TBI-BH ECHO Presentation Tips & Guidelines

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Updates in Post-traumatic Headache

2022

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Disclosures

- None

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Overview

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Overview

- Definition of post-traumatic headache
- Pathophysiology (mild TBI/concussion)
- Different headache phenotypes
- Exacerbating factors
- Treatment modalities

Objectives

- Understand the most recent updates about the pathophysiology and epidemiology of post-traumatic headaches.
- Gain insight into how new medication and non-medication treatments work.
- Apply the newest medication and non-medication treatments to patients with post-traumatic headaches.

Overview

- **Definition of post-traumatic headaches**

- Advances in post-traumatic headaches :
  - Epidemiology
  - Pathophysiology
  - Treatments, including clinical applications of new treatments

Patient case

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Adam

- Adam is coming to the medical center with chief complaint of headaches that worsened 3 days after he was hit by a car while cycling to UW
- He fell off the bicycle and was able to get back on the bike
- He had no LOC
- He has severe one-sided headache that is constant with nausea, light and sound sensitivity, and he has other medical issues such as disrupted sleep, dizziness, and others
- He is currently unable to bike due to fear of having another accident
- He has been taking daily acetaminophen (Tylenol) and seen multiple other providers; nothing is helping his symptoms

Adam would like to know his headache diagnosis

- Does Adam meet criteria for post-traumatic headache?

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Question:

- Please enter on the chat is you think he meets PTH diagnosis, why and why not
- If yes, why yes
- If no, why no

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Definition of Post-traumatic headache

IHS CLASSIFICATION ICHD-3

7 days

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Definition of Post-Traumatic Headache

IHS CLASSIFICATION ICHD-3

- Secondary headache defined by the onset of headache or worsening of pre-existing headache within:
- 7 days following trauma or injury,
- Or 7 days after recovering consciousness,
- And/or within 7 days after recovering the ability to sense and report pain

- In some studies, extension of the time interval beyond 7 days has been proposed, however currently it is 7 days

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Questions for Patient

IHS CLASSIFICATION ICHD-3

- Did your headache start or worsen within 7 days following trauma or injury?
- Did your headache start or worsen 7 days after recovering consciousness?
- Did your headache start or worsen 7 days after recovering the ability to sense and report pain?

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Persistent Post-Traumatic Headache

IHS CLASSIFICATION ICHD-3

Headache that resolves within 3 months of onset → acute PTH
Headache that persists beyond 3 months → persistent PTH

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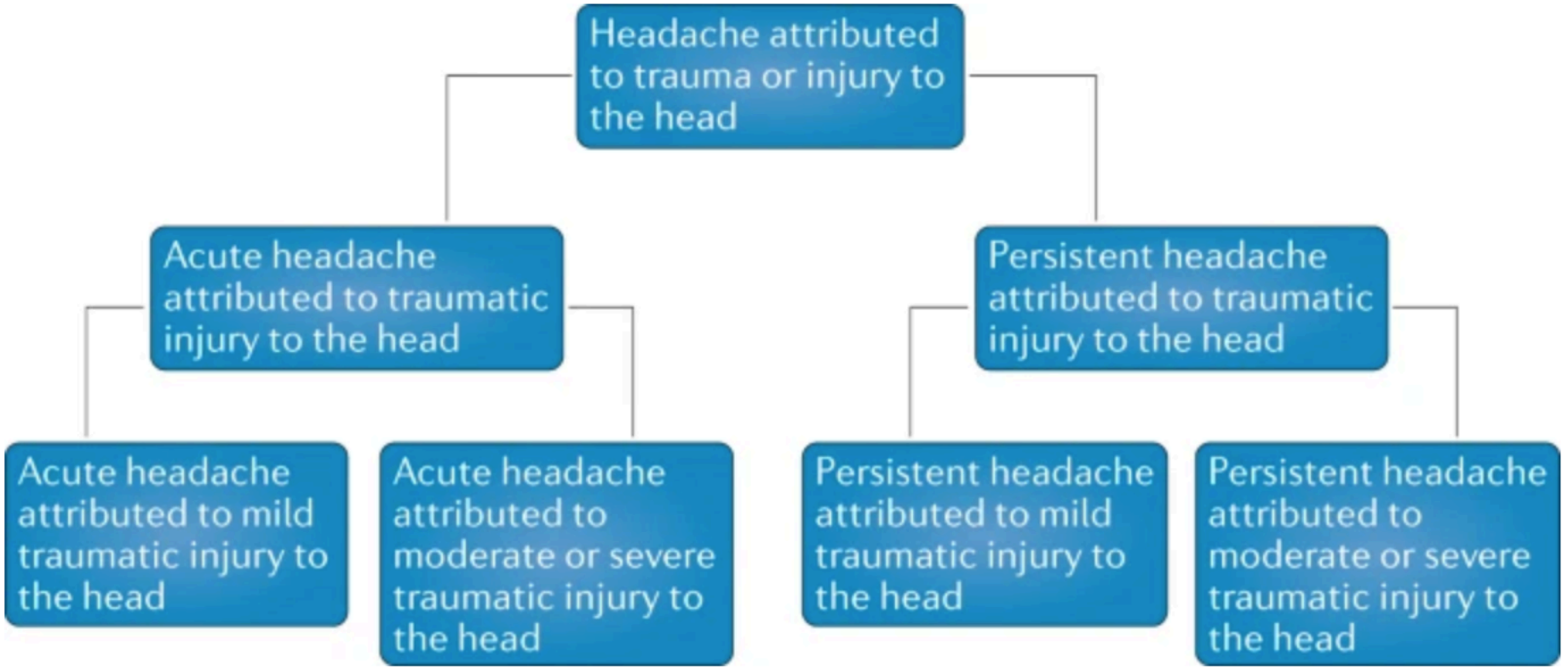
Comment Re: Duration

- How long is the duration of your new or worsening headache?
- Best is to ask for specific date of the accident and document this, instead of saying 3 months ago…

Classification of Post-traumatic headache

IHS CLASSIFICATION ICHD-3

Fig. 1: Classification of headache attributed to trauma or injury to the head.



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Definition of Post-traumatic headache

- PTH also includes headache attributed to whiplash or craniotomy

Headache Phenotype

IHS CLASSIFICATION ICHD-3

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Headache Phenotype

IHS CLASSIFICATION ICHD-3

Once we determine that someone has PTH, we look into phenotype.

The most common PTH phenotypes are:

- Migraine-like headache
- Tension-type like headache
- Less common: cluster-type headache and cervicogenic headache (we will not address these in this lecture)

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Definition of Migraine

IHS CLASSIFICATION ICHD-3

Recurrent headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

Diagnostic criteria:

A. At least five attacks[1] fulfilling criteria B-D

B. Headache attacks lasting 4-72 hr (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 (eg, 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.

Definition of Migraine

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   4. aggravation by or causing avoidance of routine physical activity (eg, 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.

This is a disabling condition that affects individuals in the prime of their lives.

Definition of Migraine with Aura

IHS CLASSIFICATION ICHD-3

- A. At least two attacks fulfilling criteria B and C
- B. One or more of the following fully reversible aura symptoms: visual, sensory, speech and/or language, motor, brainstem, retinal
- C. At least three of the following six characteristics:
  - At least one aura symptom spreads gradually over ≥5 minutes
  - Two or more aura symptoms occur in succession
  - Each individual aura symptom lasts 5-60 minutes1
  - At least one aura symptom is unilateral2
  - At least one aura symptom is positive3
  - The aura is accompanied, or followed within 60 minutes, by headache
- D. Not better accounted for by another ICHD-3 diagnosis.

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Definition of Chronic Migraine

IHS CLASSIFICATION ICHD-3

- Headache occurring on 15 or more days/month for more than 3 months, which, on at least 8 days/month, has the features of migraine headache.

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Definition of Tension-Type Headache

IHS CLASSIFICATION ICHD-3

- A. Headache may last minutes to days (30 minutes to 7 days)
- B. Headache has at least 2 of the following characteristics:
  - -Bilateral location
  - -Pressing/tightening (non-pulsating) quality
  - -Mild or moderate intensity
  - -Not aggravated by routine physical activity
- C. Both of the following:
  - -No more than one of photophobia, phonophobia
  - -No nausea nor vomiting
- D. Not better accounted for by another ICHD-3 diagnosis.

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Case Presentation

Adam would like to know his headache diagnosis

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Migraine Phenotype as an Example of PTH

- Adam meets criteria of both post-traumatic headache, and his headache phenotype is migraine type headache
- We enter both diagnoses of PTH and migraine in patient's chart
- There is not enough information to know if this is acute or persistent post-traumatic headache, and if this is episodic or chronic migraine

Migraine Phenotype as an Example of PTH

- Adam meets criteria of both post-traumatic headache, and his headache phenotype is migraine type headache
- We enter both diagnoses of PTH and migraine in patient's chart
- His headache is more than 3 months duration: persistent post-traumatic headache, and and he has chronic migraine with daily headaches

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Overview

- Definition of post-traumatic headaches
- Advances in post-traumatic headaches:
  - **Epidemiology**
  - Pathophysiology
  - Treatments, including clinical applications of new treatments

Epidemiology

nature reviews neurology

Review Article | Published: 16 September 2019

Post-traumatic headache: epidemiology and pathophysiological insights

Håkan Ashina, Frank Porreca, Trent Anderson, Faisal Mohammad Amin, Messoud Ashina, Henrik Winther Schytz & David W. Dodick ✉

Nature Reviews Neurology **15**, 607–617 (2019) | Cite this article

Ashina, H., Porreca, F., Anderson, T., Amin, F.M., Ashina, M., Schytz, H.W. and Dodick, D.W., 2019. Post-traumatic headache: epidemiology and pathophysiological insights. *Nature Reviews Neurology*, *15*(10), pp.607

Epidemiology

- Post-traumatic headaches
- Accounts for ~4% of all symptomatic headache disorders
- Headache is the most common sequelae of mild traumatic brain injury (mTBI) also known as concussion
- Estimate: 69 million people per year experience a traumatic brain injury (TBI) worldwide
- Patients with PTH commonly suffer from comorbidities such as anxiety and depression, both of which are among the leading causes of disability worldwide

Ashina, H., Porreca, F., Anderson, T., Amin, F.M., Ashina, M., Schytz, H.W. and Dodick, D.W., 2019. Post-traumatic headache: epidemiology and pathophysiological insights. *Nature Reviews Neurology*, *15*(10), pp.607

Adam

- Adam is currently not working due to daily headaches, fatigue, low energy, anxiety, depression and and new fear regarding riding his bike
- Adam would like to know what is his likelihood of being able to get back to work

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Epidemiology

- Among patients with PTH, 35% have not returned to work by 3 months after the injury (meaning 65% will return to work)
- Given that >3.8 million people are diagnosed with concussion in the USA annually
- The negative impact of PTH on work life and social activities is considerable

Ashina, H., Porreca, F., Anderson, T., Amin, F.M., Ashina, M., Schytz, H.W. and Dodick, D.W., 2019. Post-traumatic headache: epidemiology and pathophysiological insights. *Nature Reviews Neurology*, *15*(10), pp.607

Adam would like to know what is his likelihood of being able to get back to work

- Adam would like to know what is his likelihood of being able to get back to work
- 65% will return to work who had PTH by 3 months of injury

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Epidemiology

- The most common causes of TBI causing PTH
  - Traffic accidents (24–58%)
  - Falls (24–45%)
  - Sports (3–18%)
  - Violence (5–7%)

- About 40% of patients with TBI who reported acute PTH developed persistent PTH

Ashina, H., Porreca, F., Anderson, T., Amin, F.M., Ashina, M., Schytz, H.W. and Dodick, D.W., 2019. Post-traumatic headache: epidemiology and pathophysiological insights. *Nature Reviews Neurology*, *15*(10), pp.607

Pathophysiology

What are the updates in post-traumatic headache pathophysiology?

Possible disease mechanisms of PTH include:

- impaired descending modulation
- neurometabolic changes
- activation of the trigeminal sensory system

Pathophysiology

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5248 Accesses | **74** Citations | **82** Altmetric | Metrics

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Review Article

Posttraumatic Headache: Basic Mechanisms and Therapeutic Targets

Joshua Kamins, MD; Andrew Charles, MD

Frequent or continuous headache, often refractory to medical therapy, is a common occurrence after head trauma. In addition to being the most common acute symptom after traumatic brain injury (TBI), headache is also one of the most persistent and disabling symptoms. Different studies indicate that 18-58% of those suffering a TBI will have significant headache at 1 year following the trauma. In addition to being disabling on its own, posttraumatic headache (PTH) is a predictor of overall outcome after concussion. Despite its remarkable prevalence and associated social and economic costs, many fundamental and important questions about PTH remain unanswered. The purpose of this review is to identify key questions regarding the clinical characteristics of posttraumatic headache, its basic mechanisms, and its optimal management. We discuss phenotypic features of PTH, pathophysiological mechanisms of TBI including potential overlaps with those of migraine and other primary headache disorders, and potential novel targets for treatment. We suggest different strategies to finding answers to the questions regarding PTH in order to advance the understanding of the disorder and develop more effective therapies.

Kamins, J. and Charles, A., 2018. Posttraumatic headache: basic mechanisms and therapeutic targets. *Headache: The Journal of Head and Face Pain*, *58*(6), pp.811-826.

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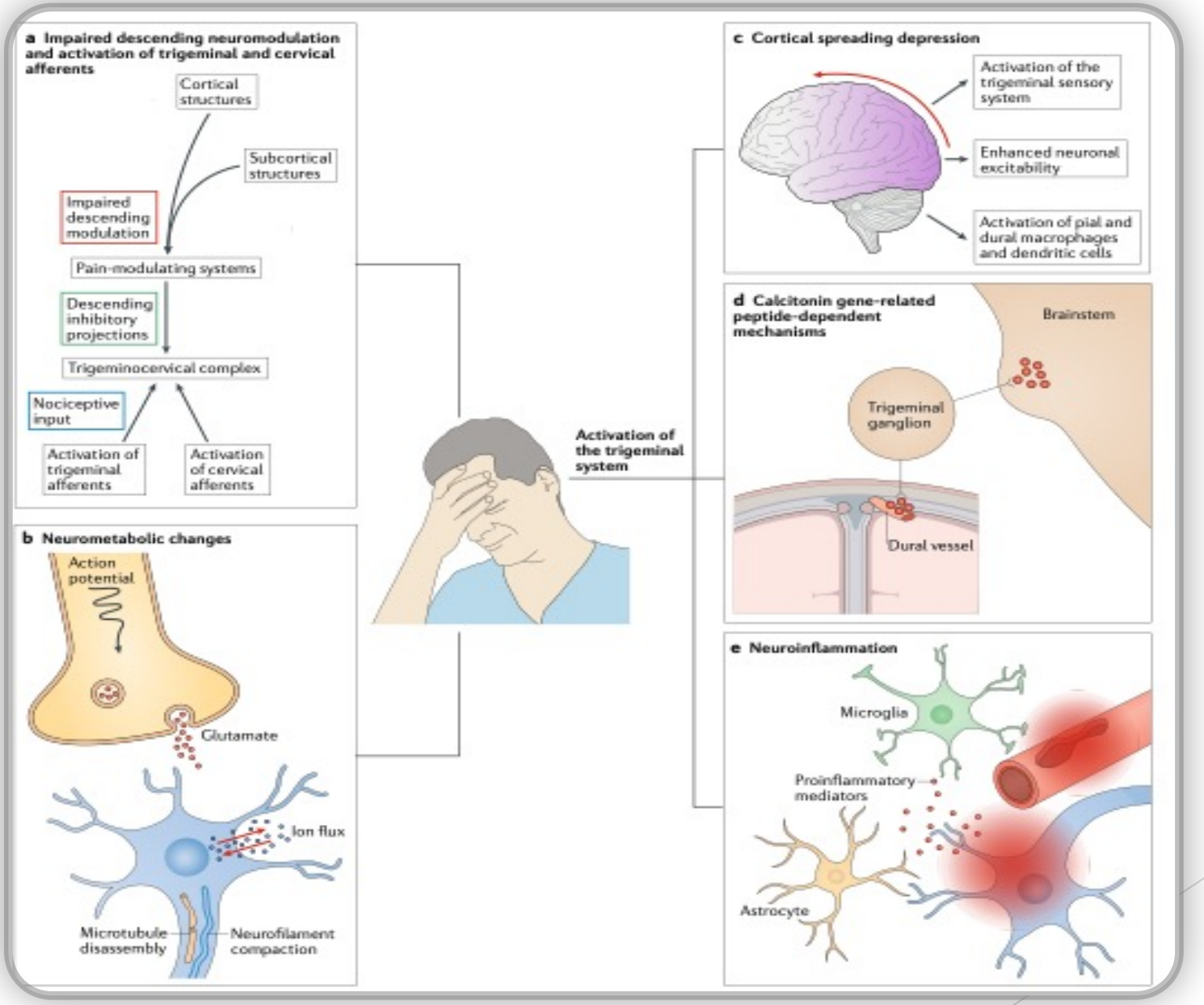
Proposed Pathophysiology of Post-Traumatic Headache



Ashina, et. al. Nature reviews 2018

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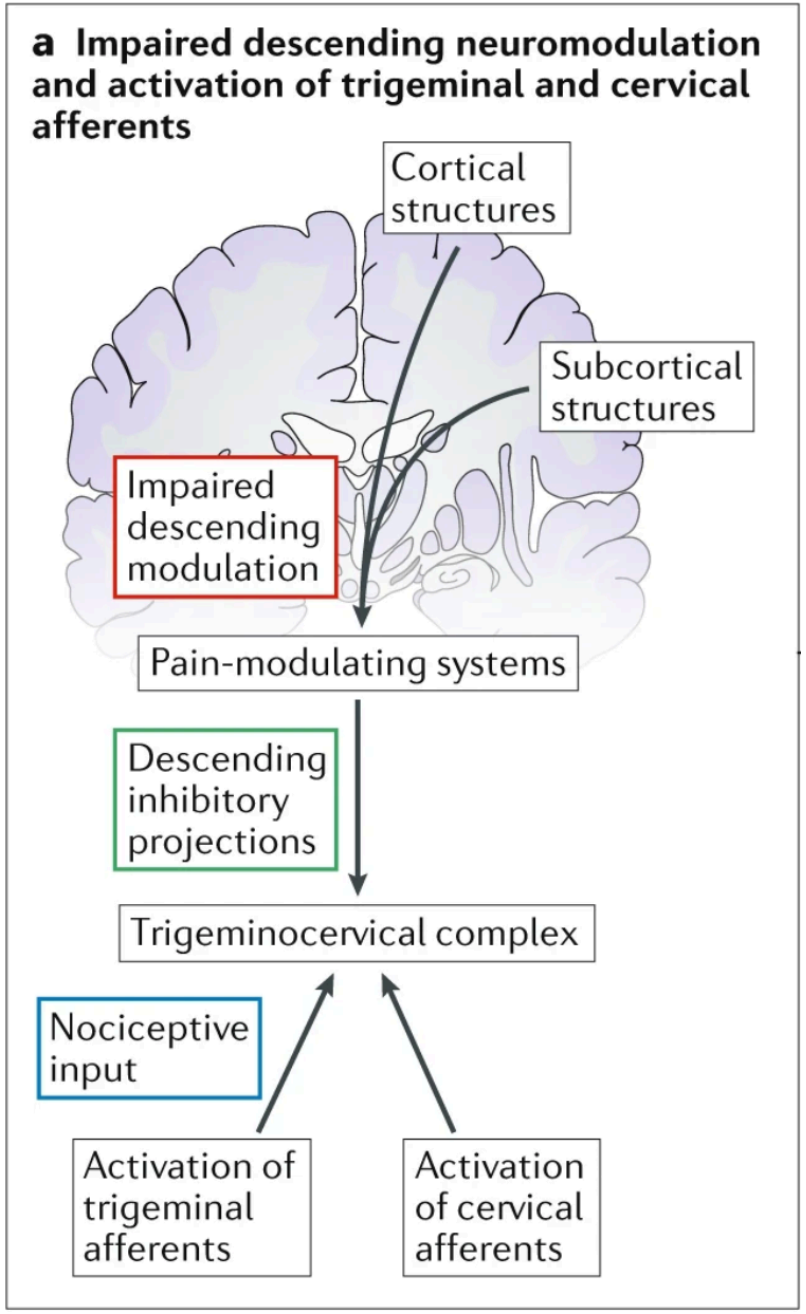
Impaired Descending Neuromodulation



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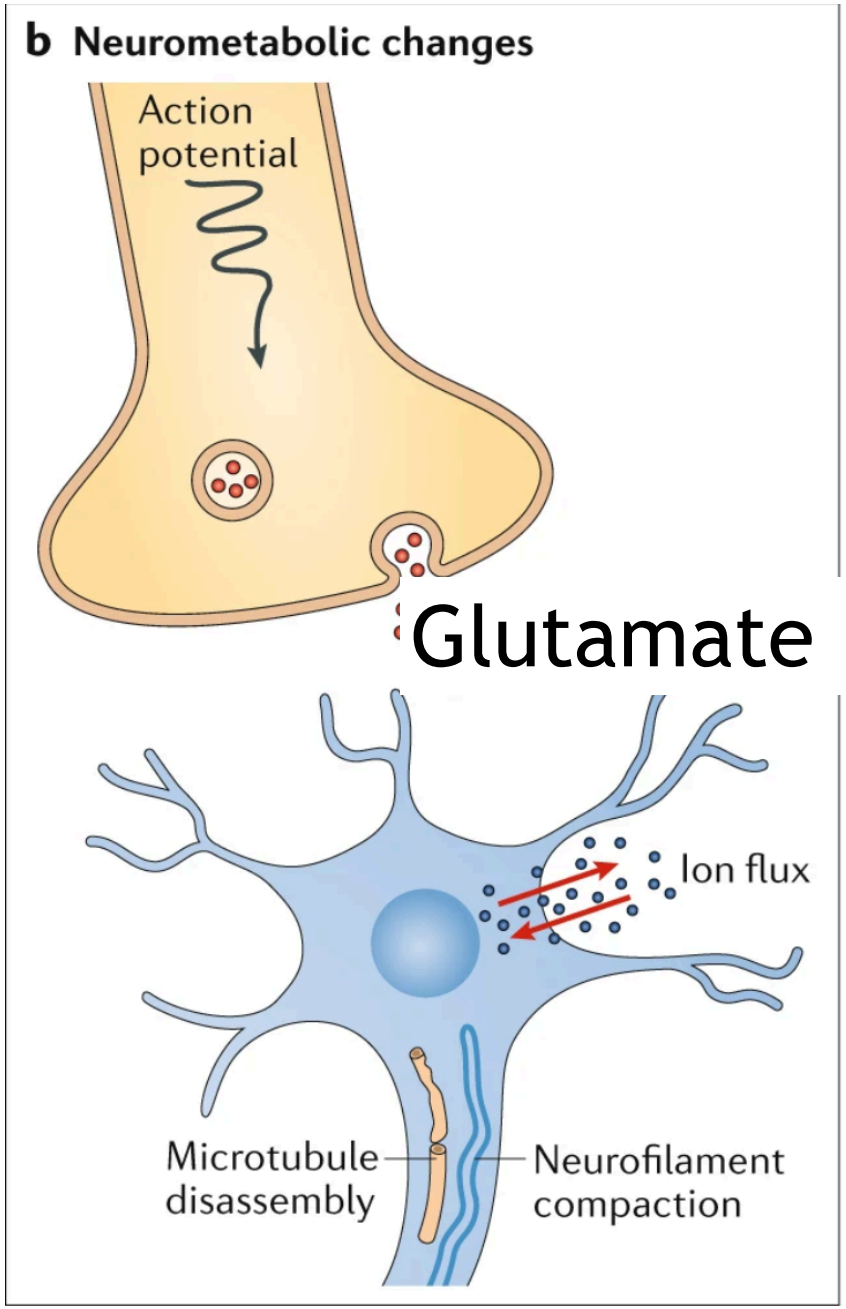


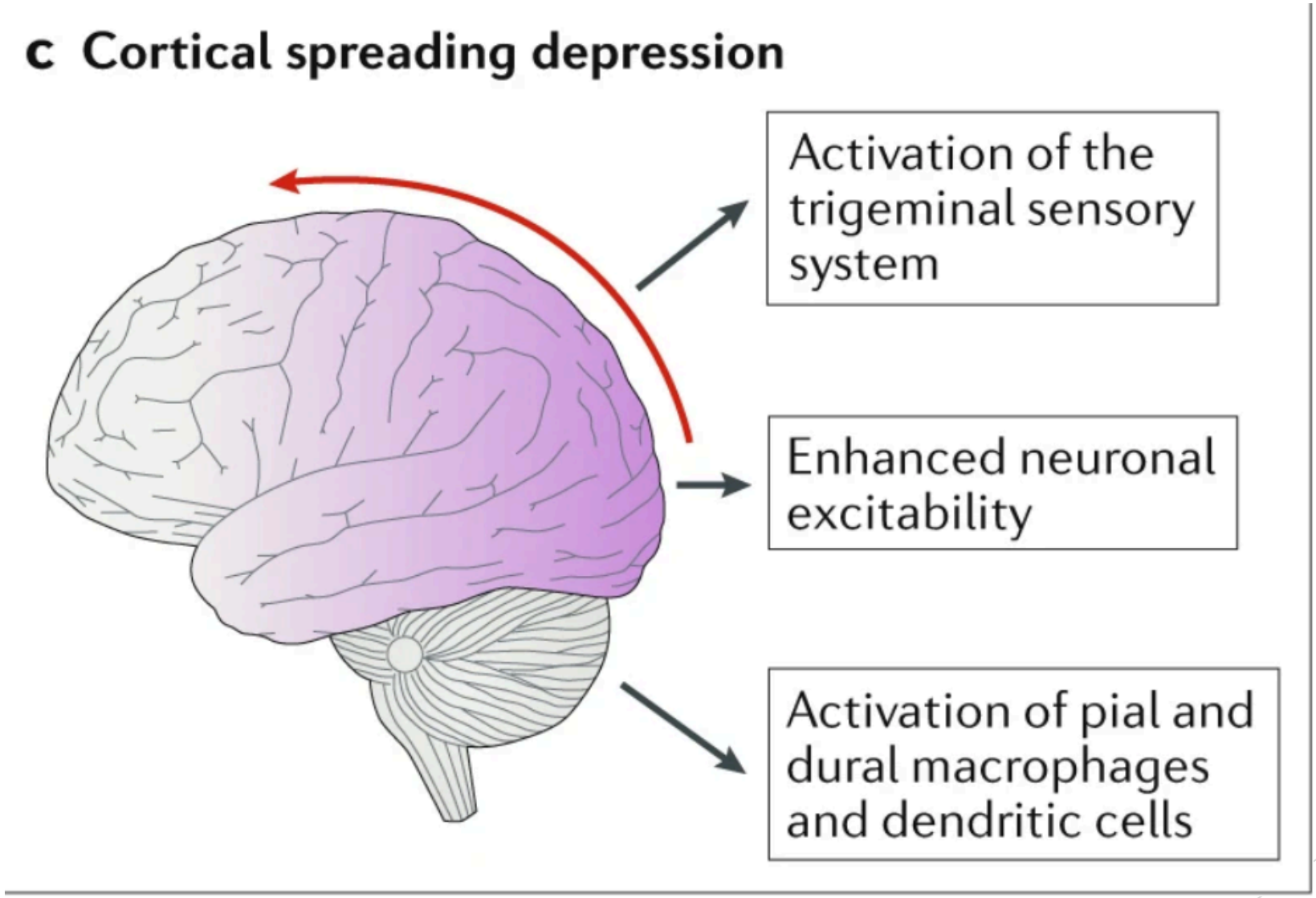
- release of glutamate
- excitatory neurotransmitters

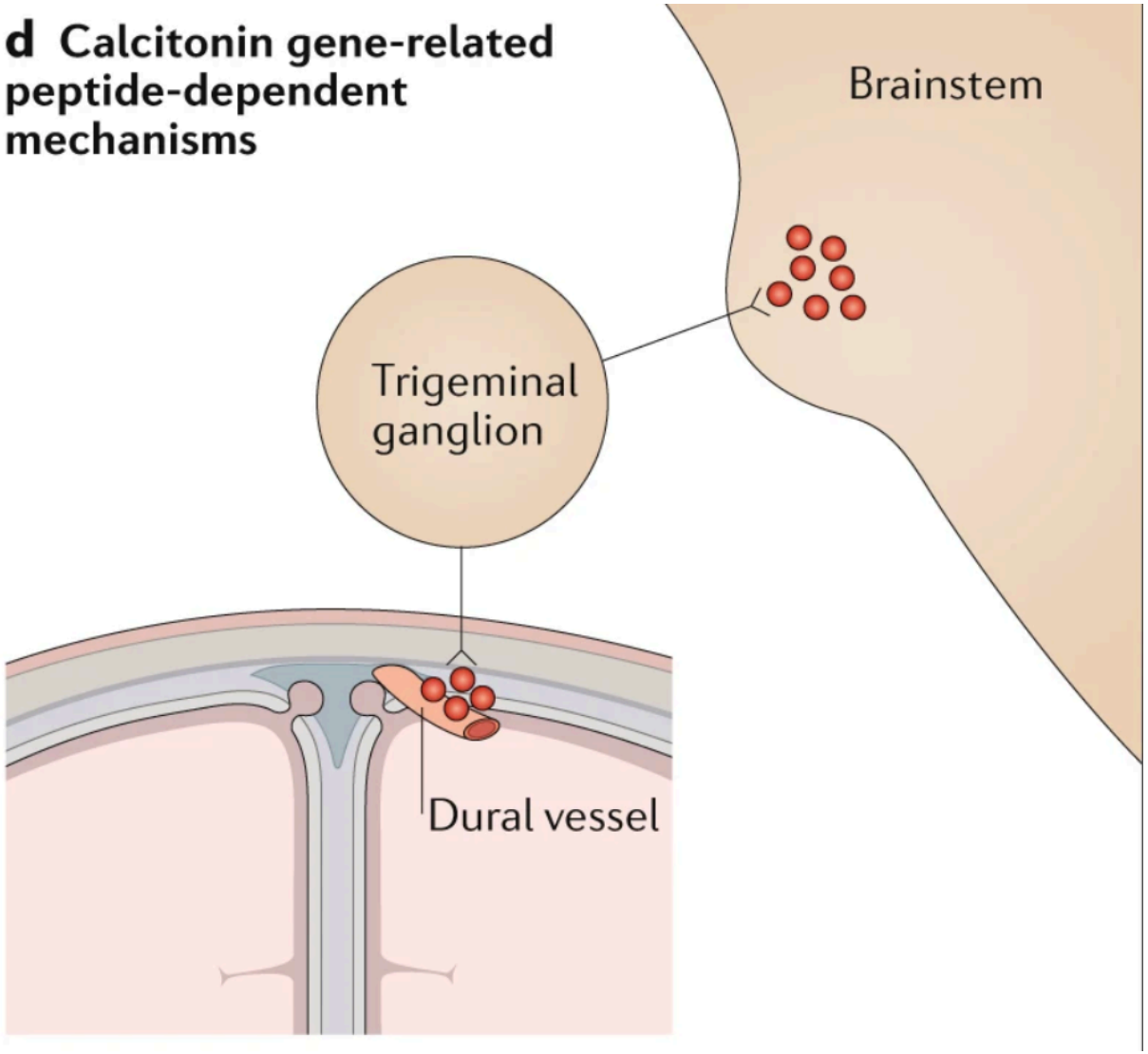
Also:

- neurofilament collapse
- microtubule disassembly

→ causing axon damage

Ashina, H., Porreca, F., Anderson, T., Amin, F.M., Ashina, M., Schytz, H.W. and Dodick, D.W., 2019. Post-traumatic headache: epidemiology and pathophysiological insights. *Nature Reviews Neurology*, *15*(10), pp.607


c Cortical spreading depression
Activation of the trigeminal sensory system
Enhanced neuronal excitability
Activation of pial and dural macrophages and dendritic cells



- Calcitonin gene-related peptide (CGRP) release-dependent mechanisms
- CGRP is also involved in migraine

Advances in the understanding of the CGRP pathway in migraine

Prof Dr Lars Edvinsson

Dept of Medicine

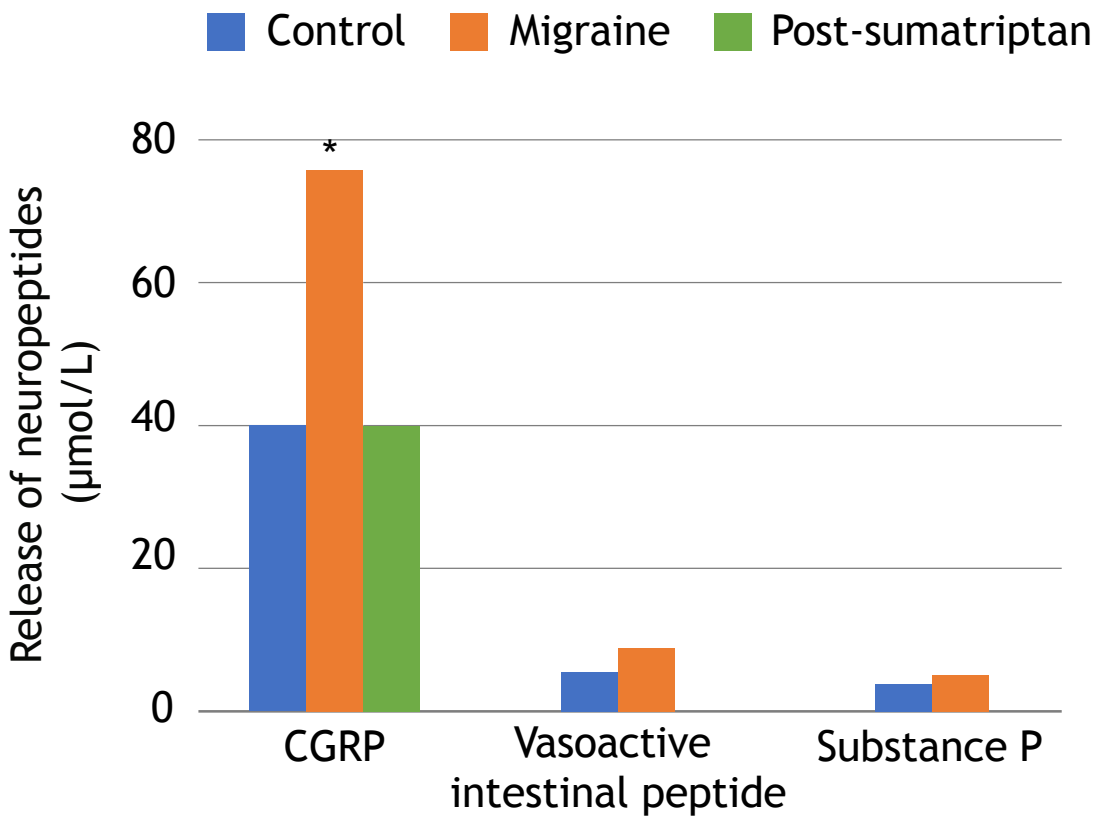
Lund University

Lund, Sweden

CGRP changes are associated with migraine

- CGRP levels doubles with migraine
- The CGRP levels normalize after a triptan, BOTOX or new anti-CGRP treatment with migraine

CGRP Changes Are Associated with Migraine



Clinical studies where Dr. Edvinson And his team collected blood from External jugular vein of patients Coming to ED with severe headache And migraine attack, on the left all Patients had CGRP increase in Parallel with headache

Patients were treated with triptans, and after headache disappeared, the CGRP levels normalized. So there was a clear association of CGRP and migraine attack. VIP and substance P were not as involved.

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Why is CGRP released during migraine?

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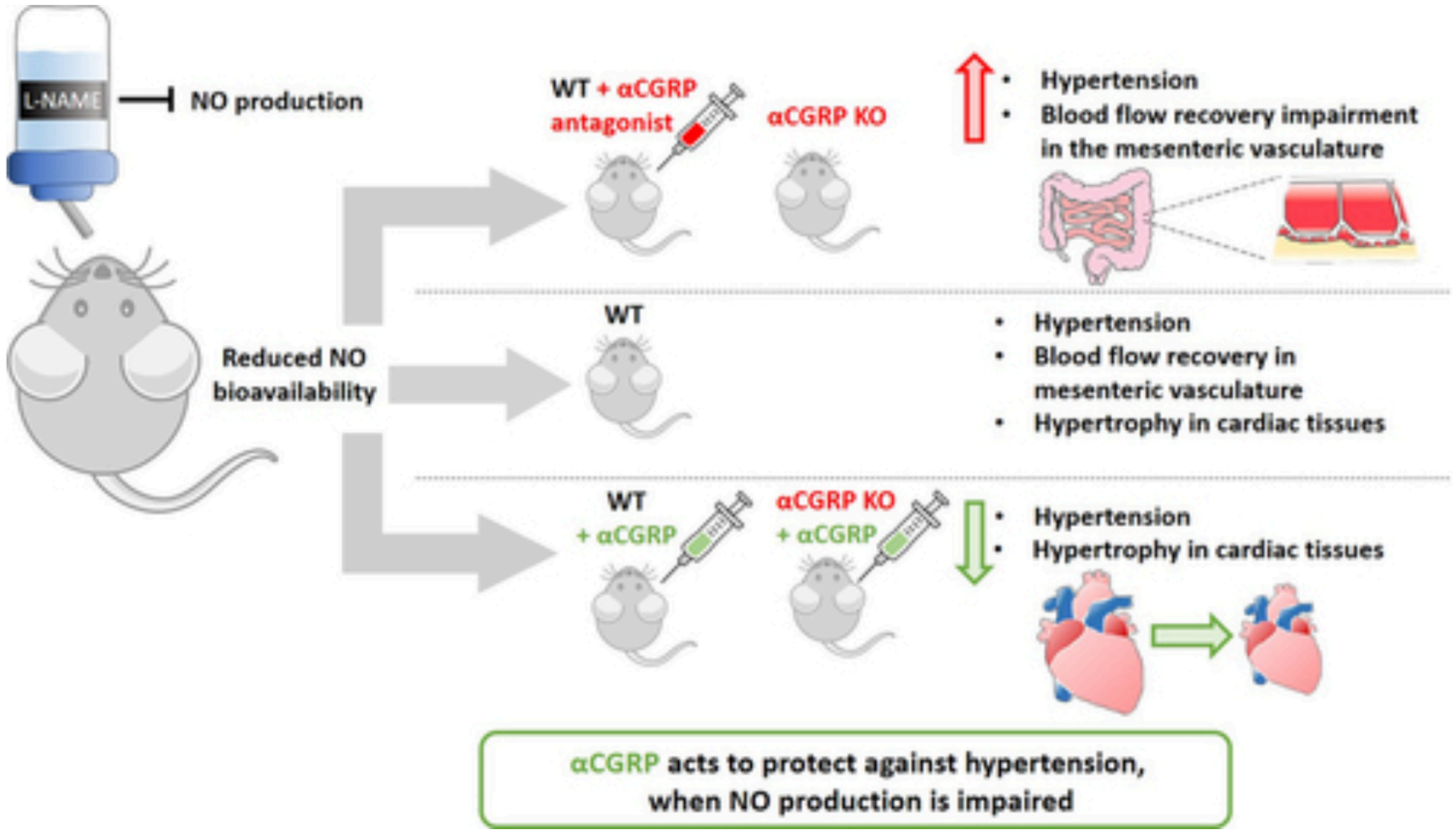
CGRP- Calcitonin gene-related peptide

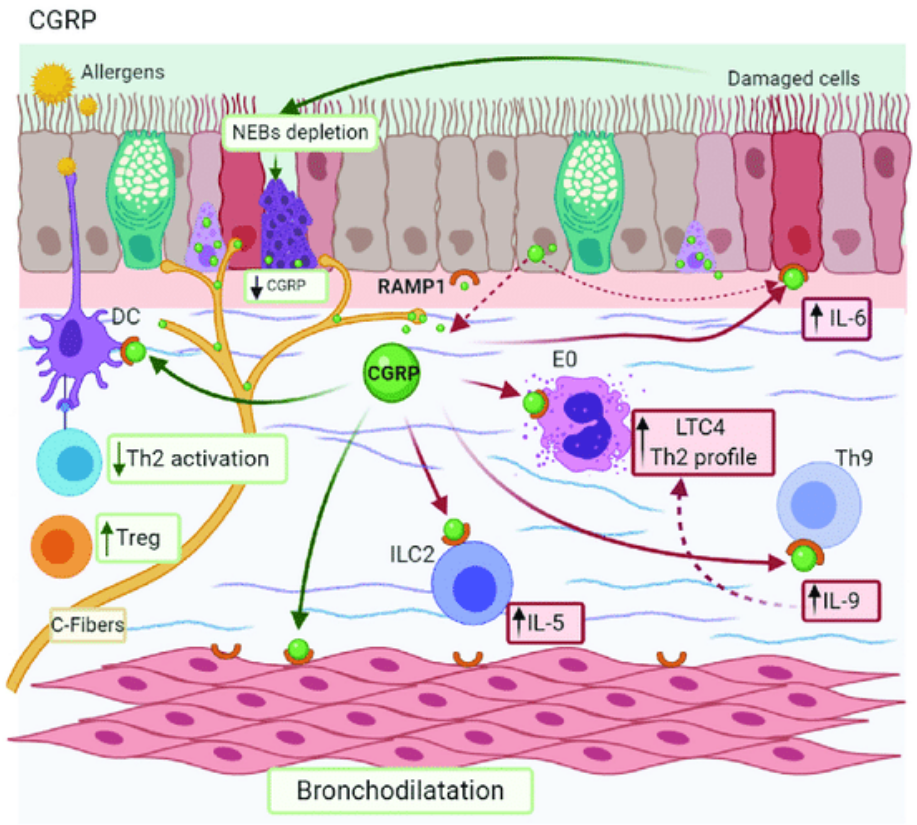
- Role in cardiovascular homeostasis
- Protects brain from hypertension
- Nociception
- In the heart, CGRP acts as a chronotrope by increasing heart rate
- Modulate the autonomic nervous system
- Plays a role in ingestion
- Has moderate effects on calcium homeostasis compared to its extensive actions in other areas, such as the autonomic nervous system

CGRP acts to protect against hypertension



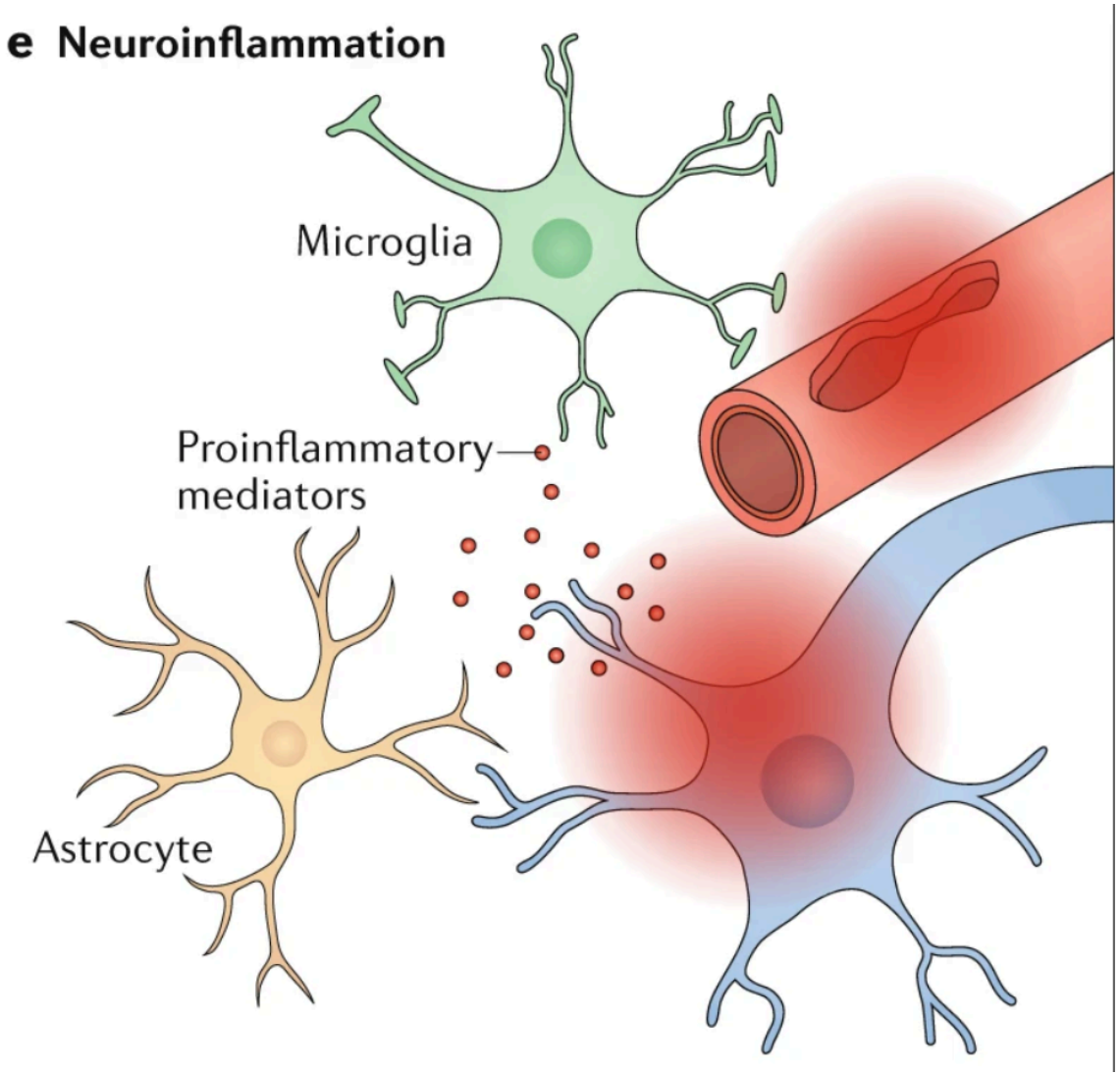
Argunhan, F., Thapa, D., Aubdool, A.A., Carlini, E., Arkless, K., Hendrikse, E.R., de Sousa Valente, J., Kodji, X., Barrett, B., Ricciardi, C.A. and Gnudi, L., 2021. Calcitonin gene-related peptide protects against cardiovascular dysfunction independently of nitric oxide in vivo. *Hypertension*, *77*(4), pp.1178-1190.

CGRP and asthma



- CGRP is synthesized and stored in C-fibers, neuroepithelial bodies (NEBs), and the epithelium.

Pavón-Romero, Gandhi F., Nancy Haydée Serrano-Pérez, Lizbeth García-Sánchez, Fernando Ramírez-Jiménez, and Luis M. Terán. "Neuroimmune pathophysiology in asthma." *Frontiers in cell and developmental biology* (2021): 1174.


e Neuroinflammation
Microglia
Proinflammatory mediators
Astrocyte

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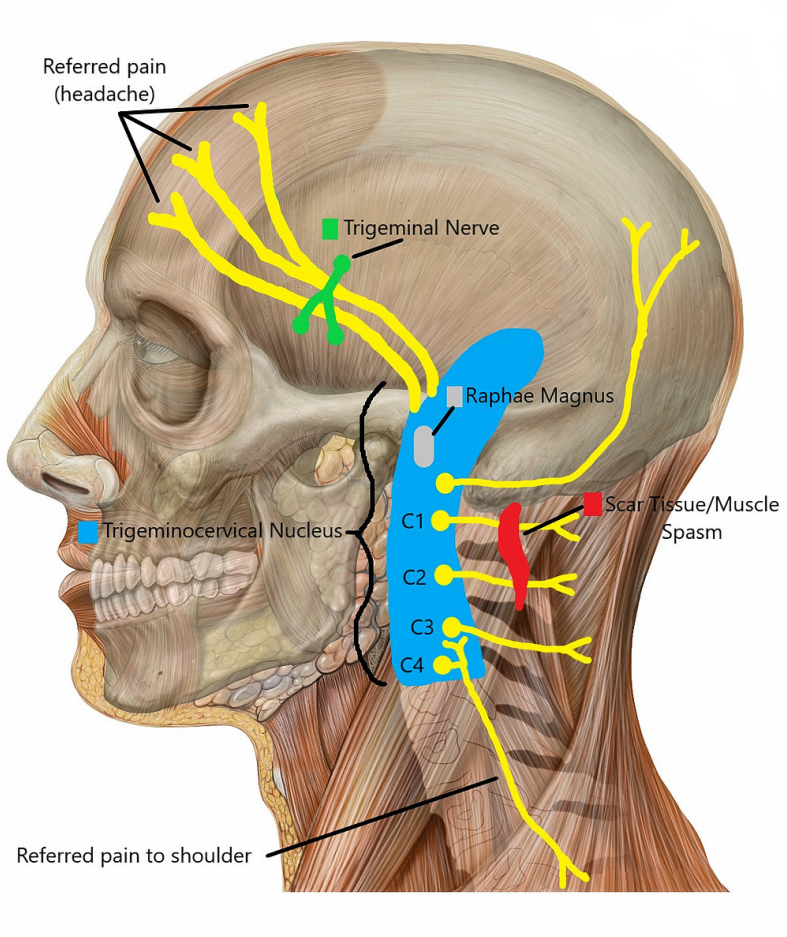
Trigemino-cervical complex



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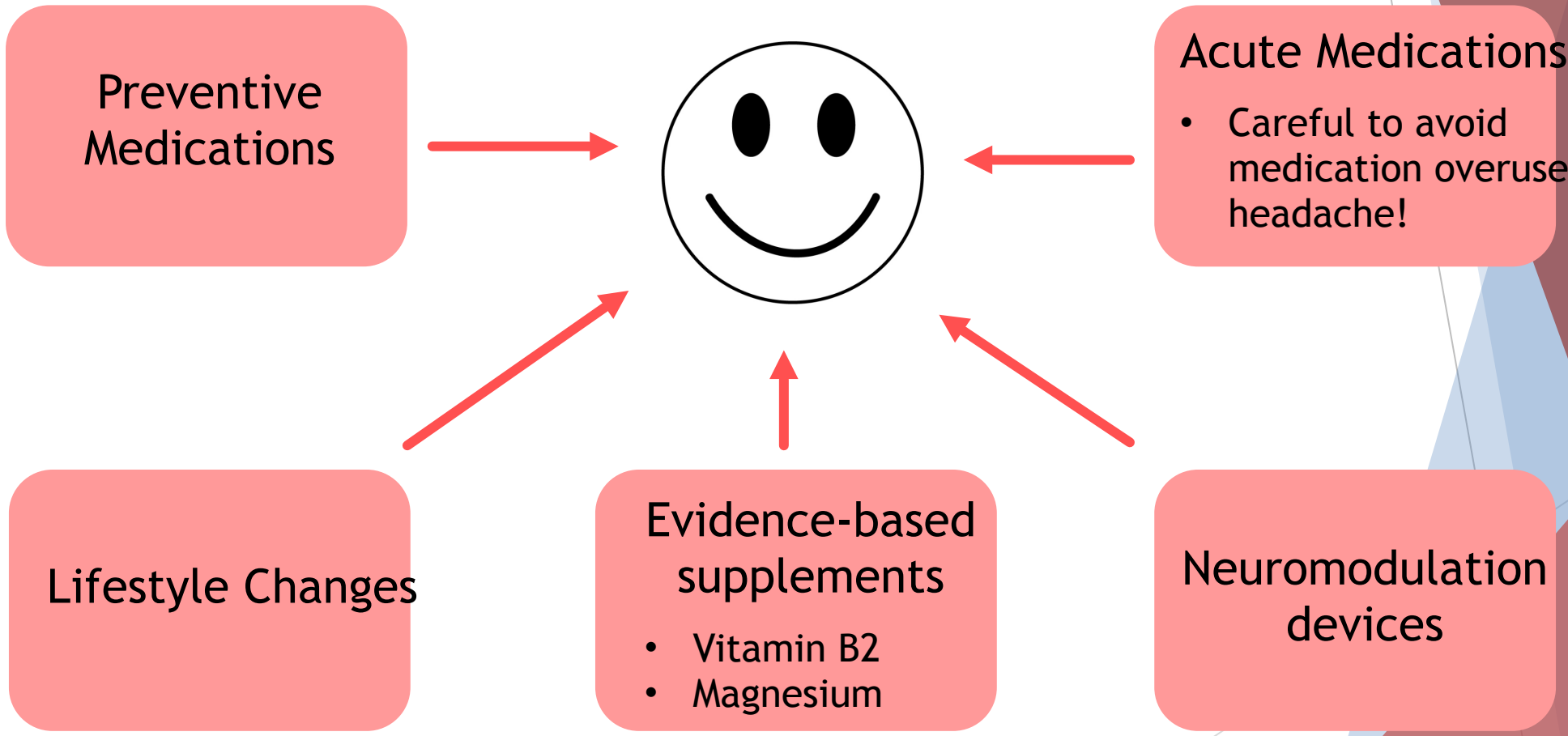
What exacerbates post-traumatic headaches?

What exacerbates post-traumatic headaches?

- Medication overuse headache
- Stress/ Mood
- Deconditioning
- Hormonal fluctuations in females
- Sleep disruption
- Lifestyle disruption
- PMH of migraine, mood disorders (depression/anxiety)
- FH of migraines and mood disorders

Treatments: Multimodal Approach



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Memantine (Namenda)

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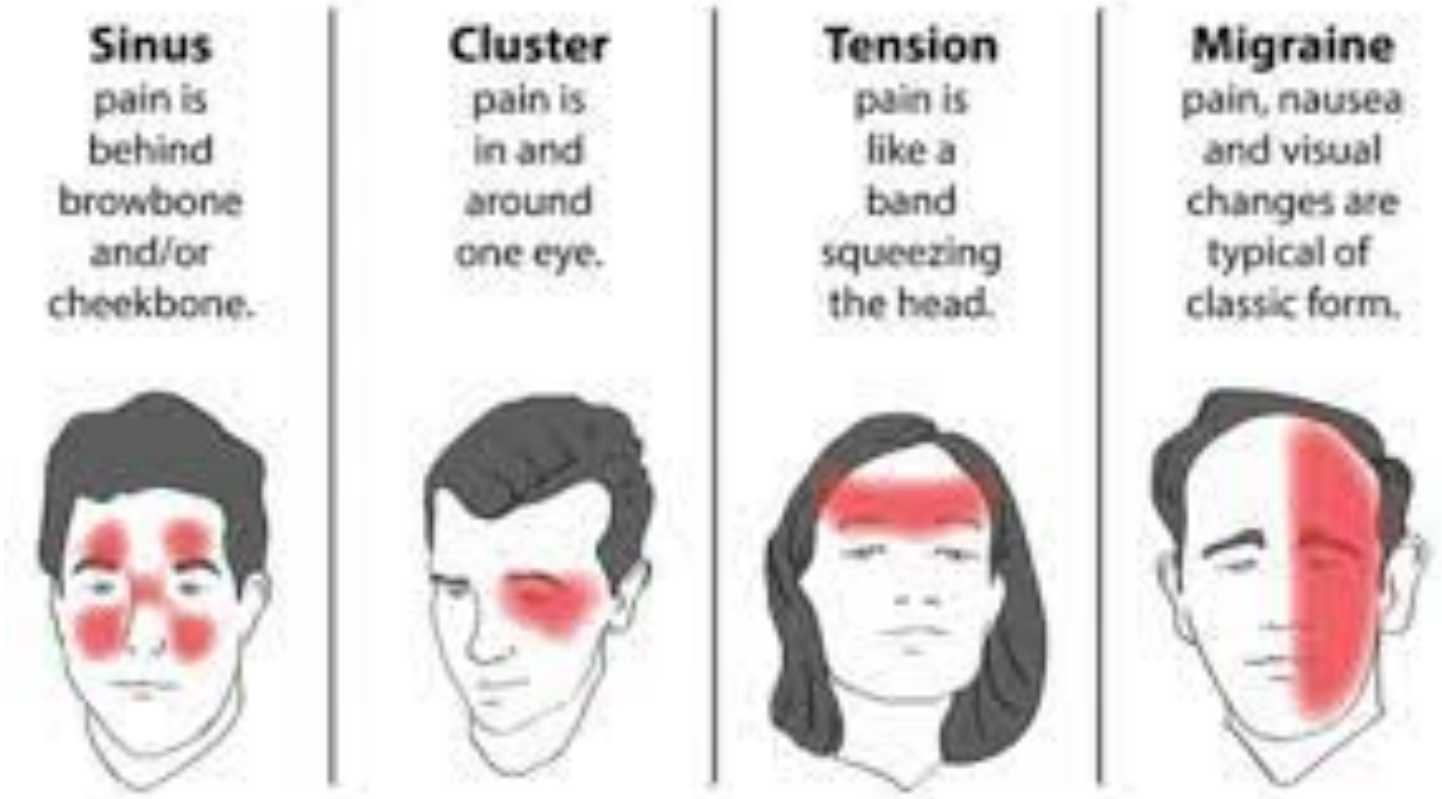
Memantine (Namenda)

- Inhibitor of the NMDA subtype of glutamate receptors
- Glutamate is the primary excitatory neurotransmitter in CNS
- Since an increase in glutamate occurs in the early response to trauma, early treatment may therefore be important.

Kamins, J. and Charles, A., 2018. Posttraumatic headache: basic mechanisms and therapeutic targets. *Headache: The Journal of Head and Face Pain*, *58*(6), pp.811-826.

If Migraine-like headache treat like Migraine



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Treatments: Preventive

- When to start a preventive medication?
  - Decision based upon:
    - Number of headache days per month
    - Disability (as based on MIDAS, MPFID, or HIT)

Received: 16 March 2021 | Accepted: 9 May 2021
DOI: 10.1111/head.14153

AHS CONSENSUS STATEMENT

The American Headache Society Consensus Statement: Update on integrating new migraine treatments into clinical practice

Jessica Ailani MD[1] | Rebecca C. Burch MD[2] | Matthew S. Robbins MD[3] | on behalf of the Board of Directors of the American Headache Society[4]

TABLE 4 Criteria for identifying patients for preventive treatment[8]

Prevention should be …	Headache days/ month	Degree of disability required[a]
Offered	6 or more	None
	4 or more	Some
	3 or more	Severe
Considered	4 or 5	None
	3	Some
	2	Severe

[a]As can be measured by the Migraine Disability Assessment Scale, Migraine Physical Function Impact Diary, or Headache Impact Test.

Treatments: Preventive

- What are the updated preventive medications?

TABLE 6 Medications with evidence of efficacy in migraine prevention[a,20,85]

Established efficacy[b]		Probably effective[c]	
Oral	**Parenteral**	**Oral**	**Parenteral**
Candesartan	Eptinezumab (IV)	Amitriptyline	OnabotulinumtoxinA + CGRP mAb[d,e]
Divalproex sodium	Erenumab (SQ)	Atenolol	
Frovatriptan[f]	Fremanezumab (SQ)	Lisinopril	
Metoprolol	Galcanezumab (SQ)	Memantine	
Propranolol	OnabotulinumtoxinA[d] (SQ)	Nadolol	
Timolol		Venlafaxine	
Topiramate			
Valproate sodium			

Nurtec (1 tab every other day)

Atogepant (1 tab daily)

Treatments: Preventive

- What are the updated preventive medications?

TABLE 6 Medications with evidence of efficacy in migraine prevention[a,20,85]

Established efficacy[b]		Probably effective[c]	
Oral	Parenteral	Oral	Parenteral
Candesartan	Eptinezumab (IV)	Amitriptyline	OnabotulinumtoxinA + CGRP mAb[d,e]
Divalproex sodium	Erenumab (SQ)	Atenolol	
Frovatriptan[f]	Fremanezumab (SQ)	Lisinopril	
Metoprolol	Galcanezumab (SQ)	Memantine	
Propranolol	OnabotulinumtoxinA[d] (SQ)	Nadolol	
Timolol		Venlafaxine	
Topiramate			
Valproate sodium			

Nurtec (1 tab every other day)

Atogepant (1 tab daily)

CGRP

Anti-CGRP monoclonal

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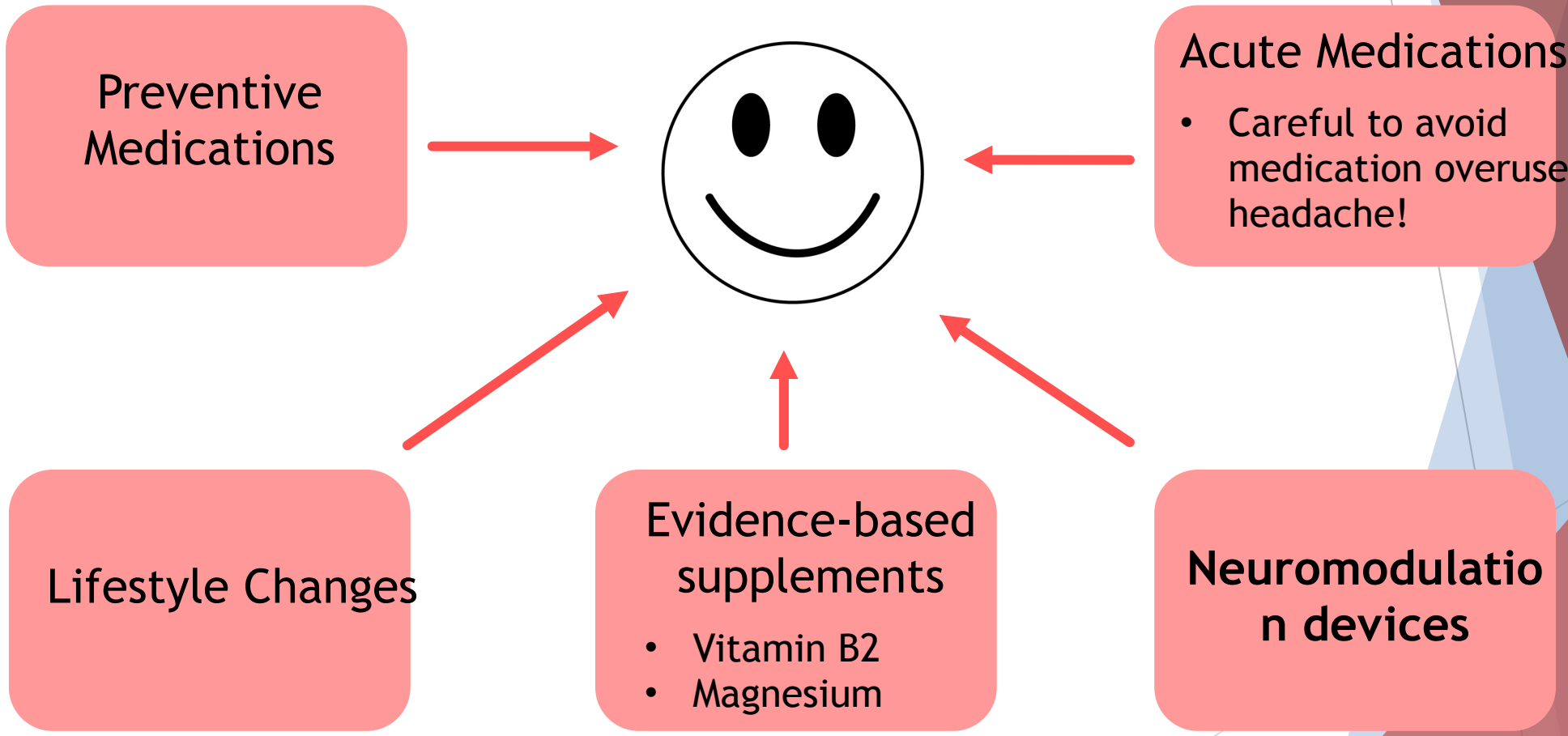
Treatments: Multimodal Approach



Treatments: Neuromodulation

- **Use as stand-alone or add-on treatment for migraine**
  - Stimulate the nervous system centrally or peripherally with an electric current
  - FDA-approved for migraine

CEFALY

- Age ≥ 18
- Acute and preventive treatment
- Mechanism:
  - Stimulates trigeminal nerve

GammaCore

- Age ≥ 12
- Acute treatment
- Mechanism:
  - Stimulates vagus nerve

Nerivio

- Age ≥ 12
- Acute treatment
- Mechanism:
  - Remote electrical neuromodulation affecting descending inhibitory pain pathways brainster

Relivion

- Age ≥ 18
- Acute treatment
- Mechanism:
  - Stimulates trigeminal and occipital nerves

Treatments: Neuromodulation

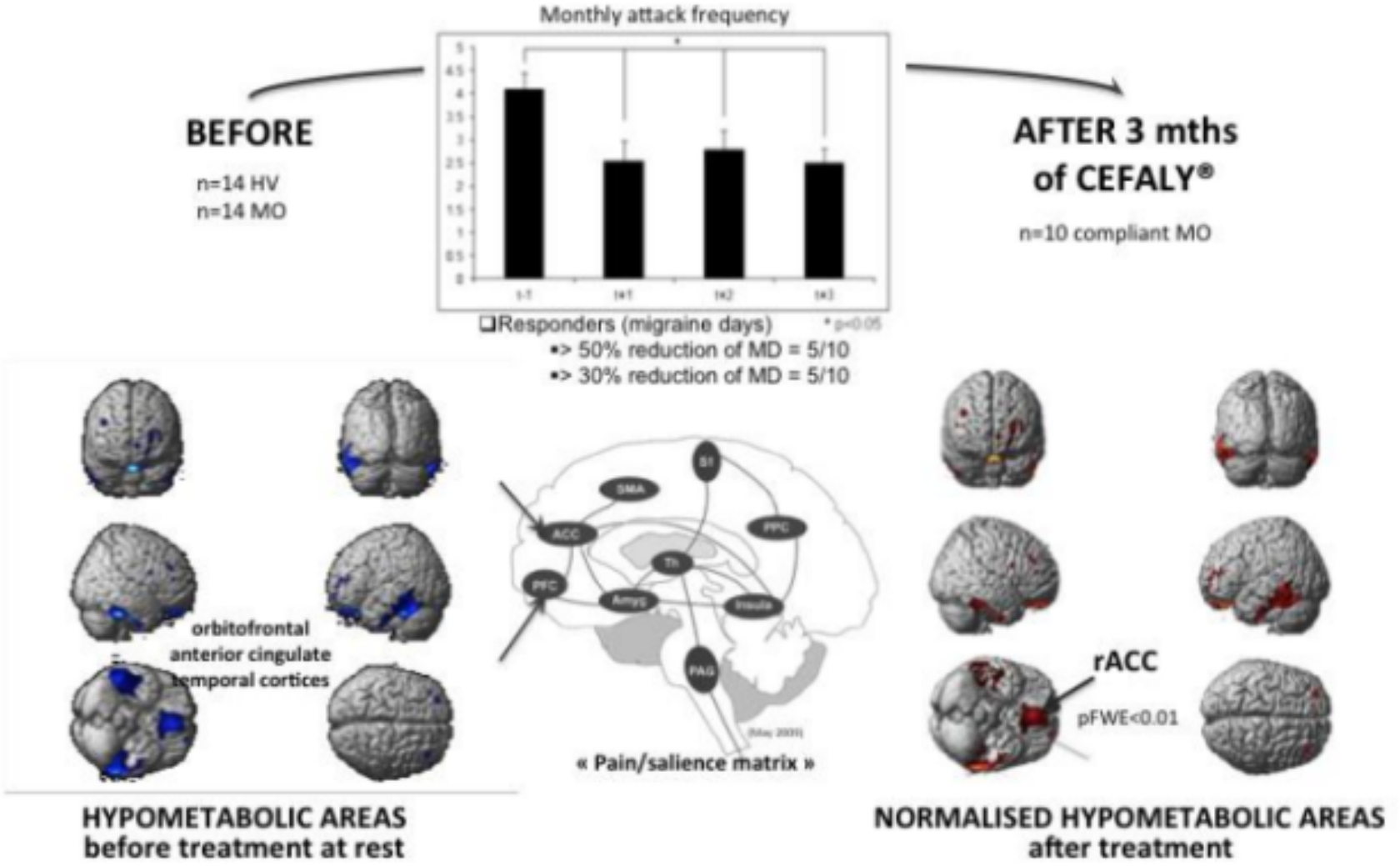
Internal Medicine Review Migraine treatment with external trigeminal nerve stimulation March 2017

Migraine treatment with external trigeminal nerve stimulation: current knowledge on mechanisms

Jean Schoenen MD, PhD

- **CEFALY**
  - Normalizes brain function in medal frontal cortical areas controlling affective and cognitive dimensions of pain

CEFALY



Treatments: Neuromodulation

Cephalalgia
International Headache Society
Original Article

Acute migraine therapy with external trigeminal neurostimulation (ACME): A randomized controlled trial

Cephalalgia
2019, Vol. 39(1) 3–14

DOI: 10.1177/0333102418811573
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SAGE

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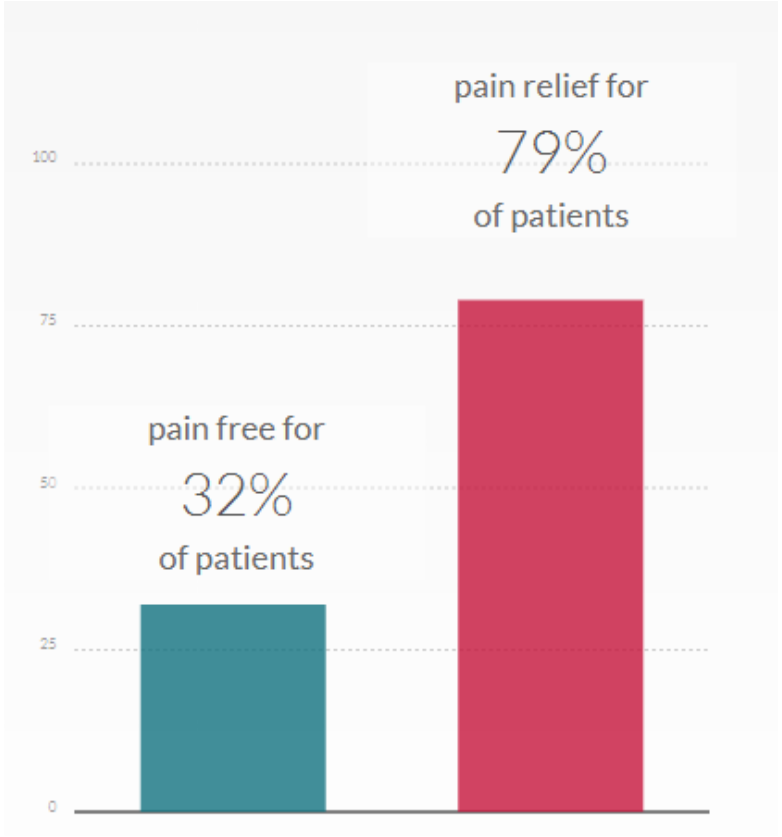
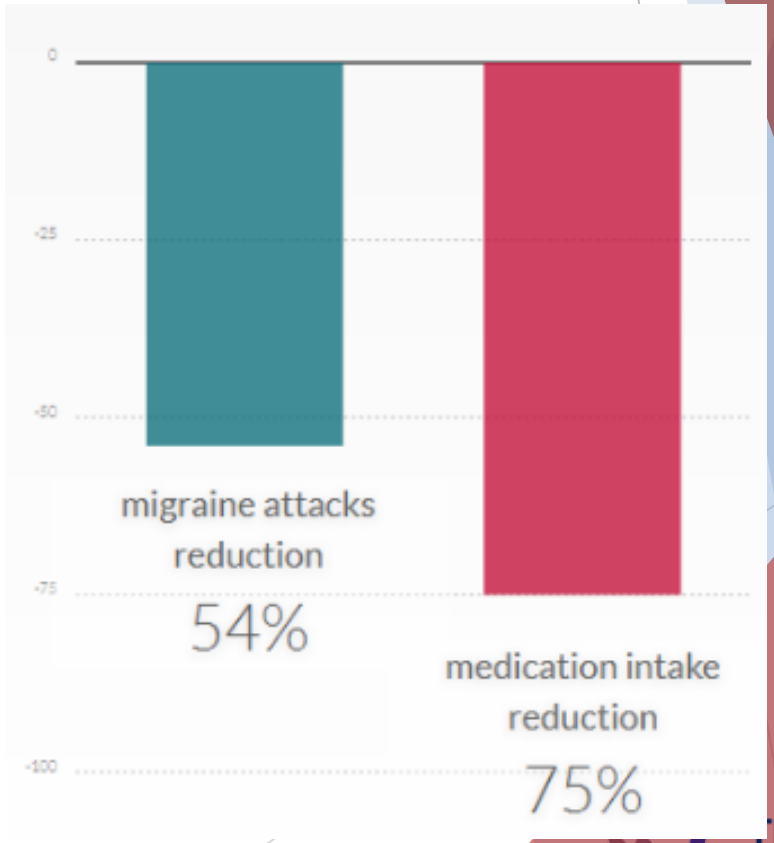
- **CEFALY**
  - Normalizes brain function in medal frontal cortical areas controlling affective and cognitive dimensions of pain

CEFALY



Acute Management



Preventive Management

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Treatments: Neuromodulation

- **Relivion**
  - Combined trigeminal nerve -occipital nerve stimulator
  - ? More effective at decreasing holocephalic pain
    - Episodic and chronic migraine subjects ($N = 55$)
      - Randomized to active ($n = 27$) or sham ($n = 28$) treatment

Relivion

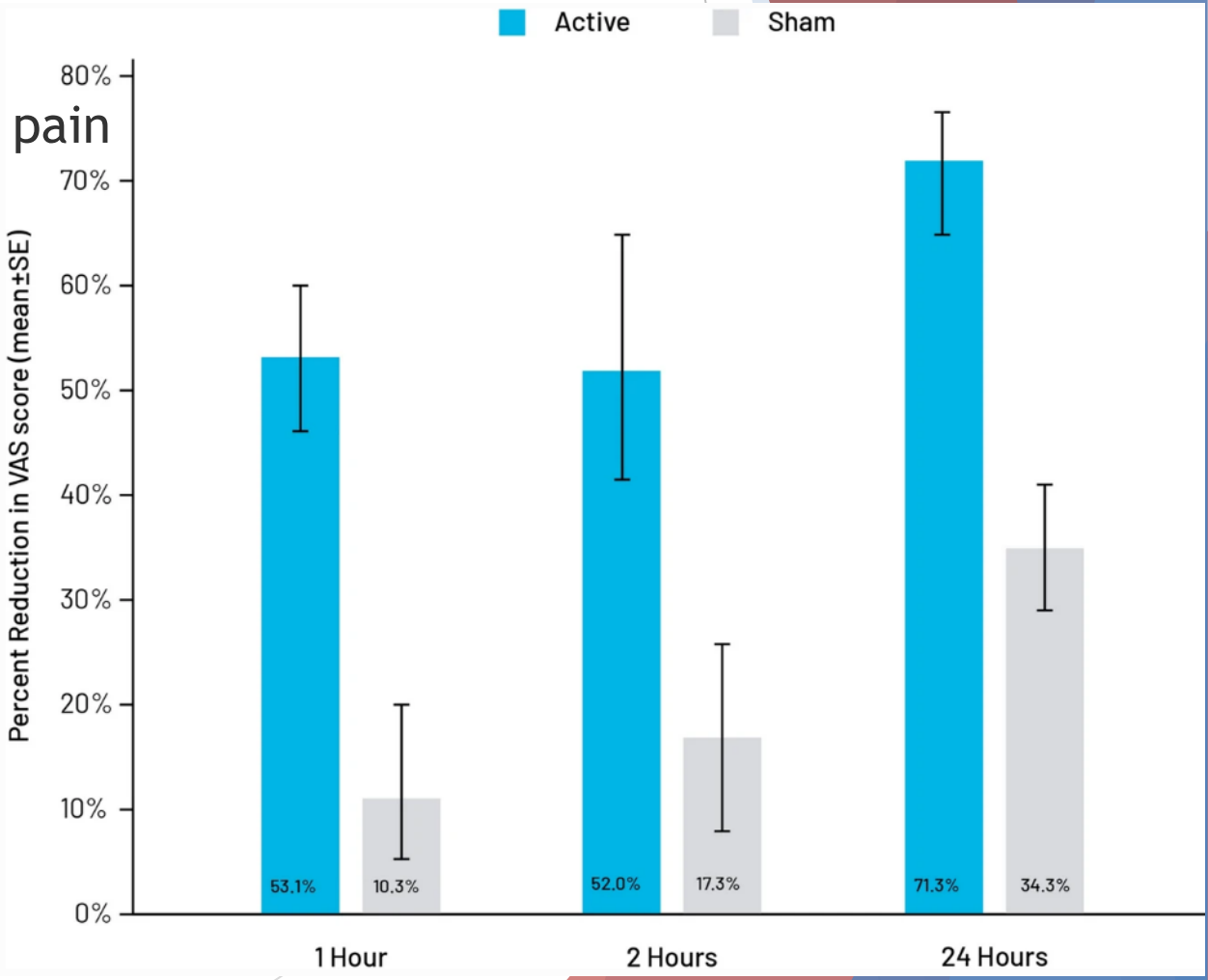
- Results
  - Active stimulation was more effective than sham stimulation at 1 hour (53% vs 10%), 2 hours (52% vs 17%) and 24 hours (71 vs 34%)
  - Comparable to triptans

Original Research | Open Access | Published: 04 June 2022

External Concurrent Occipital and Trigeminal Neurostimulation Relieves Migraine Headache: A Prospective, Randomized, Double-Blind, Sham-Controlled Trial

Oved Daniel, Stewart J. Tepper, Lisa Deutsch & Roni Sharon

Pain and Therapy **11**, 907–922 (2022)



Treatments: Neuromodulation

Which patients should use these devices?

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Treatments:Neuromodulation

Which patients should use these devices?

- Patients who are looking to **bolster their acute and preventive approaches** for treatment of migraine
- Patients **at risk for medication overuse** headache (due to headache or other body pain)
- Patients who prefer not to use medications/injections
- Patients with **medical conditions** preventing them from taking traditional medications for headache
- Patients who have **limited access/interest in acute care services** for severe headache

Treatments: Gepants

What are the side effects and contraindications?

Side effects:

- Intolerance to feeling of CEFALY on the forehead: 1.25%
- Fatigue during / after the session: 0.65%
- Headache after one session: 0.52%
- Irritation of the skin on the forehead: 0.22%

Contraindication

s:

- Implanted metallic / electronic device in head
- Pain of unknown origin
- A cardiac pacemaker or implanted or wearable defibrillator (may cause interference with pacing, electric shock, or death)

Treatments: Neuromodulation

Often not covered by insurance

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Adam

- Adam is coming to the medical center with chief complaint of headaches that worsened 3 days after he was hit by a car while cycling to UW
- He fell off the bicycle and was able to get back on the bike
- He had no LOC
- He has severe one-sided headache that is constant with nausea, light and sound sensitivity, and he has other medical issues such as disrupted sleep, dizziness, and others
- He is currently unable to bike due to fear of having another accident
- **He has been taking daily acetaminophen (Tylenol) and seen multiple other providers; nothing is helping his symptoms**

Adam

- Adam has been taking daily acetaminophen (Tylenol) and nothing is helping his symptoms

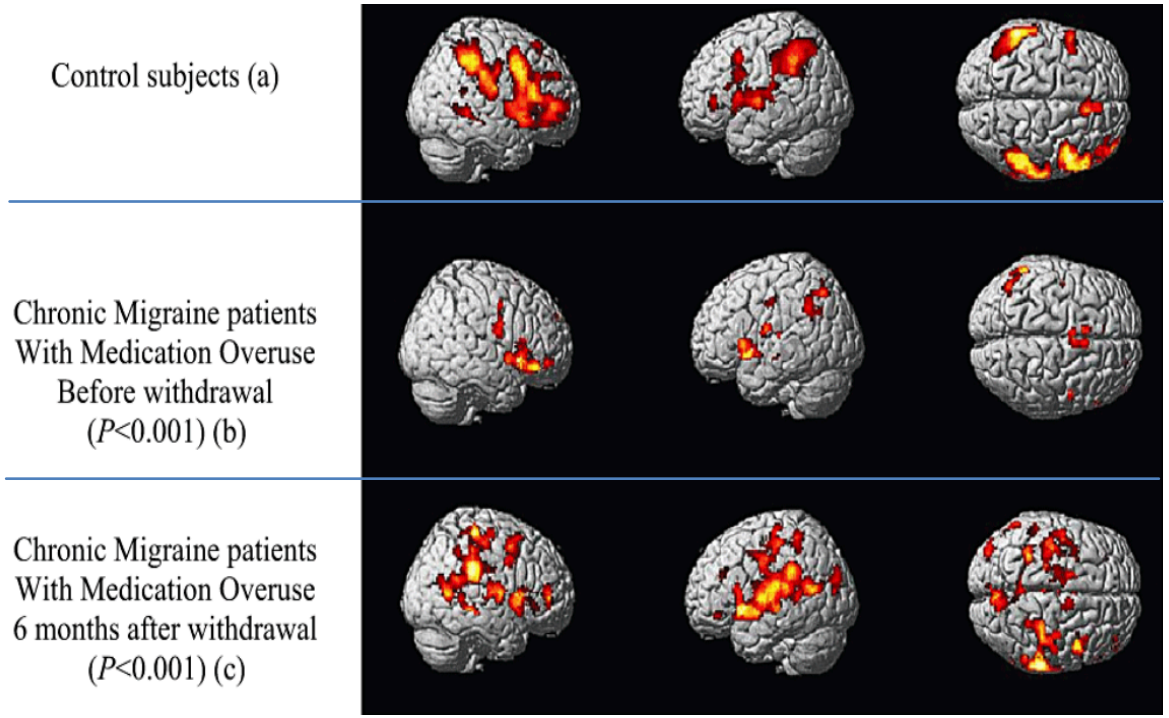
Adam

- Adam has been taking daily acetaminophen (Tylenol) and nothing is helping his symptoms
- What other diagnosis is important to diagnose and treat?

Medication Overuse Headache

- Develops in individuals with pre-existing primary headaches (e.g., migraine or tension type headache)
- Headache increases in frequency to ≥ 15 days per month
- Caused by acute medication use on ≥ 10 or 15 days/month (depending on medication) for > 3 months

How does Medication Overuse affect Brain



Grazzi, Licia, et al. "Chronic migraine with medication overuse pre-post withdrawal of symptomatic medication: clinical results and fMRI correlations." *Headache: The Journal of Head and Face Pain* 50.6 (2010): 998-1004.

- **Functional brain imaging**
  - **Hypometabolism** in several brain structures
  - Withdrawal from overused medications shows **reversible changes** to a large extent in these areas

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Medication overuse headache

How much acute medication is safe?

- **The amount of acute medication "allowed" varies from person to person**
- **International Classification of Headache Disorders**
  - Non-opiates: 15+ days/month in combination
  - Opiates +/- non-opiates: 10+ days/month
  - Triptans: 10+ days/month
- **However, as few as 6-8 days per month can cause MOH in patients with anxiety/stressors**

Supplements

Acute

Acute and preventive

Preventive

Preventive

Preventive

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Neuromodulation

Cefaly

Nerivio

gammaCore

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Use Multimodal approach

Sleep

Exercise

Biofeeedback/relaxation

Anti-Inflammatory Nutrition

Summary

- Use a multi-modal approach, incorporate medications for PTH, evidence-based supplements, neuromodulation devices, and lifestyle changes

- Neuromodulation devices
  - Not covered typically covered by insurance
  - Safe, non-medication approaches that are as effective as medication

References

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Questions?

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