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PAIN, NEUROPATHIC PAIN, HEADACHE

Josef Bednařík

General neurology questions:

3. Pain (anatomy, characteristics, types of pain)

7. Cranial nerve V (anatomy, function, signs and symptoms of lesion)

Special neurology questions:

37. Headache (definition, classification)

38. Migraine, cluster headache

39. Trigeminal and glossopharyngeal neuralgias

Pain, neuropathic pain

Pain is probably the most frequent and one of the earliest medical symptoms at all. There is also increasing awareness of a neuropathic pain as a specific neurological syndrome with high prevalence in a population (estimated up to 8%)

2015		2020
– Google		
– Pain: 863.000.000 references		– 2.020.000.000 references
Neuropathic pain: 1.200.000 references		– 5.250.000 references
– Medline		
– Pain: 6610 articles		– 185.000 articles
– Neuropathic pain: 260 articles		– 8507 articles

Definition of pain

The revised IASP definition of pain (2020):
“**An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage**.”

Clinical classification of pain: acute and chronic pain

- **Acute pain**:
  - It lasts several days or weeks and is usually well localised.
  - It is a sign of tissue involvement caused by a trauma or disease.
  - In higher intensity it serves as a great psychic burden to the patient.
  - Therapy directed against the original cause together with analgesic therapy leads usually to the diminution or replacement of acute pain.
- **Chronic pain:**
  - The relation between the cause and the pain is not usually seen.
  - It lasts longer (more than 3 or 6 months), is unproportional to the evoked stimulus.
  - Badly localised, social and psychological factors play important roles.
  - It has no signal meaning but becomes the disease itself and the therapy is directed exclusively against the pain.

Patophysiological classification of pain: nociceptive and neuropathic pain

- To great extend pathophysiological classification overlaps with a clinical one: acute pain is mostly nociceptive and most chronic pain cases belongs to neuropathic pain type or has at least neuropathic component.
- Nociceptive pain: pain arises from actual or threatened damage to non-neural tissue. Nervous system function is normal. It is usually time limited, meaning when the tissue damage heals, the pain typically resolves. It tends to respond well to treatment with opioids. Example is inflammation or trauma.

Nociceptive and neuropathic pain

- **Neuropathic pain**: it is the **result of a lesion or disease of the peripheral or central nervous system**. The pain may persist for months or years beyond the apparent healing of any damaged tissues and is frequently **chronic**. In this setting, pain signals no longer represent an alarm about ongoing or impending injury, instead the alarm system itself is malfunctioning.
- Usually, neuropathic problems are not fully reversible, but partial improvement is often possible with proper treatment.

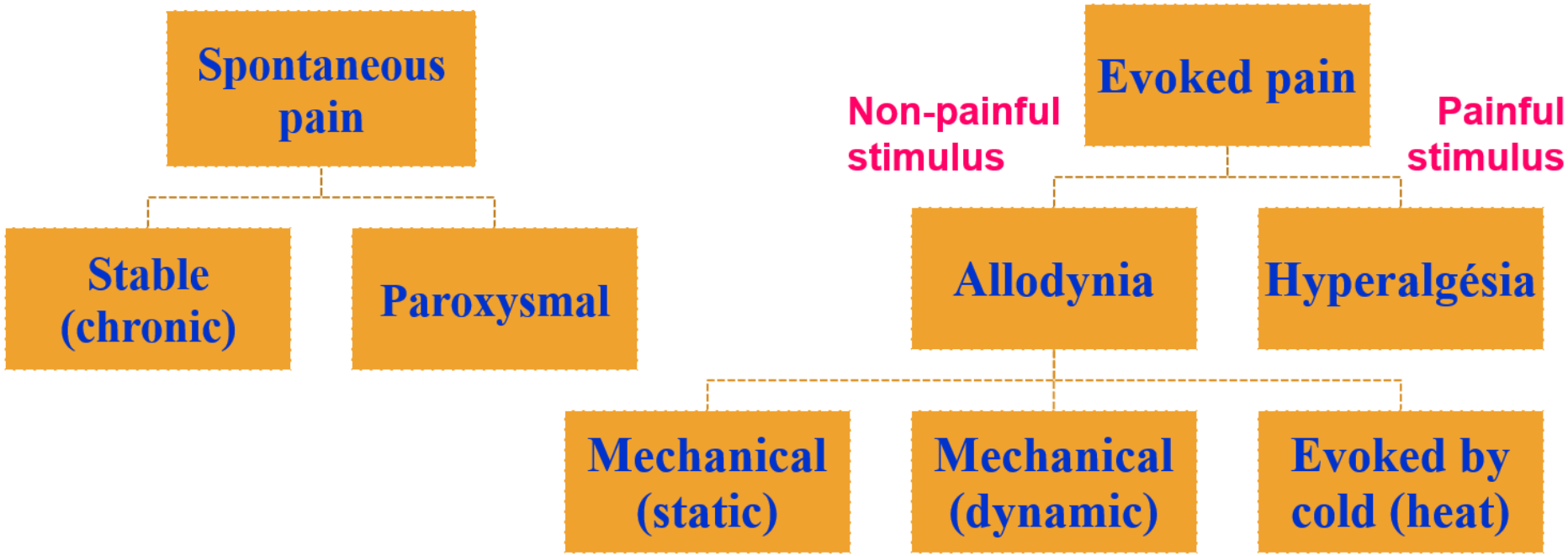
Definition of neuropathic pain

Definition (IASP 2012):

"....neuropathic pain is caused by a lesion or disease affecting the somatosensory system."

Neuropathic pain is a clinical syndrome with different types of pain and frequent and important co-morbidities, such as depression, anxiety and sleep disturbances.

Clinical symptoms and signs as a part of a neuropathic pain



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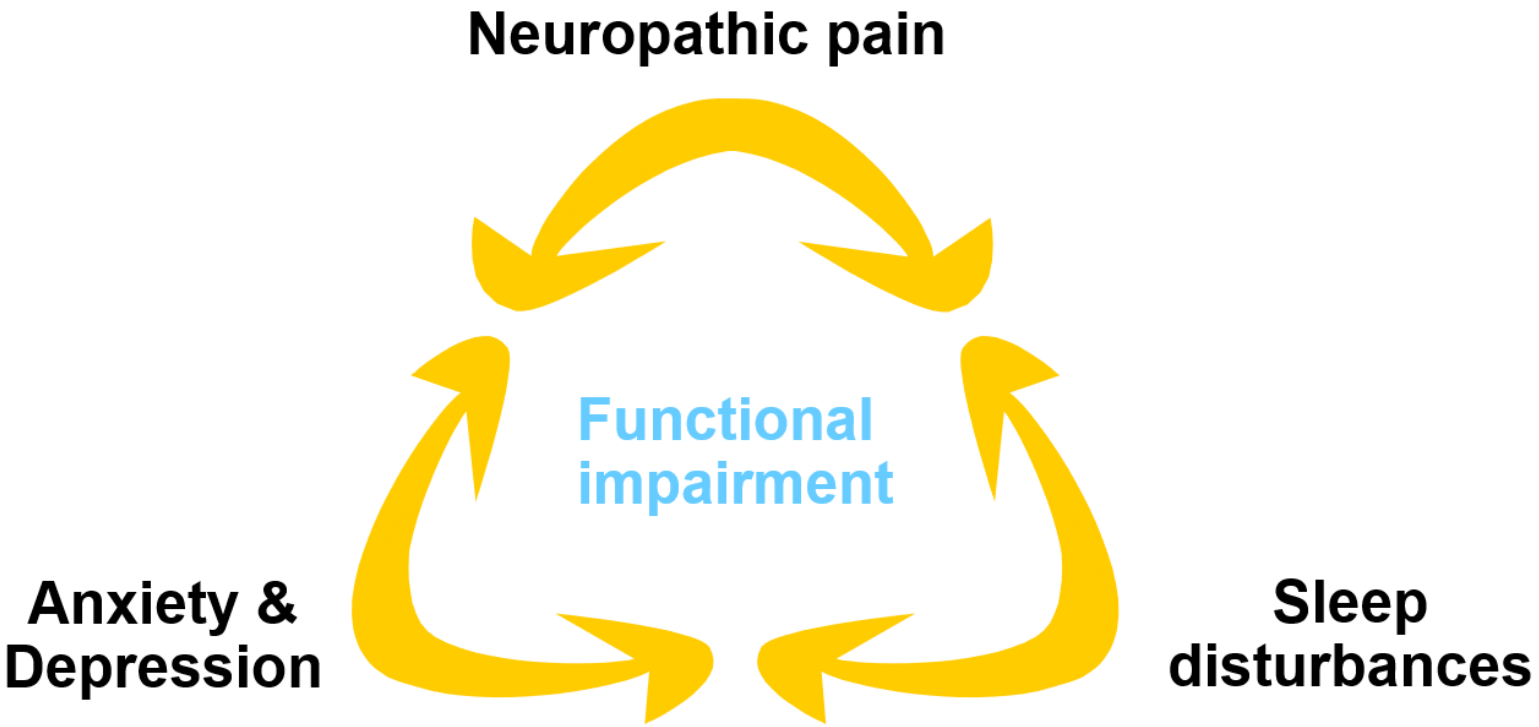
Terminology

- Pain can be **spontaneous** or **evoked**

- Evoked pain:
  - The term **hyperalgesia** refers to an increased sensitivity and a lowering of the threshold to painful stimuli.
  - The term **allodynia** refers to pain evoked by a stimulus that usually does not evoke pain (i.e. light touch).

Comorbidities of neuropathic (chronic) pain



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Epidemiology of neuropathic pain

- 20-24% of diabetics suffer from painful diabetic polyneuropathy
- 25-50% of patients older >50 years with herpes zoster develop chronic postherpetic neuralgia (≥3 months after healing of skin rush)
- Up to 20% post-mastectomy patients suffer with post-mastectomic pain
- 1/3 of patients with carcinoma suffer from neuropathic pain (isolated or in combination with nociceptive pain)
- 7% of patients with low back pain has neuropathic component of pain

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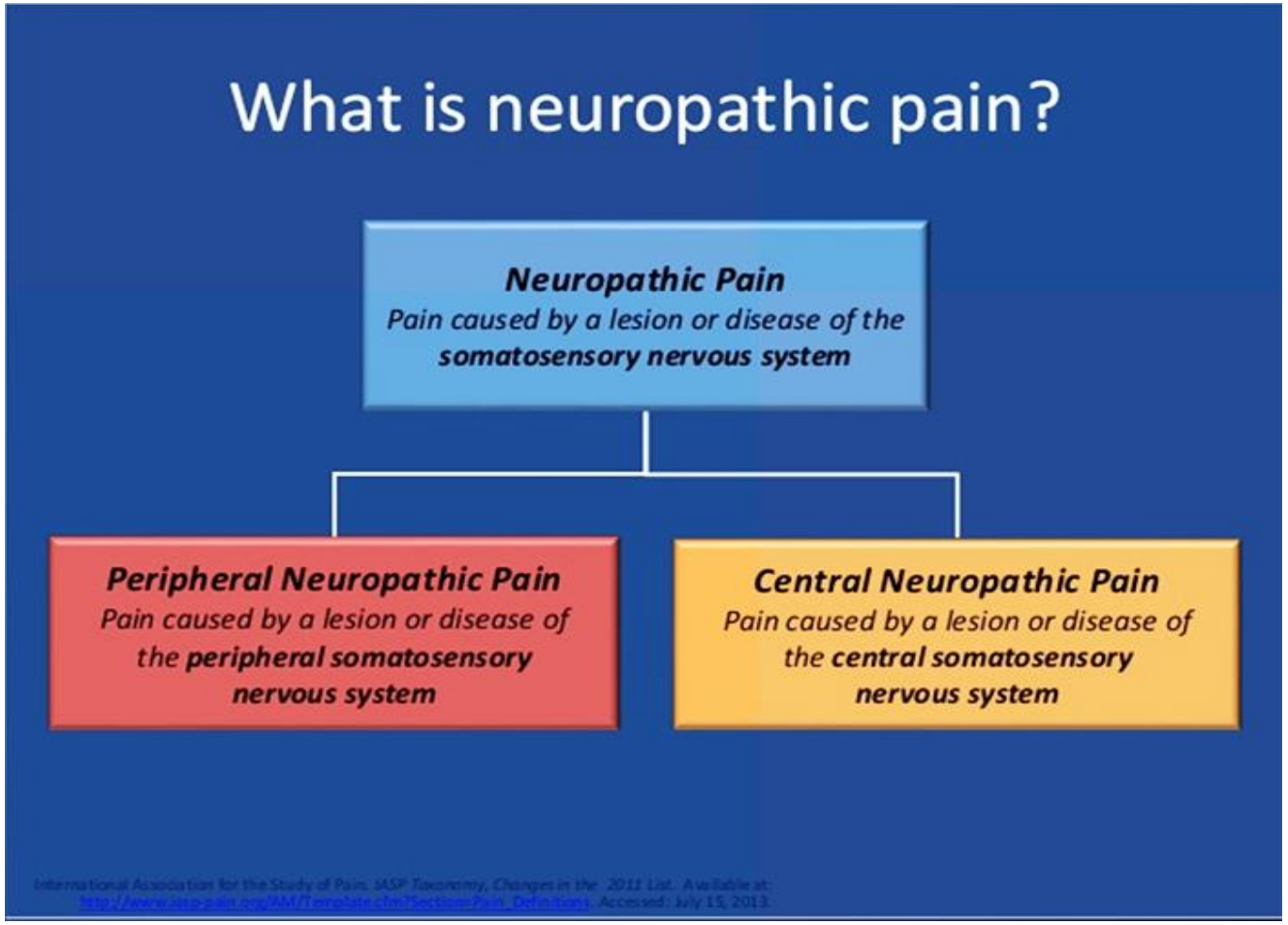
Epidemiology of neuropathic pain

According to neuropathic pain prevalece of neuropathic pain in population ranges between 1.5 - 6-8 % of population and serves as an important socioeconomic problem

Disease or clinical syndrome with neuropathic pain	**Prevalence/incidence of neuropathic pain**
Diabetic polyneuropathy	15-25% of diabetic population
HIV polyneuropathy	35 % of HIV-positive individuals
Postherpetic neuralgia	11-40/100 000/year
Trigeminal neuralgia	5-28/100 000/year
Carpal tunnel syndrome	180/100 000/year
Cervical radiculopathy	83/100 000/year
Stroke	8-10%
Multiple sclerosis	28-80% (trigeminal neuralgia 2-6 %) of MS cases
Spinal cord injury	10-80% of spinal cord injury cases

Anatomical correlate of neuropathic pain



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Pain receptors

Receptor characteristics				
Histology	**Type**	**Adequate stimulus**	**Nerve fiber type**	**Sensory quality**
Naked endings	Mechano-sensitive	Noxious mechanical stimuli	Small myelinated	Sharp fast pain
Naked endings	Polymodal	Noxious stimuli: 1.mechanical 2.thermal-above 43º C and below 14º C 3. various chemicals	Unmyelinated	Dull or burning sloww pain, itch
Naked endings	Termosensi-tive	Thermal 34-50º C	Unmyelinated	Warmth
Naked endings		Thermal	Small myelinated	Cold

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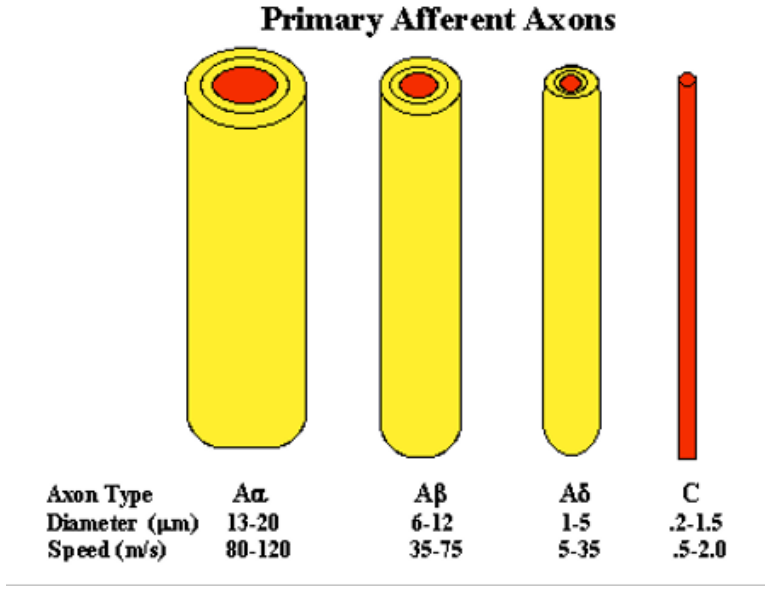
Specific pain receptors and nerve fibers

- Pain receptors (nociceptors) are naked nerve endings
- Pain stimuli are transmitted by unmyelinated (C) and thinly myelinated (A delta) afferent sensory fibres.



Picture taken from: https://faculty.washington.edu/chudler/cv.html

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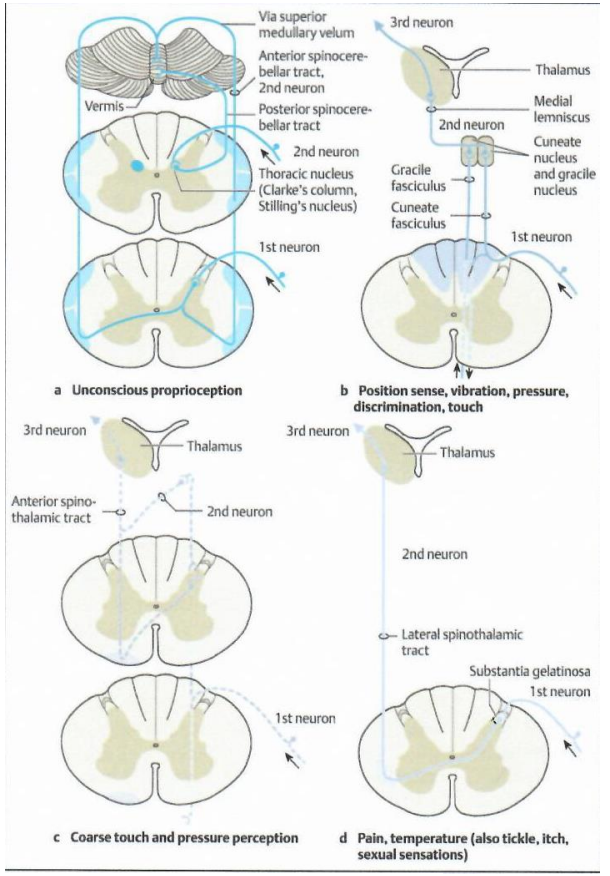
Anatomical dissociations of somatosensory pathways at spinal cord level

- There is an anatomical dissociation of two main afferent somatosensory pathways at spinal cord level:
  - spinothalamic pathway, formed by 2nd sensory neuron which after interpolation in the dorsal horns and crossing via anterior commisure goes up via contralateral anterolateral spinal columns (and contributing to the perception of pain and temperature),
  - dorsal columns and medial lemniscus system formed by fibres of the 1st sensory neurons going up via ipsilateral dorsal columns.



Baehr M, Frotscher M. Topical diagnosis in Neurology. 6th ed. Thieme 2014

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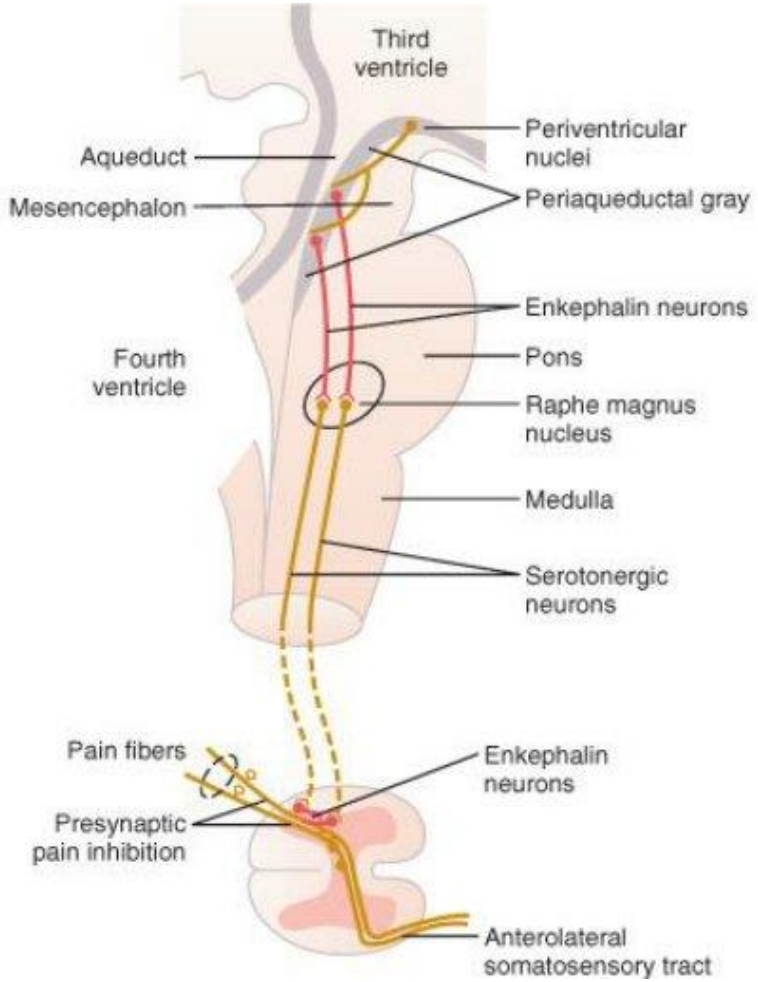
Inhibitory descending pain pathways

- There are also **descending fibers** from the brainstem structures that have an **inhibitory effect on pain**.

- These neurons are opioidergic and serotoninergic.



De Vrij, E. Acupuncture and pain relief. 2010

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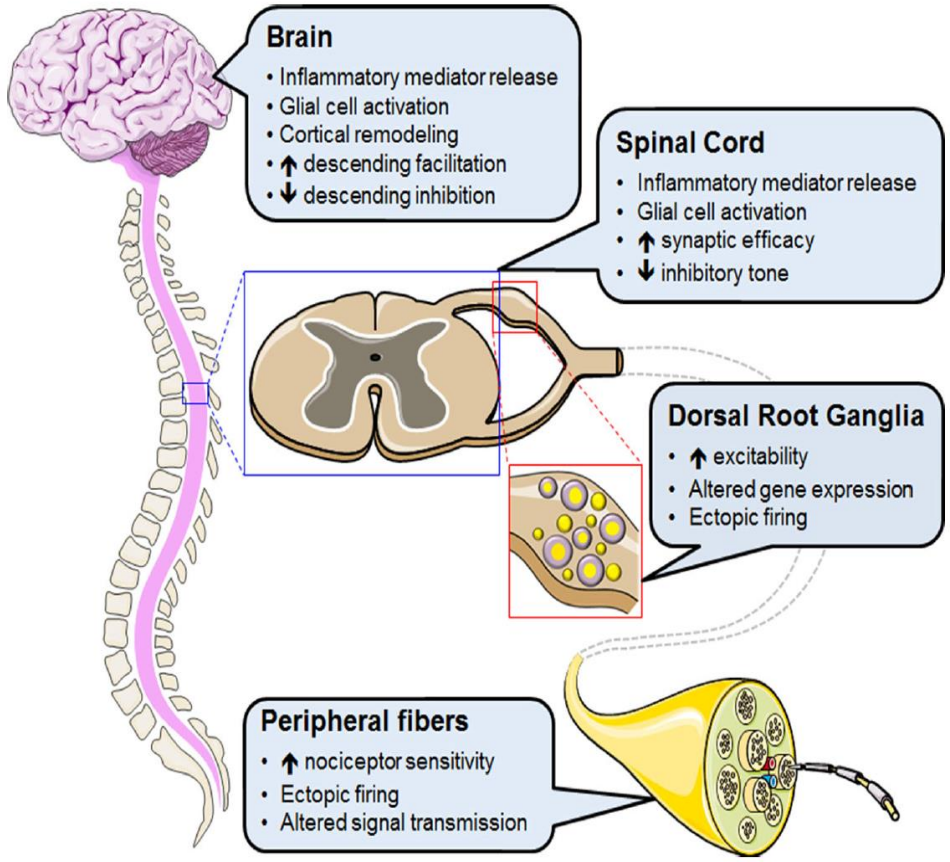
Pathophysiology of neuropathic pain

- Lesion or disease of the somatosensory system is a prerequisite for development of neuropathic pain, but different pathophysiological mechanisms may be involved:
  - **Peripheral sensitization**
  - **Hyperexcitability (ectopic discharges)**
  - **Central sensitization**
  - **Synaptic reorganization**
  - **Denervation hypersensitivity**
  - **„Wind-up“ phenomenon**
  - **Loss of inhibitory control**

- Such a lesion, however, does not necessarily lead to neuropathic pain!!!



Meacham et al. Curr Pain Headache Rep 2017

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Pharmacotherapy of neuropathic pain

- In contrast to nociceptive pain, pharmacoterapy of neuropathic pain is based on s.c. atypical analgesics (co-analgesics).

- They are very often used and known also as antiepileptics (anticonvulsants) or antidepressant drugs.

Czech national guideline for pharmacotherapy of neuropathic pain 2011

Painful clinical syndrome	1. Choice drugs		2.Choice drugs		3.Choice drugs	
Painful polyneuropathy incl. Painful diabetic polyneuropathy	Calcium blocker modulators (A)	pregabalin	tramadol/opioids: Independently or in combination with paracetamol/ Drugs of 1. choice (A)	tramadol	antiepilep-tics	phenytoin (C)
		gabapentin		morphin		
	TCA (A)	amitriptylin		oxycodon		Carbamaze-pine (C)
		nortriptylin		fentanyl		
		imipramin			NMDA receptor inhibitors	dextromethor-fane (B)
		klomipramin				
	SNRI (A)	duloxetin venlafaxin			Thioctic acid (B)	

Adopted from: Bednařík J, Ambler Z, Opavský J, Keller O, Rokyta R, Mazanec R. Klinický standard farmakoterapie neuropatické bolesti. 2011.

Pharmacotherapy of neuropathic pain

Binder and Baron, 2016

The pharmacotherapy of neuropathic pain: number of trials, number of patients, number needed to treat, evidence levels (GRADE [27]), and common side effects (modified from [6])

	Number of trials	Number of patients	Number needed to treat [95% CI]	Evidence level (GRADE)	Examples of common side effects (may vary depending on drug and manufacturer)
Tricyclic antidepressants	15	948	3.6 [3.0; 4.4]	High	Drowsiness, fatigue, dizziness, hypotension, weight gain
Serotonin-norepinephrine reuptake inhibitors	10	2541	6.4 [5.2; 8.4]	High	Nausea, dry mouth, somnolence, headache
Pregabalin	25	5940	7.7 [6.5; 9.4]	High	Drowsiness, somnolence, peripheral edema, weight gain
Gabapentin	14	3503	7.2 [5.9; 9.1]	High	Somnolence, dizziness
Tramadol	6	741	4.7 [3.6; 6.7]	Intermediate	Dizziness, nausea
High-potency opioids	7	838	4.3 [3.4; 5.8]	Intermediate	Sedation, dizziness, headache, constipation, nausea, itch
Capsaicin 8% patch*	6	2073	10.6 [7.4; 18.8]	High	Pain or erythema at the site of application

*Only peripheral neuropathic pain. CI, confidence interval. Only evidence of high or intermediate quality was considered in the construction of this table

Definition and classification of headaches

- Headache, i.e. pain perceived in the head region (or propagated to it), is very frequent. Almost all individuals experience headache at least in the short-term. Headache thus belongs among the most frequent neurological symptoms or diseases.
- **The main classification of headaches according to etiology comprises:**
- Primary headaches (disease)
- Secondary headaches (symptom)
- Painful cranial neuropathies (i.e. pain in the distribution of a cranial nerve) and other facial pains.

Classification of headache

ICHD-3

Cephalalgia
An International Journal of Headache

International Headache Society

Cephalalgia
2018, Vol. 38(1) 1–211

DOI: 10.1177/0333102417738202
journals.sagepub.com/home/cep

SAGE

Headache Classification Committee of the International Headache Society (IHS)

The International Classification of Headache Disorders, 3rd edition

https://ichd-3.org/

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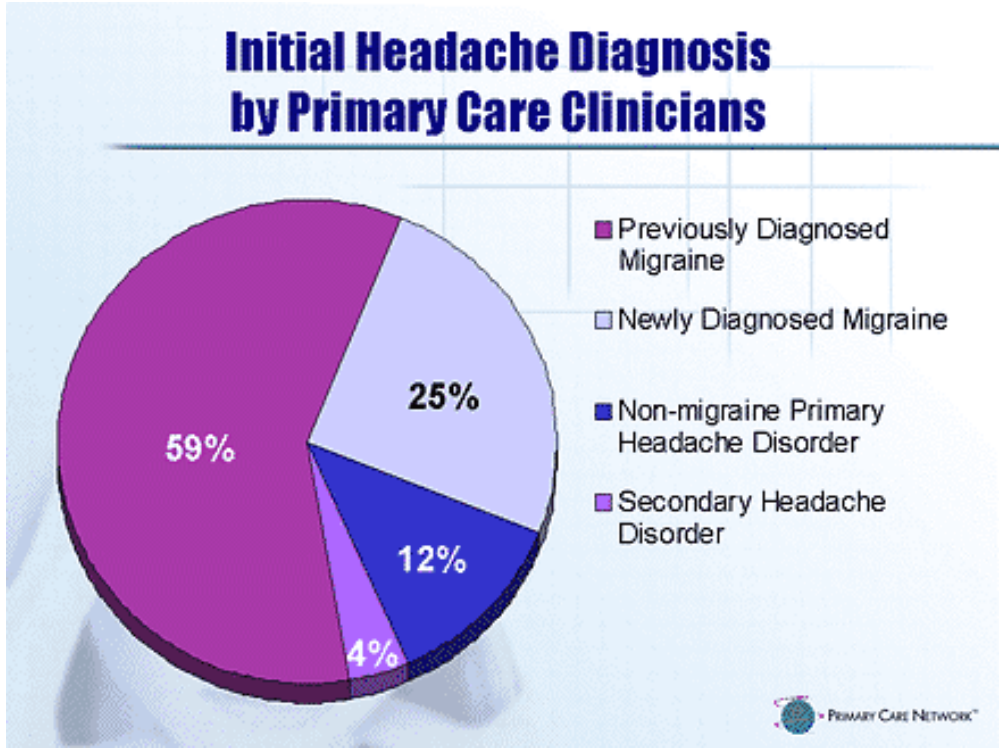
3rd International classification of headache disorders (2018)

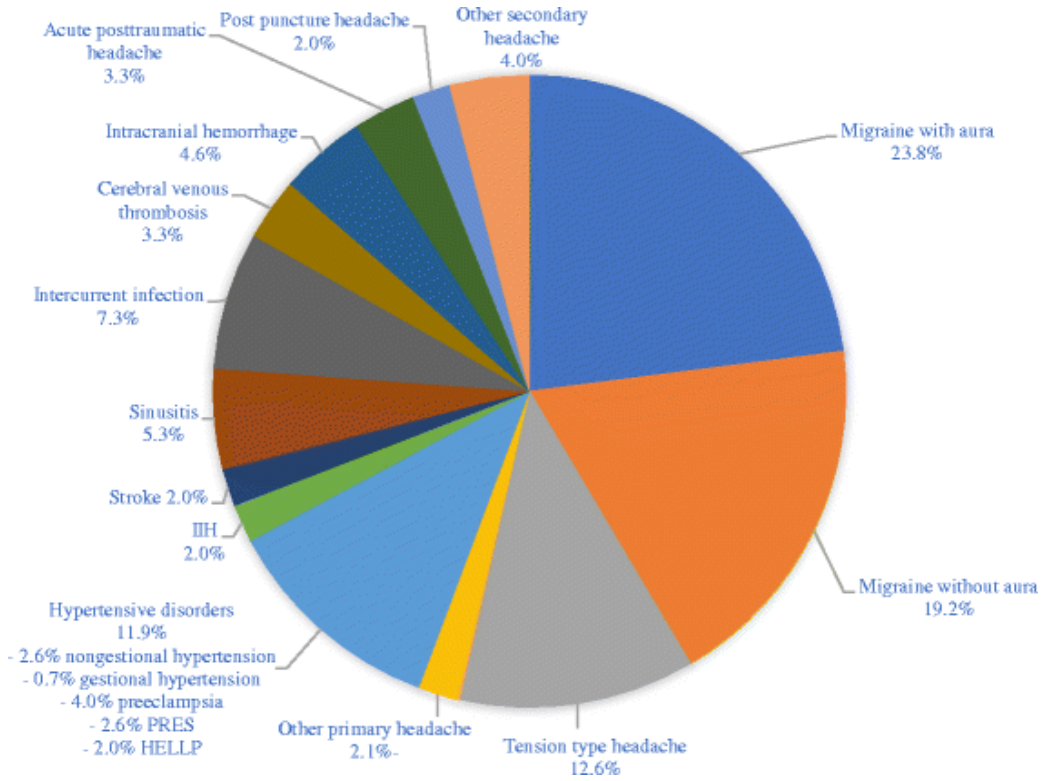
1. Migraine
2. Tension type headache
3. Trigeminal autonomic cephalalgias (incl. Cluster headache)
4. Other primary headache disorders
5. - 12. Secondary headaches
13. Painful lesions of the cranial nerves and other facial pain (incl. Trigeminal neuralgia)
14. Other headache disorders

Epidemiology of headaches

– Primary headaches form more than 90% of headaches cases.



Picture taken from: https://www.medscape.org/viewarticle/451273_2



Acute headaches in pregnancy. Raffaelli et al. J Headache Pain 2017.

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Epidemiology of migraine

- Migraine is one of the most prevalent and disabling medical illnesses in the world.
- WHO ranks migraine as the **third most prevalent medical condition** and the **second most disabling neurological disorder** in the world.
- The annual and lifetime prevalence are 18% and 33% in women, respectively, and 6% and 13% in men.
- Migraine affects approximately 10% of school-aged children (5–18 years), and at prepubertal ages (<13 years) the rate of onset of migraine is slightly higher in boys than in girls.

Classification of migraine

1. Migraine

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 brainstem aura
- 1.2.3 Hemiplegic migraine
  - 1.2.3.1 Familial hemiplegic migraine (FHM)
    - 1.2.3.1.1 Familial hemiplegic migraine type 1 (FHM1)
    - 1.2.3.1.2 Familial hemiplegic migraine type 2 (FHM2)
    - 1.2.3.1.3 Familial hemiplegic migraine type 3 (FHM3)
    - 1.2.3.1.4 Familial hemiplegic migraine, other loci
  - 1.2.3.2 Sporadic hemiplegic migraine (SHM)
- 1.2.4 Retinal migraine

Classification of migraine

1.3 Chronic migraine
1.4 Complications of migraine
- 1.4.1 Status migrainosus
- 1.4.2 Persistent aura without infarction
- 1.4.3 Migrainous infarction
- 1.4.4 Migraine aura-triggered seizure

1.5 Probable migraine
- 1.5.1 Probable migraine without aura
- 1.5.2 Probable migraine with aura

1.6 Episodic syndromes that may be associated with migraine
- 1.6.1 Recurrent gastrointestinal disturbance
  - 1.6.1.1 Cyclical vomiting syndrome
  - 1.6.1.2 Abdominal migraine
- 1.6.2 Benign paroxysmal vertigo
- 1.6.3 Benign paroxysmal torticollis

Diagnostic criteria for migraine

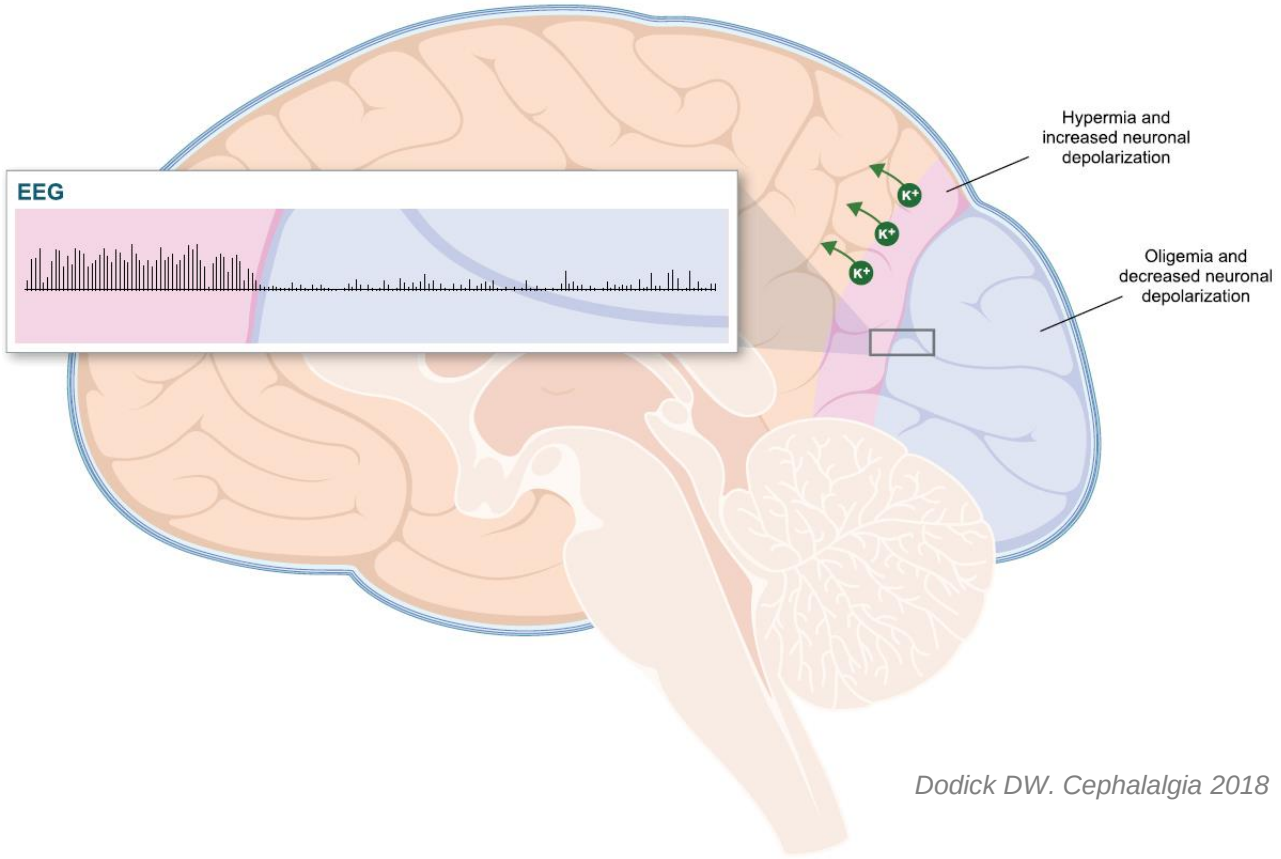
1.1 Migraine without aura

A. At least five attacks[1] fulfilling criteria B–D
B. Headache attacks lasting 4–72 hours (when untreated or unsuccessfully treated)[2,3]
C. Headache has at least two of the following four characteristics:
   1. unilateral location
   2. pulsating quality
   3. moderate or severe pain intensity
   4. aggravation by or causing avoidance of routine physical activity (e.g. walking or climbing stairs)
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.

1.2 Migraine with aura

A. At least two 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 three of the following six characteristics:
   1. at least one aura symptom spreads gradually over ≥5 minutes
   2. two or more aura symptoms occur in succession
   3. each individual aura symptom lasts 5–60 minutes[1]
   4. at least one aura symptom is unilateral[2]
   5. at least one aura symptom is positive[3]
   6. the aura is accompanied, or followed within 60 minutes, by headache
D. Not better accounted for by another ICHD-3 diagnosis.

Pathophysiology of migraneous aura: „cortical spreading depression“



Dodick DW. Cephalalgia 2018

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Migrenous visual aura

Positive symptoms:

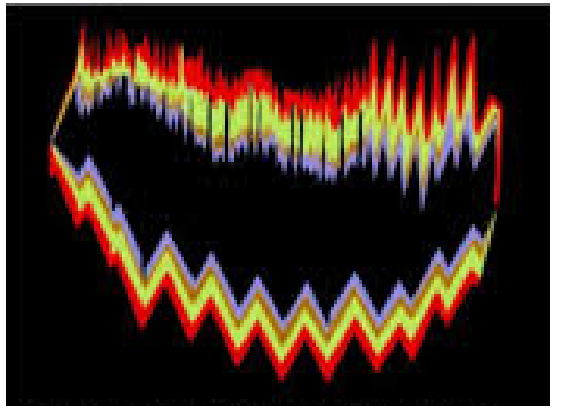
- Photopsy (flashes)
- Teichopsy („fortification spectra“)

Negative symptoms:

- Scotomas

Combined symptoms:

- Scotoma scintillans

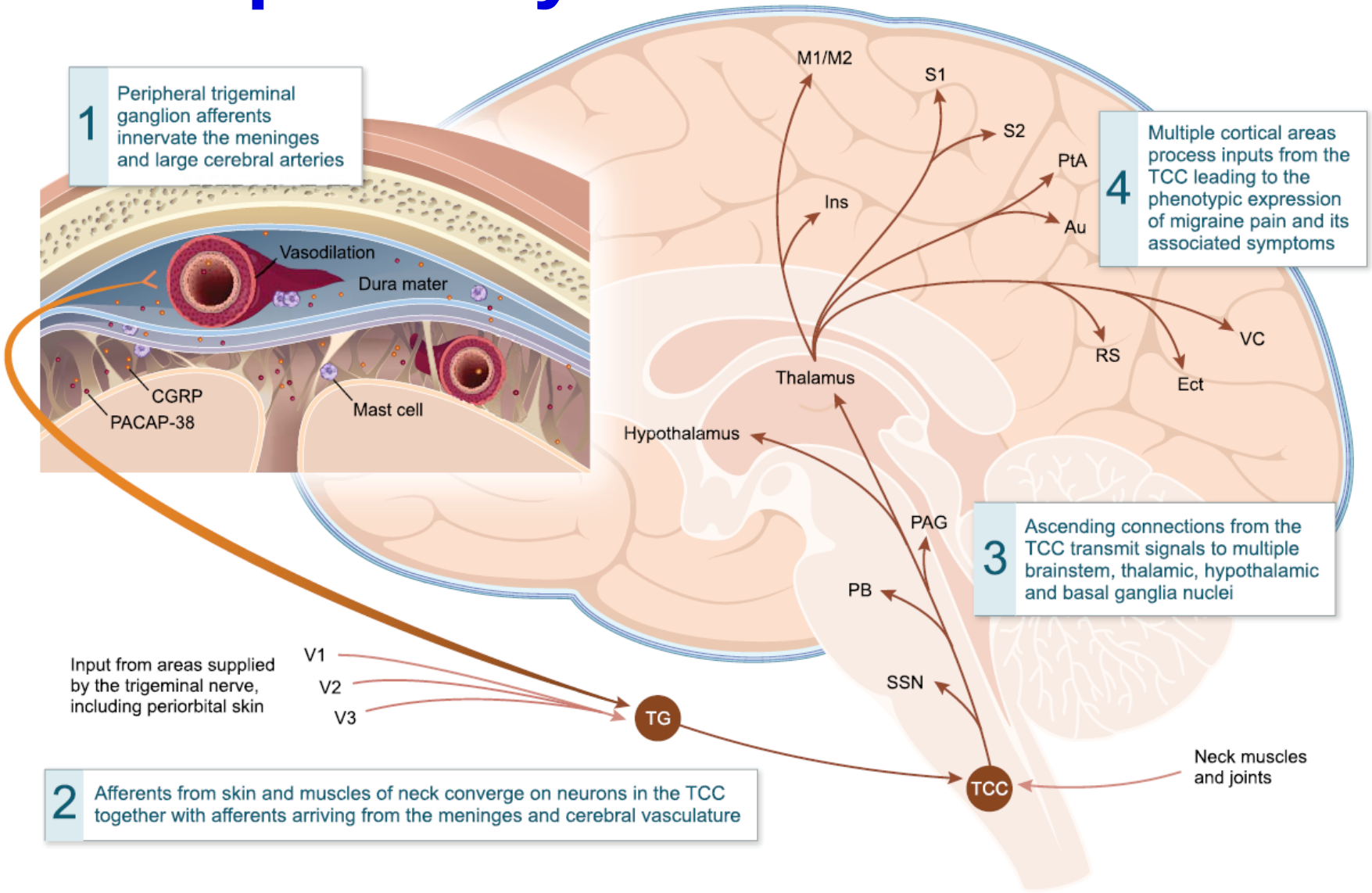


Chawla J. Drugs and Diseases, 2019

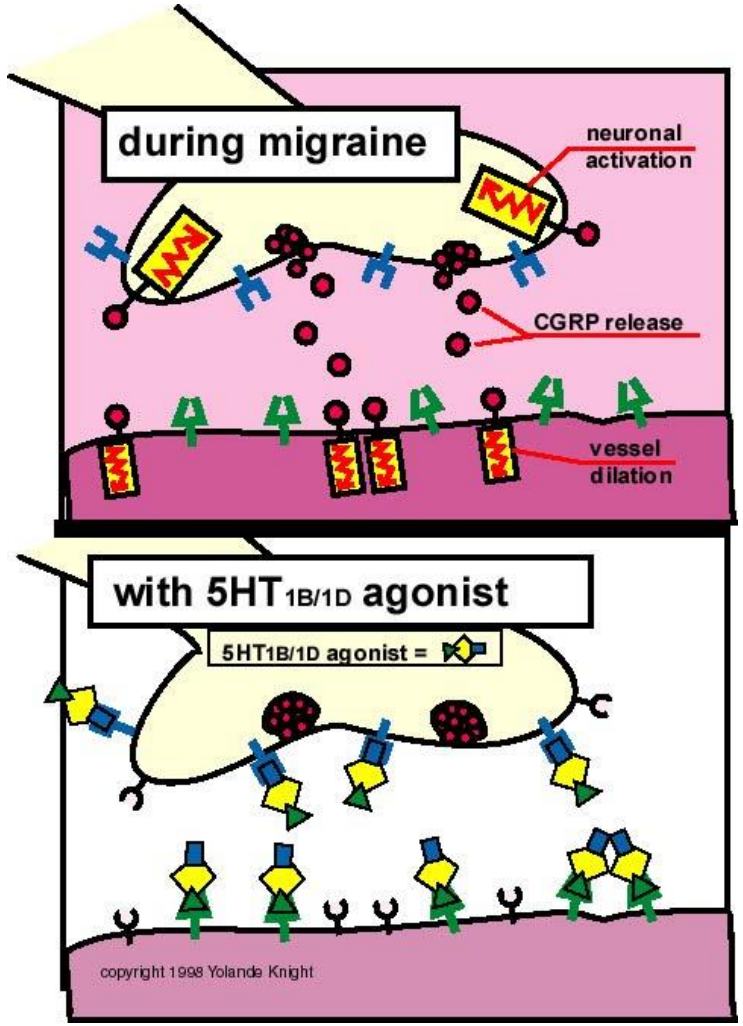
Banyas GT, Review of Optometry, 2015

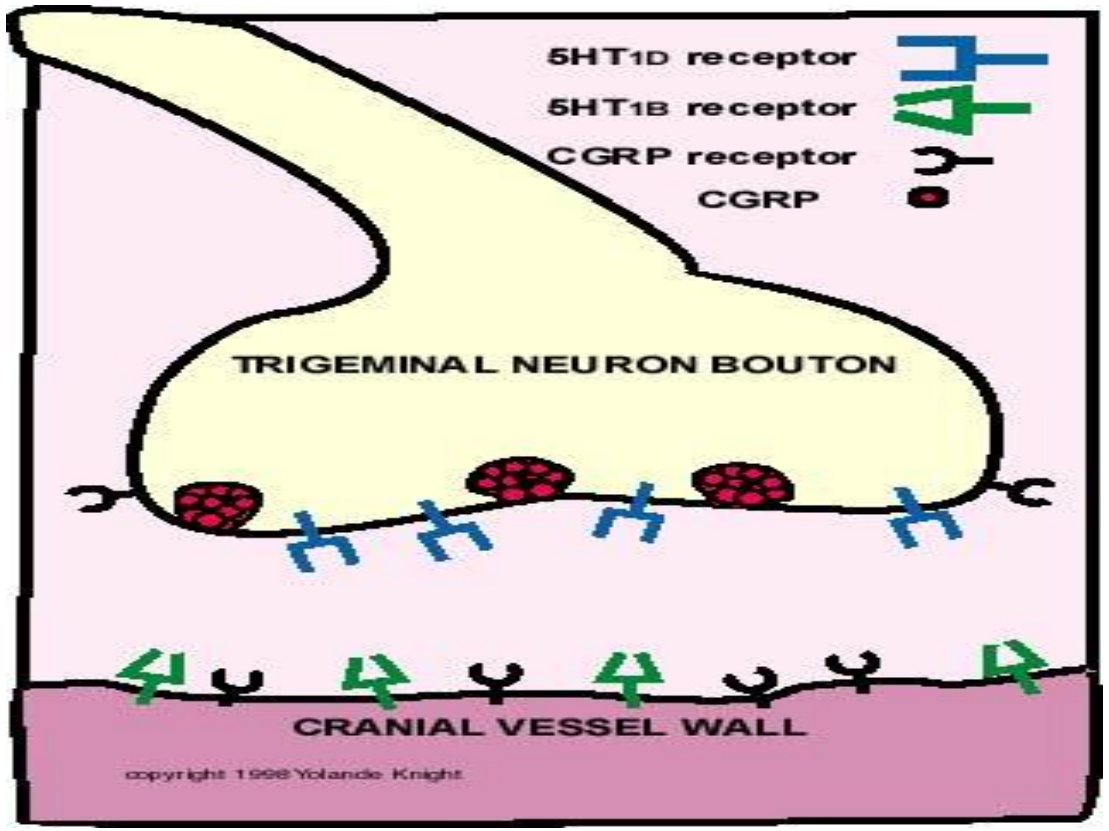
Triggering of pain via activation of trigeminovascular pathway

Dodick: Headache 2018



Trigeminovascular neurogennic inflammation model: serotonine receptors and triptans





Pictures taken from: Yolande Knight, UCL Queen Square Institute of Neurology, 1998.

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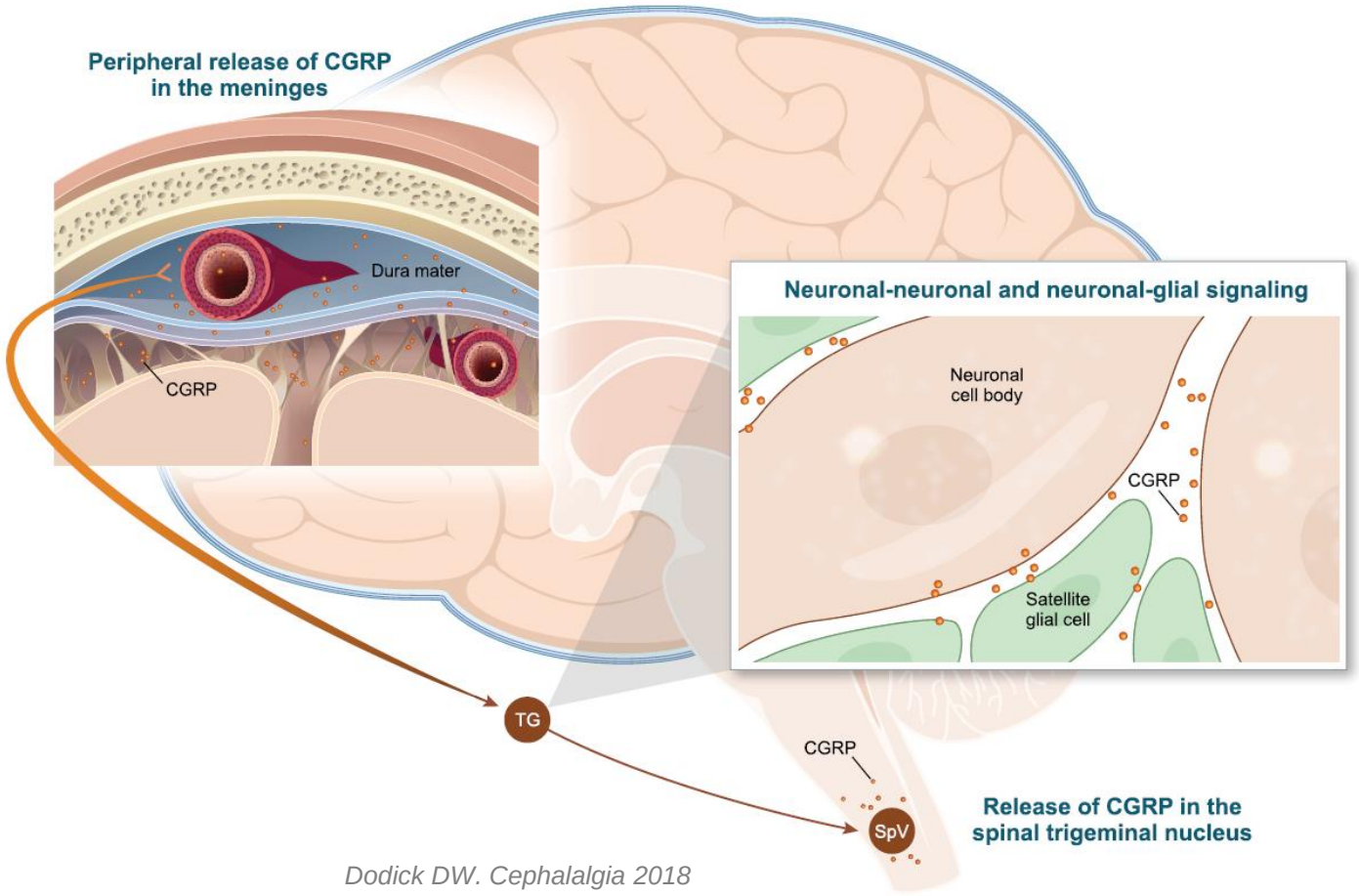
The role of CGRP in the pathophysiology of migraine



Dodick DW. Cephalalgia 2018

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Non-pharmacological treatment of migraine

- The avoidance of identified agravating (long-term effect) or triggered (short-term < 48 hours) factors:
  - **stress**
  - **the menstrual cycle**
  - **certain foods**
  - **trauma**
  - **caffeine withdrawal**
  - **alcohol**
  - **lack of sleep**
- If there is a reproducible trigger than its elimination will reduce the frequency of headaches.

  Unfortunately, this is often not possible because of the lack of a single reproducible trigger.
- Other non-pharmacological treatments has been suggested for migraine patients, including relaxation exercises, biofeedback, massage, acupuncture, chiropractic, osteopathy, and naturopathy, but their effect is not proven.

Non-pharmacological Treatment of Migraine

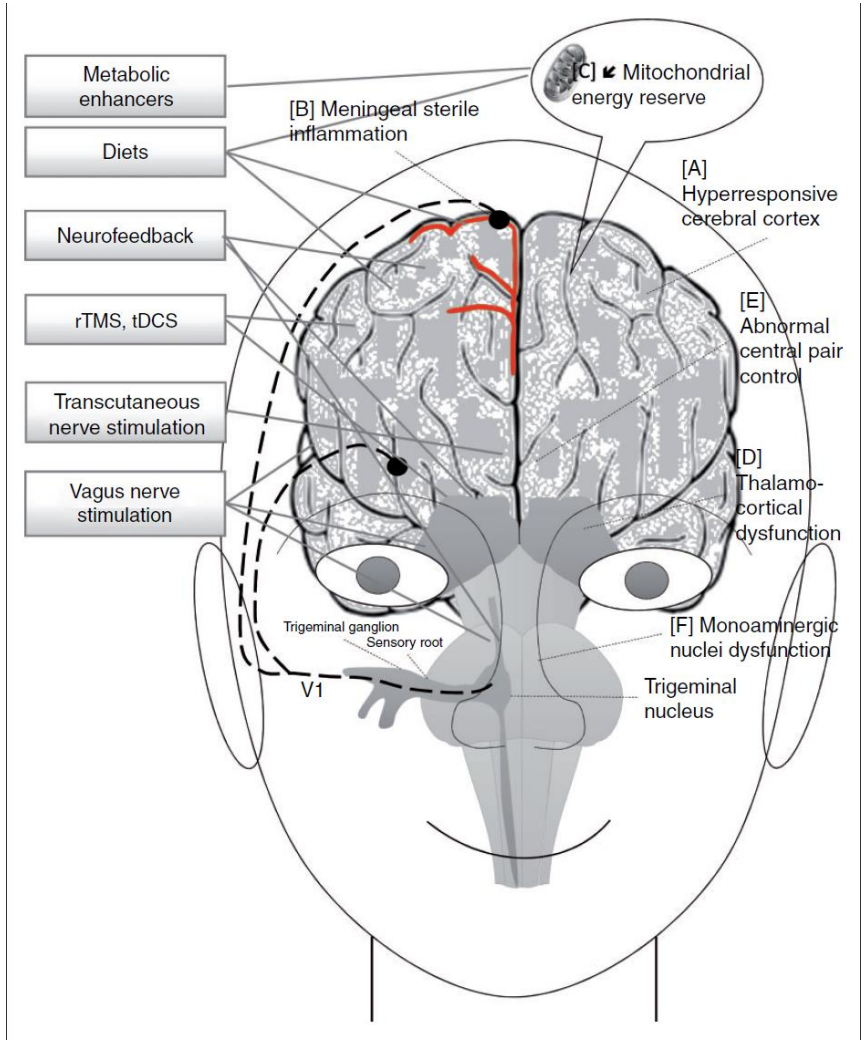

Figure 1. Scheme of migraine pathophysiological targets in migraine for non-pharmacological interventions. Diets could act by modulating neuronal excitability (a), mitigating sterile inflammation at the level of the trigeminovascular system (b) or enhancing mitochondrial energy metabolism (c). Nutraceuticals enhancing oxidative phosphorylation can augment the activity of mitochondrial complexes 1 and 2 (c). rTMS and tDCS are able to modify cortical responsivity (a) and thalamocortical circuits (d). Transcutaneous nerve stimulation may act by inducing long-term plasticity changes in central pain control centers (e). Vagus nerve stimulation is able to modulate the thalamus (d), the brainstem monoaminergic nuclei (f) and the cerebral cortex (a). Neurofeedback may act via neuroplastic changes in interconnected cerebral areas, such as the thalamus (d), brainstem (f) and various cortical networks (a), including executive, salient and attentional networks.
rTMS: repetitive transcranial magnetic stimulation; tDCS: transcranial direct current stimulation.

Coppola et al.: Cephalalgia 2015

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Pharmacological treatment of migraine

A. Treatment of acute attacks

1. Non-specific treatment

- Analgesic drugs
  - paracetamol
  - codeine phosphate
- Anti-inflammatory drugs
  - acetylosalicylic acid
  - ibuprofen
  - diclofenac
  - naproxen
  - ketorolac
- Anti-emetics: metoclopramide

2. Specific antimigraine treatment

- Ergotamines
  - ergotamine, dihydroergotamine
- Agonists of 5-HT_{1B} and 5-HT_{1D}: **triptans**
  - sumatriptan
  - zolmitriptan
  - naratriptan
  - rizatriptan
  - eletriptan
  - almotriptan
  - frovatriptan

Pharmacological treatment of migraine

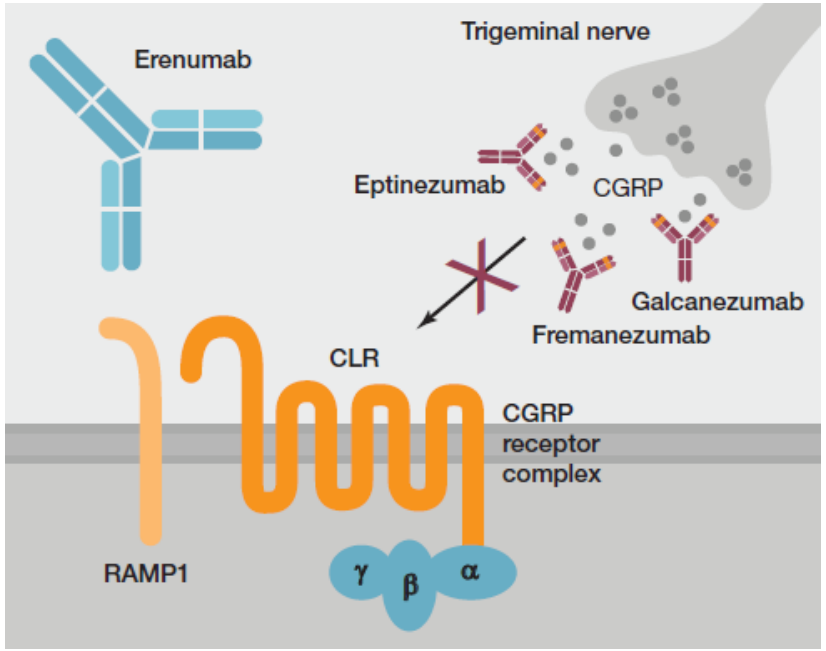
B. Preventive treatment

- beta blockers (propranolol, metoprolol, atenolol
- calcium blockers (verapamil, flunnarizine)
- anticonvulsants (gabapentin, topiramate, valproic acid)
- antidepressants (tricyclic antidepresants, venlafaxin)
- angiotensin converting enzyme inhibitors or angiotensin receptor blockers (lisinoprin, candesartan, cyproheptadine, ibuprofen, ketoprofen, naproxen)

Pharmacological treatment of migraine

B. Preventive treatment: blockade of CGRP or CGRP receptors

- **mABS bindings to CGPR**:
  - Galcanezumab
  - Eptinezumab
  - Frenezumab
- **mAB binding to the CGRP receptor**:
  - Erenumab
- This mABS proved to be efficient in reduction of both number and severity of both headache attacks in recurrent migraine, and in reduction of days with headache in chronic migraine



Edvinsson L, Cell 2018

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Classification of headache

2. Tension-type headache (TTH)

2.1 Infrequent episodic tension-type headache
- 2.1.1 Infrequent episodic tension-type headache associated with pericranial tenderness
- 2.1.2 Infrequent episodic tension-type headache not associated with pericranial tenderness

2.2 Frequent episodic tension-type headache
- 2.2.1 Frequent episodic tension-type headache associated with pericranial tenderness
- 2.2.2 Frequent episodic tension-type headache not associated with pericranial tenderness

2.3 Chronic tension-type headache
- 2.3.1 Chronic tension-type headache associated with pericranial tenderness
- 2.3.2 Chronic tension-type headache not associated with pericranial tenderness

2.4 Probable tension-type headache
- 2.4.1 Probable infrequent episodic tension-type headache
- 2.4.2 Probable frequent episodic tension-type headache
- 2.4.3 Probable chronic tension-type headache

Tension-type headache:

- **is not of pulsating quality**
- **Is not unilateral**
- **Is not aggravated by physical activity**
- **Is not of severe intensity**
- **Is not accompanied by nausea, vomitting, photofobia or phonofobia**

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Classification of headache

3. Trigeminal autonomic cephalalgias (TACs)

3.1 Cluster headache
- 3.1.1 Episodic cluster headache
- 3.1.2 Chronic cluster headache

3.2 Paroxysmal hemicrania
- 3.2.1 Episodic paroxysmal hemicrania
- 3.2.2 Chronic paroxysmal hemicrania

3.3 Short-lasting unilateral neuralgiform headache attacks
- 3.3.1 Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT)
  - 3.3.1.1 Episodic SUNCT
  - 3.3.1.2 Chronic SUNCT
- 3.3.2 Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)
  - 3.3.2.1 Episodic SUNA
  - 3.3.2.2 Chronic SUNA

3.4 Hemicrania continua
- 3.4.1 Hemicrania continua, remitting subtype
- 3.4.2 Hemicrania continua, unremitting subtype

3.5 Probable trigeminal autonomic cephalalgia
- 3.5.1 Probable cluster headache
- 3.5.2 Probable paroxysmal hemicrania
- 3.5.3 Probable short-lasting unilateral neuralgiform headache attacks
- 3.5.4 Probable hemicrania continua

Paroxysmal hemikrania: respond absolutely to indomethacin!!!

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Classification of headache

3.1 Cluster Headache

A. At least five attacks fulfilling criteria B–D

B. Severe or very severe unilateral orbital, supra-orbital and/or temporal pain lasting 15–180 minutes (when untreated)[1]

C. Either or both of the following:
   1. at least one of the following symptoms or signs, ipsilateral to the headache:
      a) conjunctival injection and/or lacrimation
      b) nasal congestion and/or rhinorrhoea
      c) eyelid oedema
      d) forehead and facial sweating
      e) miosis and/or ptosis
   2. a sense of restlessness or agitation

D. Occurring with a frequency between one every other day and eight per day[2]

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

Classification of headache

4.Other primary headache disorders

4.1 Primary cough headache
4.1.1 Probable primary cough headache
4.2 Primary exercise headache
4.2.1 Probable primary exercise headache
4.3 Primary headache associated with sexual activity
4.3.1 Probable primary headache associated with sexual activity
4.4 Primary thunderclap headache
4.5 Cold-stimulus headache
4.5.1 Headache attributed to external application of a cold stimulus
4.5.2 Headache attributed to ingestion or inhalation of a cold stimulus
4.5.3 Probable cold-stimulus headache
4.5.3.1 Headache probably attributed to external application of a cold stimulus
4.5.3.2 Headache probably attributed to ingestion or inhalation of a cold stimulus
4.6 External-pressure headache
4.6.1 External-compression headache
4.6.2 External-traction headache
4.6.3 Probable external-pressure headache
4.6.3.1 Probable external-compression headache
4.6.3.2 Probable external-traction headache
4.7 Primary stabbing headache
4.7.1 Probable primary stabbing headache
4.8 Nummular headache
4.8.1 Probable nummular headache
4.9 Hypnic headache
4.9.1 Probable hypnic headache
4.10 New daily persistent headache (NDPH)
4.10.1 Probable new daily persistent headache

The secondary headaches

5. Headache attributed to trauma or injury to the head and/or neck
6. Headache attributed to cranial and/or cervical vascular disorder
7. Headache attributed to non-vascular intracranial disorder
8. Headache attributed to a substance or its withdrawal
9. Headache attributed to infection
10. Headache attributed to disorder of homoeostasis
11. Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structure
12. Headache attributed to psychiatric disorder

Secondary headache	
Type	*Prevalence (%)*
Systemic infection	63
Head injury	4
Drug-induced headache	3
Subarachnoid hemorrhage	<1
Vascular disorders	1
Brain tumor	0-1

Rasmussen et al. 1995

New classification of trigeminal neuralgia

- Etiology
  1. Classical TN (neurovascular conflict – NCV - with morphological changes of trigeminal nerve due to compression)
  2. Secondary TN (other pathology or disease as a cause)

- Clinical form (phenotype):
  1. Pure paroxysmal form
  2. TN with concomitant continuous pain

Classical trigeminal Neuralgia

- It involves mostly 2. and 3. trigeminal branch, 1. branch is involved in 5% only.
- Pain is sharp, lancinating, like electrique shocks, sometimes continues as ongoing dull pain.
- Pain is mostly unilateral (bilateral in 3% only).
- Trigger zone is present in 50% of patients. Pain could be triggered by chewing, cleaning the tooth, speaking, washing, yawning, laughing, blowing one‘s nose.
- There is no motor or sensory deficit in trigeminal zone.
- Remisions could last months or years.

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Classical trigeminal neuralgia: diagnostic criteria

Diagnostic criteria are based onthe characteristics of pain, normal neurological findings and the absence of clear cause of pain.

A. Attacks of pain lasting from a fragment of second to 2 minutes in an area of one or more trigeminal branches and complying with criteria B and C.

B. Pain has to have one of the following characteristics:

- Intense, sharp, superficial, stabbing
- Induced from a trigger zone or triggeering factors

C. Atacks are stereotypic in an individual patient

D. No other pathology or disease as a cause of pain.

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Classical trigeminal neuralgia: etiology

Neurovascular confict is a cause – a compression of trigeminal nerve by a vessel (mostly a. cerebelli superior, less frequently by a. cerebelli anterior inferior or a. basilaris) 4-6 mm after the exit from the brainstem (transitional zone from central – oligodendroglia – to peripheral – Schwann cells – myelin.

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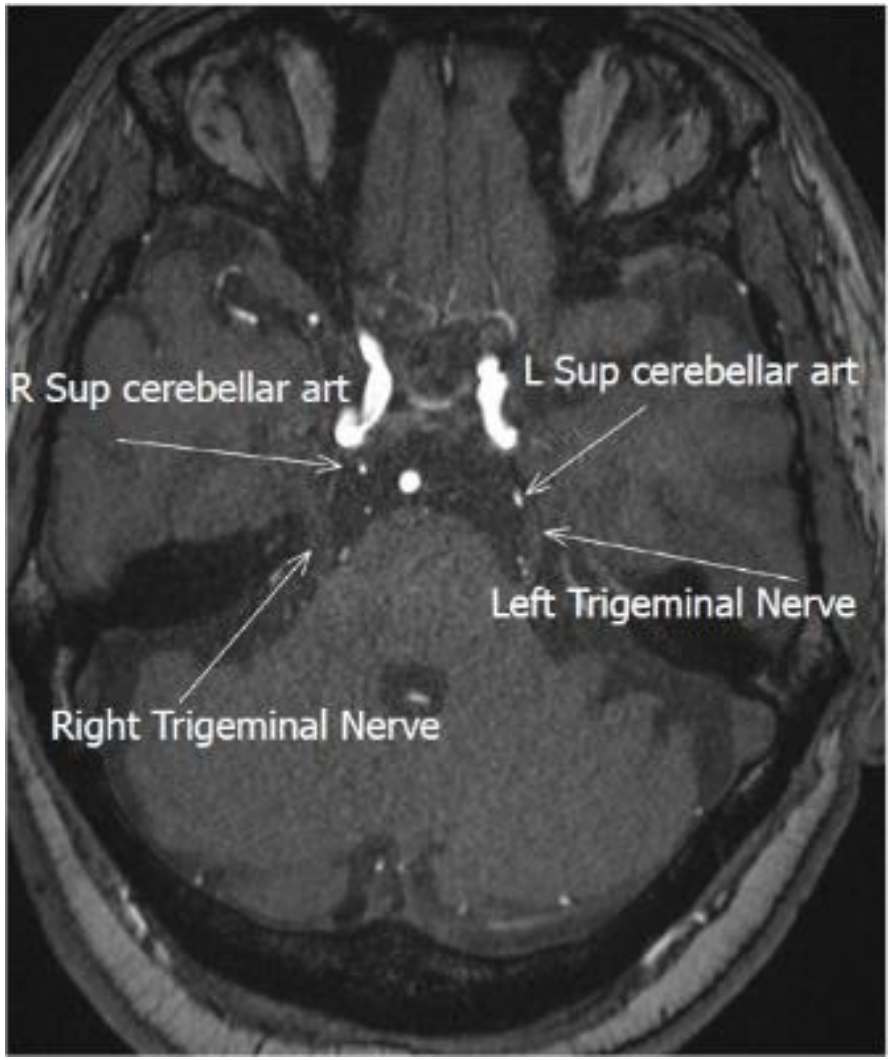
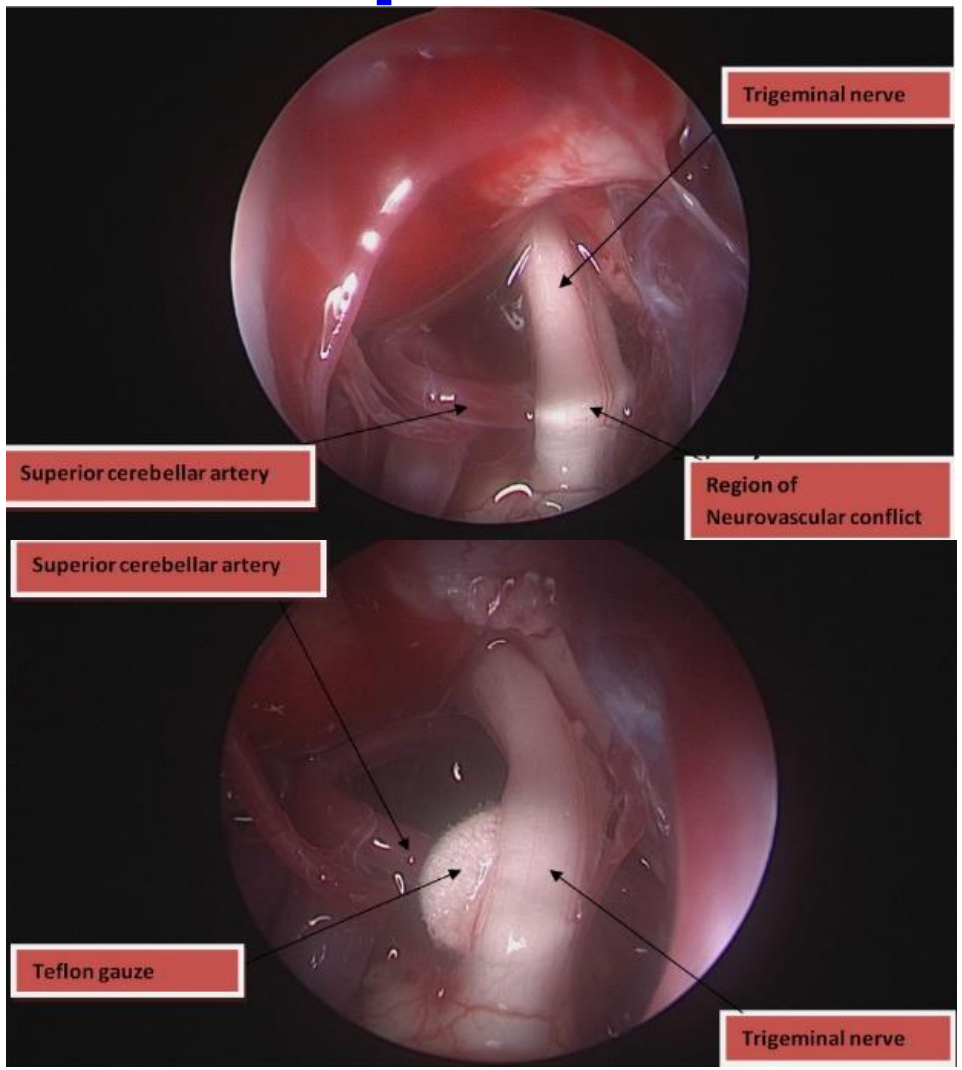
Neurovascular conflict



Microvascular decompression



Turton M, Malan-Roux, P. Stomatological Dis Sci 2019

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Secondary trigeminal neuralgia

It is a symptom of another disease.

- Is is possible to reliably clinically distinguish secondary and classical TN?

There are samo clues to secondary TN:

- younger age
- worse therapeutical response
- involvement of 1. trigeminal branch
- sensory deficit
- According to last guideline it is NOT possible to clinically differentiate classical and secondary TN!!!

MRI focused on NCV and other causes of TN should be a part of routine diagnostic algorithm.

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Secondary trigeminal neuralgia

- 3% of TN is caused by multiples sclerosis (MS), especially between 20-40 years; 1% of MS patients develop TN
- Painful ophthalmoplegia syndrome (Tolosa-Hunt) – granulomatous inflammation of the cavernous sinus)
- Compression of the trigeminal nerve in the cerebellopontine angle – schwannoma of n.VIII, V, meningeoma
- Other brainstem lesions – syringobulbia, basilar aneurysm
- Postherpetic neuralgia (herpes zoster ophthalmicus)

Trigeminal neuralgia: therapy

A. Acute treatment

- I.v. phenytoin or lidocain (low evidence)

B. Chronic treatment

- 1. Carbamazepine, oxcarbazepine (high evidence)
- 2. Lamotrigine, baclofen, phenytoin, pregabalin, gabapentin, Botox (low evidence)
- 3. Microvascular decompression
- 4. Gamma knife radiosurgery
- 5. Ablative neurosurgical techniques

C. Causative treatment in secondary TN

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