Migraine on the Mind: When the Brain and Eyes Collide

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Course Objectives

1. Understand the pathophysiology and neurovascular mechanisms underlying migraine headaches, including their impact on visual function and ocular health.
2. Recognize the diagnostic criteria and clinical manifestations of migraines, emphasizing the role of optometry in differentiating between primary and secondary headache disorders.
3. Develop comprehensive treatment plans for migraine patients, integrating optometric interventions, pharmacological therapies, and multidisciplinary approaches to optimize patient outcomes and quality of life.

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Warning

- As optometrists, we *do not* diagnose migraine(s), however, we may have patients that suffer from migraines that require us to refer to other providers (neurology/PCP/pediatricians/PMR etc) to further workup or diagnose our patients
- Migraines have visual symptoms
- Migraines can be influenced by visual tasks
- Migraine-photophobia may be manageable by optometrists
- Migraine is related to dry eye

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INTRODUCTION

Epidemiology and Prevalence of Migraine

- One of the most common neurological diseases worldwide and a leading cause of disability
- Global prevalence 1.1 billion in 2019
  - Increasing from 721.9 million in 1990
- Prevalence in US (2024):
  - 11.7-14.7% overall
  - 17.1-19.2% in women
  - 5.6- 7.2% in males
- Prevalence is higher in females than men
- Highest incident rate in age group 10-14 years old, peaked in 30-34 age group and then declined

Cohen et al. Headache 2024, Safiri et al. Pain 2022., Steiner et al Headache 2020., Goadsby Physiol Rev. 2017

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INTRODUCTION

Thoughts on "headache"

- One of the most common physical complaints seen in medical practices
- The most common physical complaint reported after concussion
- Most headaches are not dangerous or ominous but negatively quality of life
  - Leading cause of disability and time missed from work

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INTRODUCTION

What's the difference between a headache and a migraine?

- Headache" is one of many symptoms of migraine
- A migraine is a type of headache
- Not all headaches are migraines
- And not all migraine have headache

- Many patients may tell you they have "migraines" and don't
- Many patients may have triggered symptoms (nausea, dizziness) and not realize they have migraines

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INTRODUCTION

Headache Subtypes

- Migraine
- Tension Headache
- Cluster Headache
- Sinus Headache
- Oculomotor Headache
- Neuritic or Neuralgic Headache
- Musculoskelatal
  - Myofascial
  - Temporomandibular Joint Dysfunction
  - Cervicogenic
- Medication-related Headache (Medication overuse/Medication-induced)

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Headache Classification

Primary Headache

IHS Classification ICHD-3

Primary Headache Subtype

- Migraine
- Tension-Type Headache
- Trigeminal Autonomic Cephalgia
  - Cluster Headache
  - Paroxysmal Headache
  - Short-lasting unilateral neuralgiform headache (SUNCT/SUNA)
  - Hemicrania Continua

- "Other" Primary headaches
  - Cough
  - Exercise
  - Sexual Activity
  - Thunderclap
  - Cold-stimulus
  - External pressure
  - Stabbing
  - Nummular
  - Hypnic

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Headache Classification

Secondary Headaches

IHS Classification ICHD-3

- Trauma to head/neck
  - Whiplash
  - Craniotomy
- Cranial or Cervical Vascular Disorders
  - Cerebral ischemic event
  - Non-traumatic intracranial hemorrhage
  - Unruptured vascular malformation (aneurysm, AVM, etc)
  - Arteritis (GCA)
  - Cervical carotid or verternal artery disorder
  - Cranial venous disorder
  - Pituitary apoptexy
- Non-vascular intracranial disorder
  - Increased/Decreased CSF pressure
  - Non-infectious inflammation
  - Intracranial neoplasia
  - Epileptic seizure
  - Chiari Malformation
- Substance or withdrawal
  - NO
  - Phosphodiesterase inhibitor
  - Carbon monoxide
  - Alcohol
  - Cocaine
  - Histamine
  - CGRP
  - Medication over-use (ergotamine, triptan, acetaminophen, NSAID)
  - Opioids
  - Withdrawal – caffeine, opioid, estrogen)
- Infection
  - Meningitis
  - Encephalitis

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Headache Classification

Secondary Headaches

IHS Classification ICHD-3

- Homeostasis disorder
  - Hypoxia (high altitude, diving, sleep apnea, airplane travel)
  - Dialysis
  - Arterial hypertension
  - Hypothyroidism
  - Fasting
  - Cardiac cephalalgia
- Psychiatric disorder (psychotic/somatization disorder)
- Lesions of the cranial nerves
  - Trigeminal neuralgia
  - Glossopharyngeal neuralgia
  - Occipital neuralgia

- Facial pain due to disorder of
  - Cranium
  - Neck
  - Ears
  - Nose
  - Sinuses
  - Teeth
  - Mouth or other facial or cervical structure
  - Eyes
    - Acute angle closure glaucoma
    - Refractive error
    - Ocular inflammatory disorder
    - Trochlear headache
    - Optic neuritis
    - Paratrigeminal oculosympathetic syndrome – Raeder's syndrome
    - Recurrent painful ophthalmoplegic neuropathy

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Headache Classification

What's a migraine?

- It's more than "just a bad headache"
  - "Migraine" is the neurologic condition
  - "Migraine Attacks" = hours to days of increased symptoms
- Familial, episodic, recurrent complex sensory processing disturbance with a constellation of symptoms
  - *Hallmark symptom is headache
  - But could also include vomiting, nausea, sensory sensitivity (smell, sound, light, irritability, depression, extreme fatigue
- Symptoms vary from person to person

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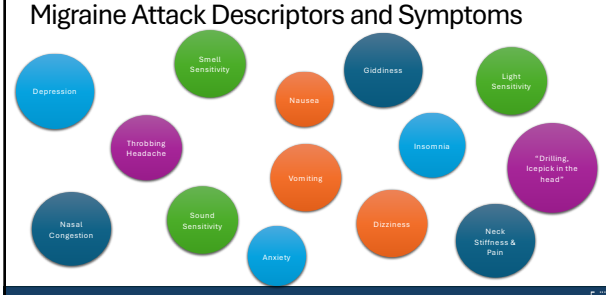
Symptomatology

Migraine Attack Descriptors and Symptoms



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Symptomatology

Cranial Autonomic Symptoms (CAS) in Migraine

Table 1. Prevalence of cranial autonomic symptoms (CAS) in patients with migraine with CAS and cluster headache.

CAS	Migraine with CAS	Cluster Headache
Lacrimation	14–45% [6,7,10–14]	50–91% [11,15–17]
Facial sweating/Flushing	6–39% [6,7,11–13]	32–56% [11,15–17]
Conjunctival injection	2–36% [6,7,10,12–14]	52–86% [11,15–17]
Eyelid edema	7–43% [7,10–14]	21–74% [11,16,17]
Nasal congestion	9–32% [6,7,10,12,13]	45–75% [11,16,17]
Aural fullness	13–27% [7,12,13]	9% [16]
Rhinorrhea	5–18% [6,7,10,12,13]	41–72% [11,15–17]
Ptosis	4–17% [6,7,10,12,13]	15–74% [15–17]
Miosis	2–6% [6]	6–29% [15–17]

Vicente, B.N et al. Cranial Autonomic Symptoms and Neck Pain in Differential Diagnosis of Migraine. Diagnostics 2023, 13, 590.

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Symptomatology

Migraine Onset

IHS Classification ICHD-3

- Typical onset of migraine is adolescence or early adulthood
  - Risk increases during reproductive years, particularly women
  - Decreases after the age of 50 (usually)
    - Can impact anyone at any age

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Symptomatology

Migraine in Children & Adolescents (<18yo)

IHS Classification ICHD-3

- Usually more often bilateral pain
  - Unilateral pain emerges in late adolescence or early adult life
- Pain is usually frontotemporal or small subset with facial pain
  - Occipital headache in pediatric patients is rare → diagnostic caution
- Migraine attack may last 2-72 hours

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Migraine Pathophysiology

Theories

- Neurovascular Theory
- Cortical Spreading Depression (CSD) Theory
- Neurogenic Inflammation Theory
- Serotonin Dysregulation Theory
- Genetic Predisposition Theory
- Hormonal Theory

Subalakshmi et al, J Neuroimmune Pharm. 2025

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Migraine Pathophysiology

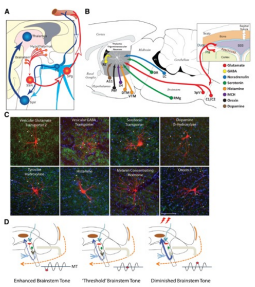

Image from:
Burstein et al. Migraine: Multiple processes, complex pathophysiology. J Neuroscience. 2015. 35(17):6619-6629

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Common triggers and Risk Factors of migraine

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Risk Factors

- Biological
  - Hormonal imbalances – estrogen dysregulation, cortisol dysregulation
  - Demographics – age, female sex
  - Metabolic – obesity, dyslipidemia, diabetes, hypertension
  - Genetics – MTDH, MEF2D, PRDM16 genes
- Psychological – anxiety, phobia, panic, stress
- Miscellaneous

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Hormones - Estrogen

- Migraine occurrence can be affected by menstruation, pregnancy, and menopause, in addition to the use of hormonal contraceptives and hormone replacement therapies
- Worse migraine outcomes associated with high-estrogen levels, high estrogen fluctuations and hormonal replacement therapy
- Menstrual migraine - specific condition when the timing of the attacks are associated with drops in estrogen levels a few days before menstruation
  - Estrogen administration
    - Pro: Can decrease occurrence of migraine headaches
    - Con: Raises risk of stroke, cardiac disease, vascular mortality
- Changes in migraines can effect CGRP
  - When estrogen decreases → release of CGRP

Lay et al. Neurol Clin. 2009., Ornello R et al. Neuropsychiatr Dis Treat. 2021., Barth et al. Front Neurosci. 2015., Sacco et al., J headache Pain 2012, Sheikh HU et al., J Head Face Pain 2018., Brandes JL JAMA 2006.,

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Hormones - Thyroid

- Migraine may result due to high TSH levels which can lead to pituitary growth and compression of intrasellar structures.
- Migraine and hypothyroidism considered comorbidities

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Triggers

- Strong Smells or Fumes
- Loud sounds or noises
- Too much or not enough sleep
- Barometric pressure changes/changes in the weather
- Stress – Physical or emotional
- Motion sickness
- Low Blood Sugar or Skipped meals
- Tobacco
- Alcohol
- Hormonal changes
- Medications
- Bright or flashing lights

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Foods that Trigger Migraine

ALCOHOL
- Red wine and champagne are most likely to cause headaches.
- Vodka, rum, liqueurs, beer and white wine are less likely to cause headaches.

CHEESES/DAIRY
- American, cheddar, brie, stilton, camembert and blue cheese, yogurt, and sour cream should be avoided.
  - Cottage cheese and cream cheese are less likely to cause headaches.

PROCESSED MEATS (with Nitrite)
- Hot dogs, bologna, bacon, salami, canned ham and sausage should be avoided.

OTHER FOODS
- Nuts, peanut butter,
- Pickled or marinated foods
- Bananas
- Chicken livers, shellfish, excessive pork, excessive coffee or tea, monosodium glutamate (Accent,MSG
- Onions or garlic in excess
- Diet cola
- Avocado
- Vitamins A,E,and Niacin
- Fresh bread or rolls
- Beans and pea pods.

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Migraine Risks and Triggers

Sleep

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Migraine Risks and Triggers

Caffeine

- Passes through all biological membranes including the blood-brain barrier
- Rapidly and completely absorbed (99%) within 1 hour of oral consumption
- Half life 5 hours
- Metabolized by cytochrome P450 system
  - Isoenzyme CYP1A2 responsible for 90% of caffeine clearance
- Antagonize adenosine receptors (ARs) A1R, A2AR
  - Adenosine is in every cell (ADP/ATP)

Alstadhaug KB and Andreou AP (2019) Caffeine and Primary (Migraine) Headaches—Friend or Foe? Front. Neurol. 10:1275

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Migraine Risks and Triggers

Adenosine and Sleep

- Adenosine has sleep promoting effects – adenosine accumulates in basal forebrain with sustained and prolonged wakefulness→ reduces cortical activity
  - Blocking A2AR in nucleus accumbuns inhibits the GABAergic output to the lateral hypothalamus, tuberomamullary nuclei in the hypothalamus and locus coeruleus → activation and arousal effect of caffeine
- Adenosine can cause headaches

Alstadhaug KB and Andreou AP (2019) Caffeine and Primary (Migraine) Headaches—Friend or Foe? Front. Neurol. 10:1275

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Migraine Risks and Triggers

Caffeine and Pain

- Caffeine at doses between 25-100mg/kg have intrinsic antinociceptive effects
  - 600-1200mg of caffeine may have anti-nociceptive benefits in caffeine-naïve humans
  - Adjuvant, caffeine ≥ 65mg can potentiate analgesic properties of other medications.
  - May be beneficial in tension-type headaches
- Caffeine increases dopamine release
  - Potential to cause physical dependence
  - Sudden withdrawal following chronic consumption can lease to time-limited headache, mood-changes, difficulty focusing, nausea, and muscle stiffness

Alstadhaug KB and Andreou AP (2019) Caffeine and Primary (Migraine) Headaches—Friend or Foe? Front. Neurol. 10:1275

Sawynok J. Caffeine and pain. Pain. (2011) 152:726–9.
Bendtsen L et al. . EFNS guideline on the treatment of tension-type headache - report of an EFNS task force. Eur J Neurol. (2010) 17:1318–25.

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Migraine Risks and Triggers

Caffeine

- Low trigger for people with migraine ~6.3%
  - May improve migraines for some
- Common trigger in migraine-associated vertigo attacks ~69.6%
- Possible mechanisms
  - Activation of sympathetic nervous system or parasympathetic withdrawal
  - May precipitate another trigger
    - Sleep dysfunction
- Conclusion: Caffeine can both induce and alleviate headaches/migraines

Mollaoglu M. Trigger factors in migraine patients. J Health Psychol. 2013;18:984-994.
Kuwahara K et al. Effects of modern eating patterns on the cardiac autonomic nervous system in young Japanese males. J Physiol Anthropol. 2011;30:223-231.
Omer Saglam Uket al.. The role of lifestyle modifications in the management of migraine associated vertigo. J Clin Anal Med. 2015;6:763-765.

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Migraine Triggers

Alcohol

- Alcohol can trigger migraine and tension-type headache
- Red wine most common trigger in US/UK
  - But other cultures have found white wine (France, Italy) and champagne (France) are more common triggers
- Secondary Alcohol Headache types
  - Immediate (within 3 hours)
  - Delayed (after blood alcohol level declines or reduces to zero)
    - 1 day after alcohol consumption

Table 4 Contents and actions of principal alcoholic drinks, possibly interested in headache provocation

	Histamine	Tyramine	Sulphites	Flavonoids	5-HT release
Red wine	++	+	+	++	++
White wine	+	+	++	+	+–
Beer	+	+	+	+	–

Panconesi A. Alcohol and migraine: trigger factor, consumption, mechanisms, a review. J Headache Pain (2008). 9:19-27

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Migraine Risks and Triggers

Smells

Groups of Odors that Trigger Migraine

Fetid
Cooking products
Oil derivatives
Shampoo and Conditioner
Hair styling products
Perfumes, insecticides, Rose smell
Laundry detergent/Fabric softener (florals)

Odors	Rate of association (%)
Perfumes	56.4
Tobacco	47.5
Fabric softener	32.7
Body odor	32.7
Garbage	24.8
Hairdressing products	22.8
Automobiles	22.8
Sweat	19.8
Garlic	16.8

Imai et al. Scientific Reports | (2023) 13:8469

Table 2. Details of odors or odors reported to be associated with migraine attacks.

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Migraine Risks and Triggers

Stress

- No definite evidence that stress incites the incidence or progression of migraine or directly triggers migraine attacks
  - General understanding that is worsens migraine burden
  - Higher migraine frequency is associated with higher levels of perceived stress
  - High
- Indirect etiology?
  - Missed medications, meals
  - Reduced sleep
  - High job strain → decreased lack of time for leisure or personal care and decreased sleep
- Stress leads to central sensitization and hyperalgesia
  - May enhance nociceptive responses → increase clinical characteristics (throbbing pain, motion sensitivity, hyperalgesia, allodynia)

Stubberud et al. Is there a causal relationship between stress and migraine? Current evidence and implications for management. J Headache Pain. (2021) 22:155.

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Migraine Risks and Triggers

Neck Pain and Migraine

- Neck pain is more prevalent in patients with severe headache or migraine (38%) than the general population (11%)
- Etiology
  - Trigeminocervical complex (TCC)
    - C1, C2, C3
    - Trigeminal nucleus caudalis
  - Neck muscle induced-pain causes pain in both cervical and trigeminal areas.
  - Migraine attacks may lead to central cervical sensitization and lower pressure pain thresholds associated with increased neck muscle tension
  - Increased stress may contribute to increase tension
  - Excessive forward head posture → reduces cranio-cervical angle
    - Due to repeated tasks in poor ergonomics and shortening of cervical extensor muscles.

Vicente, B.N et al. Cranial Autonomic Symptoms and Neck Pain in Differential Diagnosis of Migraine. Diagnostics 2023, 13, 590.

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Migraine Risks and Triggers

Comorbidities

- Comorbidities
  - Insomnia
  - Depression
  - Anxiety
  - Gastric ulcers and/or gastrointestinal bleeding
  - Angina
  - Epilepsy

Buse et al., J Headache Pain 2020.

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Migraine Classification

Type of Migraine

Migraine without aura	• Common migraine • Hemicrania Simplex
Migraine with Aura	• Classic or Classical migraine • Ophthalmic, Hemiparesthetic, hemiplegic, aphasic migraine • Migraine Accompagnee • Complicated Migraine
Migraine with Brainstem Aura	• Basilar artery migraine • Basilar Migraine • Basilar-Type Migraine
Retinal Migraine	
Chronic Migraine	

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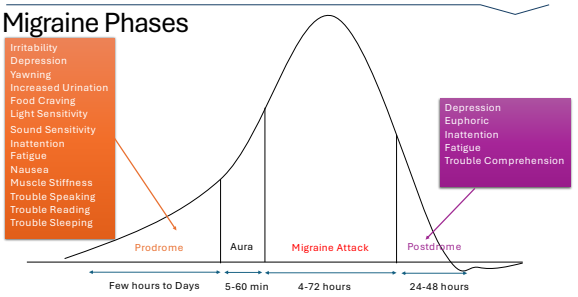
Migraine Phases



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Migraine Types

Migraine

Without Aura

- At least 5 attacks
- Headache attack lasts 4-72 hour (untreated)
- Headache has at least 2 of 4 characteristics:
  - Unilateral location
  - Pulsating quality
  - Moderate or severe pain intensity
  - Aggravation by or causing avoidance of routine physical activity
- During the headache at least one of the following:
  - Nausea and or vomiting
  - Photophobia and phonophobia

With Aura

- At least two attacks
- One or more of the following fully reversible aura symptoms
  - Visual
  - Sensory
  - Speech and/or language
  - Motor
  - Brainstem
  - Retinal
- At least three of the six characteristics
  - At least one aura symptom spreads gradually over >5 min
  - 2 or more aura symptoms occur in succession
  - Each individual aura lasts 5-60 min
  - At least one aura symptom is unilateral
  - At least one aura symptom is positive
  - The aura is accompanied by or followed within 60 minutes by a headache

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Migraine with Aura

Migraine Aura

- Aura – complex of neurological symptoms that occurs before the migraine headache
  - May being after the headache phase has commenced or continue into the headache
- When aura symptoms are multiple, they usually follow one another in succession:
  - Visual→ Sensory → Aphasic
- Duration ~20 min to 1 hour
  - Motor symptoms may last longer
- Pathophysiology
  - Regional cerebral blood flow is decreased in the cortex corresponding to the clinically affected area and often over a wider area.
  - Blood flow reduction usually starts posteriorly and spreads anteriorly and is usually above the ischaemic threshold.
  - After one to several hours, gradual transition into hyperaemia occurs in the same region. Cortical spreading depression is the likely underlying mechanism.

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Migraine with Aura

Clinical Features of Visual Migraine Aura

Table 2 List of Elementary Visual Symptoms (EVSs) of migraine aura as reported in literature and the range of their frequency in the studies

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

Viana et al., Clinical features of visual migraine aura: a systematic review. *J Headache Pain*. 2019. 20(64):1-7

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Migraine with Aura

Clinical Features of Visual Migraine

Table 3 Proposed list of all EVS of migraine aura and their description

Proposed Name	Description
1. Bright light[c]	Single area of bright light
2. Foggy/blurred vision	Foggy or blurred vision
3. Zigzag lines[ac]	Zigzag or jagged lines
4. Scotoma	Single blind area
5. Scotomata	Several blind/black areas
6. Small bright dots[c]	Small bright dots/stars
7. White dots/round forms[abc]	Medium sized white dots/round forms
8. Colored dots/round forms[abc]	Medium sized coloured dots/round forms
9. Lines (colored lines)[abc]	Lines (colored lines)
10. Geometrical shapes[abc]	Geometrical shapes
11. 'Like looking through heat waves, water or oil'	'Like looking through heat waves, water or oil'
12. Visual snow	Dynamic, continuous, tiny dots usually black/gray on white background and gray/white on black background
13. 'Bean-like' forms[abc]	'Bean-like' forms like a crescent or C-shaped
14. Hemianopsia	Blindness of half of the visual field
15. Deformed images	Deformed images (alteration of lines/angles)
16. Tunnel vision	Blindness in the whole periphery
17. Oscillopsia	Movement of stationary objects
18. Mosaic vision	Seeing mosaic-like
19. Fractured objects	Seeing fractured objects
20. Corona effect[abc]	An extra edge on objects
21. Anopia	Total blindness
22. Micropsia	Objects appear smaller or more distant than they actually are
23. Macropsia	Objects appear larger or closer than they actually are
24. Like a negative film	Seeing like a negative film
25. Complex hallucinations[ac]	Visual perception of something not present (e.g. objects, animals, and persons)

Viana et al., Clinical features of visual migraine aura: a systematic review. *J Headache Pain*. 2019. 20(64):1-7

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Migraine with Aura

Migraine with Typical Aura

- Migraine with aura (visual +/- sensory +/- speech/language symptoms) but no motor weakness
  - Duration of each symptom is no longer than one hour
  - No motor, brainstem or retinal symptoms
- Gradual onset (over 5 minutes)
- Symptoms increase over time
- Last 5-60min
- Usually bilateral
- +/- Headache
- +/- "Positive" Phenomena
  - Sparkles
  - Zig-Zags
- (-) LOC

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Migraine with Aura

Migraine with Brainstem Aura

- Migraine with at least two of the following symptoms during an attack
  - Vertigo
  - Diplopia
  - Poor muscle coordination (ataxia)
  - Slurred speech (dysarthria but not aphasia)
  - Tinnitus
  - Hearing loss (hypacusis)
  - Fainting (decreased level of consciousness GCS < 13)

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Migraine with Aura

Hemiplegic migraine

- Rare and severe form of migraine
- Aura = reversible motor weakness (<72 hrs)
  - Temporary paralysis (can last several days) on one side of thebody
  - Paralysis happens before or during a headache
- Familial/genetic mutations

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Migraine with Aura

Retinal Migraine

- Repeated attacks of MONOCULAR visual disturbance (scintillations, scotoma/blindness) associated with migraine
- Temporary vision loss or changes in eyesight
  - Spreads gradually over > 5 min
  - Symptoms last 5-60min
  - Headache within 60 minutes
- Need to exclude
  - Amaurosis fugax
  - Carotid artery occlusive disease
  - Arteritic Disease (GCA)

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Migraine with Aura

Typical Migraine Aura without Headache

- Acephalgic migraine vs TIA vs other serious disease.
  - If the aura occurs for the first time after age 40
  - When symptoms are exclusively negative (hemianopia)
  - Aura is prolonged >1 hour or very short

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Ocular manifestations of migraine: visual disturbances and ocular findings

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Differential diagnosis of migraine Aura

Stroke
- Symptoms instantaneous
- Maximal symptoms at onset
- +/- "Negative" phenomena
- Loss of vision
- Numbness/weakness
- Loss of speech
- +/- Headache

Amaurosis Fugax
- Unilateral
- Sudden onset blindness/vision fades to black
- Absence of "Positive" Phenomena

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Differential Diagnosis of Migraine Aura

Occipital Epilepsy
- Lasts seconds to minutes
- +/- Loss of consciousness

Retinal/Vitreous Tear/Detachment
- Seconds
- Unilateral

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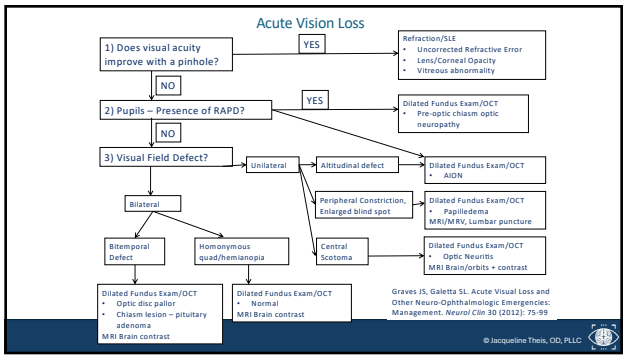

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Migraine Types

Status Migrainosus

- Migraine attack lasting > 72 hours
- Often cause by medication over use

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Difference between a Retinal vs. Ophthalmic vs. Ocular vs Acephalgic Migraine

Ocular Migraine	• Headache specialists dont use this anymore
Retinal Migraine	• Rare condition, migraine • Repeated bouts of short-lasting partial visual loss before or during the headache
Acephalgic Migraine	• Migraine Aura without the pain

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Migraine Aura

Acephalgic Migraine

- Migraine symptoms WITHOUT the pain
  - "Retinal" or "Ocular" Migraine
  - Vestibular Migraine
- Hypothesis
  - Visual aura → cortical spreading depression temporally impacting electrical impulses of neural tissue
  - Migraine pain→ vasospasm of cerebrovasculature

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Migraine Aura

Acephalgic Migraine

- Risk Factors
  - (+) Family history of migraine
  - Hormonal status – adolescence, menopause, menses cycle
  - Weather changes
- Triggers
  - Fatigue
  - Skipping a meal
  - Caffeine withdrawal
  - Certain foods
  - Unknown

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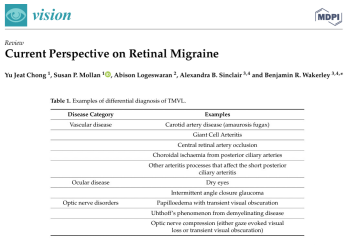
Migraine Aura

Transient Monocular Vision Loss


vision
MDPI
Review
Current Perspective on Retinal Migraine
Yu Jeat Chong [1], Susan P. Mollan [1,*], Abison Logeswaran [2], Alexandra B. Sinclair [3,4] and Benjamin R. Wakerley [3,4,*]
Table 1. Examples of differential diagnosis of TMVL.
Disease Category | Examples
Vascular disease | Carotid artery disease (amaurosis fugax)
Giant Cell Arteritis
Central retinal artery occlusion
Choroidal ischaemia from posterior ciliary arteries
Other arteritis processes that affect the short posterior ciliary arteritis
Ocular disease | Dry eyes
Intermittent angle closure glaucoma
Optic nerve disorders | Papilloedema with transient visual obscuration
Uhthoff's phenomenon from demyelinating disease
Optic nerve compression (either gaze evoked visual loss or transient visual obscuration)
Citation: Chong, Y.J.; Mollan, S.P.; Logeswaran, A.; Sinclair, A.B.; Wakerley, B.R. Current Perspective on Retinal Migraine. Vision 2021, 5, 38. https://doi.org/10.3390/vision5030038

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Role of optometry in diagnosing and managing migraine-related visual symptoms

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Optometric Evaluation

Examination

- Visual Acuity
- Refraction
- Pupils
- Visual Field
- Oculomotor Evaluation
- Ocular Health
- Tint Assessment

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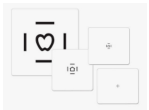
Visual Acuity

- Distance OD/OS
- Monocular OD/OS
- Optotypes
  - Letters vs. Lea
  - Isolated (single symbol) vs Crowded
- Pinhole?
  - Ocular Health



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Examination

Afferent Visual Pathway

- *Visual Acuity*
- Color Vision
  - Ishihara/AOHRR
  - Red Cap Desaturation
- Visual Field
  - Confrontation
  - FDT/HVF
  - Amsler
- Pupils
  - Size in Dark/Light
  - Direct/Indirect Response
  - +/-RAPD
  - Accommodative Response

Kerr NM et al. Diagnostic accuracy of confrontation visual field tests. *Neurology*. 2010;74:1184-1190
Rucker JC, Kennard C, Leigh RJ. The Neuro-ophthalmological Examination. *Handbook of Clin Neurol*. 102(3): 71-94

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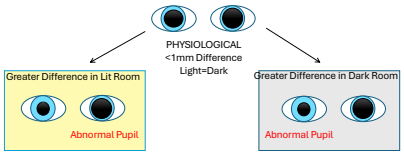
Pupils



- Horner Syndrome

- Traumatic
- Adie's Pupil
- CN III Palsy
- Pharmacologic

Ehlers J, Shah C. Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. Lippincott Williams & Wilkins. 5th ed

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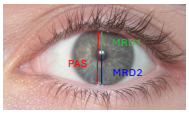
Examination



Orbital Testing

- Eyelid Measurements
  - Palpebral Aperture ~9-12mm
  - MRD1~4-5mm
  - MRD2
  - Prolonged upgaze
- Exophthalmometry
  - Base, mm OD, mm OS
  - >2mm difference of >21mm abnormal

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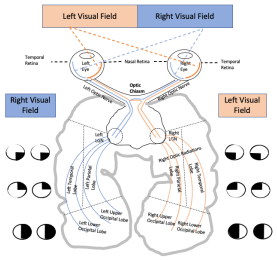
Visual Fields – The Afferent Visual Pathway

- Confrontation
- FDT/HVF
- Amsler



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Oculomotor Headaches

- Accommodative insufficiency
- Accommodative Spasm
- Cervical oculomotor
- Saccadic
- Vestibular-spinal
- Convergence spasm
- Convergence insufficiency

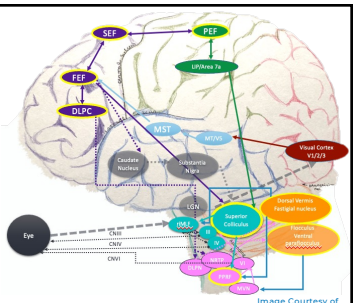

Image Courtesy of Jacqueline Theis

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Ocular Health Examination

- Anterior Segment
  - Orbit - Eyelids
  - Conjunctiva – Injection, Chemosis
  - Cornea/Ocular Surface
  - IOP
- Posterior Segment
  - ONH – Edema, pallor, hemorrhages
    - Spontaneous venous pulsation (SVP)
      - Cessation of SVP is sensitive marker of raised intracranial pressure
      - Presence of SVP indicates it is unlikely to have ONH edema, and CSF pressure <190mmH20
        - Should be present ~90% of patients
  - Macula

Jacks AS, Miller NR. Spontaneous retinal venous pulsation: aetiology and significance. *J Neurol Neurosurg Psychiatry.* 2003. 74(1):7-9.

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Ocular Health and Migraine

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Dry Eye, TBI, Migraine and Sleep

- Dry eye is common post-TBI
  - a meta-analysis showed that US military veterans with TBI were more likely to have a diagnosis of dry eye(37.2%) compared with their counterparts without TBI (29.1%) (Lee 2018)
- Dry eye is common in Migraine and other pain disorders – chronic regional pain syndrome, fibromyalgia
- Dry eye is common in psychiatric conditions – depression and anxiety
- Dry eye can be caused by sleep deprivation
  - Sleep deprivation compromises the lacrimal gland and induces dry eye within 2 days
  - Staying up all night could induce tear hyperosmolarity and reduce tear secretion
- Sleep apnea is associated with dry eye

Galor A, Feuer W, Lee DJ, Florez H, Carter D, Pouyeh B et al. Prevalence and risk factors of dry eye syndrome in a United States veterans affairs population. Am J Ophthalmol 2011; 152: 377–384 e372
Lee C, Felix E, Levitt R, et al. Traumatic brain injury, dry eye and comorbid pain diagnoses in US veterans. Br J Ophthalmol 2018;102:667–673.
Lee YB, Koh JW, Hyon JY, Wee WR, Kim JJ, Shin YJ. Sleep deprivation reduces tear secretion and impairs the tear film. Invest Ophthalmol Vis Sci 2014; 55: 3525–3531
Li S, Ning K, Zhou J, Guo Y, Zhang H, Zhu Y, Zhang L, et al. Sleep deprivation disrupts the lacrimal system and induces dry eye disease. *Exp & Molec Med.* 2018, 50:e451, 1-12.

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CLINICAL TRIAL

OPEN

A randomized crossover trial: The impact of ocular lubrication on migraine severity in persons with dry eye disease and migraine

Nur Amalina Md Isa, MOptom,[1] Arun V. Krishnan, MD, PhD,[2,3] Alessandro S. Zagami, MD,[2,3] Shyam S. Tummanapalli, PhD,[1] Katherine Spira, MD,[2] Eric B. Papas, PhD, FAAO,[1] Azadeh Tavakoli, PhD,[1] and Maria Markoulli, PhD, FAAO[1]

(Optom Vis Sci 2025;00: 00–00)

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Migraine and Photophobia

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Photophobia

"Photosensitivity" or "Light Sensitivity" - Defined

- "Photophobia" - "Fear of Light"
- True photophobia – light exposure to the eye induces or exacerbates pain (Lebensohn)
- Dazzle-induced photophobia – visual discomfort based on light diffusion through ocular media or lack of adaptation
  - Uncomfortable sense of excessive brightness without pain
  - Ocular disease: toxic amblyopia, albinism, rod monochromatism, corneal/lenticular/vitreous opacities
- "Photo-oculodynia" – pain or discomfort in the eye from a light source that should NOT be painful under normal circumstances (Digre)
  - 1) avoidance of light behavior
  - 2) Reason for behavior is because of eye/head pain/discomfort, or soothing feeling of being in the dark

Lebensohn JE, Bellows J. The nature of photophobia. Arch Ophthalmol. 1934;12(3):380-90.
Digre KB, Brennan K. Shedding light on photophobia. J Neuroophthalmol. 2012;32(1):68-81.

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Photophobia

Epidemiology

Light sensitivity can degrade the quality of life for patients

- Prevalence
  - 10% of general population
  - 50-55% of TBI population
    - Prevalence increases is exposed to >1 blast
    - Present in acute and chronic TBI (Truong)
      - Reduction in photosensitivity with time
        - 10% in first year
        - 40% after the first year
          - 42% stable
          - 3% increased
          - 5% waxed/waned
  - 80-90% of migraine population
    - Tension headache
    - Cervicogenic headache

Capo-Aponte JE, Urosevich TG, Temme LA, Tarbett AK, Sanghera NK. Visual dysfunctions and symptoms during the subacute stage of blast-induced mild traumatic brain injury. Military Medicine 2012;177:804-813
Magone MT, Kwon E, Shin SY. Chronic visual dysfunction after blast-induced mild traumatic brain injury. J Rehabil Res Dev. 2014;51(1):71e80
Shepherd D, Landon J, Kalloor M, Barker-Collo S, Starkey N, Jones K, Ameratunga S, Theadom A, BIONIC Research Group. The association between health-related quality of life and noise or light sensitivity in survivors of a mild traumatic brain injury. *Qual Life Res*. 2020;29(3):665-672.
Truong JQ, Ciuffreda KJ, Han MH, et al. Photosensitivity in mild traumatic brain injury (mTBI): a retrospective analysis. Brain Inj. 2014;28(10):1283–7
Vanagaite V. Photophobia and phonophobia in tension-type and cervicogenic headache. *Cephalalgia*. 1998:18(6):313-318
Wu Y, Hallet M. Photophobia in neurologic disorders. *Trans Neurodegen*. 2017:6(26):1-6

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Intrinsically Photosensitive Retinal Ganglion cells (ipRGCs)

- Retinal ganglion cell with melanopsin photopigment
  - 1-3% of ganglion cells in the retina
  - Also found in the iris
- Peak activation is at 480nm (between green and blue cones) but can also be stimulated by input from rods and cones
- Send axons to
  - Thalamus (light-pain matrix)
  - Pretectal Nucleus/Edinger-Westphal nucleus (pupillary light response)
  - Hypothalamus/Suprachiasmatic nucleus (circadian rhythm)
- Hypothesis of connections between ipRGCs and TBI symptoms of headaches, ocular pain, light sensitivity and sleep disturbances

Hattar S, Liao HW, Takao M, Berson DM, Yau KW. Melanopsin-containing retinal ganglion cells: architecture, projections, and intrinsic photosensitivity. Science. 2002;295(5557):1065e70
Ellenberger J, Kim B, Castr-Abegar A, Rex TS. Connections between intrinsically photosensitive retinal ganglion cells and TBI symptoms. Neurology. 2020;95:826-833
Benson DM, Dunn FA, Takao M. Phototransduction by retinal ganglion cells that set the circadian clock. Science.2002;295(5557):1070e3
Kawasaki A, Kardon RH. Intrinsically photosensitive retinal ganglion cells. J Neuroophthalmol. 2007;27(3):195e204
Noseda R, Constandil L, Bourgeais L, Chalus M, Villanueva L. Changes of meningeal excitability mediated by corticotrigeminal networks: a link for the endogenous modulation of migraine pain. J Neurosci. 2010;30(43):14420e9
Noseda R, Kainz V, Jakubowski M, et al. A neural mechanism for exacerbation of headache by light. Nat Neurosci. 2010;13(2):239e45

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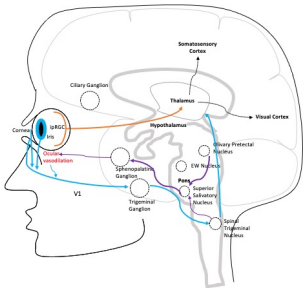
Photophobia

Retino-Thalamo-Cortical Pathway

- Retinal ipRGCs project directly to the thalamus → increased dura-sensitive trigeminovascular neurons →multiple cortical areas (somatosensory and visual)
- Implicated in photophobia that occurs with
  - Migraine triggered by photophobia
  - Exacerbation of headache by light



Albilali A, Dilli E. Photophobia: When light hurts, a review. *Curr Neur Neurosci Rep*. 2018;18:62

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Blind patients with migraine

- Intensification of headache by light only perceived by blind migraineurs with an intact optic nerve – who could perceive ambient light illumination but not image formation due to rod/cone degeneration
- Blind migraineurs with optic nerve damage did not have light intensify migraine.
  - Also have fragmented or irregular sleep patterns and deficient pupillary light response
- ipRGCs trigger photophobia in migraine

Moseda R, Kainz V, Jakubowski M, et al. A neural mechanism for exacerbation of headache by light. *Nat Neurosci.* 2010;13:239-245

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The Evolution of the Lighting Industry

- Fire ~ 500,000 years ago
- Wax candle ~700 BC
- Incandescent light sources ~1801
  - Generates light through heat
  - Similar to spectrum from the sun
  - High energy consumption
  - Emits energy towards high-wavelength
- Fluorescent light sources ~1940
  - Generates light through excitation of individual atoms
- Lower energy consumption
  - Compact fluorescent lamps (CFLs)
  - Light emitting diodes (LEDs)

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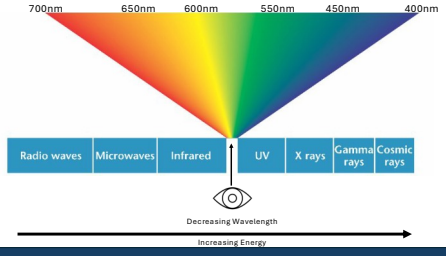
Visible Spectrum of Ligh



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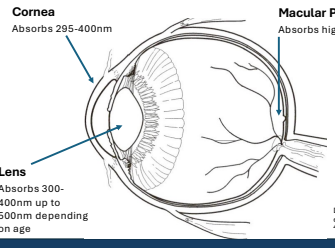
Transmission Characteristics of the Eye



*Our eyes cant Block blue light (400-500nm)

Dillon J. The photophysics and photobiology of the eye. *J Photochem Photobiol B.* 1991;10(102):23-40.

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Blue Light Blockers

- "Blue-Blockers" or "Blue-Attenuating" filters are not well-defined or standardized (cut off filter vs notch filter)
- Important properties of a filter
  - Luminous transmittance (%)
  - Melanopsin transmittance (%)
  - Color Shift
  - Color Gamut

Splitschan M, Lazar R, Cajochen C. Visual and non-visual properties of filters manipulating short-wavelength light. Ophk Physiol Optics. 39:2019:459-468

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Photophobia

Blue Light Blockers vs. Computer (Night-Mode) Modifications

- Compared the radiation produced by smartphones that reaches the eye when using night-mode functions vs blue light reducing lenses
- To determine impact they had on visual and nonvisual (circadian) parameters to compute a melatonin suppression value (MSV)
  - Night-mode functions reduced MSV by up to 93%
    - Warmest mode produced the least suppression
  - Blue light reducing spectacles reduced melatonin suppression by 33%
    - Coated lenses more efficient than tinted lenses

Teran, E., Yee-Rendon, C. M., Ortega-Salazar, J., De Gracia, P., Garcia-Romo, E., & Woods, R. L. (2020). Evaluation of Two Strategies for Alleviating the Impact on the Circadian Cycle of Smartphone Screens. *Optometry and vision science : official publication of the American Academy of Optometry*, 97(3), 207–217. https://doi.org/10.1097/OPX.0000000000001485

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Enhancing our natural blue light blockers

MDPI

Article

Macular Carotenoid Supplementation Improves Visual Performance, Sleep Quality, and Adverse Physical Symptoms in Those with High Screen Time Exposure

James M. Stringham [1,*], Nicole T. Stringham [2] and Kevin J. O'Brien [3]

[1] Nutritional Neuroscience Laboratory, Department of Psychology, University of Georgia, Athens, GA 30602, USA
[2] Interdisciplinary Neuroscience Program, Biomedical and Health Sciences Institute, University of Geo Athens, GA 30602, USA; ntstringham@gmail.com
[3] Vision Sciences Laboratory, Department of Psychology, University of Georgia, Athens, GA 30602, USA Obrien.psych@gmail.com
* Correspondence: jim.stringham@gmail.com; Tel.: +1-210-204-4189

Academic Editor: Rotimi Aluko
Received: 19 June 2017; Accepted: 28 June 2017; Published: 29 June 2017

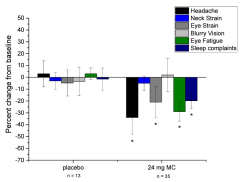

Figure 2. Percent change from baseline for physical indicators of excessive screen time (ST) (including sleep quality), at 6 months for both placebo and treatment groups. Error bars are +/− 1 SD. Asterisks denote statistically significant difference from placebo ($p < 0.05$).

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Evidence for Tilted Filters - FL41

- Developed in England in the 1980s for fluorescent light sensitivity
- Transmission minimum is 480nm
- Wavelength of light plays a role in the degree of discomfort in Migraine
  - Short (blue) and long (red) wavelengths can be uncomfortable for migraine patients
  - 480nm is particularly triggering in migraine patients
- FL41 is helpful in patients with
  - Migraine
  - Benign Essential Blepharospasm
  - Vestibular dysfunction/migraine

Wilkins AJ, Nimmo-Smith I, Slater AI, Bedocs L. Fluorescent lighting, headaches and eyestrain. Lighting Res Technology. 1989;21:11e8
Blackburn MK, Lamb RD, Digre KB, et al. FL-41 tint improves blink frequency, light sensitivity, and functional limitations in patients with benign essential blepharospasm. Ophthalmology. 2009:116(5):997e1001
Tatsumoto M, Eda T, Ishikawa T, Ayama M, Hirata K. Light of intrinsically photosensitive retinal ganglion cell (ipRGC) causing migraine headache exacerbation. Cephalalgia. 2013;33(8 Suppl):2. (abstract OR-3).

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Photophobia

Migraine and Blue Light

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- Transmission minimum is 480nm
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Botox

Blepharospasm

- Focal dystonia
- Involuntary, bilateral, synchronous, symmetrical contraction of orbicula (preseptal and pretarsal)
- Decreased spasms and blink rate (increased eyes open) with reading an
  - Occipital cortical activation may modulate orbicularis oculi spasms through the basal ganglia to reduce activity in the trigeminal blink re
- DDx
  - Tics
  - Hemifacial spasm (unilateral, microvascular compression of facial
  - Meige syndrome (involuntary contraction of both upper and lower fa
  - Apraxia of eyelid opening (inability to open eyes)
- Tx
  - Botox
  - FL41
  - Orals: Benzodiazepines, anticholinergics, tetrabenazine, baclofen

Defazio G, Hallett M, Jinnah HA, et al. Blepharospasm 40 years later. Mov Disord 2017; 32:498–509
Ferrazzano G, Conte A, Belvisi D, et al. Writing, reading, and speaking in blepharospasm. J Neurol 2019; 266:1136–1140.

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Critical Flicker Frequency

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Migraine Mimickers

- Blepharospasm
- Tension Headache
- Giant Cell Arteritis
- Carotid Artery Dissection
- Acute Angle Closure
- Increased intracranial Pressure/Pseudotumor

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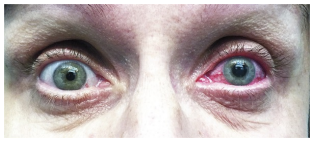
Migraine Mimickers

Acute Angle Closure

- Symptoms:
  - Headache
  - Left eye pain
  - Nausea
  - Vomiting
  - Blurry vision
  - Halos around lights



https://www.nejm.org/doi/full/10.1056/NEJMicm1712742

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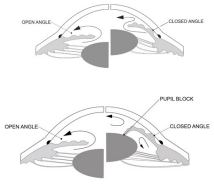
Acute Angle Closure



- Significant elevated IOP
- Diffuse corneal edema
  Fixed mid-dilated pupil
- Closed iridocorneal angle (assessed by Gonio or SLE)
- Shallow anterior chamber
- Convex iris appearance

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Migraine Mimickers

Angle Closure Kit

- Topical glaucoma meds
  - Beta Blocker (timolol, combigan)
  - Alpha 2 agonist (Brimonidine, apraclonidine)
  - *Prostaglandin analogue (Latanoprost, travoprost, bimatoprost)*
- Oral glaucoma meds
  - Diamox 250mg tablets
- Topical pilocarpine 2% or 4%
- Topical Steroids
- Hypertonic solutions
- Atropine 1%
- Ondansetron orally disintegrating tablets 8mg
- Emesis bag
- TB syringe
- 30 gauge needle

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Migraine Complications

Angle Closure and Medications

- Antidepressants
  - Amitryptyline
  - Nortriptyline
- Antiepileptics
  - Topiramate
- Antimigraine
  - Acetaminophen
- Antihistamines

Na KI, Park SP. Association of Drugs With Acute Angle Closure. JAMA Ophthalmol. 2022 Nov 1;140(11):1055-1063. doi: 10.1001/jamaophthalmol.2022.3723. PMID: 36136326; PMCID: PMC9501771.

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Migraine Mimickers

Acute Angle Closure-

Research Guideline
- Diamox/Acetazolamide: two 250mg
- Topical: effective under 50mmHg
- Quickest efficacy: Timolol and apraclodine
- Gonioscopy or tactile compression
- Supine Positioning
- Pilocarpine: effected with IOP below 40mmHg
- LPI – treatment/prophylative tx: Nd:Yag Laser typically done temporally
- Steroid: once appropriate IOP is obtained helps with inflammatory response post-event and especially needed after LPI

Academic Guideline
- Event:
- Ophthalmic Beta Blocker and Iopidine/Apraclonidine
- Indentation Gonio
- Re-evaluate pressure every 15-30 mins
- Oral CAI Diamox or glycerin
- Pilocarpine – if below 40mmHg

- Maintenance dosing until LPI preformed:

1) Timolol BID
2) Diamox 500mg BID
3) Pilo 2% QID
4) Pred Acetate QID

- LPI ideally done once inflammatory reaction has calmed

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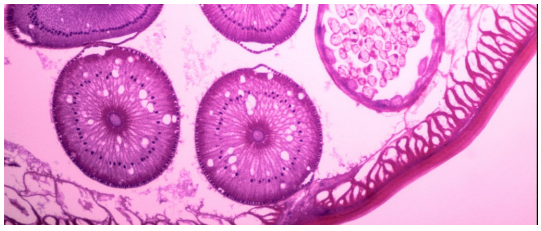
Migraine Mimicker

Giant Cell Arteritis



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Giant Cell Arteritis

Epidemiology

GCA
- Most common systemic vasculitis affecting adults >50yo
  - Rare in people <50yo
  - Average age of onset is 74-76yo
  - For each decade after 50, incidence increases from
    - 2.0 (50-60yo)
    - 11.8 (61-70yo)
    - 31.3 (71-80yo) per 100,000 persons/year
- Women affected 2-3x more than men
- More common in whites, Nordic/Northern European ancestry, and other northern latitudes

AAION
- Annual incidence of AAION from GCA is 1.3 per 100,000 in patients >50yo
- Vision loss from AAION and CRAO from GCA is severe
  - 73% present with VA worse than 20/200
  - 15% of eyes have an improvement, likely from eccentric fixation

Hoffman GS. Giant Cell Arteritis. *Ann Intern Med.* 2016;165(9):ITC65-80

Chen JJ, Leavitt JA, Fang C, Crowson CS, Matteson EL, Warrington KJ. Evaluating the incidence of arteritic ischemic optic neuropathy and other causes of vision loss from giant cell arteritis. *Ophthalmology* 2016: 123:1999-2003

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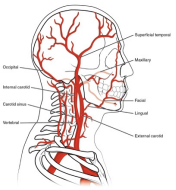
Giant Cell Arteritis

Pathogenesis



- Infectious? immune trigger in a genetically predisposed subject
- T-cell mediated granulomatous inflammation of medium- and large-vessels
  - Aorta
  - External carotid artery
    - Posterior ciliary artery → AAION
  - Temporal arteries

Ciccia F, Rizzo A, Ferrante A, Guggino G, Croci S, Cavazza A, Salvarani C, Triolo G. New Insights into the pathogenesis of giant cell arteritis. *Autoimmunity reviews*. 2017 May. Epub ahead of print. Accessed May 21, 2017.

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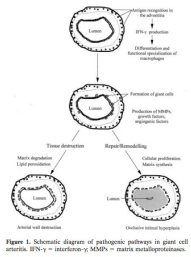
Giant Cell Arteritis

Pathogenesis

Persistent vessel wall inflammation → vascular damage → stenosis, occlusions, and aneurysms



Ciccia F, Rizzo A, Ferrante A, Guggino G, Croci S, Cavazza A, Salvarani C, Triolo G. New Insights into the pathogenesis of giant cell arteritis. *Autoimmunity reviews*. 2017 May. Epub ahead of print. Accessed May 21, 2017.

Figure 1. Schematic diagram of pathogenic pathways in giant cell arteritis. IFN-γ = interferon-γ; MMPs = matrix metalloproteinases.

Image from:
Weyand CM, Goronzy JJ. Arterial wall injury in giant cell arteritis. *Arthr & Rheum*. 1999;42(5):844-853

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Giant Cell Arteritis

Why is it an emergency?

- Blindness
  - Vision loss ~10% of patients
- Stroke
- Aortic aneurysm or dissection

- The sooner GCA is diagnosed and treated, the lower the incidence of visual loss
  - However, patients presenting with poor vision have little chance of recovery despite immediate steroid treatment
- The main goal of treatment is to prevent vision loss in the fellow eye
  - Usually occurs within days in 50% of cases of untreated GCA

Hayreh SS, Podhajsky PA, Zimmerman B. Ocular manifestations of giant cell arteritis. *Am J Ophthalmol*. 1998;125(4):509-20

Chen JJ, Leavitt JA, Fang C, Crowson CS, Matteson EL, Warrington KJ. Evaluating the incidence of arteritic ischemic optic neuropathy and other causes of vision loss from giant cell arteritis. *Ophthalmology* 2016: 123:1999-2003

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Giant Cell Arteritis

Ocular Manifestations

Symptoms

- Sudden visual loss
  - Most frequent symptom ~50% of cases
- Transient visual loss ~30%
  - Often followed by permanent visual loss
- Diplopia ~6%
- Eye pain ~8%

Signs

- AAION (6.9%) – 1.3 per 100,000 population
  - (+) RAPD
  - Pallid ON edema
  - (+/-) retinal cotton wool spots
  - Accounts for 85% of cases of permanent vision loss
- CRAO (1.6%)
- CilioRAO (0.4%)
- Posterior ION
- Ocular Ischemic Syndrome

Chen JJ, Leavitt JA, Fang C, Crowson CS, Matteson EL, Warrington KJ.

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Giant Cell Arteritis

Systemic Manifestations

Symptoms

- Jaw Claudication (48%)
- Neck pain (17%)
- Headache (57%)
- Scalp tenderness (20%)
- Unintended Weight loss (40%)
- Anorexia (31%)
- Myalgias (28%)
- Malaise (37%)

Signs

- Temporal artery tortuosity, prominence, and/or tenderness

20% of cases with permanent vision loss from GCA may present without systemic symptoms of GCA

Hayreh SS, Podhajsky PA, Zimmerman B. Occult giant cell arteritis: ocular manifestations. *Am J Ophthalmol*. 1998:125(4):521-526.

Chen JJ, Leavitt JA, Fang C, Crowson CS, Matteson EL, Warrington KJ. Evaluating the incidence of arteritic ischemic optic neuropathy and other causes of vision loss from giant cell arteritis. *Ophthalmology* 2016: 123:1999-2003

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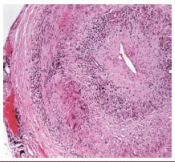
Giant Cell Arteritis

Diagnosis

- **Serological Studies**
  - Elevated ESR (85.7% sensitivity)
    - Normal in 9.2-14.3%
  - Elevated C-Reactive Protein (97.5% sensitivity)
    - Normal in 1.7%
    - ESR/CRP can also be elevated in infection and can
  - Abnormal CBC w/ differential (sensitivity <60%)
    - Thrombocytosis
    - Normocytic anemia
    - Leukocytosis
- **Fluorescein angiography**
  - Differentiate AAION from NAION
    - Choroidal hypoperfusion
    - Delayed choroidal filling
- **Temporal Artery Biopsy**
  - Gold Standard
    - BUT only 49-85% of patients with GCA have a (+) TAB
  - May need sequential if high clinical suspicion

Figure. **Temporal artery biopsy findings in giant cell arteritis include inflammatory infiltrates comprising lymphocytes, dendritic cells, macrophages, and multinucleated giant cells.**



The adventitia and media are the most intense sites of inflammation.

Lemos J, Eggenberger E. Neuro-Ophthalmological emergencies. *Neurohospitalist*. 2015;5(4):223-233.
Information and image from:
Hoffman GS. Giant Cell Arteritis. *Ann Intern Med*. 2016;165(9):ITC65-80

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Giant Cell Arteritis

Differential Diagnosis

- Common or migraine headache
- Atherosclerosis of large vessels
  - NA-AION
- Takayasu arteritis
- Other forms of vasculitis
- Polymyalgia rheumatica
  - 30-50% of patients with GCA also have PMR
  - PMR is 2-3x more common than GCA

Hoffman GS. Giant Cell Arteritis. *Ann Intern Med*. 2016;165(9):ITC65-80

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Treatment

- Steroids – IMMEDIATELY once AAION is suspected
  - Oral (60mg po) vs. IV ASAP
  - Oral taper over months to year
- Low dose Aspirin?
  - To reduce risk of ischemic events if no contraindications
- Consult PCP/internist
  - Monitor for steroid-related complications – hypertension, diabetes, osteoporosis, infection, etc.
- Smoking cessation
- Follow up – 2-4weeks

- Only 4% of patients will improve visual loss with steroids
- 4% of patients lose vision within the first 5 days, even on steroid treatment

Hayreh SS, Biousse V. Treatment of acute visual loss in giant cell arteritis: should we prescribe high-dose intravenous steroids or just oral steroids? *J Neuroophthalmol*. 2012;32(3):278-287.

Hayreh SS, Zimmerman B. Visual deterioration in giant cell arteritis patients while on high doses of corticosteroid therapy. *Ophthalmology*. 2003;110(6):1204-1215

Hayreh SS, Zimmerman B, Kardon RH. Visual improvement with corticosteroid therapy in giant cell arteritis. Report of a large study and review of literature. *Acta Ophthalmol Scand*. 2002;80(4):355-67

Jivraj I, Tamhankar M. The treatment of giant cell arteritis. *Curr Treat Options Neurol*. 2017;19(2):1-18.

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Migraine Mimickers

Swollen Optic Nerve(s)

Papilledema
- "Disc swelling from elevated intracranial pressure"

Differential Diagnosis
- Congenital Anomaly
  - Optic disc drusen
- Infection
- Inflammation
- Ischemia

Until lumbar puncture – better to diagnose "optic nerve head edema"

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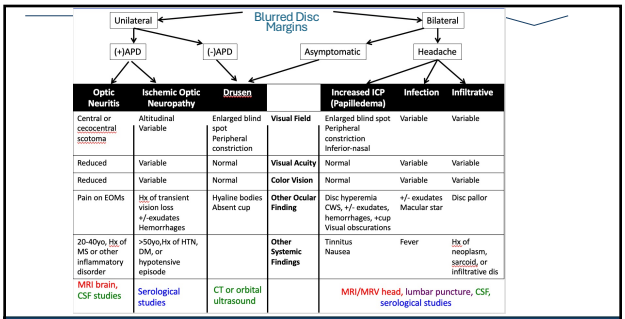

Optic Neuritis	Ischemic Optic Neuropathy	Drusen		Increased ICP (Papilledema)	Infection	Infiltrative
Central or cecocentral scotoma	Altitudinal Variable	Enlarged blind spot Peripheral constriction	**Visual Field**	Enlarged blind spot Peripheral constriction Inferior-nasal	Variable	Variable
Reduced	Variable	Normal	**Visual Acuity**	Normal	Variable	Variable
Reduced	Variable	Normal	**Color Vision**	Normal	Variable	Variable
Pain on EOMs	Hx of transient vision loss +/-exudates Hemorrhages	Hyaline bodies Absent cup	**Other Ocular Finding**	Disc hyperemia CWS, +/- exudates, hemorrhages, +cup Visual obscurations	+/- exudates Macular star	Disc pallor
20-40yo, Hx of MS or other inflammatory disorder	>50yo,Hx of HTN, DM, or hypotensive episode		**Other Systemic Findings**	Tinnitus Nausea	Fever	Hx of neoplasm, sarcoid, or infiltrative dis
MRI brain, CSF studies	Serological studies	CT or orbital ultrasound		MRI/MRV head, lumbar puncture, CSF, serological studies		

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Migraine Mimickers

Papilledema

Symptoms
- Headache
- Nausea
- Tinnitus
- Transient visual obscurations
- Double vision
- Other neurological deficits

Graves JS, Galetta SL. Acute Visual Loss and Other Neuro-Ophthalmologic Emergencies: Management. *Neurol Clin* 30 (2012): 75-99

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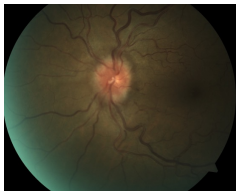
Papilledema



Signs
- Bilateral disc edema
  - Initially superior-inferior swelling
- Disc Hyperemia (its pink!)
- (+) Obscuration of retinal vessels over disc margin
- Cup is preserved
- (+) CWS/hemorrhages over time
- (-) Spontaneous Venous Pulsation
- Normal VA
- Normal Color Vision
- (+) Visual field defect
  - Enlarged blindspot
  - Peripheral field constriction

Graves JS, Galetta SL. Acute Visual Loss and Other Neuro-Ophthalmologic Emergencies: Management. *Neurol Clin* 30 (2012): 75-99

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Migraine Mimickers

Papilledema

Neuro-imaging
- Immediate Neuroimaging
- Lumbar puncture
  - Elevated intracranial (CSF) pressure

Etiology
- Mass lesion
  - Subdural hematoma
  - Epidural hematoma
  - Subarachnoid hemorrhage
- Severe cerebral edema
- Venous thrombosis
- Hydrocephalus
- Tension pneumocephalus
- AV malformation
- Pseudo-tumor Cerebri

Graves JS, Galetta SL. Acute Visual Loss and Other Neuro-Ophthalmologic Emergencies: Management. *Neurol Clin* 30 (2012): 75-99

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Migraine Mimickers

Pseudo-Tumor Cerebri Idiopathic Intracranial Hypertension

- Normal neuroimaging
  - MRI/MRV brain
- Normal CSF examination
- Elevated opening CSF pressure

Associations/Risk Factors
- Obesity
  - Recent weight gain
  - Obstructive sleep apnea
- Anemia
- h/o medication use:
  - Glucocorticoids
  - Vitamin A products
  - Tetrocycline derivatives
  - Synthetic growth hormones
- Female predilection

Graves JS, Galetta SL. Acute Visual Loss and Other Neuro-Ophthalmologic Emergencies: Management. *Neurol Clin* 30 (2012): 75-99

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Migraine Mimickers

IIH - Management

- Acetazolamide
  - May improve papilledema, visual complaints, headache
  - OR other diuretics/CAIs
- Weight loss
- Baseline automated VFs after treatment initiated
  - Progressive or severe vision loss may need more aggressive therapy
    - Ventriculoperitoneal shunt
    - Optic nerve fenestration

Banta JT, Farris BK. Pseudotumor cerebri and optic nerve sheath decompression. *Ophthalmology* 2000;107:1907–12.
Liu GT, Glaser JS, Schatz NJ. High-dose methylprednisolone and acetazolamide for visual loss in pseudotumor cerebri. *Am J Ophthalmol* 1994;118:88–96

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Migraine Management

Collaborative Care Models

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Pharmacological treatments for acute and preventive migraine management

Prophylactic

Abortive

Symptomatic

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Non-pharmacological interventions:

- Role of diet
- Lifestyle modifications
- Complementary therapies
  - Acupuncture
  - Physical therapy
    - TMJD
    - Cervical interventions
    - Photobiomodulation – cefaly/relivion
    - Vestibular
- Exercise

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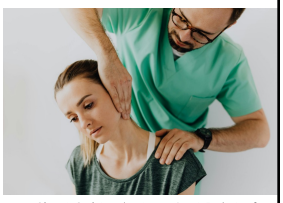
Physical Therapy

- MT + Exercise = pain intensity and QOL in CGH, Migraine, and TTH
- MT + Exercise = pain frequency in migraine
- MT + Exercise = disability in TTH
- Aerobic Exercise (continuous or HIIT) = pain intensity pain frequency in patients with migraine
- MT or a mixed intervention = pain intensity in TTH.



Herranz-Gómez, A., García-Pascual, I., Montero-Iniesta, P., Touche, R. L., & Paris-Alemany, A. (2021). Effectiveness of Exercise and Manual Therapy as Treatment for Patients with Migraine, Tension-Type Headache or Cervicogenic Headache: An Umbrella and Mapping Review with Meta-Meta-Analyses. *Applied Sciences*, *11*(15), 6854. https://doi.org/10.3390/app11156854

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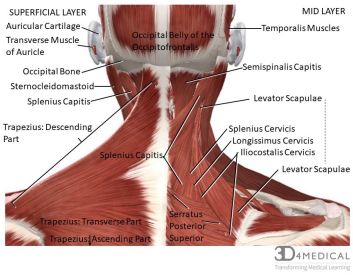
Cervicogenic

Best interventions:

- Dry needling + manipulations (intensity and duration)
- MET + Exercise (intensity)
- Soft Tissue Work + exercise (intensity)
- Dry needling + exercise (frequency)



Jung, A., Carvalho, G. F., Szikszay, T. M., Pawlowsky, V., Gabler, T., Luedtke, K. (2023). Physical Therapist Interventions to Reduce Headache Frequency, Duration, and Intensity in Adults With Episodic Migraine: A Systematic Review and Network Meta-Analysis. *Physical Therapy*, *103*(10), pzad091. https://doi.org/10.1093/ptj/pzad091

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Tension Type

Manual Therapy + Exercise = reduced headache frequency

Transcutaneous Electrical Stimulation + PT = reduced pain intensity

Jung, A., Eschke, R.-C., Struss, J., Taucher, W., & Luedtke, K. (2022). Effectiveness of physiotherapy interventions on headache intensity, frequency, duration and quality of life of patients with tension-type headache. A systematic review and network meta-analysis. *Cephalalgia*, 03331024221082073. https://doi.org/10.1177/03331024221082073

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Migraine

Physical Therapy that focuses on TMD + Aerobic exercise may be more effective than Aerobic Exercise + cervical interventions

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Lendraitiene, E., Smilgiene, L., Petruseviciene, D., & Savickas, R. (2021). Changes and Associations between Cervical Range of Motion, Pain, Temporomandibular Joint Range of Motion and Quality of Life in Individuals with Migraine Applying Physiotherapy: A Pilot Study. *Medicina*, *57*(6), 630. https://doi.org/10.3390/medicina57060630

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Vestibular Migraine

- Patients have dizziness and vestibular symptoms in addition to their headaches.
- Second most common cause of dizziness behind BPPV
- 2021 showed improvement in dizziness with vestibular rehab

Koc, A., & Cevizci Akkılıc, E. (2021). Effects of vestibular rehabilitation in the management of patients with and without vestibular migraine. *Brazilian Journal of Otorhinolaryngology*. https://doi.org/10.1016/j.bjorl.2021.07.011

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Who To Refer To:

- Cervicogenic → Ortho/Manual PT
- Migraine → Ortho/Manual PT → TMJ/TMD PT
- Vestibular Migraine → Vestibular PT
- Tension Type Headache → Ortho/Manual PT

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