Understanding Gaze Nystagmus

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Goals
- To understand eye movements in general
- To understand the HGN & VGN Tests and why they work
- To review other possible causes of nystagmus
- To prepare you for defense arguments

Definition
- **Horizontal Gaze Nystagmus** Test has 3 components
  - Lack of Smooth Pursuit
  - Distinct and Sustained Nystagmus at Maximum Deviation
  - Onset of Nystagmus Prior to 45 degrees

Anatomy & Physiology
- **Retina** is the photosensitive layer of the eye
- **Fovea** is the area of sharp central vision
- **Optic nerve** carries information out of the eye to the brain
- HGN is based on observation of eye movements: Why do we need to move our eyes?
- Acuity is poor except at the fovea
  - fovea – 20/20
  - 5 deg from fovea – 20/60
  - 10 deg from fovea – 20/100
  - 20 deg from fovea – 20/200
Eye Movement Machinery

- 6 muscles per eye working in pairs produce 3 types of eye movements:
  - horizontal
  - vertical
  - rotation

Basic Brain Structure

- 3 of the 12 pairs of cranial nerves control eye muscles
- Each nerve has its own control center deep under the brain (brainstem)
- The cerebellum acts as a gateway for information going to and coming from the brain (neural integrator)

System is fragile and may fail in many ways

- 3 to 5% of population have strabismus - eyes do not align
- About 0.5% of population have nystagmus - eyes do not stay still
- May be constant or intermittent
  - depending on gaze angle and/or viewing distance
  - strabismus will be more frequent with stress, fatigue, low O₂

Basic Eye Movements

- **Vergence** - eyes move in opposite directions
  - Convergence
  - Divergence
  - Near Triad – Convergence, Focus, Pupil Miosis

- **Version** - eyes move together in the same direction: up, down, or to the side; muscle pairs in each eye must work together
  - Saccades
  - Pursuits
Saccades

• Usually big and fast to reposition foveas, up to or greater than 300 deg/sec
• *Ballistic*: “Fire and forget”
  ¬ Brain calculates new position and fires
  ¬ No feedback during movement
  ¬ *Vision is suppressed during movement*

Smooth pursuits

• Slow, following movement, usually no faster than 100 deg/sec
• MUST have a target to follow
• Brain sends constant feedback to eye muscles
• Almost everyone can follow accurately to about 30 deg/sec
• If smooth pursuit is not fast enough, saccades will be present

Development of Eye Movements

- Babies and many animals move the whole head
- Next, infants develop saccades
- Pursuits develop last
- Eye movements are lost in reverse order with intoxication
  ¬ If smooth pursuit system is sick, saccades will be used
  ¬ Some drunks cannot hold head still during testing

Control of Eye Movements

- Conscious
- Automatic
  ¬ Reflexive
  ¬ Vestibular
Vestibular apparatus
- Small, bony, fluid-filled structures located in the inner ears
- Semicircular canals detect head / body movement
- Otolith organs detect head / body position

Vestibular System
- Links to eye position centers via 1 pair of cranial nerves to brainstem and cerebellum
- Eyes stay on target when head moves
- Vestibulo-Ocular Reflex (VOR)
- Failure produces oscillopsia

Alcohol (ETOH) alters the fluids within the vestibular apparatus
- Vestibular system sends bogus information, especially when the head is not upright

Mismatch between vision and vestibular information
Result is dizziness or "motion distress"

Common vestibular problems
- Meniere’s syndrome
  - episodes of intense dizziness & nausea
- Endolymphatic hydrops
  - chronic Meniere’s
- Benign paroxysmal positional vertigo (BPPV)
  - move head to specific position
- Tumors, vision problems, and brain abnormalities

Bogus vestibular data can result in eye position shift
- Vision says nothing moved and resets position
- Vestibular moves, eyes reset

OR, Neural integrator wants to reset eye position
- Vision or conscious effort tries to keep eyes to side

Resulting in repeated back and forth motion called...NYSTAGMUS ! ! !
Nystagmus

➢ Involuntary, repetitive eye movements
➢ Typically reduces acuity
➢ Over 40 named types, based on:
  • Type of Movement
  • Medical Condition
  • Conditions in the Environment

Classification of Nystagmus

➢ Based on Type of Movement
  • Pendular
    ➢ eyes swing back & forth almost equally in both directions
  • Jerk (a.k.a. Beat)
    ➢ eyes drift slowly and move back quickly
    ➢ may be horizontal, vertical, or rotatory
    ➢ specified by Direction, Amplitude, and Frequency

➢ Based on Medical Condition
  • Congenital – idiopathic
  • Albinism
  • Vestibular abnormality
  • Brain problem, e.g., tumor, trauma, etc.

➢ Based on Conditions in the Environment
  • Positional
    ➢ when the head or body is in a particular position (but NOT the eye!)
    ➢ Benign Paroxysmal Positional Nystagmus
    ➢ Positional Alcohol Nystagmus
• Caloric
  - temperature difference between the two inner ears
  - *Cold, Opposite; Warm, Same*
  - also present in zero gravity, BUT NOT after swimming, shower, etc.

• Rotation
  - constant angular acceleration; spinning
  - will adapt with continued stimulation

• Post-Rotation
  - when spinning stops

• Optokinetic Nystagmus (OKN)
  - everybody exhibits this under the proper conditions

• Endpoint (similar to Maximum Deviation)
  - seen at the limits of gaze
  - very common, up to 60% of population
  - typically small in amplitude, difficult to see
  - dissipates in a few seconds
  - usually symmetric in the two eyes

• Gaze-induced (a.k.a. Gaze-evoked)
  - slippage and recovery of the visual image caused by suppression of the smooth pursuit system by disease or certain drugs
  - present when the eye is looking to the side, but NOT straight ahead

• Vertical
  - jerk nystagmus present when looking up, but not straight ahead or down
Effects of Drugs

- ETOH chemically alters the fluids within the vestibular apparatus
- Depressants (including ETOH), inhalants, and phencyclidine (PCP) may affect one or more nerve control centers and/or the cerebellum
- With intoxication:
  - smooth pursuits are replaced with saccades when following a target
  - endpoint nystagmus is exaggerated and lasts longer than normal (but the cause is not well-understood)
  - jerk nystagmus is present when looking at a target to the side
  - presence of vertical nystagmus indicates use of PCP or very high doses of alcohol, depressants, and/or inhalants

Law Enforcement Applications of HGN

- Testing is done one eye at a time with one point given for each clue - maximum 6 points
- Score of 4 points or more corresponds to a BAC of 0.08 / 0.10% or higher with about 80-90% accuracy in the field
- When combined with WAT and OLS, accuracy of discriminating BAC of 0.08 / 0.10% increases to 93% or better
- VGN will be present ONLY when at least 4 clues of HGN are present
- Head (and body) should be still
  - Control of balance via vestibular system and peripheral vision
  - BUT body sway will not affect test or observation
- Pre-testing
  - Check for equal tracking
  - Check pupil sizes
- Lack of Smooth Pursuit
  - Inability to use pursuit eye movements to follow a target moving about 120 deg in 4 sec, i.e. 30 deg/sec
  - CANNOT be controlled voluntarily
Distinct and Sustained Nystagmus at Maximum Deviation
- Sustained for 4 sec or more
- Large amplitude – easy to see
- CANNOT be controlled voluntarily

Onset of Nystagmus Prior to 45 degrees
- NO nystagmus when looking straight ahead
- Jerk nystagmus when looking to the side
  - direction of fast phase = direction of gaze
- Angle of Onset before 45 deg
  - ETOH only: BAC \( \approx 50 \) – AON
  - Other DIP drugs: indicates presence but not quantity or quality of drug
- Symmetric in the two eyes
- CANNOT be controlled voluntarily

Vertical Nystagmus
- NO nystagmus when looking straight ahead or down
- Jerk nystagmus when looking up
  - ETOH only: high BAC for that person
  - Other DIP drugs: indicates presence but not quantity or quality of drug
- CANNOT be controlled voluntarily

Problems with HGN and Solutions

Insufficient officer training
- Detection of nystagmus takes training
- Must differentiate HGN from other types of nystagmus

Certified DRE/DUI officers undergo extensive training
- Participate in workshops and field certifications

The test is not 100% accurate!
- 100% accuracy will catch only high BACs, OR
- To catch all non-zero BACs will result in many false positives
Probable cause and other tests will corroborate the findings
  - This is what courts and judges are for
Suspects with neurological or systemic pathology
  - Vestibular disease/infection
  - Heart attack
Medical rule-out – officer saves a life!
Or, suspect demonstrates same signs to a doctor when sober
  - BUT get a blood or urine sample!
Other medical causes of nystagmus?
  - etc.?
NOT types of nystagmus officer expects to observe
Suspect is very fatigued
  - Extreme fatigue may reduce nystagmus onset angle by as much as 5 deg – NOT PROVEN
  - Extreme fatigue may exacerbate endpoint nystagmus – NOT PROVEN
While the suspect may not be intoxicated, s/he probably should not be driving
  - Sleep!
Suspect cannot see the officer’s finger or penlight without glasses
  - Essentially not possible unless the suspect has a cane and a dog
Officer should ask if suspect can see his/her finger or penlight before starting the test
Suspect claims influence of flashing lights or moving traffic
  - Exhibits OKN
Officer makes sure suspect does not face lights or traffic
Opposition to Law Enforcement Use of HGN

- Scientists and doctors who think that HGN is voodoo
  - Studies PROVE that cognitive reasoning is impaired with use of ETOH (and DIP drugs), AND
  - Studies PROVE that HGN failure increases with higher BACs, BUT
  - Impairment can arise from many sources
- Scientists and doctors who think that police cannot be trained to test eye movements
- Intoxicated individuals
- Those paid to oppose its use
  - Will keep us honest, but also will attempt to confuse judge, jury, prosecutor and officer
- Two commonly cited paragraphs from “Nystagmus and Saccadic Intrusions and Oscillations,” by Dell’osso and Daroff, in Duane’s Clinical Ophthalmology, Vol. 2, Ch. 11, pp. 21-22 (1997) [reprinted on last page]
  - What are the messages from these paragraphs?
    - Alcohol, other depressants and inhalants can cause nystagmus
    - Pathology can cause nystagmus
    - Drugs at levels too low to affect driving can cause nystagmus
      - Then why was the traffic stop made?
    - CURSORILY trained law officers cannot make accurate evaluations of nystagmus
      - Who can disagree with that?
    - Some suspects will be “mis-diagnosed” based on traffic stop evaluations
      - This is why the test is not 100% accurate
      - Convictions based ONLY on HGN data are unlikely

Summary

- Many things can cause nystagmus, BUT few things other than intoxication with Depressants, Inhalants, or PCP can cause the pattern of abnormal eye movements and nystagmus seen with this condition
References

- Admissibility of Horizontal Gaze Nystagmus Evidence (May 2003)
  - APRI National Traffic Law Center
  - www.ndaa-apri.org
  - www.nhtsa.dot.gov
- 1993 American Optometric Association Resolution on HGN
Excerpt from “Nystagmus and Saccadic Intrusions and Oscillations,” by Dell’osso and Daroff, in Duane’s Clinical Ophthalmology, Vol. 2, Ch. 11, pp. 21-22 (1997):

Drug-induced nystagmus is a common sequela of barbiturate, tranquilizer, phenothiazine, and anticonvulsant therapy. The nystagmus is generally regarded as gaze-evoked and is usually horizontal or horizontal-torsional in direction. Vertical nystagmus is often present on upward gaze and only rarely on downward gaze. At times the nystagmus may be dissociated in the two eyes despite the lack of structural disease to account for the asymmetry. Although primary-position nystagmus is usually indicative of severe drug intoxication, it may appear 10 hours after the oral ingestion of 100 mg of secobarbital. In addition, this amount of secobarbital can produce positional nystagmus. As mentioned, lithium can produce downbeat nystagmus, tobacco can induce upbeat nystagmus, and severe alcohol intoxication can produce downbeat nystagmus that abates during sobriety.

Unfortunately, the fact that alcohol can produce horizontal gaze-evoked nystagmus has led to a “road-side sobriety” test conducted by law-enforcement officers. Nystagmus as an indicator of alcohol intoxication is fraught with extraordinary pitfalls: many normal persons have physiologic endpoint nystagmus; small doses of tranquilizers that wouldn’t interfere with driving ability can produce nystagmus; nystagmus may be congenital or consequent to structural neurologic disease; and often a sophisticated neuro-ophthalmologist or oculographer is required to determine whether nystagmus is pathologic. It seems unreasonable that such judgments should be the domain of cursorily trained law officers, no matter how intelligent, perceptive, and well meaning they might be. As noted, meticulous history taking and drug-screening blood studies are often essential in evaluating patients with nystagmus. Toluene (glue-sniffing) can induce pendular nystagmus with both horizontal and vertical components.