The Strabismus That You May Not Know and some of your doctors don’t know

UNDERSTANDING FOR DOCTORS AND THERAPISTS.

Introduction

Jason’s story
Path guy in Optometry School
Clinician of the year SCO 1998

- Wife is sensory based peds OT
- As we say in the south “Lookin’ fer ain’ers”
- New ways of thinking about vision and visual functions
- What they teach you is not always the truth... and I like to dig deep

My Game Changers

- Pathology and disease guy gets questions he can’t answer
- Read the book Neurology of Eye Movements – Leigh and Zee leads to
- Read the book Eye Movement Disorder – Wong leads to
- Read the book Vestibular Rehabilitation – Hermann
- Pediatric/Brain injury clinic that leads to
- Understanding functional vision that leads to
- Special needs care for patients that leads to
- Nutrition understanding that leads to
- Genetics/Epigenetics that leads to...
What is a Strabismus?
- Muscle position?
  - No Studies
- Muscle tone (position) or phasic (strength) problem?
  - No Studies
- Visual motion processing problem?
  - Studies on this and pathways for this
- Vergence overaction?
  - No studies on this
- Nerve Paresis or palsy?
  - Depends
  - Some studies on this particularly with infantile esotropia

What affects a Strabismus?
- Hair covering an eye?
- Accommodation/refractive?
- Convergence?
- Motor control?
- Spatial awareness development?
- Sympathetic imbalance?
- Fixation imbalance or loss?
- Diplopia avoidance?
- Infections?
- Adaptation to any of the above?
### Prevalence of Strabismus

- **Ocular alignment of neonates study** (n=3324)
  - Orthotropia: 22.7%
  - Exotropia: 61.1%
  - Intermittent exotropia: 13.0%
  - Esotropia: 0.2%
  - Intermittent esotropia: 0.5%
  - Esodeviation to exodeviation: 2.5%

### Current State of Treatment

#### Surgical

Current treatment with surgery is for symptoms with "unknown" causes.
Cutting a muscle and repositioning when it is not a muscle problem is not optimal.
Latest surgical studies want earlier intervention... due to possibly better motion processing.

Surgical outcomes are *less than optimal*, yet considered standard of care.
There are no multi-center, randomized, double blind, placebo controlled studies for surgery, yet it is still considered standard of care.
What do you do with abduction deficits in surgery?

BCBS and Cigna policies on strabismus surgery "...despite the paucity of randomized controlled studies"!
Current State of Treatment

Surgical side effects –
- Marked overcorrection/undercorrection
- Infection
- Scleral perforation
- Foreign body granuloma at surgical site
- Allergic reaction to suture material
- Conjunctival inclusion cyst
- Conjunctival scarring
- Anesthesia (more than 2 in childhood shows delayed learning)

Current State of Treatment

Surgical side effects –
- Anterior segment ischemia
- Change in eyelid position
- Lost muscle
- Slipped muscle
- Poorly positioned muscle (vertical or horizontal)
- Nystagmus (latent)
- Oculocardiac reflex
- Others
- Why has earlier surgery been pushed in US?
  - Better surgical outcomes?
  - Anesthesiology on infants has improved
What are outcomes of early surgery?

<table>
<thead>
<tr>
<th>Condition</th>
<th>No Tx</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amblyopia</td>
<td>0-14%</td>
<td>41-72%</td>
</tr>
<tr>
<td>Stereopsis</td>
<td>Gross</td>
<td>Better quality</td>
</tr>
<tr>
<td>Fusion</td>
<td>Monofix→monofixation re GV 6-24mo no better</td>
<td></td>
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<tr>
<td>IOOA</td>
<td>15%</td>
<td>72-78%</td>
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<tr>
<td>DVD</td>
<td>2%</td>
<td>62-76%</td>
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Side effects: Development? See previous slides

Current State of Treatment

Optometric treatment
- Therapy is slow.
- Why is therapy not always successful?
- What else should we consider during treatment?
- Are deficient muscle positions/strength the CAUSE of strabismus?
  - ie-diplopia patient from hospital, int XT patients, “stubborn” ET, vertical deviations, etc…
- Others
  - OT’s, PT’s, Bodywork, DC’s, Cranial Osteopathy/Cranial-Sacral, Acupuncture, myofacial release

Optometric side effects
- Time
  - Monofixation syndrome
- Loss of surgical opportunity
Big Picture - Neurological vs. Muscular vs. Other Causes

- Treatment of causes vs. treatment of symptomology
- The simpler thing is to treat it surgically vs. developmentally
- Faster outcomes in surgery, but it may not be stable due to current adaptations and/or they have reached maximum ability (e.g., monofixation syndrome)
  - Consecutive xT following Infantile ET surgery
  - Vertical diplopia after 3.00 ATR cylinder Lasik surgery. Ophthalmologist wants to do surgery for 4th nerve palsy?
  - Really, a nerve palsy from Lasik?
  - Vertical deviation following Infantile ET surgery
  - Nystagmus after strabismus surgery

Current State of Treatment

Surgery as first approach is based on symptoms, not the cause.
Most often OD’s and OMD’s think and treat as simply ET or XT
If we can we define the type, cause, and underlying adaptations that sets the stage for the emergence of the strabismus we will have better treatment strategies.

We must have proper diagnosis before prognosis!!!
Why do we allow OMDs to make rules regarding therapy that they are ignorant of?

Types of eso strabismus

- Cranial Nerve Disease (palsy/paresis, congenital/developmental anomaly, tumor, vascular, inflammatory, trauma, immune, etc.)
  a. Nuclear Location
  b. Fasical Location
  c. Subarachnoid Location
  d. Cavernous Sinus Location
  e. Orbital Location
- Neuro-muscular Junction (Myasthenia, etc)
- Muscular Disease (congenital/developmental anomaly, tumor, vascular, inflammatory, trauma, immune, etc.)
- Orbital Disease (pulley, congenital/developmental, tumor, vascular, inflammatory, trauma, immune, etc.)
Types of strabismus

- **Esotropia**
  - Infantile Esotropia syndrome
    - Abduction deficit
    - Leading to cross fixation
    - Inflammatory or structural cause of CN6 deficit?
    - Petro-sphenoidal Ligament (Ligament of Gruber)
    - Vaccination, reports of recurrent CN6 involvement
    - Others
    - Birthing process, craniosynostosis, etc
  - Most studies are showing motion processing limited in development
  - Vestibular origin??? VOR/OKN asymmetry, adaptations...DVD/IOOA

Infantile Esotropia

- Nixon, et.al. study
- Sondi, Archer, VonNoorden study
- PEDIG 2002 - 175 infantile eT (40% reported at birth)
  - Parents observations?
- Scheiman and Wick reviewed infantile esotropia - found 28-54% of esotropia is infantile in origin
- What percentage did you say earlier?

Types of Infantile Esotropia

- Infant, constant eT, Abduction deficit, VOR no help
- Infant, constant eT, Abduction deficit, VOR helps
- Infant, No abduction deficit
  - Intermittent eT, clears spontaneously
  - Intermittent eT, straightens with gaze change, blink, nose tap
  - Intermittent eT, occasional alignment, int. cross fixator
  - Constant eT, cross fixator
- Toddler/Adult, no surgery, visual sequella less prevalent (IOOA, DVD, motion asymmetry, LN)
- Toddler/Adult, had surgery, visual sequella prevalent
- Any above combined with accommodative esotropia
What Causes Abduction Deficit?

- How long does it take to establish ET with an abduction deficit?
- When does it disappear?
- RARE-bilateral sixth nerve palsy, uni-maybe
  - Diagnoses is usually limited to those that are seen at birth through 1 year or older
- Birth trauma
- Viral infection
- Vaccination – recurrent 6th nerve paresis cases reported
- Other

Birch and Stager prism study

Compensatory prism with infantile esotropia
- Up to 4 mo, strabismics showed some stereo = normals
- After 4 mo, the stereo still dropped off with compensatory prism in place vs. normals
- Why?
  - Ocular motor may not be symmetrical
  - Development of atrophy/contracture?
  - Development of tonus to support ARC?
  - Lack of proprioceptive support?
  - No change in abduction deficit or motion asymmetry?
    - As convergence becomes more active, more input from asymmetry
  - What mechanism supports higher level of stereo?
  - Brain Periodization – pruning?

Neurologic Concerns of Early Infantile Esotropia Surgery

- Treats symptoms vs. causes
- Short vs. Long term results
  - Increases Amblyopia!
- Monofixation syndrome is considered optimal success
- Removes possibility of spontaneous recovery (Shon-2001, et.al.)…how can we reason spontaneous recovery?
Neurologic Concerns of Early Infantile Esotropia Surgery

- Effects upon proprioceptive feedback
  - Feldenstruktur fibers, 20% of EOM fibers, 1:1 and 2:1 nerve-motor ratio
- Effects upon pulley/double insertion of EOM (Brunech, Ruskell)
- Appropriate age for surgery has yet to be determined
- Complications / Side effects of surgery

Types of eso strabismus

- Accommodative Esotropia
  - Pure Refractive
  - Non-Refractive
  - Mixed
- Monofixation Esotropia Syndrome
- Basic Non-Accommodative Esotropia
- Esotropia And Visual or Neurologic Abnormality (e.g., sensory esotropia)
- Intermittent Esotropia
- Divergence Insufficiency Esotropia (pareisis, paralysis). Is this a partial form of abduction deficit?
- Mixed |Partially Accommodative| Esotropia.

Infantile vs. Accommodative ET

<table>
<thead>
<tr>
<th></th>
<th>Infantile</th>
<th>Accommodative</th>
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<tbody>
<tr>
<td>Onset</td>
<td>8-6 mo</td>
<td>&gt;6 mo to 7yrs</td>
</tr>
<tr>
<td>Angle</td>
<td>25-60PD</td>
<td>10-40PD</td>
</tr>
<tr>
<td>Refraction</td>
<td>&lt;+3.00D</td>
<td>&gt;+3.00D</td>
</tr>
<tr>
<td>Amblyopia</td>
<td>uncommon</td>
<td>common</td>
</tr>
<tr>
<td>LN, MLN</td>
<td>common</td>
<td>uncommon</td>
</tr>
<tr>
<td>DVD, IOOA</td>
<td>common</td>
<td>uncommon</td>
</tr>
<tr>
<td>Motion Asym</td>
<td>common</td>
<td>uncommon</td>
</tr>
<tr>
<td>VOR</td>
<td>common</td>
<td>uncommon</td>
</tr>
</tbody>
</table>
Eye Scan 2-3 months (VOR gain)

Eye Scan 4-6 months

DVD vs. IOOA

Dissociated Vertical Deviation (DVD) - is a vertical deviation with decreased visual input

Inferior oblique over action - is a vertical deviation when the inferior oblique is being used (i.e., left eye in right gaze or right eye in left gaze) or over elevation and abduction of the eye
Dissociated Vertical Deviation

- 50-75% of infantile eT have DVD, post surgical
- Usually not before 2 y.o.
- Can be monocular in amblyopic eye
- No dual DVD (bilateral suppression?)
- Head tilt common to same side for fusion or opposite side for suppression of hyper-deviation
- Torticollis may be a postural manifestation of DVD
- DLR is best model I’ve found to describe DVD

Inferior Oblique Overaction

- Rarely before 1 year old
- Up to 75% in infantile esotropia after surgery
- Look L, R - suppression and decreases VF
- Possibly 2ndary to abduction deficit and via Inferior Oblique secondary muscle action or DLR

Overview of Abnormal EOM

- Abduction Deficit – common in infantile eT
  - If you see them early enough!
- Motion processing – Tychsen
- DVD, IOOA, LN – Brodsky
- PEDIG studies –DVD, IOOA, LN
- *Effects of monocular patch vs. binasal?
- Binasals may attempt to equalize sensory input (or balance it?), increase alternation and promote abduction eye as lead eye
There have been attempts by some OMDs to change the approach which mainstream OMDs (AAPOS) consider as standard of care. These OMDs are renegades.

VonNoorden Am J Ophthalmol Review of Surgical Outcomes

- Before 2 yr the optimal outcomes were best
- Before 2 yr ALSO has the highest % of unacceptable outcomes
- Best overall outcome including optimal, desirable, acceptable occurs in older than 4 year old
- More recent studies on earlier surgery show no more cases with stereo, but better level of stereo
- ? type of stereopsis testing
- Why not evaluate abduction deficit/cross fixation?

Rethy, MD-Mistake of Strabology

- Miss the possibility of prevention
- Believe in unknown causes
- If a problem cannot be solved as a whole, take it apart
- Apply symptomatic tx, not causal therapy
- The turn...“will be soon overwhelmed by the sensory adaptation (ARC) more or less rapidly developing the stabilization of the tonus of convergence”
- “The adaptive stabilization of the convergence tonus has to be treated before the harmless causes can be eliminated.”
Limitations of Rethy’s Work

- Based on Donder’s work only in accommodation
- Typically +1-2 D Hyperopia end up +6-8 D
- Wide span of binasal occlusion may have limited the size of fusional fields during therapy, but emphasis is lateral abduction.
  - Often patients are head turners during therapy…but effects upon vestibular input to EOM?
- Plus increases VOR gain, thus affect N-T motion?

“Treatment should be directed toward what is best for the patient, not for the surgeon.”
Stefan Rethy, MD

The Cochrane Database of Systematic Reviews
Interventions for Infantile Esotropia
The Cochrane Library 2005 #4

The main body of literature on interventions for IE are either retrospective studies or prospective cohort studies. It has not been possible through this review to resolve the controversies regarding the type of surgery, non-surgical intervention and age of intervention. There is clearly a need for good quality trials to improve the evidence base for the management of IE.
So can WE treat esotropia successfully?

Types of exo strabismus

- Infantile Exotropia Syndrome
- Intermittent Exotropia
- Low Accommodative Convergence
- Normal Accommodative Convergence
- High Accommodative Convergence
- Monofixation Exotropia Syndrome
- Basic Exotropia
- Exotropia Associated with Visual or Neurologic Abnormality (e.g., sensory exotropia)
- Convergence Insufficiency Exotropia

Types of exo strabismus

- Cranial Nerve (palsy/paresis, congenital/developmental anomaly, tumor, vascular, inflammatory, trauma, immune, etc.)
- Neuromuscular Junction Disease (Myasthenia, etc)
- Muscular Disease (congenital/developmental anomaly, tumor, vascular, inflammatory, trauma, immune, etc.)
- Orbital Disease (pulley, congenital/developmental, tumor, vascular, inflammatory, trauma, immune, etc.)
Types of vertical strabismus

- Apparent Oblique Muscle Dysfunction
- Over-Elevation in Adduction (OEA) [Old, Inferior Oblique Overaction]
- Under-Elevation in Adduction (UEA) [Old, Inferior Oblique Underaction]
- Over-Depression in Adduction (ODA) [Old, Superior Oblique Overaction]
- Under-Depression in Adduction (UDA) [Old, Superior Oblique Underaction]

Types of cyclo-paretic strabismus

- Unilateral Superior Oblique Paresis (Congenital/Decompensated 4th)
- Superior Oblique Paresis (Non-Congenital [old "acquired"])
- Bilateral Superior Oblique Paresis
- Monocular elevation deficiency [old "double elevator palsy"]
- Monocular depression deficiency [old "double depressor palsy"]

Types of cyclo-vertical and other strabismus

- Dissociated Strabismus, Cyclo-vertical Deviation
  - Dissociated Cyclo-vertical Deviation
- Restrictive/Mechanical Strabismus
  - Cyclo-vertical Deviations Secondary to Muscular Disease
  - Cyclo-vertical Deviations Associated with Orbital Bony Disease
  - Iatrogenic Cyclo-vertical Deviations, ("Induced Adhesive Syndromes")
- Neuro-Myogenic Strabismus
  - Myasthenia Gravis
  - Chronic Progressive External Ophthalmoplegia
  - Internuclear Ophthalmoplegia (INO)
  - Skew Deviation
Types of other strabismus

- **Special Forms**
  - Co-Contractive Retraction Syndrome (CCRS, Types 1-3) [Old Duane]
  - Co-Contractive Retraction with Lower Cranial Neuropathy (CCRS, Type 4) [Old, Moebius]
  - Co-Contractive Retraction with Jaw-Eyelid Synkinesis Syndrome (CCRS, Type 5) [Old, Marcus Gunn]
  - Co-Contractive Retraction with Exotropia [Old Synergistic Divergence and “Y.” Exotropia] (CCRS Type 6)
  - Restrictive Hypotropia in Abduction (RHA) [Old, Brown Syndrome]
  - Congenital Fibrosis of the Extraocular Muscles (CFEOM)

Testing

- Neuromuscular evaluation
  - Near/far, Worth, Parks, Hess-Lancaster, OKN, VOR
- Vestibular/vision motion evaluations
  - ROM, VOR (DVA), OKN, PRN, posture, tone, binocular status
- Adaptation evaluation
  - Sitting vs. Standing
  - Prism adaptation – differences in Developmental vs. Acquired Strabismus
- Effects upon mobility

Neurology of Eye Movements

- VOR – Vestibulo-ocular reflex (16 msec)
  - Maintains fixation and stability by registering very short period of time
  - Sub-cortical response at birth
  - Gain is ratio of head to eye movement
  - Two types rotational (semi-circular canals) and translational (otoiths)
    - 1.0 at birth down to 0.6-0.8 when affected by development and other cortical responses
    - Used in therapy for strabismus and other therapies
    - Gain is changed by lenses and therapy
      - Low plus/minus
      - Prism affects in one plane
      - BU, BD, BI, BO – implications
Neurology of Eye Movements

- VOR
- Maintenance of posture
- Kinetic/transitory contractions of muscles for maintenance of equilibrium and EOM during movement - phasic
- Maintains muscular tone of EOMs - tonic posture
  - Specifically a saccule function (vertical stimulation)

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Neurology of Eye Movements

- **VOR**
  - **Testing**
    - DVA: 2 hertz
    - Head thrust on infants
    - ENG
  - Patient complaints – dizziness, lack of coordination, vertigo, reading delays, hx of ear infection, and blur with motion
  - Almost always effected with ABI
  - Sometimes causative in oculomotor deficiencies
  - Primarily reflexive early on, cortical develops control later on a continuum

**DOLL’S EYE TESTING:** When the head is rotated to the left, the endolymph moves toward the left ampulla and away from the right ampulla. When the head is rotated to the right, the endolymph moves toward the right ampulla and away from the left ampulla.
Neurology of Eye Movements

- Vestibular system is fully myelinated at birth
- Sensory system for acceleration/deceleration
- 60% of compensatory eye movements
- Supplemented with OKN and smooth pursuits to provide stable eye movements
- Smooth pursuits overrides (integrates) VOR
  - If SP is overriding mechanism, patients with poor SP ability can only marginally suppress VOR

Summary
- Short or transient eye stabilization and movement that is suppressed or integrated by SP system

Neurology of Eye Movements

- “The semicircular canals respond to angular acceleration and the otoliths respond to linear acceleration. Together they provide inputs for the VOR.”
  - r-VOR and t-VOR respectively

The Neurology of Eye Movements - Leigh and Zee

Neurology of Eye Movements

- OKN - Latency of 140 msec
  - Registers sustained stimulus through sub-cortical with cortical integration (along with the continuum of development)
  - Indirect in infants (sub-cortical) and direct SP pathway in adult type movement (cortical)
  - Stimulated by visual motion input on retina
  - Involves optokinetic system, smooth pursuit, and saccades (see latency breakdown)
  - Testing - OKN drum 60 degrees per second up to 180 degrees per second
Neurology of Eye Movements

- OKN
  - “hard wired” lateral to nasal
  - Development of nasal to lateral begins at 2-3 months
  - Can have asymmetry up to 6 months, but should be symmetric at 9 months
  - Deficiencies found in strabismus or deprivation amblyopia (commonly early onset)

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Why is OKN important?

- Birth T to N present, N to T begins 2-3 mo.
- Symmetry about 9 mo.
- Visual experience needed for maturation
- *Stereo parallels development of OKN 4-6 mo.
- *Can be used as differential test for time of onset / type of esotropia
- Likely on a continuum
- Right and left not necessarily symmetrical

Development of Motion Pathway
Neurology of Eye Movements

- **Pursuits** – 90-150 msec latency –
  - Cortical
  - Driven by retinal slip (motion)
  - 240 degrees per second
  - Thought to override VOR response
  - Motion processing area to flocculus and ventral paraflocculus in cerebellum via VM nuclei in the pontine nuclei.
  - Motor output to EOMs via the VESTIBULAR nucleus.
  - Many other pathways

- **Saccade** – 150-250 msec latency –
  - Sub-cortical and cortical
  - Smooth pursuit tracking interrupted from approximately 51 inches per second at 250 milliseconds up to 86 inches per second at 150 milliseconds performed at 1/3 meter (33 cm)
  - Mediated by the vestibular system through SP/VOR/OKN
  - Large visual-motor movement planned and implemented without peripheral feedback during movement (Brooks, 1986, p. 127)
  - Implies peripheral “map” with schema holding muscle tension, velocity, size information to get to point B.
  - Predictive saccades possible in 12-14 week olds.
Neurology of Eye Movements

- Neural Integrator (cortical control)
  - Prolongs/decreases signal from peripheral vestibular apparatus
  - Signals from SCC/otoliths
- Velocity signal aligns eye to speed of rotation (VOR)
  - Horizontal oculomotor - Nucleus Prepositus Hypoglossi
  - Vertical and torsional oculomotor - Interstitial Nucleus of Cajal
- Integrates information signals from VOR/OKN/SP to allow for normal eye movements

Neurology of Eye Movements

- "Velocity signals from SCC or acceleration signals from otoliths, need a signal encoding eye position" - Herdman, Vestibular Rehabilitation
  - Feldenstruktur fibers role?
Neurology of Eye Movements

- Feldenstruktur fibers
- New findings with electron microscopy (Dr. Richard Brunech)
- 1:1 or 1:2 neuron to motor unit
- So far only found in the ear muscles and EOMs
- 20% of all motor neurons in EOMs
- Not fully developed till 6-8 years of age
- Proprioceptive feedback loop for EOM position
- should we cut? grow back? Near work implications?

Core concepts for applications in VT

- VOR-16 msec latency-
  - Registers brief stimulus through sub-cortical and is overridden or integrated by smooth pursuit system
- OKN-140 msec latency-
  - Registers sustained stimulus through sub-cortical with cortical integration (along with the continuum of development)

Core concepts for applications in VT

- Pursuits – 90-150 msec latency –
  - Cortical (with attention)
  - Cortically Suppresses VOR
- Saccade – 150-250 msec latency –
  - Sub-cortical and cortical
- Multiple inputs with vision
  - Stationary central visual input with moving periphery, vice versa (watching a moving target)
  - Linear/rotary vestibular input with stationary eyes (driving a car)
Duality of the Systems

Spatial Worlds are all found in all sensory systems and are reciprocally interwoven

Proprioception/Tactile, Motor, Vestibular, Auditory, Visual

- Developed vs. acquired
- Differences for early onset vs. late onset strabismus vs. acquired
- When is the initial onset of amblyopia? That's the biggest
- Asymmetry small amount increased for ideal performance, but overly symmetric can be a concern as well

Motor/Proprioception theories

- Motor theory
- Tonic theory (tense motion)

Tonic vs. Phasic Receptors

- Tonic Receptors ("Slow Adapting")
  - The neuron continues to fire until the stimulus is removed
  - Example: You poke yourself with a needle. It will hurt until it is removed.
  - Tonic receptors are constantly on

- Phasic Receptors ("Fast Acting")
  - Fire only when stimulus first is perceived or removed
  - Example: Putting your clothes on; you are only aware of them for a short period of time.
  - Phasic receptors are usually "off" until a stimulus turns them "on".
    - Example: You are not aware of the temperature until it becomes hot or cold.

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FIGURE 1.10. Schematic representation based on Schor's model of the relationship of fast-fusional vergence, slow-fusional vergence, and fixation disparity.

- Elimination of retinal disparity
- Slow-fusional vergence response
- Decay of fast-fusional response
- Gradual shift of tonic position
- Fixation disparity: a steady-state stimulus
- Reduction of effort required of fast-fusional vergence

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Duality of the Systems

- Vestibular - Keiner, et al., Herdmann
  - SCCs are "phasic"; Ooliths (saccule and utricle) are "tonic"

- Visual - Sensory-Motor theories
  - Motor fusion
  - Sensory fusion
  - All appear to have both phasic and tonic components!

Duality of the Systems

- Visual - Binocular Rivalry
  - The development of OKN and stereo

- Visual - Accommodation/Convergence - Slow/Fast
  - Vergence
  - Tx - traditionally we just vergence think about ranges and adaptation?

- Visual - Spatial
  - Ambient/Focal processes
  - Size of target attended to can be variable
  - Spatial Awareness x, y and z-axis
  - Tx Considerations

Duality of the Systems

- Visual-EOM -
  - Input from EOM proprioception and vestibular input to EOM
    - HorizontalTx
    - Linear vs. Rotational
    - Why does it sometimes appear to worsen?
  - Other EOM and SCC relationships -- each one to one
  - Considerations
    - Surgical intervention upon proprioception
    - Ear infections upon abducens
Adaptations

- Short and Long Term Adaptations
  - Older patients - diplopia
  - Younger patients - may learn to suppress
  - Recovery of initial cause (decreased swelling, circulation, rewiring, etc.)
  - Fast Vergence - phasic
  - Slow Vergence - tonic
  - Muscle Length changes with contracture
  - Spreading of contracture
  - Surgical Concerns
  - Other
Types of Amblyopia

**Strabismic Amblyopia**
- Esotropic: Most likely in surgical intervention, less likely with accommodative ET
- Exotropic
- Vertical
- Cyclo

**Refractive Amblyopia**
- Hyperopia: bilateral >6D?, 9?, 12+?
- Myopia: bilateral >-6D?, -9, -12?
- Astigmatism: >3D, 4, 5?
- Anisometropia >2D
  - Spectacle compensation (match BC and CT)
  - Contact lens compensation due to anisokoria (retinal image size)
- Pathologies are not true amblyopias
  - Microphthalmia (>6.00 or so)
  - Colobomas

**Deprivation amblyopia**
- Infantile (congenital) Cataracts
- Ptosis
  - Myogenic – malinsertions (How often does this really happen?)
  - Mechanical
  - Cicatricial
  - Traumatic
- Amblyopia, unspecified – really?
Overall Treatment

General Overview
- Origin: Developmental vs. Acquired vs. Mixed
- Relate to Duality of Systems – Reciprocally Intertwined
  - Example: proprioceptive, auditory, vestibular, visual and space worlds
- Traditional treatment strategies – are they enough?
  - Look for underlying visual deficits impeding therapy
    - Example: Cross fixation and motion processing asymmetry
  - Abduction deficits
- Use other systems in attempt to reset underlying neurology
  - Example: vestibular to drive EOM, modifying cortical control
  - Secondly, yoked vestibular input to yoked EOM control

Further Treatment Considerations
- Jump prism work – relate to motion-processing deficit?
- Ear Infections/Effusion – related to strabismus?

Considerations for Pre-Treatment
- Arousal/Attention before and during each session: Vestibular, Syntonics
- Prep with motion and serotonin/dopamine - duality of systems
- Neurotransmitters
- Nutritional Counseling

Standard Treatment

OVERVIEW
- Lenses and Prisms
- Occlusion
  - Break Cross Fixation Pattern-modify distribution of light (confusion)
    - Bump or move
  - Mono and Binasals-Where do they alternate fixation? Possibility of reducing vertical
  - Bistroke temporals: to work alternating fixation across midline

Vision Therapy
- Traditional vs. Duality of Systems
Traditional Treatment

- **Traditional Visual**
  - Monocular
    - Acuity / Amblyopia
    - Eye Movements
  - Binocular
    - Squinch-spatial aspects
    - MFBF
  - Binocular
    - Levels and Ranges of Fusion

Sequential considerations, but each patient is individual depending upon needs, previous skills – phasic and tonic perspectives

- Monocular
  - Work paresis/palsy
  - Equalize VOR
    - Extend Ranges - Pursuits, Saccades, VOR, Motion-OKN
    - Check cross fixation

- Binocular
  - Watch over and undershooting
  - Cross fixation patterns

- Binocular
  - Paresis/Palsy cases extend binocular ranges, especially in angles of overshooting and/or undershooting, emphasize jump ductions
  - Stereopsis
    - Static vs. Dynamic (does it break down with movement?)
Summary of Traditional Optometric Approaches

- Refractive “Error” Correction
- Prism
- Orthoptic training
- Monocular occlusion
- Surgical referral
- What is the difference between this and OMD?

Developmental Optometry

- Developmental Visual Guidance
- Possible referral for abduction deficit
- Lenses
- Selective Occlusion / Binasal
- Vision Therapy
  - Direct
  - Passive
  - Delayed
- Alternative considerations - Syntomics

Outside the box treatment

Therapy

Use sub-cortical responses with cortical integration for all eye movement functions

Tactile/proprioception inputs
  - Motor space world
  - Posture
  - Stability for accurate eye movements
  - Breakdown of suppression via tact/prop phasic input

Cervical ocular reflex
  - Infinity walk
Outside the box treatment

Considerations in therapy

Auditory
- Auditory processing of motion - inferior colliculus to superior colliculus
- Biaural hearing as precursor to binocular vision?

Vestibular - from SCC and otolith organs
- Direct connection from SCC and otoliths to EOMs
- SCC are phasic inputs to EOMs, Otoliths are tonic inputs to EOMs
- Drives VOR, OKN, SP, Saccades, & PRN (core basis of eye movements)
- Treat phasic in direction of deviated eye: R-turn = SCC stimulation to right
- Treat tonic via linear/vertical stimulation
- Treat phasic via SCC

Outside the box treatment

- Vestibular use to drive EOM function - linear vs. rotational
  - Linear (tonic): Exotropia, affects all EOM
  - Rotational (phasic): Esotropia, each affects separate EOM
  - Mixed input: Belgau, can modify binocular ranges
  - Postural tone in ABI
  - Asymmetric PRN-related to strabismus, ear infections, developmental in origin?
Outside the box treatment

Considerations in Eye Movements

Sub-cortical and cortical motion processing

- VOR, OKN, PRN - vision requires a stable motor base to work efficiently

Neural Integrator

- Prolongs/decreases signal from peripheral apparatus
- Signals from SCC/otoliths
- Velocity signal aligns eye to speed of rotation

Horizontal oculomotor – Nucleus Prepositus Hypoglossi

Vertical and torsional oculomotor – Interstitial Nucleus of Cajal

“Velocity signals from SCC or acceleration signals from otoliths, need a signal encoding eye position” – Herdman (feldenkrais fibers role?)

Outside the box treatment

Considerations in Eye movements

Vision

Infantile ET

Asymmetric bilateral phasic input (2nd to Abduction deficit)
Asymmetric OKN in N-T direction

Accommodative ET

Initially phasic, but can convert to tonic with mid-adaptation

Partial or Non-Accommodative Strabismic ET

Is this phasic vestibular processing?

Proprioception

Outside the box treatment

Considerations in Eye movements

Vision

Exotropia

Low bilateral tonic input
Linear/vertical stimulation to increase overall tone

CI – intermittent or positional XT
Core of CI is overall muscle tonus
Otolith stimulation with prop/tactile feedback
Prevention and Guidance

- Equal Access / Alternation – bottle, feeding, crib placement (West study that the abducting eye leads binocularity)
- Stress Reduction – talk and sing
- Developmental Intruders vs. Motor Guidance
- Vestibular input – possible effect for H and V deviations?
- Observe and break the pattern
- Biochemistry

Vestibular Applications

- Parents including it daily, with fixation (mirror)
  - Consider prism with it therapeutically?
- Increase arousal – involved in therapy, postural control improved, suppression? (BO/BI ranges)
- Repeated VOR during the day
- Post rotary nystagmus to break abduction deficits
- Alternating R and L eyes between rotations, binasal sets the stage

Vestibular Applications

- Pursuits
- Saccades (eye throwing)
  - Monocular prism jumps
- OKN Cloth (motion)
- VOR – doll’s eye
- Monocular prism jumps
- Vergence if fusing (keystone binocular cards)
- Ron, et al. study on oculomotor subsystem transfer
* Binasals help sets stage for monocular work
What vestibular stimulation direction should one emphasize for infantile esotropia patients:

1 – abduction deficit
2 – motion asymmetry

GOAL OF OPTOMETRIC INTERVENTION
1 - Break Cross Fixation
2 - Improve N-T Motion Processing with sub-cortical responses
3 - Promote Alternation and Fusion

Treatment Considerations
- Partial or Non-Accommodative Esotropia
- Fast and Slow Accommodative-Vergence System (Ciuffreda and Hung)
- “Vergence Adaptation” allows decrease in angle over time
- Current view ignores the following:
  - Vestibular component
  - Proprioceptive component
  - Auditory component
Treatment

- Lifespan Management of Case
  - Preventive Care – Use of lenses, visual hygiene
  - Resilience - eT late in day, flu or ear infection, etc.
  - Education on what to expect, what to do if strabismus returns
- Others

Thank You

- Comments, criticisms, complaints, confusion...
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