant cell arteritis and other headache attributed to cranial or cervical vascular disorders Neoplasms and other non-vascular intracranial disorders	Older individuals with headache have a higher frequency of secondary etiology. The incidence of primary headache disorders is also lower in this age group
6	Pattern change or recent onset of headache	Neoplasms, headaches attributed to vascular, non-vascular intracranial disorders	A recent change of pattern or a newly developed headache can be the only signs of a secondary etiology. Diagnosis is often delayed in these cases
7	Positional headache	Intracranial hypertension or hypotension	Positional headache is the trademark of intracranial hypotension, and the most common cause is cerebrospinal fluid leak at the spinal level
8	Precipitated by sneezing, coughing or exercise	Posterior fossa lesions, Chiari malformation	Cough headache is highly predictive of Chiari malformations and posterior fossa lesions
9	Papilledema	Neoplasms and other non-vascular intracranial disorders; intracranial hypertension	A high prevalence of patients with papilledema has a serious underlying pathology
10	Progressive headache and atypical presentations	Neoplasms and other non-vascular intracranial disorders	Progressive headache and atypical headache presentation can be the only signs of serious underlying pathology
11	Pregnancy or puerperium	Headaches attributed to cranial or cervical vascular disorders; post-dural puncture headache; hypertension related disorders (e.g., preeclampsia); cerebral venous thrombosis; hypothyroidism; anemia; diabetes	Headache during pregnancy and puerperium has a higher risk of severe pathology. More than one-third of individuals presenting to acute care with headache during pregnancy will have a secondary etiology. Most common causes are hypertensive disorders followed by pituitary adenoma or stroke Other risk factors are no prior headache history, occurring during third trimester, seizures, hypertension, and fever
12	Painful eye with autonomic features	Pathology in posterior fossa, pituitary region or cavernous sinus Tolosa-Hunt syndrome Ophthalmic causes	Patients with presentations of painful eye with autonomic features should undergo neuroimaging as it can be due to a structural lesion. Even typical presentations of cluster headache (or other trigeminal autonomic cephalalgias) can derive from a structural lesion
13	Posttraumatic onset of headache	Acute and chronic posttraumatic headache; subdural hematoma and other headache attributed to vascular disorders	Headache related to trauma should always be explored
14	Pathology of the immune system such as AIDS (acquired immunodeficiency syndrome)) or medical immunosuppression	Opportunistic infections	Risk of severe pathology is dependent on the degree of immunosuppression
15	Painkiller overuse or new drug at onset of headache	Medication overuse headache; drug incompatibility	Medication overuse is the most common cause of secondary headache. Onset of headache due to a new drug can be a sign of incompatibility with the given drug

"Green flags" for secondary headaches

- The current BH was also present during childhood
- The patient indicates a pause between episodes of HD
- Most primary pain is paroxysmal
- Related to the menstrual cycle
- Similar character of HD in the family
- HD started / ended more than 7 days ago

History of headache

Side

unilateral → migraine (60%), trigeminal autonomic HD, trigeminal/occipital neuralgia, glaucoma, nummular BH, temporal arteritis, optic neuritis, trigeminal neuralgia

bilateral → migraine (40%), tension HD, primary stabbing HD, cervicogenic HD,
→ post-traumatic HD (tension BH/migraine)
→ secondary BH (iCMP, hCMP, SAK)

Location where it originates / where it spreads

Character

pressure, constriction → tension HD
throbbing → migraine
stabbing → primary stabbing HD
neuralgic → trigeminal, occipital ev. other neuralgia

Intensity

severe → migraine (↑…↑↑…↑↑↑), trigeminal autonomic HD (↑↑↑), neuralgia
moderate strong (VAS 4-5/10) → tension HD

Duration

hours → migraine (4-72 hours) vs. tension HD (30 min. – 7 days) vs. trigeminal autonomic HD (minutes)

Peaking pain? (hours…migraine vs minutes…trigeminal BH)

Was there similar pain in the past? What has changed over the years (character - not frequency)

Symptoms

Nausea / vomiting, photophobia / phonophobia, aura → migraine (trigeminal autonomic HD ↓)
Cranial autonomic symptoms (lacrimation, eye pain), trigeminal autonomic HD (migraine +/-)
glaucoma (+ eye pain, mydriasis, visual disturbances)

Disability: calm (migraine) vs. restlessness (cluster BH ine TAC)

Menstrual related HD → migraine

Does HD arise during stading up ? → intracranial hypotension syndrome

HD + transient visual disturbances (clouding, etc.) → idiopathic intracranial hypertension (**obesity** + **female**)

Wakes up with a HD → migraine, hypnic BH (older patients)

Headaches lasting several days → both migraine / tension-type BH

Medication

No response / decrease in intensity (↓ VAS) / complete relief of pain (2 hour pain free)
Adverse effects

Migraine classification

1.1 Migraine without aura

1.2 Migraine with aura

- 1.2.1 Migraine with typical aura
  - 1.2.1.1 Typical aura with headache
  - 1.2.1.2 Typical aura without headache
- 1.2.2 Migraine with trunk aura
- 1.2.3 Hemiplegic migraine (familial, sporadic)
- 1.2.4 Retinal migraine

1.3 Chronic migraine

1.4 Complications of migraine

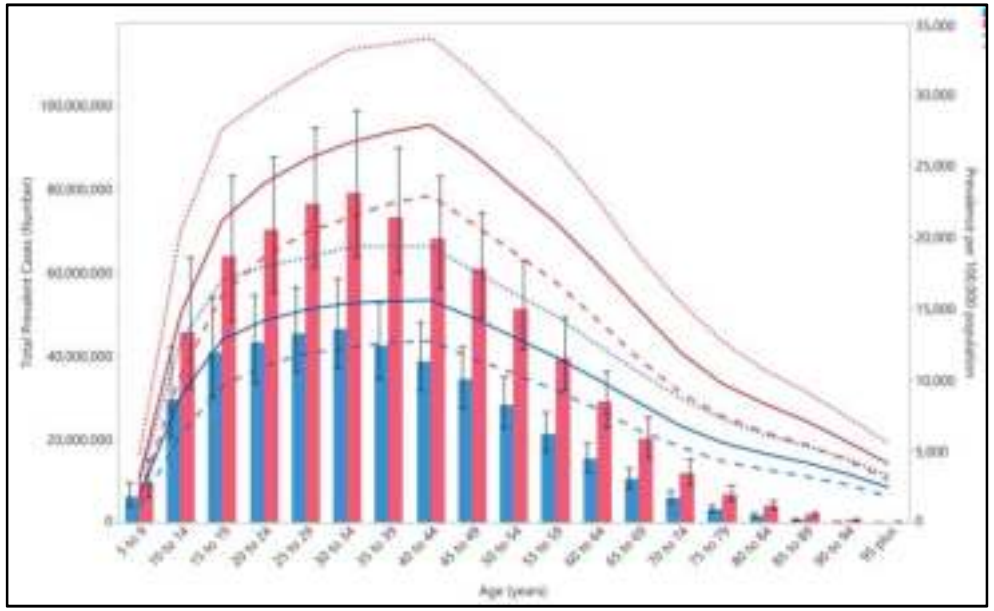
- 1.4.1 Migraine status
- 1.4.2 Persistent aura without infarction
- 1.4.3 Migrainous infarction
- 1.4.4 Migraine aura triggered seizure

1.5 Probable migraine

1.6 Episodic syndromes associated with migraine

Migraine

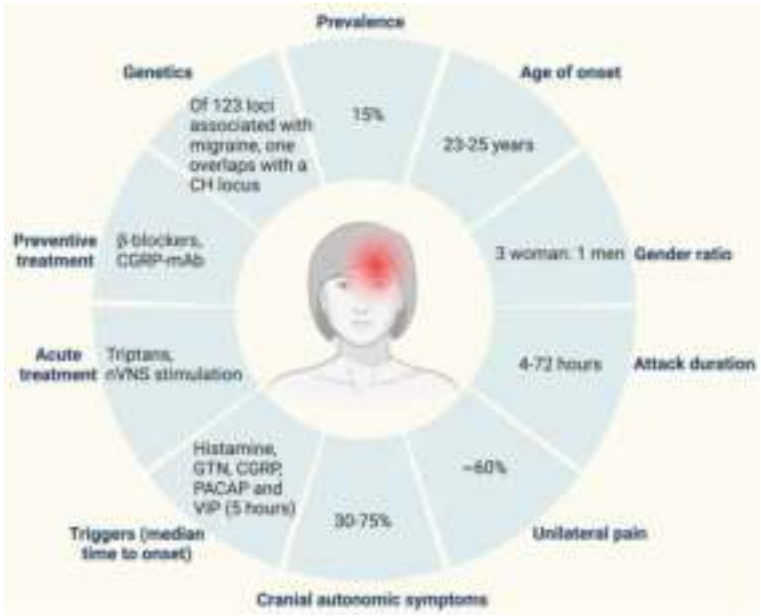
- The 3rd most common diagnosis
- The 6th cause of disability
- Prevalence in the population **12%**
  - 3x more common in women (18% vs. 6%)
- 50% first attack < 20 years of age
- 75% migraine before the age of 35
- 2% chronic migraine (>15 days/month)
- 20% menstrual migraine
- 30-50% vertigo during a migraine
- 75% cervical spine pain



Migraine prevalence (/100 000) [4]

Diagnostic criteria

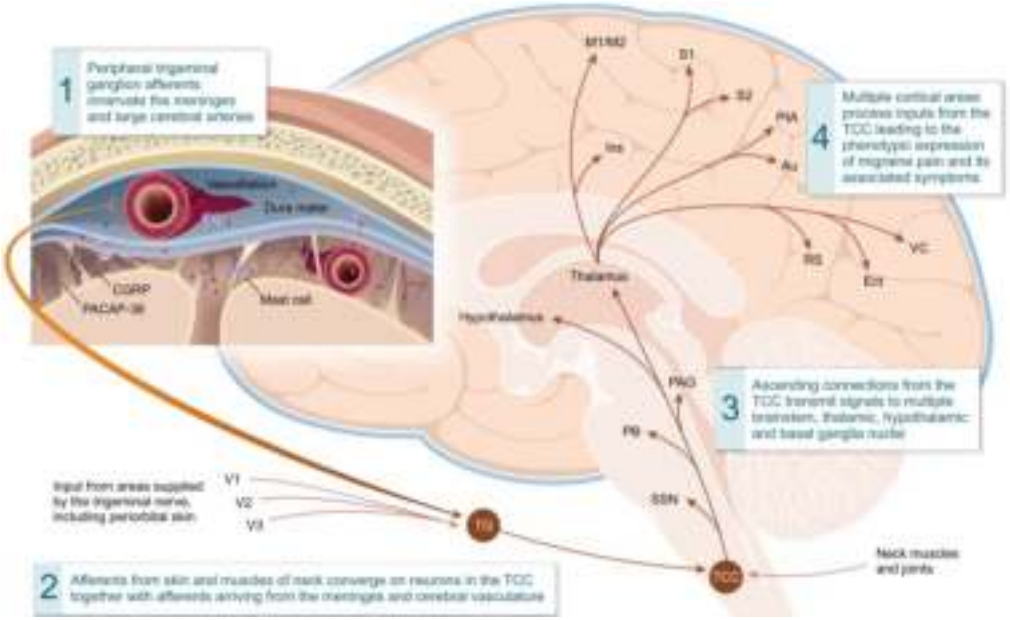
A. At least **5** attacks[1] fulfilling criteria B-D
B. Headache attacks lasting **4-72 hr** (**untreated/unsuccessfully treated**)[2;3]
C. Headache has at **least 2** of the following four characteristics:
   1. **unilateral** location
   2. **pulsating** quality
   3. **moderate** / **severe** pain intensity
   4. aggravation by or causing avoidance of routine physical activity
D. During headache at least one of the following:
   1. nausea and/or vomiting
   2. photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.



Mechanism of migraine

- The trigeminovascular pathway transmits signals from the periphery (meninges) to the cortex
- Nociceptive fibers (V1-n.V) lead signals from the meninges to gl.trigeminale → trigeminocervical complex (ncl.caudalis n.V + dorsal horns C1 and C2) → thamalus → cortex

- Stimulated nociceptive neurons (invert the dura mater on the periphery) secrete vasoactive peptides (CGRP, PACAP) → activation of the trigeminovascular pathway



The trigeminovascular system contains

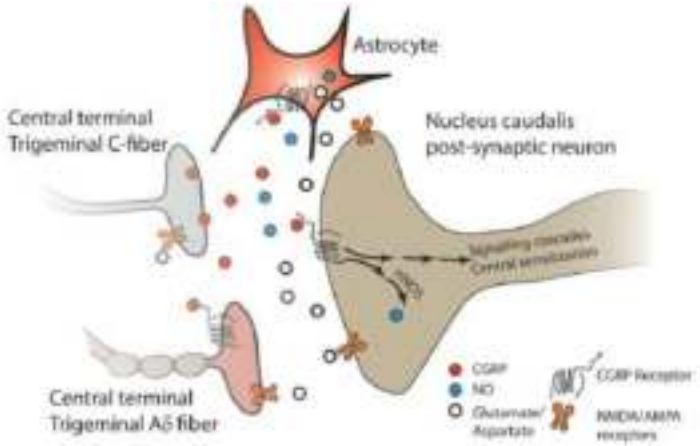
Thin C fibers: contain CGRP <u>vesicles</u>

Thick A-δ fibers: contain CGRP <u>receptors</u>

Stimulation of C fibers → release of CGRP → binding to CGRP receptor (A-δ)

↓ (cAMP)

Vasodilatation → migraine headache



Why calcitonin gene related peptide ?

- CGRP is widely expressed (cortex, hippocampus, thalamus)
- Most (35-50%) is located in **trigeminal ganglion**
- CGRP binds with high affinity to receptors (CGRP, AMY1 and others) → signal transmission (cAMP, PKA, G-protein)

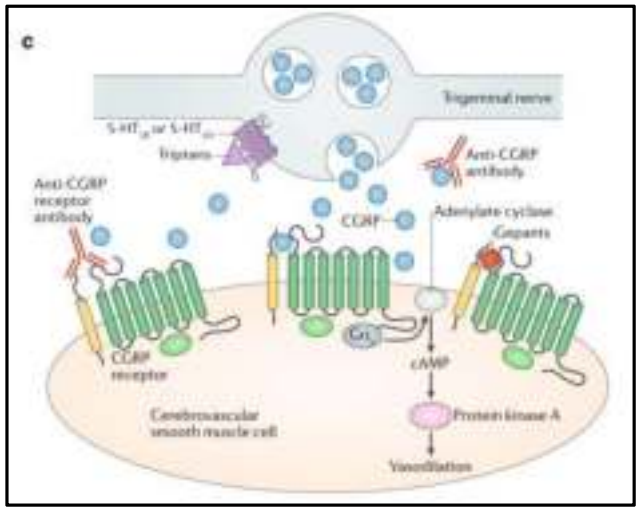
↓

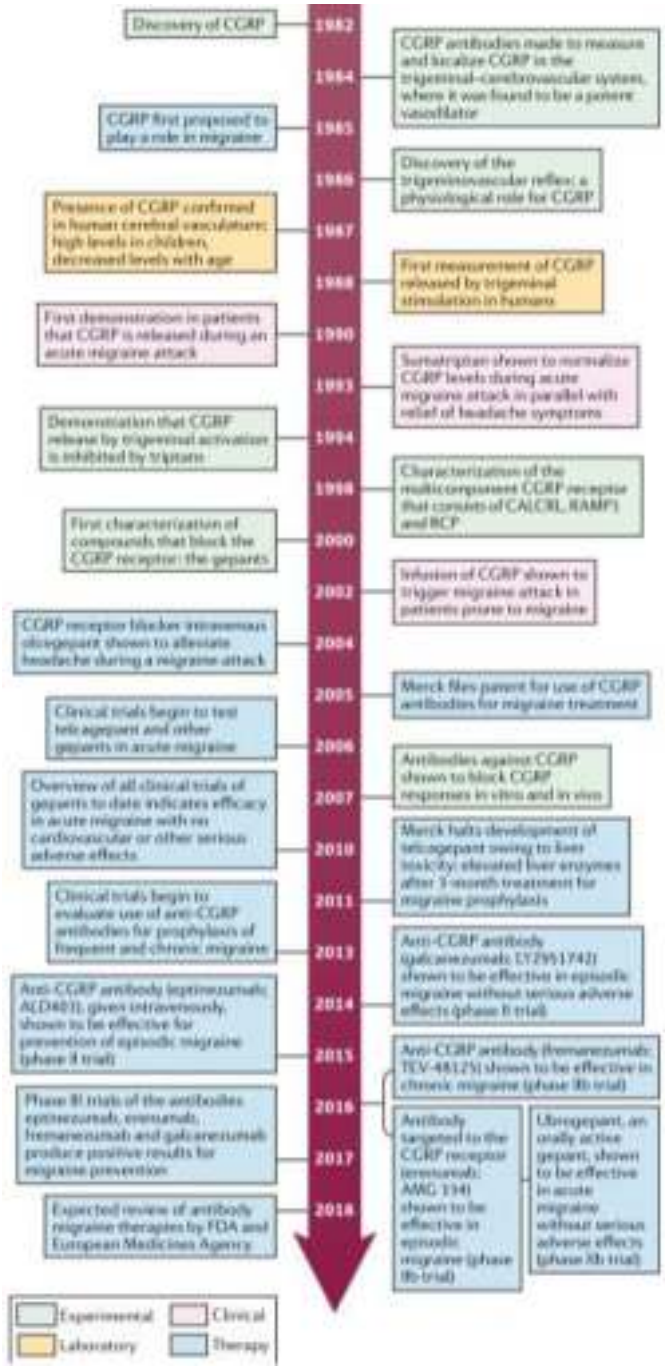
vasodilatation, neurogenic inflammation, peripheral sensitization

Vasoactive peptide release in the extracerebral circulation of humans during migraine headache

	VIP	CGRP	SP	NPY
Site				
External jugular	< 6	86 ± 4[b]	6 ± 2	130 ± 15
Cubital fossa	< 6	43 ± 6	4 ± 1	133 ± 10
Control values	< 20	< 40	< 4	< 130

- Elevated CGRP values during a migraine attack (cerebral circulation only, VJI)
- **Triptans** (including DHE) led to a **↓CGRP levels**

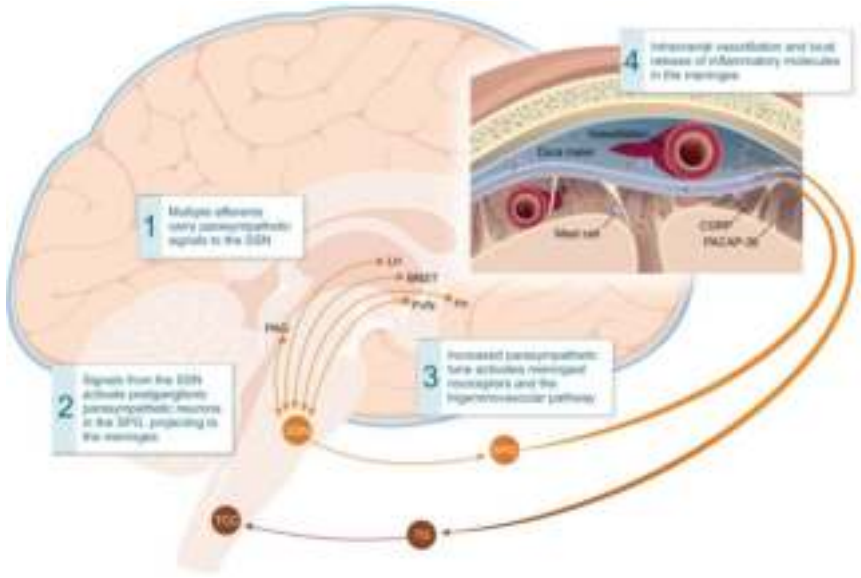




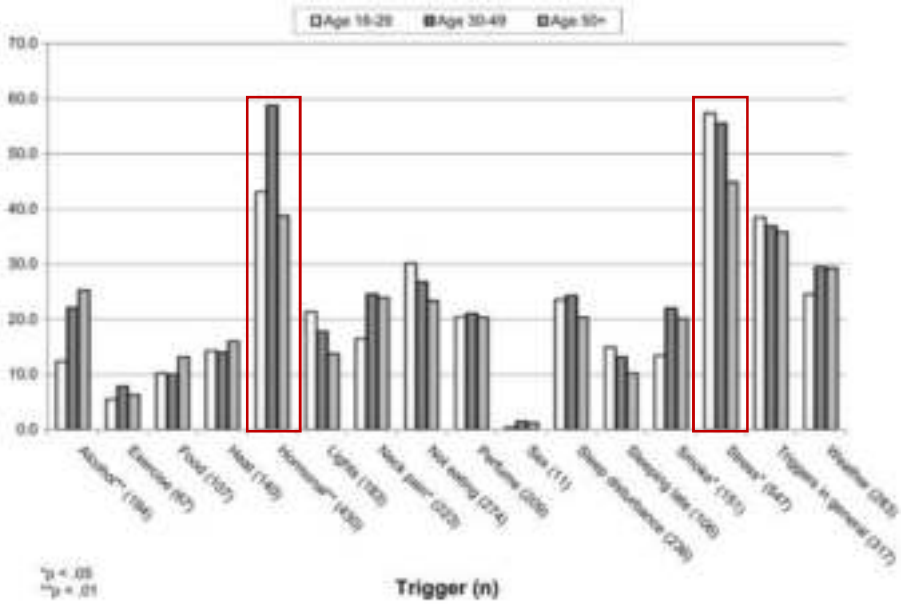
[1]Martelletti et al. 2019; [2] Edvinsson L, 2018; [3]De Vires et al., 2020

But what causes the activation of meningeal nociceptors?

- Many symptoms of migraine (nausea, vomiting, cranial autonomic symptoms) arise as a result of changes in the ASN (sympathetic vs. parasympathetic)
- Migraine triggers (stress, awakening, changes in physiological functions) activate nociceptive pathways through **↑ parasympathetic activation**

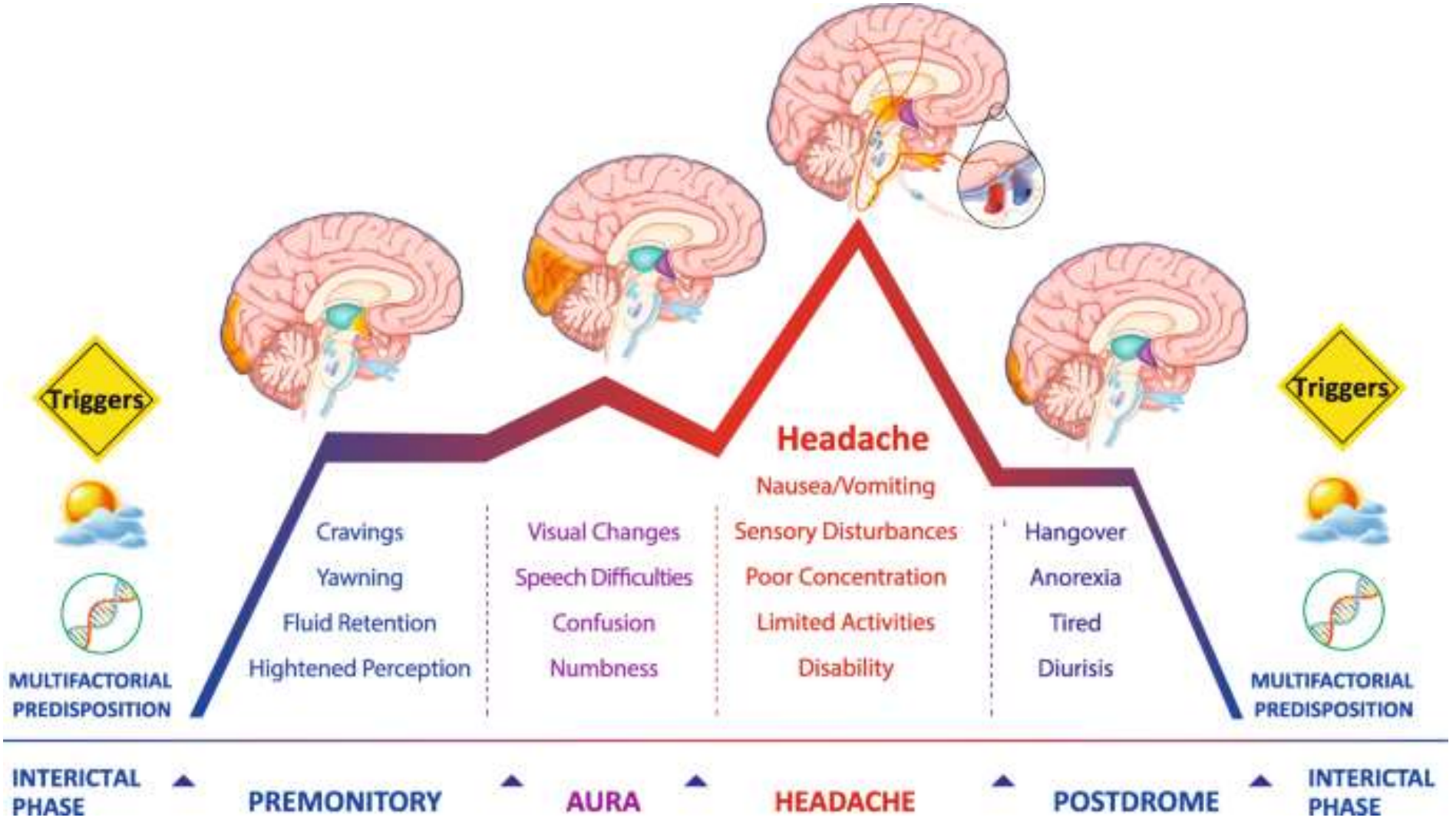


Migraine triggers Kelman L, Headache 46.7 (2006): 1161-1171.



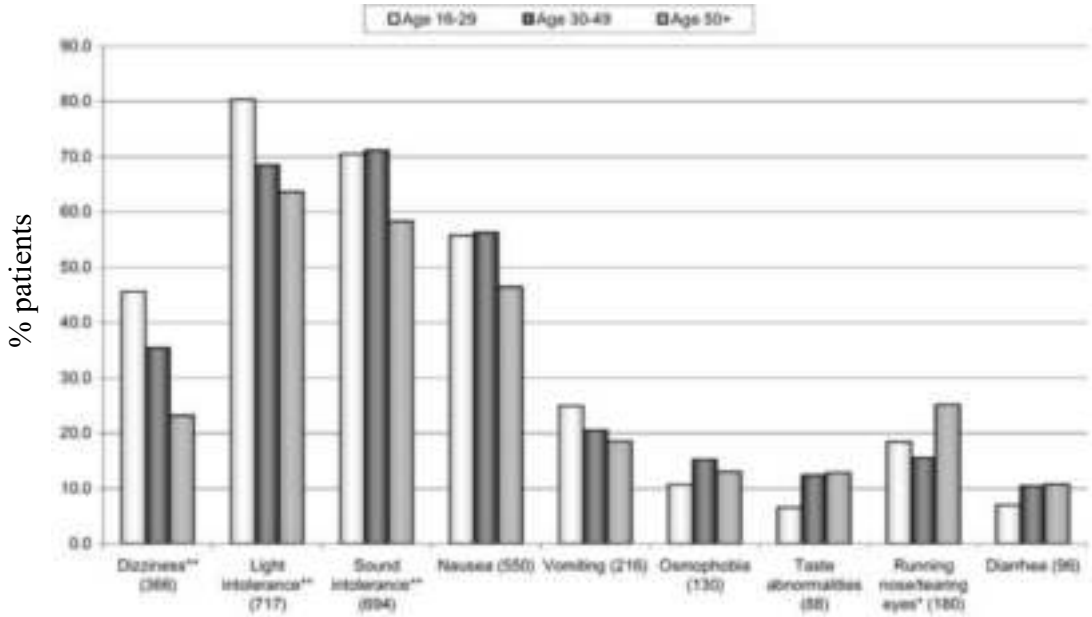
Burstein R, Jakubowski MJ Comp Neurol. 2005;493:9-14

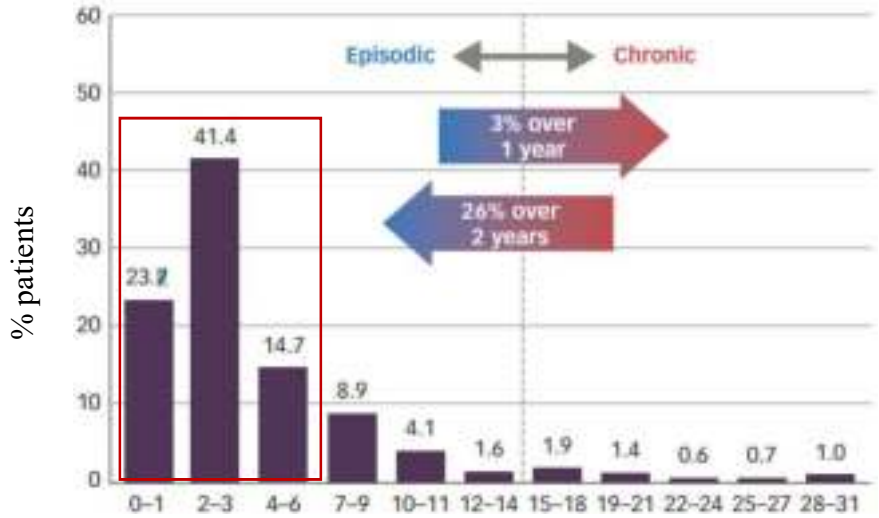
- Prodrome (30 – 80 %)
  - sensitivity to light, sound and odors
  - lethargy, yawning, tiredness
  - mood changes (eg, depression, anger, euphoria)
  - excessive thirst, anorexia
- Aura (30 %)
  - Visual, sensory, speech, motor
- Headache
- Postprodrome (80 %)



Migraine symptoms Kelman L, Headache 46.7 (2006): 1161-1171.

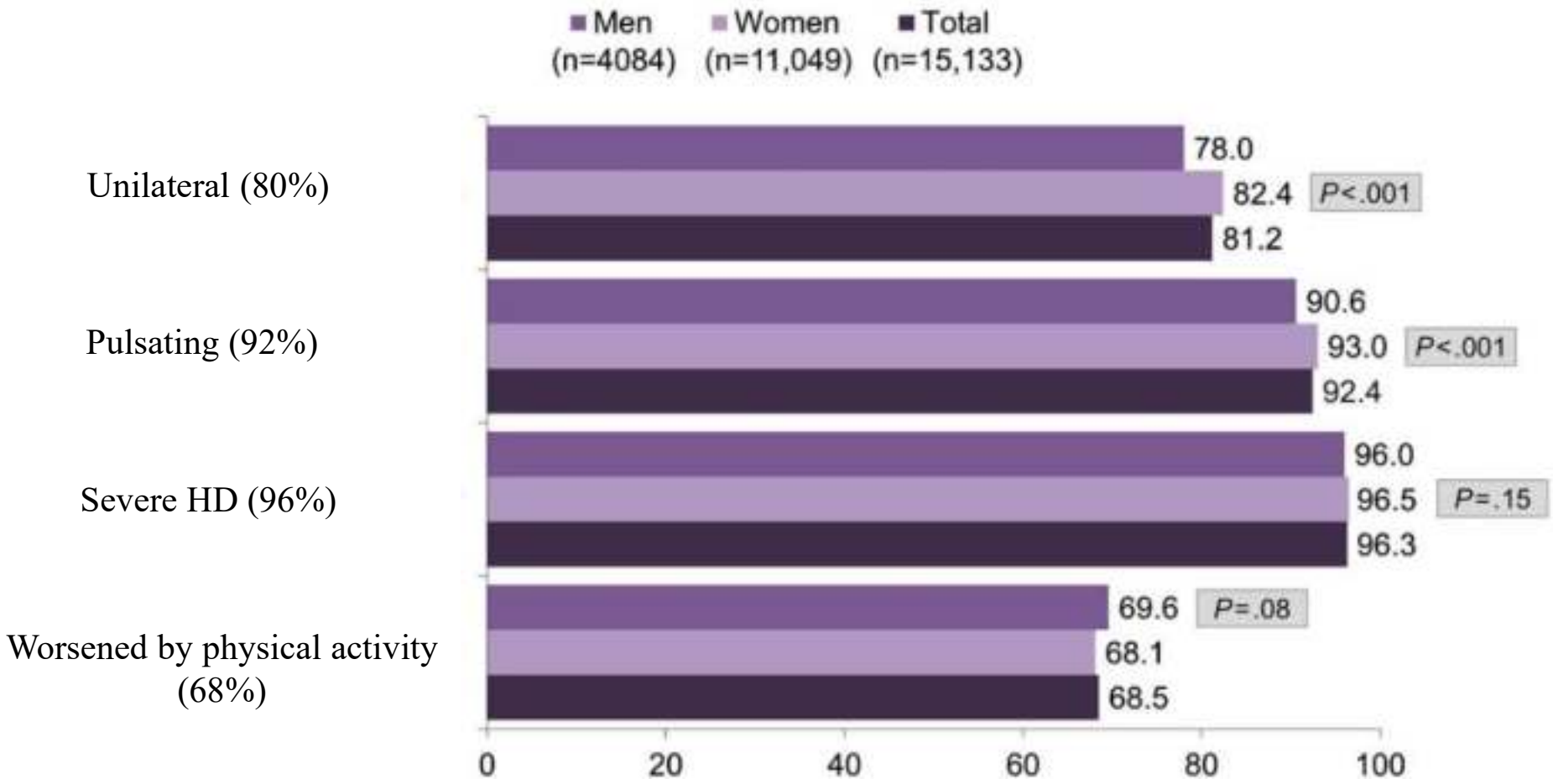
50%	Nausea
20%	Vomiting
60-80%	Fotofobia
60-70%	Fonofobia
10-15%	Osmofobia
20-45%	Vertigo



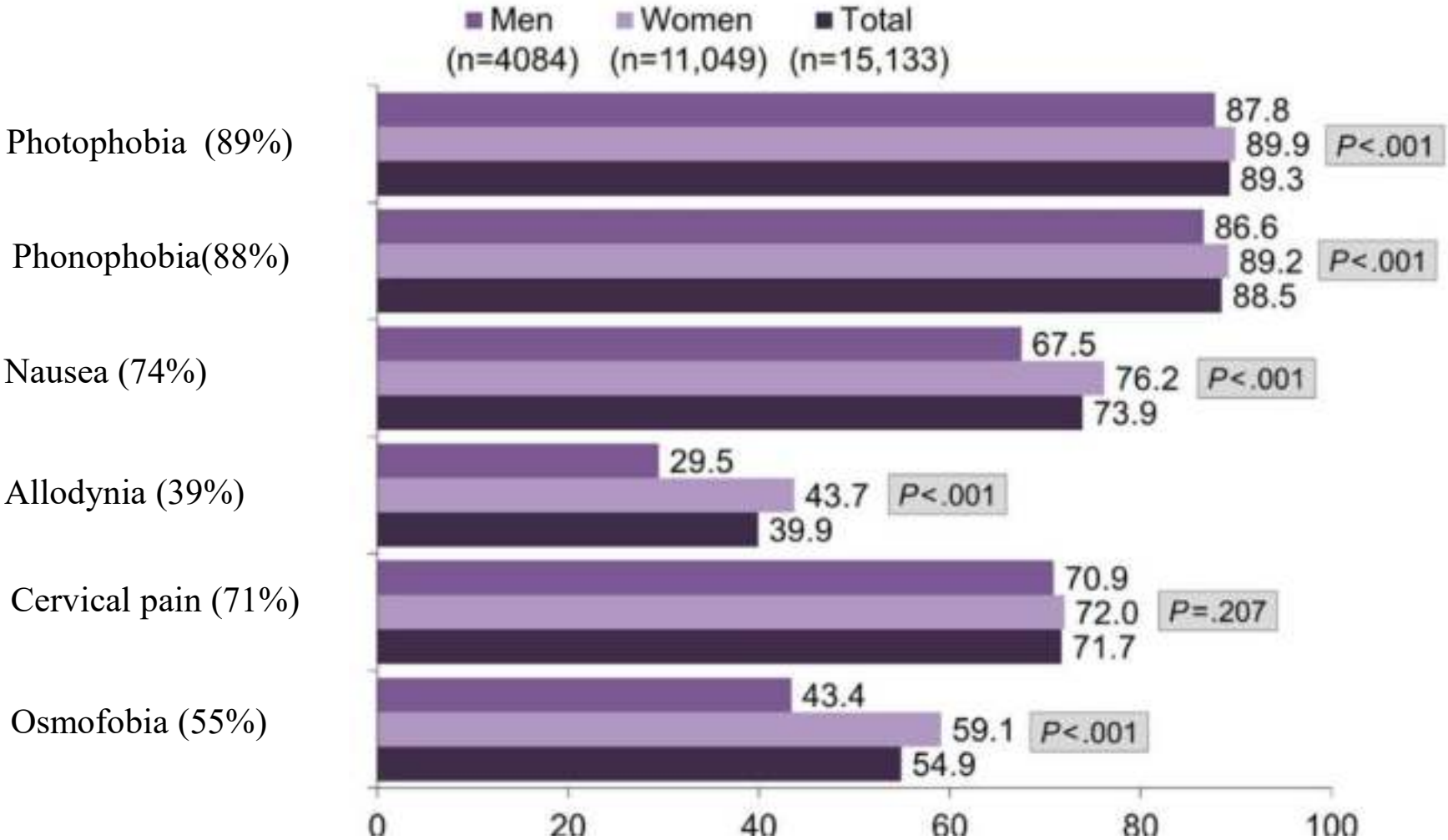


Days with migraine (/month)

Bigal M, Krymchantowski AV, Lipton RB, 2008;16
Blumenfeld AM et al., 2011;8 Manack A et al., 2011.11



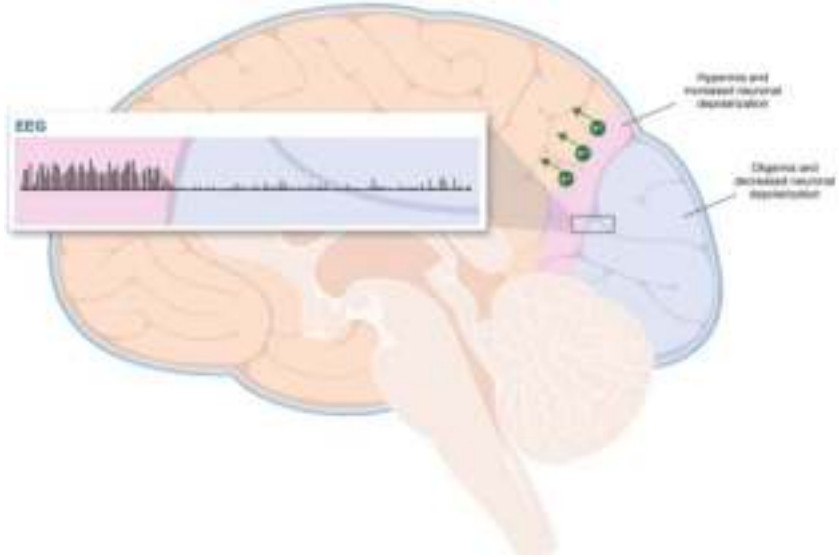
Lipton R.B.,et al., 2018;58(9):1408-1426



Lipton R.B.,et al., 2018;58(9):1408-1426

Migraine with aura (20%)

- Neurological symptoms that precede the onset of migraine
- The essence of the aura is a cortical spreading depression (CSD, Leao)
- Depolarization wave (↑ flow 1-2 min; 2-6mm; **hyperemia**) → suppression of cortical activity (↓ flow by 20-30%; **oligemia**)



Diagnostic criteria:

A. ≥ 2 attacks fulfilling criteria B and C
B. One or more of the following fully reversible aura symptoms:
   1. visual
   2. sensory
   3. speech and/or language
   4. motor
   5. brainstem
   6. retinal
C. **At least 3** of the following six characteristics:
   1. ≥ 1 aura symptom **spreads gradually over ≥5 minutes**
   2. ≥ 2 aura symptoms **occur in succession**
   3. each individual aura symptom lasts **5-60 minutes**[1]
   4. ≥ 1 aura symptom is **unilateral**[2]
   5. ≥ 1 aura symptom is **positive**[3]
   6. the aura is accompanied / followed by headache (< 60 minutes)
D. Not better accounted for by another ICHD-3 diagnosis

Burstein R, Jakubowski MJ Comp Neurol. 2005;493:9-14

Visual aura

- Most common type of aura (95%)

Positive symptoms

- phosphenes, flashing light, fortification signs

Negative symptoms

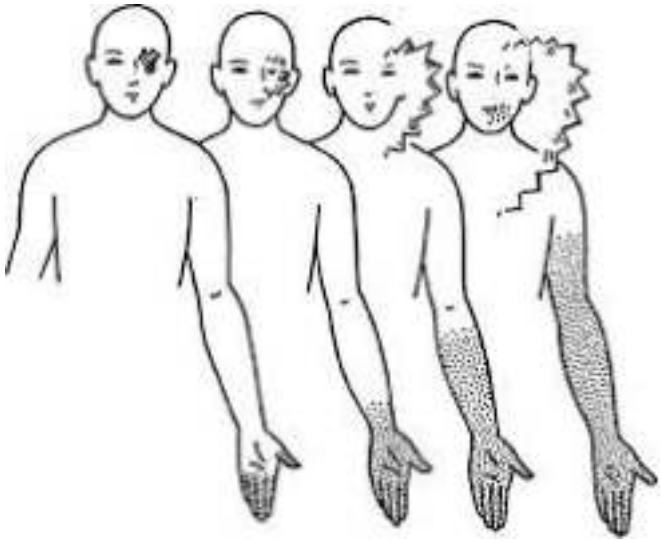
- scotoma, loss of visual field (hemianopsia, quadratopsia)

Elementary Visual Symptoms of aura	Frequency (range %)
1. Flashes of bright light / unformed flashes of light / star-shaped figures	16–38
2. 'Foggy/blurred vision or "dimness"	25–54
3. Zigzag or jagged lines	24–81
4. Scotoma	23–77
5. Blind spots (scotomata)	32
6. Black dots	3–17
7. Phosphenes (small bright dots)	19–70
8. Flickering light	12–91
9. 'Like looking through heat waves or water'	8–24
10. Visual snow	7
11. White Spots	7–22
12. 'Bean-like' forms like a crescent or C-shaped	7
13. Hemianopsia	6–24
14. Deformed images (alteration of line/ angles) / Metamorphopsia	2–6
15. 'Tunnel' vision	4–27
16. Curved or circular lines	4–18
17. Round forms	12
18. Colored dots / spots of light	3–19
19. Oscillopsia /autokinesis (movement of stationary objects)	2–4
20. Like a mosaic	13
21. Fractured Vision	1
22. Corona phenomena	2–18
23. Anopia	1–2
24. Things look farther away than they really are	1–13
25. Things look closer than they really are	1–3
26. Macropsia (things look larger than they really are)	1–3
27. Micropsia (things look smaller than they really are)	2–4
28. "Like a negative of film"	1
29. "Slanted vision"	1
30. Complex hallucinations	1–3

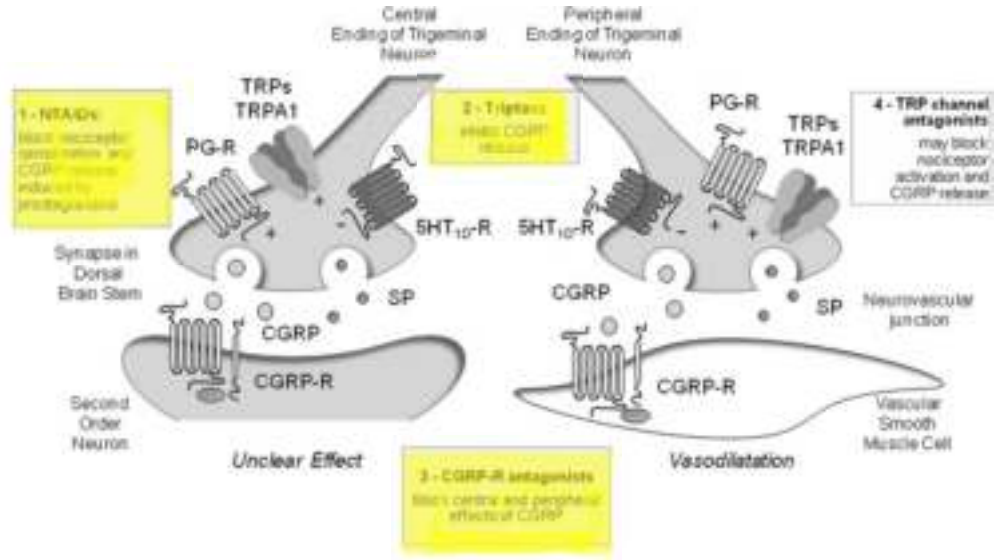
Sensory symptoms (35%)
- Positive symptoms (paresthesia) followed by negative symptoms
- 65% paresthesia
- 20% paresthesia → hypoesthesia/anesthesia

Motor symptoms (18%)
- Feelings of heavy limbs
- Hemiparesis in hemiplegic migraine (sporadic/familial)
- Dx ischemic stroke

Speech aura (17-20%)
- Expression disorders (difficulty finding words, articulation)



I.Acute treatment of migraine



1st line
NSAID + antiemetics

→ 3 consecutive migraine attacks without successful treatment contraindicated →

2st line
Triptans
(+ NSAID)

→ 3 consecutive migraine attacks without successful treatment contraindicated →

Triptan change
↓
Failure of all triptans →

3st line
Gepants
Ditans

NSAID

ASA	1000 mg
Ibuprofen	**400-600 mg**
Diclofenak	50 mg p.o.
Paracetamol	1000 mg p.o.

Domperidon	10 mg p.o
Metoklopramid	10 mg p.o.

Triptans Agonist of **5-HT $1_{B}/_{D}$** receptor
(↓ CGRP)

Sumatriptan 50 mg p.o
6 mg subcutaneous inj,
10 mg nasal

Eletriptan	40 mg p.o
Rizatriptan	10 mg p.o

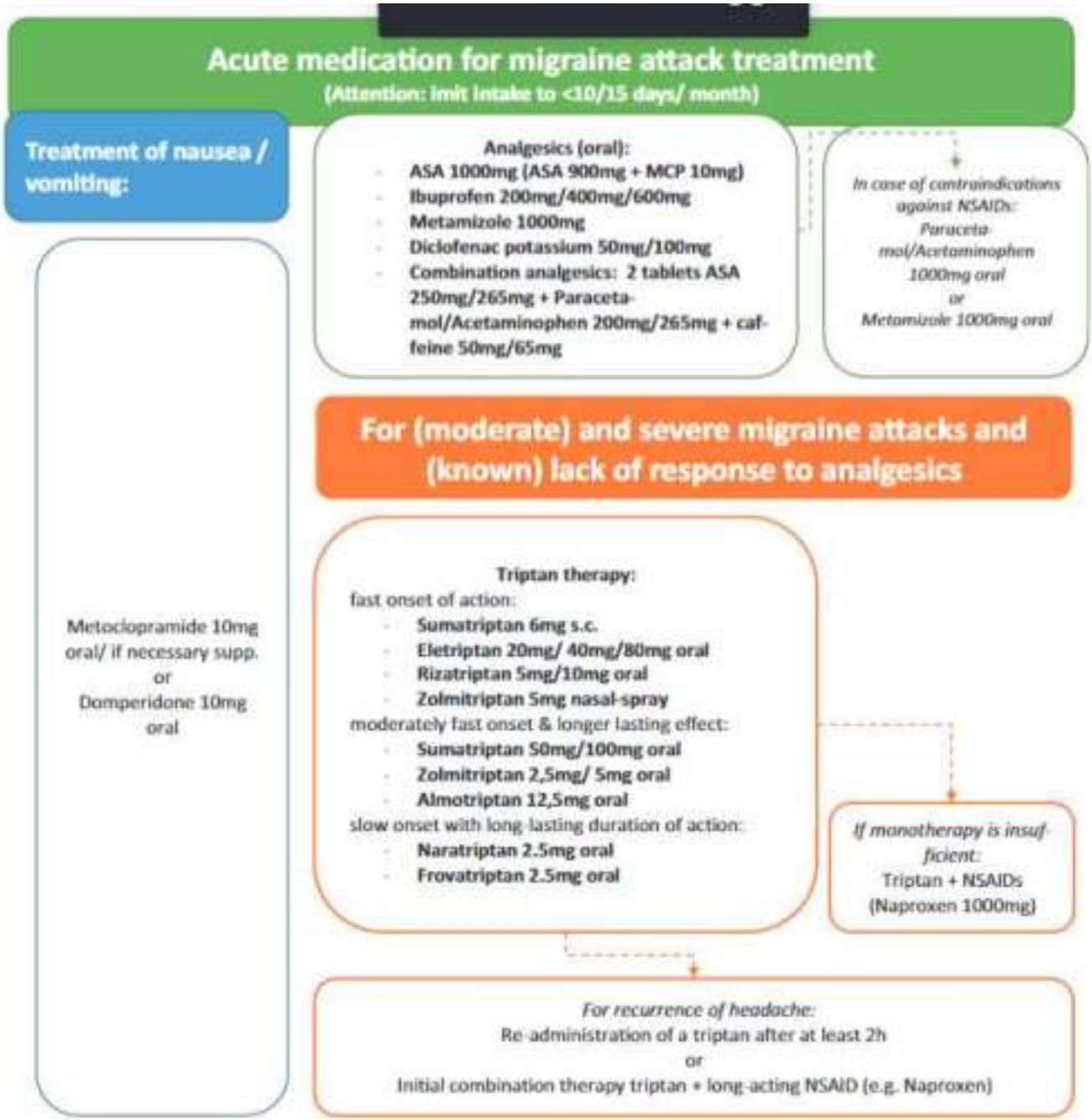
<u>Gepants</u>
Rimegepant 75 mg p.o

Ditans
Agonist **5-HT1_{F}** receptor (↓ CGRP)
Lasmiditan 50,100,200 mg

Drug	T_{max} (Hours)	Elimination Half-Life (Hours)	Hour Headache Relief	NNT: 2 Hour Pain Free	Dose (mg)†	Dosage Interval (If Repeated) and Maximum Daily Dose†
Acetaminophen	0.5-1	2	5.0	12	1000	Every 4 hours, max. 4000 mg
Acetylsalicylic acid (ASA) (tablet)	1-2	ASA: 0.25 Salicylate (active): 5-6 (after 1 g dose)	4.9	8.1	975-1000	Every 4-6 hours; max: 5.4 g/day (varies depending on indication)
ASA (effervescent)	~20 minutes	as above			975-1000	Every 4 hours; max: 8 (325 mg) tablets
Ibuprofen (tablet)	1-2	2			400	Every 4 h; max: 2400 mg
Ibuprofen (solubilized)	<1	2	3.2	7.2	400	Every 4 hours; max: 2400 mg
Naproxen sodium‡	2	14	6.0	11	500-550 (up to 825 mg)	Twice a day; max: 1375 mg
Diclofenac potassium (tablet)	<1	2	6.2	8.9	50	3-4 times a day; max: 150 mg
Diclofenac potassium (powder for oral solution)	15 min	2	5.1	7.4	50	Single dose recommended for migraine attack

†For acute migraine treatment, only 1 or 2 doses are usually recommended; doses are for adults.
‡Absorbed more quickly than naproxen.
T_{max} = time to maximum plasma concentration; NNT = number needed to treat: the number of patients that must be treated to obtain a response on a given end point over and above the response rate obtained from placebo.

Becker, W. J. (2015). *55*(6), 778-793.



Diener, H.C.,(2019). *Clinical and Translational Neuroscience*, *3*(1), 2514183X18823377.

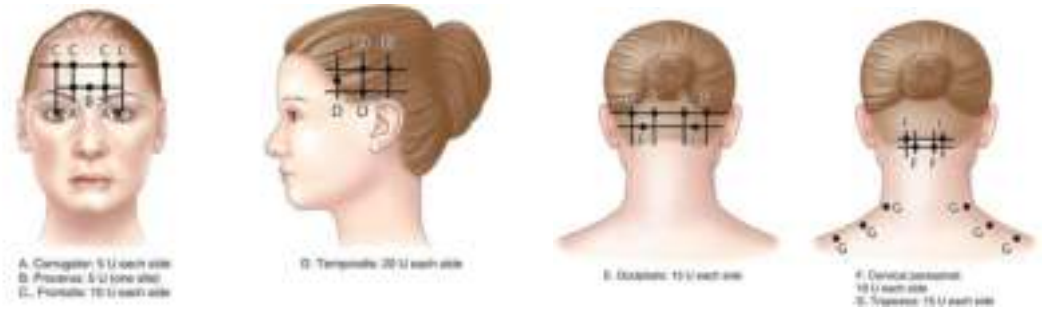
II. Preventive migraine treatment

Indications:

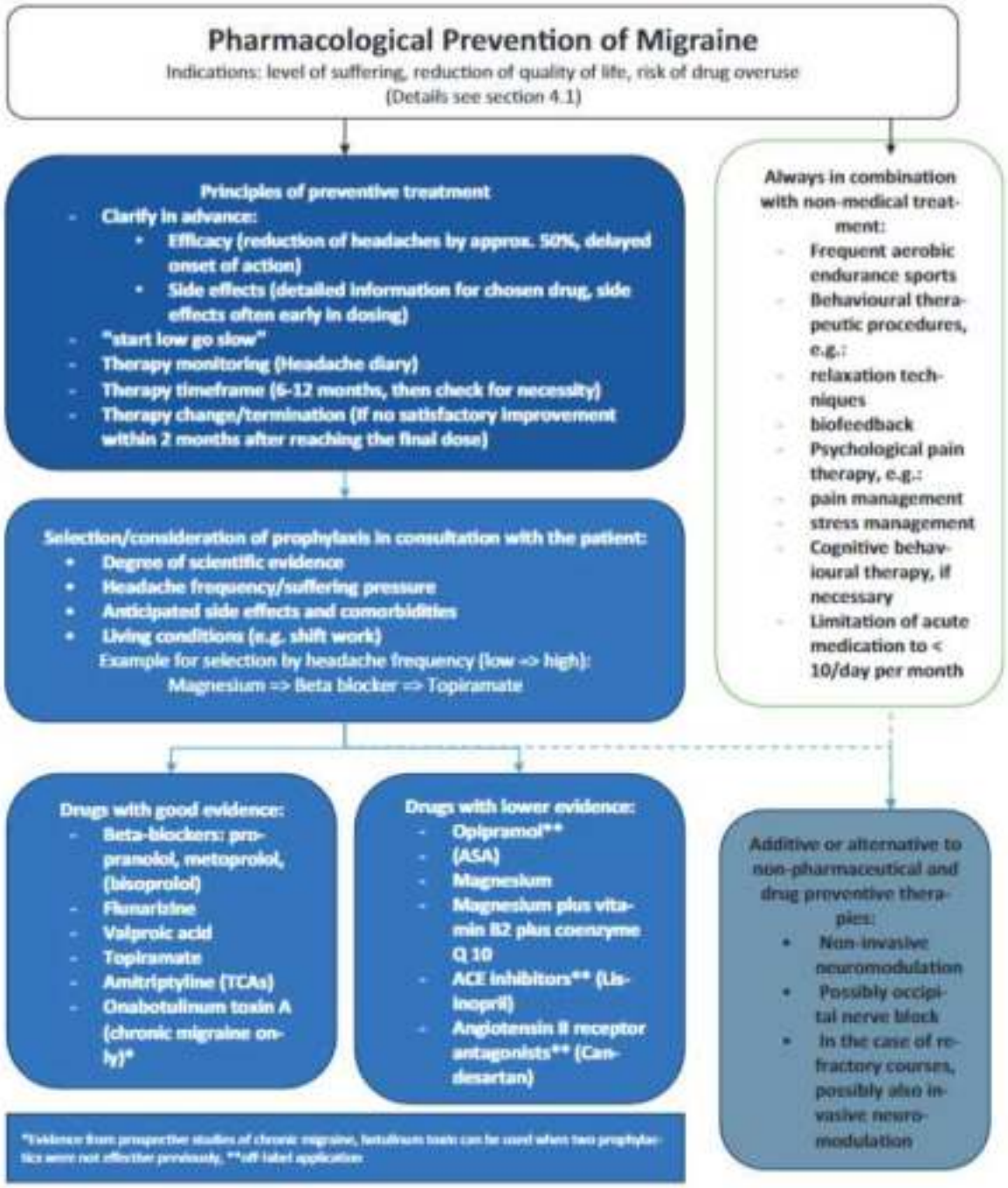
- Migraines lasting > 72 hours
- Ineffectiveness/contraindication of acute treatment
- Migraine negatively affecting the quality of life
- Rare forms of aura (hemiplegic migraine)

	Monthly migraine days				
Disability	**2**	**3**	**4-5**	**6-10**	**11-14**
None	-	-	**to consider**	to suggest	to suggest
Some	-	**to consider**	to suggest	to suggest	to suggest
Severe (bed rest)	**to consider**	to suggest	to suggest	to suggest	to suggest

- Antiepileptics (topiramate, valoproate)
- Beta-blockers (metoprolol)
- Antidepressants (amitriptyline)
- Serotonin antagonists (pizotifen)
- Botulinum toxin (BOTOX; chronic migraine



[1]Schwedgt et al. 2018



Diener, H.C.,(2019). *Clinical and Translational Neuroscience*, *3*(1), 2514183X18823377.

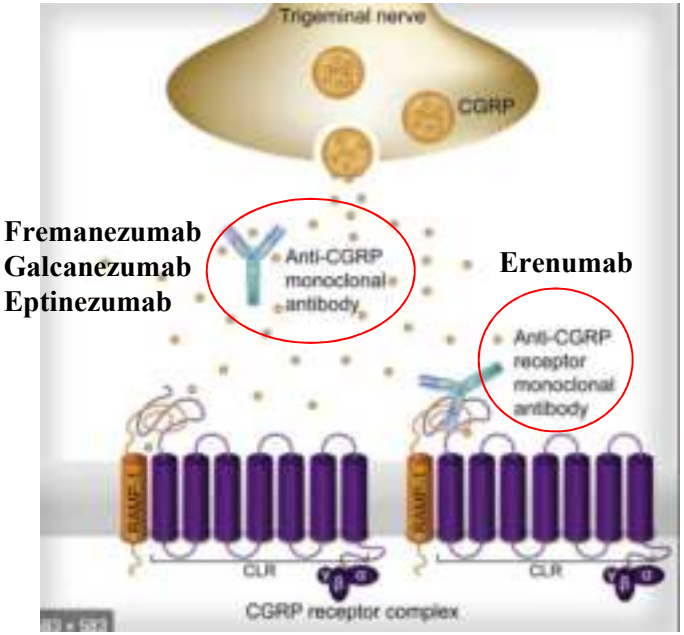
Monoclonal antibodies (mAbs) in the preventive treatment of migraine

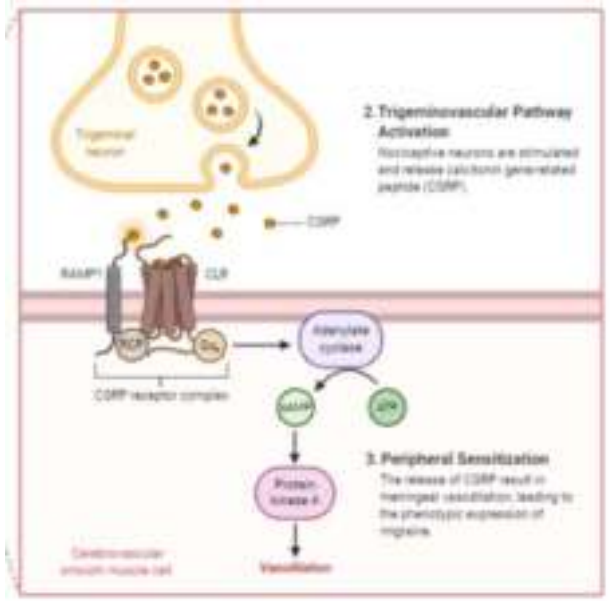
- They bind to the CGRP receptor or to the peptide itself (CGRP)

Receptor antibodies
- Erenumab (sc)

Ligand antibodies
- Fremanezmab (s.c.)
- Galcanezumab (sc)
- Eptinezumab (IV)

Advantages
- Low rate of side effects
- Administration once a month (every 3 months, AJOVY)
- The only specific preventive therapy





Medication overuse headache (MOH)

1-2% of the global population suffers from MOH
1% of the population in Europe (Greece 0.7%)
11-70% of patients with chronic HD have MOH

- the risk of MOH is only in patients with a history of headache
- 101 patients with rheumatological disease and daily use of analgesics, when only 8 patients (7.6%) developed MOH and all had a history of migraine

Each of the criteria A–C needs to be filled for the diagnosis.

A. Headache on ≥15 days/month in a patient with a preexisting headache disorder

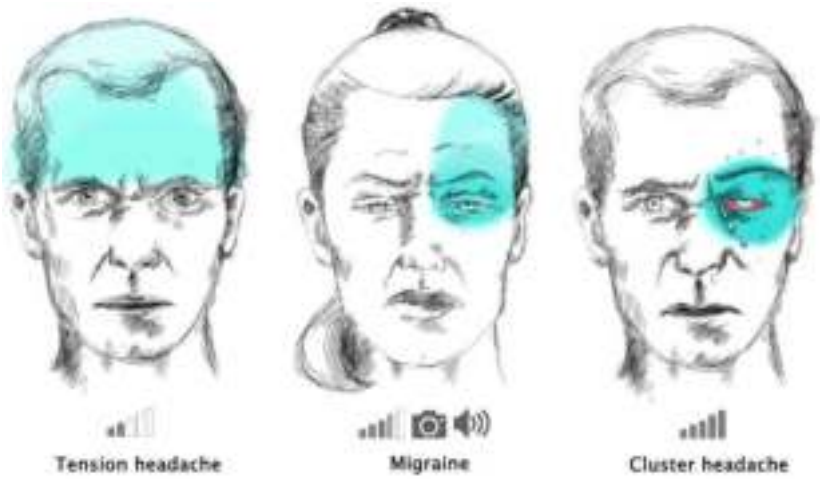
B. Regular overuse of acute and/or symptomatic headache drugs for >3 months of:

1. Ergotamines, opioids, triptans or combination of analgesics on ≥10 days/ month
2. Simple analgesics (paracetamol/acetaminophen, acetylsalicylic acid, and nonsteroidal anti-inflammatory drugs) on ≥15 days/month
3. Any combination of the above-mentioned drugs, or one or more medications other than those mentioned above, taken for acute or symptomatic treatment of headache on ≥10 days/month

C. The headache cannot be better accounted for by another ICHD-3 diagnosis

(Ashina et al.,2023;Constantinidis et al.,2023; Diener et al., 2020; Bahra a kol.,2001)

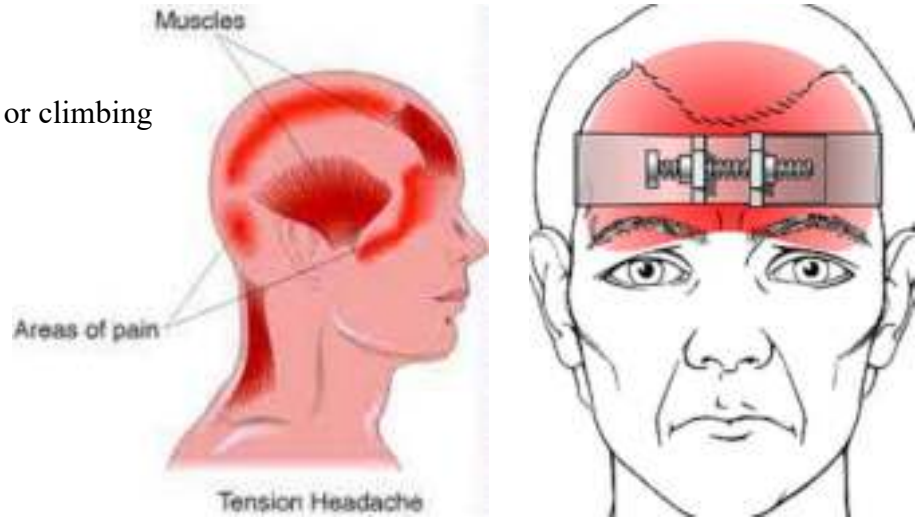
2. Tension type headache



- The most common headache (annual prevalence 40%)
- Most common in the 3rd decade of life
- Episodic or chronic (>15 days/month)
- Often described as a **band around head, weight on the head**, gradually becomes daily (chronic)

Diagnostic criteria:

A. At least **10 episodes** of headache occurring on <1 day/month on average fulfilling criteria B-D
B. Lasting from 30 minutes to 7 days
C. At least two of the following four characteristics:
    1. **bilateral** location
    2. **pressing** / **tightening** (non-pulsating)
    3. **mild** / **moderate** intensity
    4. not aggravated by routine physical activity such as walking or climbing stairs
D. Both of the following:
    1. no nausea or vomiting
    2. no more than one of photophobia or phonophobia
E. Not better accounted for by another ICHD-3 diagnosis[1].



TTH treatment

I. Acute treatment of TTH

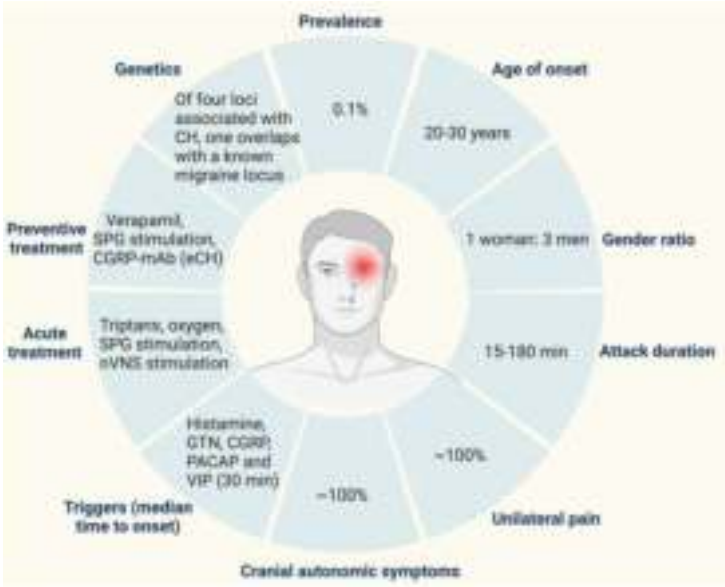
- Analgesics (acetylsalicylic acid, paracetamol, NSAID)

II. Preventive treatment of TTH

- High frequency (>7 days) or chronic (>15 days)
- Non-pharmacological procedures (physiotherapy)
- Pharmacological treatment (**amitriptyline/venlafaxine** - antidepressants)

Cluster headache

- Rare (0.1% in the population)
- M:F=3:1 (average age 30 years)
- Circadian rhytm (between 1-3 a.m.) → awaking
- Average of 1-2 attacks / day (75-88%)
- Cluster period lasts on avr. 100 minutes



A. ≥ 5 attacks fulfilling criteria B-D

B. Severe / very severe unilateral orbital, supraorbital and/or temporal pain lasting **15-180 minutes** (when untreated)[1] **vs.migraine (4-72 hours)**

C. Either or both of the following:
   A. ≥ 1 of the following symptoms or signs, ipsilateral to the HD :
      1. conjunctival injection and/or lacrimation
      2. nasal congestion and/or rhinorrhoea
      3. eyelid oedema
      4. forehead and facial sweating
      5. miosis and/or ptosis
   B. a sense of **restlessness/agitation**

D. Occurring with a frequency between one every other day and 8 / day[2]

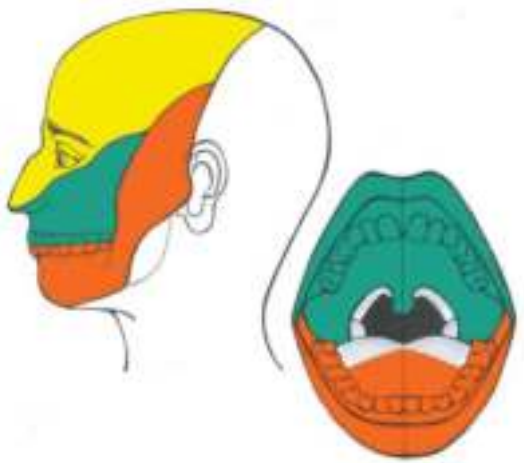
E. Not better accounted for by another ICHD-3 diagnosis.

Treatment

Acute (abortive)
- O2 inhalation (8 L/min ... 10 min.)
- Sumatriptan (subcutaneous/nasal)

Preventive treatment
- start low go slow
- 1st verapamil
- 2nd topiramate
- Other: lithium

Trigeminal neuralgia (TN)



- More frequent in **older** patients (50-60 years; incidence ↑ with age)
- F:M=2:1, bilateral 2-5%
- **Sharp**, **electrifying**, **stabbing** pain (70-95%)
- **Severe** intensity (VAS 9/10)
- Mostly **V2** branch (V3...V1)
- **Triggers** (chewing, shaving, cold air, touch)

Dx criteria:

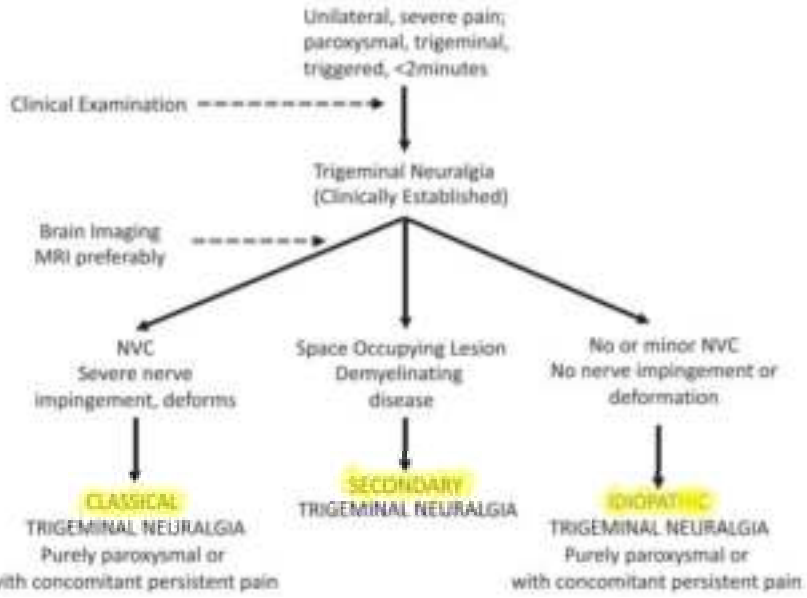
A. Recurrent paroxysms of **unilateral** facial pain in the distribution(s) of **one or more divisions of the trigeminal nerve** (no radiation beyond) and fulfilling criteria B and C

B. Pain has all of the following characteristics:
   1. lasting from a fraction of a **second to 2 minutes**[2]
   2. **severe intensity**[3]
   3. **electric shock-like**, shooting, stabbing or sharp in quality

C. Precipitated by innocuous stimuli within the affected trigeminal distribution[4]

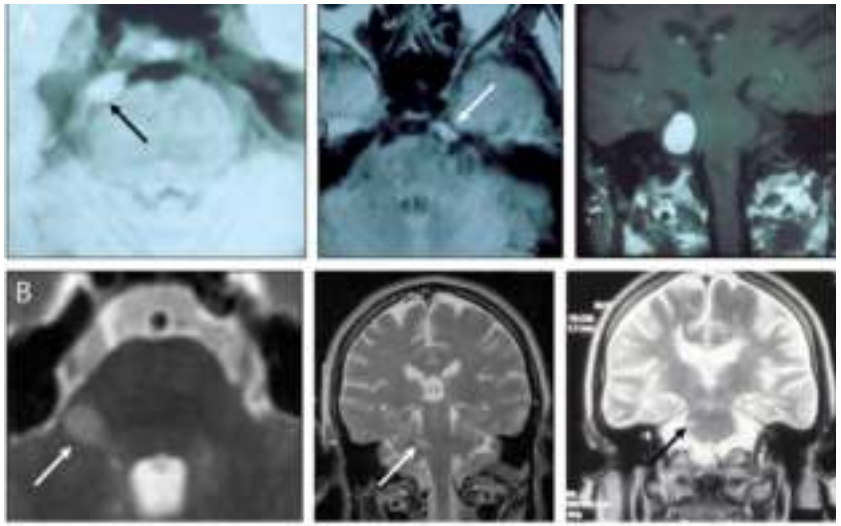
D. Not better accounted for by another ICHD-3 diagnosis.

A.Classical TN
B.Secondary TN
C.Idiopathic TN



Dx of trigeminal neuralgia

1. Brain <u>MRI with contrast</u> (+MR angiography)



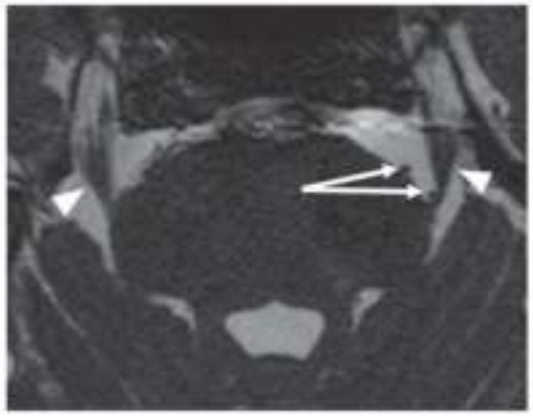
It excludes focal lesions in the area of the brainstem

- Tumor (meningioma)
- Demyelination (multiple sclerosis)

It excludes neurovascular conflict

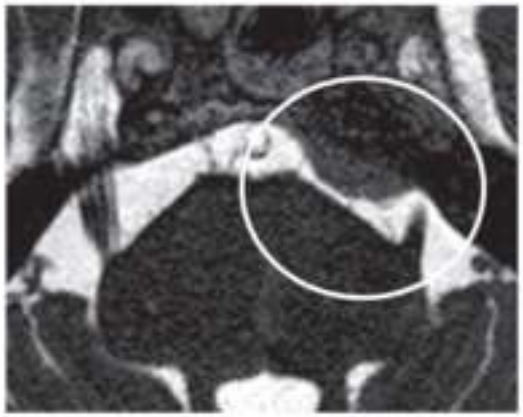
- Contact of n.V with superior cerebellar a. (SCA)/ anterior inferior cerebellar a.(AICA)

Clasical TN



Compression of left trigeminal nerve by arterial loop of AICA (arrow)

Secondary TN



Compression of left trigeminal nerve by meningioma

Dental causes	▶ Dental caries ▶ Pulpitis ▶ Dental sensitivity ▶ Periodontal disorders ▶ Pericoronitis ▶ Cracked tooth ▶ Alveolar osteitis
Sinus causes	▶ Maxillary sinusitis
Salivary gland causes	▶ Salivary stone
Temporomandibular joint causes	▶ Temporomandibular disorders
Neuropathic pain	▶ Glossopharyngeal neuralgia ▶ Nervus intermedius neuralgia ▶ Post-herpetic neuralgia ▶ Post-traumatic trigeminal neuropathy ▶ Painful trigeminal neuropathies ▶ Atypical odontalgia ▶ Burning mouth syndrome
Trigeminal autonomic cephalalgias	▶ SUNCT/SUNA ▶ Paroxysmal hemicrania ▶ Cluster headache ▶ Hemicrania continua
Other	▶ Persistent idiopathic facial pain ▶ Primary stabbing headache

Lambru, G., et al. (2021). Practical neurology, 21(5), 392-402
Maarbjerg, Stine, et al. Cephalalgia 37.7 (2017): 648-657.

Treatment of trigeminal neuralgia

I. Pharmacological (treatment of neuropathic pain)
- 1. **Carbamazepine**
- 2. **Gabapentin, pregabalin**

II. Non-pharmacological
- Microvascular decompression
- In case of proven neurovascular conflict

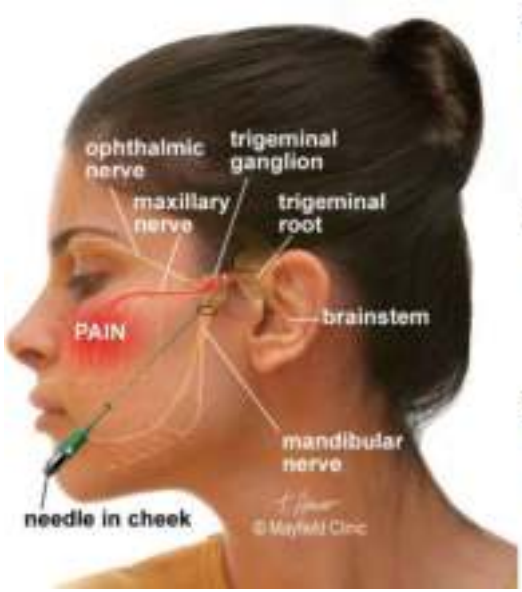
Gamma Knife surgery
- In the absence of NV conflict
- Pharmacoresistant forms
- Superficial, deep (brain stem area)

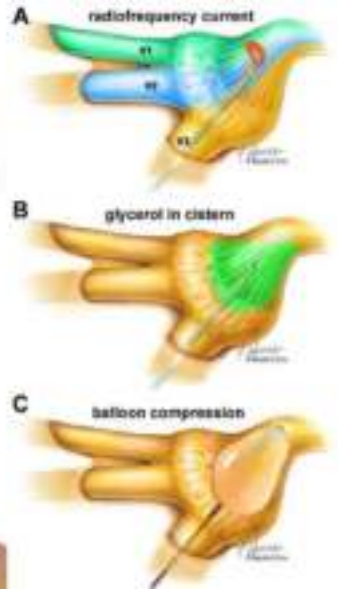
Intervention methods
- Balloon compression
- Radiofrequency thermoablation

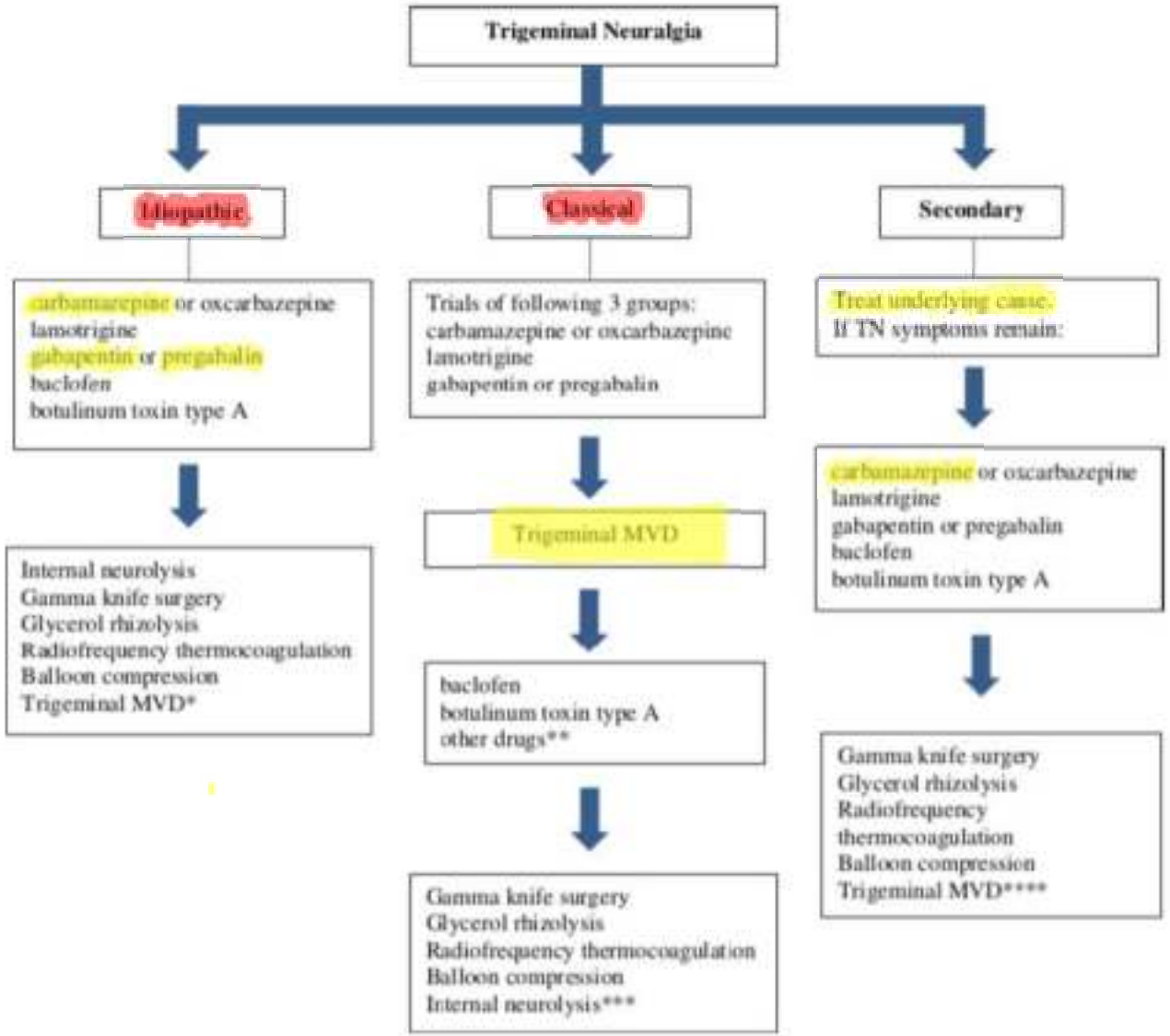
Mikrovaskulárna dekompresia

Drug	Initial dose (mg)	Target dose (mg)*	#Dose increase (titration)	Schedule
Carbamazepine	100–200	1200–1800	100–200 mg/2 days	x2-4/days
Carbamazepine-CR	200–400	1200	Usually transfer from regular format at equivalent dose	x2/days
OXC	300	1200–2400	300–600 mg/week	x2-3/days
Baclofen	5–15	30–60	5 mg/3 days	x3/days
Gabapentin	300	900–2400	300 mg/2–3 days	x3/days
Pregabalin	150	300–600	50 mg/2–3 days	x2-3/days
Lamotrigine	25	400–600	25–50 mg/week	x1-2/days

Abbreviations: CR, controlled release. OXC, oxcarbazepine; X, number of times daily.
*Titrate according to response and side effects.







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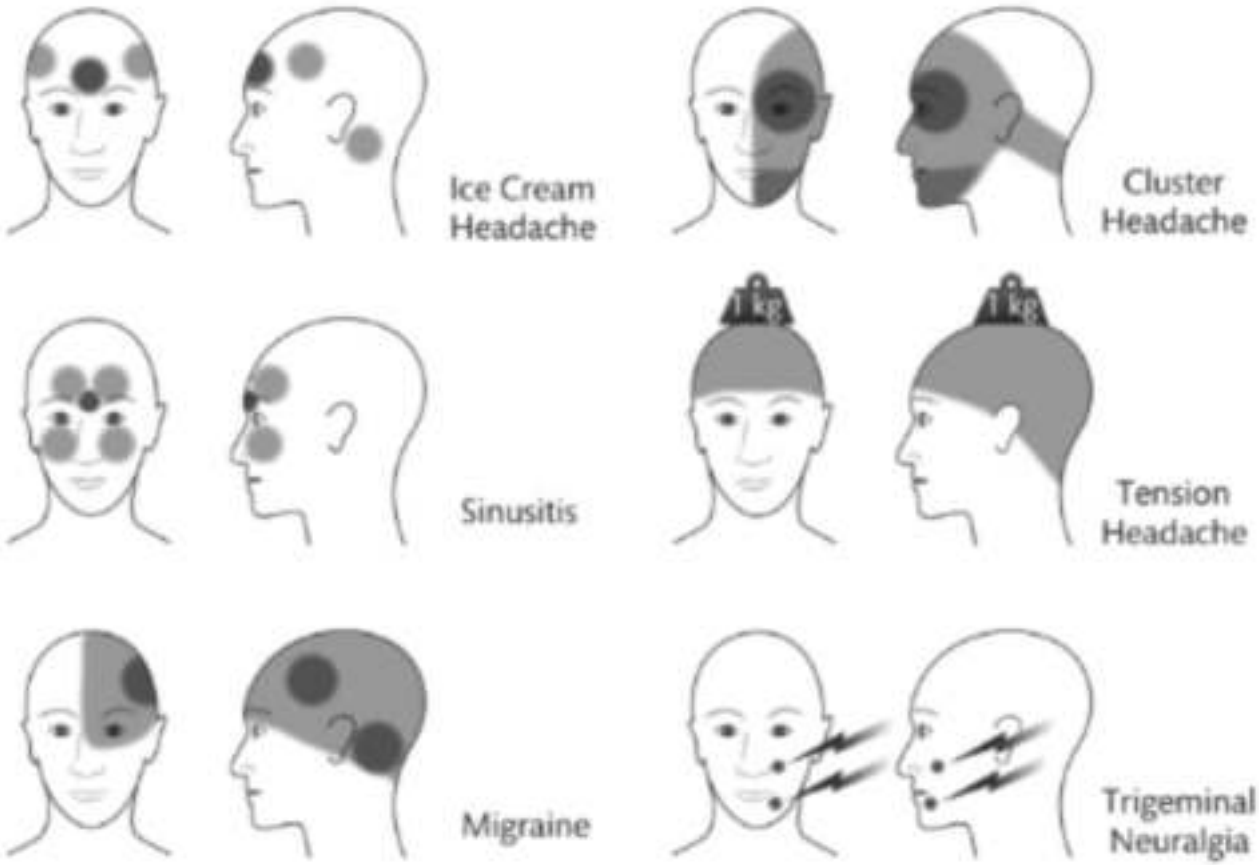
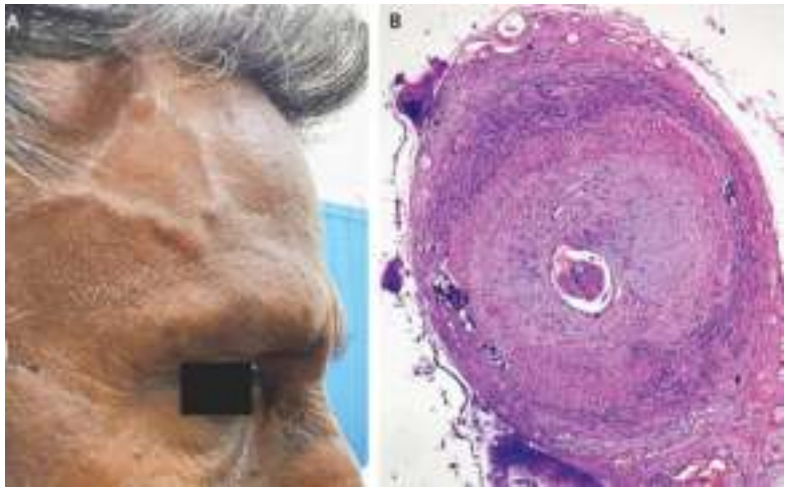

Ice Cream Headache
Cluster Headache
Sinusitis
Tension Headache
Migraine
Trigeminal Neuralgia

Giant (temporal) cell arteritis

- Horton's arteritis, granulomatous arteritis (GCA - giant cell arteritis)
- Systemic inflammatory diseases manifested by ophthalmological as well as neurological symptoms
- Affects medium to small caliber blood vessels (temporal superficial artery → temporal arteritis)
- May affect ophthalmic artery, vertebral artery
- 0.5-27/ 100,000 (> 50 years) → rarely < 50 years
- F:M=3.7:1 (average age 75 years)



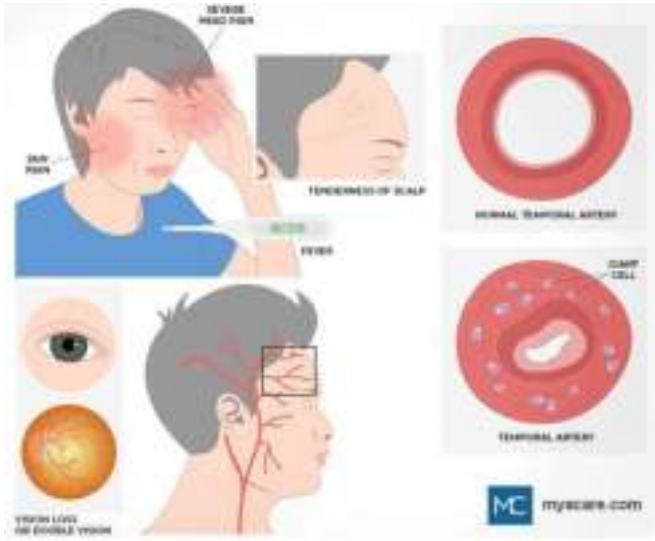
Generalised symptoms
- Systemic inflammatory disease
- Low grade fever (40%), fatigue, anorexia, weight loss

Jaw claudication (30% of patients)

Headache (≥ 75%)
- Mostly temporal (may be occipital, periorbital, holocranial)
- They progress gradually
- Local sensitivity of the scalp (pain when combing)

Neurological symptoms (TIA/stroke 30%)

Visual symptoms (15%)
- Vasculitis of ophthalmic artery → transient vision loss (amaurosis fugax)
- Partial/complete/permanent



Dx
- ↑ inflammatory parameters (CRP, Leu, IL-6)
- Biopsy (granulomatous inflammation)
- MRI manifestations of inflammation in the vessel

Tx
- Corticosteroids (iv., p.o.)
- Immunosuppressants (methotrexate)

Thank you for your attention