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Establishment of video-based case library of various nerve palsies and other oculo-motor nerve dysfunctions

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Establishment of video-based case library of various nerve palsies and other oculo-motor nerve dysfunctions

Abstract
This project was designed to start a video-based case library of various oculo-motor dysfunctions. These digital video cases will be made available to the faculty and students of Pacific University. Six existing VHS and Hi-8 videos of patients with oculo-motor dysfunctions and nerve palsies were digitized using digital video technology. These cases were then transferred via firewire cable to Macintosh Computers running imovie 3. All six cases were edited to include pertinent clips, titles, transitions, relevant case history, diagnoses and special effects. Finally, these edited cases were downloaded to iDVD and written onto DVD discs, to be viewed on any computer through iDVD or converted to mpeg format and viewed through Quick Time. Cases were formatted during the editing process to facilitate a presentation to students and doctors. Pertinent case specific information, and testing procedures were presented first, followed by a brief pause for diagnosing. The proper diagnosis was then presented at the end of the video. A general overview of each diagnosis was provided in a Word Document and written to the DVD as well. This overview details the condition in an Etiology, Subjective, Objective, Assessment, Plan format in addition to including case specific information.

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Thesis

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ESTABLISHMENT OF VIDEO-BASED CASE LIBRARY
OF VARIOUS NERVE PALSIES AND OTHER
OCULO-MOTOR NERVE DYSFUNCTIONS

By

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A thesis submitted to the faculty of the
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Abstract

This project was designed to start a video-based case library of various oculo-motor dysfunctions. These digital video cases will be made available to the faculty and students of Pacific University. Six existing VHS and Hi-8 videos of patients with oculo-motor dysfunctions and nerve palsies were digitized using digital video technology. These cases were then transferred via firewire cable to Macintosh Computers running imovie 3. All six cases were edited to include pertinent clips, titles, transitions, relevant case history, diagnoses and special effects. Finally, these edited cases were downloaded to iDVD and written onto DVD discs, to be viewed on any computer through iDVD or converted to mpeg format and viewed through Quick Time. Cases were formatted during the editing process to facilitate a presentation to students and doctors. Pertinent case specific information, and testing procedures were presented first, followed by a brief pause for diagnosing. The proper diagnosis was then presented at the end of the video. A general overview of each diagnosis was provided in a Word Document and written to the DVD as well. This overview details the condition in an Etiology, Subjective, Objective, Assessment, Plan format in addition to including case specific information.
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Case 1
Superior Third Nerve Palsy OS

Etiology of Third Nerve Palsy

- Congenital (rare)
  - Most often unilateral involvement and no other neurological abnormalities
  - Most often all of the extraocular muscles innervated by the third nerve will be affected resulting in exotropia, hypotropia, and ptosis
  - Pupillary involvement may result as a manifestation of the palsy or from aberrant regeneration.
  - Partial nerve palsy can affect superior or inferior divisions of the third nerve and therefore present differently

- Acquired (more common, but still unusual)
  - Rarely bilateral, possible causes are age dependent with trauma accounting for nearly 40% of acquired palsies in children and vascular/diabetic accounting for nearly 24% in adults.
  - Similar findings to congenital palsy, in addition to complaints of diplopia and/or visual confusion if ptosis does not obscure visual axis
  - Further neurological studies are generally indicated to rule out ominous causes such as aneurysm
  - Isolated pareses of individual muscles innervated by either branch of the third nerve are almost never indicative of other neurological pathology.

Symptoms

- Complete palsy – Inability to look nasally, superiorly, and inferiorly, ptosis, blown pupil, incomplete or absent stereopsis.
- Partial palsy – Diplopia and/or confusion. Superior division: ptosis, inability to look up,
  Inferior division - Inability to look nasally or inferiorly, blown pupil

Objective

- Depends on subdivisions involved
  - Exotropia
  - Hypotropia
  - Aberrant Regeneration
  - Altered head posture – most often chin elevation and/or contralateral face turn
  - Ptosis
  - Blown Pupil

Assessment

- Thorough Case History regarding signs and symptoms noticed and duration
- Cover Testing
- EOM's, ductions if underacting muscles
- 3-Step Testing
- Pupil Testing
- Assess adjacent cranial nerves
- Further neurological studies if indicated

Differential Diagnoses
- Myasthenia Gravis
- Brown's Syndrome
- Thyroid Eye Disease
- Midbrain Lesion
- Isolated Inferior Oblique Palsy

Plan
Nonsurgical
- Many cases exhibit at least some degree of improvement (up to 3 years)
- Elimination of diplopia
- Optimize binocularity in as many gazes as possible
- Relieve ptosis if blocking visual axis
- Monitor children closely for amblyopia
- Realistic goals and ergonomic adaptations discussed with patient

Surgical/Injections
- Botox injections into antagonist muscle(s) to prevent permanent contracture
- Surgical options when little expectation of improvement exists
- Options vary with the affected muscles/division of the third nerve

Case Specific Information
- 6 year-old female, parents concerned about left lid lag/ptosis
- Acuities equal
- No other remarkable History
- EOMS (see video) restricted upgaze OS, underaction of superior rectus OS
- Exyclotorsion noticed in ductions OS
- No head tilt in primary position
- Net vertical phoria in primary gaze (OD hyper) – deviation more noticeable when right eye is uncovered because of Hering’s law (RSR also gets the extra neural signal sent to help the LSR elevate)
Case 2
Fourth Nerve Palsy

Other Terminology
- Superior Oblique Palsy

Etiology
- Trauma – most common cause along with congenital palsies.
- vascular infarct
  - (most often from diabetes or hypertension),
- congenital
- rare causes include tumor, hydrocephalus, aneurysm and giant cell arteritis

Symptoms
- Vertical diplopia – in downgaze and in one horizontal direction. Intermittent diplopia may be a concern when the patient is tired as adaptations begin to fail.
- Reading difficulties
- Possibly asymptomatic

Objective
- Decreased inferior movement of an eye when attempting to look down and in.
- The affected eye is hyper in primary gaze. This hypertropia increases when looking in the direction of the uninvolved eye or tilting head toward the ipsilateral shoulder.
- Habitual head tilt toward contralateral shoulder
- Spread of Commitancy – a possibility with a long standing palsy. The Superior Rectus becomes tight if an eye is hyper for a long time.

Assessment
- A good case history must be taken with specific questioning regarding onset of diplopia, trauma or stroke.
- Old photos may be inspected for a head tilt.
- EOM assessment
- 3-Step Testing
- Vertical fusional amplitudes – a congenital palsy will have greater than the normal 1-3 prism diopters, maybe up to 10-15 prism diopters.
- Differential Diagnosis Testing – Tensilon test, Imaging studies, Double Maddox Rod test (if suspect bilateral SO palsy).

Differential Diagnosis
- Myasthenia Gravis
- Thyroid eye disease
- Pseudotumor cerebri
- Incomplete third nerve palsy
• Brown's syndrome

Plan
• Treat the underlying disorder
• Prisms for the hyper deviation in primary gaze
• Vision Therapy options
• Surgery – usually delayed for 6 months after the onset for stabilization and possible resolution.

Case Specific Information
• Assessment
  o 22 year-old female with complaints of occasional diplopia. “My eyes just don’t seem to work well together.”
  o Has always had this problem
  o EOM’s and 3-Step Testing (see video)
• Plan
  o Increase fusion capability by working ranges in VT
  o Prism
Case 3
Complete Third Nerve Palsy

Etiology of Third Nerve Palsy
- Congenital (rare)
  - Most often unilateral involvement and no other neurological abnormalities
  - Most often all of the extraocular muscles innervated by the third nerve will be affected resulting in exotropia, hypotropia, and ptosis.
  - Pupillary involvement may result as a manifestation of the palsy or from aberrant regeneration.
  - Partial nerve palsy can affect superior or inferior divisions of the third nerve and therefore present differently.

- Acquired (more common, but still unusual)
  - Rarely bilateral, possible causes are age dependent with trauma accounting for nearly 40% of acquired palsies in children and vascular/diabetic accounting for nearly 24% in adults.
  - Similar findings to congenital palsy, in addition to complaints of diplopia and/or visual confusion if ptosis does not obscure visual axis
  - Further neurological studies are generally indicated to rule out ominous causes such as aneurysm.
  - Isolated pareses of individual muscles innervated by either branch of the third nerve are almost never indicative of other neurological pathology.

Symptoms
- Complete palsy – Inability to look nasal, superior, and inferior, ptosis, blown pupil, incomplete or absent stereopsis
- Partial palsy – diplopia and/or confusion.
- Superior division – ptosis, inability to look up
- Inferior division – Inability to look nasally or inferiorly, blown pupil

Objective
- Exotropia
- Hypotropia
- Aberrant Regeneration
- Altered head posture – most often chin elevation and/or contralateral face turn
- Ptosis
- Blown Pupil

Assessment
- Thorough Case History regarding signs and symptoms noticed and duration
- Cover Testing
- EOM’s, ductions if underacting muscles
• 3-Step Testing
• Pupil Testing
• Assess adjacent cranial nerves
• Further neurological studies if indicated

**Differential Diagnoses**
• Myasthenia Gravis
• Browns Syndrome
• Thyroid Eye Disease
• Midbrain Lesion
• Isolated Inferior Oblique Palsy

**Plan**
• If new, refer immediately for neurological workup.
• Maximize remaining function and minimize symptoms with surgery and/or prism.

**Case Specific Information**
• 43 year-old
• Severe CVA involving brainstem a few months prior to video
• OD – Nuclear (complete) right third nerve palsy
• OS – Superior rectus non-functioning due to cross over of the contralateral SR fibers in the nucleus.
• Blown pupil OD
• Prisms and surgical consult
Case 4
Oculocutaneous Albinism

Main Types of Albinism Affecting the Eyes
- Oculocutaneous Albinism
  - Tyrosinase-negative Albinism – not able to synthesize melanin
  - Tyrosinase-positive Albinism – able to synthesize variable amounts of melanin
- Ocular Albinism

Etiology of Albinism
- Genetically determined heterogeneous group of disorders involving deficiency in the enzyme protein tyrosinase, which mediates the conversion of tyrosine to melanin
- Oculocutaneous Albinism
  - Autosomal recessive
- Ocular Albinism
  - X-linked, or less commonly autosomal recessive
  - Female carriers are asymptomatic and have normal vision, although they may show partial iris transillumination, macular stippling, and scattered areas of depigmentation and granularity mid-peripherally.

Symptoms
- Photophobia
- Decreased acuity
- Nystagmus
- Poor depth perception

Objective
- Oculocutaneous (Tyrosinase-negative)
  - Light blue iris
    - transilluminates completely
    - gives rise to a ‘pink-eyed’ appearance
  - Ophthalmoscopy
    - Lack of pigment, giving rise to conspicuously large choroidal vessels
    - May be a marked reduction of vessels forming the perimacular arcades
    - Fovea and/or optic disc may be hypoplastic
  - Visual Acuity
    - Reduced, often to 20/60 or worse
  - Nystagmus
    - Usually pendular and horizontal, increasing on bright illumination
- Severity may lessen with age
  - Refractive Errors
    - Both myopia and hyperopia are common
  - No, or greatly reduced stereopsis

- **Oculocutaneous (Tyrosinase-positive)**
  - Iris Color
    - Can be any color with variable degrees of iris transillumination
  - Ophthalmoscopy
    - Fundus hypopigmentation is variable.
  - Visual Acuity
    - Usually impaired due to lack of differentiation of the fovea.

- **Ocular Albinism**
  - Often difficult to diagnose as pigmentation may be normal, nystagmus mild, and VA’s near normal.

**Assessment**
- Thorough Case History with special emphasis on Family History
- Observation of hair color, including brows and lashes
- VA’s
- EOM’s, noting possible nystagmus
- Iris Transillumination
- Ophthalmoscopy with special attention to:
  - Pigmentation & visibility of choroidal vessels
  - Possible foveal hypoplasia
  - Possible optic disc hypoplasia

**Plan**
- Maximize patient function and minimize symptoms
  - Best correction
  - Tinted lenses for photophobia
  - Yoked prism (base towards nystagmatic gaze)

**Case Specific Information**
- Most likely Tyrosinase-positive Oculocutaneous Albino
- Nystagmus in left gaze only
  - Null point ~30° right gaze
  - Dampens upon convergence
- Habitual left head turn to compensate
- Photophobic
- VA’s 20/25
  - Initially prescribed 10° yoked base left, one year later changed to 8° yoked base left.
  - Refer to chiropracter for head turn.
Case 5
Dissociated Vertical Deviation (DVD)

Other Terminology

- Dissociated Vertical Divergence
- Alternating Hyperphoria
- Double Hypertropia

Etiology

- Idiopathic
  - One possible explanation is that the Inferior Rectus loses innervation, leaving the Superior Rectus unopposed.
  - The normal versions and ductions seen in DVD imply a defect in supranuclear control of eye position.
  - Correlated with infantile esotropia, but may occur with any strabismus that develops early in life
  - May occur as an isolated defect
  - Latent or manifest nystagmus commonly occurs with DVD in congenital esotropia.
  - Seldom (if ever) present at birth, typically appears at 2-3 years of age

Symptoms

- Vary greatly from asymptomatic to physical discomfort produced by a large up drift
  - Often poor binocularity
  - Often poor motor fusion
  - Possible diplopia, especially upon fatigue

Objective

- Spontaneous upward deviation of either eye while it is unilaterally covered
  - Breaks Hering’s Law
  - Binocular vision is restored within seconds when occlusion is removed.
  - The amplitude of deviation is usually unequal between the two eyes, with the non-dominant eye usually deviating more.

Assessment

- History of infantile esotropia, or any other strabismus
- History of diplopia with or without daydreaming or fatigue
- Unilateral and Alternating Cover Tests
- EOM’s, looking for elevation in adduction, producing an apparent Inferior Oblique overaction
- May wish to perform stereoacuity, vertical and horizontal ranges, and other binocularity tests
Differential Diagnoses
• Overacting Inferior Oblique
• Skew Deviation

Plan
• Non-surgical:
  o Eliminate any concurrent strabismus
  o Optimize vision through accurate Rx and treatment of any amblyopia
  o May give prism:
    ♣ Don’t give the full amount
    ♣ Prescribe the difference between the two eyes
• Indications for Surgery
  o Symptomatic
  o Physical discomfort from a large deviation
  o Disfigurement produced by up drift
• Surgical Procedures
  o None is entirely satisfactory
  o One or more of the following may be utilized:
    ♣ Resection of the Inferior Recti
    ♣ Tuck of the Superior Obliques
    ♣ Large recession of the Superior Recti
    ♣ Anterior Transposition of the Inferior Oblique insertion
    ♣ Recession of the Superior Rectus using the Faden Procedure

Case Specific Information
* Nevus on the lower left iris is an incidental finding in this patient.

• Symptoms of physical discomfort, “eyes not working together,” and intermittent diplopia upon fatigue
• DVD revealed with cover testing
• Prism prescribed
Case 6
Congenital Oculomotor Apraxia
(COMA)

Other Terminology
- Congenital Saccadic Palsy

Etiology
- Supranuclear ocular motility disturbance
- Familial gaze palsies are shown to have autosomal recessive inheritance patterns.
- Individual cases, such as MR, indicate a possible autosomal dominant pattern with variable expressivity.
- Tends to affect males > females

Symptoms
- Inability to initiate horizontal saccades
  - Normal pursuits and vertical movements
- Difficulty reading
- Difficulty with speech is common

Objective
- Most apparent during infancy
- Head thrusts to initiate horizontal eye movements (but only 57% of OMA’s exhibit this behavior)
- Frequent blinking
- Tilted head posture
- No quick-phase nystagmus
- Most affected children are slow in attaining early general developmental milestones.
- Later, tend to have poor motor skills

Assessment
- Normal visual workup, with special attention to:
  - Pursuits & Saccades
  - EOM’s
  - Nystagmus Testing
  - Thorough Family Case History
  - Other neurological testing
Differential Diagnoses
- Gaze Palsies
- Slow Saccades
- Visual Field Defects
- Poor Eccentric Gaze Holding

Plan
- Eye Movement Therapy
- Expect mild/moderate educational difficulties, so enlisting the help of special educators may prove valuable.
- Monitor, as the signs and symptoms decrease with age.

Case-Specific Information
- MR presented at eight months old because her mother was worried about possible vision dysfunction, based on excessive turning to view faces and other objects.
- VA’s at eight months: 20/100 with PL
- Gross Visual Fields: Good responses
- Hirshberg/Bruckner: Equal and Central fixation, OU
- Cycloplegic Refraction: +2.50 D Sph OU
- Ocular Health: Unremarkable
- OKN Testing: No response
- Denver Developmental Profile: Delayed in language, but also had a congenital sensorineural hearing loss, and a possible gross motor delay
- Head thrusts used to initiate horizontal eye movements 90% of the time.
References


