Differentiating Vestibular Migraine from other Vestibular Disorders

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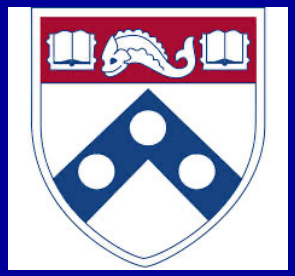
Assistant Professor Otolaryngology

Charleston Vestibular Update 2023



Wilmington, Delaware, USA

Goals

- Become familiar with migraine in its “classic” and atypical forms
- Review the currently accepted pathophysiologic model of migraine
- Highlight comparisons of VM with other vestibular disorders
- Establish familiarity and comfort with sound migraine therapies

Outline

- Overview of common vestibular disorders
- Migraine Epidemiology and Pathophysiology
- Mechanisms of vestibular symptoms in VM
- Treatment of VM
- Q&A

Diagnosis and Management of Balance Disorders

There can be few physicians so dedicated to their art that they do not experience a slight decline in spirits on learning the patients complaint is of dizziness.

WB Matthews

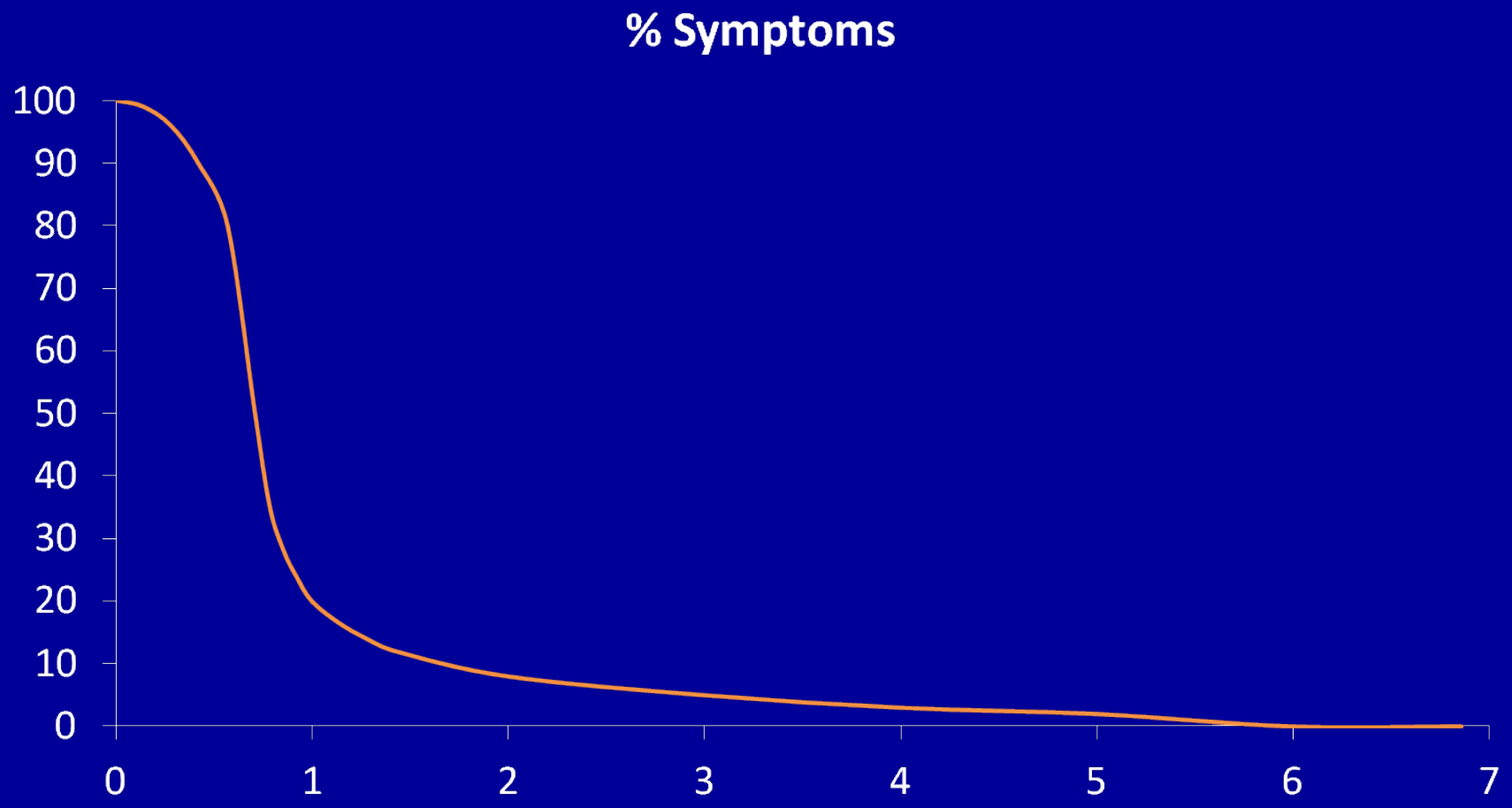
Neurologist

Pattern Recognition

Time course of vestibular disorders

Time Course of Symptoms

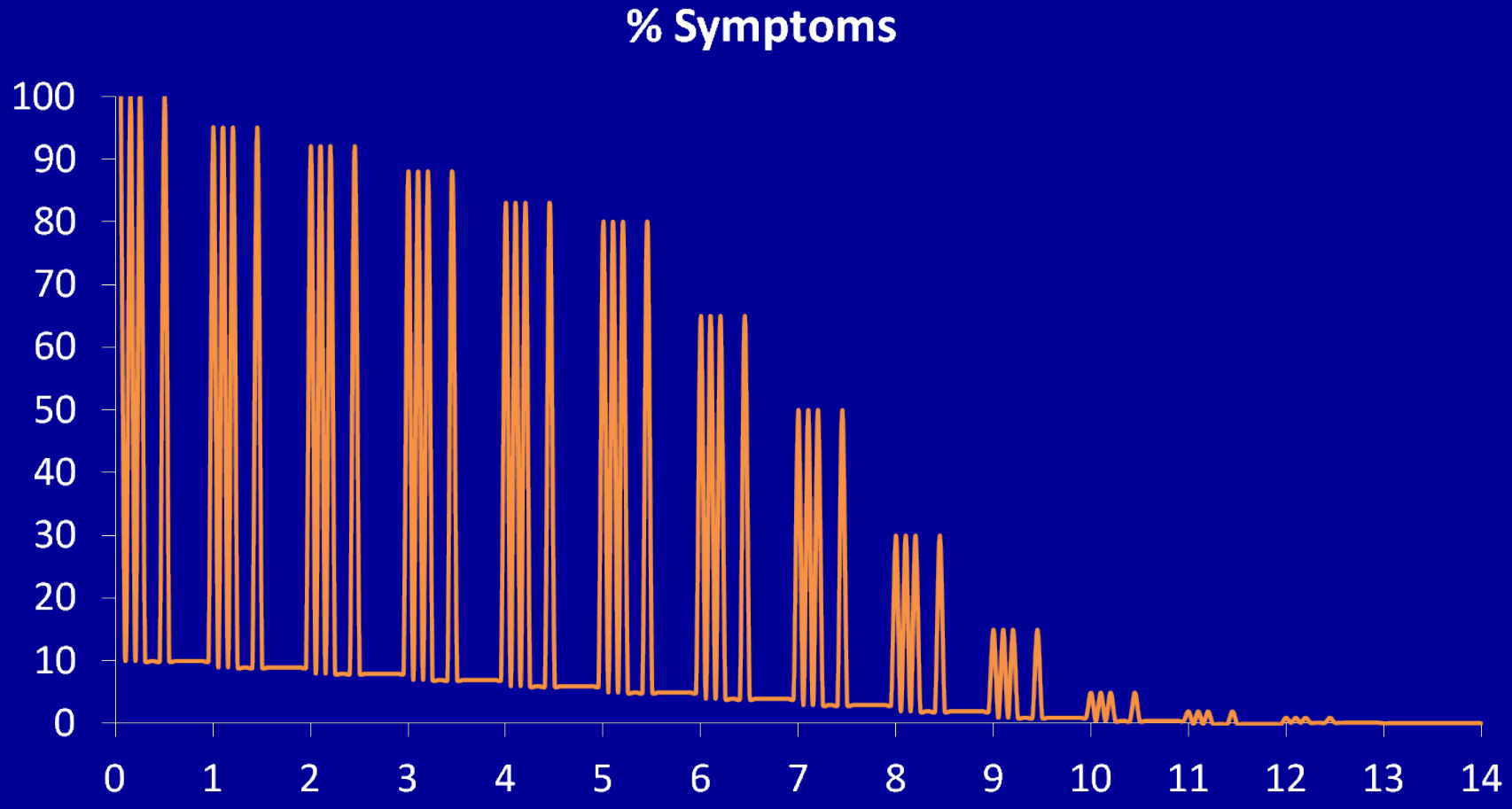
Vestibular Neuritis



Weeks from onset of symptoms

Time Course of Symptoms

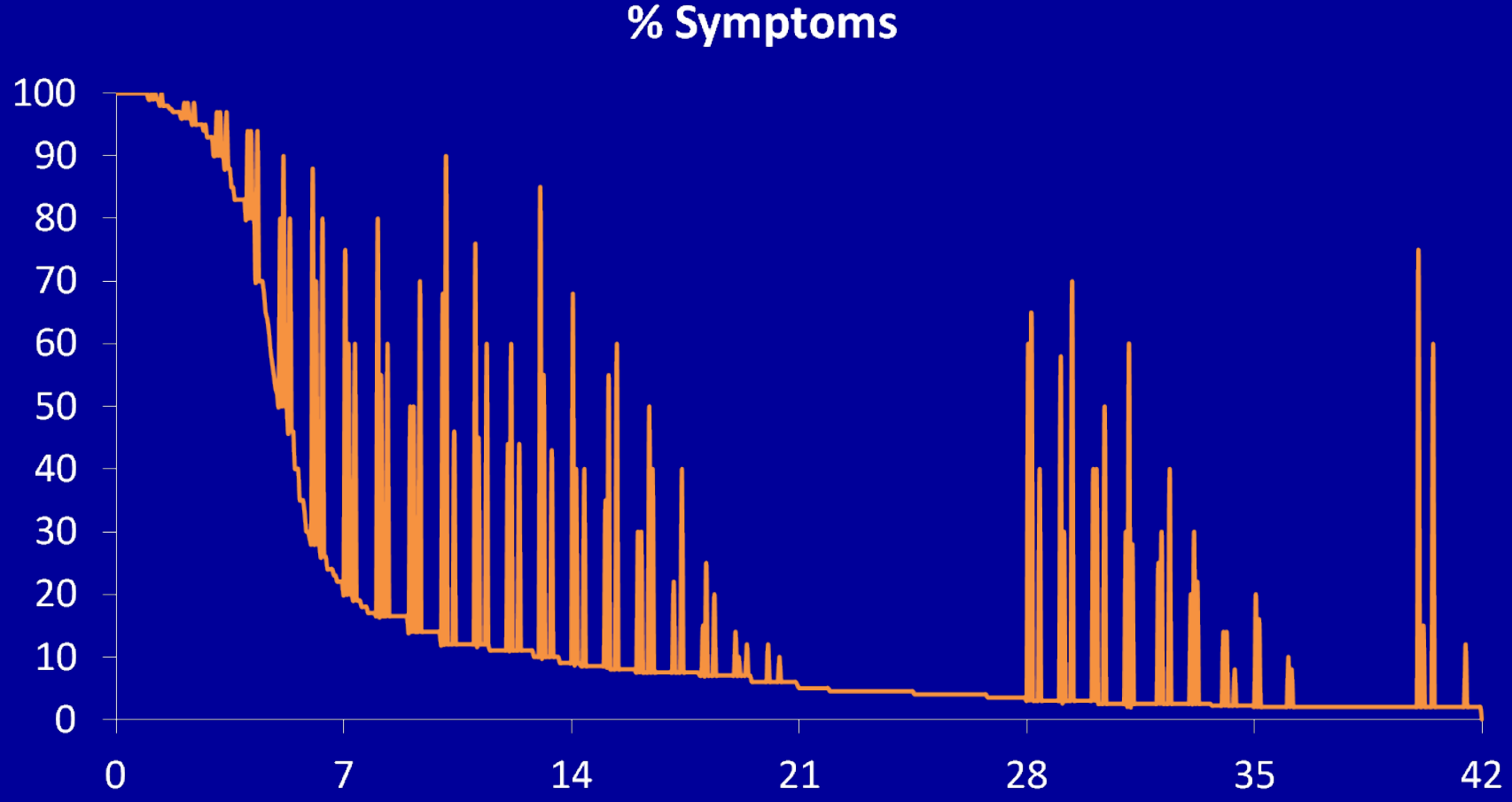
BPPV



Days from onset of symptoms

Time Course of Symptoms

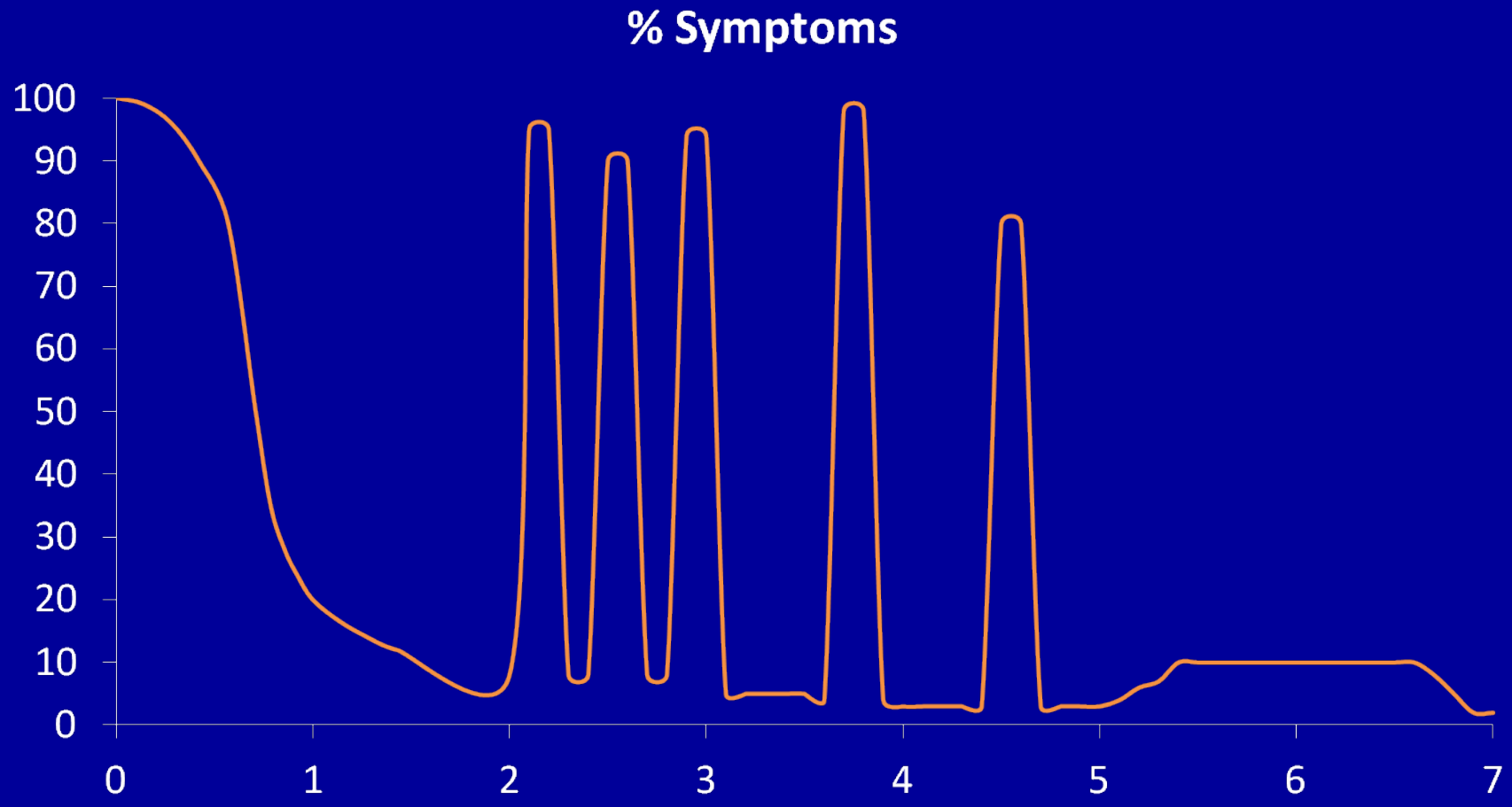
Vestibular Neuronitis with secondary BPPV



Days from onset of symptoms

Time Course of Symptoms

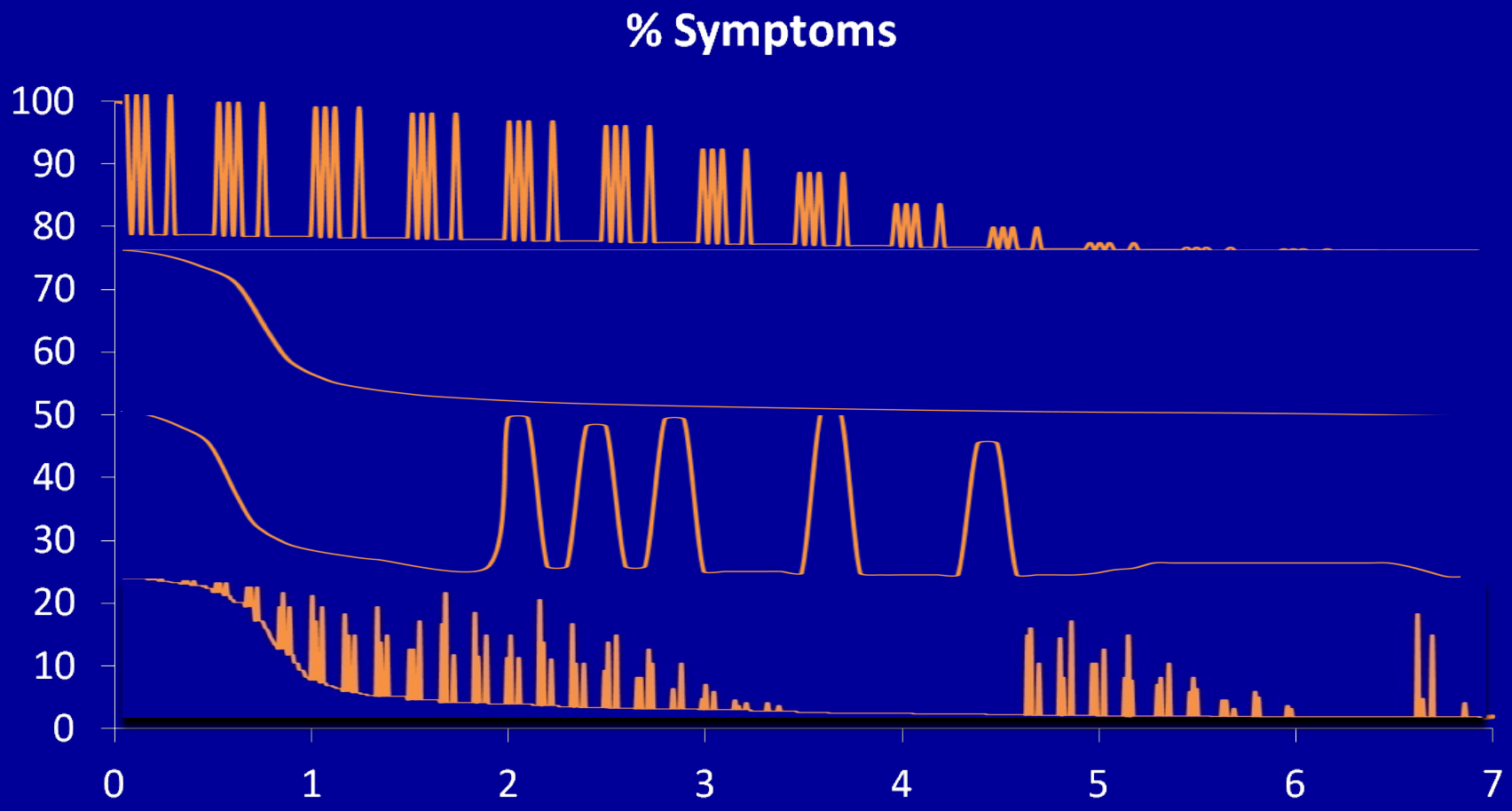
Meniere's Disease Symptoms



Weeks from onset of symptoms

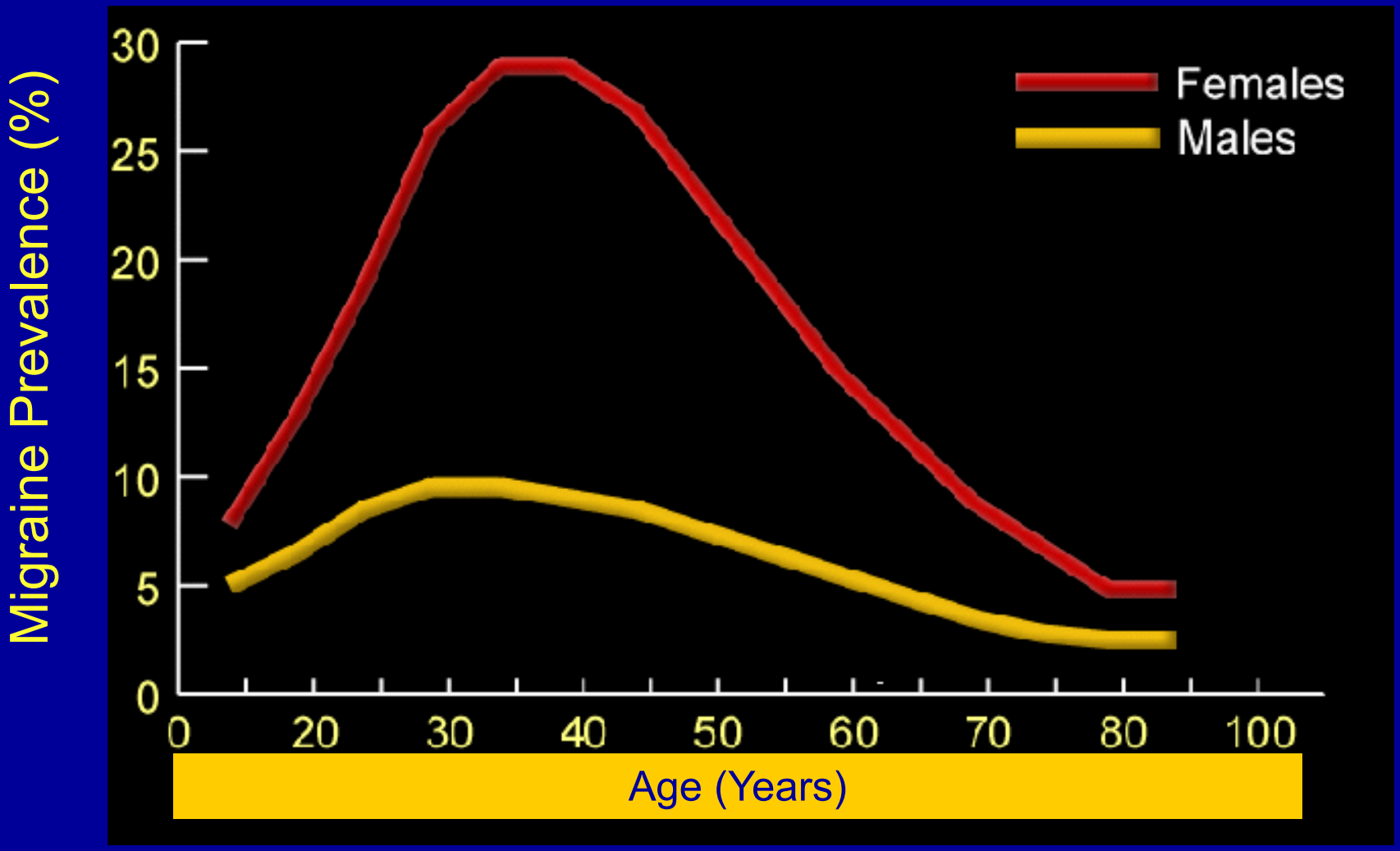
Time Course of Symptoms

Vestibular Migraine Symptoms



Migraine Epidemiology and Pathophysiology

AGE- AND GENDER-SPECIFIC PREVALENCE OF MIGRAINE



Lipton RB et al. *Headache*. 2001.

Undertreatment of Migraine

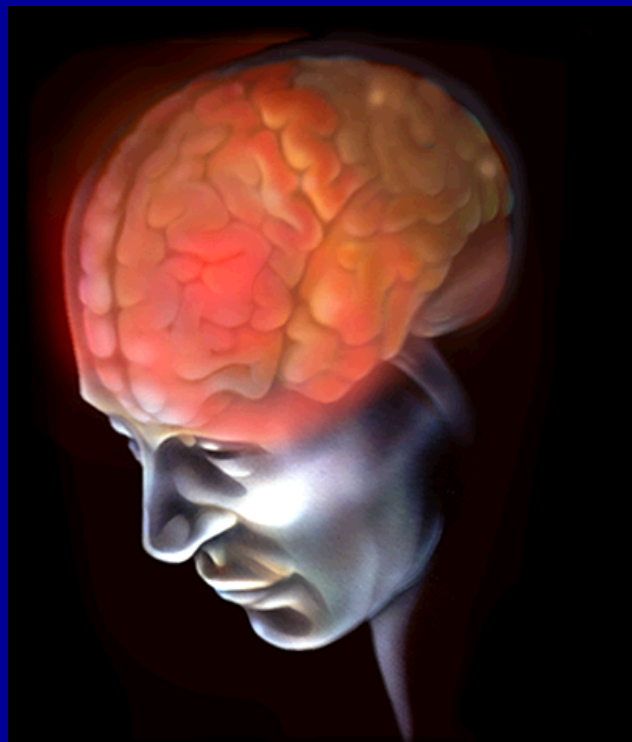
- International Headache Society (IHS) criteria for migraine
  - Designed as inclusion criteria for patients in drug efficacy trials and epidemiologic studies
  - Inappropriately used as exclusion criteria for migraine treatment by many clinicians
- Data presented so far considers only clinically obvious migraine that meets IHS criteria. Of these:
  - Only 50% are diagnosed
  - Only 15% are satisfactorily treated
- Most migraine patients seen in the otolaryngology office will *not* meet IHS criteria but *will* respond to migraine treatment

Otolaryngologic Presentations of Migraine

- Common
  - Vestibular Migraine
  - “Sinus headache”, Facial Pressure, Atypical Facial Pain
  - Aural Pressure
  - Otalgia
  - Hyperacusis
- Emerging Awareness
  - Recurrent BPPV
  - Sudden Hearing Loss
  - Meniere’s Syndrome
  - Tinnitus

WHAT IS MIGRAINE?

- Disorder characterized by episodic attacks of head pain *with* associated symptoms, such as nausea, sensitivity to light, sound, or head movement
- Highly variable presentation
- Headache not necessarily present in every attack



How Common is Migraine?

American Migraine Study II
29,727 survey responses

- There are currently 28 million migraine sufferers age 12+ in the United States
  - 21 million female
  - 7 million male
  - Overall prevalence 13%

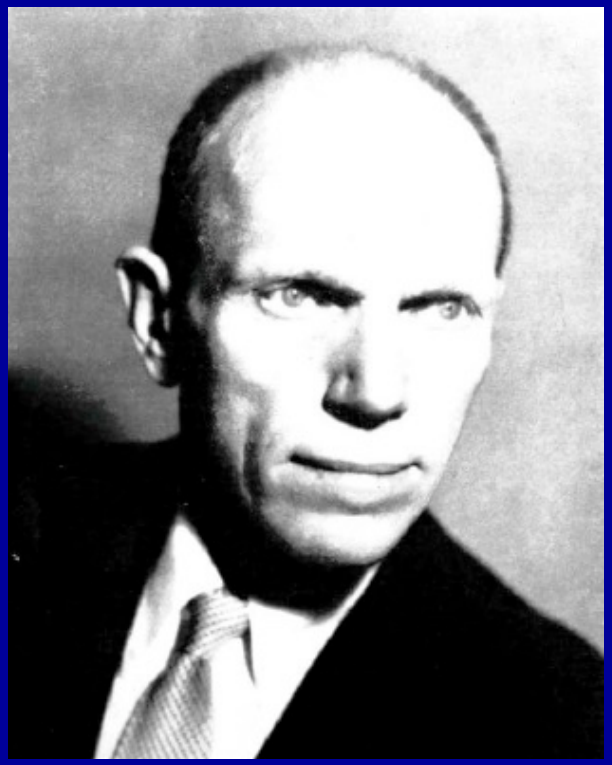
- Nearly 1 person affected in every…
  - 4 households
  - 5 women
  - 16 men
  - 11 children

Lipton RB et al. *Headache.* 2001.

Migraine Pathophysiology

What Causes Migraine Pain?
The old dogma

- Migraine is a vascular headache.
- Vasoconstriction causes ischemia and neurological symptoms (auras).
- Vasodilatation causes headache.
- *But these notions are simply incorrect in light of modern findings.*



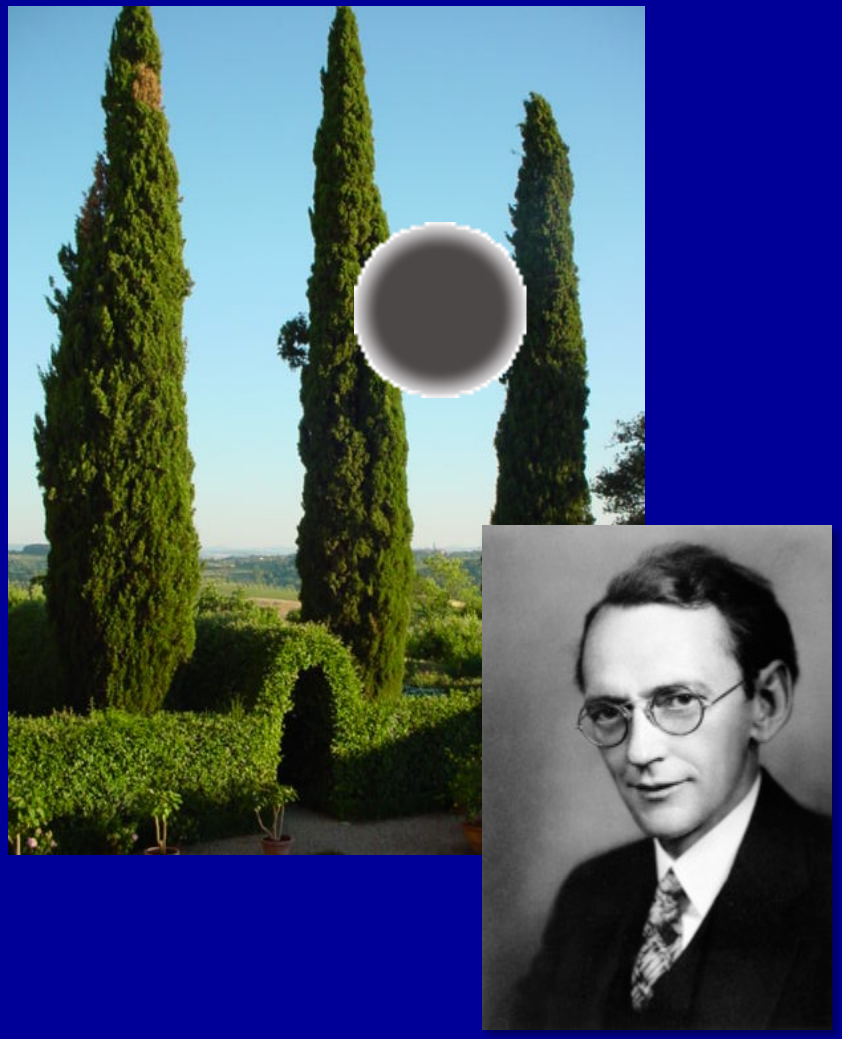
Dr. Harold Wolff

1898 - 1962

What Causes Migraine?
New Evidence

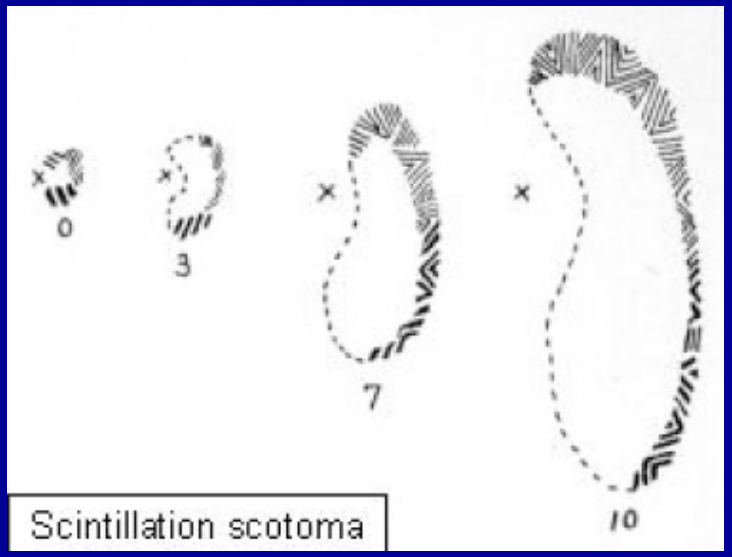
- **Spreading neuronal depression**
  - Spreading excitation, then inhibition, of brain neuronal activity correlates with visual auras.
- **Trigeminovascular efferent activity**
  - Release of vasoactive peptides by trigeminal endings causes local inflammation, including plasma extravasation.
- **Brainstem nuclear changes**
  - Altered activity of brainstem nuclei that project widely throughout the brain may explain global symptoms, e.g.- fatigue, disequilibrium
- **Hypersensitization of sensory nuclei**
  - Convergence of sensory modalities may magnify noxiousness of normally comfortable sensory stimuli.

Scintillating scotomas



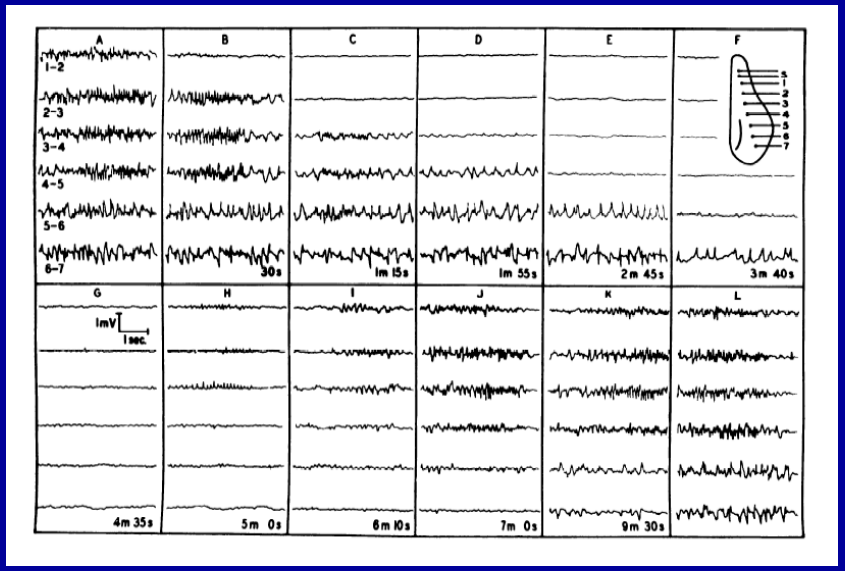
- Lashley hypothesized in 1941 that the spreading growth of bright/dark patterns might result from the spread of neuronal excitation/inhibition in the visual cortex.

Do visual auras result from cortical electrical disturbance?

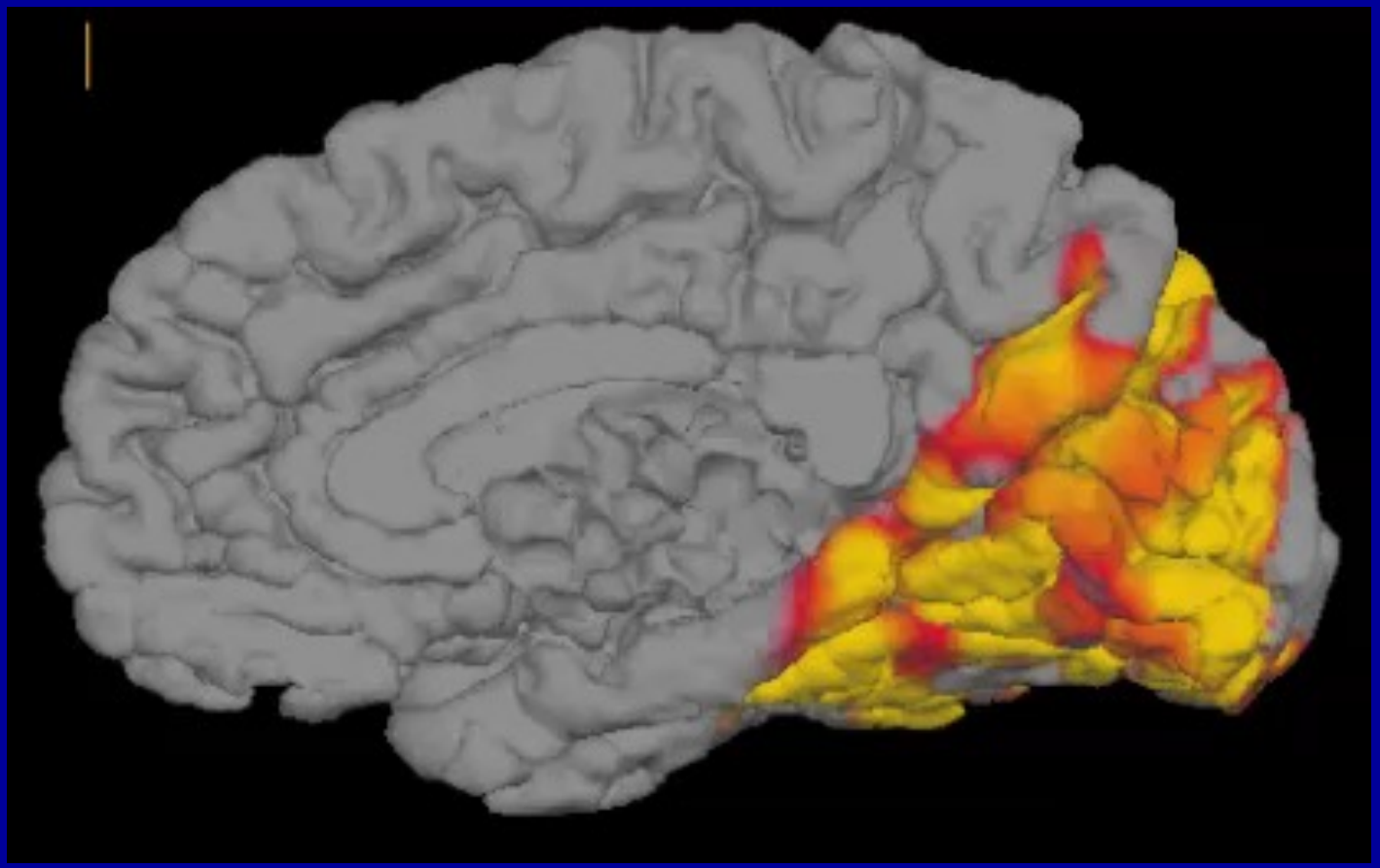


Scotoma of Karl S. Lashley, 1941

Spreading depression in auditory cortex, Leão in 1944.



MIGRAINE AURA



Hadjikhani N, et al. *Proc Natl Acad Sci USA*. 2001.

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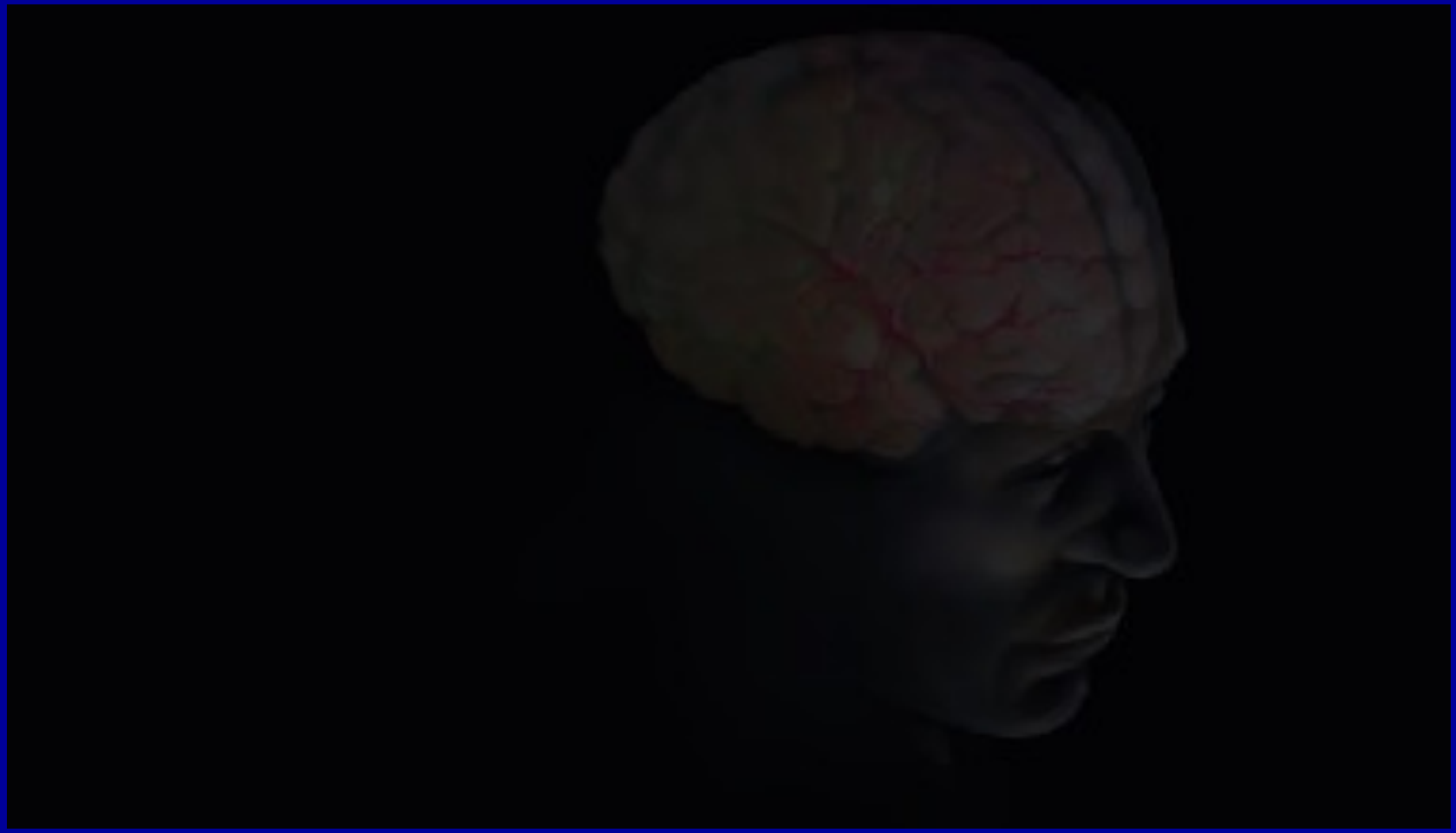
THE “OLD” VASCULAR THEORY DOES NOT FIT THE DATA



Olesen J, et al. *Ann Neurol*. 1990.

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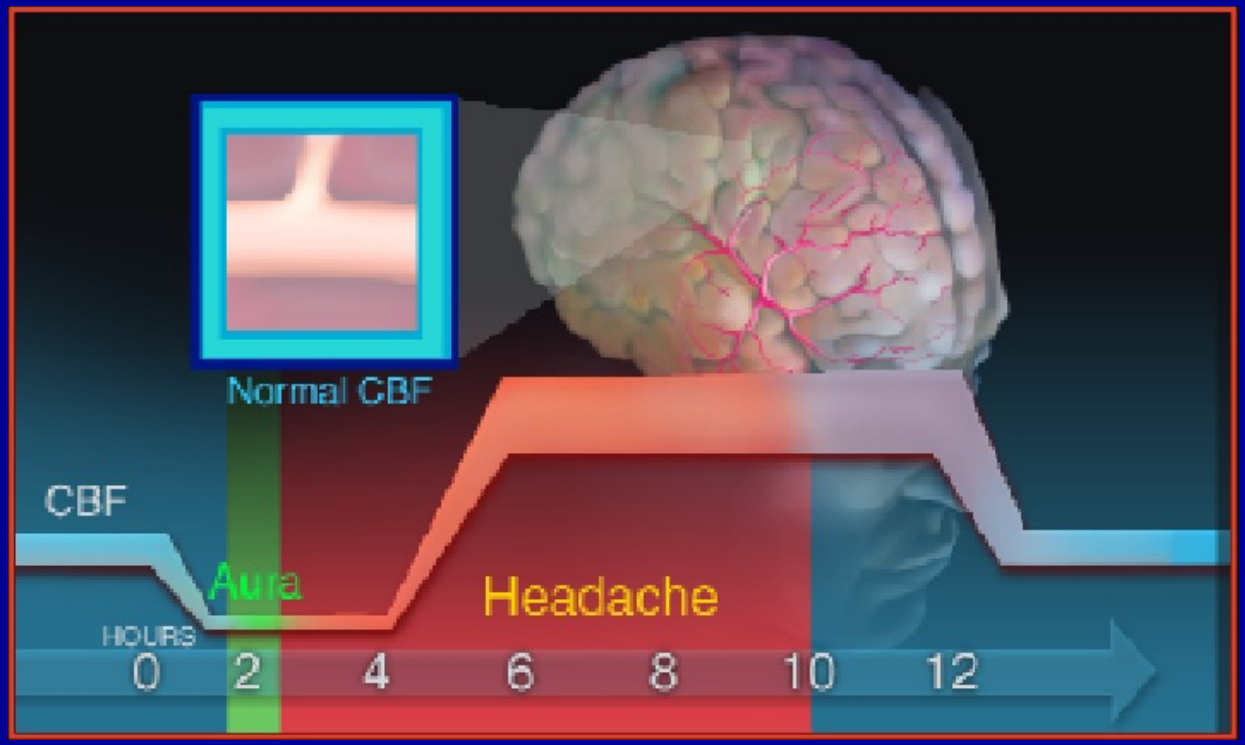
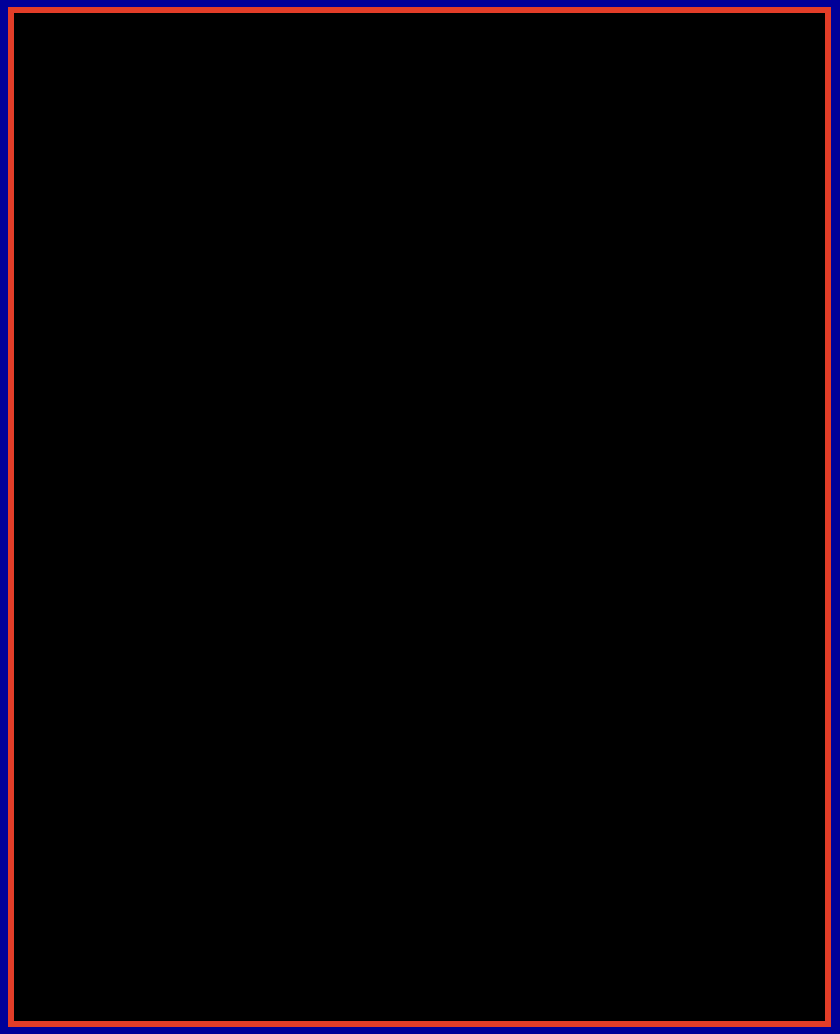


- Few patients have aura
- Headache starts during oligemia
- Headache ends during hyperemia
- Non-vascular drugs are effective (NSAIDS)

Olesen J, et al. *Ann Neurol*. 1990.

Trigeminal outflow causes local tissue inflammation

- Trigeminal outflow results in extravasation of plasma proteins and inflammatory neuropeptides around intracranial vessels. One of these (CGRP) is a potent vasodilator.
- Referred pain from dura mater and blood vessels to V1 and C2 cause typical occipital and fronto-parietal pain distribution



Goadsby, PJ, et al. *NEJM* 2002

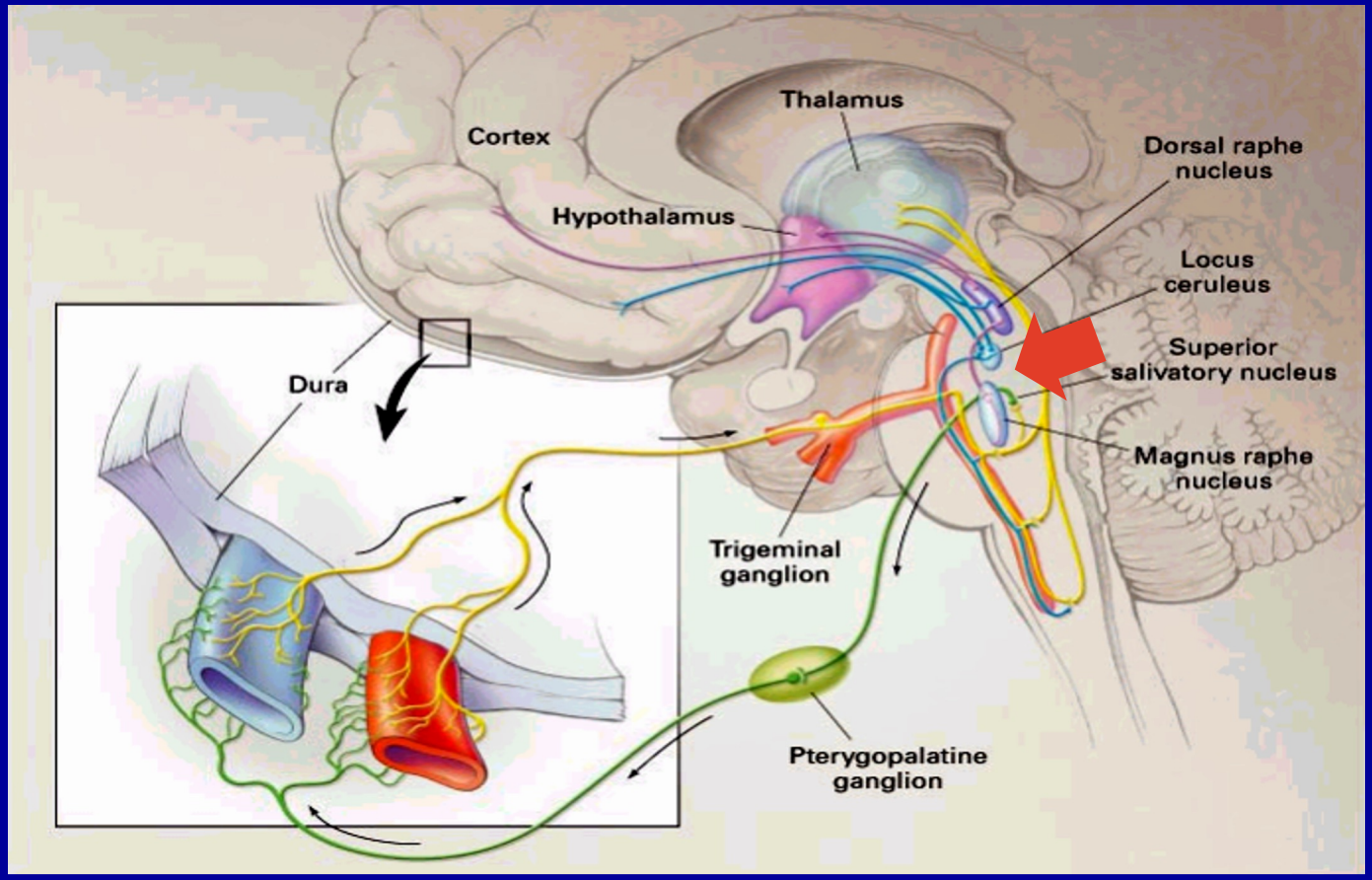
Cutrer FM, et al. *Headache: Bluebooks of Practical Neurology. Vol. 17.* 1997.

PAIN-PRODUCING INTRACRANIAL STRUCTURES

- Large cranial vessels
- Proximal cerebral vessels and dural arteries
- Large veins and venous sinuses

(after Wolff 1940)

Feedback loops in migraine



Goadsby, PJ, et al. Migraine - Current Understanding and Treatment, Jan. 24, 2002 New England Journal of Medicine, No. 4, Volume 346:257-270 Copyright (C) 2002. Massachusetts Medical Society. All rights reserved.

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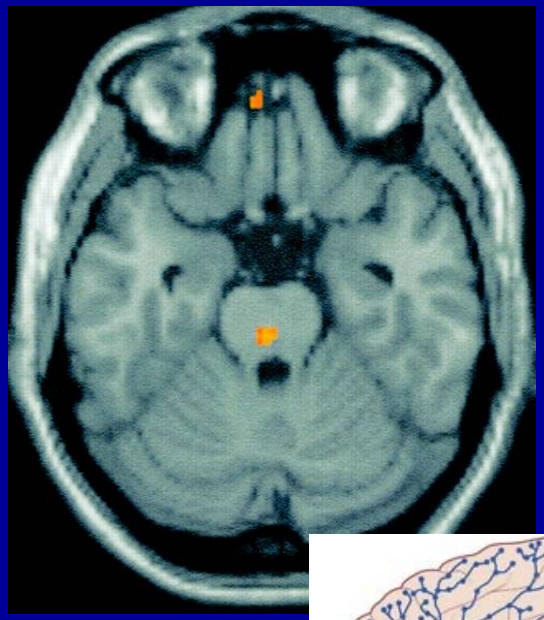
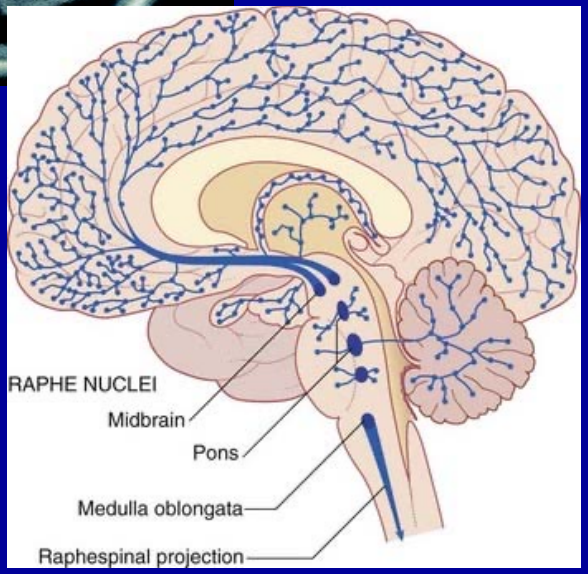
Migraine activates brainstem sites





Raphe
- *Serotonin source*

Reticular formation
- *Ascending: alertness*
- *Descending: vestibular and oculomotor motor control*

Medial lemniscus
- *Touch and proprioception fibers ascend to thalamus*

Locus coeruleus
- *Norepinephrine source*

Salivatory nucleus
- *Parasympathetic outflow*

Autonomic Dysfunction in Migraine

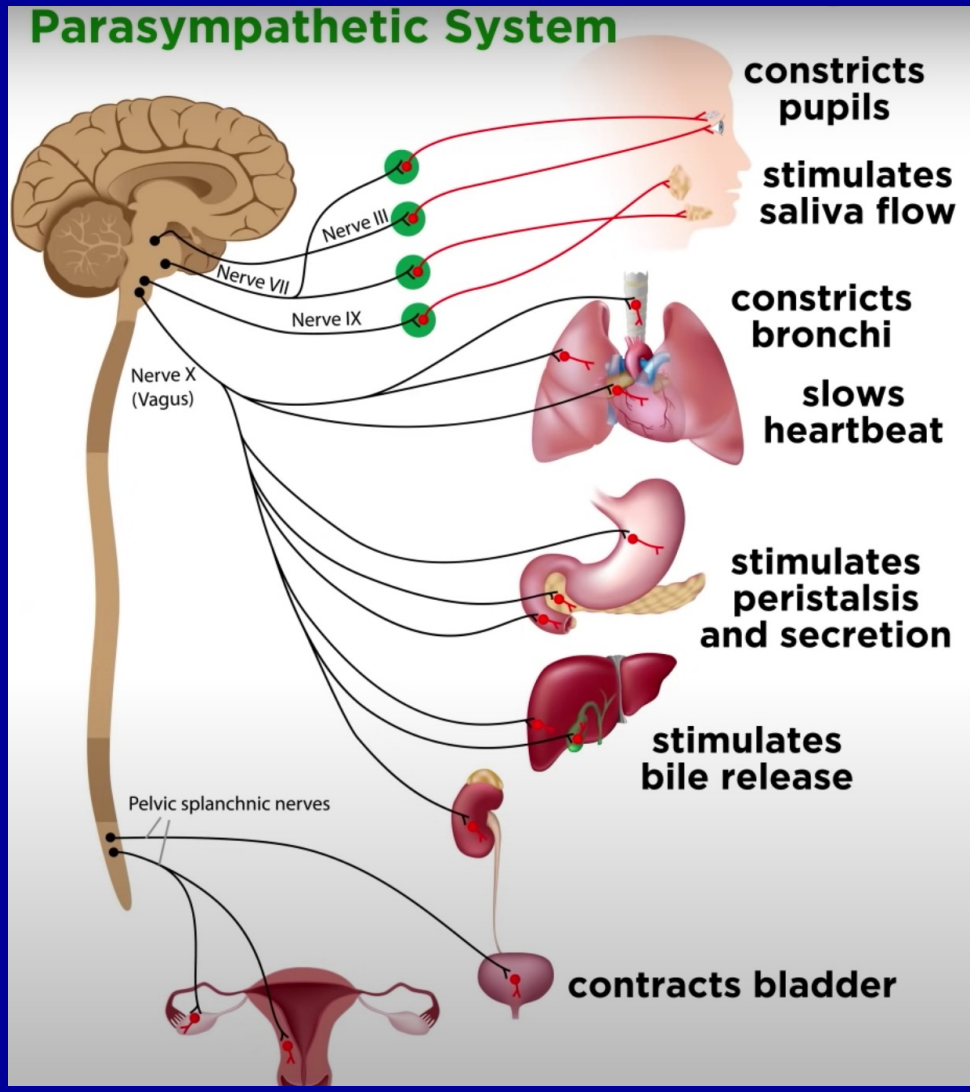
- Parasympathetic tone is increased in migraineurs and increases during attacks
  - Local Symptoms
  - Cardiac Symptoms
  - GI symptoms
  - Cardiovascular Symptoms (POTS)

- Trigemino-parasympathetic reflex - facial and sinonasal vasodilatory and secretory responses in response to pain
  - mediated by parasympathetic innervation

Migraine Symptoms
Parasympathetic Outflow

The migraine attack is associated with increased parasympathetic outflow from activity in the superior salivatory nucleus.

- Lacrimation
- Rhinorrhea
- Nasal congestion
- Soft tissue swelling
- Fluid retention
- GI symptoms
- Cardiac symptoms
- Orthostasis
- Motion Intolerance



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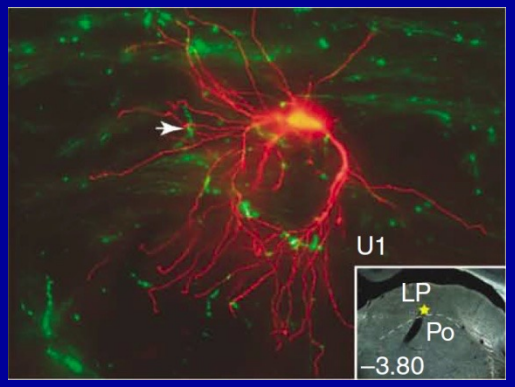
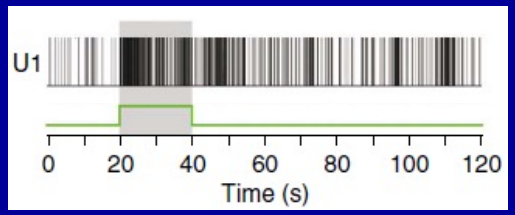
Photophobia and Phonophobia

http://i.telegraph.co.uk/telegraph/multimedia/archive/01422/migraine_1422924c.jpg

http://cdn.sheknows.com/articles/woman

A mechanism for photophobia



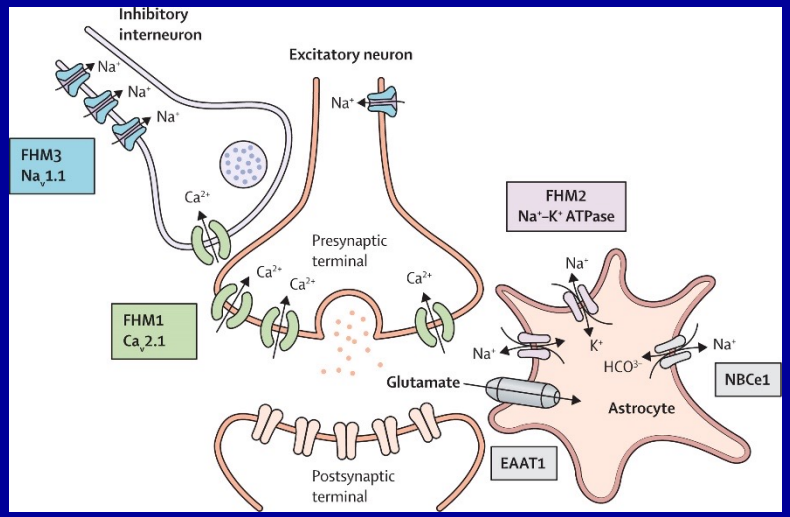


- Burstein et al have shown evidence that photophobia in migraine is due to axonal projections of retinal afferents that converge on dura-sensitive neurons in posterior thalamus.
- Bright light increases firing of these dura-sensitive neurons.

Nosda et al. Nat Neurosci 2010

Genetics – an ion channelopathy?

- Familial hemiplegic migraine
  - Rare, autosomal dominant migraine with auras that include motor weakness
  - Mutations in several genes involved in ion transport
  - Net result: neuronal hyperexcitability



- Model for common migraine?

  Migraine sufferers have altered neurophysiologic responses between attacks.
  Concept of ‘a sensitive brain’

Russell & Ducros, *Lancet Neurol* 2011,10(5):457-470

Migraine is Polygenetic.

- Genome Wide Association studies
- 8 major groups:
  - ion channels membrane potential - 11.2%
  - homeostasis of blood vessels - 26.5%
  - metabolism of neurotransmitters - 11.2%
  - transport/reception of neurotransmitters - 24.5%
  - neurogenesis - 5.1%
  - inflammation - 8.2%
  - sex hormones - 5.1%
  - other - 8.2%

Kondratieva, N et al, J Neurol Sci. 2016 Oct 15;369:63-76. doi: 10.1016/j.jns.2016.08.008.

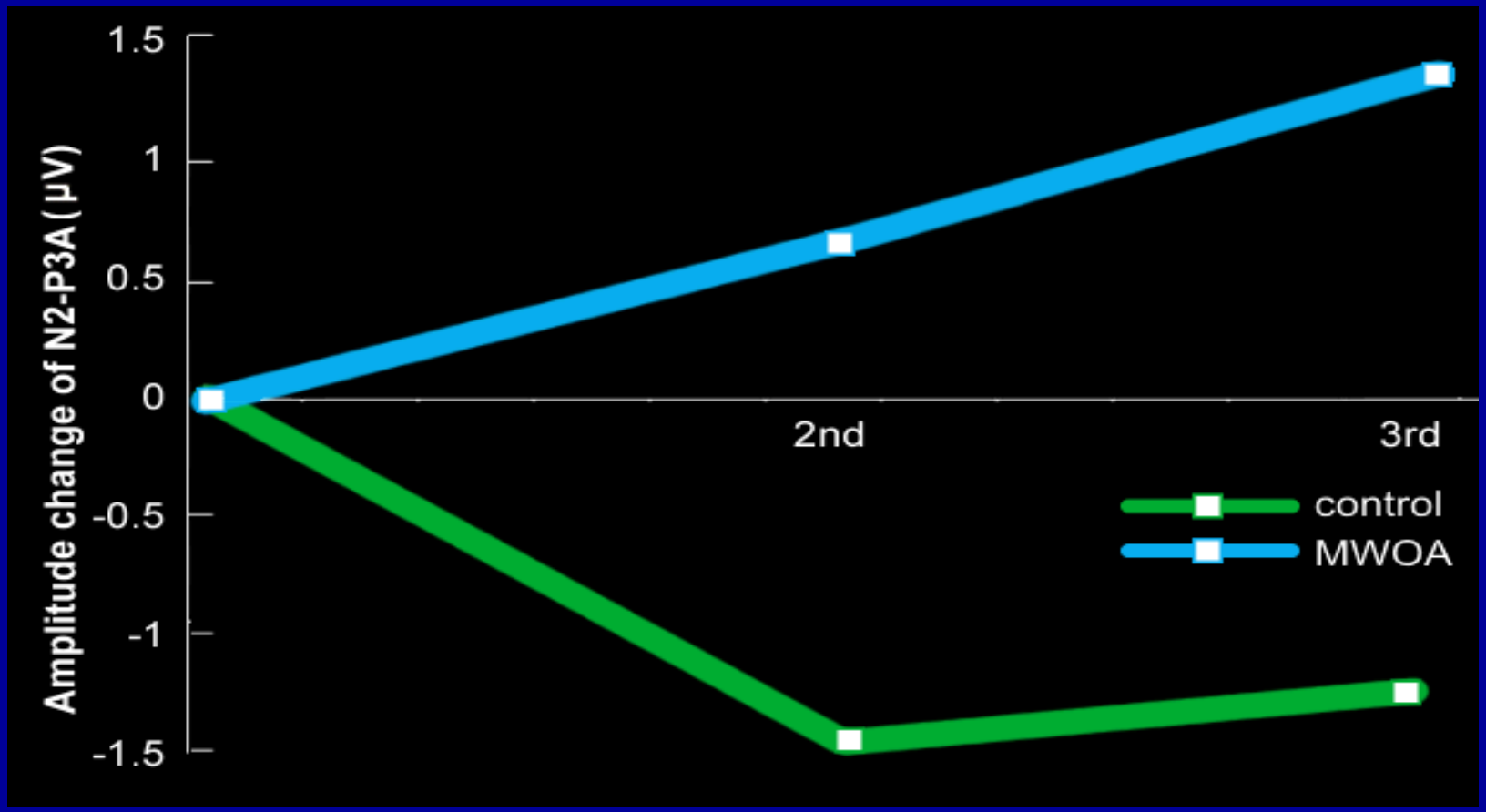
Genetics of Common Migraine

If one of your parents has migraine you are 40% more likely to have them too.

If both your parents have migraine that number jumps to 90%.

Potentiation vs. Habituation of Sensory Responses in Migraine

Passive “Oddball” Auditory ERP in Migraine



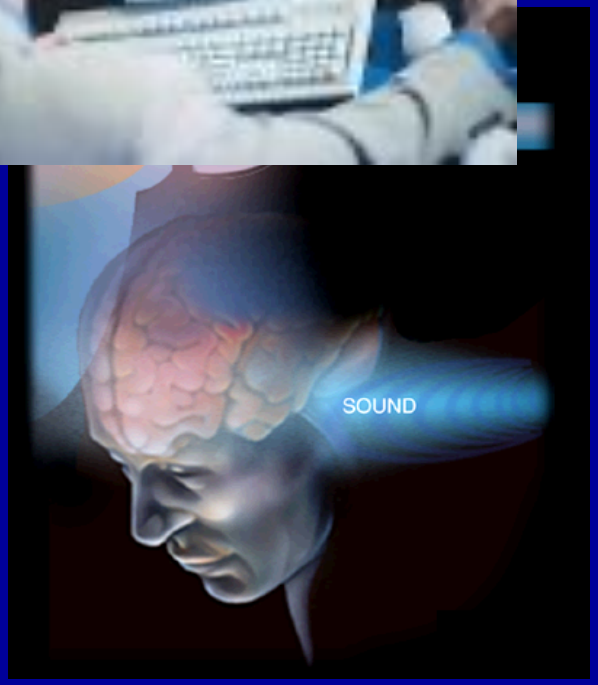
Wang W, Schoenen J. *Cephalalgia*. 1998.

Triggering Migraine- Categories of Migraine Triggers

- Dietary triggers
- Environmental triggers
- Physiologic triggers

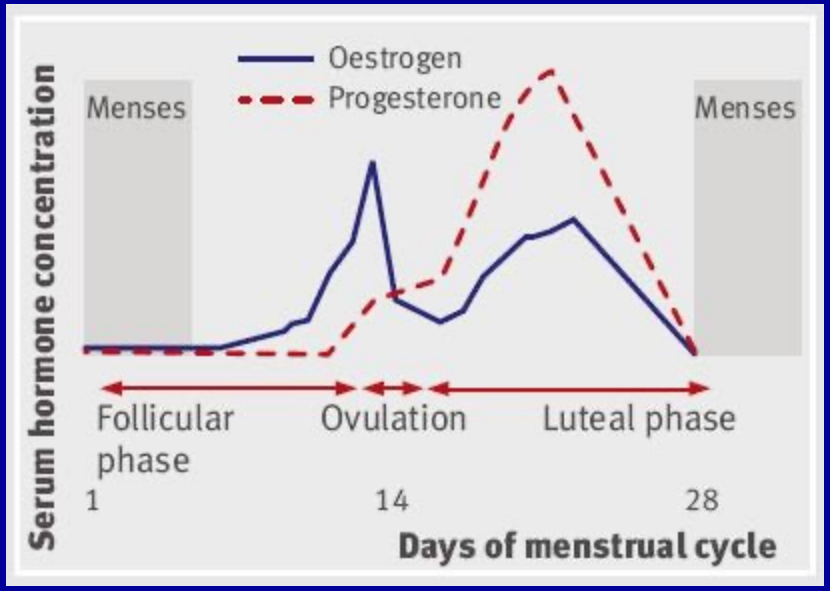
Common Environmental Triggers for Migraine Symptoms

- Odors
- Bright Lights- computer
- Noise
- Excessive head motion
- Excessive motion of visual surround
- Weather changes
- Altitude



Common Physiologic Triggers for Migraine Symptoms

- Anxiety, stress
- Fatigue, lack of sleep
- Oversleeping
- Hunger
- Exercise
- Hormone changes



- Pain- especially C2 cervical pain (whiplash), TMJ, and sinusitis or ostia obstruction
- Travel- Stress, letdown, altitude, alcohol, dehydration, time change

Common Food Triggers for Migraine Symptoms

- Byproducts of food aging – e.g. tyramine
  - Fermented products like red wine
  - Aged cheese
  - Yeast in fresh bread, live yogurt
- Amines or chemicals similar to our own neurotransmitters
  - Caffeine
  - Nitrates and other preservatives (lunchmeat)
  - MSG
  - Chocolate

Common Food Triggers for Migraine Symptoms

- Many surprise foods: Bananas, nuts, peanut butter, citrus
- Effects may come immediately or even days later
- Dietary, Physiologic and Environmental triggers are additive and synergistic
  - Chocolate - no problem
  - Red wine - no problem
  - Chocolate + red wine - problem

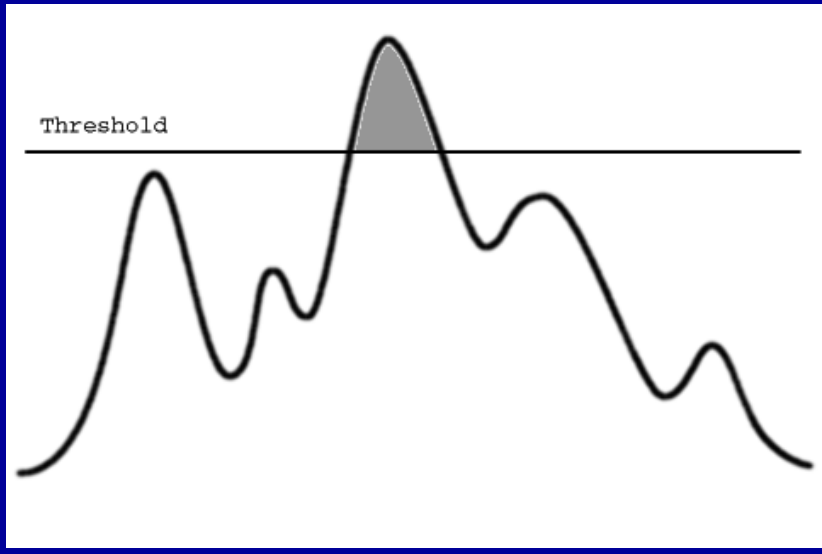
IgG mediated food allergy in migraine

- Diet restriction in migraine, based on IgG against foods: A clinical double-blind, randomized, cross-over trial
- 266 foods tested
- Crossover provocation and elimination diets
  - Headache days decreased in the elimination diet period (from 10.54.4 to 7.53.7; P<0.001)
  - Migraine attacks decreased in the elimination diet period (from 9.04.4 to 6.23.8; P<0.001)

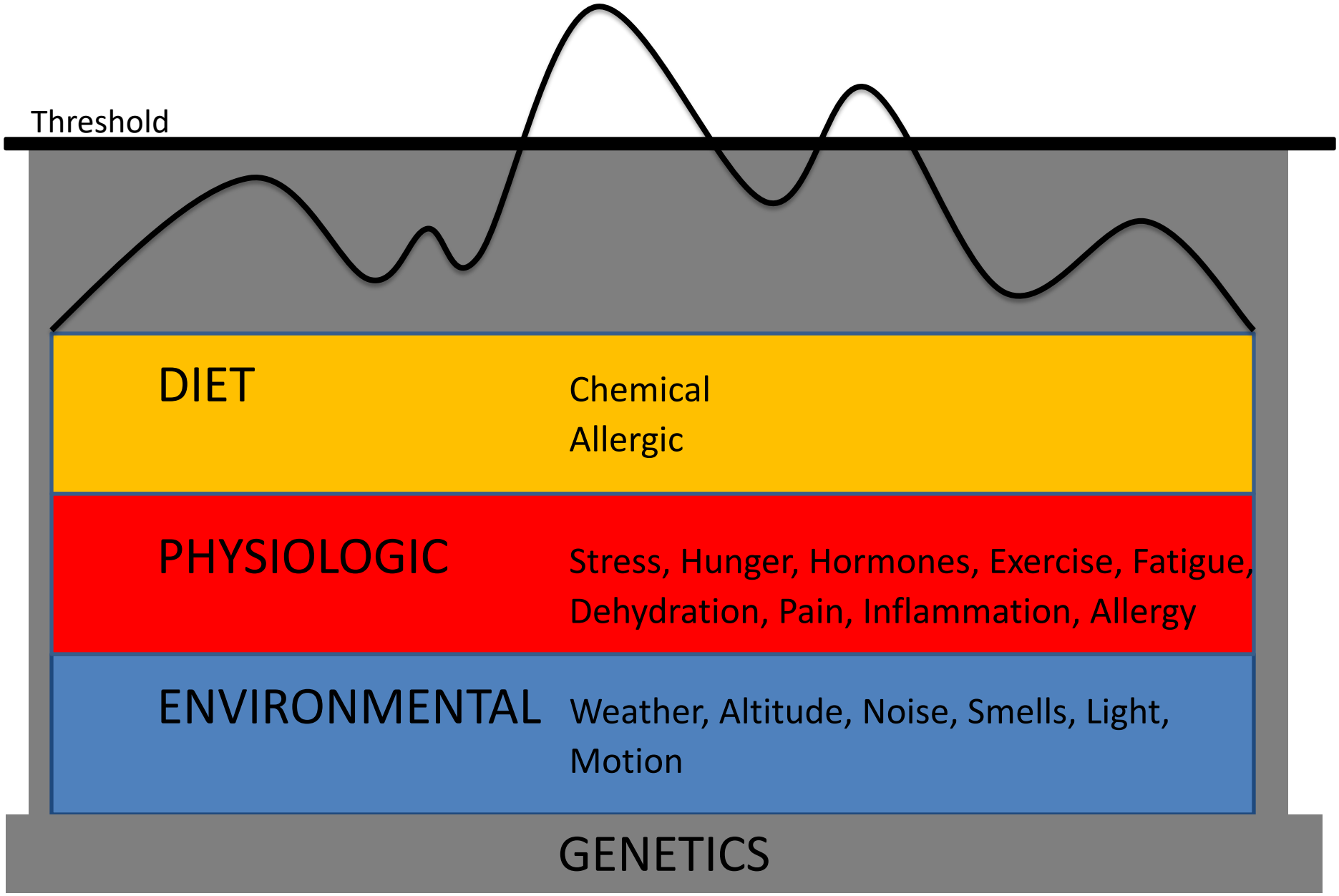
Alpay K, et al, Cephalalgia 30(7) 829–837 2010

Migraine Threshold

- Genetic variability determines a threshold to migraine activation
- Combinations of partial triggers create a total stimulus burden that causes the threshold to be exceeded



A Migraineur: Partial Triggers and Susceptibilities



Migraine and Vertigo

Vestibular Migraine

- A 70-year-old man presented with a 30-year history of dizziness and episodic vertigo. Some of his vertigo attacks were clearly positional, others spontaneous and lasting for hours. These were not associated with changes in his hearing, but he did report left aural fullness, a "bobble-head"/rocking sensation, photophobia, and nausea with the attacks. Symptoms progressed to constant disequilibrium.
- Family Hx: migraines and dizziness occurred in his mother, in his sister, and in all 3 of his children.
- Audio: Normal through 4k, then R>L downsloping SHNL, Normal ENG, normal MRI.
- Symptoms resolved on nortriptyline

Is it central or peripheral?

Maybe both!

Where do vestibular symptoms from migraine arise?

- Cortex
- Thalamus
- Limbic System

Supratentorial

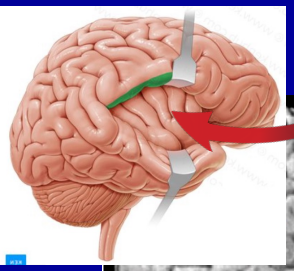
- Brainstem nuclei
- Cerebellum

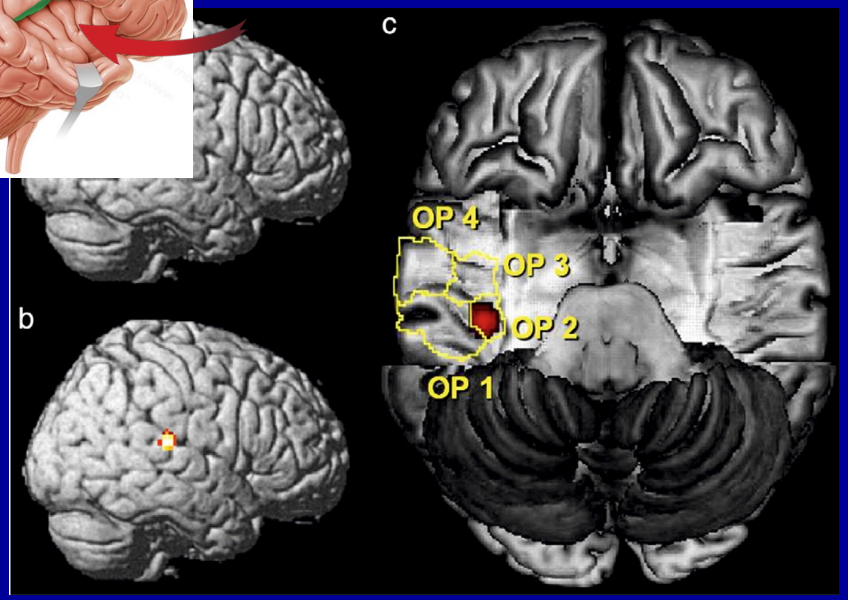
Infratentorial

- Inner ear

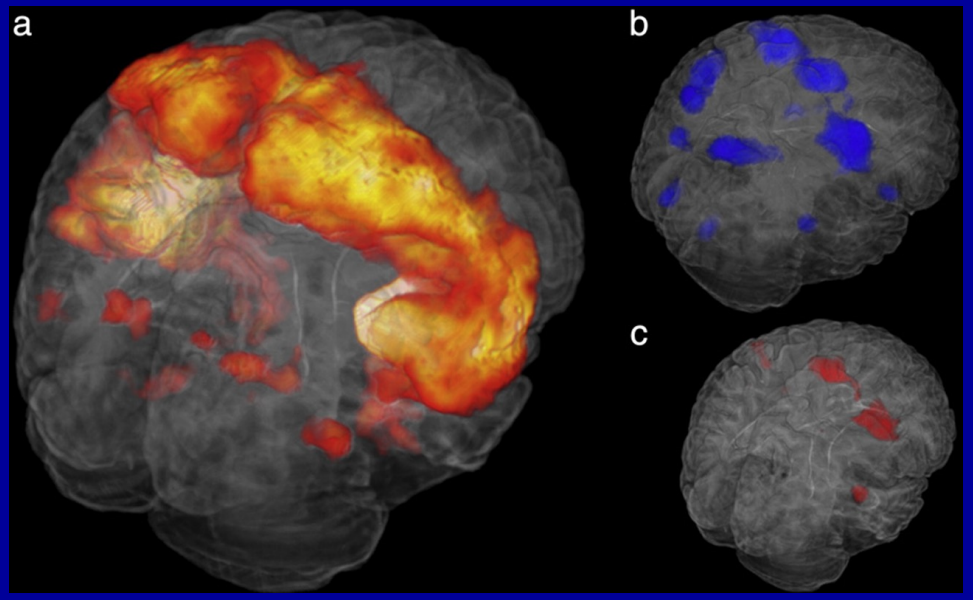
Labyrinthine

Migraine Aura can cause Dizziness





Canal stimulation: right hemispheric parietal opercular area OP 2



Saccadic eye movements

Vestibular responsive cortical areas are multi-modal: can be stimulated by vestibular, somatosensory, visual stimuli and eye movements(like the vestibular nuclei).

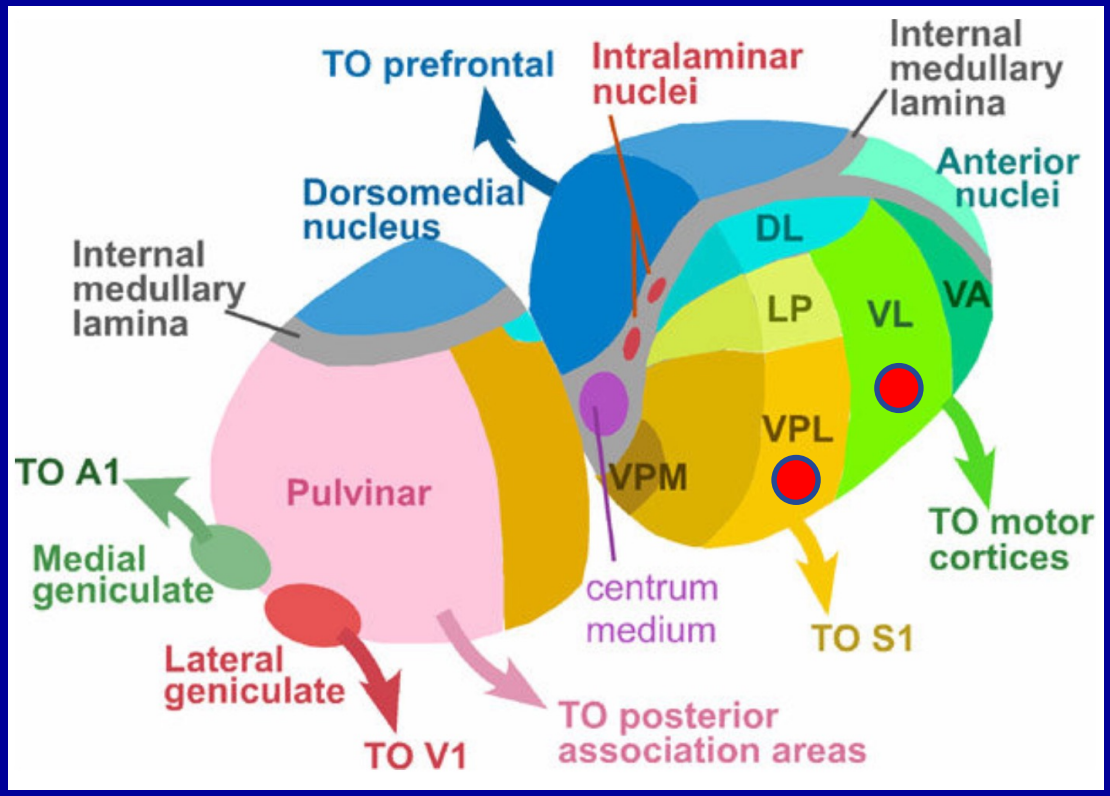
Meta-analytical definition and functional connectivity of the human vestibular cortex P. zu Eulenburg, S. Caspers , C. Roski b S.B. Eickhoff, Neuroimage 60 (2012)

Dizziness from the Thalamus

- Best explanation for:
  - Alice in Wonderland syndrome
  - ”Displaced in space.”
  - Walking on Foam
  - “Bouncy House”
  - PPPD?

Clinical case-Thalamic lesion causes proprioceptive distortion

- 78 yo M with intractable essential tremor
- Focused ultrasound to ventral intermediate nucleus of thalamus
- Left side successful, right side unsuccessful
- New proprioceptive distortion on right

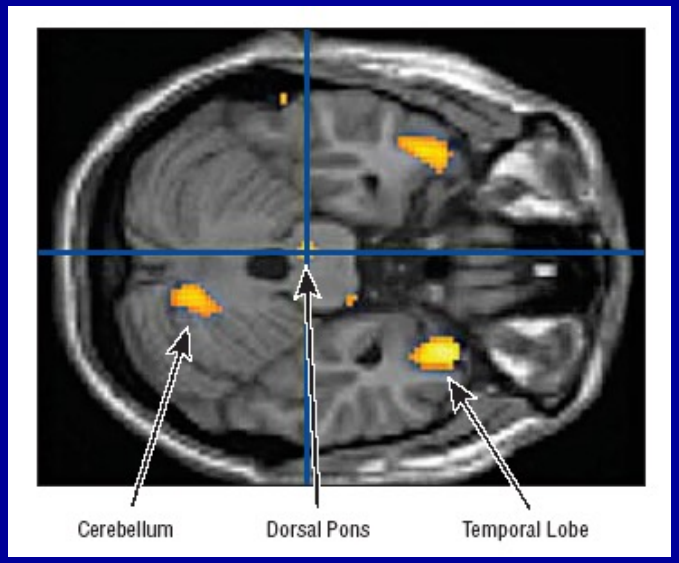


https://www.researchgate.net/figure/Overview-of-thalamic-nuclei

A possible central mechanism

Neuronal electrical disturbances include the pons:

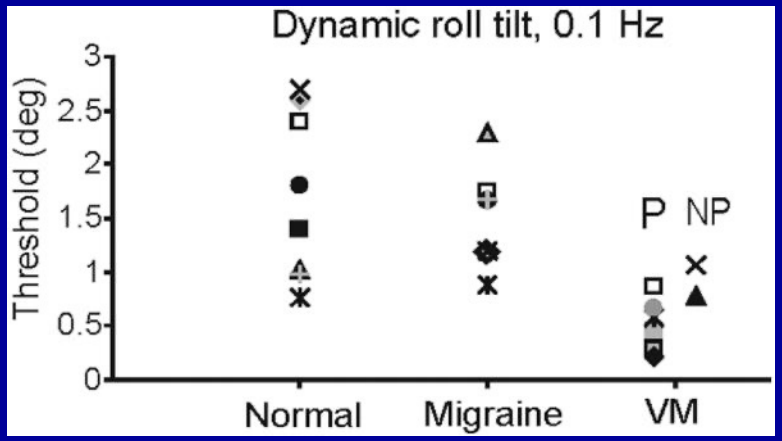
- Vestibular nuclei
  - Disequilibrium
  - Head motion intolerance
  - Visual motion intolerance
  - Frank vertigo
  - Unresponsive to typical peripheral vestibular suppressants



Afridi SK et al. *Brain* 2005.

Enhanced Sensitivity in VM

Patients with vestibular migraine have dramatically lower thresholds (much greater sensitivity) to motion in certain planes than do normal people and migraineurs without vertigo.

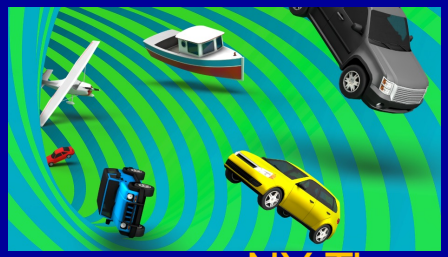


Lewis RF, Priesol AJ, Nicoucar K, Lim K, Merfeld D. Dynamic tilt thresholds are reduced in vestibular migraine. J Vestib Res 2011, 21:323-330.

King S, Wang J, Priesol AJ, Lewis RF. Central integration of canal and otolith signals is abnormal in vestibular migraine. Front Neurol 2014, 5:233.

Extreme OKN sensitivity in VM

- OK reflex supplements the VOR and is important for gaze stabilization in constant motion environments(no acceleration to stimulate SCCs)
- Visual scene drift acts as a surrogate for SCC stimulation even after the cupola has come to rest
- Results in nystagmus and in a perceived vection
- *Velocity storage allows continued perception of motion below the mechanical properties of the cupola*
- *This seems to be impaired in VM patients*
- *Often very averse to OKN stimuli in real life situations*



NY Times

VM and Motion Sickness

- Biological significance unclear -only in artificial motion environments, never by normal locomotion or everyday postural reactions.
- Conflict between 2 streams of information, *and with expected* based on experience
- Habituation results in symptom improvement(learning)
- Symptoms return on return to stable ground(mismatch of learned adaptation and expectation)
- *Symptoms overall are autonomic*
- *In most chronic migraine there is dysautonomia with increased parasympathetic outflow(superior salivatory nucleus)*
- In most VM hx of motion sickness is lifelong

A possible peripheral mechanism: inflammation in the inner ear

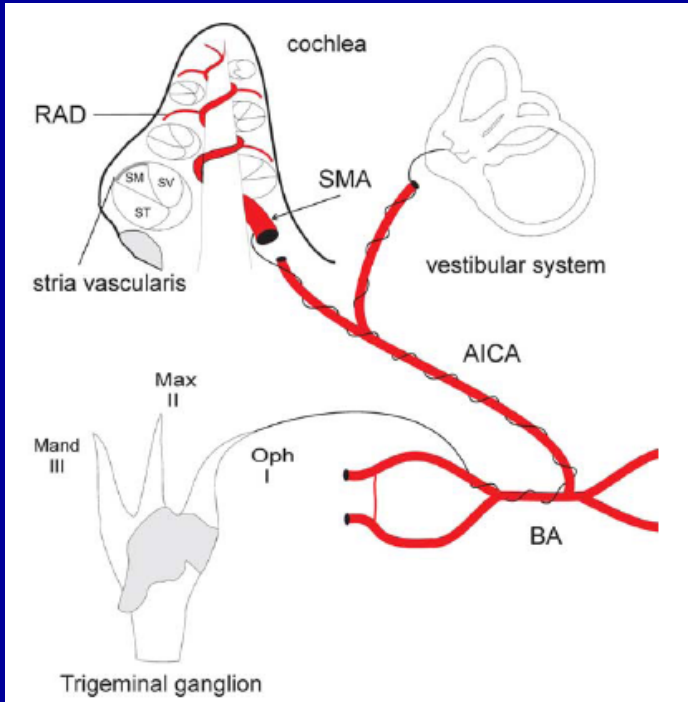
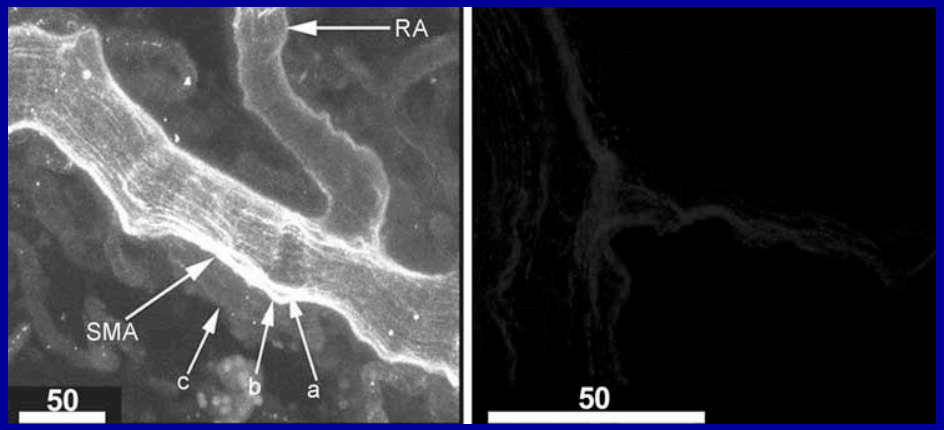


Fig. 1. Schematic of the anatomical relationship of the trigeminal ganglion, cochlea, vestibular labyrinth and BA. The cochlea and vestibular labyrinth receive innervation from the ophthalmic branch (Oph) of the trigeminal ganglion, which may pass along the BA and AICA. Mand, mandibular branch of trigeminal ganglia; Max, maxillar branch of trigeminal ganglia; SM, scala media; ST, scala tympani; SV, scala vestibuli.

Z. Vass et al. / Neuroscience 124 (2004) 919–927

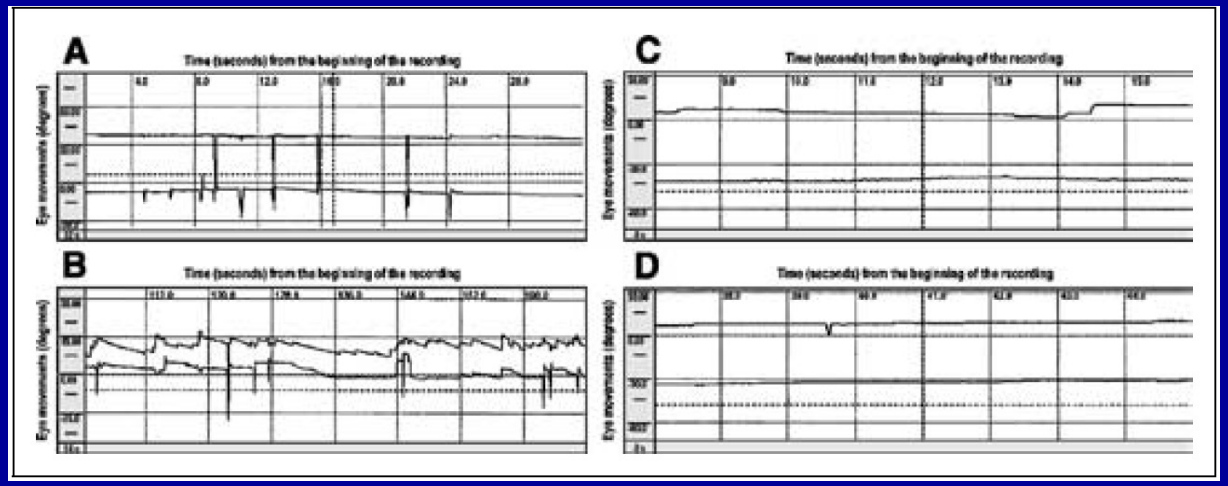
- Trigeminal efferent neurons reach the cochlea- V1
- Electrical stimulation of the trigeminal nerve causes plasma extravasation in spiral modiolar artery.



stimulated control

Trigeminal Stimulation can Induce Nystagmus

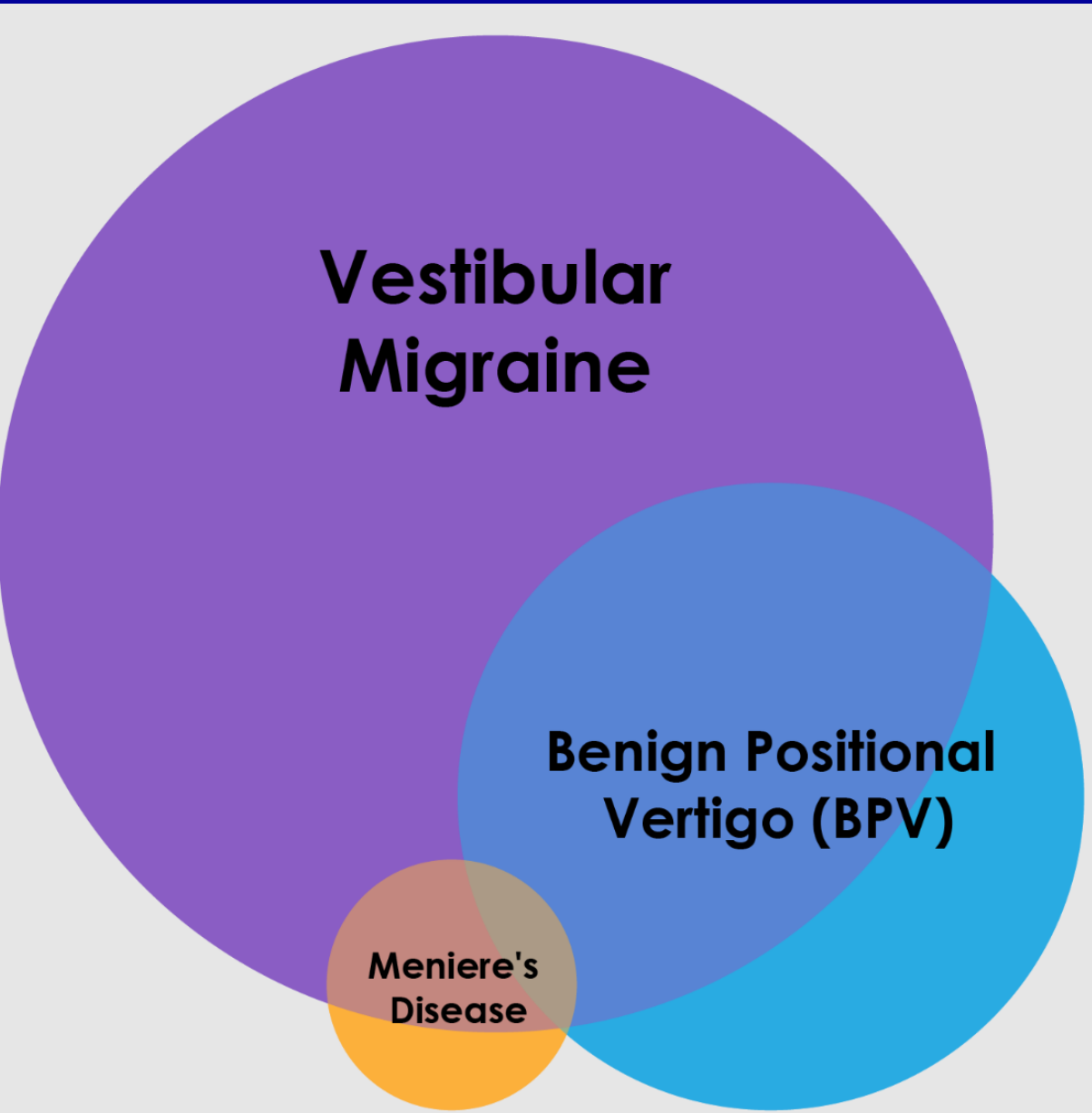
- Supraorbital nerve stimulation induced or modified Ny in all of 10 migraine patients.



- Mean latency 25 s. Mean duration 120 s
- Visual suppression. Linear slow phase.
- Ipsilateral excitatory effect.

Marano et al, *Headache*, Volume 45, Issue4 April 2005, Pages 325-331

Prevalence of Vestibular Migraine



VM prevalence in Germany(survey 4869 Adults)

- 0.89% of adults
- 0.98% lifetime

Neuhauser et al, *Neurology*, 2006; 67:1028-33

VM prevalence in the US (NHIS survey 21K)

- 2.7% of adults (6.1 million)

Formeister,EJ, Rizk,HG, et al, Otology & Neurotology,2018;39:1037-44

Prevalence of Vestibular Migraine

- Migraine occurs at some point in time in 15-20% of adults in the USA.
- 25% of migraine sufferers report attacks of vertigo.
- Thus, migraine associated vertigo is VERY common.
- Most otolaryngology textbooks focus much more on Ménière's disease, but, in fact, migraine-associated vertigo is probably 10-20 times more common!

Vestibular Migraine

- The symptoms may be described as vertigo (spinning, rocking, swaying, even positional) or simply disequilibrium.
- Motion sensitivity is very common, often from childhood.
- The symptoms may be quite variable in duration, lasting minutes to days in episodic cases, or may present as constant disequilibrium lasting for months.
- Intolerance to movement of the head or the visual world is a frequent finding.

Nystagmus in Vestibular Migraine

- Nystagmus can be provoked with positional testing in 100% of symptomatic patients.
- Positional nystagmus most commonly was sustained, of low velocity, and could be horizontal, vertical or torsional.
- The nystagmus dissipates when the patient is free of symptoms.

Polensek SH, Tusa RJ. Nystagmus during attacks of vestibular migraine: an aid in diagnosis. Audiol Neurootol. 2009;15:241–246.

Vestibular Migraine VNG Findings

- VNG may show a caloric weakness in 15-35%
- Abnormal saccades, sinusoidal tracking in a young patient
- Cannot tolerate OPK
- Cannot finish caloric testing
- VNG may provoke a long incapacitating attack

Cutrer, *Headache*, 1992;32
Kuritzky, *Headache*, 1981;21
Bir et al, *J Otolaryngol*, 2003;32

Is Vestibular Migraine Associated with Headache?

- Typical migraine headaches – occur during the vertigo spells in only about half of cases.

- Often the patient has a prior history of migraine headaches, but feels that they have resolved.
  - Milder head or neck pain or pressure may replace the pounding headaches and accompany the dizzy symptoms.

- A family history of migraine may be helpful in the diagnosis, as may a history of unexplained falling spells and motion sensitivity as a child.

Does vertigo respond to migraine treatment?

- Patients with migrainous vertigo (MV) and complex dizziness of undetermined etiology (CDUE) responded to migraine treatments.
  - 46% responded to nortriptyline
  - 25% responded to topirimate
  - 16% responded to caffeine cessation
- 75% of MV and 50% of CDUE responded to these treatments

Mikulec AA, et al., Am J Otolaryngol. 2011 Jun

Rocking Dizziness

- Highly associated with migraine
- Mal de debarquement syndrome is a form
- Uniquely responsive to PT(OKN retraining)
- Responds to concurrent migraine management

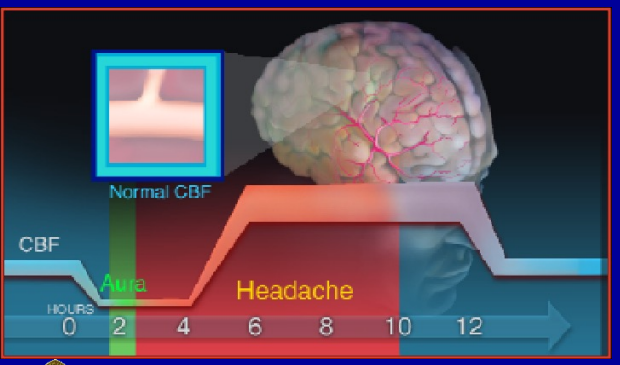
Dai M, Cohen B, Smouha E, Cho C. Front Neurol. 2014; 5:124. Epub 2014 Jul 15.

Ghavami , Djalalian et al ,*Laryngoscope*. 2017 Jul;127(7):1670-1675. Management of mal de debarquement syndrome as vestibular migraines.

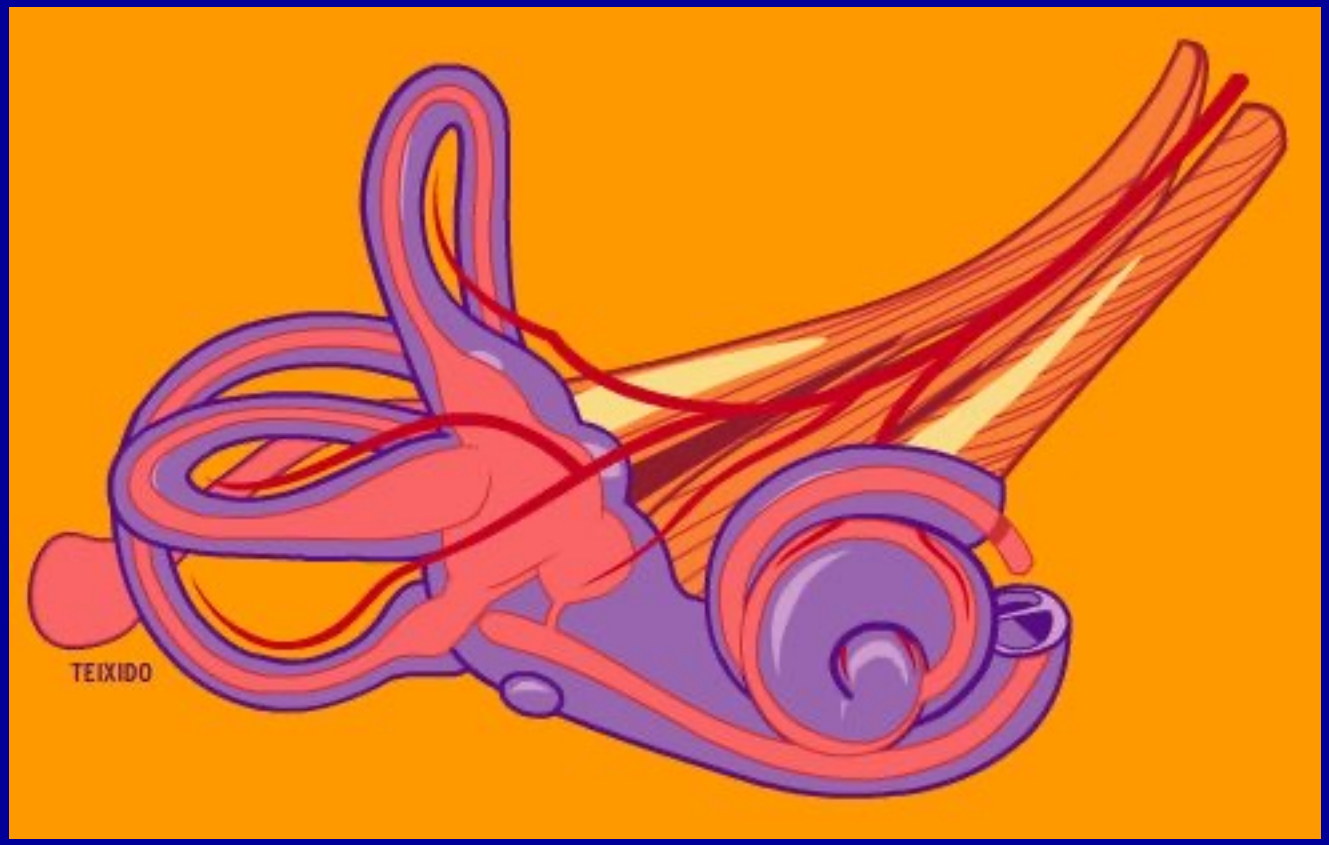
Migraine and Ménière's Disease

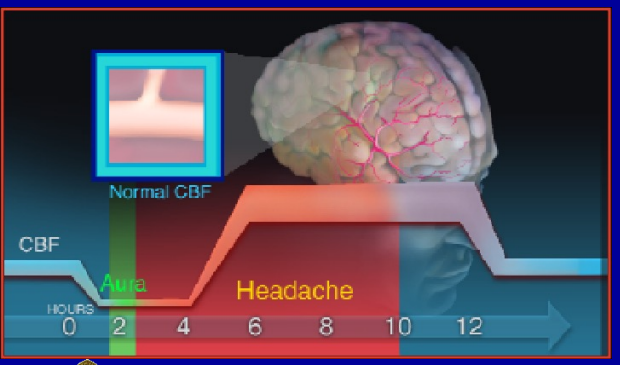
Migraine and Ménière's disease

- If plasma extravasation occurred in both the vestibular and cochlear blood vessels, would the patient have symptoms of Ménière's disease?
- Could endolymphatic hydrops result from repeated episodes of plasma extravasation?
- Note the similarities between MD and migraine:
  - Dietary and other triggers
  - Excitatory followed by inhibitory phase of nystagmus

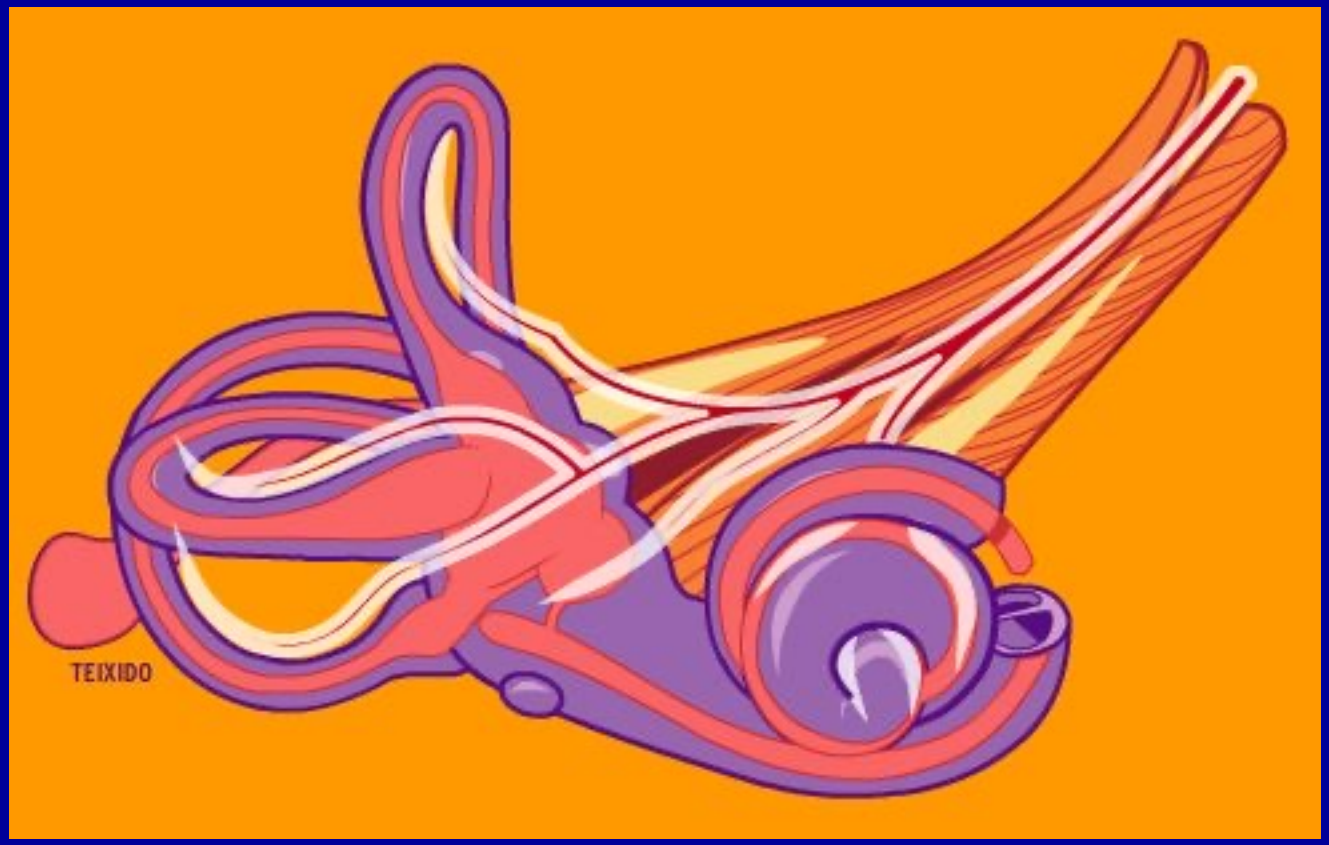


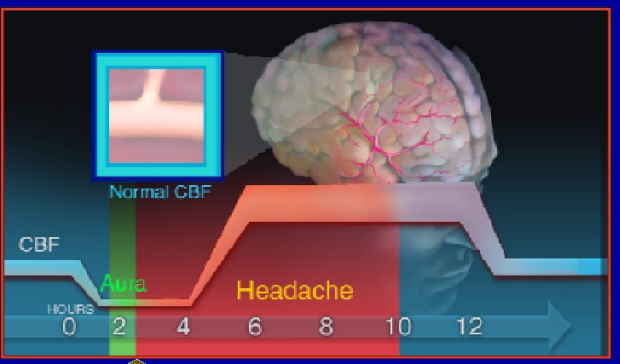
Migraine in the Inner Ear?



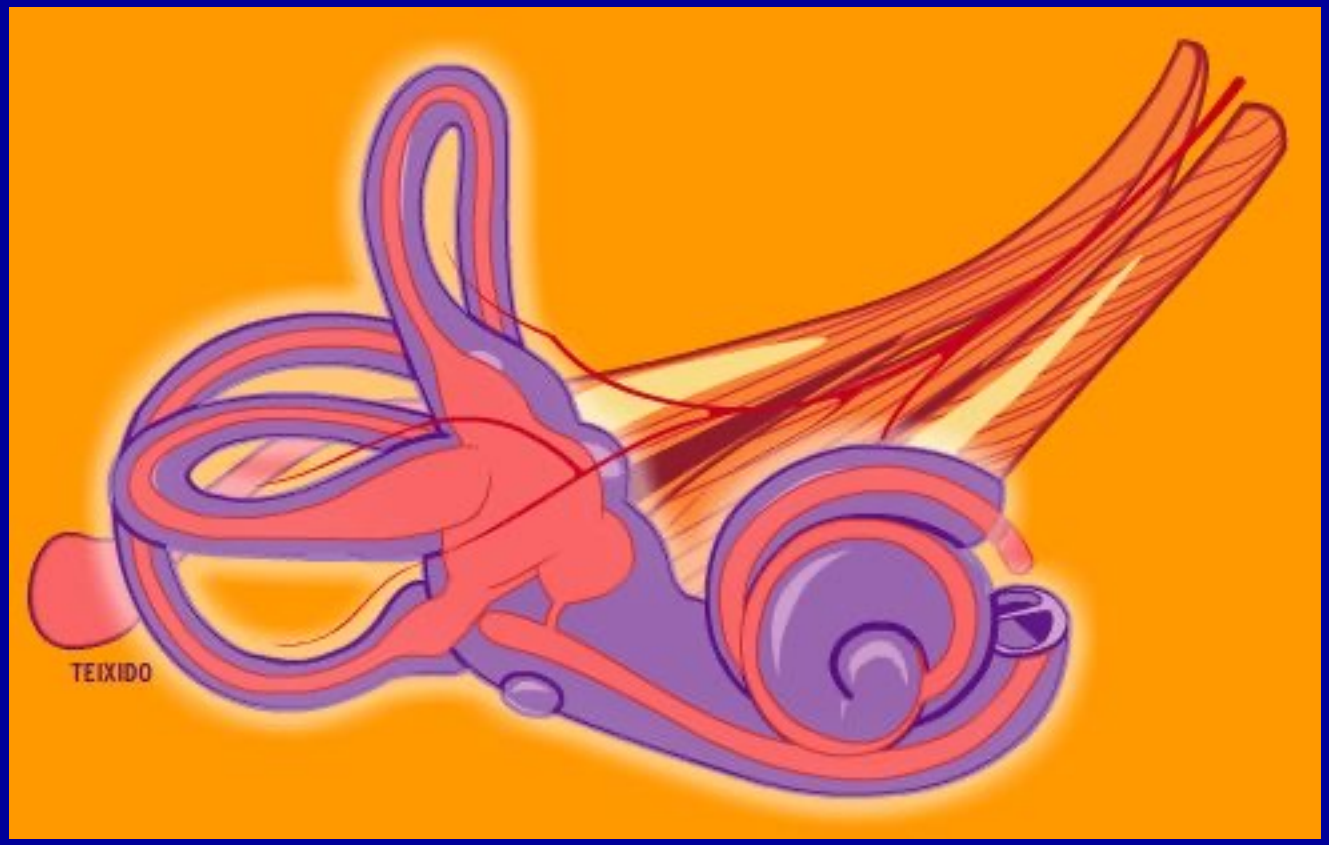


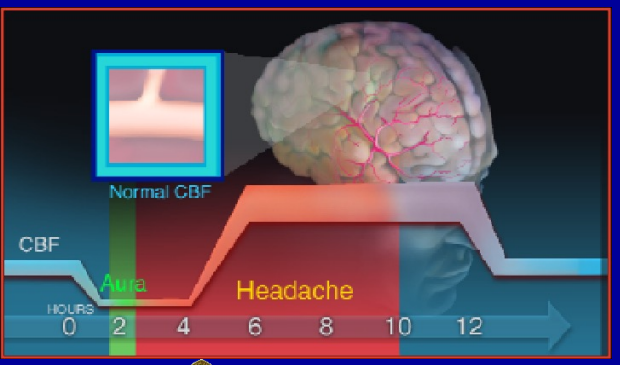
Migraine in the Inner Ear?



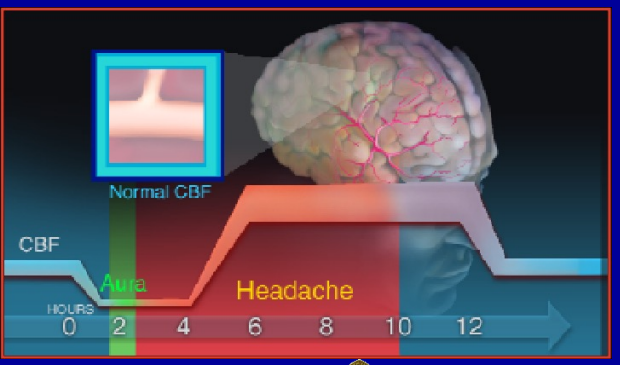


Migraine in the Inner Ear?

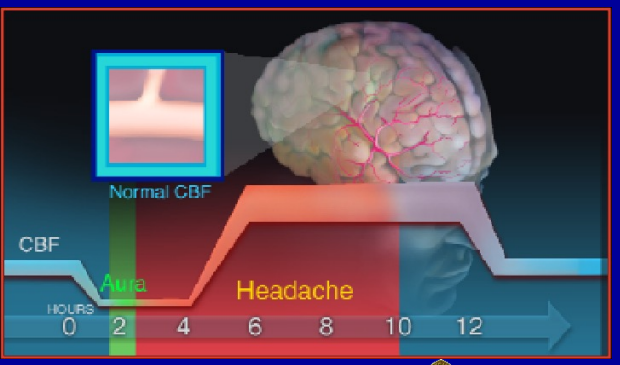




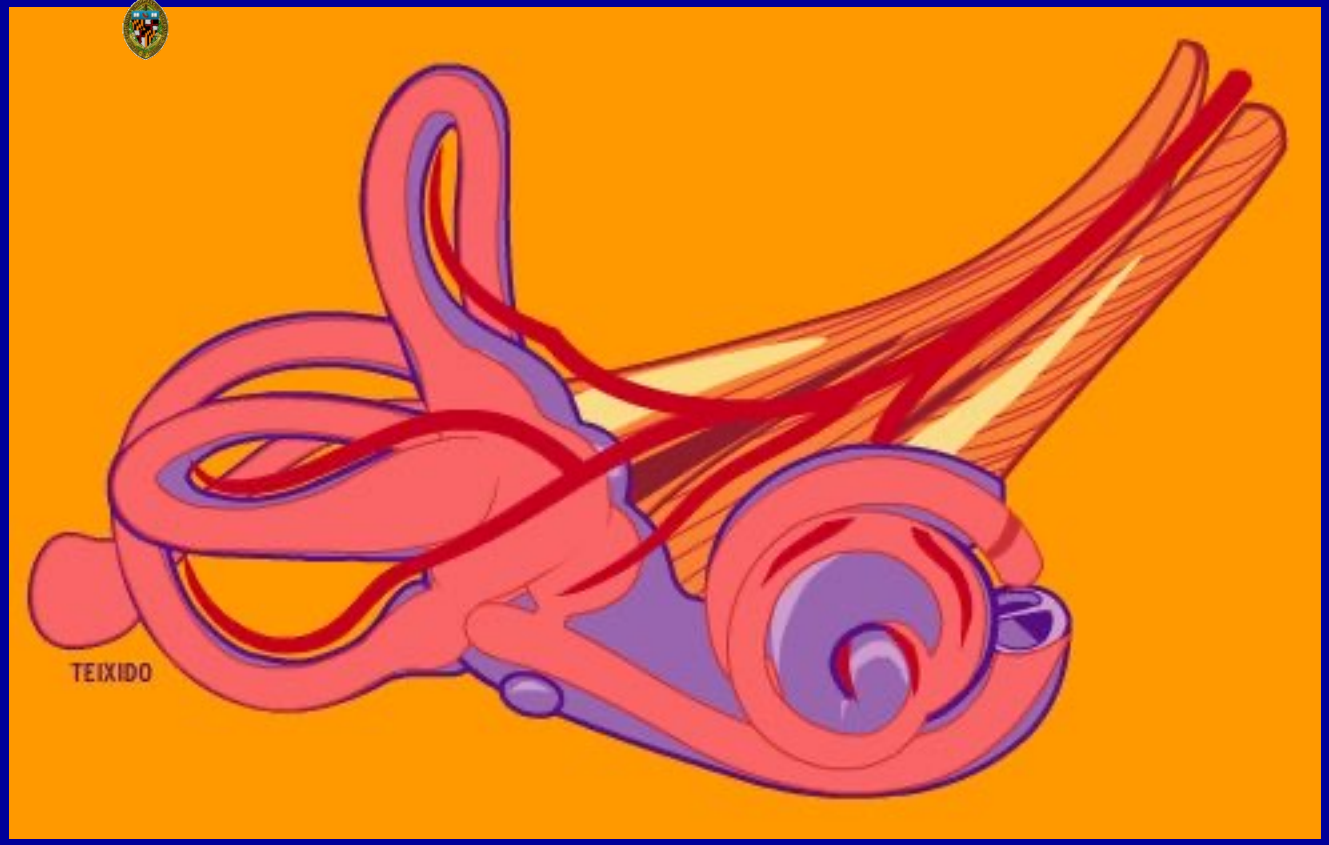
Migraine in the Inner Ear?

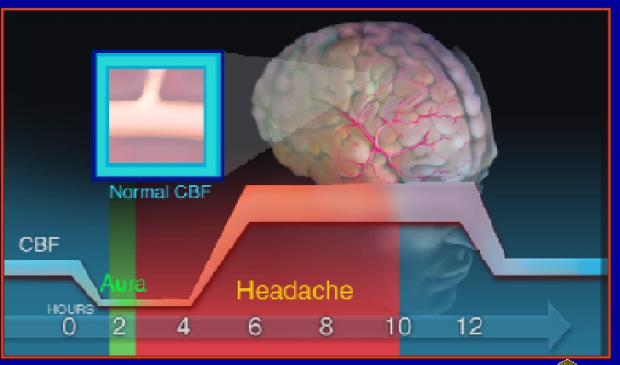


Migraine in the Inner Ear?



Migraine in the Inner Ear?





Migraine in the Inner Ear?

Migraine and Ménière's Disease

- The lifetime prevalence of migraine in patients with Ménière's disease is 56%, compared with 25% in controls ($p<0.001$).
- Ménière's attacks are accompanied by at least one migrainous symptom (headache, photophobia, or aura symptoms) in 45% always, and only sometimes in 11%.
    - Radtke et al. Migraine and Ménière's disease: Is there a link? *Neurology* 2002;59;1700-1704

Migraine and Ménière's disease

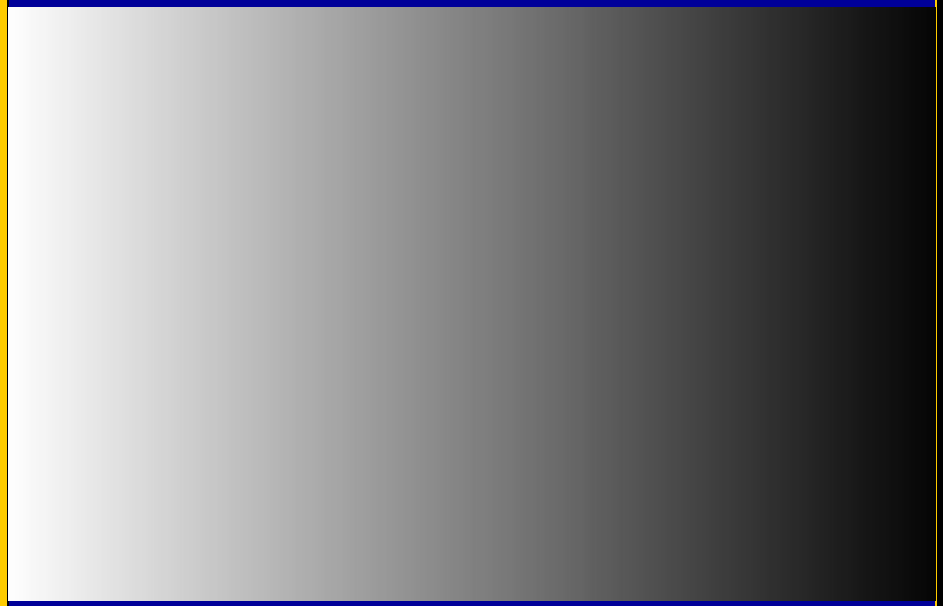
- Is this all coincidence?
  - Not likely!
- Perhaps there is a causal link between Ménière's disease and migraine.
- Some patients fit clear diagnostic criteria for one, the other, or both.
- But many patients are in a gray zone in between Ménière's disease and migraine.

The spectrum of disease

Vestibular Migraine:

1) At least 5 episodes with vestibular symptoms of moderate to severe intensity, lasting 5 minutes to 72 hours.
2) Current or previous history of migraine with or without aura according to the ICHD.
3) One or more migraine symptoms with at least 50% of the vestibular episodes:
4) The first headache with at least 2 of the following characteristics: one-sided location, pulsing quality, moderate or severe pain intensity, aggravation by routine physical activity.
5) Photophobia and phonophobia
6) Visual aura

Cephalalgia 33(9) 629–808, 2013.



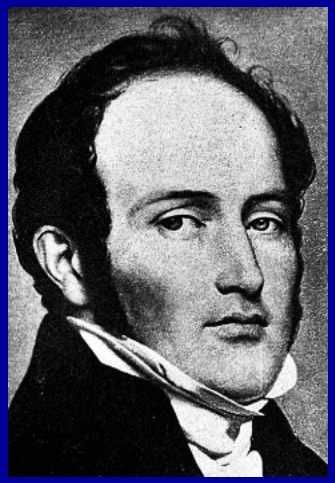
Ménière's disease:

1) 2+ spontaneous episodes of vertigo each lasting 20 minutes or longer
2) hearing loss documented by audiogram on at least one occasion
3) tinnitus or aural fullness in the affected ear
4) other causes excluded (AAO-HNS Criteria)

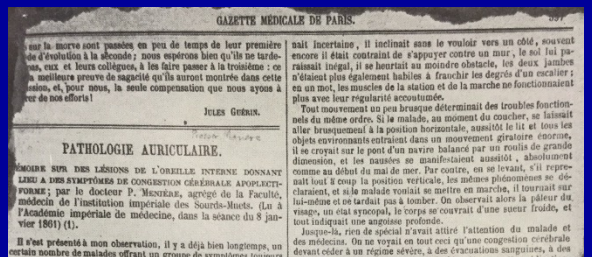
Migraine and Ménière's disease

- When migraine or migrainous vertigo and Ménière's disease co-occur, it is important to treat both.
- It is especially important to address migraine before proceeding to any ablative therapy for Ménière's disease.
- Many patients with Ménière's disease will find that their attacks are triggered by some of the typical foods that trigger migraine.

Ménière's disease and Migraine



"If it is incontestable that individuals who are prey to vertigo…syncope... and vomiting have at the same time head noises and rapidly become deaf, is it not less certain that cerebral states, called migraine, give place in the end to similar attacks, and the deafness which arises in these circumstances would seem to us inevitably to be related to a disease of the same nature."



GAZETTE MÉDICALE DE PARIS.

eur la morve sont passées en peu de temps de leur première de d'évolution à la seconde; nous espérons bien qu'ils ne tarde- nt, eux et leurs collègues, à les faire passer à la troisième : ce la meilleure preuve de sagacité qu'ils auroit montrée dans cette estion, et, pour nous, la seule compensation que nous ayons à cer de nos efforts!

JULES GUÉRIN.

PATHOLOGIE AURICULAIRE.

MÉMOIRE SUR DES LÉSIONS DE L'OREILLE INTERNE DONNANT LIEU A DES SYMPTÔMES DE CONGESTION CÉRÉBRALE APOPLECTI- FORME; par le docteur P. MENIÈRE, agrégé de la Faculté, médecin de l'institution impériale des Sourds-Muets. (Lu à l'Académie impériale de médecine, dans la séance du 8 jan- vier 1861) (1).

Il n'est présenté à mon observation, il y a déjà bien longtemps, un certain nombre de malades offrant un groupe de symptômes toujours

nait incertaine, il inclinait sans le vouloir vers un côté, souvent encore il était contraint de s'appuyer contre un mur, le sol lui pa- raissait inégal, il se heurtait au moindre obstacle, les deux jambes n'étaient plus également habiles à franchir les degrés d'un escalier; en un mot, les muscles de la station et de la marche ne fonctionnaient plus avec leur régularité accoutumée.

Tout mouvement un peu brusque déterminait des troubles fonction- nels du même ordre. Si le malade, au moment du coucher, se laissait aller brusquement à la position horizontale, aussitôt le lit et tous les objets environnants entraient dans un mouvement giratoire énorme, il se croyait sur le pont d'un navire balancé par un roulis de grande dimension, et les nausées se manifestaient aussitôt, absolument comme au début du mal de mer. Par contre, en se levant, s'il repre- nait tout à coup la position verticale, les mêmes phénomènes se dé- clairaient, et si le malade voulait se mettre en marche, il tournait sur lui-même et ne tardait pas à tomber. On observait alors la pâleur du visage, un état syncopal, le corps se couvrait d'une sueur froide, et tout indiquait une angoisse profonde.

Jusque-là, rien de spécial n'avait attiré l'attention du malade et des médecins. On ne voyait en tout ceci qu'une congestion cérébrale devant céder à un régime sévère, à des évacuations sanguines, à des

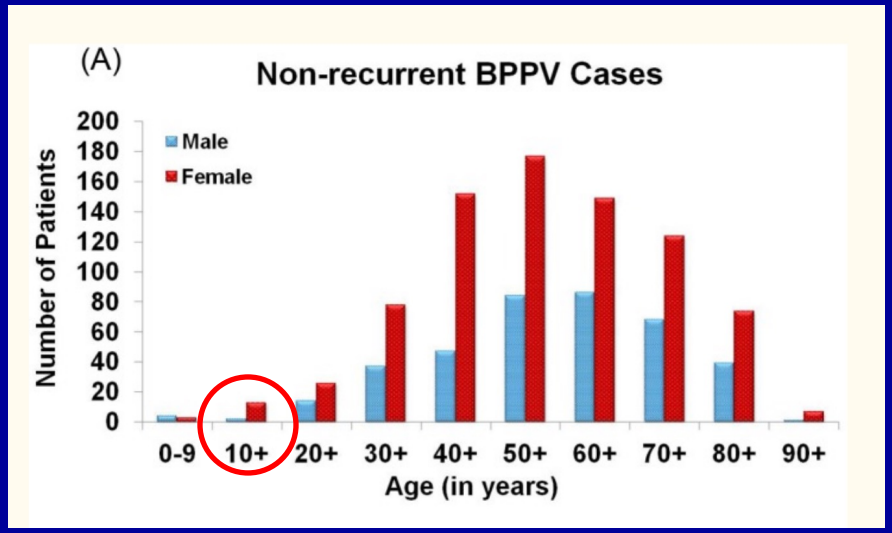
P. Ménière, Gaz Med de Paris, 1861: 16

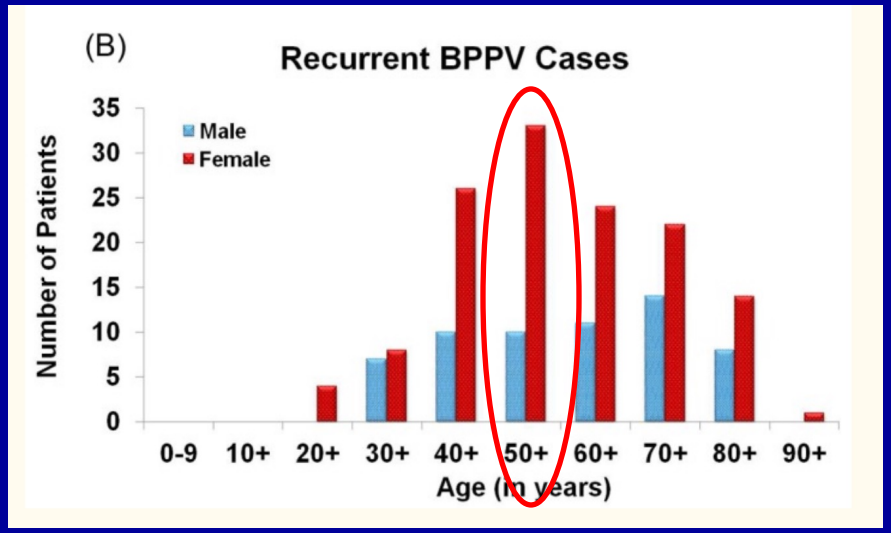
Migraine and BPPV

There is also a coincidence of migraine and BPPV

- Migraine is 3X more frequent in idiopathic BPPV than in BPPV secondary to head trauma or surgery.
  - Ishiyama A, Jacobson KM, Baloh RW. Migraine and benign positional vertigo. Ann Otol Rhinol Laryngol 2000;109:377–80.
- Migraine is 2X more prevalent in patients with BPPV than in age and sex matched controls.
  - Lempert T, Leopold M, von Brevern M, et al. Migraine and benign positional vertigo. Ann Otol Rhinol Laryngol 2000;109:1176.
- The odds ratio for BPPV in individuals with migraine is 7.5 (95% CI: 3.9–14.2) over age- and sex-matched controls.
  - von Brevern et al. Epidemiology of benign paroxysmal positional vertigo: a population based study. *J. Neurol. Neurosurg. Psychiatry* 2007;78;710-715
- BPPV affects females twice as often as males.
  - PubMed Health. 30 January 2014. Retrieved 25 July 2016

BPPV affects females twice as often as males- hormones may play a role
1,377 BPPV patients





33.3% reported headache immediately preceding first BPPV symptoms n=227

Hormonal fluctuations may increase the tendency to develop BPPV

6.8F:1M at puberty

3.2F:1M at menopause

Ogun OA, Janky KL, Cohn ES, Büki B, Lundberg YW. Gender-based comorbidity in benign paroxysmal positional vertigo. PLoS One. 2014 Sep 4;9(9)

Ogun OA, Büki B, Cohn ES, Janky KL, Lundberg YW Menopause and benign paroxysmal positional vertigo. Menopause. 2014 Aug; 21(8):886-9.

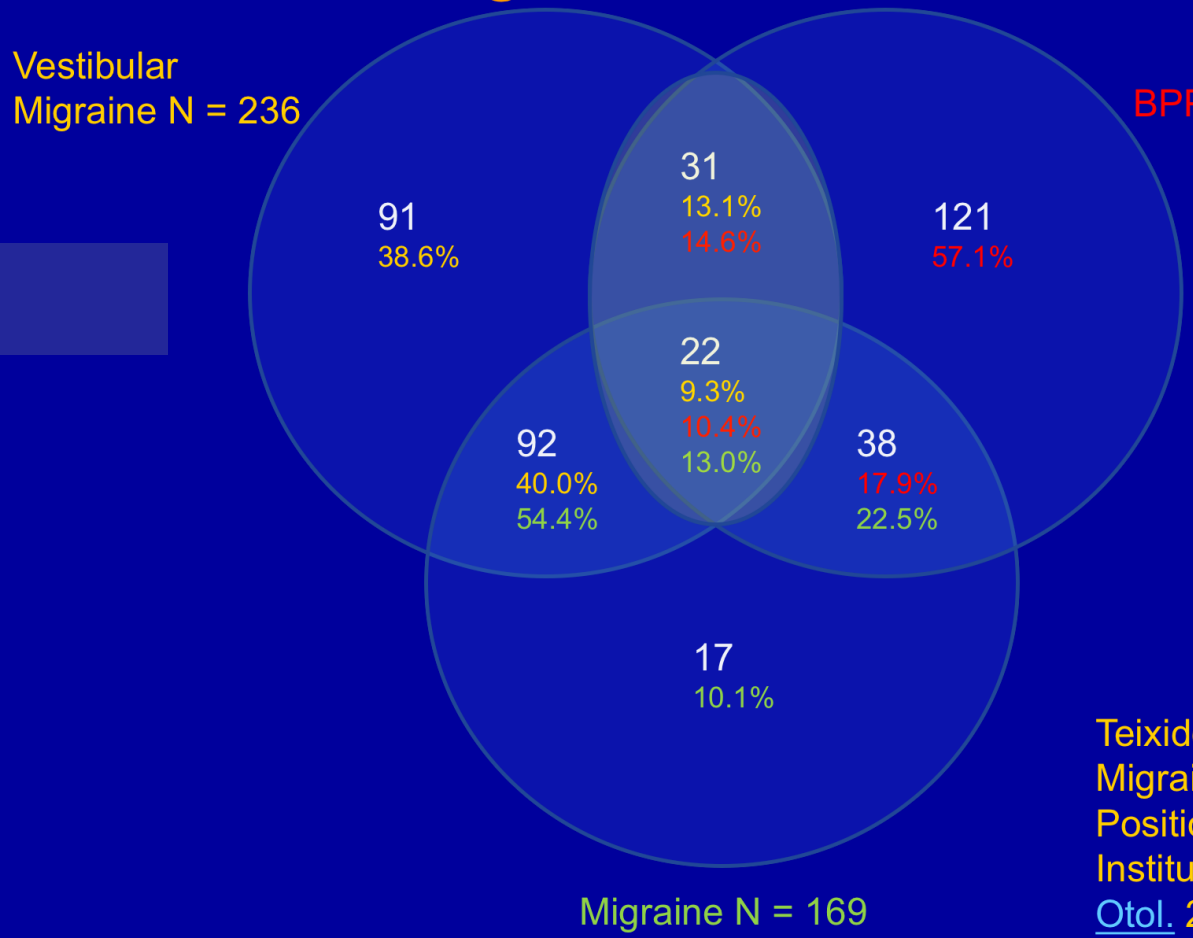
Boys Town National Research Hospital

Migraine and BPPV

- Can migraine cause BPPV?
  - Plasma extravasation or other peripheral inflammation could damage the utricle.
  - Release of otoconia could start BPPV.
- Migraine is associated with recurrent BPPV
  - Especially in young patients

Otolaryngologic Presentations of Migraine- Recurrent BPPV

The Intersection of Headache, Vestibular Migraine and BPPV in 500 pts



Teixido MT, Baker A, Isildak H, Migraine and Benign Paroxysmal Positional Vertigo: A Single Institution Review, J Laryngol Otol. 2017 Jun;131(6):508-513.

Symptom Variability in Migraine Cortex +/- Vessels +/- Brainstem

- The full package
  - Aura + Pain + Brainstem symptoms
- Other packages:
  - Aura, pain, or brainstem symptoms alone
  - Atypical pain, pressure, or fullness
  - Parasympathetic symptoms
    - congestion, GI, dysautonomia
  - Fluctuating or constant symptoms
  - Any region with trigeminal or parasympathetic innervation
    - ear, sinuses, eye, heart, blood vessels, gut

Symptom Variability in Migraine

- Headaches may become less severe over time.
- Neurological symptoms may become more prominent
- IHS criteria – which are research criteria - are not flexible enough to encompass all of the presentations that otolaryngologists are likely to see.

Neuhauser H, Leopold M, von Brevern M et al. The interrelations of migraine, vertigo, and migrainous vertigo. Neurology 2001;56:436-441

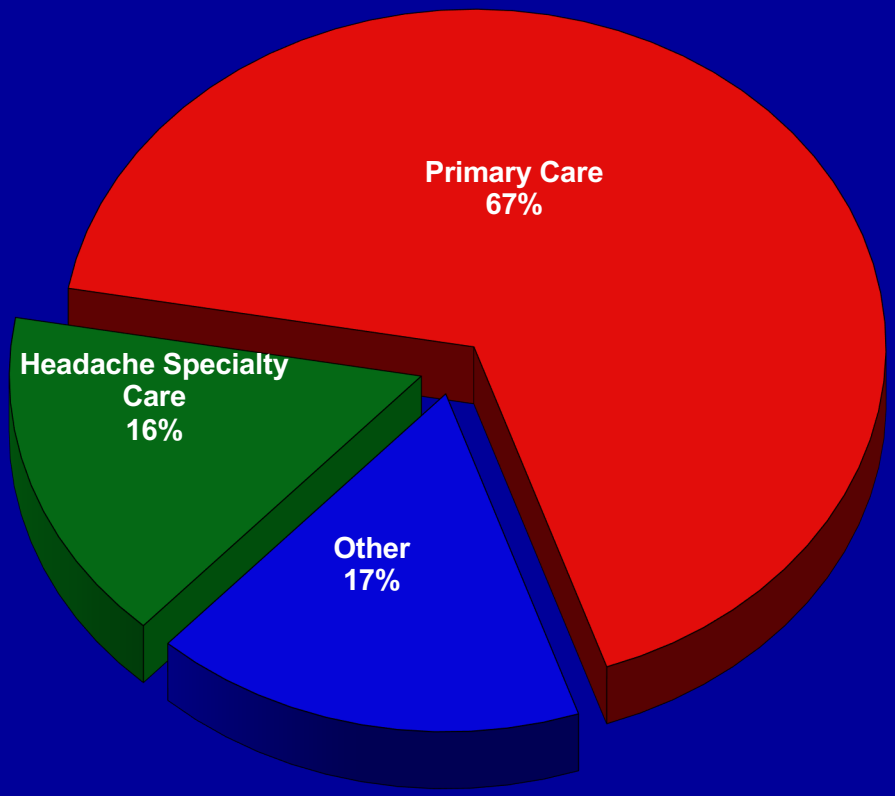
Kelman L, Migraine changes with age: IMPACT on migraine classification. Headache. 2006 Jul-Aug;46(7):1161-71.

So, how can I be sure it's migraine?

Maintain a high degree of suspicion in the patient with:

- New onset dizziness, motion sickness concurrent with headache activity
- Pattern of symptoms not fully explained by BPPV, Ménière's d., neuronitis
- Associated neurological symptoms – e.g.-sinus headache with vertigo
- No hearing loss
- Triggerability
- Past history of migraine
- Family history of migraine or symptoms similar to the patient's

WHERE DO MIGRAINE SUFFERERS SEEK MEDICAL CARE?



General Practice

Internal Medicine

Pediatrics

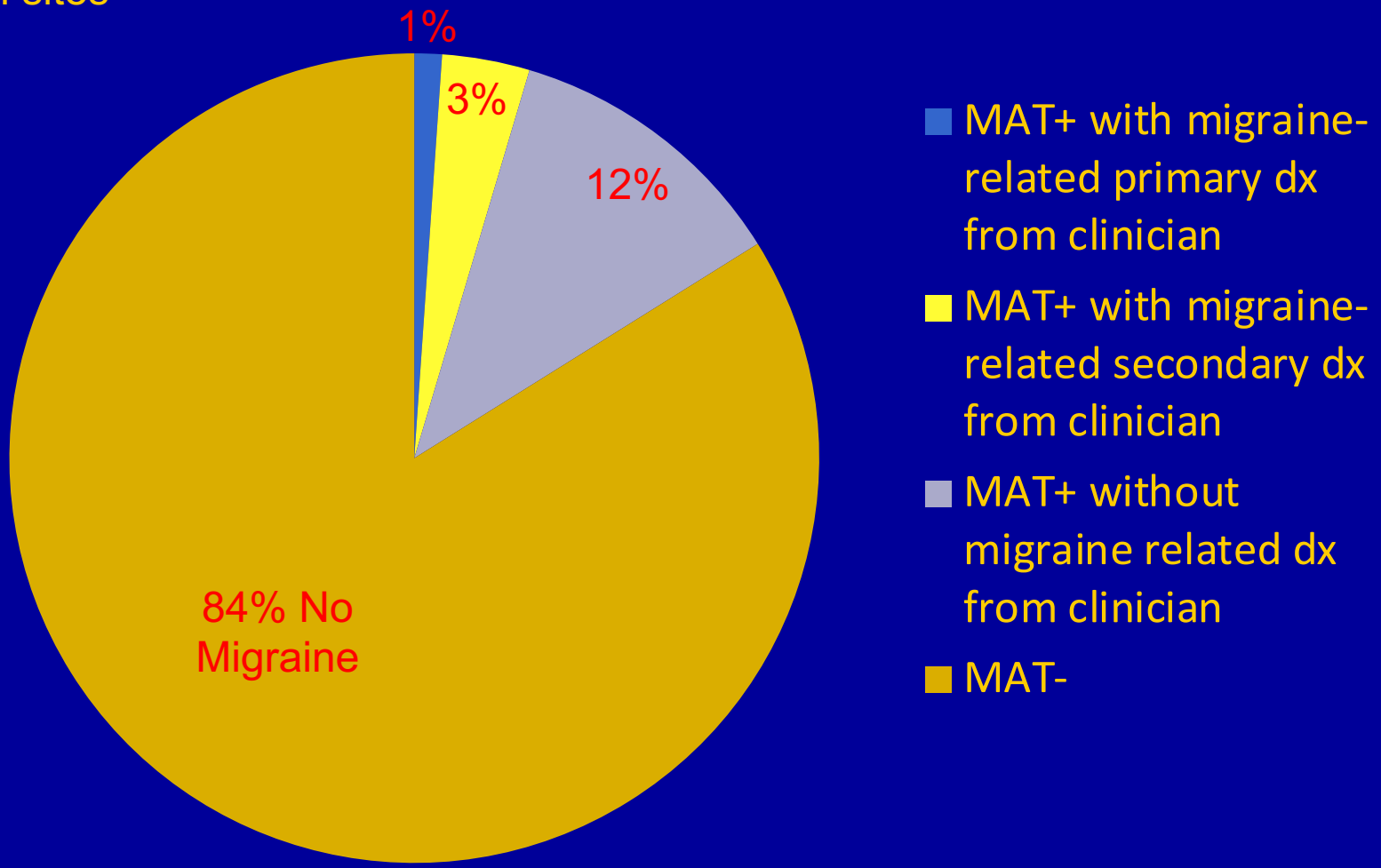
Neurology

Lipton RB et al. *Headache*. 1998.

Patterns of Migraine Disease in Otolaryngology: A CHEER network study
Schulz , Esmati, Godley, Hill, Monfared, Teixido, Tucci, Witsell OTO-HNS 2018 July

1458 Patients Randomized at 14 clinical sites

Distribution of Study Patients



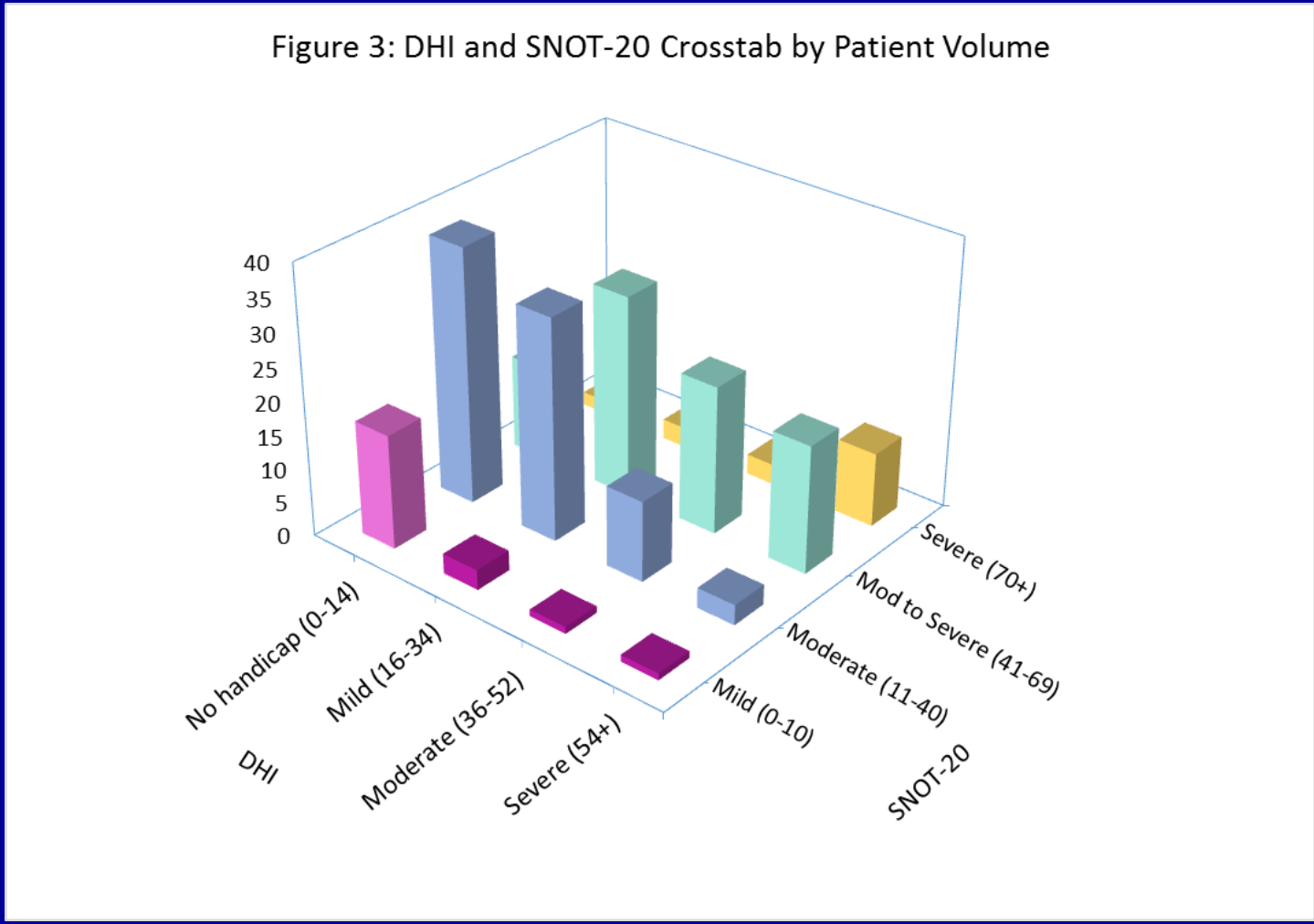
16% of ENT pts have migraine / 28.6% have chronic migraine

12- month prevalence among adults

- Migraine: 11.5%
- Probable migraine: 7%,
- Total combined migraine + PM: 18.5%.
- Migraine with aura: 4.4%
- Chronic migraine: 0.5% (**4.7% in ENT**)
- Tension-type headache: 13%

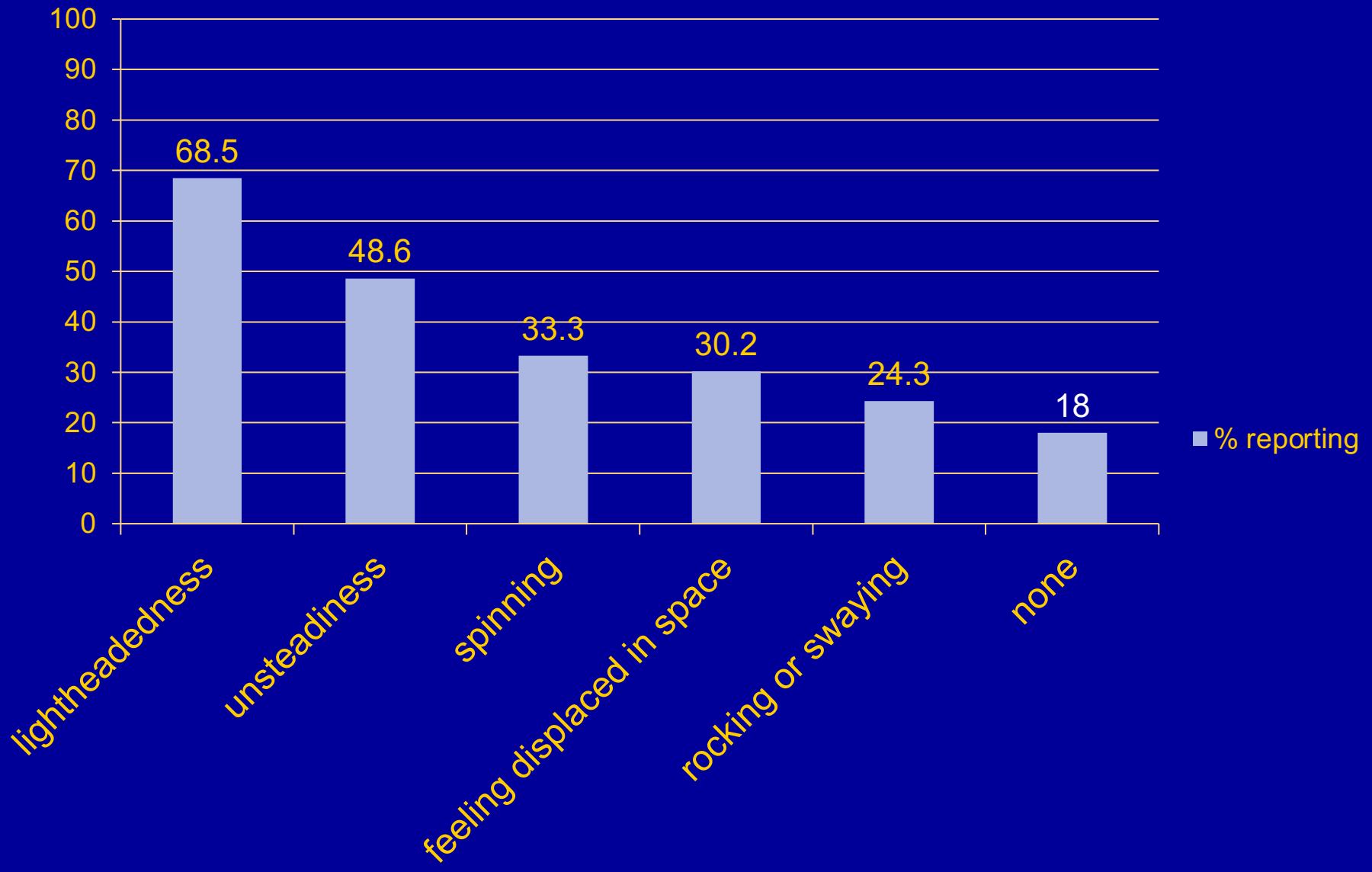
Merikangas KR. Contributions of epidemiology to our understanding of migraine. Headache. 2013;53(2):230-46.

Sinus and Dizziness symptom burden are highly correlated in migraineurs n=235



Patterns of Migraine Disease in Otolaryngology: A CHEER network study
Schulz , Esmati, Godley, Hill, Monfared, Teixido, Tucci, Witsell 2017

Vestibular Symptoms in MAT+ Patients



Patterns of Migraine Disease in Otolaryngology: A CHEER network study
Schulz , Esmati, Godley, Hill, Monfared, Teixido, Tucci, Witsell 2017

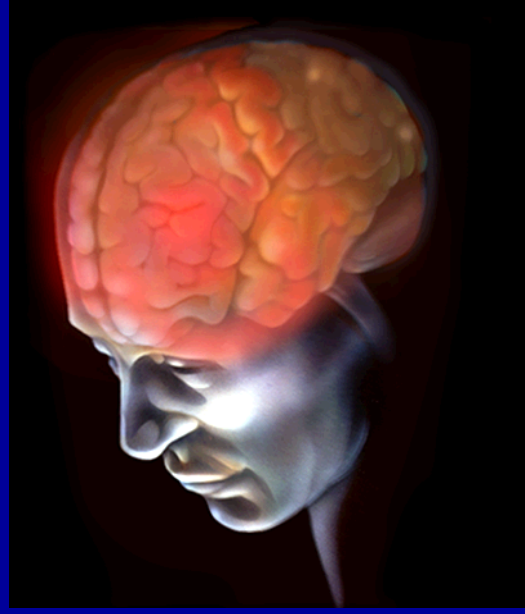
Vestibular Migraine Treatment

Buy - In

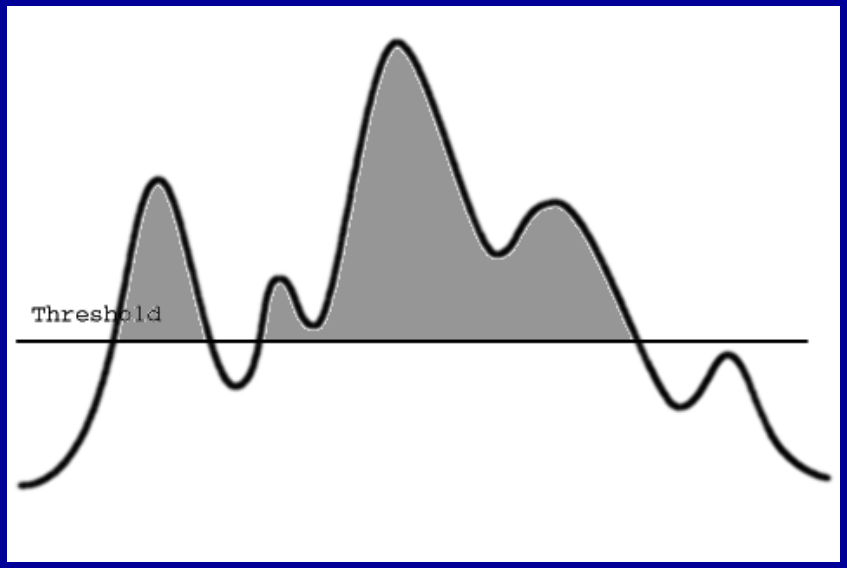
- Our goal is to convince these patients –and doctors - that migraine does not equal headache; rather, that migraine is a neurologic syndrome. Patient handout at: http://www.entad.org/wp-content/uploads/2016/03/Migrainehandout.pdf
- Headache is certainly the most common manifestation, but it is not the only one.
- Other manifestations might include:
  - Sinus congestion, sinus headache
  - Dizziness: spontaneous vertigo, recurrent BPPV, MD
  - Sudden hearing loss, hyperacusis, fluctuating tinnitus
  - Otalgia, aural pressure, atypical facial pain

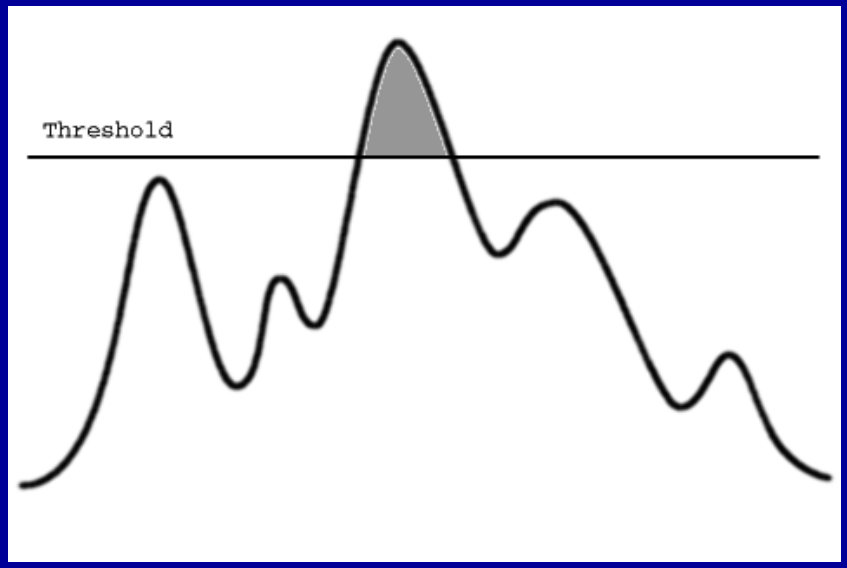
VM Treatment

Testing the Migraine Hypothesis



- Trigger reduction
- Threshold elevation
- Event management





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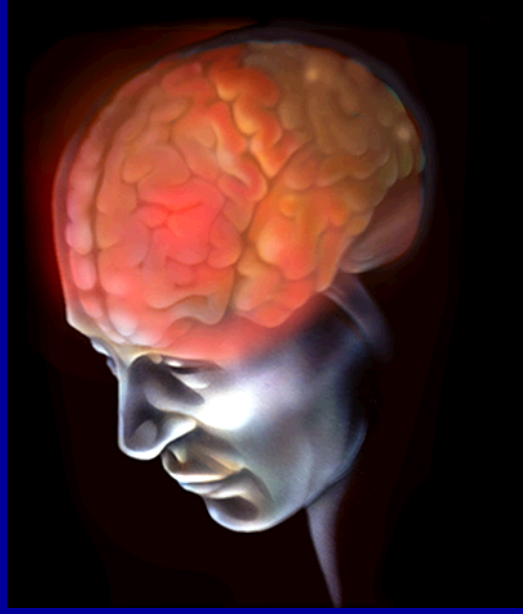


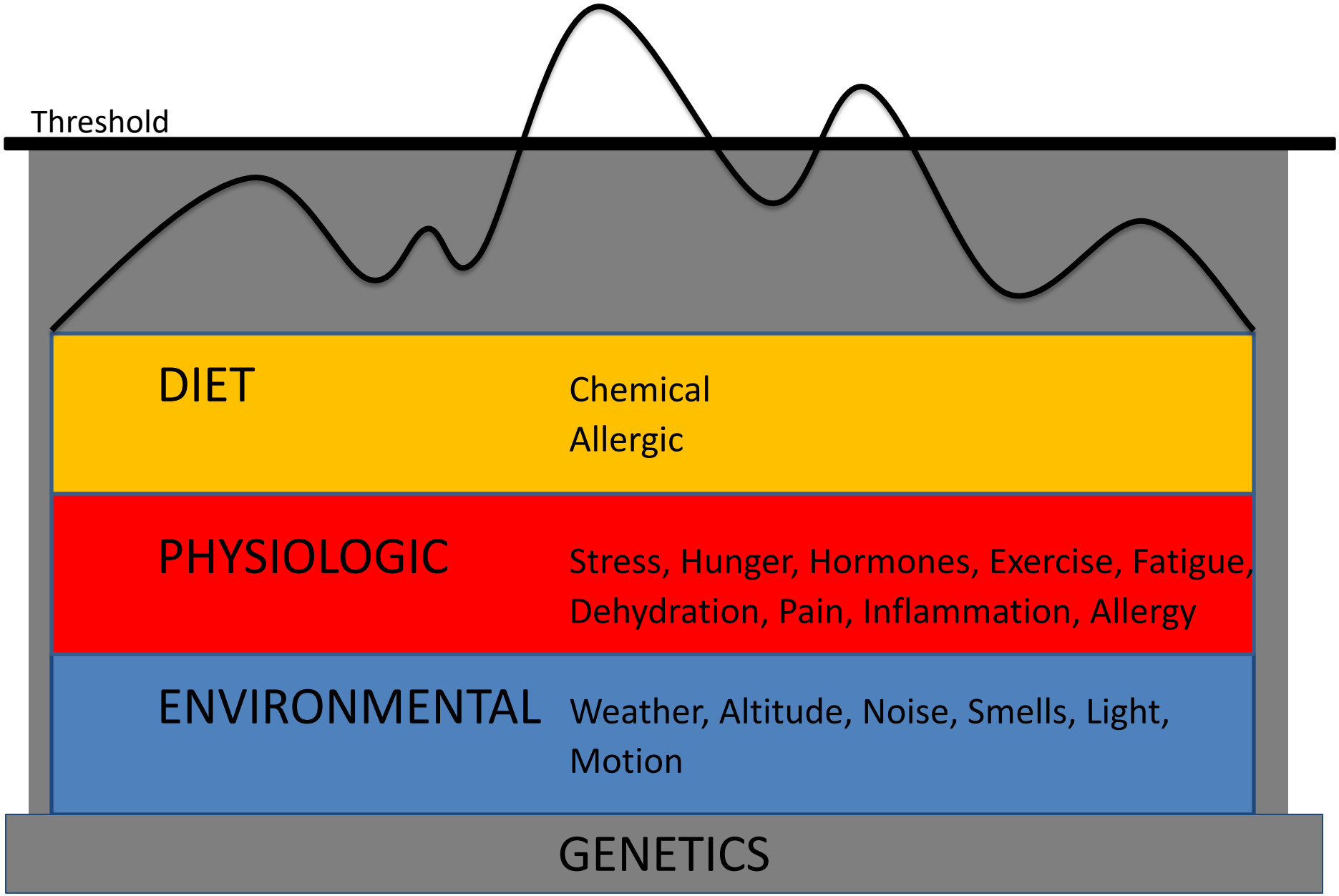
- Education
- Teamwork
- Trigger identification and avoidance
- Migraine prophylaxis usually needed
- Abortive therapy-
  - Steroids/ promethazine
  - Migraine abortive agents not useful
- Lifestyle hygiene
- PT can be helpful in some cases
- Cycle Breaking Procedures- ie, Botox, blocks generally not useful

Vestibular Therapy for VM

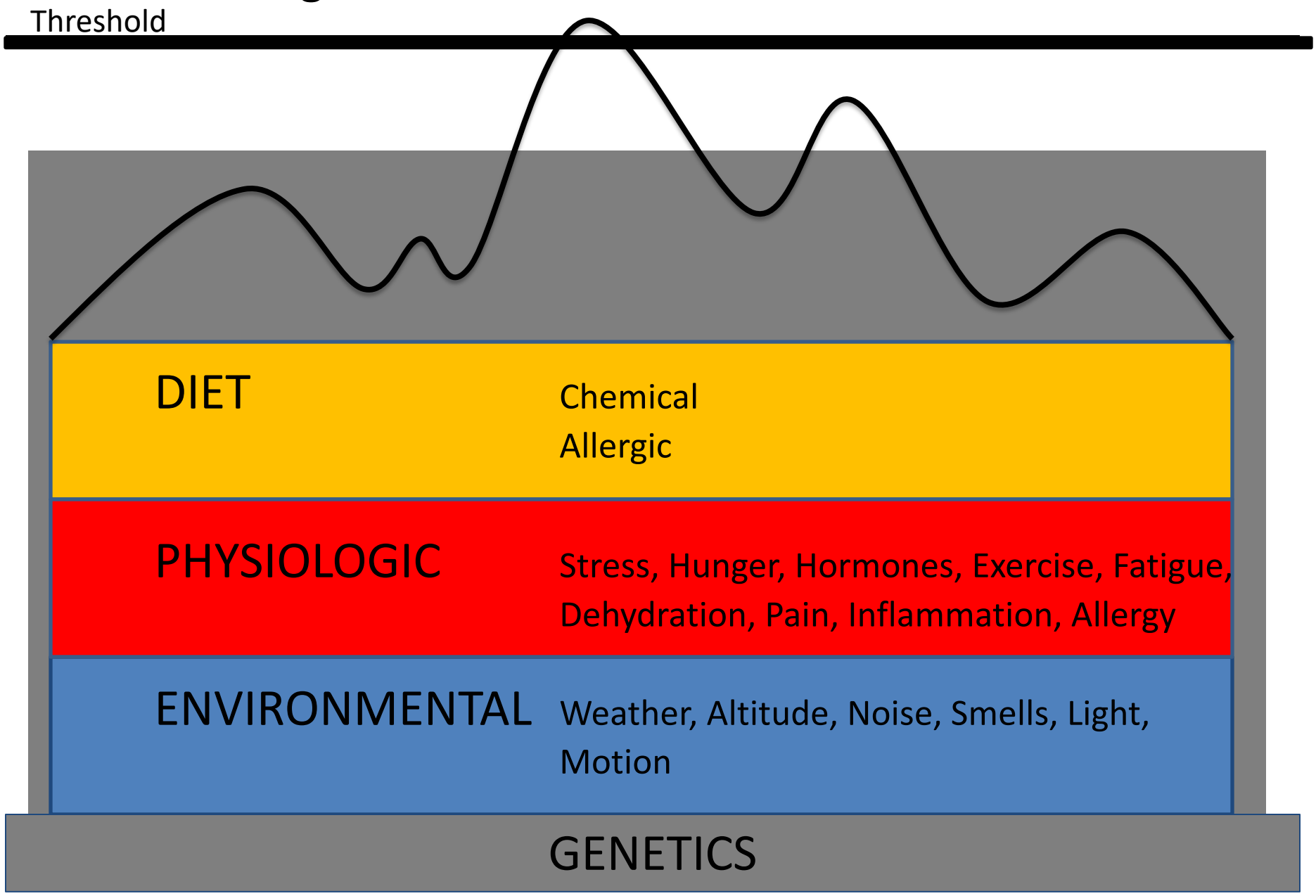
- Mixed results in 6 studies
- VM patients do sustain vestibular injury
  - uncompensated weakness/impaired compensation ability
  - BPPV
- Theoretically most effective when combined with chronic headache management
- May help patients adapt to presence of symptoms

Alghadir A, Anwer S. The Effects of Vestibular Rehabilitation in the Management of Vestibular Migraine: A Review. Frontiers in Neurology. 2018;9:article 440

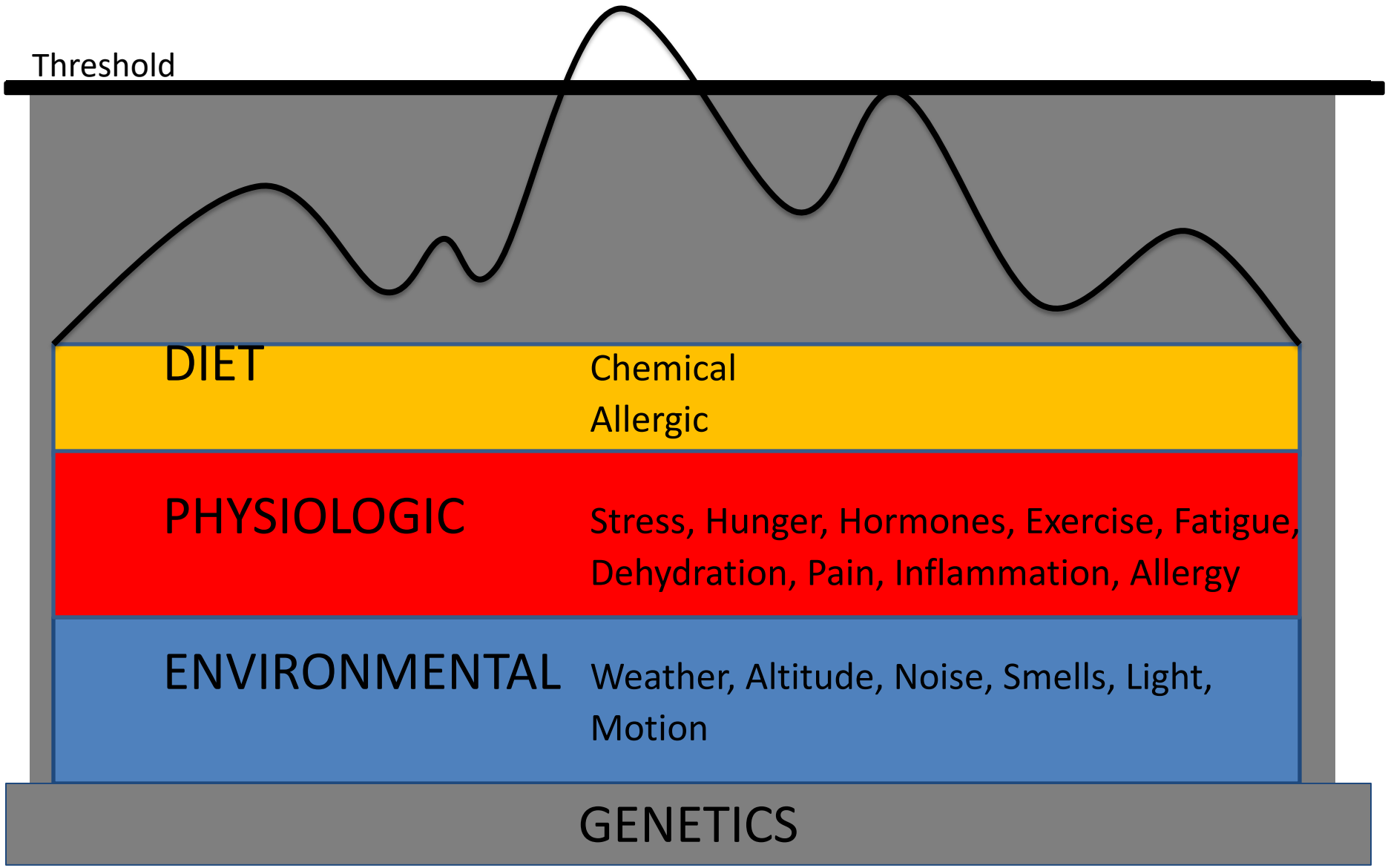
A Migraineur: Partial Triggers and Susceptibilities



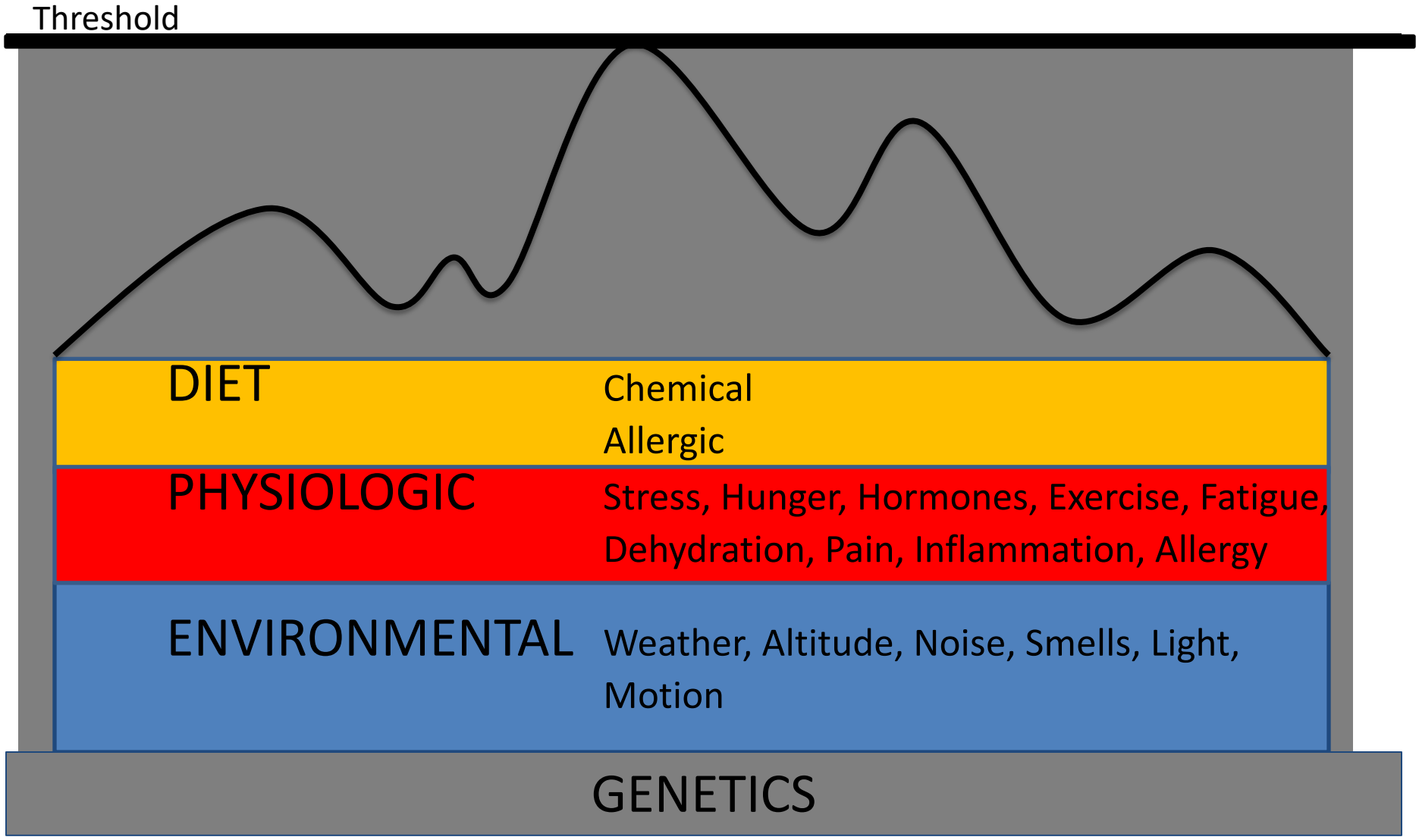
A Migraineur: Medical Threshold Elevation



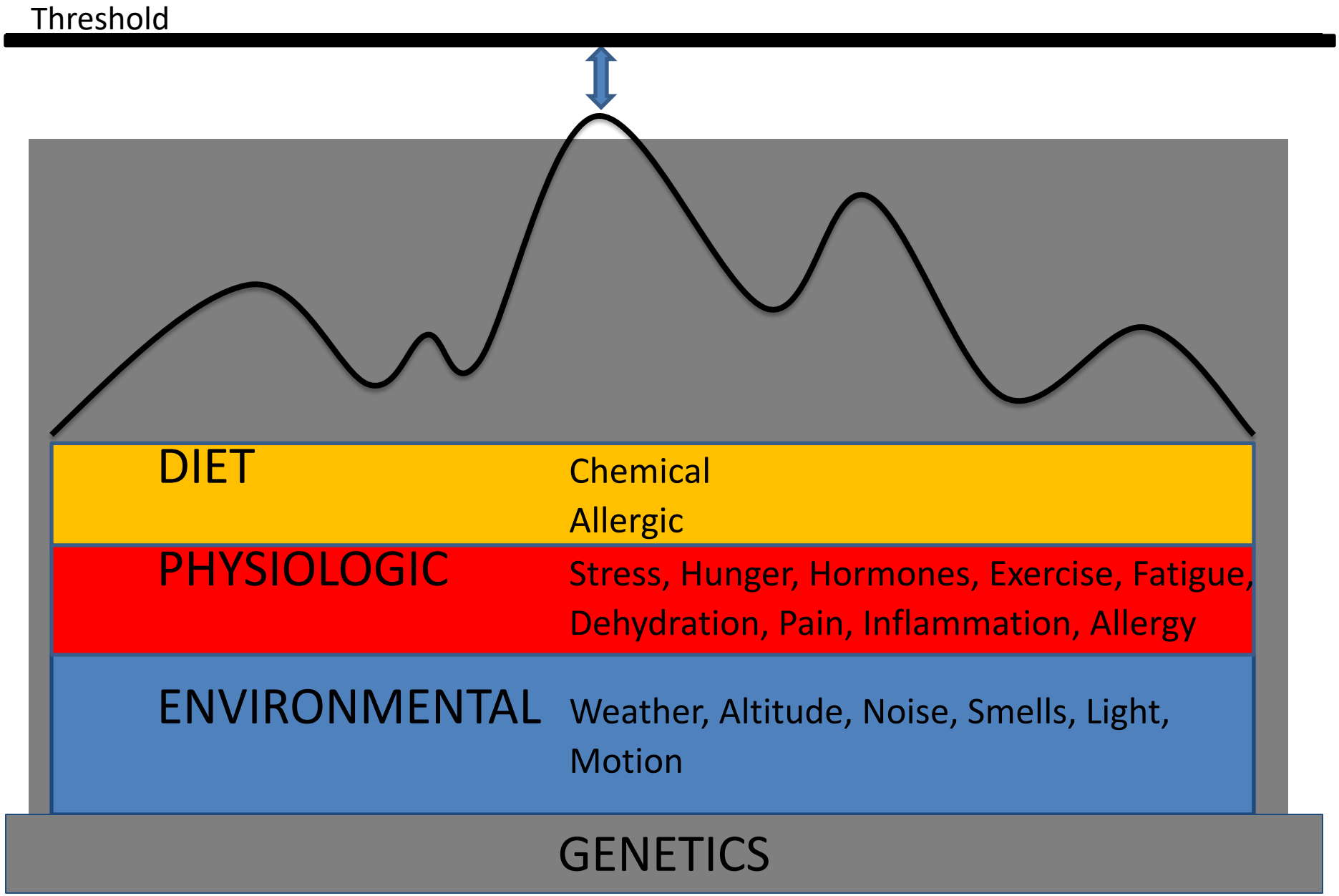
A Migraineur: Reducing Dietary Triggers



A Migraineur: Reducing Dietary and Physiologic Triggers



A Well Controlled Migraineur



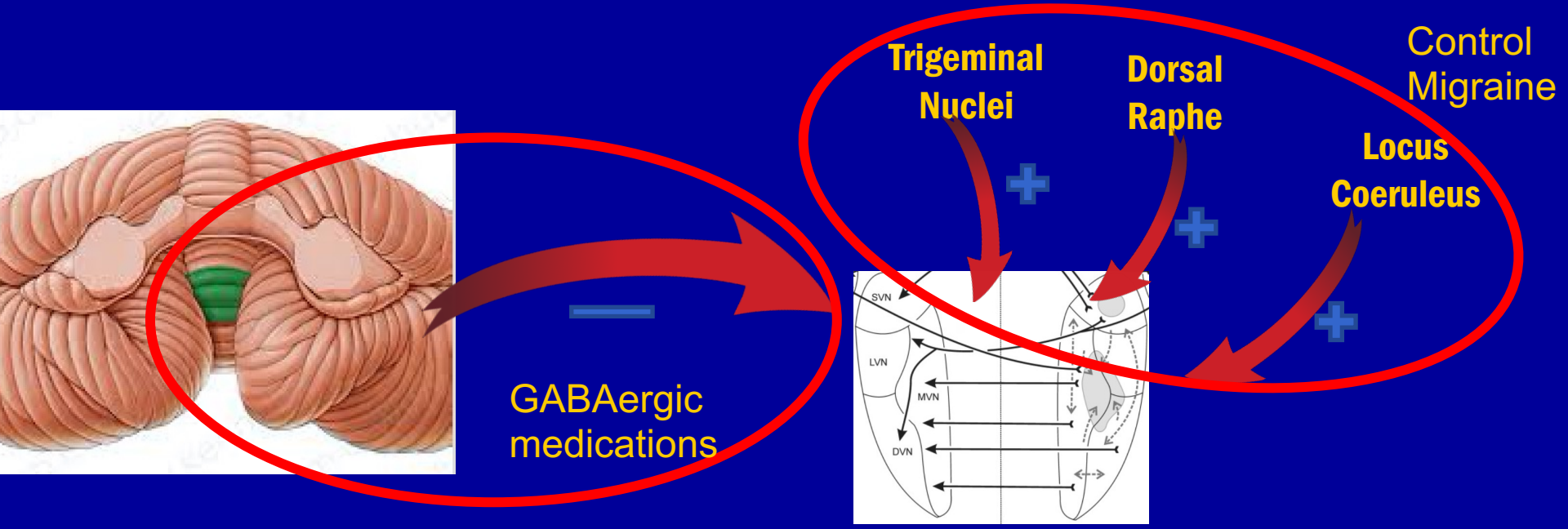
VM Treatment
Trigger Identification and Avoidance

- Highly effective even in severe/refractory cases
- Requires patient education and motivation
- Migraine diary
- Safe
- Associated with neck sx – treat neck
- Associated with new TMJ sx – treat TMJ etc.

Migraine Prophylaxis- When to Use

- Useful when symptoms are frequent, disabling, or so brief abortive measures cannot be
- Daily medications usually well tolerated
- Select drugs based on co-existing medical conditions
- Response may take up to 6 weeks
- Patients should continue trigger identification and avoidance
- Goal- decrease frequency/severity by 50-70%
- Can be used for hypothesis testing in individual patients

Optimum Treatment of VM: Enhance Cerebellar Inhibition Decrease Vestibular Nucleus Sensitization



A range of patient states may be determined by the relative contributions of inhibitory (cerebellar) and excitatory (brainstem) projections to the vestibular nuclei

King, Priesol, Davidi, Merfeld, Lewis, **Self-motion perception is sensitized in vestibular migraine: pathophysiologic and clinical implications**
Sci Rep. 2019 Oct 4;9(1):14323.

Well Tolerated Medications for Migraine Prophylaxis

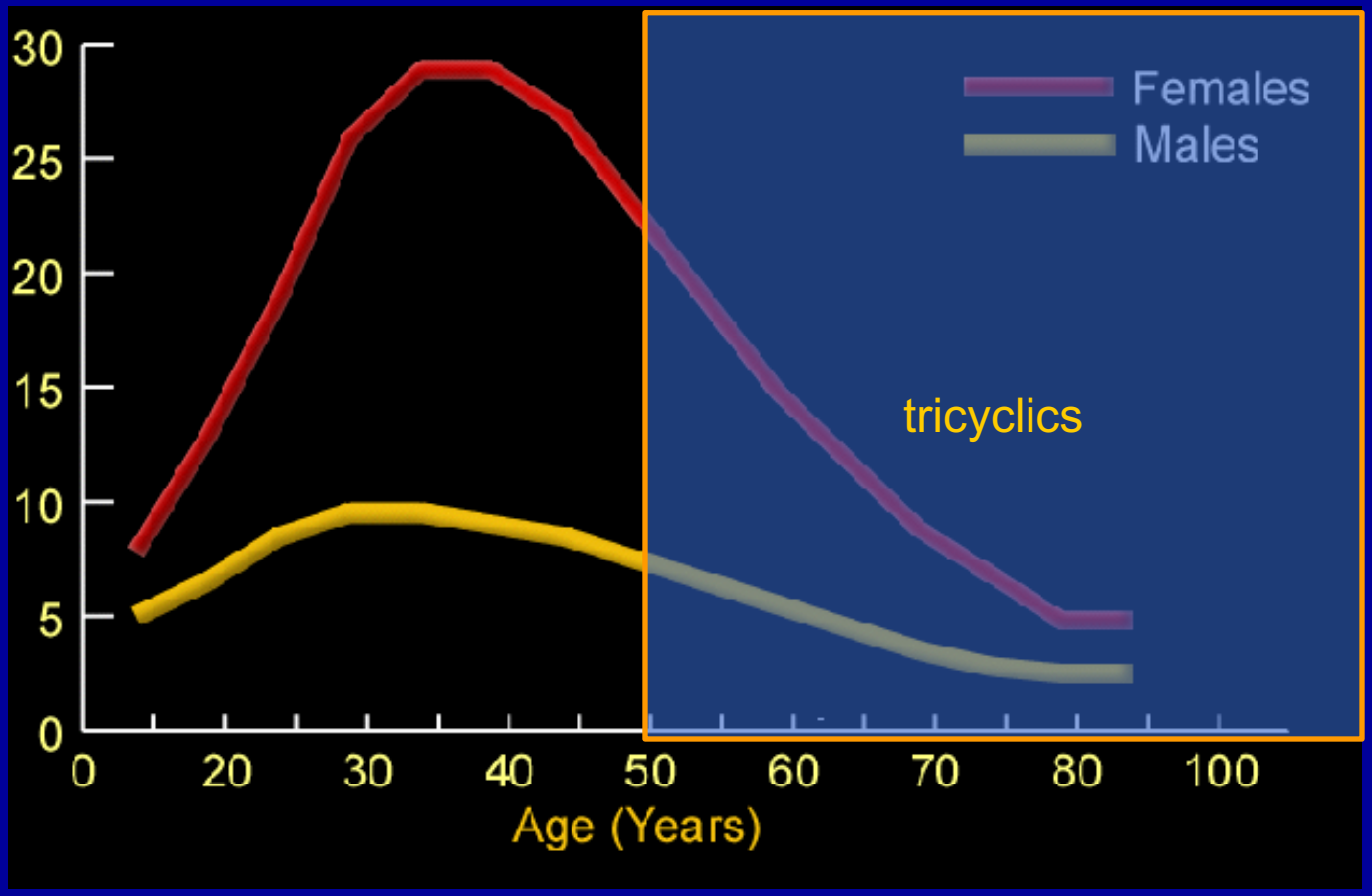
- Beta-blockers: propranolol, propranolol SR
- Calcium channel blockers: diltiazem, verapamil
- Tricyclic antidepressants(Ca/Na blockers): nortriptyline, amitriptyline
- SNRIs- venlafaxine
- Anticonvulsants(Na blockers): valproic acid, topiramate, pregabalin (Lyrica)
- Carbonic anhydrase inhibitor: acetazolamide
- If family pattern of disease use medications effective for family members

Medications for Migraine Prophylaxis Tricyclic Antidepressants

- Nortriptyline – safe / effective
- Serotonin-norepinephrine reuptake inhibitor (SNRI)
- Inhibits sodium channels and L-type calcium channels
- Anticholinergic
- Improves sleep
- Often good response at low doses (20mg)
- 10mg HS x 7d then 2 HS.
  Increase over weeks as needed/tolerated to 50 mg.
  Some need up to 150 mg.
- Dry mouth, weight gain at higher doses
- If morning sedation take HS dose earlier in evening
- Amitriptyline- similar dosing and side effects
  - Tablet- can be divided; useful in some patients with extreme sensitivity to medications

Medications for Migraine Prophylaxis Tricyclic Antidepressants

- Work best for patients 50+



Medications for Migraine Prophylaxis Anticonvulsants

Topiramate (Topamax)

- Anticonvulsant and carbonic anhydrase inhibitor
- Good evidence for efficacy as 1st line agent
- Slow dose escalation needed
- Cognitive side effects can be limiting
- Start 25 mg daily, then BID, then increase weekly to 100 BID.
- Risks include kidney stones, rare form of glaucoma
- Weight loss a plus!
- Best starting drug for young women
- New long acting formulation (Trokendi)

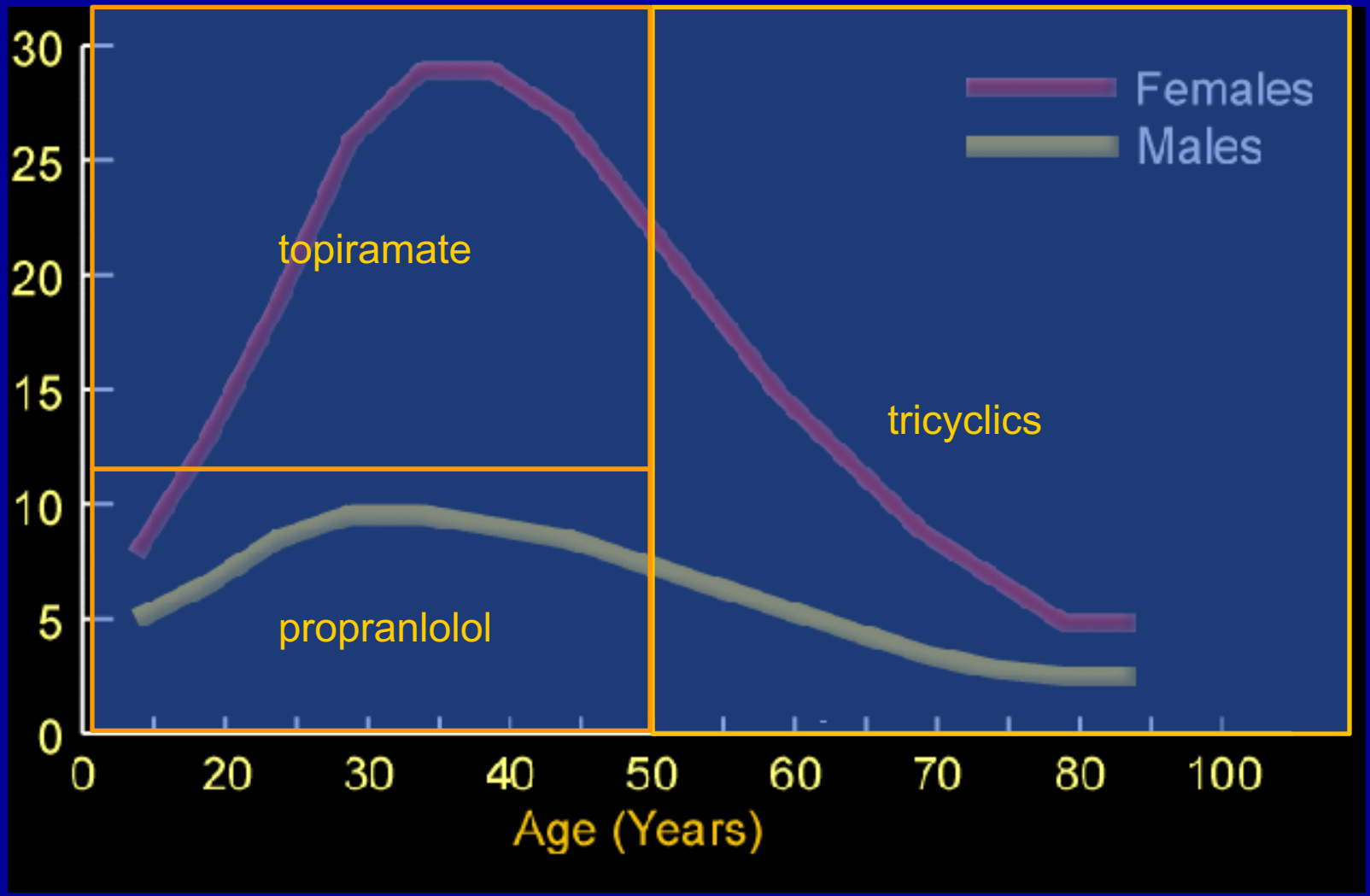
Medications for Migraine Prophylaxis Beta-blockers

- Do not use with asthma, diabetes
- Propranolol LA 60-120mg/day starting dose
- Increase as needed to 240mg/day
- May worsen depression
- Exercise limitation
- Well tolerated by younger men

Medications for Migraine Prophylaxis Calcium Channel Blockers

- Well tolerated often used as first line
- Diltiazem CD 120mg starting dose
- Increase as needed to 240-480 mg/day
- Divide dose at higher doses
- Constipation, hypotension(rare)
- Verapamil often cheaper but tid dosing

Medications for Migraine Prophylaxis



Ca blockers a good second line for all / Any drug can be used as tolerated

Testing the Migraine Hypothesis: Treatment Navigation

- Trigger identification and avoidance
- Diet
- Nortriptyline, propranolol or topiramate
- Diltiazem
- Venlafaxine
- Dual therapy (cannot take Ca and beta blocker together)
- Clonazepam

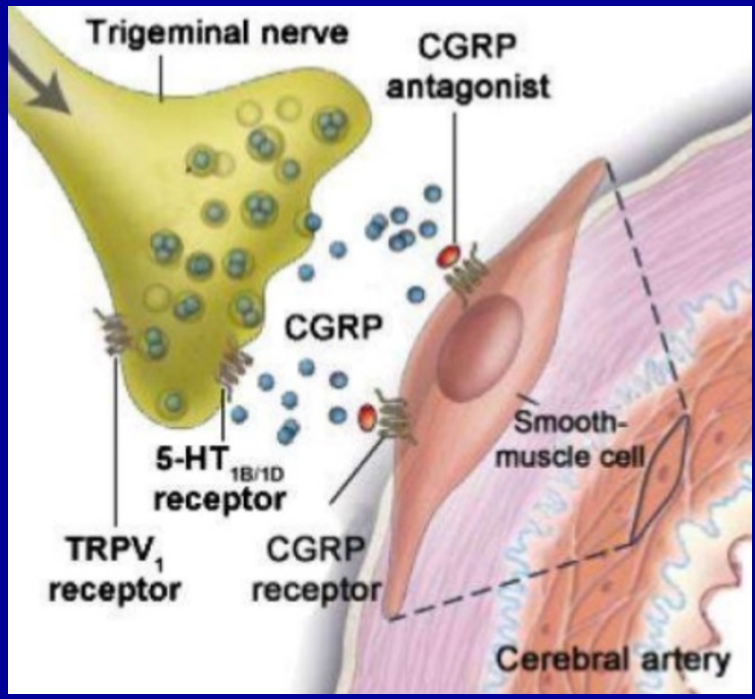
Rocking Dizziness

- Highly associated with migraine
- Mal de debarquement syndrome is a form
- Uniquely responsive to PT(OKN retraining)
- Responds to concurrent migraine management
- Clonazepam helpful-0.25-0.5mg BID
- Venlafaxine helpful(Effexor XR) 37.5mg
  - Increase by 1 tab weekly to 150mg

New Drugs

- CGRP receptor antagonists- first migraine specific treatment, monoclonal antibody, monthly IM injection, $8500/yr, 2018
  - Galcanezumab (Eli Lilly)
  - Erenumab (Amgen/Novartis)
  - Fremanezumab (Teva)
  - Eptinezumab (Alder)
- May be used with traditional preventives

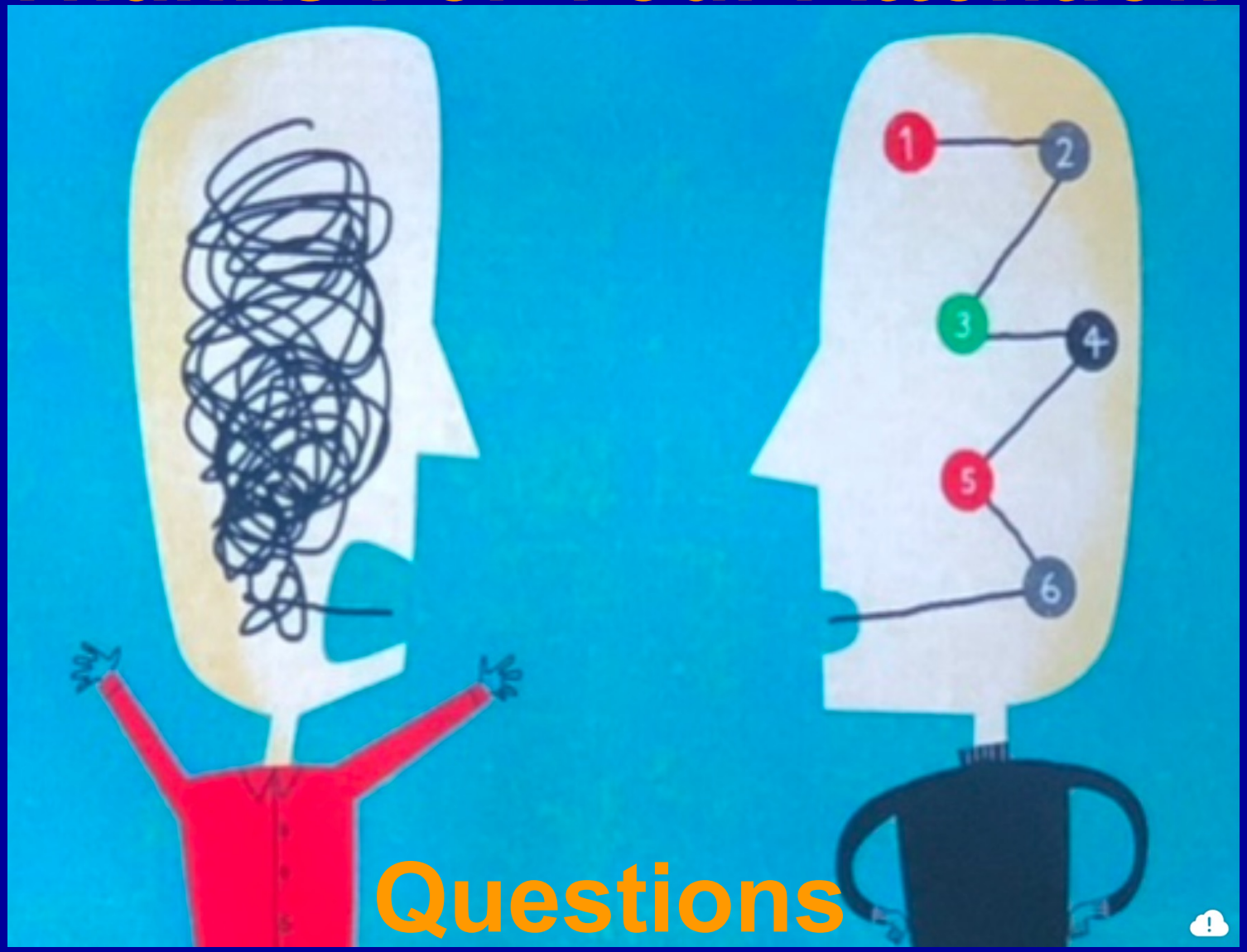
No information on utility in VM



Summary

- Vestibular Migraine is a common clinical problem
- Defined neurovascular mechanisms are responsible for symptom generation
- Many atypical presentations of dizziness are vestibular migraine
- Otolaryngologists should be comfortable initiating migraine therapy

Thanks For Your Attention



Questions