

Visual Disturbances Following mTBI/concussion – What the Health Care Provider Needs to Know

**2016 TRAUMATIC BRAIN INJURY CONFERENCE
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Objectives:

Visual disturbance following m-TBI

1. Recognize pre-accident visual issues.
2. Mild concussion (m-TBI): common visual symptoms.
3. Rarer, more severe visual injuries.
4. Management of common m-TBI visual issues: less is more.
5. 'Neuro-optometry' – their concepts (PTVS, VMSS).
6. 'Neuro-optometry' – their devices, their therapies.
7. 'Neuro-optometry' – their literature, evidence.
8. A reality check.









Pre-accident visual issues

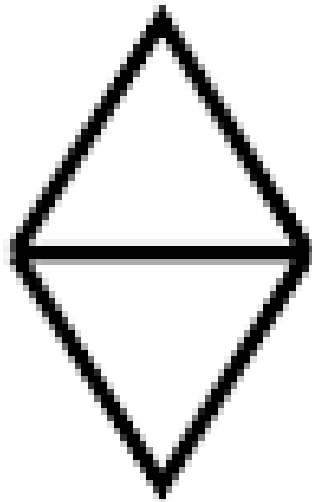
1. Refractive error: myopia, hyperopia, astigmatism.
2. Strabismus; amblyopia (lazy eye).
3. Congenital nystagmus.
4. Rare congenital palsies: Duane's congenital retraction syndrome.

Distance refractive error:
myopia / hyperopia / astigmatism

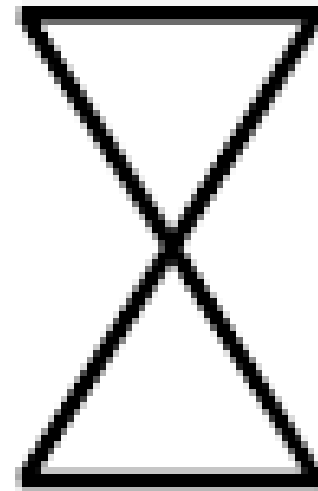


Distance refractive error -- corrective lenses:
Convex (for hyperopes) Concave (for myopes)

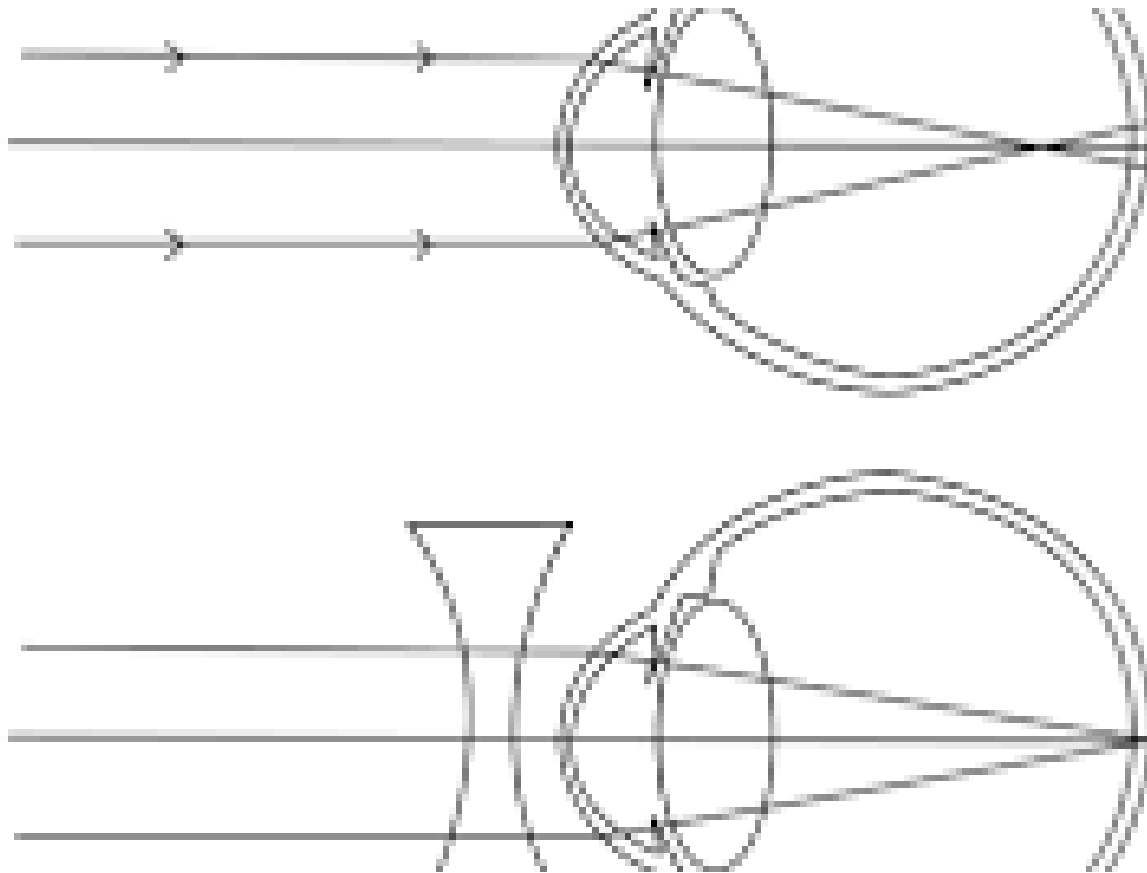
+ LENS



- LENS



Myopia: correction with concave (-) lens



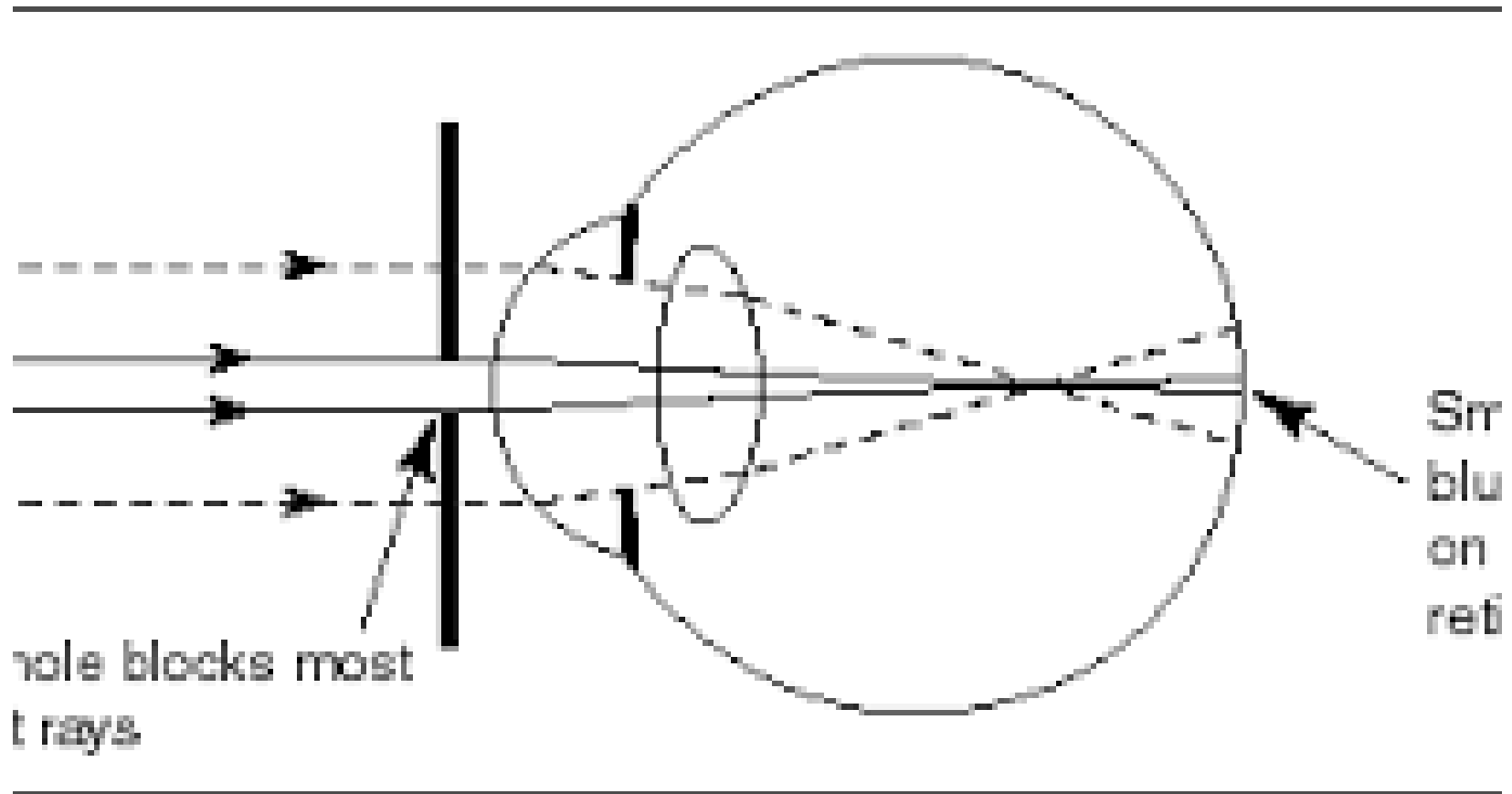
“Best-corrected” vision

What the neuro-ophthalmologist tries to obtain.

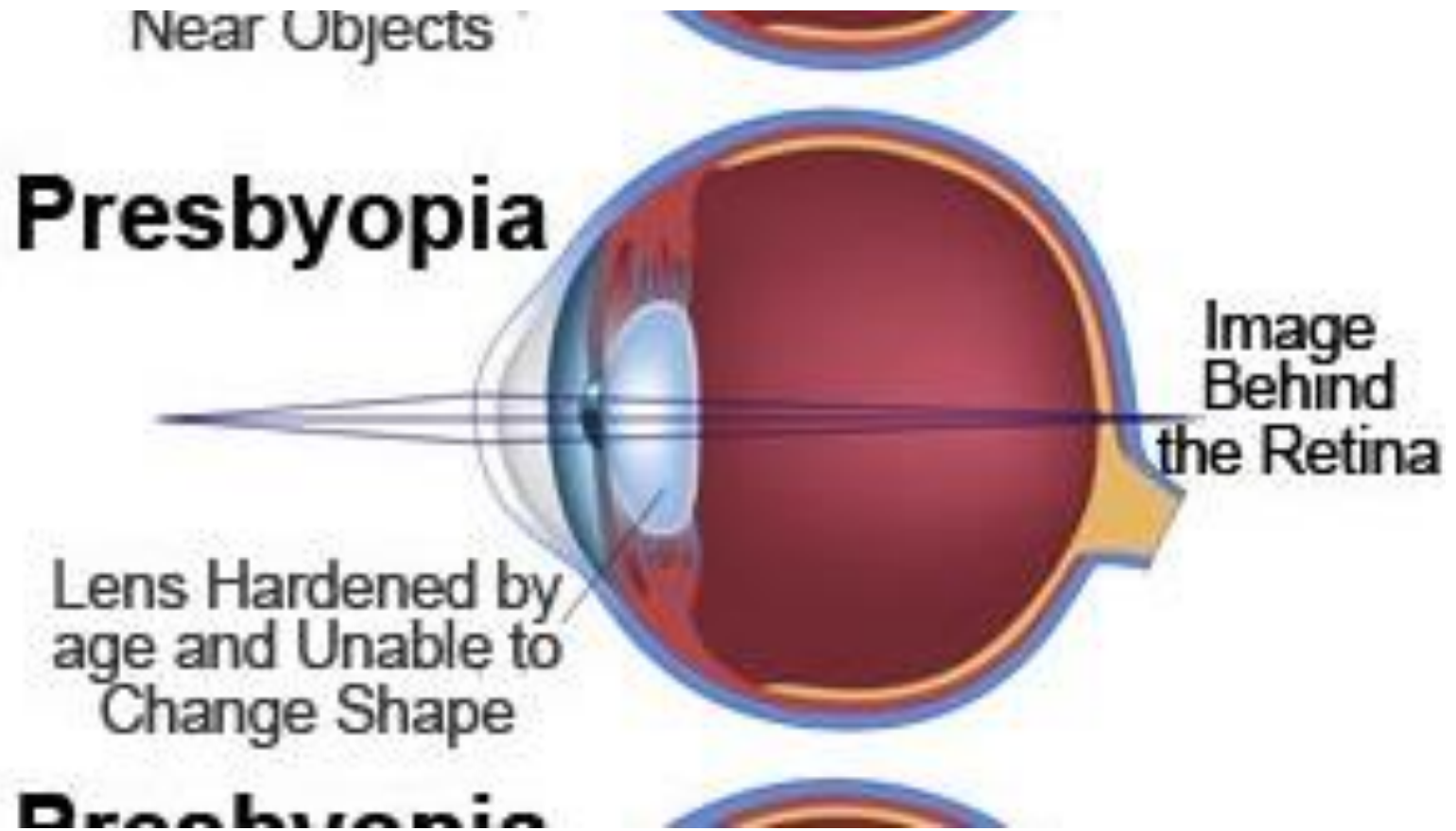
“What the brain (optic nerve) sees.”

Also, corrects for blurring / monocular diplopia/distortion.

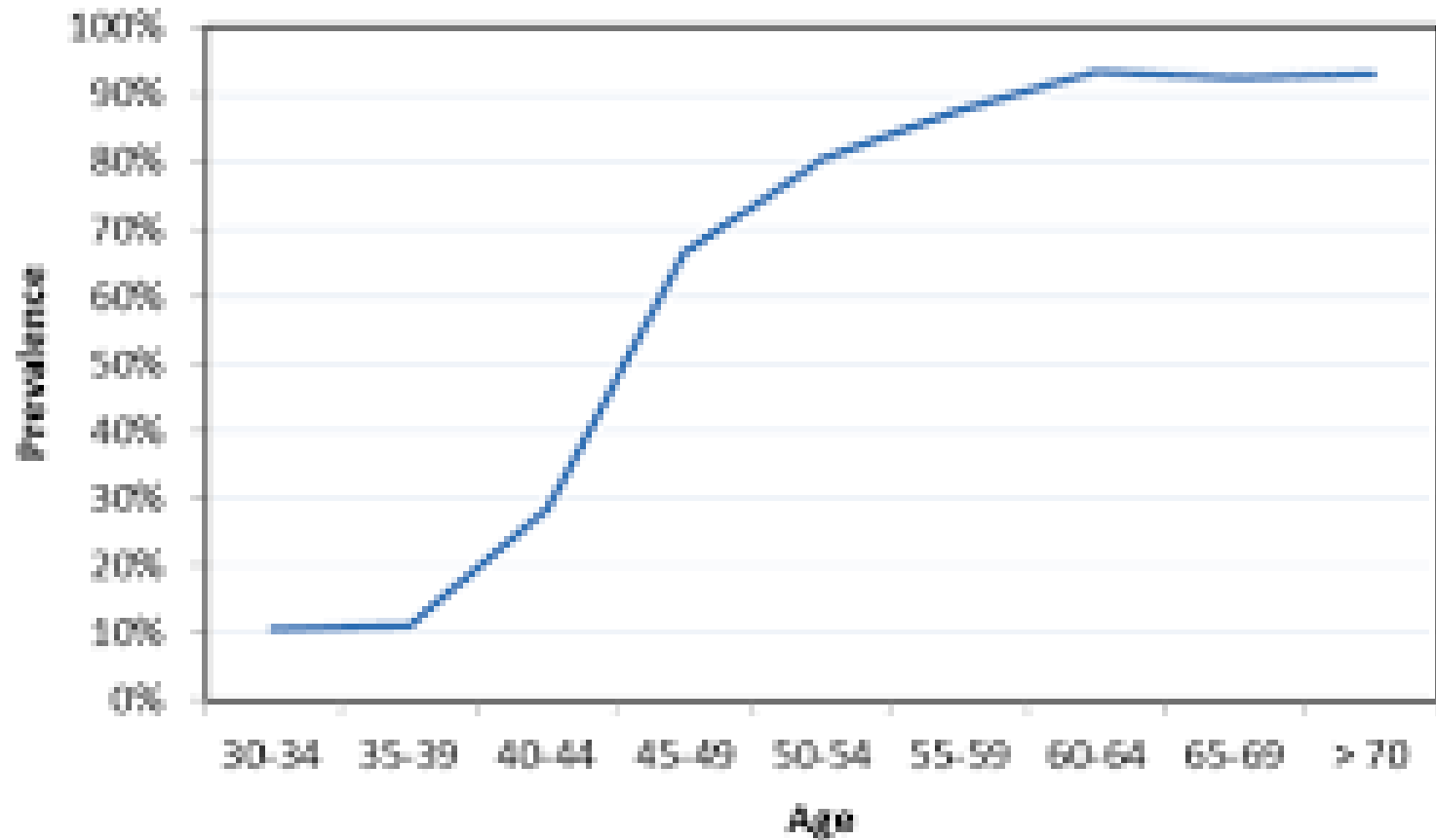
Pinhole -- how it sharpens uncorrected distance vision



Presbyopia: optics



Presbyopia: incidence with age



Near point of convergence (NPC)

Age (mean)	13 yr.	22 yr.	30 yr.
NPC (obj. break) (cm)	6.3	8.0	8.3

NPC recedes with age.

Abraham NG et al (2015)

Bifocal glasses



Pre-accident visual issues

1. Refractive error: myopia, hyperopia, astigmatism.

2.Strabismus; amblyopia (lazy eye).

3.Congenital nystagmus.

4.Rare congenital palsies: Duane's congenital retraction syndrome.

Exotropia: before



Exotropia: after



Duane's congenital retraction syndrome (Type I)



Duane retraction syndrome

- Absent or hypoplastic 6th nerve nuclei
- Aberrant innervation of lateral rectus
- ***Generally do not report diplopia***
- Amblyopia in 15%
- Type I (80%): limited abduction
- Type II (7%): limited adduction
- Type III (15%): both abduction and adduction limited

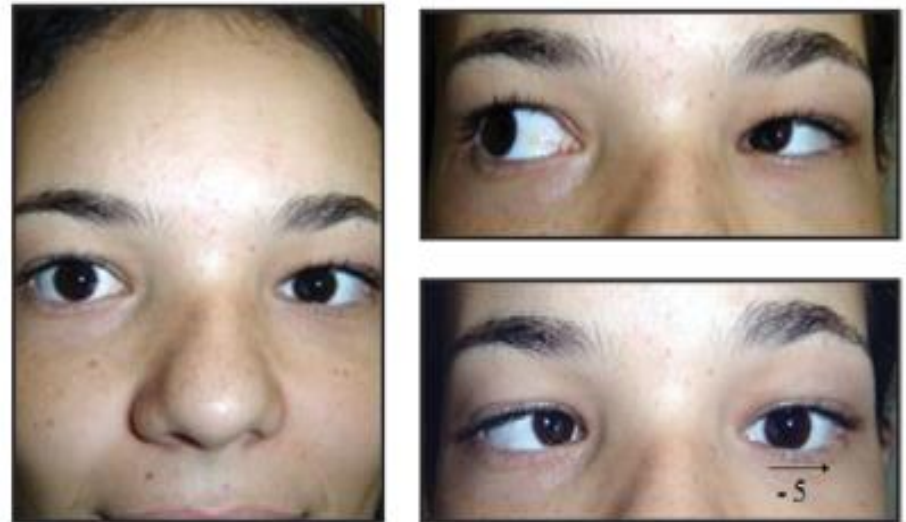
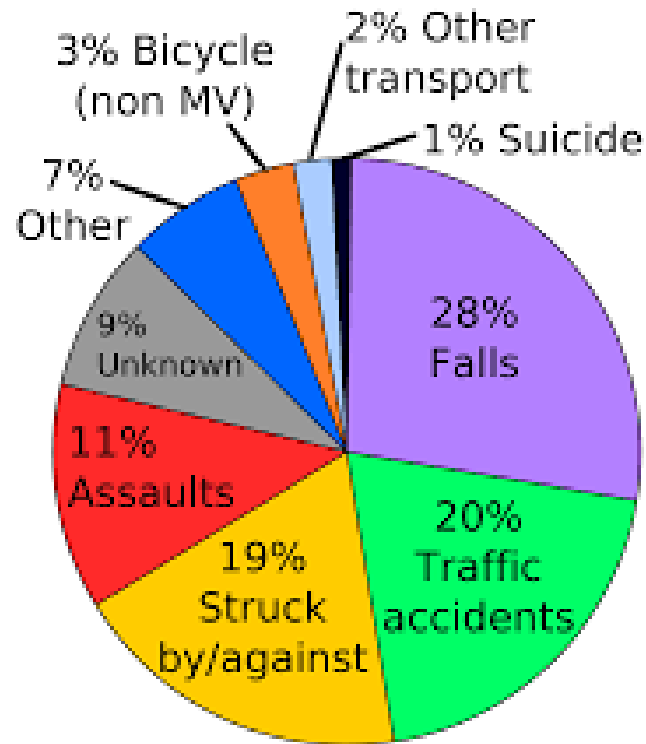


Figure 2: A little worse Duane's syndrome type I (eso Duane's); A little more fibers leave the medial rectus nerve toward the lateral rectus; Less asymmetric co-contraction; stronger retraction and small upshoot on adduction

TBI: causes

Traumatic brain injury causes



TBI: Vision impairments (common)

1. Photosensitivity.
2. Migraine (+ visual aura).
3. Convergence/accommodation insufficiency.

Cerebral effects presenting as visual impairment:
reduced concentration; visual fatigue;
impaired comprehension

Photosensitivity treatment = SG

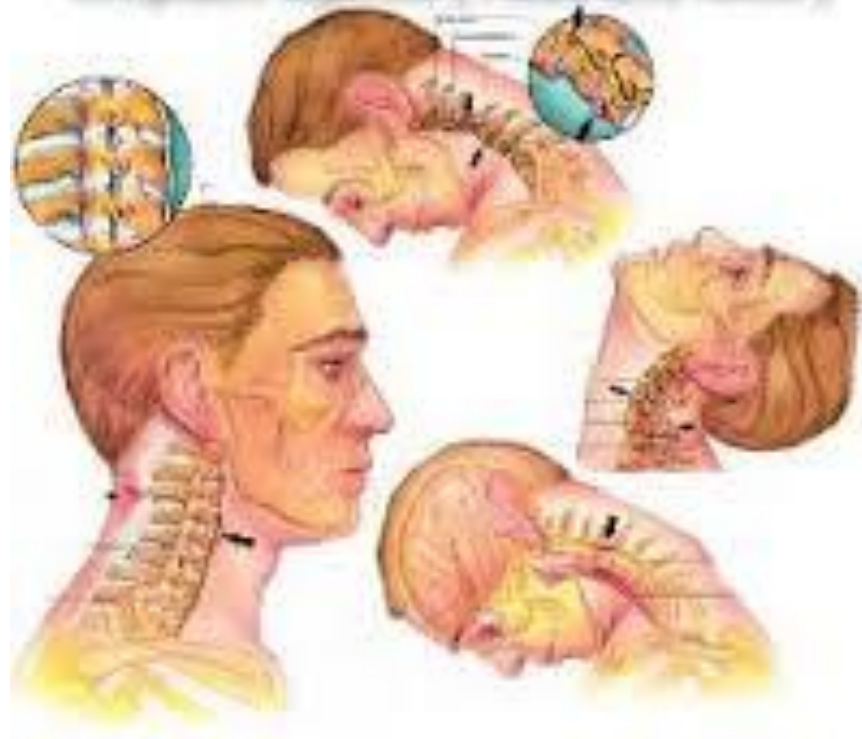


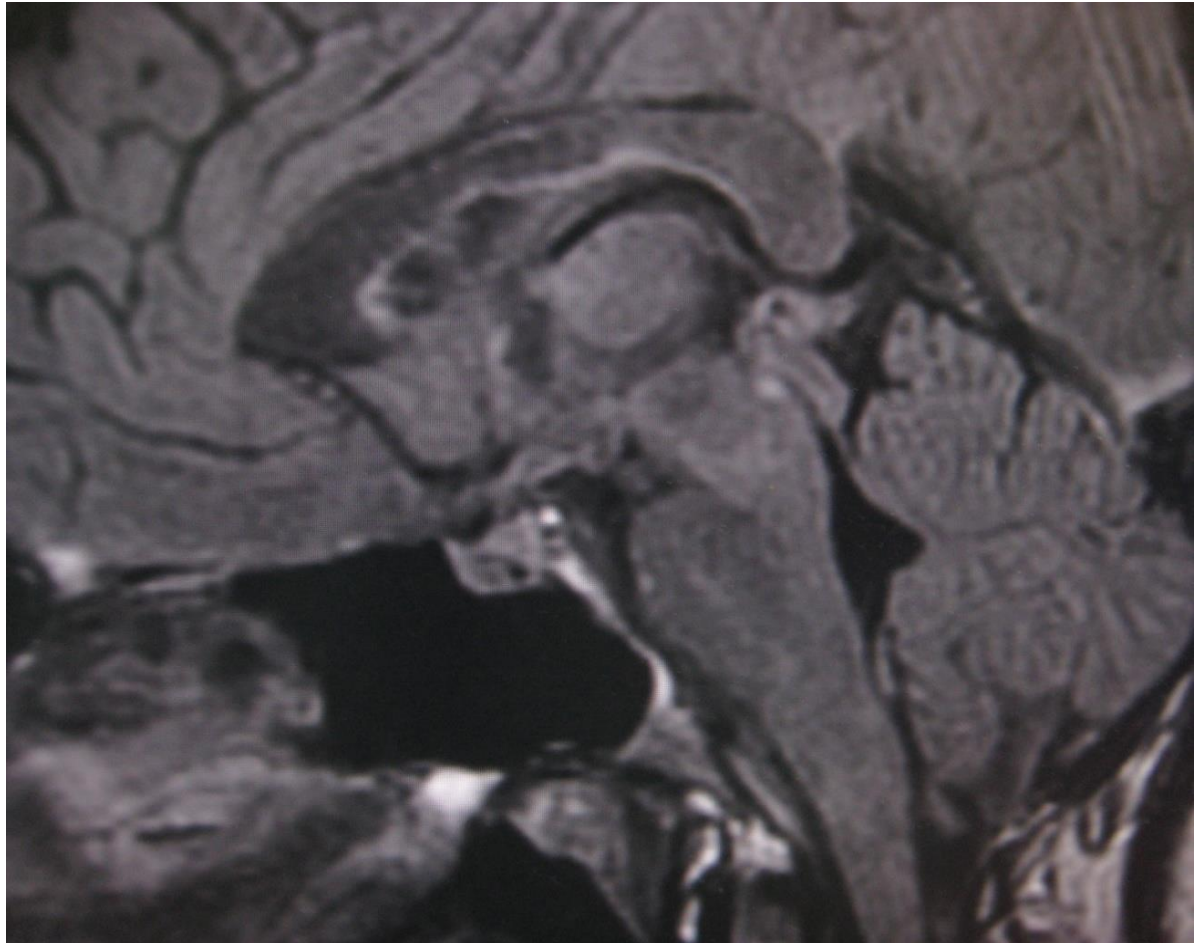
Migraine visual aura



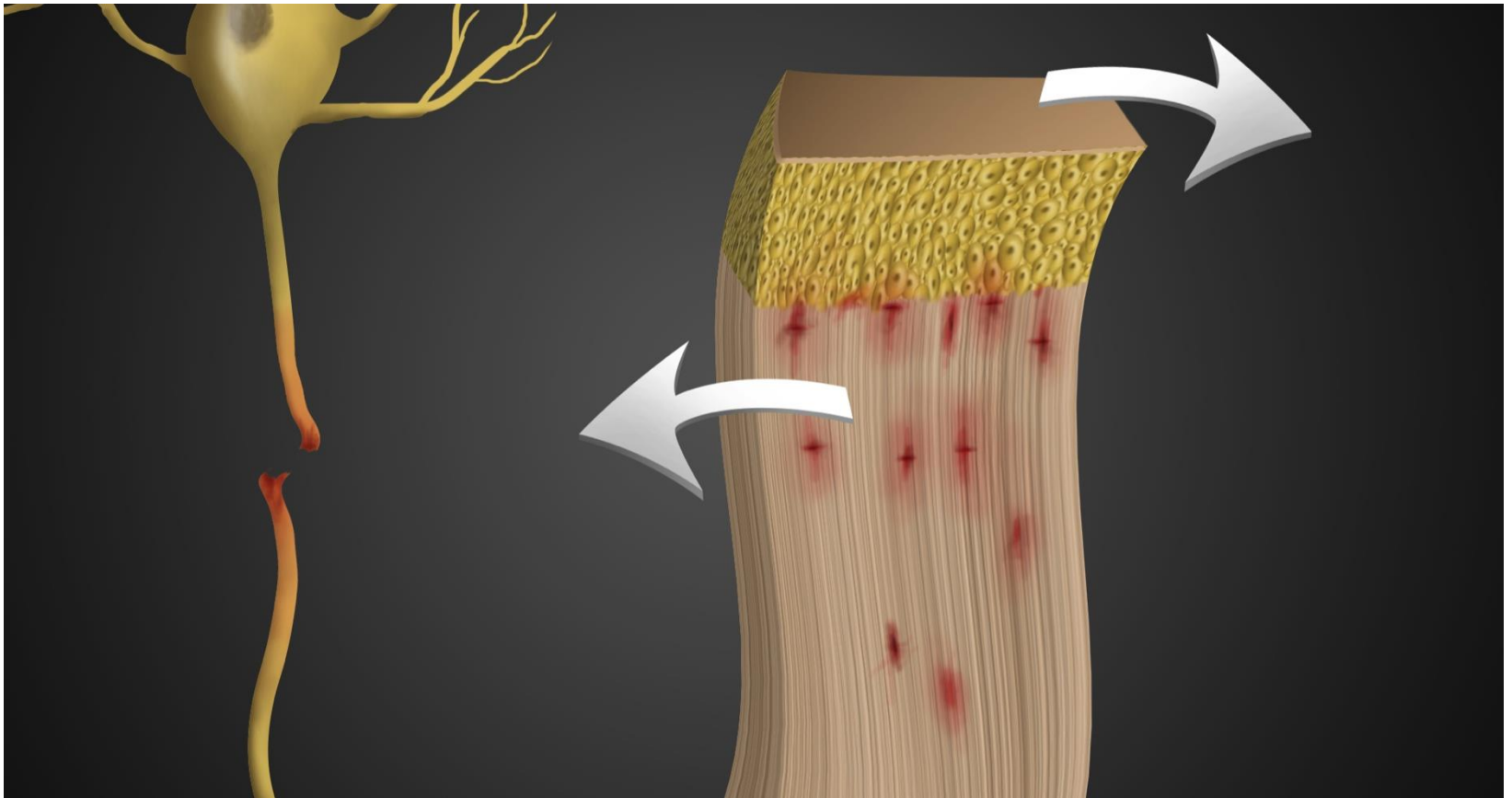
Whiplash extension-flexion force: potential effect on brainstem

Whiplash Injuries (Head and Neck)

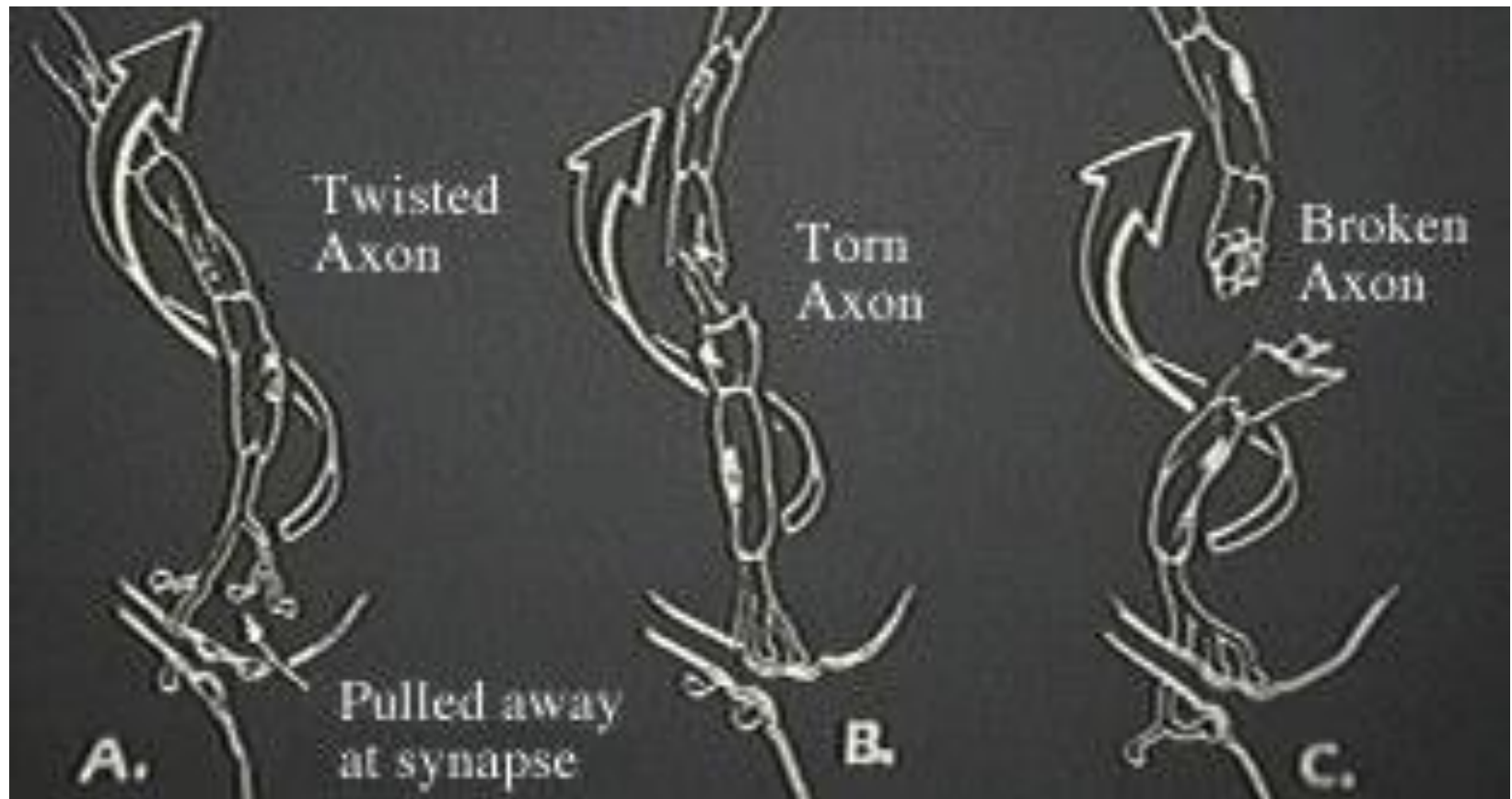




Diffuse axonal injury (DAI)



DAI: mechanical disruption of axons



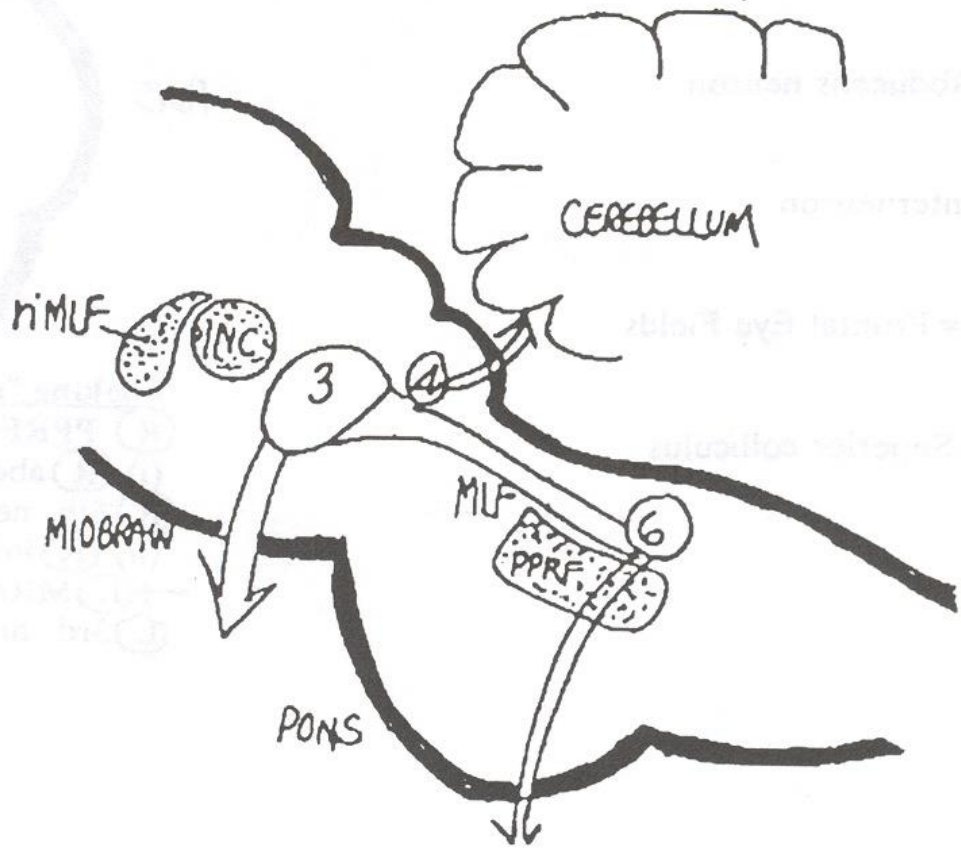
SNTF – a possible m-TBI biomarker?

- DAI deformation injures white matter axons.
- cytoskeleton extrudes . . . Enzymes cleave off N-terminal fragment from alpha-II spectrin.
- from extracellular space, it leaks into blood.

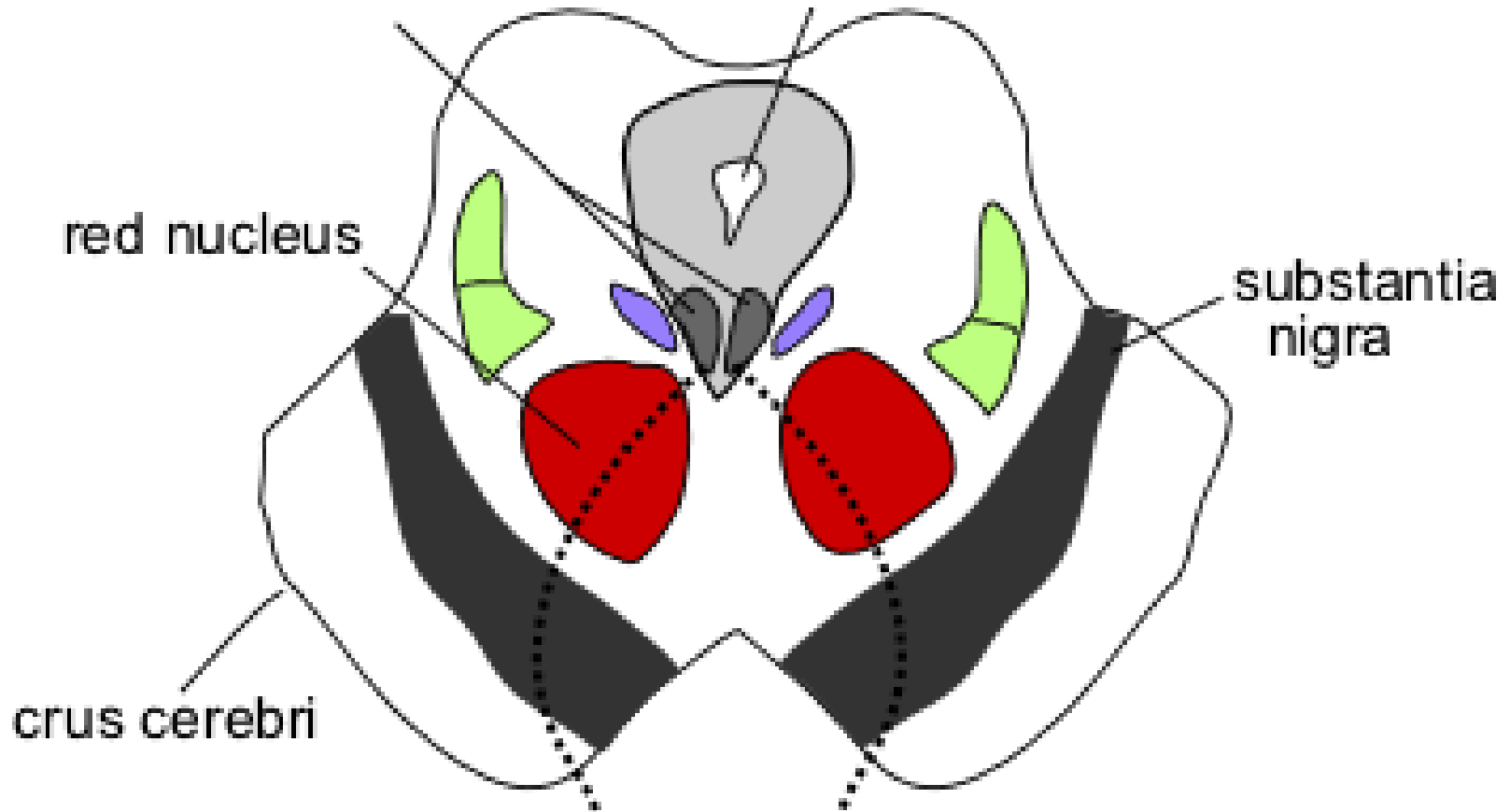
After severe-TBI, both APP (amyloid precursor protein) and SNTF localize to damaged axons.

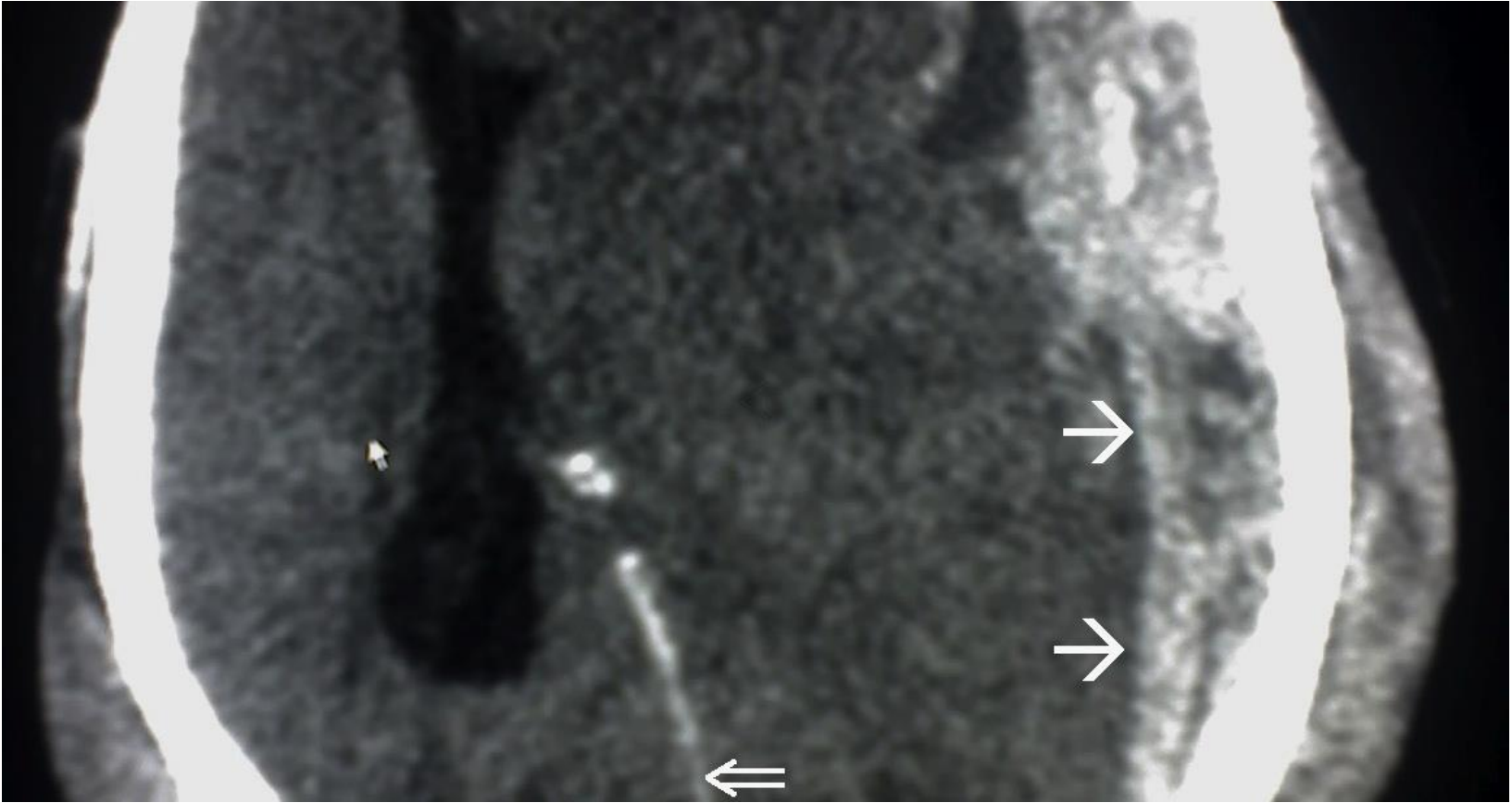
But after m-TBI, a subpopulation of axons is marked by SNTF, undetected by APP.

Brainstem
in sagittal
section



Edinger-Westphal nucleus: accommodation, convergence, pupillary constriction

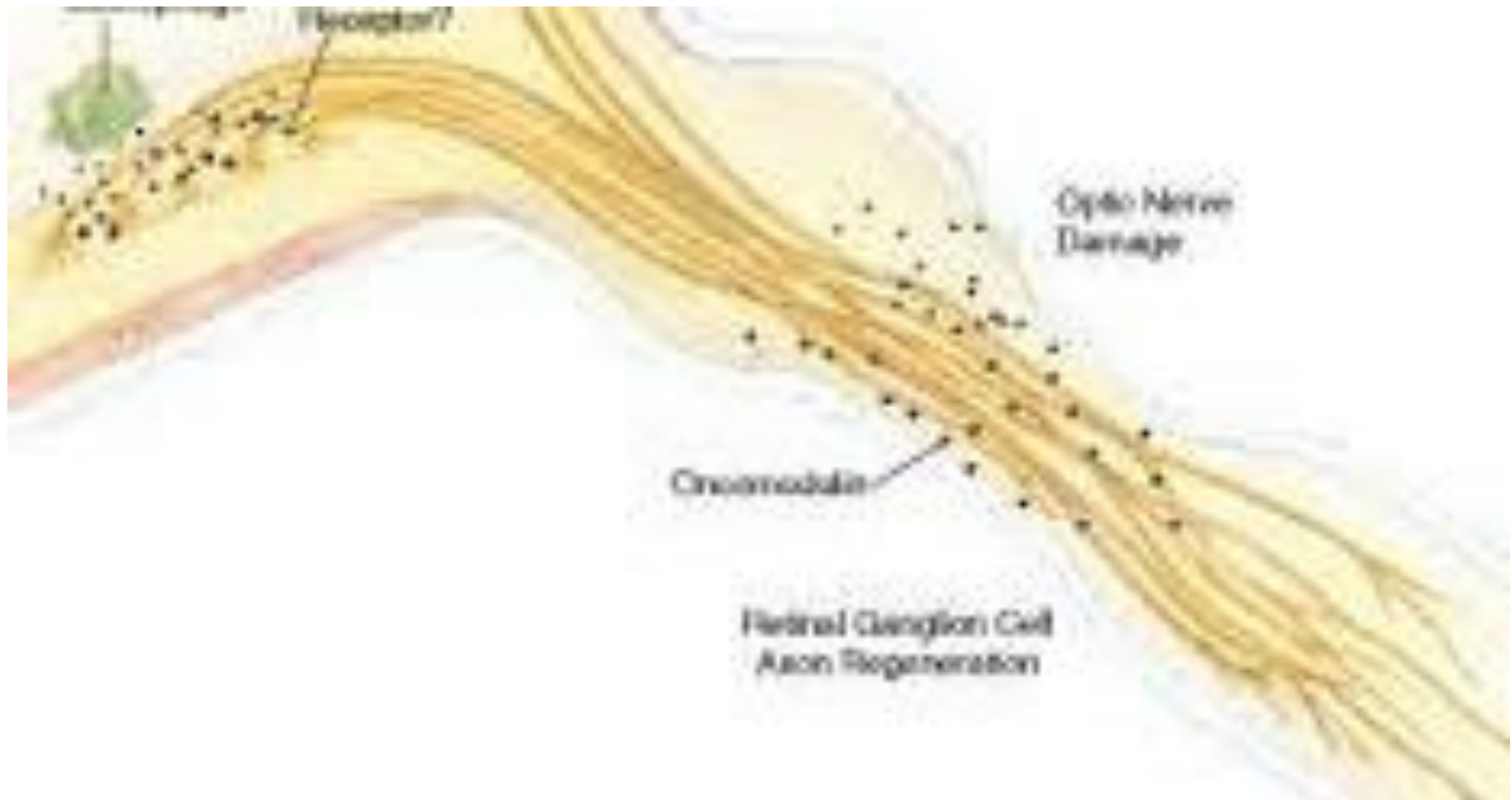




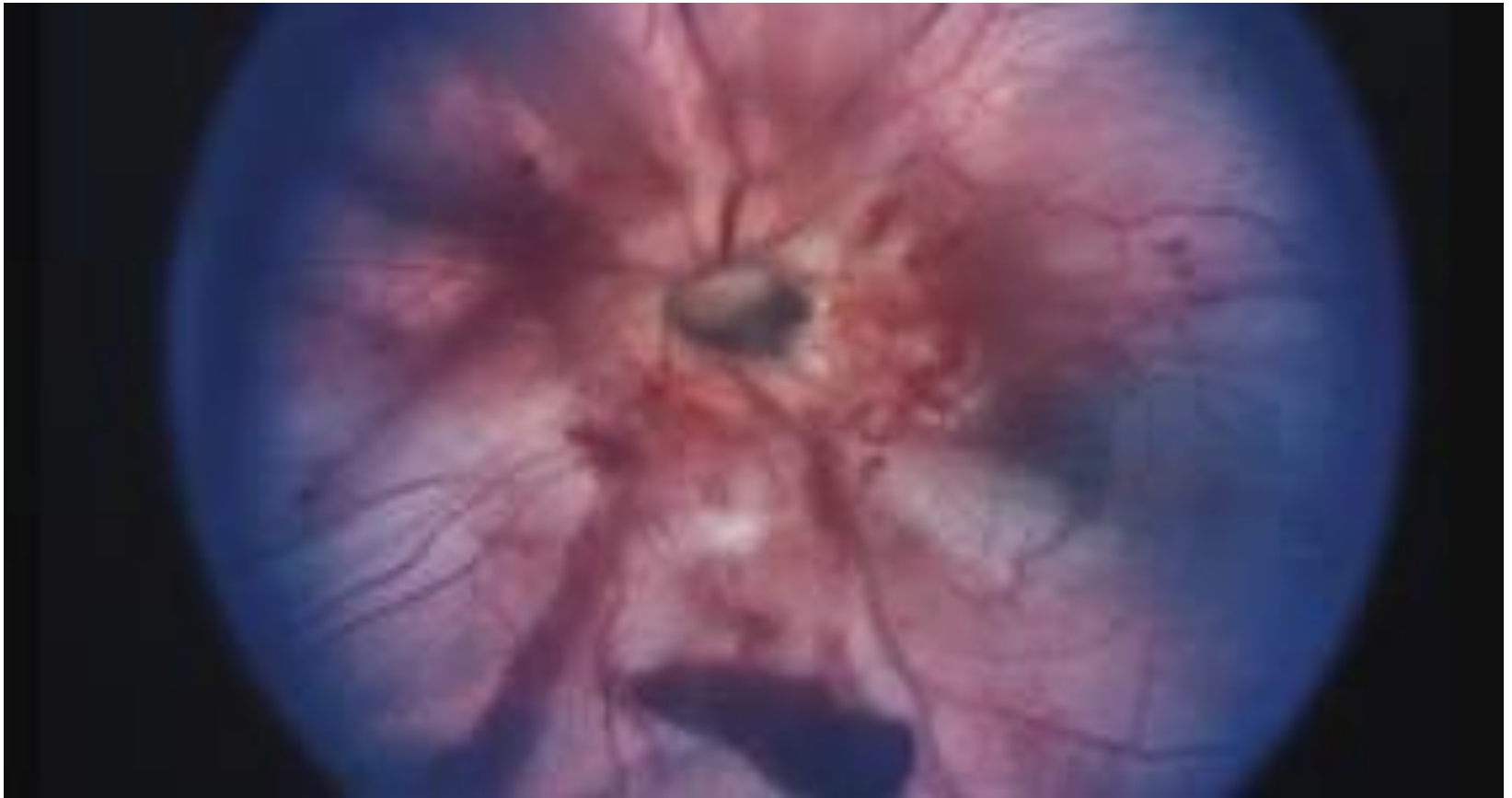
Severe TBI affecting vision: recognized syndromes

1. **Afferent vision: optic nerve.**
2. Afferent vision: central visual pathways
3. Diplopia: ocular misalignment
 1. Orbital/facial derangement
 2. Cranial neuropathy (4, 6, 3)
 3. Brainstem damage

Traumatic optic neuropathy



Traumatic optic neuropathy



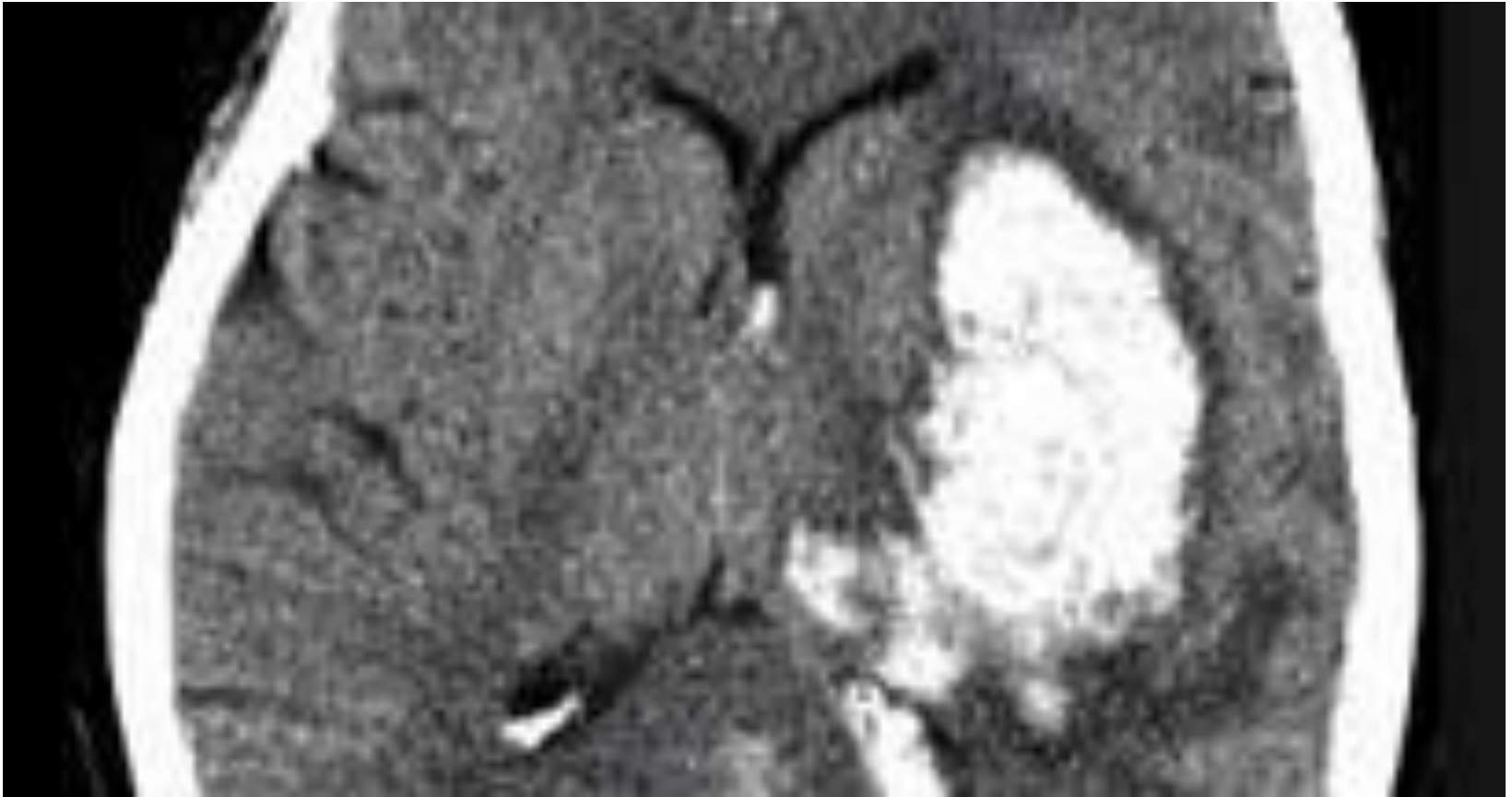
Late optic nerve trauma: pallor



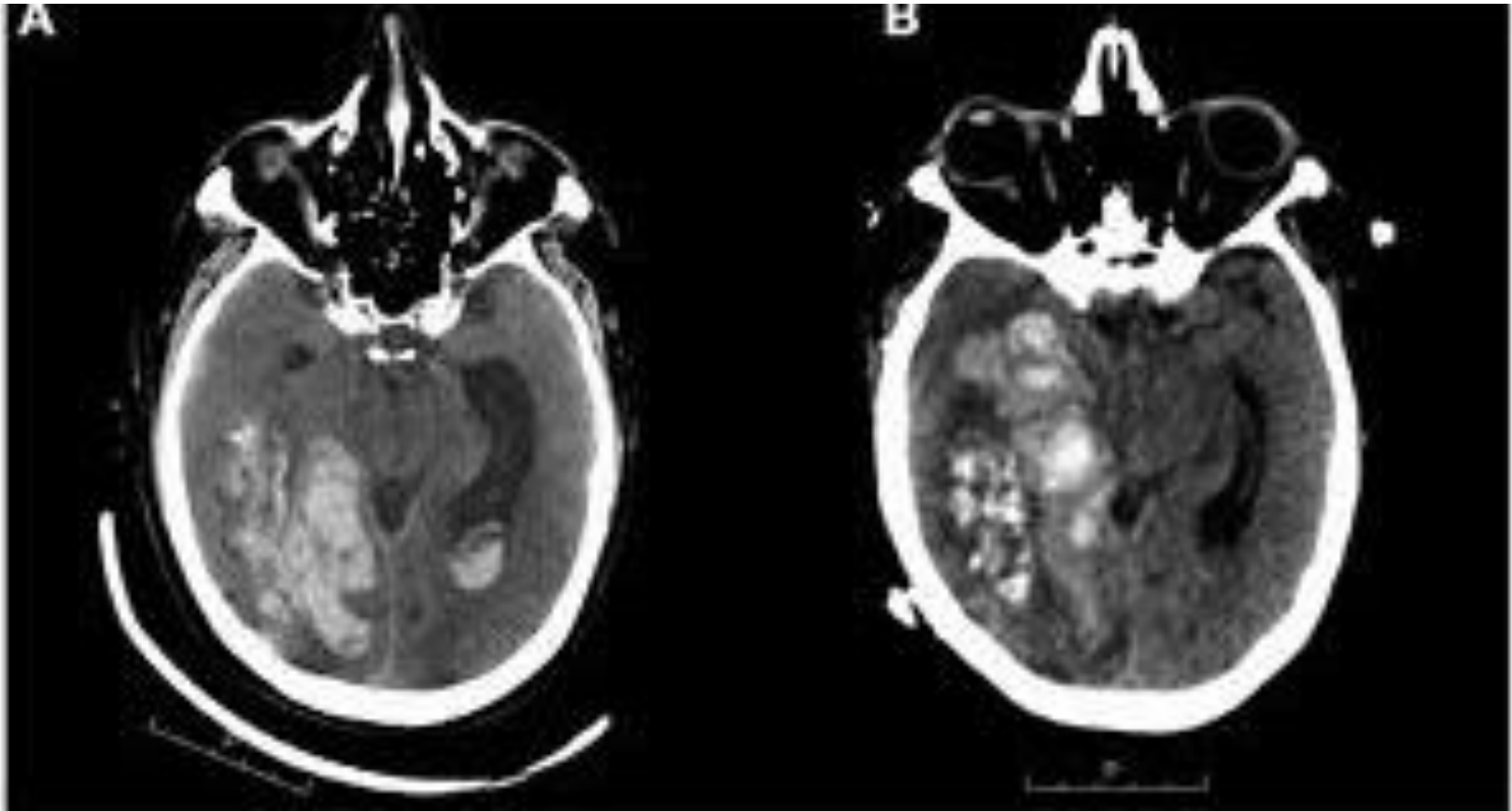
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Trauma: Visual radiations



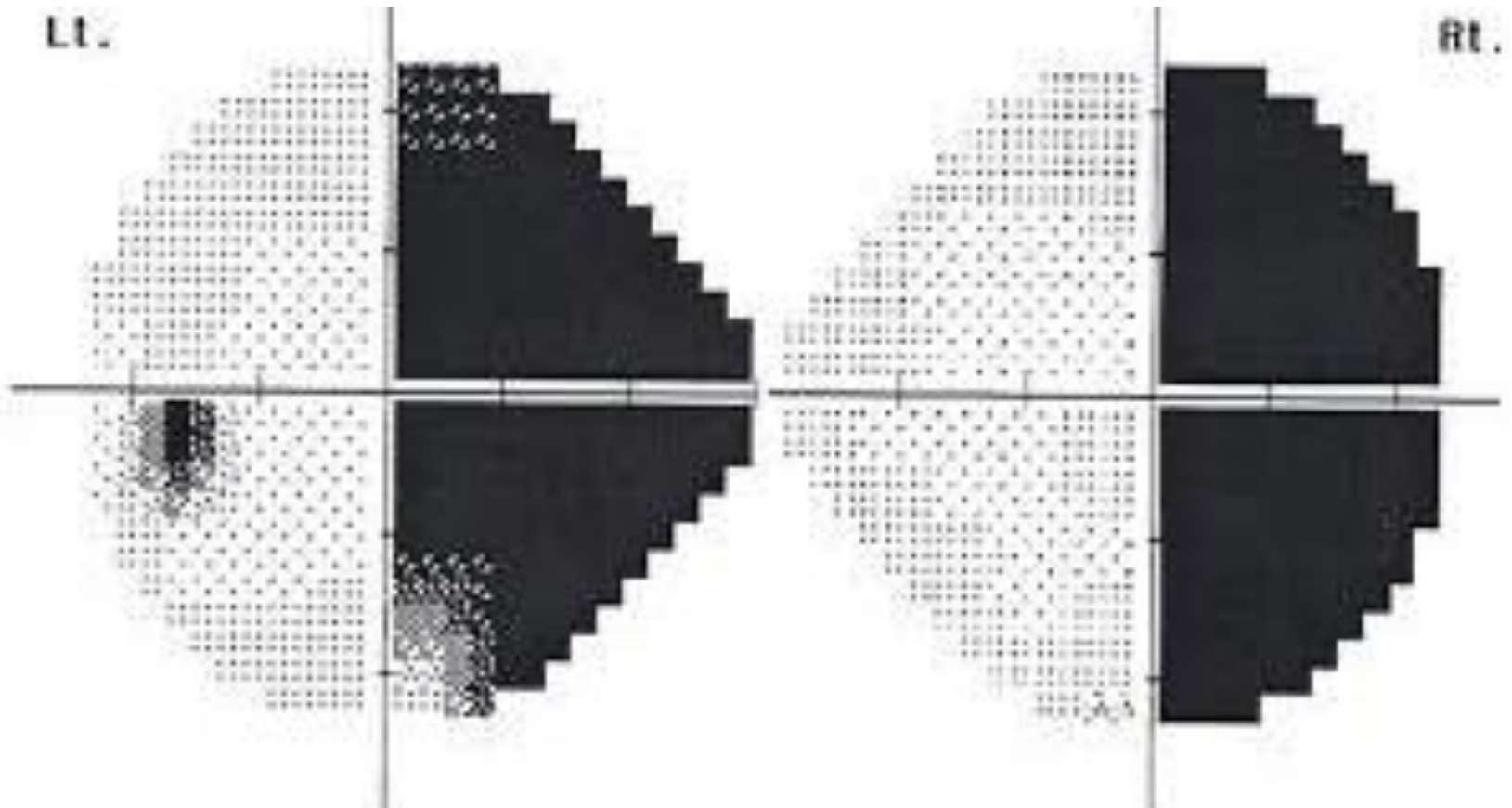
Trauma: occipital cortex



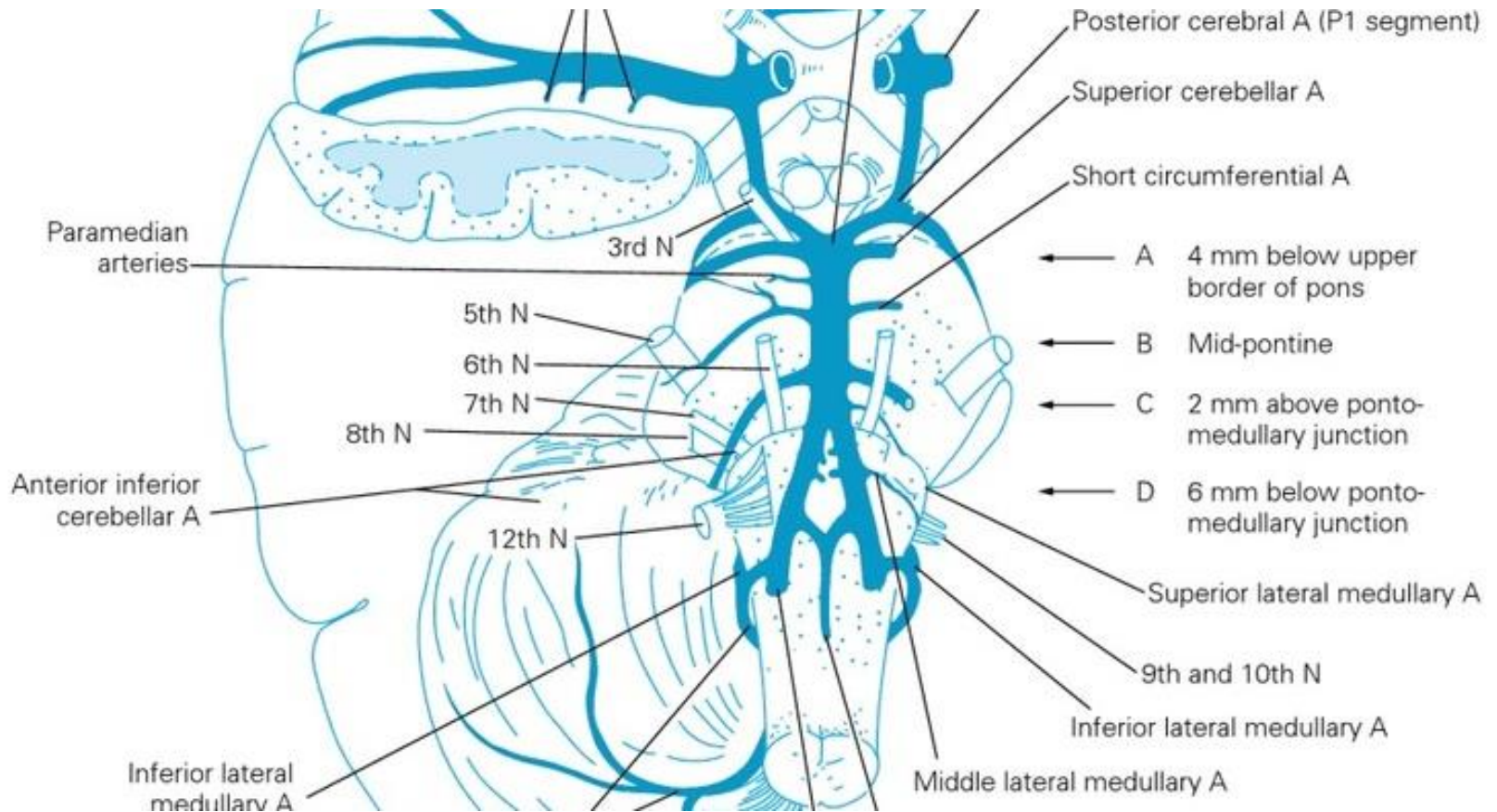
A patient with homonymous hemianopia (artistic rendering)



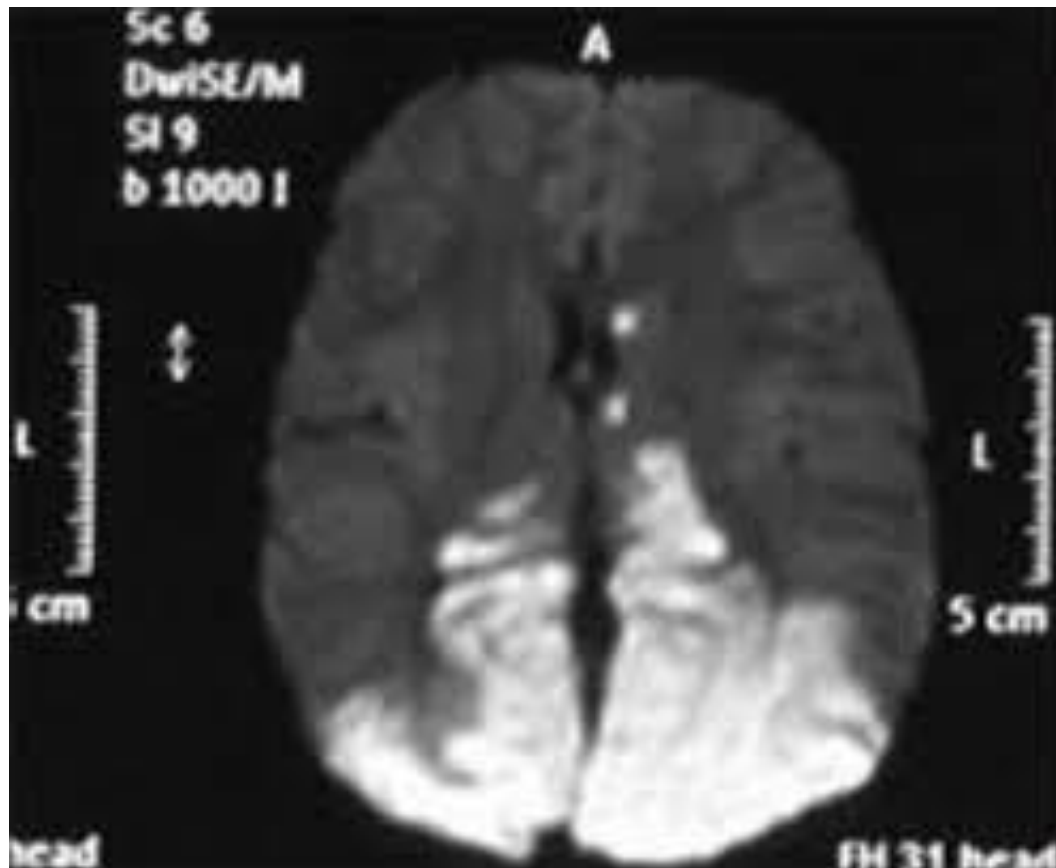
HVF: Homonymous hemianopia



Brain swelling in trauma: rostral-caudal herniation compresses both posterior cerebral arteries



Bilateral occipital lobe infarction: cortical blindness



Trauma affecting vision: recognized syndromes

1. Afferent vision: optic nerve; central visual pathways.
- 2. Diplopia: ocular misalignment**
 - 1. Orbital/facial derangement**
 - 2. Cranial neuropathy (4, 6, 3)**
 - 3. Brainstem damage**
3. Convergence/accomodation impairment
4. Photosensitivity.

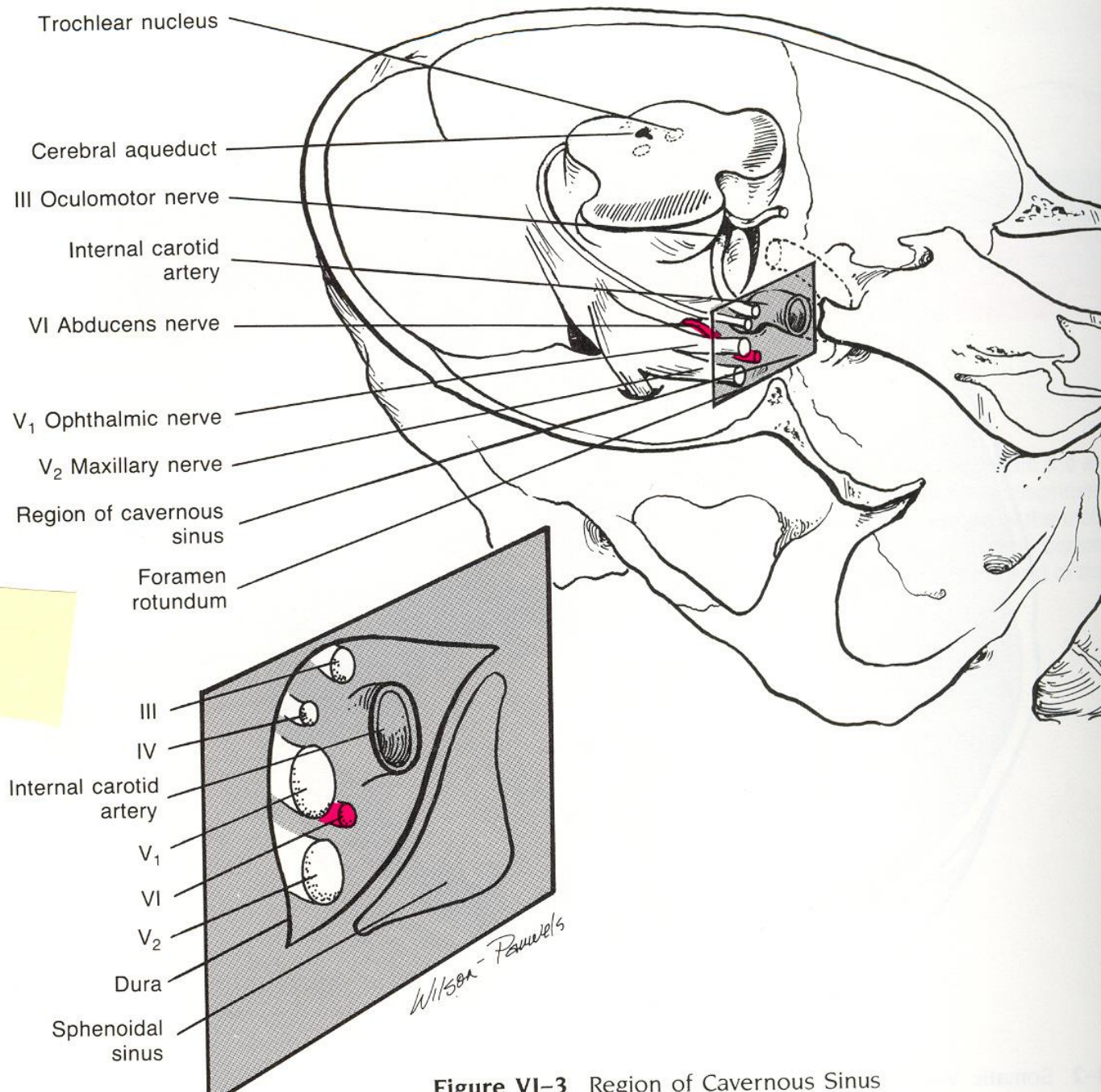


Figure VI-3 Region of Cavernous Sinus

6th Cranial nerve palsy

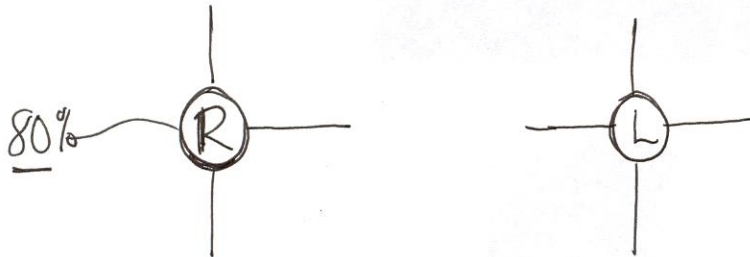
Younger **Inflammatory**

Older **Ischemic**

Any age **Traumatic**

Less common: tumour, aneurysm, MS

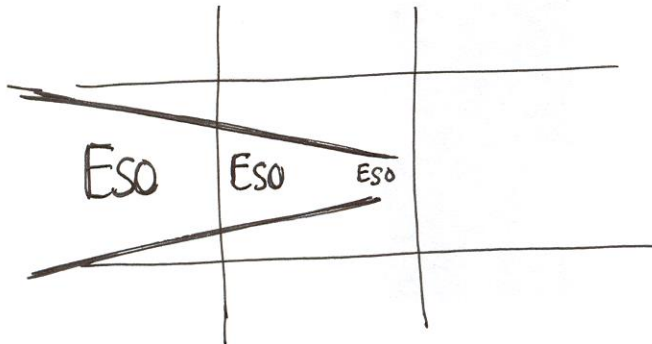
Diplopia: horizontal case



- Limited (R) abduction

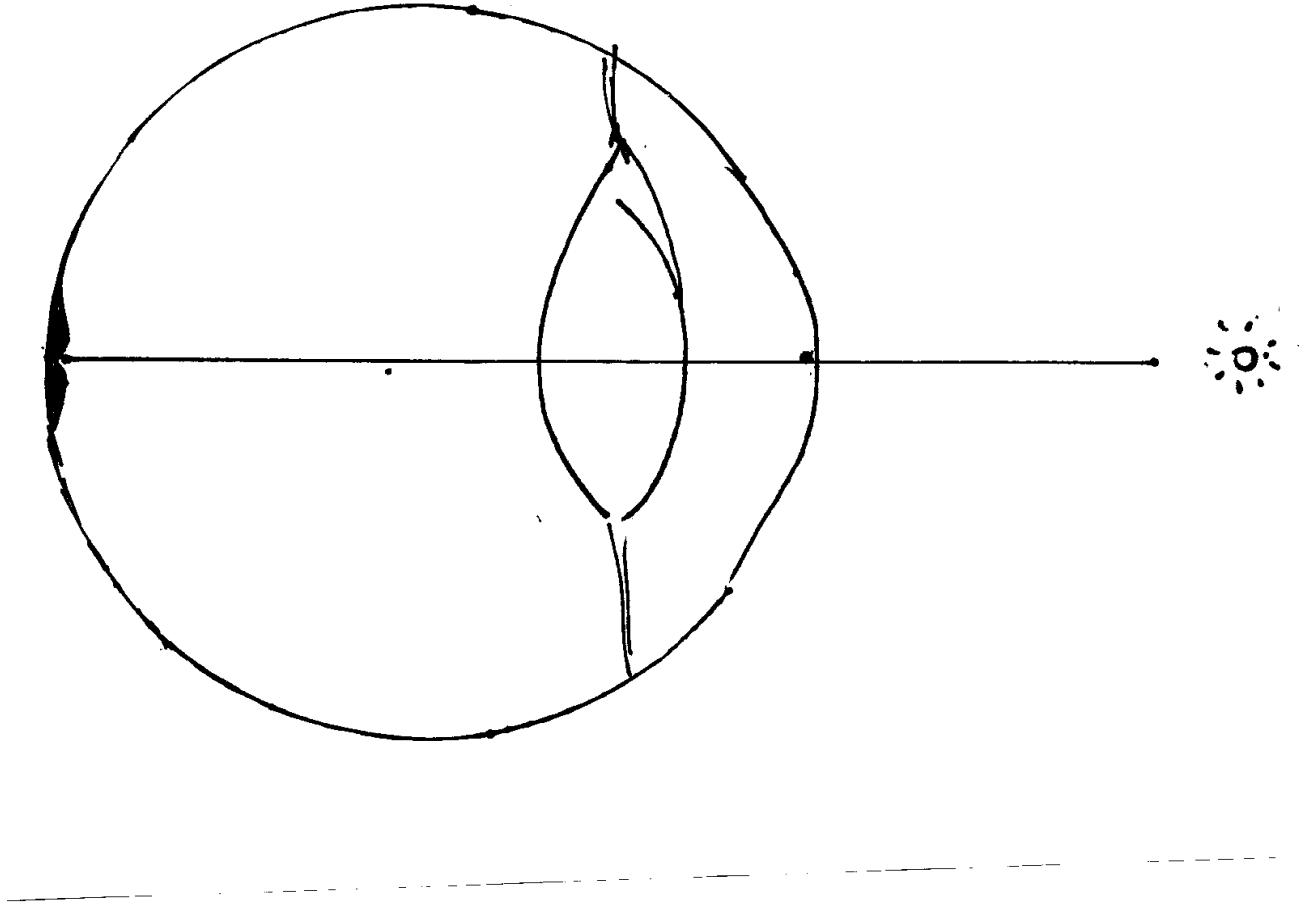
– = (R) LR weakness

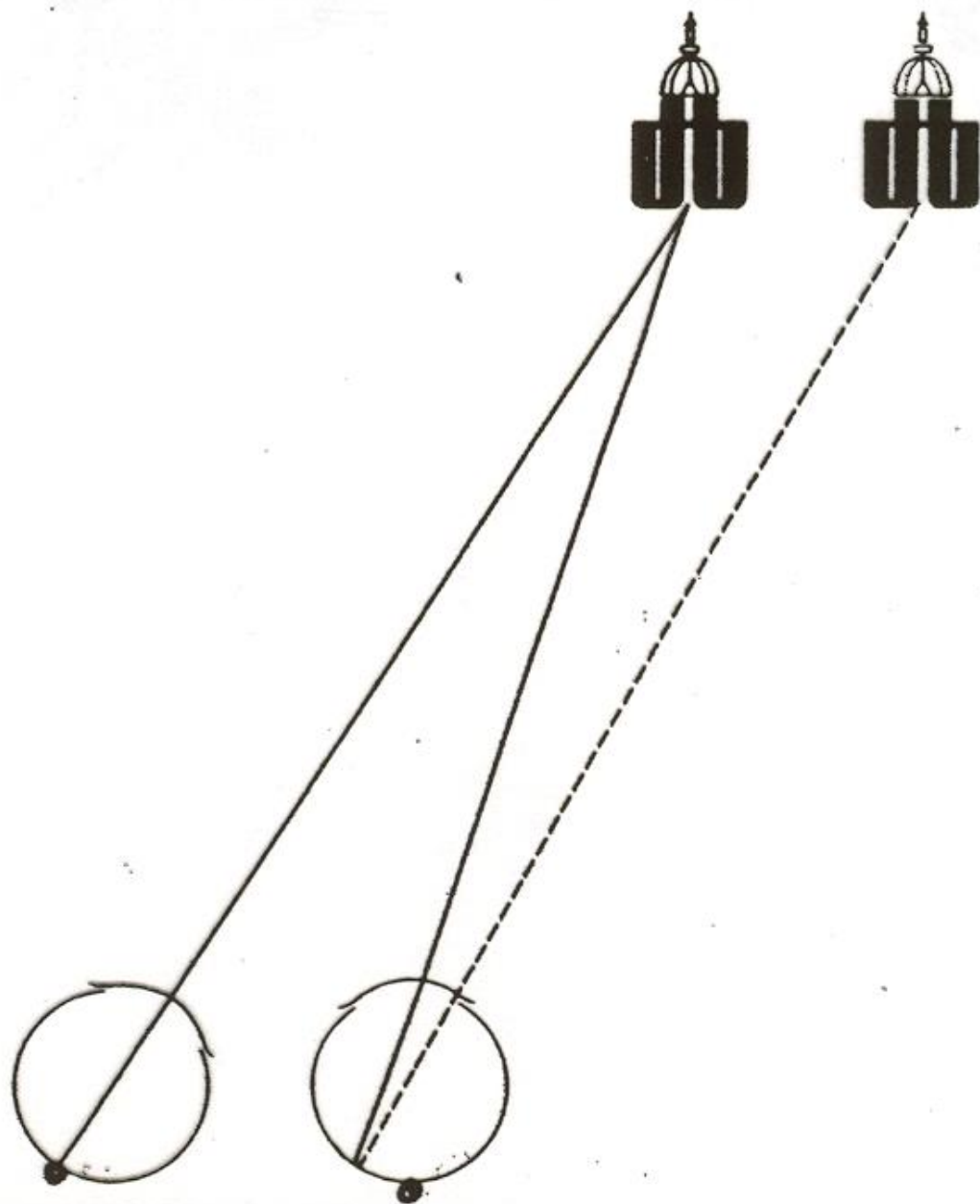
– Confirmed with incommittant esotropia, max. in (R) gaze



Giant aneurysm of intra-cavernous carotid artery







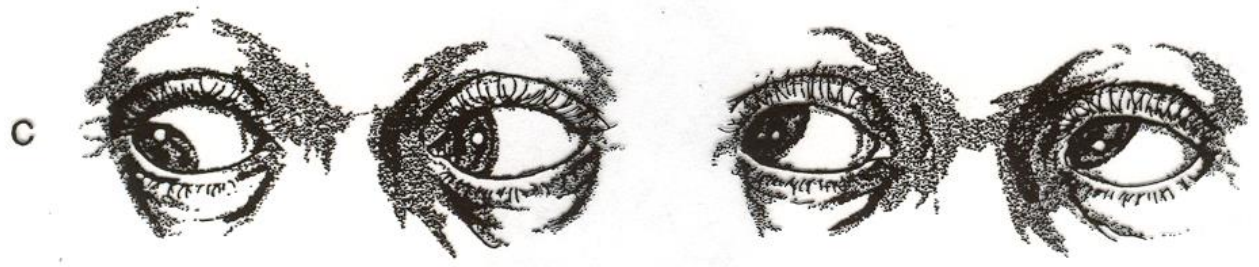
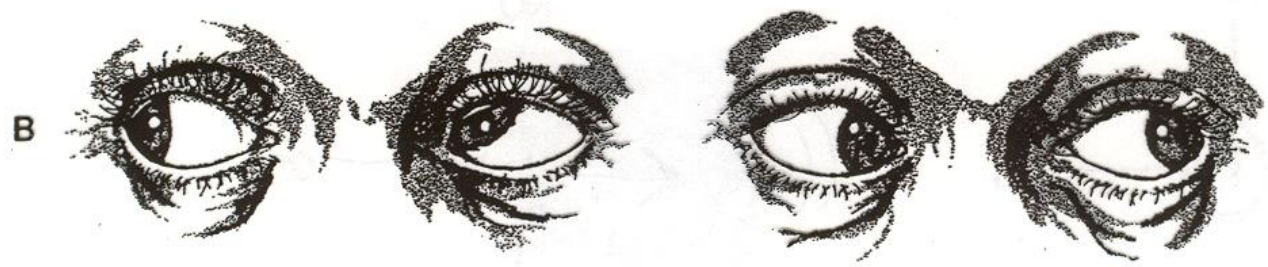
4th Cranial nerve palsy

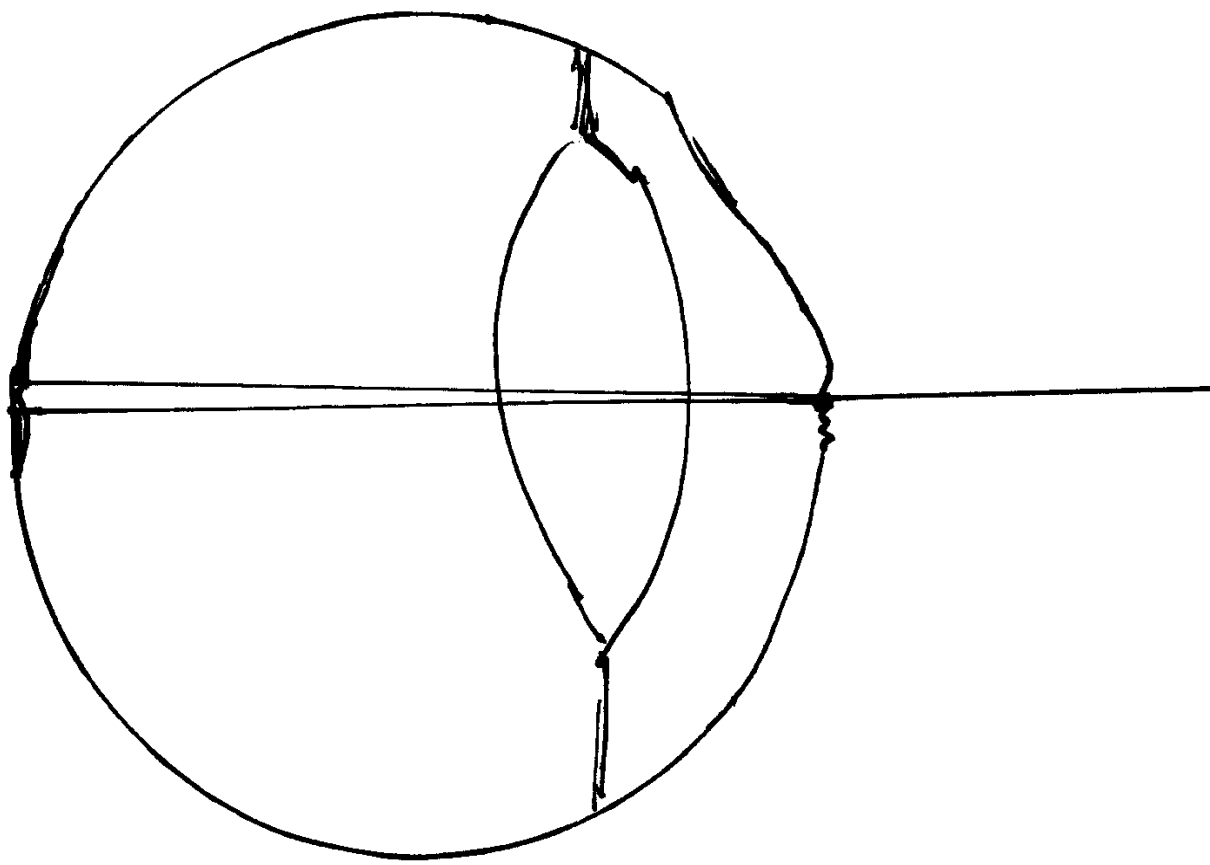
Trauma

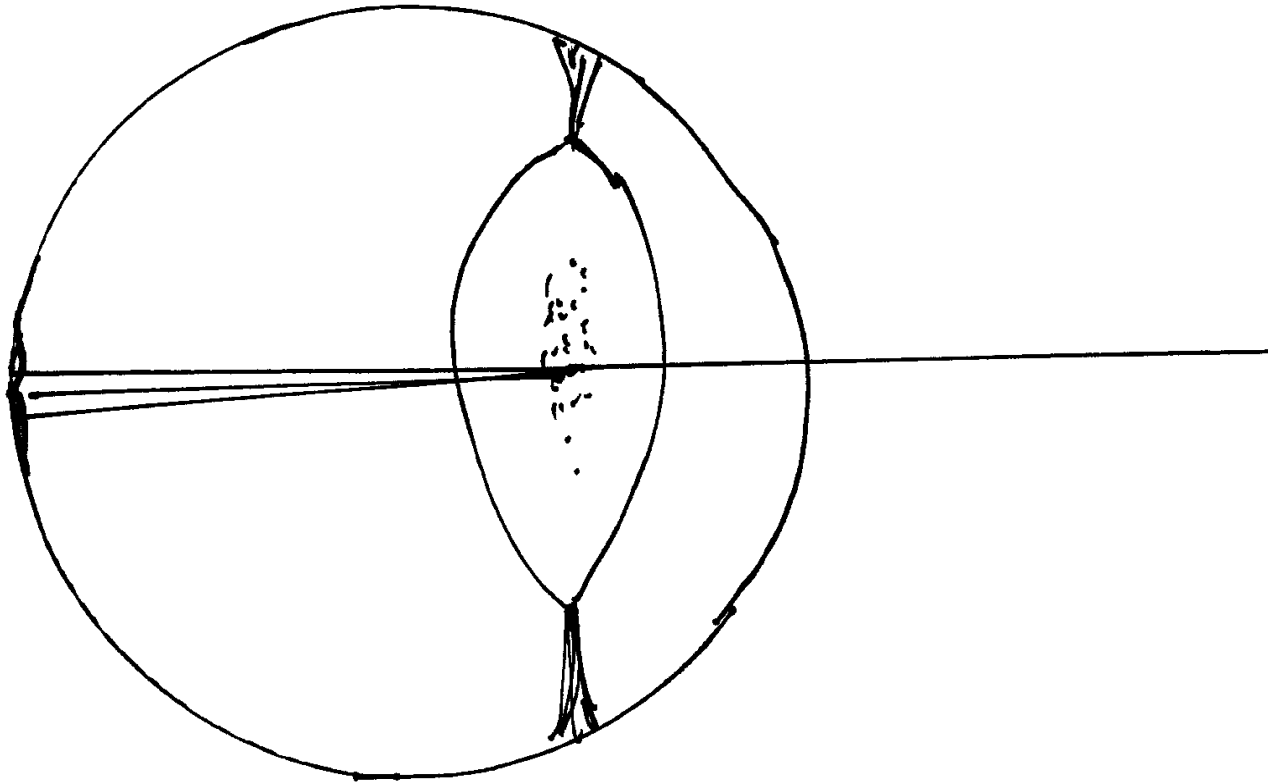
Congenital

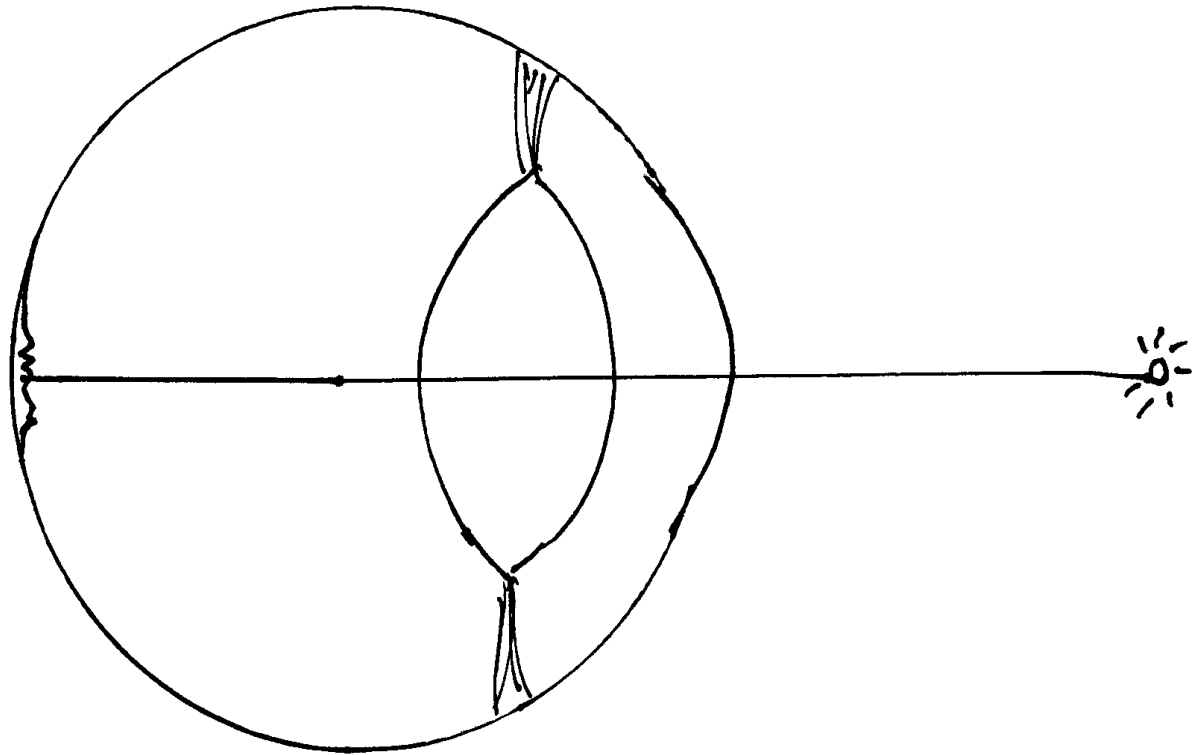
Ischemic, inflammatory (presumed)

Tumour (4 %)









Monocular diplopia

-- Cornea

- Astigmatism
- Scar
- Ulcer
- Dry eye

-- Anterior chamber

-- Lens

- early nuclear sclerosis

-- Vitreous

-- Retina: macular disease (AMD; diabetic)

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Treatment of concussion

MAYO CLINIC

“Rest is the most appropriate way to allow your brain to recover from a concussion

This means avoiding general physical exertion, including sports or any vigorous activities, until you have no symptoms

This rest also includes activities that require thinking and mental concentration, such as playing video games, watching TV, schoolwork, reading, texting or using a computer . . .

As your symptoms improve, you may gradually add more activities”

TBI management principle:
a need to withdraw from environmental stimuli



Post-traumatic vision syndromes:
common, self limited

Photosensitivity

Intolerance of visual motion

Less common:
convergence/accommodation insufficiency
(confounder: presbyopia)

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‘Post Trauma Vision Syndrome’

A dysfunction between the ‘focal’ and
‘ambient’ visual process.

‘Ambient’ process loses its ability to
organize information spatially.

Individuals visually ‘fixate’ on individual
letters; have difficulty ‘releasing’ their
vision.

Visual Midline Shift Syndrome

“ a disconnect between the position of their perceived ego-centre and their true center of gravity.”

Q. Where does this concept come from . . .
?

ambient process and focal process. These two systems are responsible for the ability to organize ourselves in space for balance and movement, as well as to focalize on detail such as looking at a traffic light. Post Trauma Syndrome results when there is dysfunction between the ambient and focal process causing the person to over emphasize the details. Essentially individuals with PTVS begin to look at paragraphs of print almost as isolated letters on a page and have great difficulty organizing their reading ability. It has been found that the use of prisms and binasal occlusion can effectively demonstrate functional improvement, while also being documented on brain wave studies by increasing the amplitude (this is like turning up the volume on your radio). Visual Midline Shift Syndrome also results from dysfunction of the ambient visual process. It is caused by distortions of the spatial system causing the individual to misperceive their position in their spatial environment. This causes a shift in their concept of their perceived visual midline. This will frequently cause the person to lean to one side, forward and/or backward. It frequently can occur in conjunction with individuals that have had a hemiparesis (paralysis to one side following a TBI or CVA). The shifting concept of visual midline actually reinforces the paralysis, by using specially designed yoked prisms that can be prescribed, the midline is shifted to a more centered position thereby enabling individuals to frequently begin weight bearing on their affected side. This works very effectively in conjunction with physical and occupational therapy attempting to rehabilitate weight bearing for ambulation.

Visual Midline Shift syndrome: theory

“Following a neurological event, such as a hemiparesis or hemiplegia . . . The ambient visual process changes its orientation to concept of the midline.”

“[An] event such as a CVA causing a hemiparesis or hemiplegia, information from one side of the body becomes interfered with.”

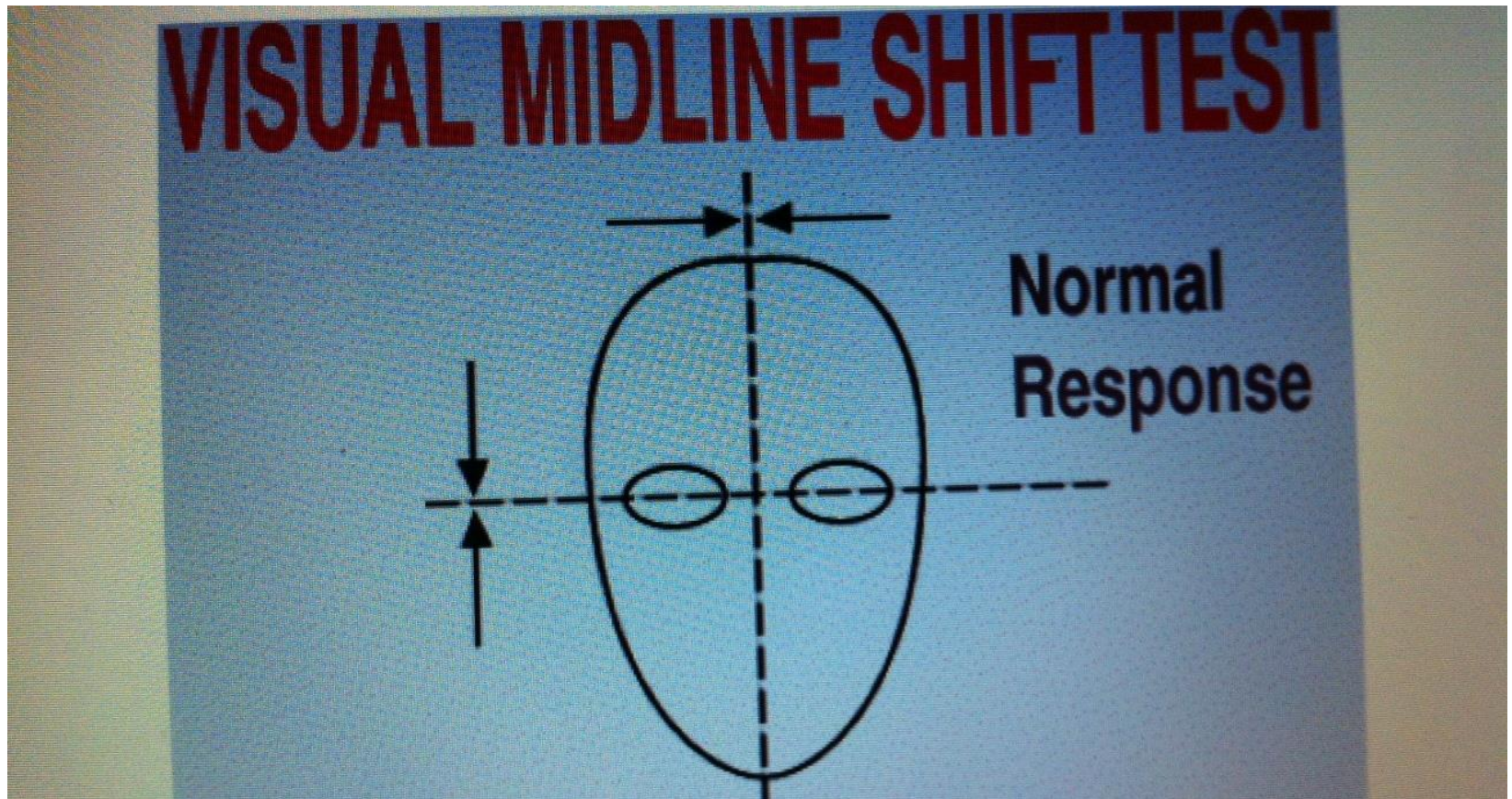
“The ambient visual process attempts to create balance by expanding its concept of space on one side of the body compared to the other. In so doing a perceived amplification of space occurs internally on one side and a perceived compression of space occurs on the other side. This phenomena [sic] causes a shift in concept of midline that usually shifts away from the neurologically affected side.”



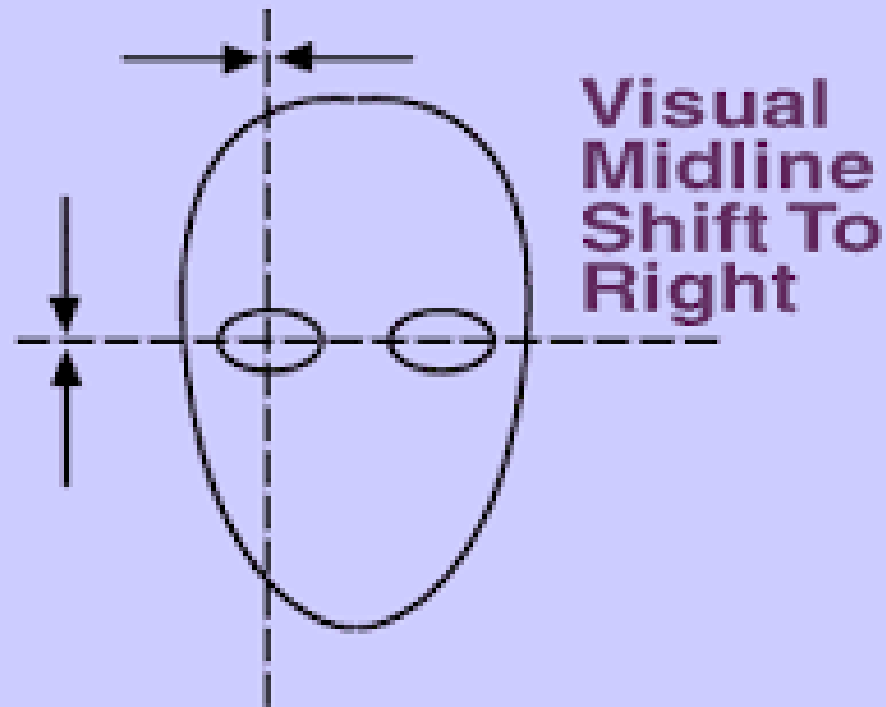




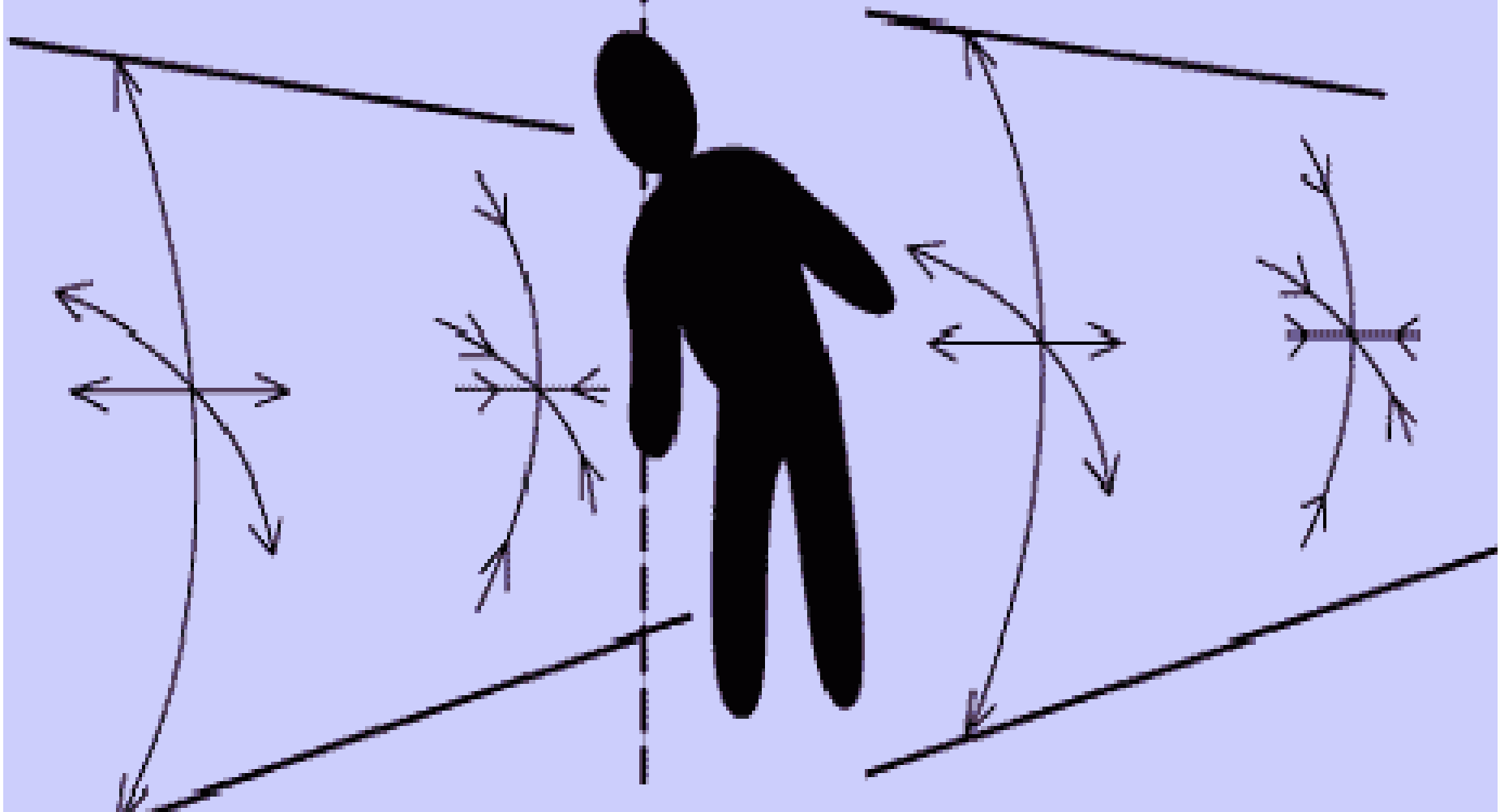
The 'Padula' test



VISUAL MIDLINE SHIFT TEST



Right Visual Midline Shift



“Yoked prism therapy”

Alleged treatment for the “Midline Visual shift”

A prism is inserted in both the right and left lenses.

The prisms *are in the same direction*.

Designed to “reduce ambient collapse”.

Visual Midline Shift Syndrome

Biological plausibility: extremely unlikely

Validated evidence for yoked prisms: 0

Wm. Padula, OD



Padula Institute

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Reality check: the vestibular system

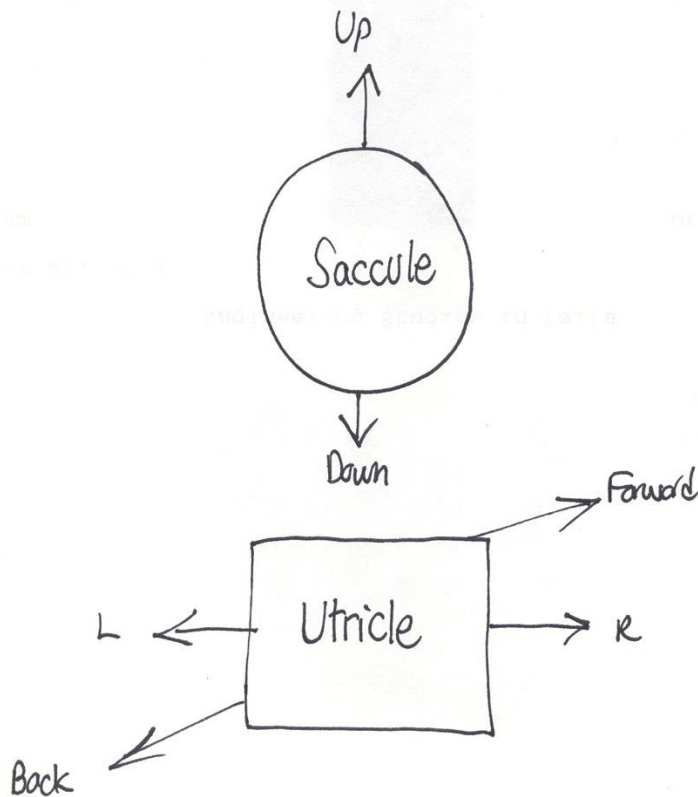
Complicated

Would need to be implicated in any type of
'shift' of body's perception

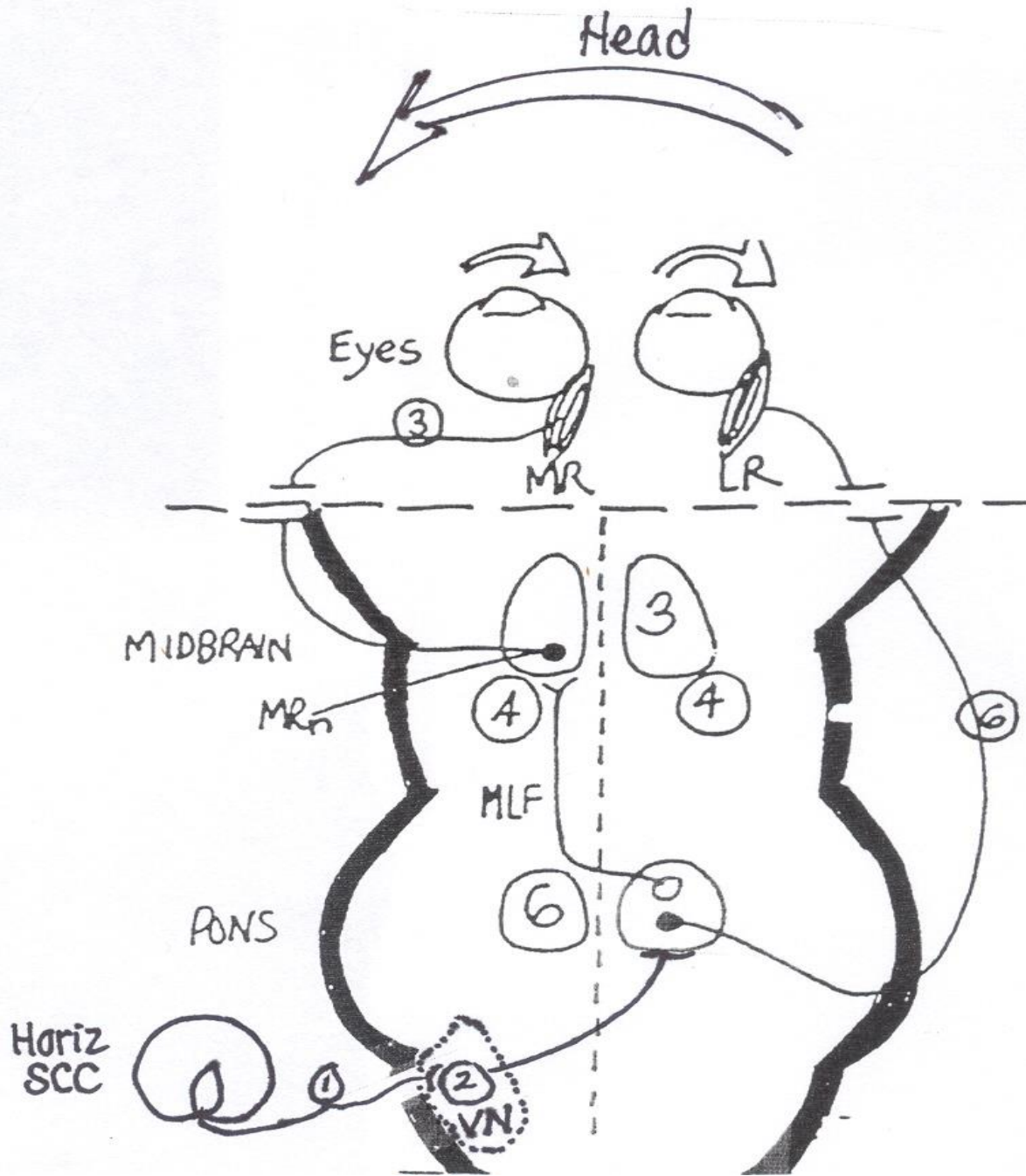
Accompanied by well-known additional
vestibular/eye movement signs and
symptoms

Otoliths

The linear pulsion centres



- **Saccule** detects vertical linear acceleration (gravity, elevators, roller coaster hills)
- **Utricule** detects horizontal linear acceleration (right, left, forward, back)

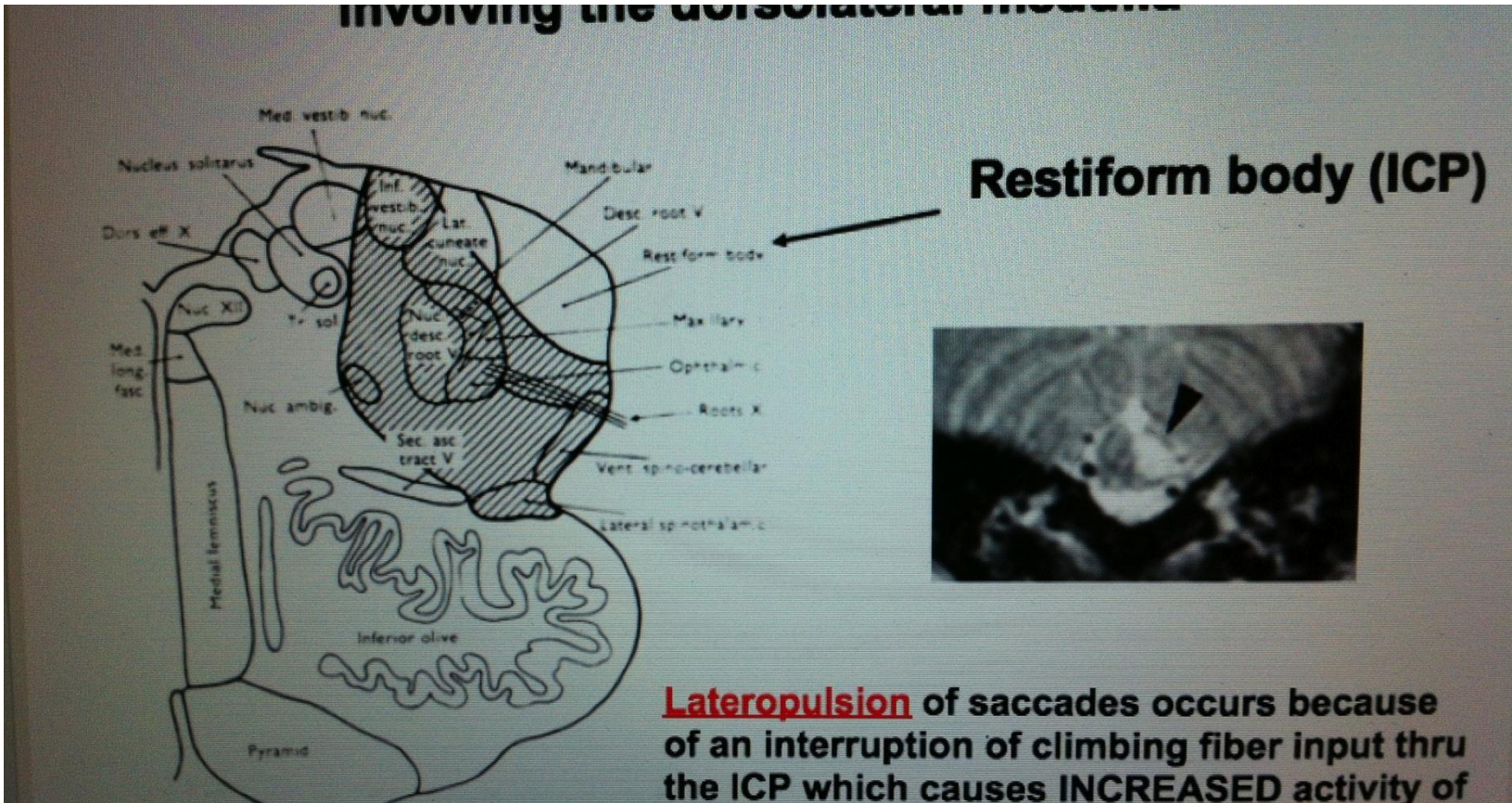


The high-frequency/acceleration head heave test in detecting otolith diseases

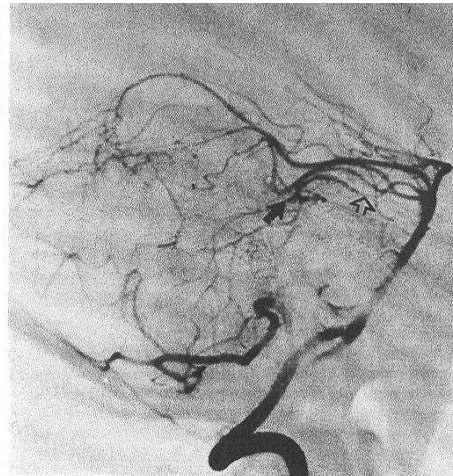
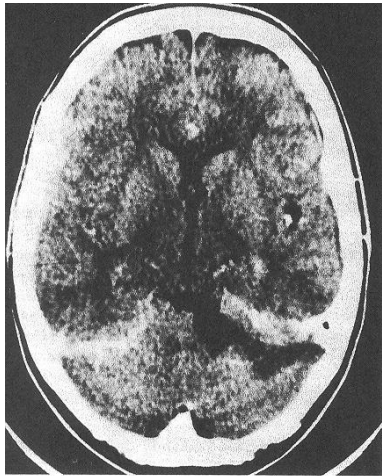
Kessler P, Tomlinson D, Blakeman A, Rutka J, Ranalli P, Wong A
Otology and Neurotology 2007 28:896-904



Wallenberg (lateral medullary) syndrome



Contrapulsion: left vermal lesion



- Occlusion of L superior cerebellar artery (SCA)
- L cerebellar hemisphere infarction
- Includes L vermis
- Signs:
 - L arm and leg dysmetria
 - R lateropulsion

Neuro-Visual Processing (Optometric) Rehabilitation and Visual/Postural Dysfunction Following a Neurological Event



Presented by William V. Padula, OD, FNAP, FAAO, FNORA and Raquel Munitz, MS, COVT

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Vertical 'Heterophoria' syndrome



Dizziness Ameliorated With Prism Treatment of Vertical Heterophoria

Arthur Rosner, MD¹; Debby Feinberg, OD²; Mark Rosner, MD, ^{3,4}

¹The Oakland University William Beaumont School of Medicine, Rochester, MI ²Vision Specialists Institute, Bloomfield Hills MI ³St. Joseph Mercy Hospital, Ann Arbor, MI ⁴University of Michigan, Ann Arbor, MI

ABSTRACT

The visual system is an integral part of the balance system. However a specific visual disorder causing dizziness and headache has not been described in the otolaryngology literature. Vertical Heterophoria (VH) is a binocular vision disorder with symptoms of headache, dizziness, neck pain and reading difficulties, treatable with prismatic lenses. The study's objective was to quantify dizziness and associated symptom reduction after prismatic lens treatment in patients with a chief complaint of dizziness concomitantly diagnosed with VH.

Retrospective analysis of 40 patients presenting to a vision specialist with a chief complaint of dizziness and who were simultaneously diagnosed with VH between August 2009 and May 2011. Pre / post-treatment data was collected from validated survey instruments (Headache Disability Index (HDI), Dizziness Handicap Inventory (DHI), Zung Self-Rating Anxiety Scale (SAS); from the Vertical Heterophoria Symptom Questionnaire (VHSQ) (a validated self-administered VH symptom assessment instrument developed by the authors to determine VH symptom burden); from a subjective rating (0-10 scale) of headache, dizziness and anxiety severity; and from a sub-analysis of VH symptom questions that pertain specifically to headache, dizziness and anxiety. Upon conclusion of treatment, subjective assessment of overall improvement of VH symptoms was obtained utilizing a 10 cm visual analog scale (VAS).

Effects were analyzed using paired t-tests. Following prismatic lens treatment, there was a 50% decrease in DHI score (p<0.0001); 46.9% reduction in Zung score (p<0.0001); 49.4% reduction in VHSQ score (p<0.0001); 19.9% reduction in HDI score (p<0.0001); 60.4% reduction in VHSQ headache questions (p<0.0001); and anxiety reduction in VHSQ anxiety questions (50.2%; p<0.0001).

Patients with dizziness with prismatic lenses score reduction of the validated headache and anxiety, which correlated with overall VH symptom reduction. Prismatic lenses are needed to further validate determine prevalence of VH in

INTRODUCTION

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METHODS AND MATERIALS

Retrospective analysis of 40 patients presenting to an optometric binocular vision subspecialist with a chief complaint of dizziness and who were simultaneously diagnosed with VH between August 2009 and May 2011. Pre / post-treatment data was collected from validated survey instruments (Headache Disability Index (HDI), Dizziness Handicap Inventory (DHI), Zung Self-Rating Anxiety Scale (SAS); from the Vertical Heterophoria Symptom Questionnaire (VHSQ) (a validated self-administered VH symptom assessment instrument developed by the authors to determine VH symptom burden); from a subjective rating (0-10 scale) of headache, dizziness and anxiety severity; and from a sub-analysis of VH symptom questions that pertain specifically to headache, dizziness and anxiety. Upon conclusion of treatment, subjective assessment of overall improvement of VH symptoms was obtained utilizing a 10 cm visual analog scale (VAS).

RESULTS

Treatment effects were analyzed using paired t-tests. Following prismatic lens treatment, there was a 50% decrease in DHI score (p<0.0001); 46.9% reduction in Zung score (p<0.0001); 49.4% reduction in VHSQ score (p<0.0001); 19.9% reduction in HDI score (p<0.0001); 60.4% reduction in VHSQ headache questions (p<0.0001); and anxiety reduction in VHSQ anxiety questions (50.2%; p<0.0001).

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% Reduction of Anxiety, Dizziness and Headache Metrics With Prism Lenses

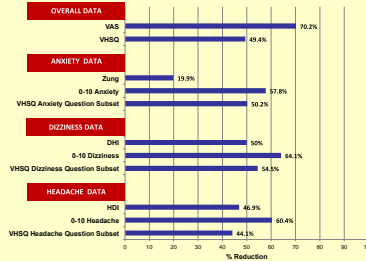


Figure 1.

HISTORY and PHYSICAL EXAM

Patients present with a combination of non-vertiginous dizziness, facial pain around the eyes, headache, neck pain, and anxiety. Reading difficulties are common, but often not the chief complaint (see Figure 2 for full symptom set and prevalence). Given the diverse symptom set, they have seen many different specialists prior to being diagnosed with VH.

Patients have skew deviation (vertical eye misalignment), head tilt to the side, and slight dissociate gaze. Symptoms are often duplicated by having the patient move their eyes into convergence.

PRISM CHALLENGE

The current tests used to identify VH perform inconsistently.^{1,2,3} To improve diagnosis, the authors developed the Prism Challenge, a dynamic process between the optometrist and the patient to determine the optimal prismatic lens prescription.⁴ One quarter units of prism are incrementally added to a trial frame (see Figure 3) in the vertical and horizontal direction until the patient's symptoms are minimized.



Figure 3. Trial Frames.

VH Symptoms in Dizzy Patients

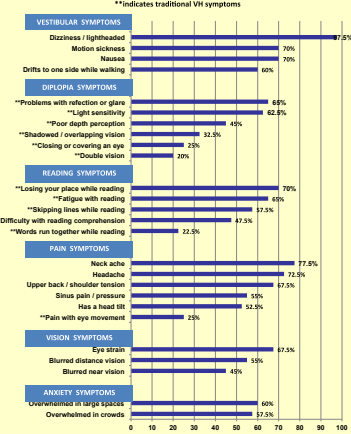


Figure 2.

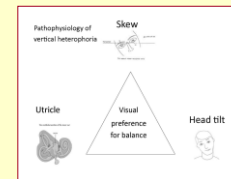


Figure 4. Pathophysiology of Vertical Heterophoria

PATHOPHYSIOLOGY

Vertical heterophoria seems to be caused by a combination of deviation of the eyes, a roll head tilt, and otolith dysfunction (utricle). This is combined with a visual preference for balance (Figure 4). This has been described in visual vertigo patient.

Thirty percent of the population has one eye higher than the other. Only 4% of the population becomes symptomatic. Patient tilt or isolated utricle dysfunction often do not have symptom dysfunction causes skew, head tilt and ocular torsion. In a head roll tilt is likely present to stabilize the retinal image. A head roll tilt is the second most stabilizing posture. Head tilt backward. This head tilt causes the balance eyes to be misaligned with gravity.

Motion sickness is common in these patients. It is likely an asymmetric vertical optokinetic stimulation/nystagmus, asymmetric in both time and angle. Vertical optokinetic nystagmus has been shown to be one of the most potent stimuli for motion sickness.

CONCLUSIONS

Treatment of dizziness with prismatic lenses resulted in a reduction of the validated and other metrics for dizziness, anxiety, which correlated with a marked reduction of overall symptom level. These patients have had many other diagnoses with VH including: failure to compensate dizziness, vestibular migraine, visual vertigo, and motion sickness. Close collaboration between vision providers and otolaryngologists is needed to serve this population. Prospective studies to further validate this intervention to assess for placebo effect and determine prevalence of VH in dizziness patients.

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- Gall R, Wick B. The Symptomatic Patient with Normal Phorias Near: What Tests Detect a Binocular Vision Problem? Optometry. 2005;74:309-322
- Doble JE, Feinberg DL, Rosner MS, Rosner AJ. Identification of Vision Dysfunction (Vertical Heterophoria) in Traumatic Brain Injury: Effects of Individualized Prismatic Spectacle Lenses in the Treatment of Postconcussive Symptoms: A Retrospective Analysis. PM R. 2011
- Bronstein AM. Visual Vertigo Syndrome: Clinical and Postural. J Neurol Neurosurg Psychiatry. 1995;58:472-476

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248-844-2936 www.VSOm.com

Vertical heterophoria syndrome

Biological plausibility Nil

Validated evidence 0

Post Trauma Vision Syndrome

Essentially, individuals with PTVS begin to look at paragraphs of print almost as isolated letters on a page and have great difficulty organizing their reading ability. It has been found that the use of prisms and bi-nasal occlusion can effectively demonstrate functional improvement, while being documented on brain wave studies by increasing the amplitude (this is like turning up volume on your radio).

Convergence Disorders Affecting Reading Patients may experience reduced convergence after stroke or head injury. Our eyes must turn in together accurately as a team to prevent double vision and eye fatigue in reading. Prisms may aid some patients. Optometric vision therapy may aid some, but not all patients with convergence insufficiency will respond fully to therapy due to the variation in the extent of trauma which may be present. Test: Padula near point of convergence Result: convergence insufficiency

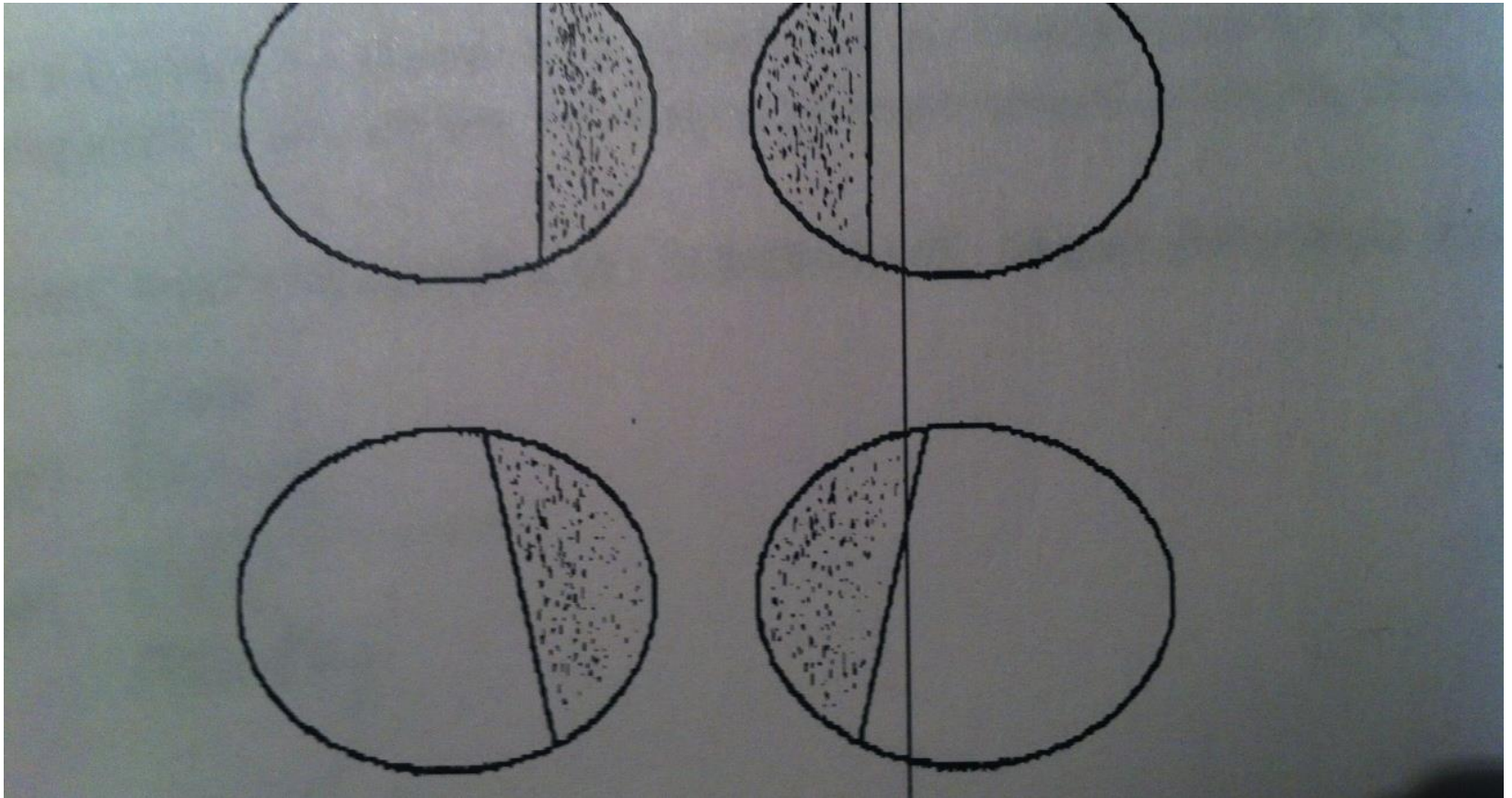
Unstable Ambient Vision Brain injury patients may present with vertigo, sensitivity to light, extreme sensitivity to motion around them. Trying to sustain reading becomes very difficult. The patient may experience nausea, loss of attention, difficulty fixating on the words and fatigue. Unstable ambient vision is a hallmark of Post Trauma Vision Syndrome.

Post Trauma Vision Syndrome/Visual Midline Shift Syndrome

Any such “difficulty organizing their reading ability”
in a TBI patient is likely cerebral.

Validated evidence that this is helped by prisms
and bi-nasal occlusion..... 0.

Binasal occlusion



Binasal Occlusion (BO)

Louis Jacques (1950) – introduced for strabismus patients, mainly for esotropia.

Theory: “removed visual inhibition and suppression”, and allowed the opportunity to establish the basic vision patterns of the normal human being.”

Much discussion over: shape (flat, tapered?); density (opaque, translucent?); symmetric/asymmetric

Tassinier J. Binasal occulsion. J Behav Optom 1990; 1(1):16-21

Binasal occlusion literature: typical article

Proctor A. Traumatic brain injury and binasal occlusion.
Optom Vis Dev 2009;40(1):45-50

Male, 46.

Referred to TBI Clinic, Oklahoma Coll Optom.

(no trauma details)

Dizzy, poor depth perception.

Complete eye exam >>> vision therapy 6 sessions.

Binasal Occlusion (BO) “suggested by
optometrists presenting at the Invitational Lens
Symposium at Tahlequah, OK.”

BO strips inserted into patient’s glasses – asked to
walk down the hallway . . .

Binasal Occlusion article (cont.)

“He seemed to feel more confident”, and “he reported feeling better.”

Advised to wear them for all activities, including riding as a car passenger.

1 week later: the BO helped mobility, “but he did not feel comfortable driving with them on.”

BO were adjusted, given more VT, sent home.

“Due to scheduling issues, he discontinued the therapy but returned 3 months later.” BO adjusted, encouraged to use for all activities.

Binasal Occlusion article (cont.)

“The patient, unfortunately, did not follow through with this plan of action.”

After a 6 month absence, he was called by telephone: he claimed he wore the BO intermittently. Still complained of how they restricted his peripheral vision while driving.

Offered 1-year follow-up, but he declined, citing transportation difficulties.

Binasal Occlusion (BNO)

Biological plausibility Nil

Validated evidence 0

DEM SCORE SHEET

DOB 03-29-1985 AGE 30.2 GRADE College

ARTICULATION PRE-TEST

Y N

NUMBER KNOWLEDGE PRE-TEST

Y N

/ = substitution error
 2 = addition error

o = omission error
 <or> = transposition error

TEST A

TEST B

TEST C

3	4	6	7	3	7	5	9	8
7	5	3	9	2	5	7	4	6
5	2	2	3	1	4	7	6	3
9	1	9	9	7	9	3	9	2
8	7	1	2	4	5	2	1	7
2	5	7	1	5	3	7	4	8
5	3	4	4	7	4	6	5	2
				9	2	3	6	4

“The Readalyzer”

A device that purports to measure eye movements while reading.

Creates scores such as:

of fixations / 100 words

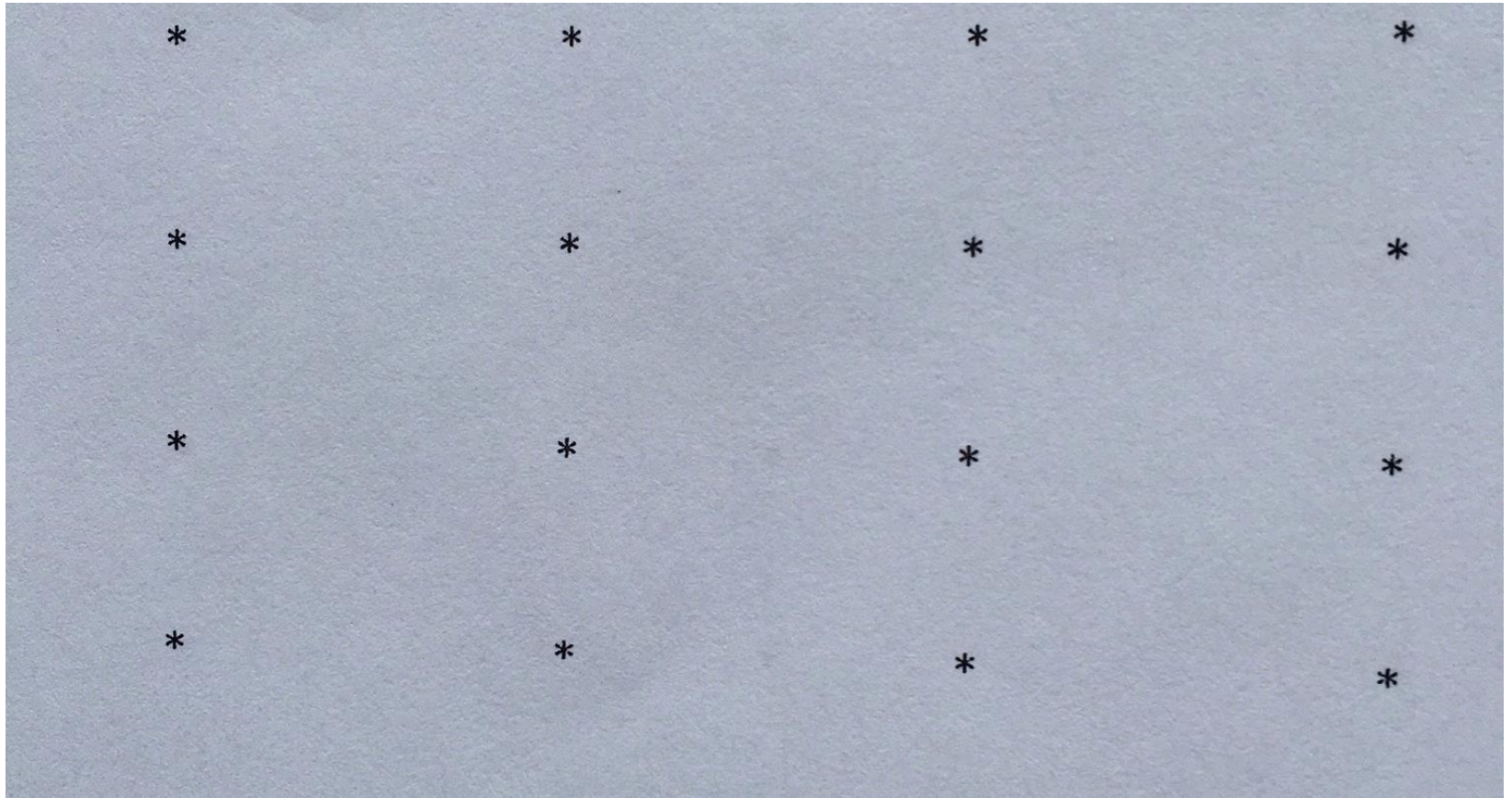
of ‘regressions’ / 100 words

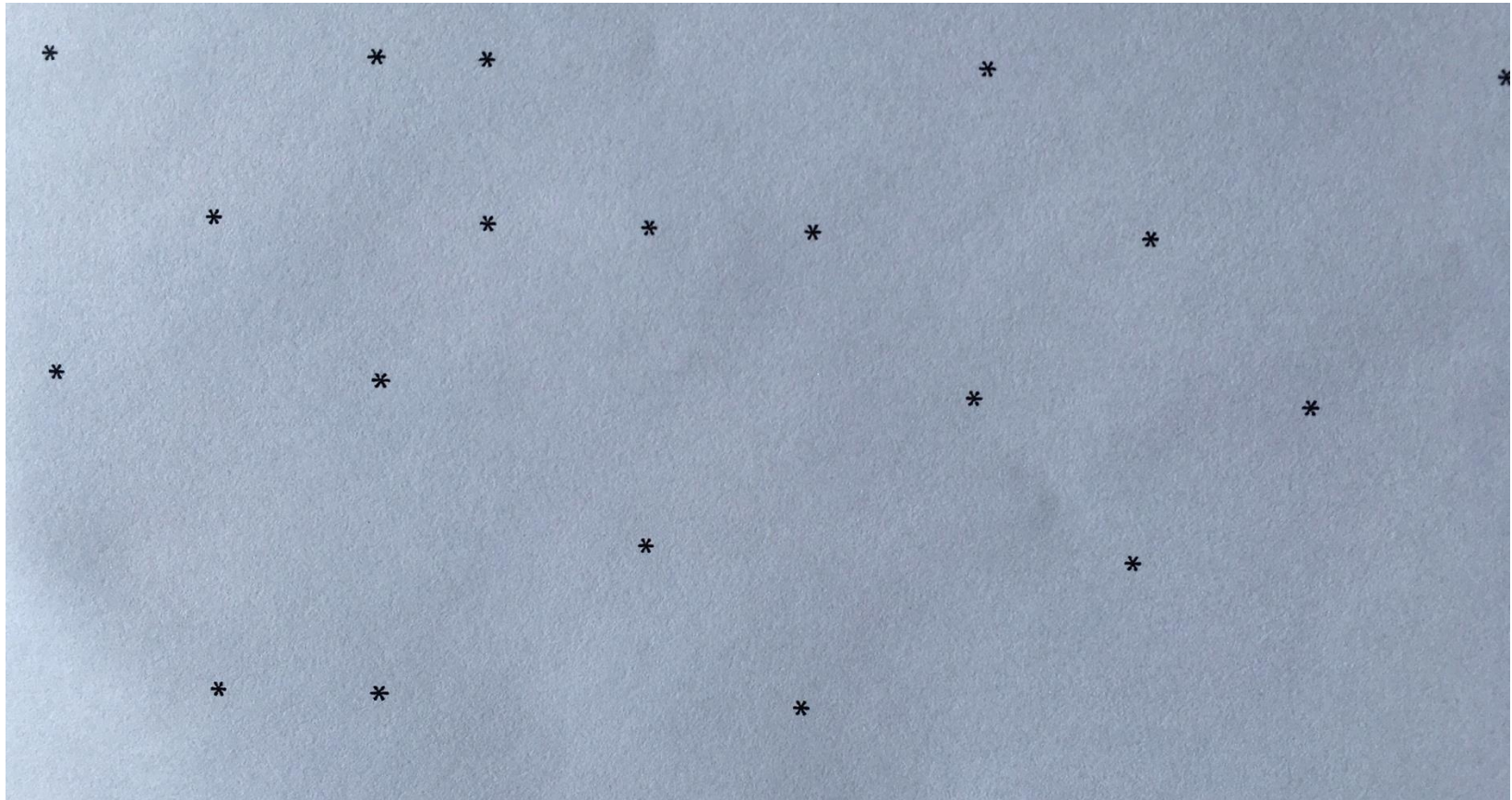
Comment: Where to begin??

th a new prescription she is able to see 20/20 in both eyes. With her
as seeing 20/20 in the right eye and 20/25 in the left eye. She is
distance and near, no vertical deviation. Anterior and posterior
remarkable.

amination was done for her on May 30, 2014. That testing indicated
in tracking. She is reading at a 11-12 year old level with DEM test.
g showed she has a lot of regression before continuing to read and
words per minute compared to the normal adult reading rate of 340
of fusional ranges for near showed she has a weak Base-In and Base-
tion disparity.

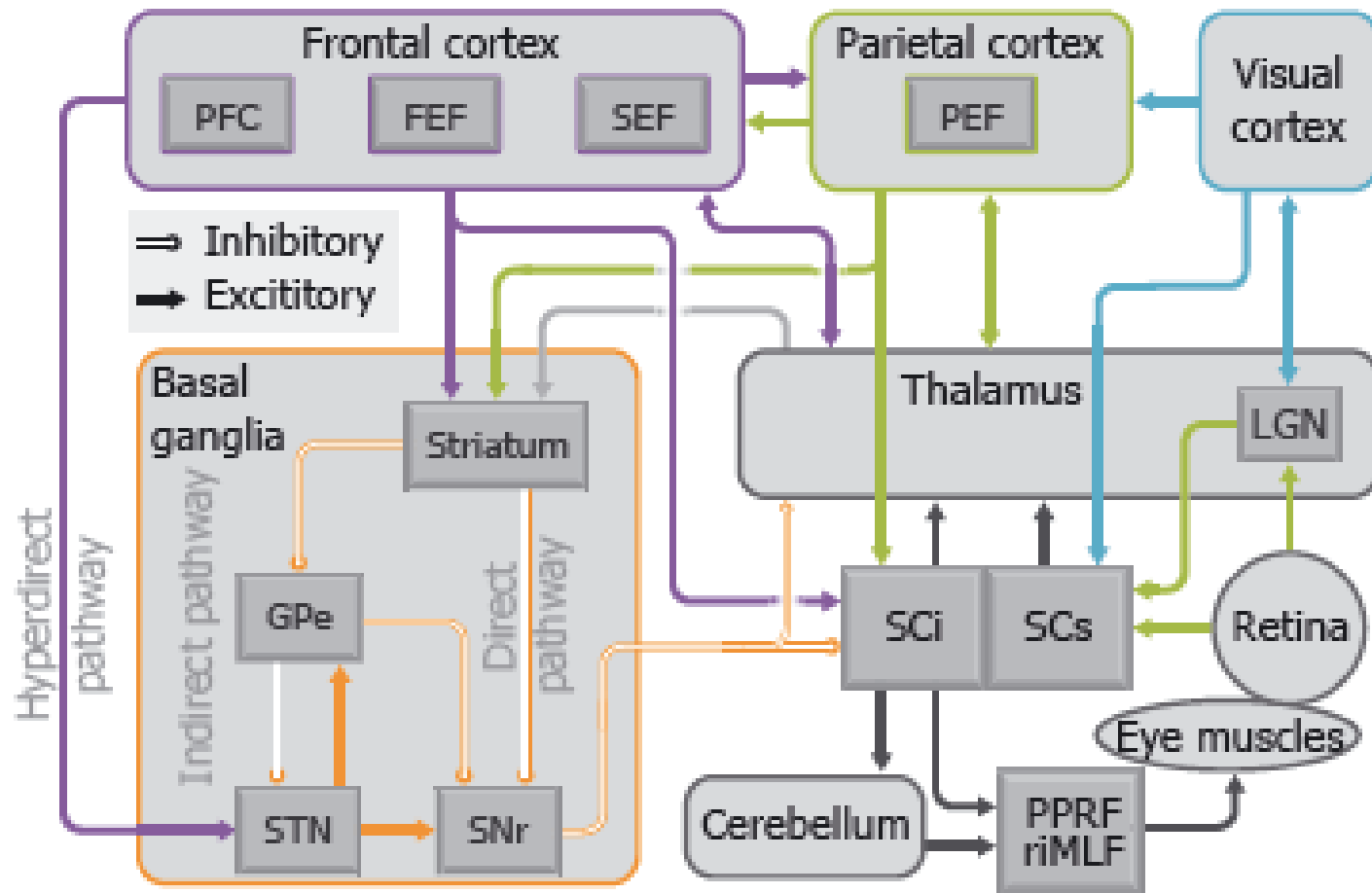
voked Potential Testing (VEP) which is an objective test for the
that she has interference with big targets and no delay in the



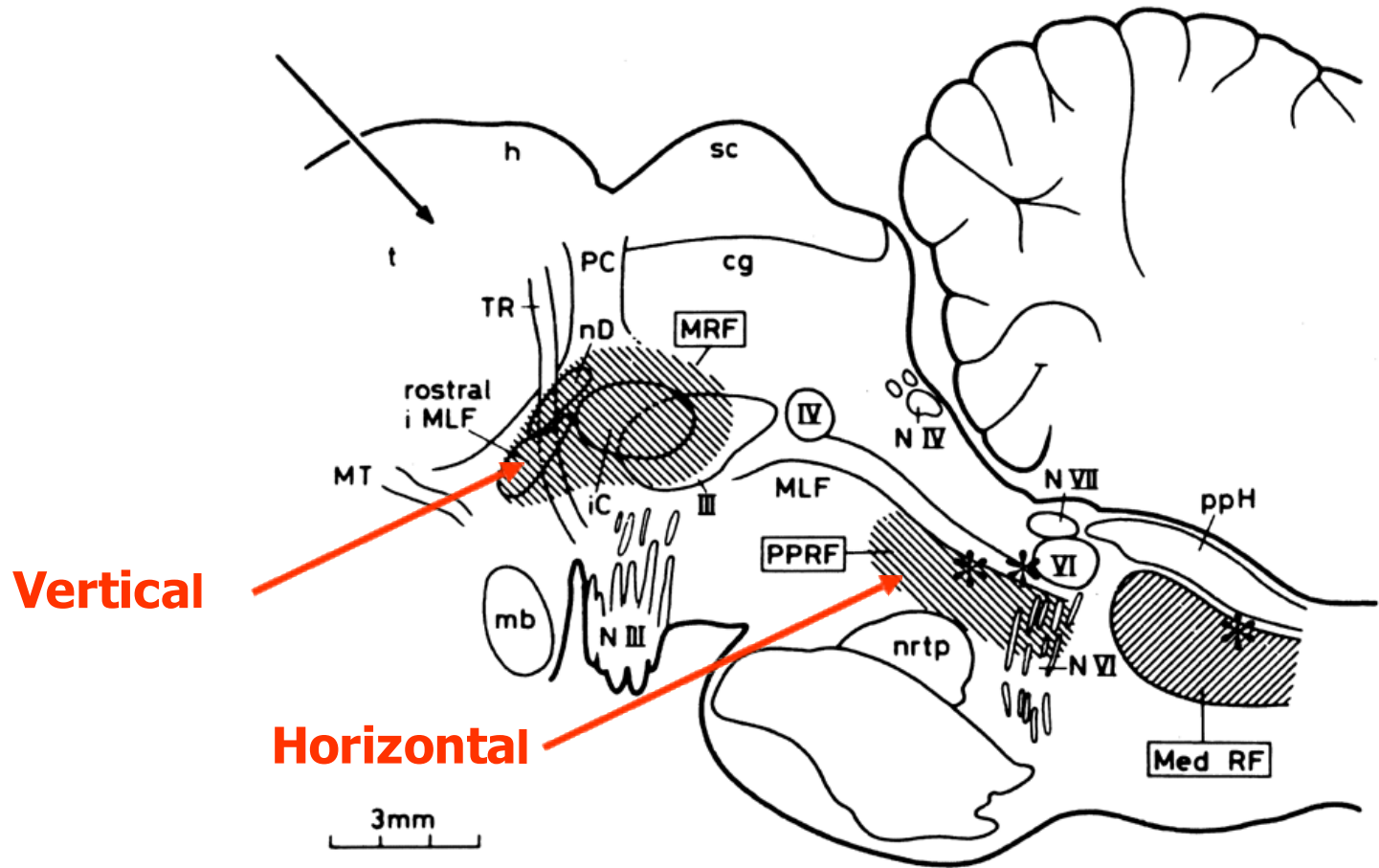


Z L G R D N O Z W R Y I
T H W F M B K A P J H R
X L M T A N C W G B P P
C P V D H Z E T O A R K
R E J P W X F P N V M K
B G M T K O S E U R Q W
O O P A C N V X D L T X
Z H C F L R W T E B S Z
C W V Y P A O D K G A V
L F R Z M S Y N W T E T
Y A Y O T U T R S W M Y
U Z T C B I X A Q E C F
W O S D R A N O H A G I
I K X N G M C O O

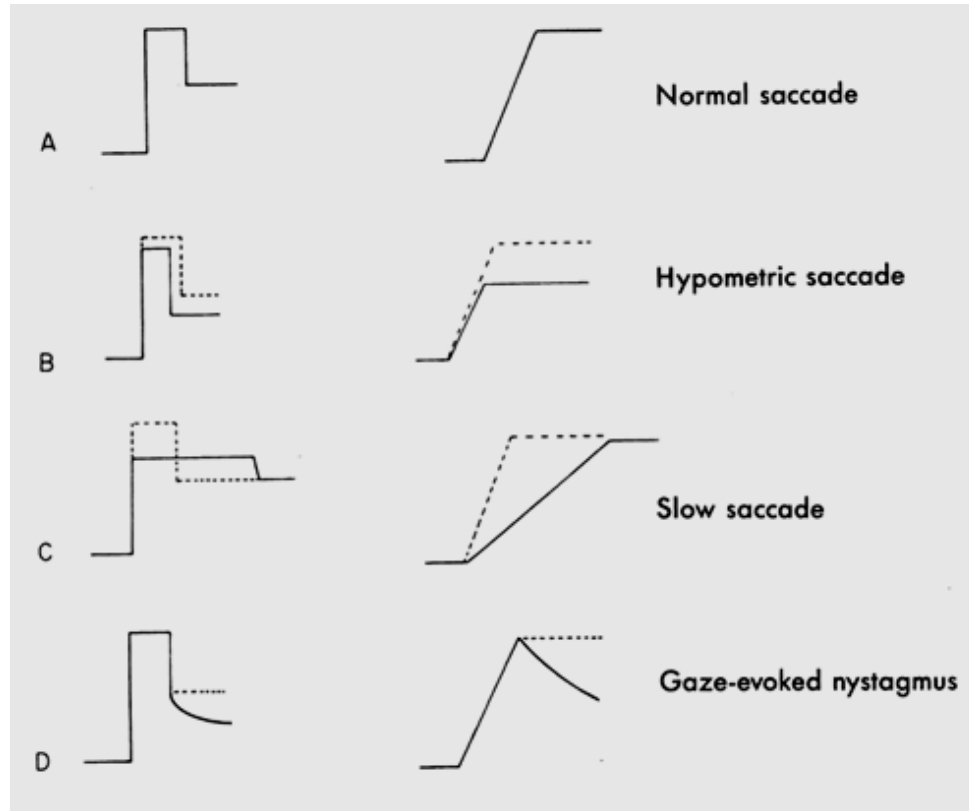
Control of saccades



Burst neurons for saccades: generate the Pulse (velocity) command



Signal processing for normal and abnormal saccades



Saccade analysis / reading scores based on saccade counting, accuracy by unvalidated test instruments . .

.

Biological plausibility Minimal

Validated evidence for saccadic ‘training’
. 0

“Pinhole glasses” from a neuro-optometrist

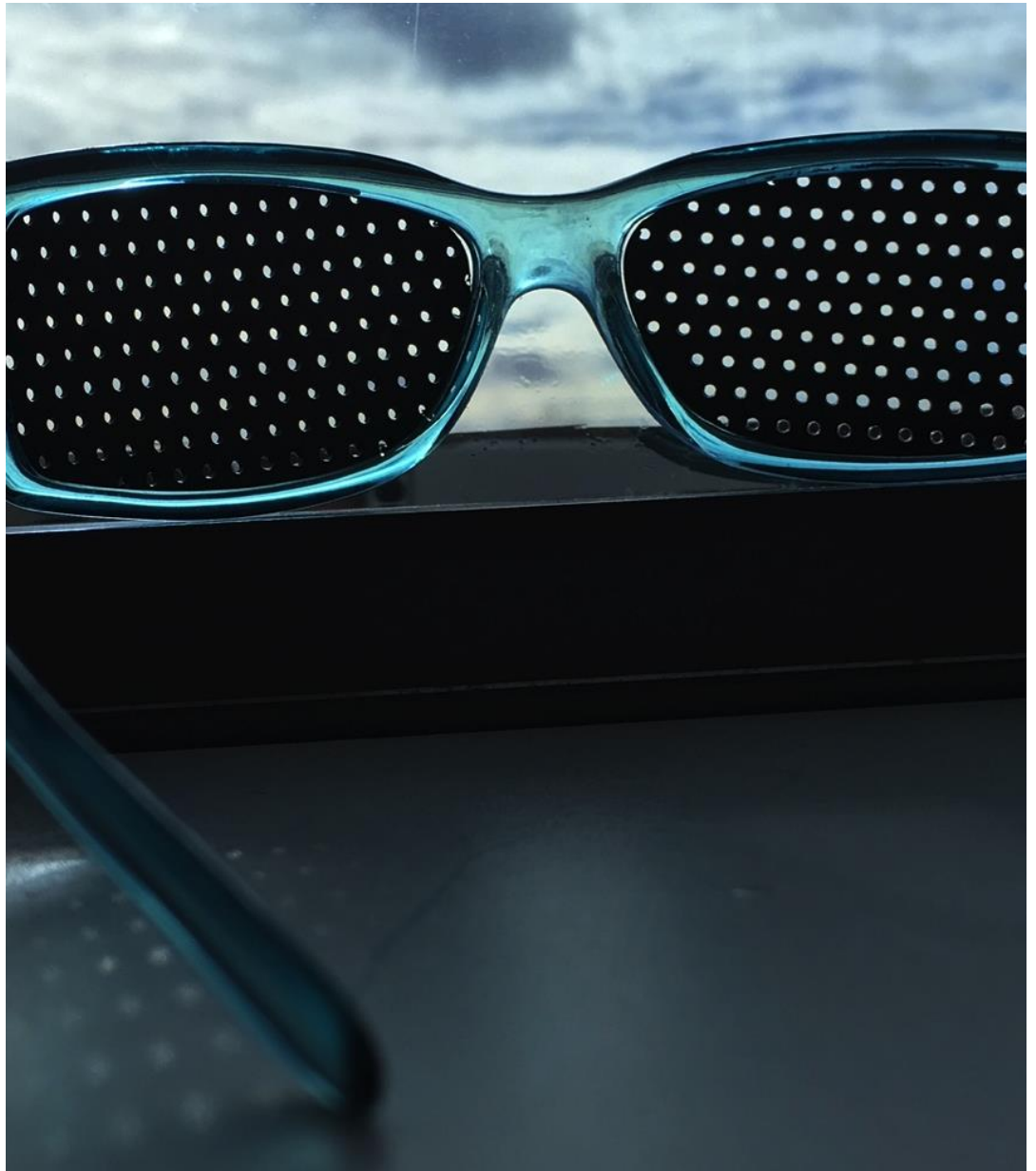
.. And other gear:

Compression vest (for reading)

Elastic bands wound around the shoulders (for reading)

Padula cube

Validated evidence: 0



Padula Transformation Cube

Padula T-cube

During postural or vision therapy, fixate on the cube.

Q. “Which side seems more forward, red or green?”

If “red”, patient is asked to “allow the green side to come forward”. The patient must “utilize the ambient visual process to release the focalization maintaining one side forward. The release is simultaneous with the anticipatory response by the ambient process to allow the opposite side and color to come forward. A patient who has difficulty is deemed to have ‘focal binding’ caused by PTVS, and needs NVPT to release the binding and re-establish “the ambient process as the anticipatory process for change.”



More unusual concepts

“ . . . Her neurological system is not in balance as shown by the red cap desaturation and omega pupil.

There is an asymmetric collapse of the peripheral system as noted with the visual field errors only on the right eye.”

The top references

References:

A. Cluffroda KJ, et al.: Vision therapy for oculomotor dysfunctions in acquired brain injury: a retrospective analysis; *Optometry*. 2008 Jan;79(1):18-22.

B. Freed S, Hellerstein LF: Visual electrodiagnostic findings in mild traumatic brain injury; *Brain Inj*. 2008 Jan;11(1):25-36.

Hellerstein LF, Freed S, Maples WC.: Vision profile of patients with mild brain injury; *J Am Optom Assoc*. 1995;66(10):634-639.

Kapoor N, et al.: Vision Disturbances Following Traumatic Brain Injury; *Curr Treat Options Neuro Neurol*. Jul;4(4):271-280.

C. Padula WV, et al.: Visual evoked potentials (VEP) evaluating treatment for post-trauma vision symptoms (PTVS) in patients with traumatic brain injuries (TBI); *Brain Inj*. 1994 Feb-Mar;8(2):125-33.

Padula WV, Munitz R & Magrun WM: *Neuro-Visual Processing Rehabilitation: An Interdisciplinary Approach*; CRC Press 2013.

Ciuffreda KJ et al: Vision therapy for oculomotor dysfunction in acquired brain injury: a retrospective analysis. Optometry 2008 79(1):18-22

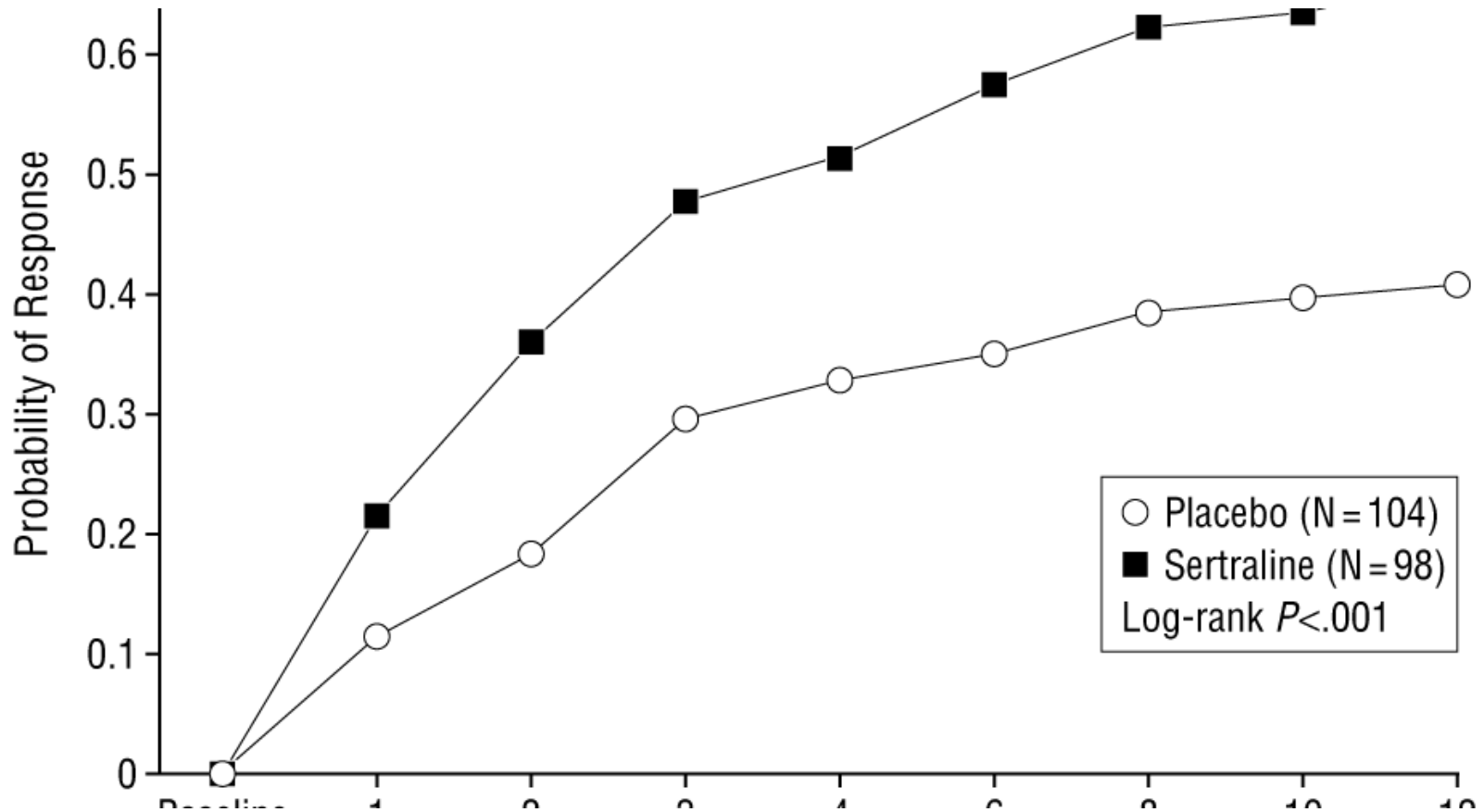
Open label.

No controls.

Computer-based review of 40 of their chosen treatment patients.

Up to 8 months of therapy.

Therapeutic gain: the post-traumatic placebo group improves over time



Freed S et al. Visual electrodiagnostic findings in mild traumatic brain injury. Brain Inj. 1997; 11(1):18-22

Padula WV et al. Visual evoked potentials (VEP) evaluating treatment for post-trauma vision syndrome (PTVS) in patients with traumatic brain injuries (TBI). Brain Inj 1994;8:125-33

VEP have no validated role in assessing the cerebral effects of brain injury on vision.


Suspicious that a vision-treatment trial did not report *functional* outcomes.

CITT trial: supportive reference

For literally 100s of peer-reviewed references on this point (i.e. the link between binocular vision dysfunction and learning issues) as a single pdf document with abstracts visit www.covd.org (website for the College of Optometrists in Vision Development) and go to the "Research and White Papers" section. The CITT (Convergence Insufficiency Treatment Trial), which is a double blind, randomized, multi-centre trial (highest level of research in the clinical arena and is published by both ophthalmology and optometry) clearly shows that eye teaming problems can be treated effectively with vision therapy.

If you have any queries or concerns, please feel free to contact me. If you have questions regarding the area of vision therapy, please visit www.visionhelp.com or www.covd.org for more details.

Sincerely,



CITT: Convergence Insufficiency Treatment Trial

Arch Ophth 2008

NEI funded.

Randomized, but not exactly blinded.

221 subjects – assigned into 4 blocks of about 55 each.

a) Home pencil-pushups (PP).

b) Home PP + computer vergence/accom. vision therapy.

c) Office placebo therapy.

d) Office vision therapy + home reinforcement.

CITT: outcome

Office-based Vision therapy was superior to the other 3 methods.

Outcome measures:

- a) A symptom-based score
- b) Improvement in fusional vergence

CITT: The catch . . .

It was *children* (and adolescents)

subjects aged 9-17; avg. 12 yrs.

They were *born* with convergence
insufficiency

Children entered with a certain minimum degree
of exo-deviation at near, and a high CI symptom
score

. . ie. They're not post-head-injured
adults

Dr. Paul Sieving MD PhD
Director of NEI

“The CITT will provide eye care professionals with the research they need to *assist children with this condition.*”



In-office VT benefit in adults

Birnbaum MH et al. *J Am Optom Assoc* 1999;70(4):225-232

Subjects: adult males with ‘asthenopic symptoms’

Subjective scoring.

Primum non nocere



- The inevitable effects of fatigue and frustration, due to the relentless, repetitive nature of therapies that are unvalidated with respect to efficacy.
- Spending precious ‘brain capital’ at a time of recommended withdrawal and rest.
- An open question: might these activities be prolonging recovery?

Neuro-optometry: **Summary**

1. PTVS: *a recent construct.*
2. Visual injuries in real life: major (rare); minor syndromes (common). *Well known, established.*
3. ‘Neuro’-optometrists: their terms, their claims. *No credible biological model. No validated results.*
4. The source: Padula, and his tests. *Lack neurophysiological rigor.*

Objectives:

Visual disturbance following m-TBI

1. Recognize pre-accident visual issues.
2. Mild concussion (m-TBI): common visual symptoms.
3. Rarer, more severe visual injuries.
4. Management of common m-TBI visual issues: less is more.
5. 'Neuro-optometry' – their concepts (PTVS, VMSS).
6. 'Neuro-optometry' – their devices, their therapies.
7. 'Neuro-optometry' – their literature, evidence.
8. A reality check.