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Definitions and Types of Nystagmus and Calculations

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This report will present an organized classification of nystagmus and calculations used in its evaluation. Three major types of nystagmus are discussed: ocular, peripheral vestibular, and central vestibular. Ocular nystagmus can be subdivided into congenital, gaze, central, and optokinetic nystagmus. Peripheral vestibular nystagmus can be subdivided into latent, spontaneous, and induced nystagmus. Types of central vestibular nystagmus include: inverted nystagmus, perverted nystagmus, vertical nystagmus, periodic alternating nystagmus, dissociated nystagmus, and cervical nystagmus. Induced nystagmus can be elicited by caloric, galvanic, rotatory, positional, and cervical stimulations. It can be normal or pathological and may be used to detect dysfunction in the ocular, vestibular, central nervous, or somatosensory systems.

The strength of nystagmus has been measured in terms of duration, frequency (beats per 10 seconds), velocity (of the slow component), and total velocity (amplitude multiplied by frequency). Before the advent of clinical electronystagmography, duration was the commonest parameter used. Today, the velocity of the slow component has become the standard because it most directly measures the vestibular response.

Several sets of calculations are commonly performed on nystagmus elicited by caloric stimuli. Of these, unilateral weakness, hyperactivity, ocular fixation, and determination of the decruitment ratio are the most useful. Directional preponderance is of little value, and multiple studies have shown its lack of pathologic localization. Its use in the clinical evaluation of patients should be done only with caution.

PHYSIOLOGY OF THE OCULOVESTIBULAR SYSTEM

Body coordination results from a combination of inputs from the ocular, vestibular, and proprioceptive systems. Dysfunction in any of these systems can give rise to a balance disturbance which may be characterized by vertigo and nystagmus. Generally nystagmus existing without physiologic stimuli is pathologic, as is a reduction or absence of this reflex during adequate stimulation.

The Oculomotor System The oculomotor system allows a stationary subject to fixate on a moving object. When tracking moving objects, an individual will fixate on the object and follow it using slow movements of the eyes (smooth pursuit). This activity is mediated by the anterior occipital lobe and directed to the occulomotor nuclei with input from the retina, cerebellum, and reticular formation (1). If during smooth pursuit the eyes lose the object, a saccade, or rapid eye movement, is produced to reposition the object on the fovea of the macula. This is a preprogrammed conjugate movement dependent upon the frontomesencephalic pathways with input from the frontal lobes, cerebellum, and reticular formation (1-3). Selective damage to the pathways which produce smooth pursuit will result in the production of a series of small saccades (3). This phenomenon is called saccadic pursuit or cogwheeling. Damage to the frontal eye fields will produce a transitory loss of saccade function (2), and loss of cerebellar control produces ocular dysmetria (3, 4), resulting in the production of multiple small and inaccurate saccades to accomplish the task of a single normal saccade.

The Vestibular System The vestibular system's primary function is to detect motion and allow the body to compensate for it. Thus, it allows a moving subject to fixate on a stationary object. The paired end organs produce a resting discharge and increases or decreases in this activity is perceived as motion. Thus, a destructive lesion will not only decrease the labyrinth's sensitivity to detect motion but will send a message and produce a false sensation of motion. The cerebellum acts as a fine tuner of the system, allowing for more accurate tracking. It also dampens extreme variations of vestibular input and diminishes the initial symptoms that a patient suffers after an acute vestibular insult (5). The resting vestibular discharge is integrated in the brain stem (four vestibular nuclei) with input from the optic system, spinovestibular tracts, cerebellar tracts, contralateral labyrinth, and reticular formation. This information is then transmitted by the medial longitudinal fasciculus and reticular formation to the oculomotor nuclei producing eye movement, opposite head motion, and facillitating fixation. In man, the eye movement is always slower than the head movement so optokinetic and pursuit reflexes are also called into play. If the subject is moving too fast for the slow component to produce fixation, a quick eye motion is produced which allows the eyes to catch up and refixate (6). Hallpike and Hood (7) found that the velocity of the slow component corresponded to the calculated deflection of the cupula in the labyrinth and McCabe (6) has shown the quick component to be mediated by the reticular formation in the brain stem. The independent generation of slow and quick components is emphasized by the "doll's eyes" reflex in

the lightly comatose or anesthetized patient which is pure slow component.

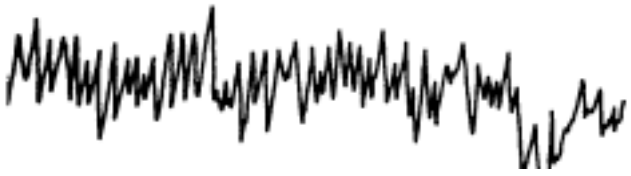
DEFINITIONS OF NYSTAGMUS

The term nystagmus is derived from the Greek word nystagmos "to be sleepy" and nystazein "to nod." It was coined because of its similarity to the jerking movements of a person's head who is falling asleep in the sitting position. First, the head slowly falls then the person momentarily awakens and the head quickly assumes the upright position. Medically, it refers to a repetitive involuntary oscillatory movement of the eyes. Nystagmus may be undulating (pendular nystagmus-sinusoidal movements of the eyes) or rhythmic (jerk nystagmus-eye movements which have a slow velocity in one direction and a fast velocity in the opposite direction) (Fig. 1). While usually a conjugate eye motion, unilateral nystagmus can exist and is most often the result of central nervous system (CNS) disease. The most common way of describing nystagmus is in terms of the plane of eye movement (i.e., horizontal, vertical, and rotatory) and direction of eye movement (right, left, up, down, clockwise, counterclockwise). By definition, the direction of the nystagmus is named after the direction of the quick component. It should be remembered that the quick component is mediated by the reticular formation and not produced in the labyrinth.

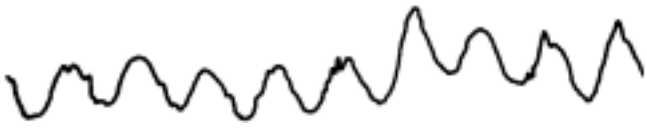
A multitude of classification systems for nystagmus have been proposed. This has resulted in confusion and overlap in terminology. The authors choose to think of nystagmus in these categories: Ocular (visual), peripheral vestibular, and central vestibular. The latter includes projection from multiple other systems and includes cervical nystagmus.

Ocular (Visual) Nystagmus

The term ocular nystagmus can be defined as nystagmus which is produced by reflexes affecting the retina, eye muscles, optic nerve, and their central projections. It also includes nystagmus induced by an orbital or optic stimu



Rhythmic Nystagmus



Undulating Nystagmus

Figure 1. Rhythmic and undulating nystagmus.

lus. We include in this definition congenital, gaze, central, and optokinetic (OKN) nystagmus.

1. Ocular-Congenital Ocular Nystagmus Characterized by an undulatory (sinusoidal movement of the eyes with an equal velocity in all directions) or rhythmic nystagmus (which has a slower velocity in one direction than the other) (Fig. 1). A characteristic feature of this nystagmus is its variability, especially with changes in direction of gaze. Both undulating and rhythmic nystagmus can be found in the same patient (8, 9). For example, when looking to the left the fast component may beat to the left, when looking up the nystagmus may be undulatory, and when looking to the right the fast component may beat to the right. Vertical nystagmus is not seen in congenital ocular nystagmus (10), and upward gaze usually produces a horizontal (10) or undulatory nystagmus (8, 9). Other important characteristics of congenital nystagmus are: the presence of a null point located off central gaze where the nystagmus is markedly diminished or disappears (10) and the marked reduction or absence of nystagmus with convergence (8-10). An important clinical feature of congenital nystagmus is that affected individuals do not complain of symptoms characteristic of oscillopsia. Ocular nystagmus present at birth is seldom caused by visual impairment. Blindness will cause a "searching" undulatory nystagmus which presents itself when the subject is 3 to 4 months old (9). A similar phenomenon is Miner's Nystagmus (10). This nystagmus is undulant and due to prolonged light deprivation. It used to be found in miners working in poorly illuminated mines.

2. Ocular-Gaze Nystagmus (Nystagmus of Eccentric Fixation) Refers to nystagmus produced by changes in eye position (11). We define it as ocular because it is dependent upon the position of the globe. The presence of nystagmus in extreme lateral and medial gaze is normal. The fast component is toward the direction of gaze, the slow component is away. The presence of nystagmus at a gaze of less than 30° is considered pathologic. Vestibular insults may produce nystagmus which is present in only certain directions of gaze. Coats (11) classifies a vestibular nystagmus that is present only in contralateral gaze, as a gaze nystagmus. Alexander et al (12) classifies this type of nystagmus as a 1 ° spontaneous nystagmus. If a gaze nystagmus is present bilaterally, it will beat in opposite directions with right and left gaze. Gaze nystagmus is not present with the eyes in the midline position. Kornhuber (13) requires a gaze nystagmus to be bilateral and beat in opposite directions. If Kornhuber's definition is used, then gaze nystagmus is a strong indicator of central disease (14).

3. Ocular-Central Ocular Nystagmus Rebound nystagmus is a type of central gaze nystagmus which is opposite beating and occurs in the position of primary gaze after a gaze nystagmus is elicited. It is felt to be due to cerebellar dysfunction (15). Central ocular nystagmus can also be produced by dysfunction of the medial longitudinal fasciculus. An example of this is Brun's Nystagmus which is sometimes found in patients with acoustic neuromas. This nystagmus is characterized by a gaze nystagmus when looking toward the lesion (from damage to the ipsilateral pons) and a vestibular nystagmus when looking

away (from damage to the ipsilateral vestibular nerve and nuclei) (16).

4. Ocular-Optokinetic Nystagmus An induced rhythmic nystagmus resulting from attempting to follow repetitive visual targets moving across the visual field. The stimulus may be monocular or binocular. There is clearly interaction between the two sides during binocular examination. A rotating drum with contrasting stripes parallel to the axis of rotation is often used to elicit OKN; however, the stimulus may be as simple as a line of moving newsprint or as sophisticated as computer generated targets.

Vestibular Nystagmus

Peripheral vestibular nystagmus is caused by an imbalance in the resting discharge rate of the paired peripheral endorgans, either the semicircular canals or otolith organs. The nystagmus may be spontaneous or induced by a myriad of stimuli. The physiologic stimulus for the labyrinth is a change in velocity (acceleration) as with the rotatory stimulus of a Barany Chair. Unilateral stimuli which allows comparisons from side to side include caloric and galvanic stimuli. In certain pathologic conditions, nystagmus may be induced by a static change in head position due to stimulation of the spinovestibular tract or gravitational effects on the semicircular canals.

1. Vestibular-Latent Nystagmus Refers to nystagmus which is only present when the subject has no visual fixation (eyes closed or dark room) and is alerted. Alerting is the process of having the subject perform a mental task during an electronystagmogram. This has been shown to increase the maximum slow component velocity of the nystagmus (17). Usually, latent nystagmus is asymptomatic and only detectable during an ENG (electronystagmogram) or with the use of Frenzel Glasses.

Although considered a sign of vestibular imbalance, latent nystagmus has been reported in 11 of 53 normal (asymptomatic subjects) by Milojevic and Allen (18) and in 8 of 60 normal subjects studied by Kumar (14). The nystagmus had a frequency of 6 to 16 beats/ 10 sec (14). Coats (19) considers a pathologic latent nystagmus to be greater than 10°/sec and Kumar (14) considers latent nystagmus to be pathologic whenever it is recorded in patients with cochleovestibular symptoms. (It should be noted that in the above articles the authors use the term spontaneous to describe what we would classify as latent nystagmus, i.e., nystagmus that is only present without ocular fixation.)

2. Vestibular-Spontaneous Nystagmus Defined by Nylen (20) as nystagmus that was not affected by changes in head position. Spontaneous nystagmus is persistent and does not require an eliciting stimulus. It is defined by Coats (1 1) as nystagmus which is present with eyes open or closed and fixed in the midline. Our definition is similar to Coats (11) except that we classify nystagmus which is only present in the eyes closed condition as "latent nystagmus." Alexander et al. (12) classified spontaneous nystagmus as: 1 °, only present in lateral gaze in the direction of the fast component (for example, away from a labyrinthine lesion); 2°, present in midline gaze and in the direction of the fast component; 3°, present in lateral, midline, and medial gaze (21). It should be noted that according to Coats (11), Alexander's 1 ° spontaneous nystagmus would be classified as a gaze nystagmus. Disorders producing spontaneous nystagmus include labyrinthitis, Meniere's disease (endolymphatic hydrops), brain tumors, and trauma. Spontaneous nystagmus is more often indicative of significant pathology than latent nystagmus. However, Kumar (14) does report its occurrence in 4 of 53 normal (asymptomatic) subjects.

Spontaneous nystagmus resulting from permanent nonprogressive unilateral decrease in end organ output should only last 2 to 3 weeks, after that central compensation will occur and the nystagmus will cease. A latent nystagmus may continue to exist for years in this situation.

3. Vestibular-Secondary Phase Nystagmus A rhythmic nystagmus which occurs after, and in the opposite direction of, an induced nystagmus (18). It was recorded by Milojevic and Allen (18) in 30 of 53 normals after bithermal caloric testing [Fitzgerald-Hallpike caloric test (22)J. They felt that secondary phase nystagmus may be due to habituation or to a transient imbalance of the vestibular nuclei. This phenomenon can be a confounding factor which adds to the inaccuracy in calculating directional preponderance by caloric stimulation. Although secondary phase nystagmus is a normal finding, premature caloric reversal is an indicator of central disease. It is defined as a secondary phase nystagmus which occurs in the first 140 sec following a Fitzgerald-Hallpike caloric and has a slow component velocity of more than 7°/second (23).

4. Vestibular-Positional Nystagmus Defined in 1931 by Nylen (20) to indicate nystagmus produced by changes in head position. It can be further subdivided, as suggested by Rubin et al (21), into nystagmus produced by changes in the labyrinth in relationship to gravity, and nystagmus produced by changes of the labyrinth in relationship to the body. In this report, we will refer to the former as positional nystagmus and the latter as cervical nystagmus. Positional nystagmus is described in terms of its latency (time period from change in head position to initiation of the nystagmus), duration (total length of time that the nystagmus beats), fatigability (shorter duration of nystagmus on repeated stimulation), and direction. The parameters of latency and duration for positional induced nystagmus may not have the same properties as for caloric induced nystagmus. This is due to vascular and anatomical influences on diffusion of caloric induced convection currents which are not present in positional testing. The direction of nystagmus can be defined anatomically (right or left) or in relationship to gravity (geotrophic, toward earth; ageotrophic, away from earth).

Nylen (20) proposed a classification of nystagmus in 1931. Seiferth (24) first proposed the terms of "directionchanging" and "direction-fixed" nystagmus in 1937. These terms are included below in Nylen's classification:

Nylen Type I. Direction changing positional nystagmus. The nystagmus beats in only one direction in each head position but the direction changes with different head positions.

Nylen Type 11. Direction fixed nystagmus. Nystagmus beats in the same direction in all positions. It is present in only one position or shows marked attenuation in intensity in the other positions.

Nylen Type Ill. Irregular positional nystagmus. Nystagmus that may change direction in a given position. This group also includes nystagmus not included in types I and II.

Aschan et al (25) modified Nylen's classification by including the terms persistent and transitory nystagmus. Persistent nystagmus is defined as nystagmus with an infinite duration. Transitory nystagmus is defined as nystagmus with a short duration [usually less than 60 sec (26)].

Aschan Type 1. Direction-changing, persistent nystagmus.

Aschan Type 11. Direction-fixed, persistent nystagmus.

Aschan Type III. Transitory nystagmus, direction-changing or direction-fixed.

Using this classification Aschan type I positional nystagmus has the highest probability of having a central etiology and Aschan type III has the highest probability of a peripheral (labyrinthine) etiology. McCabe and Ryu (27) state that 20% of patients with a direction-changing nystagmus will have a central lesion. Direction-fixed, transient [less than 60 sec (26)] positional nystagmus which has a long latency [up to 5 to 6 sec (28)], fatigability on repeated stimulation, and is geotrophic (toward gravity), usually has a vestibular rather than central etiology. Benign paroxysmal positional vertigo (28, 29) is the most common presentation and is classified as a type III positional nystagmus. The most common type of positional nystagmus is rotatory or elliptical (21, 28). Rotatory nystagmus is described as clockwise or counterclockwise, or as right and left. Right and left refers to the direction of the quick component's vector along the globe at 12 o'clock (28). Thus, a clockwise nystagmus can also be described as to the left (Fig. 2). Horizontal direction-fixed positional nystagmus of up to 6°/sec (slow phase velocity) is normal in adult subjects evaluated with their eyes closed, provided it is present in only two of five head positions (erect, supine, right lateral, left lateral, head hanging) (30).

5. Vestibular-Positioning Nystagmus This has various definitions in the literature. Rubin et al (21) define it as vertigo produced by changes of the labyrinth in relationship to gravity. According to this definition, benign paroxysmal positional vertigo should be referred to as benign paroxysmal "positioning" vertigo (21). Barber and Stockwell (30) refer to positioning nystagmus as that elicited by quick movements of the head, i.e., Hallpike maneuver. This is very confusing because cervical vertigo elicited by a Hallpike maneuver would be defined by Rubin et al (21) as positional (nystagmus produced by changes of the labyrinth to the subject's body) and by Barber as positioning (elicited by quick changes in head position). Uemura et al (31) defines positional nystagmus as persistent nystagmus which is induced by different head positions, and positioning nystagmus as a transitory nystagmus elicited by changes in head position. Thus, the use of the term "positioning nystagmus" is confusing and needs definition whenever mentioned in the literature.

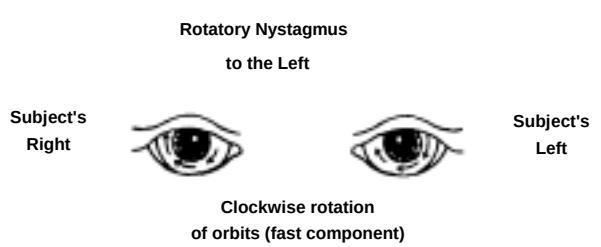


Figure 2. Description of the direction of rotatory nystagmus. Arrows indicate the direction of the fast component.

6. Vestibular-Provocative Nystagmus Positional nystagmus which becomes spontaneous after repeated positional testing (32). The spontaneous nystagmus will subside after a period of time. However, the positional nystagmus will still be present. Aschan and Stahle (32) have reported this finding in patients with vestibular neuritis.

Central Vestibular Nystagmus

Central nystagmus refers to nystagmus that is not characteristically elicited or found in normal subjects or subjects with end-organ disease. Central vestibular nystagmus is produced by dysfunction of the central vestibular system. We include cervical and other forms of somatosensory induced nystagmus in this definition because the neurodysfunction has major central connections with the vestibular nuclei.

1. Central Vestibular-Inverted Nystagmus This refers to nystagmus which beats in the opposite direction than expected; perverted nystagmus is the production of vertical, rotatory or oblique nystagmus from caloric stimulation of the horizontal canal.

2. Central Vestibular-Periodic Alternating Nystagmus

Characterized by a rhythmic nystagmus which builds to a maximum velocity in one direction then slowly diminishes and starts beating in the opposite direction. This cycle is repeated indefinitely. Periodic alternating nystagmus is associated with pathology located in the cerebellomedullary region (33-35). Periodic alternating nystagmus is distinguished from direction changing, positional nystagmus in that the former is recorded during testing for latent and spontaneous nystagmus and the latter is elicited during positional testing. It must also be distinguished from caloric induced nystagmus that is recorded along with an opposite beating spontaneous or latent nystagmus.

3. Central Vestibular-Vertical Nystagmus Also strongly indicates a central lesion. Downbeating nystagmus suggest a lesion in the medulla or upper cervical spinal cord (16, 36). [Note: A downbeating gaze nystagmus can also be seen in upper brain stem lesions, drug intoxication, and Wernicke's syndrome (16).] Upbeating nystagmus suggests a lesion in the posterior fossa or drug intoxication (16, 36).

4. Central Vestibular-Dissociated Nystagmus Characterized by the eyes moving in dissimilar planes. It can result from posterior fossa tumors or lesions involving the medial longitudinal fasciculus (16). Patients with this type of nystagmus usually have disturbing oscillopsia.

5. Central Vestibular-Cervical Nystagmus Refers to nystagmus produced by changes of the labyrinth in relationship to the subject's body. This nystagmus is often pathologic and felt to be from the input of the spinovestibular tracts on the vestibular nuclei in the brain stem. This process gives rise to the clinical correlate of cervical vertigo. It can be found normally in newborn children (37) and in adults who are examined in the dark (38).

ELECTRONYSTAGMOGRAM

While any system capable of measuring changes in eye position over time can document nystagmus, this is most commonly accomplished by recording changes in the corneoretinal potential. An orbital potential was recorded by Schott (39) in 1922 using a galvanometer. Meyers (40) proposed in 1929 that these potentials were derived from the extraocular muscles. However, it was Mowrer et al (41) in 1936 and Fenn and Hursh (42) in 1937 who accurately described these potentials as being derived from the cornea of the eye which is electrically positive in relationship to the retina. Aschan et al (25) in 1956 described a systematic test called nystagmography which utilized this potential to produce graphic records of vestibular function. Varying electrode arrays have been used to record nystagmus in different planes. The horizontal plane is the most common clinical placement of electrodes. Torok et al (43) described photoelectric nystagmography in 1951. This procedure detects eye movement by reflecting an infrared light from the sclerocomeal region of the eye onto a photocell. Eye movements are detected by changes in electrical activity generated by the photocell. Other optical systems to record eye position have utilized digital cameras.

During an ENG, calibration takes place first, followed by the measurement of spontaneous and latent nystagmus. After this, the optokinetic, cerebellar, and vestibular systems are tested. Optokinetic nystagmus is elicited in a manner described above. Cerebellar function is evaluated both during the calibration of the ENG and during pendular tracking. Cerebellar dysfunction will often present as either a calibration overshoot (ocular dysmetria) (3, 4, 44) or an ataxic eye-tracking pendular pattern (45). The labyrinth is evaluated by positional and caloric stimulation. Caloric stimulation can be used for the measurement of canal paresis, ocular fixation, directional preponderance, and decruitment. During all of these tests, alerting is important to reduce central inhibition of nystagmus (17).

Measurement of Nystagmus Strength

Whether one induces nystagmus by rotation, changes in position, or calorics, a method of quantifying the nystagmus response is needed. Early researchers relied on the duration of nystagmus to measure nystagmus strength (22, 46, 47) (Fig. 3). However, this measurement cannot be applied to a continuing stimulus (for example: a rotatory drum stimulus). Fitzgerald and Hallpike (22) also used "briskness" to decide borderline cases of canal paresis. Briskness corresponds to the velocity of the slow component and to the number of beats per unit time (frequency). Although these later measurements were more accurate, they were not widely used in clinical practice until the advent of ENG (25). Measurement of nystagmus frequency is reported by Torok (48, 49) and Kumar (50) and involves calculating the maximum number of nystagmotic beats per 5 sec (Fig. 3). Culmination frequency is defined as the total of the two highest consecutive frequency measurements (48, 49) Nystagmus frequency is related to the velocity of both the slow and quick components. Torok advocates using frequency to estimate the strength of caloric induced nystagmus since he found this parameter to be more consistent and to have smaller standard deviations than the slow-phase velocity (51). He also stated that there is no relationship between the beat frequency of nystagmus and the parameters of duration or amplitude (52).

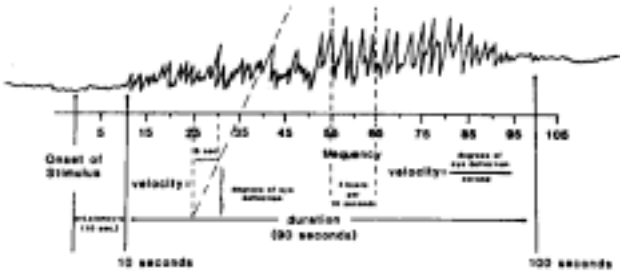


Figure 3. Measurements of nystagmus strength by the parameters of slow component velocity (degrees of eye deflection per second), duration (time period from the onset to the cessation of the induced nystagmus), and frequency (nystagmic beats per 10 sec).

The velocity (slope) of the slow component is the parameter most widely used today. Its use was advocated by Henriksson (53) in an extensive study reported in 1958 (see Fig. 3). He compared the parameters of velocity of the slow component to duration, in estimating the strength of nystagmus elicited by a caloric stimulus. Nystagmus elicited by the Fitzgerald-Hallpike caloric test (22) had a mean duration equal to 155 sec with a standard deviation of 27 sec and a variation coefficient of 18%. The mean velocity was found to equal 29°/sec with a standard deviation of 11°/sec, and a variation coefficient of 38%. Although duration had a much smaller standard deviation and variance, it did not predict changes in vestibular excitation as well as velocity measurements. Varying the caloric stimulus from 29°C to 35°C changed the duration of nystagmus from 128 to 172 sec, as the velocity changed from 9.6°/sec to 31 °/sec. Henriksson concluded that the duration of nystagmus was closely related to the time it takes to restore the temperature in the ear canal, and not to the magnitude of the vestibular reaction. The smaller variance found in subjects is due to "exactitude in restoring normal temperature" (53). The velocity of the slow component was much more sensitive than duration, and slight variations in delivery or labyrinth temperature would produce changes in vestibular response. For this reason, Henriksson advocates the use of the slow component velocity, in estimating nystagmus strength (53). The slope of the slow component is calculated by measuring

the maximum degree of eye deflection per unit time (usually 1 sec) (Fig. 3). Three methods have been used to calculate the velocity of the slow component: (1) the maximum velocity observed in any one beat, (2) the maximum velocity obtained by averaging 10 successive beats, (3) the maximum velocity obtained by averaging all beats in 10 successive seconds (culmination velocity).

The measurement of amplitude (total degrees of eye deflection) has received very little attention in the literature because this parameter has no measurement of time, and only represents the largest eye deviation that is recorded. For example, a strong stimulus can elicit a nystagmus with a large frequency (fast beating) that has a small amplitude. Several investigators advocate using the amplitude multiplied by the frequency (AF) to estimate nystagmus strength (54, 55). This measurement calculates the total degrees of eye deflection (amplitude) per unit time (frequency). The AF is equal to one-half of the total eye velocity (average velocity of the fast and slow component). It can be argued that slow phase velocity is a more accurate estimate of vestibular reaction than AF because it is not influenced by the fast component, which is produced by the reticular formation in the brain stem.

When determining the strength of induced nystagmus by the parameters of amplitude, frequency, or velocity, one must take into account any spontaneous or latent nystagmus which is present. If the preexisting baseline nystagmus is in an opposite direction as the induced nystagmus, its magnitude is added to the magnitude of the induced nystagmus. If it is in the same direction as the induced nystagmus, its magnitude is subtracted from the magnitude of the induced nystagmus. When nystagmus strength is evaluated by the parameter of duration, it must be measured to the recurrence of the baseline nystagmus.

Calibration and Measurement of Spontaneous and Latent Nystagmus

Before recording an ENG, the strip chart must be calibrated. Calibration is necessary in order to accurately measure slow component velocity and to compare one patient to another. This is accomplished by having the patient look to the right and left in a 20° arc. The amplifier is adjusted so each 20° arc of gaze produces a deflection equal to 20 boxes on the ENG recording strip. After calibration, spontaneous and latent nystagmus is recorded. It is very important to alert the subject during this process (17). If latent nystagmus is detected its strength is calculated and used as a correction factor for measuring induced nystagmus.

Measurement of Positional and Cervical Induced Nystagmus

Positional nystagmus is first evaluated by log rolling the patient in the right, left, and supine positions, keeping the head and chest in the same plane. If no nystagmus is produced, cervical nystagmus is tested by turning the head on the body to the right and left and by placing the head in the hanging position. Positional nystagmus should be tested first. If cervical nystagmus is tested before positional nystagmus and nystagmus is only elicited during the first test, it will not be known if the person has cervical nystagmus or positional nystagmus which adapts rapidly. The Hallpike maneuver may also be used to elicit nystagmus. This maneuver consists of rapid turning of the head from the upright to the right or left head hanging position. This maneuver is a very strong vestibular stimulus and not only elicits cervical nystagmus but also can elicit positional nystagmus, which was not produced by log rolling the patient. The latency and duration of the nystagmus should be recorded along with repeating the test to. document adaptation. Since positional nystagmus is usually rotatory, visual inspection is also performed because a perfectly symmetric rotatory nystagmus will not be recorded on ENG. Direction changing positional nystagmus refers to nystagmus which changes in direction with different positions or on retesting the same position. Only the nystagmus which is elicited in the three positions (head right, head left, and head hanging) apply, because it is normal for an opposite beating nystagmus to be produced when the patient sits up.

Measurement of Caloric-Induced Nystagmus

Brown-Sequard (56) was the first researcher to describe vertigo after cold caloric stimulation and Breuer (57) reported the associated production of nystagmus. The fact that the fast component of nystagmus is toward the ear with warm stimulation was first described in 1906 by Barany (58). He was the first to propose a theory that the caloric reaction in the labyrinth is caused by convection currents in the endolymph which mimics rotatory motion (58). Warm and cold vestibular stimulation (in relationship to body temperature) was first described by Dusser de Barenne and de Kleyn in 1923 (59). The utilization of 27 and 48°C stimuli was reported by Lundberg (46), and testing with 30 and 44°C stimuli was reported by Fitzgerald and Hallpike (22) and Jongkees (47). Both Fitzgerald and Hallpike, and Jongkees, described the use of directional preponderance in site-of-lesion testing. Simultaneous bilateral irrigations of 25°C water was described in 1937 by Aubry and Ombredanne (60). Bookler (61) later described the use of simultaneous binaural, bithermal caloric testing using the stimuli described by Fitzgerald and Hallpike (22). Since simultaneous bilateral, bithermal calorics present the same stimulus to opposite ears, a normal response is the absence of nystagmus.

Caloric stimulation, delivered by air or water, can elicit nystagmus by forming convection currents in the semicircular canals (58) which deflect the cupula in one direction or the other. Temperature changes in the vertical canals are smaller than those in the horizontal canal and result in minor convection currents. This is particularly true for the posterior canal (22). The superior canal also responds poorly as reported by Veits (62) who could not record rhythmic nystagmus from this canal during caloric irrigations. Clinically, caloric induced nystagmus is limited to the evaluation of the horizontal semicircular canal. During testing the patient's head is elevated 30° while he is laying supine. This positions the horizontal canal vertically, which is optimal for convection current formation. It appears that cold convection currents cause the endolymph

to fall away from the vestibule (Utriculofugal) and decrease the activity in the ampulla of the horizontal canal. The resultant nystagmus is away from the stimulated ear. The opposite process takes place for warm caloric stimuli. Early researchers have reported the strength of nystagmus is stronger for a cold than warm caloric stimulus. This difference was felt to be due to changes in vascularity caused by the variations in ear canal temperature (47). However, Henriksson (53) explained this difference as being caused by a fall in temperature of the caloric stimulus as it travels through the delivery tube to the ear.

Five calculations have been described for evaluating caloric induced nystagmus. They are: (1) interaural asymmetry in nystagmus to a monothermal, bithermal, or simultaneous binaural bithermal stimulus; (2) asymmetry in total left compared to total right beating nystagmus; (3) bilateral hypoactivity and hyperactivity; (4) calculation of visual suppression of nystagmus; (5) measurement of recruitment and decruitment.

Measurement of Vestibular Responses to Monothermal Vestibular Stimulation

Barber et al (63) and Jacobson and Means (64) described a monothermal warm caloric screening test. Jacobson and Means used a 44°C stimulus of 250 ml of water delivered over 40 sec. A difference in the maximum slow component velocity (averaged from 10 consecutive beats), elicited from the right and left ears, of greater than 29.5% was considered significant. This test diagnosed 75% of subjects with an abnormal bithermal stimulation. When combined with criteria to detect labyrinth hypoactivity the test's sensitivity was 95 %. Barber et al (63) used a 44°C stimulus and considered intra-aural differences greater than 25% significant. They averaged the maximum slow component velocity of the three fastest beats in the 10 sec period of maximum response. This technique produced a 4.8% false-negative rate, and when criteria to detect labyrinth hypoactivity was added, the false-negative rate dropped to 0.7%.

McCabe and Ryu (65) advocate using a ice water caloric (0°C) for determining unilateral vestibular weakness (5 ml of ice water is given over 5 sec). If this does not produce an adequate response, then 10 and finally 20 cc are given. Right and left aural differences of 30% or greater are felt to be significant. Lower values may be due to adaptation and subject variation. As with all caloric tests, the subject must be alerted during the procedure. The following equation is used to calculate unilateral weakness:

$$\text{Unilateral Weakness} = \frac{RC - LC}{RC + LC} \times 100^{*}$$

Torok (66) described a 20°C monothermal caloric that he delivered to the ear in 10 cc over 5 sec (weak stimulus) and 100 cc over 20 sec (strong stimulus). Nystagmus strength was determined by measuring the culmination frequency (maximum number of beats over a 10 sec period). Using this stimulus, Kumar (50) classified seven categories of responses: normal, asymmetric, areflexia (no response), decruitment, recruitment, hypoactive, and hyperactive. Asymmetric responses were diagnosed if the ratios of strong to weak stimulus differed by more than 25% between the ears.

Measurement of Vestibular Responses to Bithermal Vestibular Stimulation

Unilateral weakness (canal paresis) is usually evaluated using a bithermal caloric proposed by Fitzgerald and Hallpike (22). A 30 and 44°C stimulus is delivered to the subject in 240 cc of water over 40 sec. At least 6 minutes should be allowed to elapse between stimuli to allow induced habituation to disappear. The normal variation between right and left sides is 25% or less (23). The following equation is used to calculate unilateral weakness.

Unilateral Weakness

$$\frac{(RW + RC) - (LW + LC)}{(RW+RC+LW+LC)} \times 100^{t}$$

Symmetric Hypoactive and Hyperactive Caloric Responses

Hypoactivity and hyperactivity of the labyrinth can indicate a variety of pathological processes. Hypoactivity can indicate either a central or peripheral process. Complete bilateral loss of activity can be caused by tumors, diabetes, ototoxins, and other disorders (67). Hyperactivity often indicates loss of central inhibition (52). Bilateral hyperactive responses can occur in lesions of the cerebellum and cerebrovestibular tracts, along with other central nervous system abnormalities (52). Unilateral hyperactivity can be of peripheral origin and found in patients with Meniere's disease or early suppurative labyrinthitis. In 24% of patients with hyperactive caloric responses (unilateral or bilateral), a peripheral or inner ear abnormality was identified and in 10% no organic disease was found (52). A loss of central inhibition for only right or left beating nystagmus is the basis of directional preponderance. In evaluating the activity of the labyrinth, the strength of stimulus is critical. Variables for caloric stimulation include the temperature, volume of water, and duration of stimulus delivery.

Hautant (68) felt a labyrinth was hypoactive if the caloric reaction had a duration shorter than 60 sec and felt it was hyperactive if it had a duration longer than 2 minutes. Barber and Stockwell (23) measured the maximum slow component velocity to warm (44°C) and cold (30°C) caloric stimulus. (In the third chapter of their book, Barber and Stockwell (69) described delivering this stimulus in 250 cc of water over 30 sec.) Hypoactivity was defined as less than 6°/sec to a cold stimulus and less than 11°/sec to warm irrigations. Hyperactivity was defined as a slow component velocity greater than 50°/sec for a cold stimulus and greater than 80°/sec for a warm stimulus (23).

* In this and followiing equations the total response to all stimuli is the denominator in order to normalize the value for comparison between individuals.

t This equation can be used for either cold (C) or warm (W) stimuli.

Table 1. Number of patients with hyperactivity as an indicator of central disease [adapted from Torok (52)]

Group	Unilateral[a] (central/total)	Bilateral[a] (central/total)
A	4/7*	10/12*
B	8/17*	17/2**
C	5/7**	Borderline

[a] * includes one patient with head trauma as a central etiology;
** includes two patients with head trauma as a central etiology.

Torok (52), using his weak (10 cc of 20°C water delivered over 5 sec) and strong (100 cc of 20°C water delivered over 20 sec) caloric stimulus, measured the culmination frequency of nystagmus. Normal individuals had a culmination frequency of 3 to 29 beats/ 10 sec for the weak stimulus and 10 to 39 beats/ 10 sec for the strong stimulus. He divided the responses into three groups. Group A: culmination frequencies equal to or greater than 30 for the weak stimulus and 42 for the strong stimulus. Group B: culmination frequency from 24 to 29 for the weak stimulus and 36 to 42 for the strong stimulus. Three percent of normal individuals will have group B hyperactivity. Group C: culmination frequency from 18 to 23 for the weak and 30 to 36 for the strong stimulus. Ten percent of normal individuals will have group C hyperactivity. Patients who had a group C response with bilateral hyperactivity were regarded as having a borderline abnormality, as unilateral group C responses were considered indicative of pathology (Table 1). Unilateral hyperactivity in association with a peripheral lesion was often contralateral and felt to be a "secondary central consequence of a unilateral peripheral lesion" (52). Overall, 65% of individuals with hyperactive vestibular responses (unilateral or bilateral) had central pathology.

Measurement of Directional Preponderance

Directional preponderance [a term coined by Fitzgerald and Hallpike (22)] was first observed in 1911 by Bauer and Lieder (70). They found that after removal of one cerebral hemisphere in the rabbit, deceleration during rotational testing produced a more intense nystagmus toward the side of the damaged hemisphere. He attributed this to an increased function of the ipsilateral labyrinth. Dusser de Barrenne and de Kleyn (59) confirmed these findings. However, they noted increased strength with only ipsilateral cold and not hot caloric stimulation. Thus, they reasoned this phenomenon was not a peripheral process.

Directional preponderance can be based on the duration, frequency (beats per unit time), or velocity (slope of slow component). Torok (49) states that frequency and velocity of nystagmus are interrelated and influenced by vestibular pathology. Nystagmus duration was felt to be controlled by the central nervous system and he presented two normal patients with abnormal directional preponderance of duration but with symmetric directional preponderance of frequency. He theorized that increased duration of caloric nystagmus resulted from loss of central inhibitory factors. An analogous situation exists with Positional nystagmus where increased duration indicates central pathology. It should be noted that directional preponderance was based on nystagmus duration by the early researchers. In current practice directional preponderance is most often based on nystagmus frequency or velocity. Thus, different phenomenon are possibly being evaluated.

The validity of directional preponderance has been debated for many years. Lundberg (46) frequently observed directional preponderance of duration in normal subjects who were studied with a caloric stimulus (27 and 48°C). Fitzgerald and Hallpike (22) further studied directional preponderance of duration using caloric stimuli. They used a right cold (30°C) and left warm (44°C) caloric stimuli (delivered in approximately 8 oz over 40 sec) to mimic nystagmus produced with deceleration to the right and a left cold and right warm caloric to mimic deceleration to the left. As with many early investigators, nystagmus strength was measured by duration. Although briskness was also important in borderline cases. They studied 20 patients with established cerebral lesions. Directional preponderance of duration was found to be present in al] 10 patients with temporal lobe lesions and absent in all 10 patients without temporal lobe involvement. Jongkees (47) found a directional preponderance for duration of 20% or greater in 17% of normal subjects (caloric stimulus of 30 and 44°C delivered in 50 cc of water over 30 sec). Hallpike (71) recorded a directional preponderance for duration in 21 % of patients with Meniere's disease (caloric stimulus of 30 and 44°C delivered over 40 sec at "a constant and fairly free rate of flow") and Anderson (72) recorded directional preponderance to duration 1 Fitzgerald-Hallpike caloric test) in 26% of Meniere's disease and 40% of supratentorial lesions. Anderson (72) also reported that in patients with unilateral supratentorial lesions. the directional preponderance was usually toward the affected side (contralateral side being hyperactive). Kirstein and Preber (73) reported 33 cases of unilateral supratentorial lesions (44 and 30°C caloric stimulus delivered in 100 cc of water over 40 sec). Twenty-five of these cases had an abnormal directional preponderance for duration greater than or equal to 20% . The directional preponderance in all cases was toward the lesion.

Directional preponderance of frequency was studied by Brookler and Pulec (74) in 839 patients with either peripheral or central disease. They found abnormal directional preponderance in 14% of 780 patients with Meniere's disease and in 15% of 45 patients with acoustic neuromas. (A directional preponderance of 30% was considered abnormal.) At this level, only 3.3% of normal subjects had a false positive test. They concluded that an abnormal directional preponderance indicates pathology but has no localizing value (right versus left or central versus peripheral).

McCabe and Ryu (27) studied directional preponderance of slow component velocity in 100 subjects with end organ and central disorders and 100 normal subjects. They found it nonspecific to the site-of-lesion and present in many of the normal subjects. They observed that directional preponderance is affected by the order of caloric stimulation that is given to a subject, and state that directional

preponderance "is worthless as a localizing, or even a general, test of vestibular disease" (27). Barber and Stockwell (23) report that 95% of all normal subjects will have a directional preponderance for velocity equal to or less than 29%. The following equation is utilized to calculate directional preponderance:

Directional Preponderance =

$$\frac{(RW + LC) - (LW + RC)}{(RW+LC+LW+RC)} \times 100$$

Measurement of the Ocular Fixation Index

Demanez and Ledoux (54) described an ocular fixation index (OFI) as a method of quantifying visual suppression of nystagmus. The following formula was used:

$$OFI = \frac{\text{Velocity of slow component (eyes open)}}{\text{Velocity of slow component (eyes closed)}}$$

Alpert (75) compared the various parameters of measuring nystagmus strength that can be used to calculate the ocular fixation index. He studied amplitude (averaged over 10 beats), frequency (averaged over 5 sec), and slow phase velocity (averaged over 10 beats) elicited by a caloric stimulus [Fitzgerald-Hallpike caloric test (22)]. The upper limit of normal, set at the 95 % tolerance level, for the OFI calculated with amplitude, frequency, and velocity was 0.6, 1.9, and 0.6, respectively. Ocular fixation indexes, calculated with slow phase velocity, most accurately discriminated between normal and abnormal patients (75). Abnormal ocular suppression of nystagmus can be seen with cerebellar lesions (76) and congenital nystagmus (10, 23). It is rarely seen with labyrinthine disease. It should be remembered that poor visual acuity can produce an abnormally high ocular fixation index.

The time of measurement of the slow phase velocity is important. Alpert (75) suggests measurement of the eye's closed condition at the maximal caloric response (culmination velocity) and the eyes open condition 10 to 20 sec after the culmination velocity has been reached. Hart (77) proposes having the subject's eyes open at 75 sec, after initiation of the caloric stimulus (Fitzgerald-Hallpike caloric test), and measuring the eyes open condition during this time period. Although this latter technique is much easier to implement in a clinical setting, it cannot be expected to be as accurate as the technique proposed by Alpert (75). The following equation is utilized to calculate the OFI:

$$OFI = \frac{\text{velocity of slow component (eyes open)}}{\text{velocity of slow component (eyes closed)}} \times 100$$

Measurement of Recruitment and Decruitment (Torok Monothermal Differential Caloric Test)

This test involves the repeated stimulation of the labyrinth with more intense caloric stimuli (50, 66, 78, 79). Each ear is irrigated with 10 cc of 20°C water over 5 sec. This is then followed by 100 cc of 20°C water over 20 sec. The maximum number of beats per two consecutive 5 sec periods is then determined (culmination frequency) for each stimulation. A ratio is then calculated (culmination beat frequency of strong stimulation/weak stimulation). A normal ratio is 1.2 to 3.5. Abnormal ratios indicating decruitment have values below 1.1 (type I, 1.1 to 1.0; type II, 0.9 to 0.1; and type III, when no nystagmus was produced with the stronger stimulus) (79, 78). Torok (79) reported 53 of 81 patients with type I decruitment, 45 of 51 patients with type II decruitment, and all 7 patients with type III decruitment had central lesions. He also reported the occurrence of decruitment in patients with hypoactive, normal, and hyperactive responses. Normal symmetric results are produced if ratios of 1.2 to 3.5 are recorded (50). Recruitment is diagnosed if the ratio is greater than 3.5 (50). The following equation is used for calculation of the recruitment ratio.

Recruitment Ratio =

$$\frac{\text{nystagmus frequency (strong stimulus)}}{\text{nystagmus frequency (weak stimulus)}}$$

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