A Behavioral Optometry/Vision Science Perspective on the Horizontal Gaze Nystagmus Exam for DUI Enforcement

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Horizontal Gaze Nystagmus (HGN) requires no specialized equipment, is easily learned, and is a reliable, readily applied psychophysical test. It is one of three standardized field sobriety tests (SFSTs). The SFSTs essentially test the skills required to safely operate a motor vehicle. The HGN exam demonstrates the absence or presence of certain characteristics of eye movements that are consistent with the subject being under the influence of depressants, inhalants, or dissociative anesthetics. The HGN exam is designed to determine whether an officer should release a driver or use the results for a probable cause for arrest.

Four clues support finding probable cause for a driving under the influence (DUI) arrest. Many devices are available that test and record the eye findings for drug impairment (Acutex, n.d.; MCJ EyeCheck, n.d.) as well as programs for training those who are not law enforcement officers for administering vision drug screenings (Safety Center, n.d.). The military, departments of transportation, and various industries implement eye testing where a drug-free work environment is essential for safety (e.g., construction, chemical labs, federal contractors, etc.) and performance (e.g., banking, casino dealers, etc.).

Some parole officers, drug counselors, and even parents are finding the eyes useful in detecting drug impairment (Mockensturm, 2001). The eyes yield physiological information that is highly indicative of impairment due to centrally acting drugs. It is relatively inexpensive and convenient, and it does not present a biohazard or the embarrassment of urine or blood testing.
Definition of Nystagmus

Nystagmus refers to an oscillation of the eyes. Pendular nystagmus is a back-and-forth movement of the eyes at essentially equal speeds. Jerk nystagmus has a slow drifting phase and then a fast corrective phase. The nystagmus in the HGN exam involves a jerk nystagmus that is evident when a target is presented under certain testing conditions. HGN does not appear without a target.

A Brief Introduction to the HGN Exam

HGN is one of the three standardized field sobriety tests well researched by the National Highway Traffic Safety Administration (NHSTA), National Institute on Drug Abuse (NIDA), and Southern California Research Institute. Sgt. Dick Studdard of the LAPD Motorcycle division was one of the main U.S. sources in beginning observations of the effects of alcohol, and he participated in related studies. Researchers in Finland did extensive studies on many tests for determining impairment from alcohol and certain drugs before the HGN exam became commonplace in the United States. Researchers found that nystagmus testing was most beneficial in detecting a blood alcohol content (BAC) greater than .06 (Penttila, Tenhu, & Kataja, 1971, 1974). Law enforcement officers receive extensive education and training in the SFSTs. The “Walk and Turn” and the “One-Leg Stand” are the other two SFSTs. The Romberg test yields further information.

Central nervous system depressants (tranquilizers, barbiturates, alcohol), inhalants, and the dissociative anesthetics such as phencyclidine (PCP) influence most brain and bodily functions, including fine and gross motor nerve systems. That includes oculomotor control such as the ability to follow a moving target and the ability to maintain eye position. The examiner observes the subject’s ability to keep his or her head steady while controlling his or her eyes to follow a moving target 12–15 inches in front of his or her eyes and slightly above eye level. If the subject is unable to maintain the head position in a steady posture and cannot complete the exam, he or she is typically impaired. The sterno cleido mastoid muscles are linked to the eye muscles, so when eye muscle function becomes difficult, the neck muscles...
are often employed as a compensatory action (Forkiotis, 1987).

Steps of a Field Sobriety Test

Questioning
The officer asks about physical defects, sickness, trauma, medications, and recent alcohol use; when the individual started drinking, when he or she was last imbibed, how much he or she drank, etc.

Preparing for the Eye Test
The following instructions are given: “I am going to test your eyes. Please remove your glasses.” Uncorrected vision is more than sufficient in following a target. The eye doctor typically performs smooth pursuit testing on patients in the exam room without spectacles—even on patients with the highest prescription lenses.

If glasses are not apparent, the examiner asks if contacts are worn. Contact lenses are not typically removed for the HGN testing. They will not fall out of the eye under the testing conditions unless there is an existing problem with the lens/eye relationship. Clinically, contacts do not cause nystagmus.

Beginning the Test
The examiner gives the driver the following instructions: “Place your feet together with your arms down and at your side. Keep your head still. Move your eyes to follow this target. Do you understand?” The examiner proceeds to demonstrate how the test is done. A penlight target or similar target is sufficient for use, as demonstrated from research and clinical experience. For officer safety considerations, a pencil, pen, or other object that may be used as a weapon is discouraged. The instrument is held 12–15 inches from the eyes, slightly higher than eye level. The lids are tied to the muscles that elevate the eye, allowing a better view of the eyes. Raising the eyes slightly enables this function. It should be noted that the 12- to 15-inch distance allows ease of viewing and provides the officer a safety zone, with space between the examiner and the subject. Being an angular measurement, the actual distance would otherwise seem arbitrary, except that the bulk of research was conducted at that range.

The pen light or instrument is moved laterally to the left of the subject in a straight line outward to maximum deviation, back across the midline to the right side maximum deviation, and back to the midline.

Normal Vision Function

The eye doctor, especially the behavioral optometrist, typically assesses the vision system of every patient. Eye-muscle testing is one of the many areas assessed in the eye exam. The behavioral optometrist is particularly interested in the oculo-motor system, from normal performance to the many abnormalities and anomalies. An eye doctor should be aware that the patient might have certain substances in his or her system that may influence the outcome of the eye exam. Substances that influence pupil size, convergence, and focusing may result in a less-than-optimum lens prescription. Some drugs/substances may decrease the intraocular pressures compared to their normal levels. This is important for the glaucoma-screening portion of the exam. If the eye doctor’s exam reveals eye movements consistent with alcohol or certain drugs as well as other physiological findings, that patient should be re-examined when not under the influence and given counsel on substance dependence programs.

The eye doctor is also able to determine visual impairment from age-related changes of the eye, such as cataracts, macular degeneration, and other vision difficulties interfering with the safe operation of a motor vehicle. The eye doctor is often requested to perform a medical examiner’s motor vehicle application evaluation of vision performance, or in many states, during an eye exam, to inform the patient and motor vehicle department of any vision-related reason that driving privileges should be suspended. Regarding drugs that influence pupil size, any substance—whether drugs acting on the central nervous system or a topical ocular drug—that causes changes to the individual’s normal pupil state may create visual impairment. For instance, the glaucoma drug pilocarpine will cause miosis (constricted pupil). The patient should be warned that driving under dim illumination might prove difficult. A drug-induced small pupil will create vision difficulties if combined with other ocular health problems such as cataracts. Ocular drugs and other drugs that produce mydriasis will also produce an undesirable effect such as increased glare and decreased acuity and lead to visual fatigue (Wood, 2003).

From clinical experience, a person exhibiting a sustained jerk nystagmus at maximum deviation who lacks the ability to smoothly follow a moving stimulus shows a distinct sustained jerk nystagmus before 45 degrees, and his or her nystagmus is more typically resulting from certain drugs, inhalants, or alcohol. (Cerebellar and brainstem disease will often, but not always, exhibit a rebound nystagmus.) These substances influence the brain, particularly the brainstem and cerebellum. Peripheral vision becomes constricted. At near viewing, within 18 inches, the eyes tend to slip apart, but over-convergence occurs at distance viewing, causing space perception and distance judgment to become increasingly difficult (Forkiotis, 1987). Tasks that divide a driver’s attention, such as attending to traffic conditions, roadside signs, and rearview mirrors, become more difficult.

If a subject is driving erratically, appears to be impaired, or has difficulty on the SFSTs, whatever the cause of impairment, the driver should be removed from the road until he or she is determined to be a safe driver. Individuals with physical, mental, or visual problems who drink and drive greatly increase their burden of vigilance and fatigue, which may drastically and adversely affect their driving ability.
The examiner observes the eyes for smooth pursuit and the ability of the subject to maintain his or her head position. The rate of movement of the target is approximately 2 seconds from center to maximum, or 20 degrees per second. The typical speed of the pursuit system is 30 degrees per second, with some individuals being capable of tracking at 100 degrees per second. The test does not exceed the typical capabilities of the unimpaired individual. The target is moved with two excursions. If the subject is unable to smoothly track the target, one clue is recorded for each eye exhibiting lack of smooth pursuit.

The target is then moved for two excursions for each eye to maximum deviation and held for approximately 4 seconds, assessing if there is a distinct and sustained nystagmus at maximum deviation. Notice this is for 4 seconds rather than 30 seconds. About 60% of the population will exhibit nystagmus at maximum deviation if held longer than 30 seconds (Duke-Elder, 1954).

**Finding the Onset**

The next step requires the target to be moved out laterally at a slower pace, looking for the onset of nystagmus. Nystagmus occurring before 45 degrees is consistent with the individual having a minimum of .08 grams of alcohol per 100 milliliters of blood—BAC (blood alcohol concentration)—if alcohol is the only drug. Typically, the closer to the midline the nystagmus appears, the higher the BAC (Southern California Research Institute, 1981).

**Finding Ocular Clues**

The SFSTs are administered to not only find the BAC, but also to determine whether ocular signs typical of impairment from alcohol, depressants, inhalants, or PCP exist. If the BAC via breath or blood is less than expected from the HGN, then a depressant, inhalant, or PCP is the likely substance causing the impairment or combination. Scoring consists of one clue for each eye exhibiting lack of smooth pursuit, one clue for each eye that sustains jerk nystagmus at maximum deviation, and one clue for each eye that displays jerk nystagmus before 45 degrees. Vertical gaze nystagmus (VGN) observed along with HGN is consistent with high amounts of depressants, inhalants, or PCP present. VGN, if present, is viewed by moving the target upward. The eyes will beat in a vertical fashion. The results are noted, but VGN does not add extra clues (VGN without HGN is typically not from drug/substance, but rather from a pathological problem).

HGN is different from most other types of nystagmus. HGN has a slow phase toward the primary position (drifting in the direction of forward), and a fast corrective phase returning into the direction of the target, thus it is a jerk nystagmus. It, unlike most other nystagmus types, requires a target to be followed. It does not require specialized equipment, is easily taught and learned, has been heavily researched, and has been found to be a valid indicator of impairment. Unlike other SFSTs, HGN cannot be diminished with practice. Scoring four or more out of a possible six clues is considered to be a failure.

**Ten Myths and Misconceptions**

Some of the misnomers, myths, or half-truths that are perpetuated in cyberspace and through urban legends follow. Research and science shows these to be outright false or not plausible.

**Myth One—**“My client’s vehicle spun around several times, so he was exhibiting post-rotational nystagmus. This jerk type nystagmus is what the officer saw, not HGN.”

This is not plausible. Rotational and post-rotational nystagmus are jerk types of nystagmus (Duke-Elder, 1954). During rotation, the vestibular apparatus will induce a jerk nystagmus. When the rotation ceases, the fluid in the semicircular canals continues in motion, stimulating the nystagmus for a short time—again, a jerk nystagmus. Before the officer administers the HGN eye test he is observing the subject. If nystagmus were present without the introduction of a stimulus or target, this would be duly noted. HGN must have a stimulus, and the fast phase of the nystagmus is in the direction of gaze, toward the right when presented in right gaze, and left in the left gaze. Post-rotational nystagmus exists with the fast phase in one direction and is short-lived.

**Myth Two—**“My client did not exhibit HGN, but did have caloric nystagmus. This jerk nystagmus was the result of one ear being in the warm vehicle, and the other exposed to the cold air in the environment.”

This also is not plausible. A 7 degree centigrade (13.4 degrees Fahrenheit) difference from the two vestibular apparatus must exist for caloric nystagmus to occur. It takes a determined effort to induce and maintain a 7 degree centigrade temperature differential between the right and left vestibular systems. This is a jerk type nystagmus, and typically is induced in the office of an ear, nose, and throat specialist, using fluid or air introduced into the ear canal. This nystagmus is not as target dependent as HGN is, and the fast phase is toward the warmer ear (Duke-Elder, 1954).

**Myth Three—**“My client was smoking heavily in the vehicle. Nicotine is known to induce a jerk type nystagmus. This is what my client exhibited, which the officer mistook for HGN.”

This is an incomplete-knowledge problem. Nicotine-induced nystagmus (NIN) produces a jerk nystagmus that moves in the vertical meridian, with the fast phase in the upward direction, thus it is an upbeat nystagmus (Sibony, 1987). The officer would not see this without specialized laboratory equipment, because this NIN occurs in the dark, and there is no visual target. Once a target is presented, this nystagmus disappears. In HGN, the nystagmus has the fast phase in the direction of gaze and has to have a target to follow.

**Myth Four—**“My client had a Meniere’s attack. The nystagmus was from this and not HGN.”

Individuals who are experiencing a Meniere’s attack are terribly dizzy and unstable on their feet. Meniere’s is a condition involving the hearing and balance apparatus of the ear. Episodes of decreased hearing, buzzing, and fullness in the ear and vertigo are typical presentations of a Meniere’s attack. Attacks may last 1–2 hours, but the nystagmus may sometimes appear 1–2 days afterward. They do have a jerk nystagmus, which most of the time is on the lateral plane. Typically, the fast phase is toward the healthy ear during the attack and is present without the introduction of a target (Hain, 2005; Duke-Elder, 1954). Because HGN has to have a target to follow and the direction of the fast phase changes with the gaze, a Meniere’s attack is discernable from HGN.
Myth Five—“My client ingested a large amount of glycerol. This induced a nystagmus that the officer mistook for HGN.”
When certain substances enter the endolymph (fluid in the semicircular canals) and change the specific gravity and when the head is positioned in a certain way, a nystagmus may be created. This jerk nystagmus may be viewed with the eyes open but occurs most easily without fixation or a target. This nystagmus must occur when the head is at an angle relative to the level, putting the semicircular canals in position to induce the effect. Alcohol and glycerol are two of the substances that may induce this (Rietz, 1987). When alcohol is the cause, it is termed positional alcohol nystagmus, type I and II (PAN I & PAN II). Type one (fast phase toward the lower ear) is when the plasma concentration of the foreign substance is higher than the endolymph. Type II (fast phase toward the upper ear) is when the concentration is higher in the endolymph than in the foreign substance. The presence of this phenomenon is not used as an indicator of impairment. The direction of the nystagmus reverses when the concentration balance changes. PAN I- and PAN II-induced positional nystagmus are not target dependent. They occur with head positions not used when administering the HGN eye test.

Myth Six—“My client has physiological micro-nystagmus. The officer saw that and thought he saw HGN.”
Physiological micro-nystagmus is an extremely small oscillation of the eyes visually fixating a point or target, preventing fatigue of the retinal cells. It is not visible to the naked eye. It is not a jerk nystagmus, as is HGN, and is not mistaken for HGN because HGN is visible to the visually unaided observer or a macroscopic observation, whereas physiological micro-nystagmus is a microscopic phenomenon.

Myth Seven—“My client did not have HGN, but had physiological endpoint or a fatigue nystagmus.”
Barany, in 1906, showed that fatigue nystagmus occurs in approximately 60% or more of the population if the stimulus is held at the position of maximum deviation (the farpoint of the eye out laterally) for 30 seconds or longer (Duke-Elder, 1954).
The portion of the HGN exam where the stimulus is held at maximum deviation is approximately 4 seconds, not 30 seconds, and sustaining nystagmus at maximum deviation is typical with the use of depressants, inhalants, PCP, alcohol, or any combination.

**Myth Eight—“My client took an antibiotic, creating a nystagmus that was indistinguishable from HGN.”**

Many substances are ototoxic/vestibulotoxic (negatively influence the organs of hearing/balance) including aminoglycoside antibiotics, aspirin, quinine, and many more. Tobacco and caffeine may contribute to the condition. These substances and other problems may contribute to problems in the vestibular system, such as labyrinthitis and benign paroxysmal positional vertigo (BPPV) (Black, Pesznecker, Homer, & Stallings, 2004; Li, 2004), where in certain head positions or changes in head position, dizziness and nystagmus may occur. BPPV nystagmus is easily discerned from HGN. The nystagmus in BPPV can typically be induced via the diagnostic Dix-Hallpike test position (as performed in an ear, nose, and throat specialist’s office) where the individual begins from sitting to supine, with an approximately 20 degree neck extension. The head is then rotated approximately 45 degrees toward the ground. A geotropic (toward the earth) nystagmus occurs with fast phase toward the top of the head, rotating toward earth. Unlike HGN, it is not target dependent. The episode (paroxysmal refers to a sudden episode) typically lasts 20–30 seconds. Trauma or otitis media may also be responsible. Labyrinthitis may create a nystagmus, but it is not head position dependent. These phenomena exist without the presentation of a target, where HGN exists only with a target.

**Myth Nine—“My client is a diabetic and the officer saw eye problems from the diabetes, not HGN.”**

When the officer is administering the HGN exam, he or she also establishes whether equal tracking (the eyes teaming together) exists or not. A diabetic with poorly controlled glucose may experience a paralysis of one of the muscles that control the movement of the eye. Commonly, it is the muscle that brings the eye outward that is affected (abducens paresis), resulting in lack of equal tracking. This is quite different from nystagmus. If that were the case, the officer would make note. There is a condition called gaze paretic nystagmus that occurs in certain conditions, most typically while an eye muscle paralysis is resolving, but the range of motion is still limited. A nystagmus will appear at a particular position of gaze. Myasthenia Gravis and Grave’s disease are two examples of diseases affecting eye muscle function, and they may be associated with gaze paretic nystagmus (Serra, 2002; Baloh, 1989). Unequal tracking is typically present when gaze paretic nystagmus is from eye muscle problems. Brain stem and cerebellar disease may give an eccentric gaze nystagmus, where the eye is held at a particular gaze. This is most observable under dark conditions with infrared imaging. If asymmetric (right and left position), the lesion is typically on the side of the greater nystagmus. The most common cause of symmetric gaze-evoked nystagmus is depressant drugs, inhalants, alcohol, and PCP (Bardorf, 2005).

**Myth Ten—“My client was experiencing nystagmus from a passing train.”**

Railroad nystagmus, or opto kinetic nystagmus (OKN), is a jerk nystagmus with the fast phase opposite of the direction of travel if something such as a train is passing (Duke-Elder, 1954). It is different from HGN. In HGN, the fast phase is in the direction of gaze. The HGN exam has the subject move his or her eyes following the target to the right and the left, with the fast phase into the corresponding directions. In the situation, if OKN were induced inadvertently in the field, the direction would be in one direction only, opposite of the direction of the moving vehicles. Officers are careful to position the subject in order to eliminate the phenomena as a factor, even though the effect does differ from HGN.

**Additional Myths**

In addition to the 10 most common myths aforementioned, other inaccurate claims of nystagmus being indistinguishable from HGN include nystagmus from hyperventilation, sound (Duke-Elder, 1954), galvanic stimulation (Duke-Elder), compression, vibration (Hamann, 1999), head shaking (Hain, 2005), and valsalva (Hain, n.d.). These are associated with nystagmus produced in clinical testing of vestibular anomalies and other diseases, are not target dependent, and are easily distinguishable. Claims of conditions such as glaucoma, color blindness, rare achromatopsia (Duke-Elder), retinal detachment, and cataracts (infantile) causing nystagmus may be true, with the qualifier that they exist as congenital or in early development, typically associated with impaired vision.

It is possible in adulthood that some of these conditions acquired later in life may cause blindness or vision impairment that may lead to nystagmus, but holding a valid driver's license would be difficult to a sufferer (Duke-Elder). In addition, vision-loss nystagmus is not target dependant. Amblyopia is said to have a nystagmus. There is a nystagmus that may be associated with strabismus (eye turn) and/or amblyopia (lazy eye). This is referred to as latent nystagmus (fusion maldevelopment nystagmus syndrome), where the better eye is occluded and occurs without a target (Duke-Elder). However, under the conditions of the HGN exam, both eyes are open. Again, latent nystagmus is quite different from HGN.

Migraines have been said to cause a nystagmus. Some migraines may be thought to have a relationship with the earlier mentioned BPPV, but the nystagmus is not typically dependant upon the presentation of a stimulus (Benson, 2006). Viral illness and/or fever have been said to elicit a nystagmus. (Otitis media may also be associated with fever, see BPPV section.) Opsoclonus is thought to be of viral origin and generates unusual, seemingly random involuntary eye movements looking nothing like HGN (Duke-Elder,1954). Tumors may be another cause of opsoclonus. Myoclonus often accompanies opsoclonus with unusual involuntary limb movement, thus coined “dancing eye, dancing feet” nystagmus. People with multiple sclerosis may have nystagmus, but typically without a stimulus (Lee 2005). Albinism presents with a pendular nystagmus in the horizontal meridian (Duke-Elder; Curtis, 2005) and exists on that horizontal plane in all directions of gaze, both with and without presenting a stimulus. Nystagmus may rarely occur during an epileptic seizure without a target, but it typically ceases with the episode (Loddenkemper & Kotagal, 2005).

Naturally occurring nystagmus is found at certain positions of gaze, but not beyond or before a certain point. The individual with this will usually notify the officer. It does not affect smooth pursuit or endpoint nystagmus.
Summary

Four or more HGN clues give an indicator of impairment from alcohol, depressants, inhalants, or PCP. Law enforcement officers are thoroughly educated and trained to understand the characteristics and conditions of the clues in HGN, and not merely cursorily trained. Law enforcement personnel are trained to be excellent observers. Anyone can learn HGN, and the authors recommend that school faculty, school nurses, emergency response workers, and students seek instruction on learning the HGN exam. Local civilian police academies are one such resource to learn the skill. Other ocular entities are distinguishable from HGN and usually occur without following a stimulus. Newly acquired nystagmus, unequal tracking of recent onset, or other ocular anomalies alert the officer that a medical situation may be involved.

Driving behavior, the roadside interview, and the SFSTs all provide information for the determination. If these evaluations provide probable cause for arrest, the subject is transported to the precinct where the intoxilizer (breath test) is performed. If the results of the intoxilizer are less than what is consistent with the level of impairment, other drugs/substances are suspected, and consequently, the Drug Recognition Expert (DRE) protocol is applied by educated and certified officers. Blood or urine is collected and sent to toxicology to corroborate the DRE exam findings. Some drugs/substances are detected/measured more efficiently under different testing methods: blood, breath, urine, hair, etc.

Many factors contribute to the decision to arrest, release, or call for medical assistance. Whether driving behavior is impaired from drugs or medical problems, the situation must be addressed.

References


Eugene Robert Bertolli, OD, FACHE, DABCHS, CMI-V, CHS-V, practices behavioral and advanced optometry, diagnoses and treats eye disease, and offers vision therapy services for his patients. He is a Life Fellow in the American College of Forensic Examiners, a Diplomate of the American Board for Certification in Homeland Security, and a Certified Medical Investigator (CMI-V), and he is Certified in Homeland Security (CHS-V).

Constantine Forkiotis, OD, FCOVD, FAAO, CHS-III, who recently passed away, was presented the George Comstock Award by the Connecticut Association of Optometrists posthumously for contributions to optometry and the community. Dr. Forkiotis practiced behavioral optometry over 50 years and contributed greatly to the field. He was a Connecticut State Police Surgeon for a span of 3 decades (pro bono) providing innovative services for troopers. He was a charter member of the Drug Recognition Expert (DRE) Section, served on the DRE Technical Advisory Panel, and was given the honor of “DRE Ambassador” by the DRE Section of the International Association of Chiefs of Police.

The fields of optometry, law enforcement, and forensic science have lost a great friend.

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