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In the bithermal caloric test, each ear is irrigated twice—once with cool water (or air) and once with warm water (or air). Each irrigation stimulates the horizontal semicircular canal of the irrigated ear and provokes a nystagmus response. Cool stimulations provoke nystagmus responses with slow phases toward the irrigated ear. Warm stimulations provoke nystagmus responses with slow phases away from the irrigated ear.

Caloric stimuli are uncalibrated. Even though all patients receive the same external stimulus, we know that some patients receive stronger semicircular canal stimulation than others, presumably because their external ear canals are larger and straighter. We do make the key assumption that all four caloric stimulations of a given patient are equally strong. Thus we expect some patients to have stronger caloric responses than others, but expect all four caloric responses to be of equal strength in a patient with normal vestibular function.

An example of normal caloric responses is shown in Fig. 1. Each response starts about 20 seconds after onset of the irrigation, reaches peak intensity about 60 seconds later, and then declines. Responses to right cool and left warm irrigations have rightward slow phases. Responses to right warm and left cool irrigations have leftward slow phases. The intensities of all four responses are exactly equal. In real life, the intensities of all four caloric are rarely exactly equal, because caloric stimuli are rarely exactly equal and also because response intensity is affected by extraneous factors, such as alertness, eye position, and recording artifacts. For clarity of presentation, the caloric responses shown in Fig. 1 (as well as in the figures that follow) are idealized responses generated by computer, not actual responses recorded from patients.

**Unilateral Weakness**

The primary purpose of the bithermal caloric test is to detect a unilateral lesion of the horizontal semicircular canal (or its afferent pathway). We can say that such a lesion exists when caloric responses of one ear are significantly weaker than those of the other ear.

An example is shown in Fig. 2. The responses of the right ear are noticeably weaker than those of the left ear.
Is this weakness outside normal limits? The preferred measure of caloric response intensity is peak slow phase velocity, that is, slow phase velocity of nystagmus at the point of strongest response. To calculate unilateral weakness, we insert these peak slow phase velocity values into a formula first proposed by Jongkees and Philipszoon (1964). First we find the difference between ears by summing the peak slow phase velocities of the two responses of the right ear and, from this sum, subtracting the sum of the peak slow phase velocities of the two responses of the left ear. Then, because unilateral weakness is proportional to caloric response strength and caloric stimuli are uncalibrated, we normalize this difference between ears, that is, we divide it by the sum of the peak slow phase velocities of all four responses. Finally we multiply the result by 100. In other words,

\[(\text{RW} + \text{RC}) - (\text{LW} + \text{LC})\]

\[\frac{\text{Eq. 1}}{\text{RW} + \text{RC} + \text{LW} + \text{LC}} \times 100 = \text{UW (in percent)},\]

where \(\text{RW}\) is peak slow phase velocity of the right warm response, \(\text{RC}\) is peak slow phase velocity of the right cool response, \(\text{LW}\) is peak slow phase velocity of the left warm response, \(\text{LC}\) is peak slow phase velocity of the left cool response, and \(\text{UW}\) is unilateral weakness. A positive \(\text{UW}\) denotes a unilateral weakness in the left ear, and a negative \(\text{UW}\) denotes a unilateral weakness in the right ear. A \(\text{UW}\) greater than about 25 percent is generally considered outside the normal limit.

Inserting peak slow phase velocity values from Fig. 2 into Eq. 1 yields

\[(20 + 20) - (40 + 40)\]

\[\frac{20 + 20 + 40 + 40}{20 + 20 + 40 + 40} \times 100 = -33\text{ percent},\]

which is outside the normal limit. This result is localizing. It indicates a lesion of the right horizontal semicircular canal or its afferent pathway.

**Gain Asymmetry**

An example of gain asymmetry is shown in Fig. 3.

Figure 3. Gain asymmetry.

Responses with leftward slow phases are clearly weaker than those with rightward slow phases.

Is this gain asymmetry outside normal limits? To calculate gain asymmetry, we use a formula that was also first proposed by Jongkees and Philipszoon (1964). First we...
find the difference between the two directions of nystagmus by summing the peak slow phase velocities of the two responses with leftward slow phases and, from this sum, subtracting the sum of the peak slow phase velocities of the two responses with rightward slow phases. Then because gain asymmetry is proportional to caloric response strength, we normalize this difference by dividing it by the sum of the peak slow phase velocities of all four responses. Then we multiply the result by 100. In other words,

\[
\frac{(RW + LC) - (LW + RC)}{RW + LC + LW + RC} \times 100 = DP \text{ (in percent)},
\]

where RW, RC, LW, and LC are the same as in Eq. 1, and DP is directional preponderance. A DP greater than about 30 percent is generally considered to be outside the normal limit. A positive DP denotes a directional preponderance to the right, and a negative DP denotes a directional preponderance to the left.

A note on nomenclature: It is customary to designate directional preponderance according to the direction of stronger fast phases. Thus when it is said that there is a directional preponderance to the left, it is meant that responses with leftward fast phases are stronger than those with rightward fast phases. However we think it makes more sense to designate directional preponderance according to the direction of weaker slow phases, since slow phases are being displayed in the chart. Thus when we say there is a directional preponderance to the left, we mean that responses with leftward slow phases are weaker than those with rightward slow phases.

Inserting peak slow phase velocity values from Fig. 3 into Eq. 2 yields

\[
\frac{(10 + 10) - (40 + 40)}{10 + 10 + 40 + 40} \times 100 = -60 \text{ percent},
\]

which is outside the normal limit. This result indicates that the gain of responses with leftward slow phases is significantly lower than the gain of responses with rightward slow phases, which is a nonlocalizing abnormality.

Gain asymmetry is extremely rare. In fact, for many years we did not believe it even existed, but recently Halmagyi et al. (2000) said they found this abnormality in 114 of 15,542 patients who underwent bithermal caloric testing. All of these patients had DP’s of at least 40 percent and none had significant unilateral weaknesses or significant spontaneous nystagmus. About half of them had Meniere’s disease or benign paroxysmal positional vertigo, and most of the others received no diagnosis. Halmagyi et al. said that gain asymmetry is generally a transient, benign abnormality. In a companion paper (Cartwright et al., 2000), they postulated that it is due to an asymmetric dynamic response of neurons in the medial vestibular nuclei on either side of the brain. We have never seen a convincing case of gain asymmetry in our own laboratories (Stockwell, 1987; Barin & Bahram, 2001), but the report of Halmagyi et al. indicates that it does in fact exist.

**BIAS**

An example of bias is shown in Fig. 4. There is no unilateral weakness and no gain asymmetry, but the baseline of all responses is shifted upward, that is, to the right, by about 20 deg/sec. This patient also has spontaneous nystagmus with rightward slow phase velocities of about 20 deg/sec with eyes closed.

Spontaneous nystagmus and bias are both due to a static baseline shift in the horizontal vestibulo-ocular system, and they always occur together. Like gain asymmetry, bias (and spontaneous nystagmus) occurs in patients with a wide variety of vestibular disorders, both peripheral and central.

Many normal individuals have a small amount of bias and spontaneous nystagmus, so how much is outside the normal limit? It is generally agreed that the normal limit is about 6 deg/sec, so the bias of 20 deg/sec seen in Fig. 4 is abnormal.

What happens when we calculate directional preponderance in this patient? Inserting peak slow phase velocity values from Fig. 4 into Eq. 2 yields
(20 + 20) – (60 + 60) \times 100 = -50 \text{ percent.}

This is a mistake. We cannot use Eq. 2 to calculate bias. This formula normalizes the difference between the two directions of nystagmus under the presumption that the difference is proportional to response strength. Bias is not proportional to response strength, so the formula is inappropriate when the difference is due to bias.

Eq. 2 is appropriate for calculating gain asymmetry, because gain asymmetry is proportional to response strength. However we must first eliminate the bias. To do this, we simply subtract the bias from the peak slow phase velocities of caloric responses in the same direction and add it to the peak slow phase velocities of responses in the opposite direction and then calculate gain asymmetry using the new values. In other words,

\[
\left(\frac{RW' + LC'}{RW' + LC' + LW' + RC'}\right) \times 100 = GA \text{ (in percent)},
\]

where GA is gain asymmetry and RW', LC', LW', and RC' are RW, LC, LW, and RC, respectively, after eliminating any bias.

To illustrate, if we eliminate the bias from peak slow phase velocity values in Fig. 4, we get

\[
\begin{align*}
RW' &= 20 + 20 = 40 \\
LC' &= 20 + 20 = 40 \\
LW' &= 60 - 20 = 40 \\
RC' &= 60 - 20 = 40
\end{align*}
\]

and when we insert these new values into Eq. 3, we get

\[
\frac{(40 + 40) - (40 + 40)}{40 + 40 + 40 + 40} \times 100 = 0 \text{ percent.}
\]

This result indicates no gain asymmetry, which is correct.

**Unilateral Weakness and Gain Asymmetry**

The three abnormalities described above can exist alone or in any combination. Fig. 5 shows an example of two coexisting abnormalities—unilateral weakness and gain asymmetry. First we calculate unilateral weakness using Eq. 1,

\[
\frac{(5 + 20) - (40 + 10)}{5 + 20 + 40 + 10} \times 100 = -33 \text{ percent.}
\]

Then we calculate gain asymmetry using Eq. 3,

\[
\frac{(5 + 10) - (40 + 20)}{5 + 10 + 40 + 20} \times 100 = -60 \text{ percent.}
\]

Note that the presence of gain asymmetry has no effect whatsoever on the value we get for unilateral weakness, and vice versa. Note also that since there was no bias, we could have used either Eq. 2 or Eq. 3 to calculate gain asymmetry.

**Unilateral Weakness and Bias**

Fig. 6 shows an example of two coexisting abnormalities—unilateral weakness and bias.
unilateral weakness and bias. The bias is about 20 deg/sec, and the patient has spontaneous nystagmus with rightward slow phase velocities of about 20 deg/sec with eyes closed. We calculate unilateral weakness using Eq. 1,

\[
\frac{(0 + 40) - (60 + 20)}{0 + 40 + 60 + 20} \times 100 = -33 \text{ percent.}
\]

Note that the presence of bias has no effect on the value we get for unilateral weakness, so we do not have to eliminate it first.

This combination of unilateral weakness and bias is characteristic of a sudden and recent unilateral peripheral vestibular lesion. Over time, the bias (and the patient’s spontaneous nystagmus) decline due to vestibular compensation, although the unilateral weakness may remain.

**Gain Asymmetry and Bias**

Fig. 7 shows an example of two coexisting abnormalities—gain asymmetry and bias. The bias is about 20 deg/sec, and the patient has spontaneous nystagmus with rightward slow phase velocities of about 20 deg/sec with eyes closed. Note that the right warm and left cool caloric responses failed to overcome the spontaneous nystagmus completely, so RW and LC are opposite the expected direction. When this happens, their values are expressed as negative numbers.

Before calculating gain asymmetry, we have to eliminate the bias,

- \( RW' = -15 + 20 = 5 \)
- \( LC' = -10 + 20 = 10 \)
- \( LW' = 60 - 20 = 40 \)
- \( RC' = 40 - 20 = 20 \)

Then we calculate gain asymmetry by inserting the new values into Eq. 3,

\[
\frac{(5 + 10) - (40 + 20)}{5 + 10 + 40 + 20} \times 100 = -60 \text{ percent.}
\]

Note that the normal limit of 30 percent for directional preponderance is based on studies that lumped together gain asymmetry and bias. We do not have a normal limit for gain asymmetry by itself, but it is probably somewhat less than 30 percent. We need new normative studies to establish a correct normal limit for gain asymmetry.

**Unilateral Weakness, Gain Asymmetry, and Bias**

Fig. 8 shows an example of three coexisting abnormalities—unilateral weakness, gain asymmetry, and bias. The bias is about 20 deg/sec, and the patient has spontaneous nystagmus with rightward slow phase velocities of about 20 deg/sec with eyes closed. The right warm and left cool caloric responses failed to overcome the patient’s spontaneous nystagmus, so RW and LC are expressed as negative numbers. First we calculate unilateral weakness using Eq. 1,

\[
\frac{(-15 + 40) - (60 - 10)}{-15 + 40 + 60 - 10} \times 100 = -33 \text{ percent.}
\]

We must eliminate the bias before calculating gain asymmetry,

- \( RW' = -15 + 20 = 5 \)
- \( LC' = -10 + 20 = 10 \)
- \( LW' = 60 - 20 = 40 \)
- \( RC' = 40 - 20 = 20 \)

Then we calculate gain asymmetry by inserting the new values into Eq. 3,

\[
\frac{(5 + 10) - (40 + 20)}{5 + 10 + 40 + 20} \times 100 = -60 \text{ percent.}
\]
SUMMARY

In the bithermal caloric test, each ear receives two irrigations—a warm irrigation that provokes a response in one direction and a cool irrigation that provokes a response in the other direction. From these four responses, we can distinguish between unilateral weakness, in which the responses of one ear are weaker than those of the other ear, and directional preponderance, in which responses in one direction are weaker than those in the other direction. Directional preponderance has two components. The first is gain asymmetry, which is proportional to caloric response strength. The second is bias, which is independent of caloric response strength. Before calculating gain asymmetry, it is first necessary to eliminate bias from the responses.

Unilateral weakness is highly localizing. It denotes a lesion of the horizontal semicircular canal or its afferents on the side of the weaker response. Gain asymmetry and bias are nonlocalizing. Both abnormalities denote a vestibular lesion, either peripheral or central.

REFERENCES


A NOTE TO ICS MEDICAL CUSTOMERS

The elimination of bias described in this article cannot be performed by current ICS Medical software and must be done by hand. This feature will be included in our next software release.
THE FIXATION SUPPRESSION TEST

Kamran Barin, Ph.D., and Laurie R. Davis, M.N.S.

The fixation suppression test is part of the standard ENG examination. It tests the patient’s ability to suppress vestibular nystagmus during fixation upon a visual target. The most commonly used test procedure is one described by Alpert (1974). For at least one right-beating and one left-beating caloric response, the patient’s nystagmus is recorded with eyes closed until shortly after the peak of the response. At that time the examiner tells the patient to open the eyes and fixate upon a small stationary target for about 10 seconds. Then the examiner computes the average slow phase velocity of three nystagmus beats just before opening the eyes (SPV\textsubscript{NoFix}) and the average slow phase velocity of three nystagmus beats during fixation (SPV\textsubscript{Fix}), as shown in Figure 1.

From these data, the examiner then calculates a Fixation Index (FI) by the formula,

\[ FI = \frac{SPV\textsubscript{Fix}}{SPV\textsubscript{NoFix}} \times 100. \]

FI is a measure of nystagmus intensity during visual fixation expressed as a percentage of nystagmus intensity just before opening the eyes. If nystagmus is completely suppressed by fixation, FI will be 0%. If nystagmus is incompletely suppressed by fixation, FI will be between 0% and 100%. If nystagmus is enhanced by fixation, FI will be greater than 100%. The normal range for FI is 60% or less, which means that, in 95% of normal individuals, visual fixation suppresses vestibular nystagmus by at least 40%.

FIXATION SUPPRESSION AND SMOOTH PURSUIT

There is strong experimental and clinical support for the idea that the pursuit system is responsible for suppressing vestibular nystagmus (Chambers and Gresty, 1982; Halmagyi and Gresty, 1979). The pursuit system is designed to keep the image of a small moving target on the fovea—the most sensitive part of retina. Movement of the target’s image across the retina causes retinal slip. When the pursuit system detects retinal slip, it triggers an eye movement that tracks the target. This tracking eye movement minimizes retinal slip and tends to keep the target’s image on the fovea. When a person with vestibular nystagmus fixates upon a stationary target, the slow phases of the nystagmus move the target’s image across the retina, thus generating retinal slip. The pursuit system reacts by moving the eyes in the direction opposite the nystagmus slow phases, thus suppressing the nystagmus.

Figure 1. Calculating average slow phase velocity of nystagmus with eyes closed (SPV\textsubscript{NoFix}) and during visual fixation (SPV\textsubscript{Fix}). EO indicates the point at which the patient opened the eyes.
Some investigators (e.g., Barnes, et al., 1978) have said that if the pursuit system is responsible for fixation suppression of vestibular nystagmus, then the fixation suppression test is really a test of the pursuit system, which means that the fixation suppression test is redundant, because the pursuit system is already being tested by two other ENG tests—the tracking test and the optokinetic test. On the other hand, Tomlinson and Robinson (1981) have said that a different system—the vestibular cancellation system—is primarily responsible for fixation suppression of vestibular nystagmus. They have said that the vestibular cancellation system reduces nystagmus intensity and places the image of the target near the fovea, and then the pursuit system eliminates any residual nystagmus. If so, then the fixation suppression test evaluates a different system and is therefore not redundant. It may reveal abnormalities not detected by the tracking and the optokinetic tests (and vice versa). Let us examine this issue by comparing the results of the fixation suppression test with those of the tracking and optokinetic tests.

**NORMAL FIXATION SUPPRESSION WITH NORMAL TRACKING AND OPTOKINETIC RESPONSES**

Figure 2 shows a patient with normal tracking, optokinetic responses, and fixation suppression.

In the tracking test (Figure 2A), the patient follows a small target oscillating back and forth in the horizontal plane. Frequencies of target motion range from 0.2 to 0.7 Hz with peak-to-peak amplitudes of 30° at all frequencies. Peak target velocities range from 20 to 70°/sec. The ratio of peak eye velocity to peak target velocity is determined for each target frequency and compared with age- and sex-matched normative values. The top panel shows a sample of target motion and superimposed eye motion at 0.2 Hz. The bottom panel shows tracking gains for rightward and leftward tracking at each target frequency. Tracking is within normal limits for both directions of target motion at all frequencies.

In the optokinetic test (Figure 2B), the patient tracks a series of small targets moving first to the right and then to the left at a constant velocity of 40°/sec. Nystagmus responses are considered normal if slow phase velocities are at least 30°/sec in both directions. In this patient, average slow phases velocities are 38°/sec for rightward moving targets and 33°/sec for leftward moving targets. Both values are within normal limits. (Note that the optokinetic test, despite its name, is not a test of the optokinetic system; it is a test of the pursuit system. A true test of the optokinetic system would require targets that subtend the full visual field.)

In the fixation suppression test (Figure 2C), FI is equal to 20% for nystagmus with rightward slow phases and 24% for nystagmus with leftward slow phases. Both values are within normal limits.

![Figure 2. Test results from a patient with normal tracking (A), optokinetic responses (B), and fixation suppression (C).](image-url)
**Abnormal Fixation Suppression with Abnormal Tracking and Optokinetic Responses**

Figure 3 shows a patient with abnormal tracking, optokinetic responses, and fixation suppression. In the tracking test (Figure 3A), the patient has saccadic pursuit and tracking gains are abnormally low for both directions of target motion. In the optokinetic test (Figure 3B), average slow phase velocity of optokinetic nystagmus is $3^\circ/\text{sec}$ for rightward moving targets and $6^\circ/\text{sec}$ for leftward moving targets. In the fixation suppression test (Figure 3C), FI is 200% for nystagmus with rightward slow phases and 100% for nystagmus with leftward slow phases. These abnormalities indicate a CNS lesion. They are seen in patients with a variety of neurological disorders involving the cerebellum, brainstem, or cerebral cortex.

**Abnormal Fixation Suppression with Normal Tracking and Optokinetic Responses**

Figure 4 shows a patient with abnormal fixation suppression, but normal tracking and optokinetic responses. In the tracking test (Figure 4A), the patient follows the target smoothly and tracking gains are within normal limits at all target frequencies. In the optokinetic test (Figure 4B), average slow phase velocity of optokinetic nystagmus is $31^\circ/\text{sec}$ for rightward moving targets and $37^\circ/\text{sec}$ for leftward moving targets. In the fixation suppression test (Figure 4C), the patient fails to suppress the caloric nystagmus adequately. FI is approximately 64% for nystagmus with rightward slow phases and 75% for nystagmus with leftward slow phases. This result is uncommon. It denotes a lesion in the central nervous system, most likely in the flocculus of the cerebellum.
Abnormal fixation suppression with normal pursuit supports the hypothesis of Tomlinson and Robinson (1981) that fixation suppression and pursuit are mediated by separate neural mechanisms, but there are other possible explanations. First, tracking and fixation suppression are under voluntary control, so perhaps the patient simply failed to fixate on the visual target during the fixation suppression test. Second, the stimuli used in the tracking and optokinetic tests have predictable trajectories, whereas the timing of nystagmus fast phases is less predictable. Predictable targets are easier to track than unpredictable targets (Leigh and Zee, 1991). Third, the velocity limit of the pursuit system is approximately 30-40°/sec for young healthy persons and closer to 20°/sec for persons over the age of 60. Therefore a patient could have normal pursuit and yet display abnormal fixation suppression if the intensity of to-be-suppressed nystagmus exceeds the patient’s pursuit velocity limit. (It should be noted that this third explanation is implausible in the case shown here, since slow phase velocity of caloric nystagmus just before opening the eyes is only about 24-25°/sec.)

NORMAL FIXATION SUPPRESSION WITH ABNORMAL TRACKING AND OPTOKINETIC RESPONSES

Figure 5 shows a patient with abnormal tracking and optokinetic responses, but normal fixation suppression. In the tracking test (Figure 5A), the patient has saccadic pursuit and lower than normal tracking gains in both directions. The tracking defect is asymmetric, being much worse when the target moves to the right. In the optokinetic test (Figure 5B), average slow phase velocity of optokinetic nystagmus is 16°/sec for rightward moving targets and 27°/sec for leftward moving targets. In the fixation suppression test (Figure 5C), nystagmus is virtually eliminated during fixation and the FI is equal to 0% for both directions of nystagmus.

This result is also uncommon. It also supports the hypothesis of separate pursuit and vestibular cancellation systems, because then it would be possible for a patient with defective pursuit to suppress vestibular nystagmus using the vestibular cancellation system. But again there might be another explanation. Weak caloric nystagmus does not pose much of a challenge to the pursuit system. Thus a patient who has a mild pursuit defect and weak caloric responses may be able to suppress caloric nystagmus, yet be unable to generate normal tracking and optokinetic responses.
**Clinical Implications**

Should we perform the fixation suppression test as part of the standard ENG examination? We think the answer is "yes." The procedure is simple and benign and does not take additional testing time. Two test results presented above (Figures 4 and 5) support the hypothesis that pursuit and fixation suppression are mediated by separate neural mechanisms. However, such results do not prove the hypothesis, since other possible explanations exist. Nevertheless, it is a fact that the fixation suppression test sometimes detects abnormalities in patients who have normal tracking and optokinetic responses and vice versa.

The fixation suppression test has a major shortcoming—the difficulty of the test depends upon how strong caloric nystagmus happens to be when the patient opens the eyes. If the nystagmus is very weak, even a patient with defective pursuit (or cancellation) can suppress it. If it is very strong, even a person with normal pursuit (or cancellation) cannot suppress it. There are other shortcomings as well. Therefore the test must be conducted with care and the results interpreted with caution. We recommend that attention be paid to the following details:

1. Perform the test during all four caloric responses. Then you will have two opportunities to observe fixation suppression for each direction of nystagmus.

2. Closely watch the tracing and ask the patient to open the eyes immediately after the response reaches peak intensity, which usually occurs between 60 and 90 seconds after the onset of the irrigation.

3. When testing fixation suppression, make sure the patient is actually fixating on a small visual target. It is not enough for the patient simply to open the eyes. Some patients are reluctant to fixate, especially if distressed by the dizziness that is part of the caloric response.

4. When measuring nystagmus slow phase velocities, select beats from a 5 second time period just before opening the eyes and a 5 second time period just after fixation. Avoid beats that occur within one second before and after opening the eyes, since these beats often contain artifact.

5. When interpreting the fixation suppression test, recognize that nystagmus intensities between 20 and 40°/sec are optimal for testing fixation suppression. Intensities above 40°/sec may overwhelm even a normal pursuit (or cancellation) system and yield false positive results, especially in elderly patients. Intensities below 20°/sec may not pose a sufficient challenge to the pursuit (or cancellation) system and yield false negative results.

**References**


Comments/Questions on Article

We would welcome any comments or questions you may have regarding this article. Please e-mail us at: edservice@icsmedical.com or send a fax to ICS Medical Educational Services at 847/534-2151. Be sure to give us your contact details so the authors may respond to you.

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We’re pleased to make available a CD containing four interesting Case Studies, which feature video clips of abnormal eye movements gathered on the ICS CHARTR VNG system. The Case Studies are presented by Preston C. Calvert, M.D., Dept. of Neurology, Johns Hopkins University School of Medicine. The CD sells for $10. For more information, please visit our website.

Dizziness Diagnostic Forum and Caloric Test Course Online

These are two valuable educational opportunities offered through the ICS Medical website. The Forum on Diagnosis and Management of Dizziness includes a complete list of disorders that cause dizziness and information about each.

The Caloric Test Course is an hour-long course on administration and interpretation of the Caloric Test.
Best Practices for the Evaluation and Management of Dizziness

Stephen P. Cass, M.D., M.P.H.

Biography

Stephen P. Cass, M.D., M.P.H., is Associate Professor in the Department of Otolaryngology at the University of Colorado Health Science Center. He is fellowship trained in Neurotology, specializing in disorders of the ear, hearing and balance. His research interest involves basic and clinical studies of the vestibular system. Dr. Cass is co-author with Dr. Joseph Furman of Vestibular Disorders: A Case-Study Approach.

We held a two-day course, “Best Practices for the Evaluation and Management of Dizziness: A Workshop with Leading Clinicians.” in Chicago on June 25-26, 2004. The course was presented by the Department of Otolaryngology, University of Colorado School of Medicine and sponsored by the University of Colorado School of Medicine, Office of Continuing Medical Education. Educational support was provided by GN Otometrics, North America. I served as course director. This is the second course in this series. The first course was held in Chicago on October 11-12, 2002. A copy of these proceedings was published as an Insights in Practice article that can be accessed on www.besirebalance.com.

In the first course we heard from clinicians in the specialties of primary care, neurotology, neurology, audiology, physical therapy, and psychiatry. We learned that there is little controversy about management, but a great deal more controversy about evaluation. Dizzy patients usually see a primary care physician first. Most of them have benign disorders that can be successfully managed by the primary care physician, but a few have serious disorders that require referral to a specialist. Most of us agreed that the specialist who accepts dizzy patient referrals should be prepared to take a comprehensive history and perform a thorough physical examination, but we disagreed about which clinical observations are required. Sometimes the history and physical examination fail to yield a definite diagnosis and we need additional information provided by laboratory tests. We disagreed about which tests should be ordered for which patients.

In the second course, I wanted to get closer to a clear definition of best practices for the evaluation and management of the dizzy patient. I assembled a faculty of experts in the specialties of neurotology, neurology, and vestibular testing. I asked them to describe their methods, to present the evidence underlying their decision-making, and to indicate where the evidence is weak. I allowed plenty of time for discussion among faculty members and the audience. In this manner, I hoped to identify areas of consensus and to hear various viewpoints in areas of disagreement.

I began the course with a quick review of vestibular anatomy and physiology. I described the anatomy of the vestibular labyrinth and orientation of the labyrinth in the head, and explained the structure and function of the sensory receptors in the ampullar of the semicircular canals and the maculae of the utricle and saccule. I traced the neural pathways of the vestibular nerve, vestibular nuclei, and the vestibulo-ocular and vestibulo-spinal systems.
causing a decrease in the neural input. Thus the level of neural input from the left ear is greater than the level of neural input from the right ear. As a result, the person has a sensation of leftward rotation, left-beating nystagmus, and a tendency to fall to the right.

Fig. 3 shows what happens when a person suffers an acute lesion of the right horizontal canal. Neural input from the right canal is abolished, whereas tonic neural input from the left horizontal canal remains. Thus the level of neural input from the left ear is greater than the level of neural input from the right ear. This asymmetry is the same as the one that occurs when a person rotates leftward and the reaction is also the same—a sensation of leftward rotation (which we now call “vertigo”), left-beating nystagmus (which we now call “spontaneous nystagmus”), and a tendency to fall to the right (which we can detect with postural tests). Acute lesions in other parts of the vestibular system cause similar (but not identical) signs and symptoms, and as we will see later, we can often localize the exact site of lesion by carefully noting the features of these signs and symptoms.

The lesion may be permanent, but nevertheless the patient's signs and symptoms abate over a period of days and weeks due to vestibular compensation. A major part of the compensation process is a reduction of the asymmetry due to the gradual reappearance of tonic neural activity in the vestibular nuclei on the side of the damaged horizontal canal, as shown in Fig. 4. Compensation is never complete, however. Our physical exam and tests still can detect subtle vestibular abnormalities for many years after the event. Some patients compensate better than others, and those who compensate poorly usually receive benefit from vestibular rehabilitation therapy.

Then I described how I take a history from the dizzy patient. It is often said that history taking is the most important part of the evaluation of the dizzy patient. I agree. Dizziness can be caused by an unusually large number of diseases and it is important to develop a working knowledge of all the medical conditions and disorders associated with dizziness. A good place to start with this task is the Compendium of Vestibular Disorders that can be accessed on www.hearbalance.com. The patient's history offers an important opportunity to gain information needed to distinguish among them and to create a working list of potential diagnoses.

I take a comprehensive (Level V) history from every new dizzy patient—a daunting task. To make it easier, I use an electronic patient records system. In the waiting room, the patient fills out an eight-page questionnaire that asks questions in a forced-choice format. An optical character recognition system yields a finished chart note. I take my laptop into the exam room and review my chart note with the patient, correcting and amplifying as needed.

A complete list of the items on my questionnaire can be accessed on www.hearbalance.com.

David Solomon, M.D., Ph.D., Assistant Professor of Neurology at the Johns Hopkins University School of Medicine, described how he conducts a physical examination of the dizzy patient.

Dr. Solomon performs a thorough examination of eye movements, as follows:

1. **Eye movement exam.** He examines the patient's eye movements with eyes open in the light and with vision denied using Frenzel lenses, or video-oculography. He looks for spontaneous (horizontal) nystagmus, vertical nystagmus, torsional nystagmus, gaze-evoked nystagmus, and dissociated nystagmus.

2. **Head thrust test.** He asks the patient to look straight ahead and then jerks the patient's head quickly rightward and leftward, looking for "catch up" saccades that denote a weak vestibulo-ocular response when the head is jerked toward the side of a labyrinthine loss.

3. **Head-shaking test.** He shakes the patient's head rapidly back and forth 15 times and then looks for nystagmus while the patient's head is held motionless. Head-shaking nystagmus usually (but not always) beats away from the side of a labyrinthine loss.

4. **Hyperventilation test.** Hyperventilation sometimes induces nystagmus in patients with a fistula or a compressive lesion (such as acoustic neuroma, cholesteroloma, or blood vessel) or an inflammatory lesion (such as multiple sclerosis) that causes demyelination in peripheral or central vestibular pathways.

5. **Valsalva maneuver.** The Valsalva maneuver sometimes induces nystagmus in patients with Arnold-Chiari malformation, perilymphatic fistula, or superior semicircular canal dehiscence.

6. **Ocular tilt reaction (OTR).** An unilateral otolithic lesion sometimes causes tonic ocular torsion and skew deviation when the head is tilted toward the side of the lesion.

7. **Dix-Hallpike maneuver.** He starts with the patient seated on the examination table with legs extended and the head turned 45 deg rightward. Then he brings the patient back rapidly to the supine position with the head still turned rightward and hanging backward over the end of the
He also conducts a general neurological examination, which includes evaluation of cranial nerves, deep tendon reflexes, distal vibration sensation, and cerebellar function (finger to nose, rapid alternating hand movements, heel to shin). He looks for dysarthria, dysphagia, dysesthesia, diplopia, Horner’s syndrome, loss of pin prick or temperature sensation on one side of the face and/or the other side of the body, intractable hiccups, visual inversion, visual loss, ocular motility disorder, mental confusion.

In addition, he performs an orthostatic blood pressure screening test, a dynamic visual acuity test, and a headache evaluation as indicated by the presenting history and symptoms.

Kamran Barin, Ph.D., Assistant Professor, Dept. of Otolaryngology and Dept. of Speech and Hearing Science, The Ohio State University, discussed laboratory vestibular testing. He said that laboratory vestibular tests provide an independent assessment of the peripheral vestibular system, the vestibular nerve, and central vestibular pathways. They detect lesions, differentiate between peripheral and central lesions, and localize peripheral lesions or further localize central lesions. They also help with devising treatment plans, monitoring the progress of treatment, and planning for acoustic neuroma, vestibular ablation, and cochlear implantation surgeries.

Dr. Barin described five commonly used laboratory vestibular tests:

1. ENG/VNG is a battery of eye movement tests. For decades, we have performed ENG (electrooculography), in which eye movements are monitored with electrodes placed on the skin around the eyes. In recent years, ENG has been largely supplanted by VNG (videonystagmography), in which eye movements are monitored by infrared video cameras mounted inside lightproof goggles. The standard ENG/VNG test battery consists of:
   a. four oculomotor tests (accadde test, tracking test, optokinetic test, and gaze test with fixation), which detect CNS lesions,
   b. two vestibular tests (static positional test and gaze test without fixation), which detect lesions of the peripheral or central vestibular system,
   c. two tests (Dix-Hallpike maneuver and pressure test), which identify specific etiologies,
   d. the caloric test, which detects and lateralizes lesions of the horizontal semicircular canal or its afferent pathways.

ENG/VNG has more clinical value than any other laboratory vestibular test. Dr. Barin recommends that it be used in the evaluation of all dizzy patients, except that it should be deferred pending treatment outcome in patients with BPPV. ENG/VNG detects one or more abnormalities in about 50% of dizzy patients and about 75% of these abnormalities specify the site of lesion. Other laboratory tests detect few of these abnormalities. A skilled clinician can detect most of them during physical examination, although physical examination does not permit quantitative analysis or yield a permanent record.

2. Rotary chair testing consists of recording horizontal eye movements as the patient is rotated about a vertical axis with the horizontal semicircular canals in the plane of rotation. The patient is usually tested under three conditions: (a) in complete darkness, (b) while viewing an earth-fixed visual surround, and (c) while viewing a head-fixed visual target. Under each of these conditions, the patient undergoes a series of sinusoidal oscillations at frequencies from about 0.01 Hz to about 1 Hz. Phase, gain, and symmetry of eye velocity relative head velocity is computed for each test frequency.

Rotary chair testing has only fair clinical value. It is useful in documenting bilateral vestibular loss, although the ice water caloric test, the active head rotation test, and the head thrust test also detect this condition. Otherwise rotary chair abnormalities are nonlocalizing.

3. Active head rotation consists of asking the patient to shake his or her head in the horizontal and vertical planes at frequencies from about 0.5 Hz to about 6 Hz while viewing an earth fixed visual target. Head movement is monitored by a head-mounted velocity sensor and eye movement is monitored by electrodes. Phase, gain, and symmetry of eye velocity relative head velocity are computed at each test frequency.

Active head rotation has about the same clinical usefulness as rotary chair testing. It costs less and tests both horizontal and vertical vestibulo-ocular responses at higher frequencies, although head and eye movements at high frequencies are difficult to measure accurately.
4. **Computerized dynamic posturography (CDP)** is comprised of two test batteries. The first is the Sensory Organization Test (SOT), in which the patient’s postural stability is measured as visual and somatosensory cues are manipulated. The second is the Movement Coordination Test (MCT), in which the patient’s postural stability is measured as the supporting surface is tilted or translated.

The diagnostic value of CDP is limited. Malingering patients tend to show an aphysiologic pattern of responses, so the test is sometimes used in medical-legal cases. Some physical therapists use it to design vestibular rehabilitation therapy and monitor its progress.

5. **Vestibular-evoked myogenic potentials (VEMP)** is a new vestibular test. It has been shown that VEMP identifies the symptomatic ear in patients with treatment. If treatment is successful and the patient has no other unexplained signs or symptoms, the patient receives no further laboratory vestibular testing.

Animal studies indicate that they arise from the saccule.

**Labyrinthitis/vestibular neuritis** is often accompanied or preceded by an upper respiratory infection. It is characterized by vertigo lasting for days, nystagmus beating away from the affected ear, and nausea and vomiting. Cochlear symptoms are present with labyrinthitis and absent with vestibular neuritis; otherwise, signs and symptoms are identical. **Dr. Parnes** treats this disorder symptomatically with antiemetics and vestibular sedatives. He treats with oral steroids if he sees the patient within 72 hours after onset of acute vertigo.

There is no evidence that anti-virals are efficacious.

**Recurrent vestibulopathy** is characterized by Meniere’s-like spells of vertigo. There are no cochlear or other localizing symptoms and no diagnostic tests that specify this disorder. A few patients with recurrent vestibulopathy go on to develop typical Meniere’s disease. Treatment is symptomatic, and symptoms resolve spontaneously over 2-3 years in most patients.

**Dehiscent superior semicircular canal syndrome (DSSCS)** is characterized by vertigo and oscillopsia in response to loud sounds (the Tullio phenomenon) or maneuvers that change middle ear or intracranial pressure. Eye movements evoked by these stimuli align with the plane of the dehiscent superior canal.

The patient may also have pulsatile tinnitus, sensitivity to body sounds, and hearing loss. Treatments are avoidance of symptom-inducing situations, middle fossa resurfacing of the affected canal, or middle fossa or transmastoid occlusion of the affected canal.

**BPPV** is usually caused by free-floating particles in the endolymph of the posterior semicircular canal (posterior canalithiasis). **Dr. Parnes** performs the Dix-Hallpike maneuver to identify the affected canal and then administers canalith repositioning treatment, as shown in Fig. 7. His success rate after a single treatment is 80%. If the patient still has BPPV at the next visit, he repeats the treatment. His success rate after three treatments is 95%.

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Meniere's disease is characterized by multiple attacks of vertigo (each lasting more than 20 minutes), unilaterally fluctuating sensorineural hearing loss, and ipsilateral tinnitus or aural fullness (or both). When one or more of these criteria is missing, the diagnosis is called "probable" or "possible" Meniere's disease.

Dr. Parnes administers the following medical treatments for Meniere's disease: low salt diet, avoidance of caffeine, nicotine, and stress, diuretics, benzodiazepines, antihistamines, histamine (betahistine), vasodilator, and corticosteroids.

If medical treatment fails, he treats with intratympanic gentamicin titration. He injects 1 ml of 40 mg/ml stock IV gentamicin solution through a myringotomy once a week. Treatments are discontinued if the audiogram shows a significant hearing drop for two successive weeks, if a new onset of persistent dizziness or imbalance occurs, if a new onset of spontaneous or head-shake nystagmus occurs, or when four treatments have been given. This treatment yields excellent control of vertigo and a low incidence of hearing loss (and no significant hearing drop for two successive weeks, if a new onset of persistent vertigo occurs).

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Dr. Solomon then discussed specific diseases, as follows:

1. 

Vertebrobasilar insufficiency

- Occurs in cases of severe hearing loss) and does not preclude further treatment if it fails.
- Yields excellent control of vertigo and a low incidence of hearing loss (and no significant hearing drop for two successive weeks, if a new onset of persistent vertigo occurs).

- He injects 1 ml of 40 mg/ml stock IV gentamicin solution through a myringotomy once a week. Treatments are discontinued if the audiogram shows a significant hearing drop for two successive weeks, if a new onset of persistent vertigo occurs, or when four treatments have been given. This treatment yields excellent control of vertigo and a low incidence of hearing loss (and no significant hearing drop for two successive weeks, if a new onset of persistent vertigo occurs).

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2. Lateral medullary syndrome (or Wallenberg’s syndrome) is caused by occlusion of the posterior inferior cerebellar artery (PICA). This artery supplies the lateral medullary plate and portions of the posterior medial cerebellum. Occlusion of the PICA in its origin causes the full-blown syndrome—vertigo, spontaneous nystagmus, skew deviation, altered subjective vertigo, dysequilibrium, and spontaneous nystagmus.

3. Pontine syndrome is caused by occlusion of the anterior inferior cerebellar artery (AICA). This artery supplies the lateral pons and part of the middle cerebellar peduncle. It also gives off the labyrinthine artery, which provides exclusive blood supply to the inner ear, as shown in Fig. 8. Occlusion of the AICA causes vertigo, nystagmus, ipsilateral tinnitus, ipsilateral hearing loss, ipsilateral gait and limb ataxia, ipsilateral Horner’s syndrome, ipsilateral vocal cord paresis, ipsilateral gaze paresis, gaze palsy, and contralateral body pain and temperature sensory loss. Occlusion of distal branches of PICA can produce a syndrome that mimics a labyrinthine disorder—vertigo, dysequilibrium, and spontaneous nystagmus.

Fig. 8. Labyrinthine arterial supply

- Loss, ipsilateral gait and limb ataxia, ipsilateral facial hemianesthesia, ipsilateral facial paralysis, ipsilateral Horner’s syndrome, and contralateral hemibody sensory loss.

4. Cerebellar infarction sometimes occurs without brainstem involvement. Since brainstem signs are absent, a mistaken diagnosis of labyrinthine pathology might be made. Key differentiating findings are normal responses on the head thrust test combined with gaze-evoked or vertical nystagmus and/or ataxia. A cerebellar infarction may affect only the inferior and medial cerebellum, causing nystagmus without ataxia, or it may affect only the cerebellar hemispheres, causing ataxia without nystagmus.
5. **Migraine** is present in about 11 million Americans, with 18% of females and 6% of males affected. The highest prevalence is at 30–45 years of age. It has been shown that migraine is undiagnosed in 41% of females and 29% of males who meet strict diagnostic criteria. Most cases of “sinus” headache are migraine. Some patients with migraine also have dizziness, either true vertigo or imbalance and motion sensitivity. Dizziness may occur before or during the headaches or it may occur independently. Dizziness is often accompanied by photophobia, phonophobia, or visual or other aurae. Acute attacks usually last for minutes to hours, seldom longer than 24 hours. Migraine may be indistinguishable from Meniere’s disease, except that accompanying hearing loss is uncommon. Treatment is both behavioral and pharmacological. Behavioral treatment includes regular sleep patterns, stress reduction, migraine diet (avoiding chocolate, cheese, red wine), and eliminating caffeine and habitual analgesic use. Pharmacological treatment to abort attacks includes combinations of caffeine, aspirin, acetaminophen and butalbital or a non-steroidal anti-inflammatory (such as ibuprofen or naproxyn sodium). Prophylactic treatments include beta blockers (propranolol), tricyclic antidepressants (nortriptyline), calcium channel blockers, and valproic acid. Acetazolamide and other anticonvulsants have also been used.

6. **Spontaneous adult-onset ataxia** can be caused by a variety of disorders, including vitamin deficiency, glutin sensitivity, thyroid disorder, paraneoplastic syndrome, and multiple system atrophy (Shy-Drager syndrome). The diagnostic evaluation includes testing for thyroid function, B12, magnesium and vitamin E levels, antilglin and antiendymium antibody, Hashimoto’s antibodies-thyroglobulin and thyroid peroxidase antibodies, antineuronal antibodies, trimnucleotide repeat for autosomal dominant spinocerebellar ataxia, anti-GAD antibodies, and TATA-binding protein. In many cases, no cause is found and even if a cause is found, no effective treatment exists.

7. **Multiple sclerosis** typically begins between 20-40 years of age. It usually presents with optic neuritis, but presents with vertigo in 5% of patients. Vertigo is a symptom sometime during the course of the disease in about 50% of patients. Bilateral internuclear ophthalmoplegia is the hallmark of multiple sclerosis, but various types of central nystagmus may also be seen. An attack of multiple sclerosis may mimic a peripheral vestibular lesion with a unilateral caloric weakness. An IV pulse of high-dose steroids may shorten an attack. Acquired pendular nystagmus may respond to gabapentin or baclofen. Infratentorial ependymomas arise from the lining of the fourth ventricle. Protracted nausea and vomiting are often present, and the classical headache is positional, with pain present while supine and relieved by sitting up. Brainstem gliomas may occur before or during the headaches or it may occur independently. Dizziness is often accompanied by photophobia, phonophobia, or visual or other aurae. Acute attacks usually last for minutes to hours, seldom longer than 24 hours. Migraine may be indistinguishable from Meniere’s disease, except that accompanying hearing loss is uncommon. Treatment is both behavioral and pharmacological. Behavioral treatment includes regular sleep patterns, stress reduction, migraine diet (avoiding chocolate, cheese, red wine), and eliminating caffeine and habitual analgesic use. Pharmacological treatment to abort attacks includes combinations of caffeine, aspirin, acetaminophen and butalbital or a non-steroidal anti-inflammatory (such as ibuprofen or naproxyn sodium). Prophylactic treatments include beta blockers (propranolol), tricyclic antidepressants (nortriptyline), calcium channel blockers, and valproic acid. Acetazolamide and other anticonvulsants have also been used.

8. **Arnold Chiari malformation** (Type 1) is characterized by unexplained sensorineural hearing loss, headache, vertigo, ataxia, dysphagia, dysphoria or other lower cranial nerve dysfunction. Gaze-evoked nystagmus, downbeat nystagmus, and defective pursuit are typical ocular motor findings. Treatment is suboccipital decompression of the foramen magnum.

9. **Neoplastic disease** can cause dizziness. Infratentorial ependymomas arise from the lining of the fourth ventricle. Protracted nausea and vomiting are often present, and the classical headache is positional, with pain present while supine and relieved by sitting up. Brainstem gliomas may occur at any age, but are most common in children. Cerebellar signs, trigeminal and lower cranial nerve involvement occurs. In children, medulloblastoma may cause non-fatiguing, paroxysmal positional nystagmus, which is usually purely vertical and accompanied by vertigo and generalized dyscoy autonomy. Vestibular schwannomas (or acoustic neuromas) account for 85-90% of all schwannomas. Presentation of vestibular schwannomas is usually insidious, with unilateral progressive hearing loss and vestibular paresthesias (with- out vertigo). Tinnitus, headache, mastoid pain, facial weakness or otalgia may be present. Paraneoplastic disease occurs when an immune response is triggered by a tumor that is usually remote from the nervous system. Anti-Yo antibodies cause a loss of Purkinje cells in the cerebellum, resulting in a syndrome of ataxia, dysarthria, and nystagmus. This may be the presenting picture, and when antibodies are detected, a search for the tumor must then begin.

10. **Wernicke’s encephalopathy** is caused by thiamine deficiency and can be brought on by poor nutrition, prolonged vomiting, alcoholism, eating disorders, or chemotherapy. Signs include vertical nystagmus, gaze-evoked nystagmus, and bilateral abducens palsies. Ataxia and mental changes are usually present as well. Signs may reverse within hours of thiamine administration.

11. **Normal pressure hydrocephalus** is characterized by dementia, incontinence, and gait disorder. MRI shows enlarged ventricles out of proportion to atrophy. Ventriculography is not helpful. Response to prolonged CSF drainage is the best predictor of improvement with a shunting procedure.

12. **Epileptic vertigo** is rare. It is characterized by episodes of vertigo lasting minutes, sometimes with associated cranial nystagmus, dysphasia, amnesia, disorientation, and visual field abnormalities.

Finally, I presented management of psychological and psychiatric aspects of dizziness. More than one-third of patients with vestibular dysfunction have anxiety symptoms. These symptoms cause decreased functioning, decreased quality of life, and prolonged recovery from vestibular disorders.

I disagree with the traditional criteria for psychogenic dizziness—vague description of symptoms, exacerbation of symptoms in certain environments, reproduction of symptoms by hyperventilation, and normal physical exam. While dizziness can be a symptom of a psychiatric disorder, dizziness without other psychiatric symptoms is insufficient for the diagnosis of psychiatric disease. Panic disorder may cause lightheadedness, but also causes palpitations and shortness of breath. Panic, general anxiety, acute stress, and post-traumatic stress disorders may cause an “unreal” feeling, but also cause anxiety, numbness, and flashbacks. Depression may cause a vague “swimming” feeling, but also causes poor appetite and insomnia. Conversion disorder may cause imbalance, but also causes tremors and other nonphysiologic behaviors.

Vestibular disorders commonly induce symptoms of anxiety or panic, especially in patients with vulnerable temperaments. Anxiety is part of the response to vestibular dysfunction, just as heart palpitations are part of the response to physical exercise. Concerns about future attacks of vertigo, possible embarrassment, serious medical illness, mental illness, and disability further increase the patient’s anxiety. There is a subset of patients with both panic disorder and vestibular dysfunction. These patients have vestibular symptoms between panic attacks, agoraphobia or height phobia, and discomfort in malls or supermarkets. Patients with chronic dizziness may also have symptoms of depression—trouble concentrating, poor sleep, fatigue, and social withdrawal.

Dismissive behavior by the clinician adversely affects the outcome of treatment. Such behavior includes: (a) failing to acknowledge that there is a problem, (b) minimizing the seriousness of the problem, (c) suggesting that the problem is “mental,” and (d) spending too little time with the patient. Dismissive behavior makes the patient anxious and angry. The opposite of dismissive behavior is validating behavior. Such behavior includes: (a) evaluating...
both vestibular and psychiatric symptoms, (b) assessing temperament, (c) avoiding suggestion of psychogenicity, (d) explaining vestibular mechanisms, (e) explaining somatopsychic mechanisms, and (f) identifying sources of secondary anxiety and providing corrective information. Validating behavior usually means spending more time with the patient.

Useful treatments are vestibular rehabilitation therapy and medication. (Clonazepam 0.5 mg PO BID is drug of choice). If evaluation reveals a true psychiatric disorder, the patient should be referred to a psychiatric professional who is knowledgeable about vestibular disorders. Such a referral should be made after counseling the patient and should not be made on the first visit.

After hearing these presentations, we broke up into small groups for practical demonstrations of diagnostic and treatment techniques. Dr. Solomon demonstrated the head thrust test; Dr. Parme demonstrated the Dix-Hallpike and canalith repositioning maneuvers; Dr. Barin demonstrated the interpretation of ENG/VNG tracings; and Timothy C. Hain, M.D., Chicago Dizziness and Balance and Associate Professor of Neurology at Northwestern University, demonstrated the interpretation of video eye movement recordings.

Dr. Parnes wrapped up the course with a presentation of difficult cases.

Summary. Most dizzy patients have benign disorders that can be successfully managed by a family physician, but some have serious disorders that require evaluation and management by a specialist. As specialists who see dizzy patients, we must be able to distinguish among many possible diagnoses, including some outside our own areas of specialty. We must be prepared to take comprehensive histories and perform thorough physical examinations, as outlined in this course. Sometimes our evaluations yield definite diagnoses, but more often they yield lists of possible diagnoses, and to distinguish among them, we must seek information provided by laboratory testing. We rely primarily upon imaging studies and laboratory analyses of blood and other body fluids to help confirm or refute possible diagnoses. Audiometric and vestibular tests are also useful, especially for determining the functional state of the vestibular system, but rarely crucial for diagnosis. Our goal is to make the correct diagnosis and treat accordingly. However, in a subset of patients we need to treat without a definite diagnosis.

It has been a terrific privilege to organize these conferences and interact with our outstanding faculty. Our attendees have been highly motivated learners and active participants. No one left early; in fact, we had to be asked to leave by the hotel staff as they anxiously readied for an evening wedding. For our faculty, and me, the reward is in seeing and feeling the intense attention, interest, and urge to learn of our participants, all with the goal to better care for their patients with dizziness. I invite you to consider joining us for the 3rd conference planned for June 2006.

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Interpretation of Static Position Testing in VNG/ENG

Kamran Barin, Ph.D.

Biography
Kamran Barin, Ph.D. is the Director of Balance Disorders Clinic at the Ohio State University Medical Center and Assistant Professor, Department of Otolaryngology - Head and Neck Surgery, Department of Speech and Hearing Science, and Biomedical Engineering Program.

Dr. Barin has taught national and international courses and seminars in different areas of vestibular assessment and rehabilitation. He serves as a consultant for Otometrics.

Abstract:
The purpose of static position testing, also known as the positional test, in the video- and electro-nystagmography (VNG/ENG) test battery is to determine the presence and characteristic of nystagmus when the patient’s head is placed in different orientations with respect to gravity. Although the test procedure is relatively simple (see Barin, 2006, for details), the interpretation of the results may not be straightforward to inexperienced VNG/ENG examiners. This article provides a basic step-by-step algorithm for the interpretation of static position testing.

The flow chart in Figure 1 is a summary of the interpretation process. Additional information is provided below for the numbered items on the flow chart.

1. Sometimes moving the head from one position to another provokes a transient nystagmus, either immediately or after a short delay. As the purpose of the static position test is to detect the steady-state nystagmus that is present as long as the head remains in the critical head position, transient nystagmus should not be included in the interpretation of this test. Instead, the examiner should perform the appropriate maneuver and interpret the results as a part of the dynamic position testing.

2. Traditionally, static position testing has been limited to the interpretation of horizontal eye movements. The main reason for this is that the vertical channel in ENG is often noisy and contaminated by eye blinks. In VNG, the differences in the noise level and resolution between horizontal and vertical channels are relatively insignificant. Therefore, VNG users should consider vertical nystagmus in the interpretation of the static position test. ENG users may wish to skip this step because of the high level of artifacts in the vertical channel. The flow charts in Figures 2 and 3 show the interpretation for horizontal and vertical nystagmus, respectively.

When nystagmus has both horizontal and vertical components, the examiner must make sure that it represents true eye movements and is not due to crosstalk. Crosstalk occurs when eye movements in one channel generate activities in the other channel. Those activities do not represent true eye movements and are usually caused by the misalignment of the electrodes in ENG or...
Figure 1. Interpretation Summary for the Static Position Test.

1. Transient nystagmus?
   - Yes: Do not include in the interpretation of static position testing. Perform appropriate maneuvers and interpret the results as a part of the dynamic position test.
   - No: Interpretation of horizontal nystagmus

2. Interpretation of vertical nystagmus

3. Report and describe clinical significance of any abnormal nystagmus
4. Report presence of any normal nystagmus
5. If applicable, report the effect of neck rotation
6. If applicable, report presence of transient nystagmus

End
the misalignment of cameras or goggles in VNG. Crosstalk can be recognized when the patient is asked to make purely horizontal or vertical eye movements. Any activities in the other channel represent crosstalk. The examiner should either repeat the test or subtract the effect of the crosstalk from the tracings.

3 Static position testing should include examining the eye movements in at least four different positions: sitting, supine, head right, and head left. If fewer head positions are tested, a partial interpretation is still possible according to the guidelines of the flow chart. The examiner should be aware that the position testing for some head positions may be embedded in other parts of VNG/ENG. For example, the results from the spontaneous nystagmus test or the gaze test may be used in place of the position test in the sitting position as long as the fixation conditions are taken into account. Sometimes it is necessary to include other head positions such as body right, body left, or head hanging. Some laboratories routinely include those and other head positions. The interpretation algorithm is also applicable to those cases.

4 The static position test is typically performed in the absence of fixation. However, the effect of fixation is the single most useful factor in differentiating nystagmus that is generated by central lesions from other types of nystagmus. Therefore, some laboratories routinely test the patient with and without fixation. The algorithm allows for this possibility. However, even when position testing with fixation is not available, the effect of fixation for the sitting position can be determined from the gaze test or the spontaneous nystagmus test.

5 Horizontal nystagmus with fixation is always abnormal. No other criterion, such as nystagmus intensity, is needed and the simple presence of nystagmus with fixation is enough. To localize the lesion, the nystagmus with fixation should be compared to the nystagmus without fixation. If the nystagmus intensity does not increase significantly (at least double) when the fixation is eliminated, the results are consistent with a central lesion. If the nystagmus intensity increases significantly without fixation, the nystagmus with fixation should be considered “leak through” of the strong nystagmus without fixation. Therefore, the interpretation should follow the same path as that of nystagmus without fixation.

6 Horizontal nystagmus that changes direction in a single head position is always abnormal. This type of nystagmus, called periodic alternating nystagmus, is usually present both with and without fixation and changes direction about every 2-4 minutes. This finding is consistent with a central lesion. Detection of periodic alternating nystagmus is hampered by the fact that typical recording of eye movements in each head position is much shorter than 2-4 minutes. Instead, the examiner must look for inconsistencies in the direction of nystagmus for parts of the VNG/ENG test battery that provide equivalent test conditions. For example, the nystagmus direction in the gaze or spontaneous nystagmus test without fixation is expected to be the same as the nystagmus direction in the sitting position. When such inconsistencies are observed, the duration of the recording for the suspected head position should be extended to determine if the nystagmus changes direction.

7 Horizontal nystagmus without fixation does not always indicate an abnormality. A number of studies have shown that some form of nystagmus without fixation is present in many individuals without a history of dizziness or other balance disorders (Barber and Wright, 1973). The most important characteristic that differentiates normal from abnormal nystagmus without fixation is the nystagmus intensity. Other criteria, such as the number of head positions in which the nystagmus is persistent or intermittent, are outdated and should not be used. The presence and intermittency of nystagmus are related to technical issues such as the patient’s level of alertness or gaze direction. Therefore, it does not seem logical to include those factors in identifying abnormal nystagmus without fixation. To avoid such technical issues and to obtain a valid static position test, it is important for the
Figure 2. Interpretation of Horizontal Nystagmus.
examiner to maintain a steady level of patient alertness throughout the test.

Nystagmus intensity is defined as the velocity of the nystagmus slow-phase. Traditionally, a threshold of 6°/sec has been used for pathologic nystagmus without fixation (Barber and Stockwell, 1980). This limit has been derived based on ENG testing and there is a question whether the same limit should apply to VNG (Hain, 2008). In a yet unpublished study of 40 normal individuals in our laboratory, we found the normal limit using VNG to be about 4°/sec. However, until this finding is confirmed by more large-scale studies of VNG findings in the normal population, we continue to use the normal limit of 6°/sec for both ENG and VNG.

Abnormal horizontal nystagmus without fixation does not provide localizing information. It can be caused by lesions in the peripheral as well as central vestibular pathways.

Horizontal nystagmus without fixation can be classified based on its direction and intensity in different head positions. For example, nystagmus that has the same direction and intensity in different head positions is usually classified as spontaneous nystagmus whereas nystagmus that has the same direction but different intensity in different head positions is classified as positional nystagmus. At times, nystagmus can beat in different directions in the head right and head left positions. When the nystagmus beats toward the ground (right-beating in head right and left-beating in head left), it is classified as geotropic nystagmus. When the nystagmus beats away from the ground (left-beating in head right and right-beating in head left), it is classified as ageotropic or apogeotropic nystagmus.

Over the years, different classes of nystagmus without fixation have been associated with lesions in the peripheral or central vestibular pathways. For example, ageotropic nystagmus has been considered a central finding by some laboratories. However, there are many counter-examples to such assumptions. For example, positional alcohol nystagmus, which is caused by the change of the cupula density with respect to the endolymph density within the peripheral vestibular system, can result in either geotropic or ageotropic nystagmus depending on the elapsed time since the ingestion of alcohol.

As a result, abnormal horizontal nystagmus without fixation, including all of its variations of spontaneous, positional, geotropic, and ageotropic, is a non-localizing finding that can originate from the peripheral vestibular system in either ear or central vestibular pathways.

The overall interpretation of static position testing for horizontal nystagmus is based on the combination of findings for each head position. For example, the overall interpretation indicates a central lesion if a central finding is identified for any head position. In the absence of a central finding, all other abnormalities indicate a non-localizing finding.

When horizontal nystagmus is not present in the sitting or supine positions but appears in the head right or head left position, the effect of neck rotation should be examined by testing the patient in the body right or body left positions. If the nystagmus disappears in the body right or body left position, it should be attributed to the neck rotation. Otherwise, neck rotation has no effect.

Vertical nystagmus with fixation is always abnormal (Baloh and Honrubia, 1990). No other criterion is needed and the simple presence of nystagmus with fixation is enough. This type of nystagmus, either down-beating or up-beating, is consistent with a central lesion.

Vertical nystagmus without fixation has been reported in both healthy individuals with no prior history of dizziness or balance disorders as well as in patients with various abnormalities (Barber and Wright, 1973; Kim et al, 2000). Currently, there are no established normal limits for vertical nystagmus without fixation. In the previously mentioned study, we found vertical nystagmus without fixation to be common in our sample of 40 normal individuals. The 95% confidence limit of this sample was about 7°/sec. This limit was established using VNG but we currently use the normal limit of 7°/sec for vertical
Figure 3. Interpretation of Vertical Nystagmus.
nystagmus without fixation for both ENG and VNG.

Even when vertical nystagmus without fixation is abnormal, currently there is not enough information to determine its localization and clinical significance. In our laboratory, we report presence of abnormal vertical nystagmus without fixation and state that its clinical significance is unknown at this time.

The overall interpretation of static position testing for vertical nystagmus is based on the combination of findings for each head position. For example, the overall interpretation indicates a central lesion if a central finding is identified for any head position. In the absence of a central finding, the localization and clinical significance of all other abnormal findings for vertical nystagmus are unknown.

The report for static position testing should include descriptions of all types of abnormal horizontal and vertical nystagmus and their clinical significance. The report should also include a description of any nystagmus that is present even when it is within normal limits. When applicable, the report should describe the effect of neck rotation on the nystagmus that is absent in the sitting and supine positions but appears in the head right or head left position. Finally, the report should include a description of any transient nystagmus that is provoked as a result of moving from one position to another.

References

Technical note
ICS Chart 200 software 6.2 and higher implement a version of the above algorithm in the Interpretation Assistant. To use the algorithm, the user must record the eye movements during tests that are specifically identified as “w/ vision” and “w/o vision”.

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Baseline Shift and Gain Asymmetry in the Caloric Test

Kamran Barin, Ph.D.

Introduction

In the standard bithermal caloric test, right warm and left cool irrigations are expected to generate right-beating nystagmus while left warm and right cool irrigations are expected to generate left-beating nystagmus. In a normal individual, the intensities of all four caloric responses are approximately the same and therefore, there is no significant difference between right-beating and left-beating responses. Some patients however, have directional preponderance (DP) in which responses in one direction are significantly greater than the responses in the opposite direction. DP is defined as the normalized (scaled) difference between the peak nystagmus slow-phase velocities (SPVs) from irrigations that are expected to generate right-beating nystagmus and those from irrigations that are expected to generate left-beating nystagmus. Mathematical formulas for calculating DP and other caloric parameters are provided in the Appendix.

Interpretation of DP

The normal limits reported for DP from different studies have ranged from as low as 20% to as high as 50%. Currently, most laboratories consider DP of less than 30% to be within normal limits (Sills et al., 1977).

There has been a controversy about the interpretation and clinical value of abnormal DP. Initially, abnormal DP was considered a central finding but this conclusion was reached based on caloric responses that were obtained in the presence of fixation (Fitzgerald and Hallpike, 1942). Therefore, what was considered...
to be abnormal DP was actually related to asymmetric failure of fixation suppression. Subsequent studies in which the caloric test was performed in the absence of fixation found DP in both peripheral and central pathologies (Coats, 1966; Baloh et al., 1977). Therefore, abnormal DP in its current form is considered a non-localizing finding. Abnormal DP has also been reported in normal individuals, which further casts doubt on its clinical value (Coats, 1965).

Due to its low sensitivity to pathologies and lack of specificity to central versus peripheral abnormalities, some laboratories do not include DP in the interpretation of the caloric test. However, it has now become clear that DP can be caused by two different types of abnormalities. Therefore, the low clinical value of DP should not come as a surprise because the current method of calculating DP does not distinguish between these two abnormalities. It seems worthwhile to define new parameters that can separately quantify these abnormalities.

Different types of DP

Figure 1 shows two different types of DP. In Figure 1A, caloric responses are shifted in one direction indicating presence of nystagmus at the beginning of all four irrigations. The caloric stimulus in an ear with an intact tympanic membrane does not reach the labyrinth for at least 10 seconds from the onset of the irrigation. Therefore, this baseline shift represents a pre-existing nystagmus in the standard caloric position. That is, this patient has some form of spontaneous nystagmus. This nystagmus is added to the caloric-induced nystagmus when they are in the same direction and subtracted from it when they are in opposite directions. As a result, significant DP is generated because the peak caloric responses for two irrigations (right cool and left warm, in this case) are greater than those for the other two irrigations (right warm and left cool, in this case). This type of DP is called Bias or Baseline Shift (BS).

Figure 1. Different types of DP: A) BS, B) GA. For clarity of presentation, simulated caloric responses are used instead of actual patient test results.
**Figure 1B** shows a different type of DP in which the caloric responses in one direction are truly stronger than the responses in the opposite direction. There is no spontaneous nystagmus as the SPVs at the onset of all four caloric irrigations are zero. This type of DP has been described in the literature but it is an extremely rare finding (Sills et al., 1977). Halmagyi et al. (2000) found this type of DP in less than 1% of patients who underwent vestibular testing whereas BS constituted the remaining 99% of the cases with clinically-significant DP. They termed this type of DP, Gain Asymmetry (GA).

**Quantification of BS and GA**

Although the caloric responses in **Figure 1A** and 1B represent two different abnormalities, UW and DP parameters are the same. In order to differentiate between these two cases, new parameters are needed to quantify BS and GA.

The formula for DP can be partitioned into two components with the first component related to spontaneous nystagmus and BS and the second component related to GA. However, the representation of BS in this form has a major shortcoming as the intensity of spontaneous nystagmus is divided by the sum of four caloric responses (Barin and Stockwell, 2002). Because spontaneous nystagmus is independent of the caloric irrigations, it does not seem logical to scale or normalize its intensity based on the caloric responses. The consequence of normalizing the intensity of spontaneous nystagmus is shown in **Figure 2**. The same level of spontaneous nystagmus can generate a wide range of values for DP that can be normal (**Figure 2A**) or abnormal (**Figure 2B**). That is, the caloric test parameters are different despite the fact that underlying abnormality is the same in **Figures 2A** and 2B.

![Figure 2](image)

**Figure 2.** The effect of normalizing the intensity of spontaneous nystagmus or BS on DP: A) Strong caloric responses, B) Weak caloric responses. For clarity of presentation, simulated caloric responses are used instead of actual patient test results.
The most appropriate method for quantifying BS appears to be the SPV of spontaneous nystagmus. This can be accomplished in different ways. First, the intensity of spontaneous nystagmus can be calculated from the supine position in the static position testing because this position is similar to the standard caloric test position (Figure 3B). A better alternative is averaging of the nystagmus SPVs from the first few seconds of each irrigation. This will account for any potential calibration change from the position test to the caloric test. The averaging of SPVs can be done computationally but a graphical approach simplifies the process. It involves finding a best-fitting horizontal line that passes through the SPV points at the beginning of each irrigation (Figure 3A). The intersection of this line with the vertical axis represents BS.

The intensity of spontaneous nystagmus (and by extension, BS) depends on the gaze position and the level of alertness. That is the reason in actual patient testing (Figure 3) the BS levels from different irrigations are approximately the same but they are not exactly the same as in the idealized responses (Figure 1 and 2). Using a best-fitting line or averaging of the SPVs addresses this issue. On the other hand, the direction of spontaneous nystagmus does not change in a single head position. Therefore, any difference in the direction of BS from one irrigation to another usually represents a technical error (such as not waiting long enough between irrigations). In very rare cases, periodic alternating nystagmus, which represents a central abnormality, can cause changing of nystagmus direction in a single head position. If the direction of BS is different in different irrigations and technical errors have been ruled out, the presence of periodic

**Figure 3.** A) Graphical method for estimating BS. The green line represents a best-fitting horizontal line for the SPV points within the first few seconds of each irrigation (dotted black boxes). B) The static position test result for the same patient showing left-beating nystagmus in the supine position with an average SPV of 10 deg/sec.
Alternating nystagmus can be verified by repeating the position test in a single head position and recording the nystagmus for an extended period of time (typically, 5 minutes or longer).

The true asymmetry in the intensity of right-beating versus left-beating nystagmus can be quantified using the same formula for the DP after removing the contribution of the spontaneous nystagmus from each of the peak caloric responses. This is indeed the definition of GA. Note that dividing of the difference between right-beating and left-beating nystagmus intensities by the total caloric responses is appropriate because after removing the contribution of the spontaneous nystagmus, all of the parameters in the formula for GA represent caloric-induced SPVs.

One more note on the somewhat confusing terminology for expressing BS and GA. BS is usually expressed with respect to the direction of stronger slow phases whereas GA is usually expressed with respect to the direction of stronger fast phases. For example, BS in Figure 1A is to the right whereas GA in Figure 1B is to the left.

Interpretation of BS and GA

Because BS and GA are independent parameters, they should be interpreted separately. BS and spontaneous nystagmus represent the same abnormality and therefore, their normative limits and interpretation are the same. Most laboratories use SPVs of less than 4-6 deg/sec as the normal limit for spontaneous nystagmus, which can be used directly as the normal limit for BS.

In many cases, abnormal BS occurs concurrently with abnormal UW, which indicates an acute or uncompensated peripheral vestibular lesion. In the absence of an abnormal UW, abnormal BS and spontaneous nystagmus indicate a non-localizing finding involving peripheral or central vestibular pathways.

The normative values and the interpretation of GA are not well-established because there are very few studies that have examined GA independent of DP. Halmagyi et al (2000) used values of greater than 40% for abnormal GA but again, that limit was based on their normal limits for DP. In our laboratory, we use a somewhat arbitrary limit of less than 25% for normal GA. More studies are needed to establish the normal limits of GA in a more definitive manner.

Abnormal GA is an extremely rare finding. Halmagyi et al (2000) found less than 1% of the patients who underwent vestibular testing demonstrated abnormal GA. Baloh and Honrubia (2001) have suggested that abnormal GA denotes a central lesion. Halmagyi et al (2000) did not address this issue directly but found abnormal GA in a variety of both central and peripheral lesions. In their study of patients with abnormal GA, the majority of those with peripheral vestibular lesions had a diagnosis of either benign paroxysmal positional vertigo or Meniere's disease. In a companion paper, Cartwright et al (2000) suggested that abnormal GA was due to a dynamic asymmetry in the secondary vestibular neurons. They further suggested that such an asymmetry in peripheral vestibular lesions was brought on by a faulty compensation mechanism in response to fluctuations of vestibular function. If we accept this notion, the concept of abnormal GA representing a central lesion is still plausible because both the secondary vestibular neurons and the compensation mechanisms reside within the central vestibular pathways.

However, further studies are needed to determine the value of GA in identifying the site of lesion.

Summary

There are two distinct abnormalities that can cause a significant DP in the caloric test. Because the current method of calculating DP does not distinguish between these two abnormalities, new parameters were defined that can separately quantify these abnormalities. Further studies are needed to determine whether GA and BS are clinically more useful than DP. Nonetheless, identifying the distinct components of DP is a logical step and addresses major shortcomings of DP.
Appendix

The method for quantifying GA is presented here. The conventional formula for DP is:

\[
DP = \frac{\text{TotRB} - \text{TotLB}}{\text{TotRB} + \text{TotLB}} \times 100,
\]

where \( \text{TotRB} \) represents total responses from the irrigations that are expected to generate right-beating nystagmus and \( \text{TotLB} \) represents total responses from the irrigations that are expected to generate left-beating nystagmus. The parameters in the above formula are determined from the peak nystagmus SPVs for right warm (\( \text{PeakRW} \)), left warm (\( \text{PeakLW} \)), right cool (\( \text{PeakRC} \)), and left cool (\( \text{PeakLC} \)) irrigations:

\[
\begin{align*}
\text{TotRB} &= \text{PeakRW} - \text{PeakLC}, \\
\text{TotLB} &= \text{PeakRC} + \text{PeakLW}
\end{align*}
\]

Note that the above formulas are algebraic operations and peak values are signed numbers with positive numbers representing rightward slow-phase or left-beating nystagmus and negative numbers representing leftward slow-phase or right-beating nystagmus (Figure 4). Sometimes caloric irrigations produce nystagmus in the opposite direction of what is expected (usually due to presence of strong spontaneous nystagmus). The above formulas are still applicable as long as the correct signs are used for the peak values of those responses. The common terminology for \( DP \) is to express it with respect to the direction of stronger fast phases.

When there is spontaneous nystagmus, the caloric response is a combination of the caloric-induced nystagmus and the spontaneous nystagmus. That is,

\[
\text{PeakXX} = \text{CalXX} + \text{SN},
\]

where \( \text{Cal} \) is the maximum SPV of caloric-induced nystagmus, \( \text{SN} \) is the average SPV of spontaneous nystagmus and XX stands for \( \text{RW} \) (right warm), \( \text{LW} \) (left warm), \( \text{RC} \) (right cool), and \( \text{LC} \) (left cool) irrigations. If we apply this concept to \( \text{UW} \), the contribution of spontaneous nystagmus completely disappears from the formula for \( \text{UW} \). That is, \( \text{UW} \) is defined as the relative difference of caloric-induced nystagmus.

\[
\text{UW} = \frac{\text{CalRW} - \text{CalLC}}{\text{CalRW} + \text{CalLC}},
\]

\[
\text{UW} = \frac{\text{CalLW} - \text{CalRC}}{\text{CalLW} + \text{CalRC}}.
\]

Figure 4. Signed values of SPV: A) Positive numbers for rightward slow-phase or left-beating nystagmus, B) Negative numbers for leftward slow-phase or right-beating nystagmus.
between the peak caloric responses of the right and left ears and quantified by:

\[
UW = \frac{\text{TotRE} - \text{TotLE}}{\text{TotRE} + \text{TotLE}} \times 100.
\]

\(\text{TotRE}\) represents total responses from the right ear and \(\text{TotLE}\) represents total responses from the left ear:

\[
\text{TotRE} = \text{PeakRC} - \text{PeakRW},
\]

\[
\text{TotLE} = \text{PeakLW} - \text{PeakLC}.
\]

Replacing the peak values:

\[
\text{TotRE} = \text{PeakRC} - \text{PeakRW} = \text{CalRC} + \text{SN} - \text{CalRW} - \text{SN} = \text{CalRC} - \text{CalRW},
\]

\[
\text{TotLE} = \text{PeakLW} - \text{PeakLC} = \text{CalLW} + \text{SN} - \text{CalLC} - \text{SN} = \text{CalLW} - \text{CalLC}.
\]

Therefore, \(UW\) is appropriately based on the caloric-induced nystagmus alone without any contamination by spontaneous nystagmus:

\[
UW = \frac{(\text{CalRC} - \text{CalRW}) - (\text{CalLW} - \text{CalLC})}{(\text{CalRC} - \text{CalRW}) + (\text{CalLW} - \text{CalLC})} \times 100.
\]

In fact, the rationale for performing bithermal caloric testing is to cancel out the effect of spontaneous nystagmus in calculating \(UW\). In the above formula, the difference between the responses of right and left ears is divided by the total of all four caloric responses to scale or normalize \(UW\). This is logical in view of the fact that there is considerable variability among the individual caloric responses from one person to another.

Replacing the peak values in the formula for \(DP\) yields:

\[
\text{TotRB} = -\text{PeakRW} - \text{PeakLC} = -\text{CalRW} - \text{CalLC} - 2 \times \text{SN},
\]

\[
\text{TotLB} = \text{PeakRC} + \text{PeakLW} = \text{CalRC} + \text{CalLW} + 2 \times \text{SN},
\]

\[
\begin{align*}
DP &= \frac{-4 \times \text{SN}}{(-\text{CalRW} - \text{CalLC}) + (\text{CalRC} + \text{CalLW})} + \\
&= \frac{(-\text{CalRW} - \text{CalLC} - (\text{CalRC} + \text{CalLW}))}{(-\text{CalRW} - \text{CalLC}) + (\text{CalRC} + \text{CalLW})} \times 100.
\end{align*}
\]

The first component in the \(DP\) formula is related to spontaneous nystagmus and was discussed earlier. The second component represents the true asymmetry in the intensity of right-beating versus left-beating nystagmus after removing the contribution of the spontaneous nystagmus. Therefore, the appropriate quantification for \(GA\) is:

\[
\begin{align*}
\text{GA} &= \frac{(-\text{CalRW} - \text{CalLC}) - (\text{CalRC} + \text{CalLW})}{(-\text{CalRW} - \text{CalLC}) + (\text{CalRC} + \text{CalLW})} \times 100.
\end{align*}
\]

where \(\text{CalXX}\) can be calculated by subtracting the BS from the corresponding \(\text{PeakXX}\). Note that dividing of the difference between right-beating and left-beating nystagmus intensities by the total caloric responses is appropriate because all of the parameters in the above formula represent caloric-induced SPVs.
References


Technical Note

ICS Chartr software versions 6.0 and higher are capable of calculating BS and GA.