Vestibular Asymmetry

Some Theoretical and Practical Considerations

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• **Pathological vestibular asymmetry can be divided into static and dynamic types. *Static asymmetry* results from a unilateral change of the resting neural input. Acute, chronic, and recovery stages can be recognized if one interprets the direction and intensity of the resultant spontaneous nystagmus relative to the clinical picture. Static asymmetry is additive with induced asymmetry and manifests itself as directional preponderance or as the direction-fixed or direction-changing feature of positional nystagmus. *Dynamic asymmetry* refers to abnormal asymmetry induced by normal head movements. For example, with unilateral hypofunction, a greater gain is observed with head movement toward the unaffected side, suggesting nonlinearity as specified by Ewald's second law. *Visually induced vestibular asymmetry* is a form of dynamic asymmetry generated by convergence of visual-vestibular information, and causing symptoms in certain "motion-active" visual environments.**

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The vestibular system is a sensory system whose function is to monitor how the head moves or is positioned relative to inertial space. Under normal circumstances, the semicircular canals have been assigned the task of detecting angular head motion. With respect to normal system function, it is well known that the recognition of such angular motion depends on the generation of asymmetry at the level of the peripheral sensors.

Asymmetry can also be generated in the peripheral vestibular system (ie, at the hair cell or first-order neuron level) as a result of various pathological states. Pathological asymmetry, with respect to semicircular canal input, can be interpreted erroneously by the CNS as a state of angular motion. On the response side, such asymmetry is manifest by symptoms and signs such as vertigo, visual and postural instability with head movement, and spontaneous nystagmus.

In the clinical evaluation of vestibular disorders, one makes an assessment and arrives at a diagnosis partly on the basis of the type and pattern of these signs and symptoms. In order to understand the basis for the symptomatology, it is useful to think in terms of the source of the vestibular asymmetry, since similar clinical pictures may be present for quite different pathophysiological reasons. This report will attempt to examine some interesting aspects of pathological asymmetry in the peripheral vestibular system, both from a theoretical and a practical point of view.

For convenience of discussion, pathological vestibular asymmetry can be divided into static and dynamic types. A third category of asymmetry, which one might wish to include as a variant of the dynamic type, is that which originates on the basis of peripheral visual inputs.

STATIC PATHOLOGICAL VESTIBULAR ASYMMETRY

Static pathological vestibular asymmetry occurs as a result of a unilateral peripheral lesion. This asymmetry exists in the absence of head movement and is due to the fact that the resting input on the affected side is altered. The objective indicator of static asymmetry is spontaneous nystagmus. It has been our practice to define *spontaneous nystagmus* as any nystagmus observed with the patient in a neutral position (ie, sitting, supine, caloric test position) and with no externally applied stimulation. This is similar to the definitions given by Coats,[1] Baloh and Honrubia,[2] and Kumar.[3]

From a clinical point of view, the symptomatology associated with static asymmetry can be observed in different stages depending on the chronicity of the asymmetry (a factor that influences the degree of adaptive

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change) and the occurrence of any ongoing change of the asymmetry. Change can be in the form of an increasing asymmetry (such as occurs in the early stages of an acute lesion or with a slowly progressive lesion such as acoustic neuroma) or in the form of a decreasing asymmetry when recovery of function occurs on the affected side. For practical purposes, one can recognize three stages.

Acute

It is well known that with the unilateral loss of vestibular function, notable symptoms develop in the patient, including vertigo and a horizontal component of spontaneous nystagmus that beats with fast phase toward the unaffected side. Such a nystagmus is persistent, at least in the initial acute stage, and is due to a decrease in the resting input on the affected side. Spontaneous nystagmus beating toward the affected side has been reported early in acute lesions,[4,5] supposedly due to an irritative effect that generates an increase in resting discharge. However, in the course of monitoring spontaneous nystagmus during acute Meniere's attacks, McClure et al[6] have never observed this so-called early irritative effect.

Chronic

It is also well known that when a unilateral loss of function is permanent, then over a period of time, the signs and symptoms associated with the acute stage of the disorder gradually decline. This is due to the fact that the CNS adapts to the asymmetrical resting input. Precht et al[7] studied the phenomenon of adaptation in hemilabyrinthectomized cats and attributed it to a restoration of resting activity at the level of the second-order neuron on the affected side.

It has been our experience that some patients with a permanent unilateral loss of vestibular function continue to show a persistent low-intensity spontaneous nystagmus that can beat in either direction irrespective of the direction of the asymmetry as defined by caloric tests. We reviewed the results for a five-year period from patients who had a persistent (defined as four months or longer) unilateral asymmetry on repeated caloric testing and whom we classified as idiopathic because there was no definite cause for the asymmetry based on historical and other evidence. In this group, there were 28 patients who had had repeated tests separated by four to 28 months (mean, 9.5 months). These tests included caloric tests and examination for spontaneous nystagmus in the caloric test position with eyes closed using DC electronystagmography and adequate mental alerting. On the repeated tests, 14 (50%) had no residual spontaneous nystagmus, five (18%) had a low-intensity spontaneous nystagmus beating toward the normal side, and nine (32%) had a low-intensity spontaneous nystagmus beating toward the hypoactive side.

A special case of chronic asymmetry is that of the slowly progressive unilateral lesion (eg, acoustic neuroma). Low-intensity spontaneous nystagmus with eyes closed is common in this situation. For example, a review of our clinic records on 30 consecutive patients with acoustic neuroma disclosed that 24 (80%) had a spontaneous nystagmus recorded with eyes closed using DC electronystagmography and adequate mental alerting. Of those with spontaneous nystagmus, 17 (71%) had nystagmus that beat toward the unaffected side.

Recovery

Sometimes, unilateral peripheral vestibular lesions are temporary, and partial or complete recovery of function occurs. The most familiar situation where one encounters recovery is the acute attack of Meniere's disease. Figure 1 illustrates the pattern of spontaneous nystagmus that one observes with a Meniere's attack. During the initial acute stage, one observes a spontaneous nystagmus beating toward the unaffected side. As the attack subsides, the nystagmus reverses direction and beats toward the ear with the active disease. The reversal of the nystagmus (defined as recovery nystagmus) results from a return of the resting discharge on the affected side toward normal after there has been substantial adaptation to the asymmetrical state. Provided some degree of adaptation has occurred, a return of the resting discharge is interpreted by the CNS as a stimulus in the opposite direction relative to the original asymmetry. A more detailed discussion of recovery nystagmus can be found in previous publications.[6,8]

Recovery is not restricted to Meniere's disease but can occur with other conditions.[4] Figure 2 illustrates a nystagmus reversal following an acute viral labyrinthitis, suggesting occurrence of recovery several days after the acute stage of the disorder.

DYNAMIC PATHOLOGICAL VESTIBULAR ASYMMETRY

As the name implies, dynamic pathological vestibular asymmetry occurs as a result of a peripheral lesion but exists only in association with head movement. In other words, the asymmetry generated with normal head movements is abnormal.

In recent years, the use of rotational tests has allowed a better assessment of the vestibular system in the dynamic state. One method of assessment makes use of a frequency response analysis whereby sinusoidal stimuli are applied either as discrete sinusoids or in the form of more complex waveforms combining multiple sinusoids. The system response is measured in terms of the slow-phase eye velocity, and the system characteristics can then be described at different frequencies by gain (ratio of maximum eye velocity to maximum head velocity) and phase (head velocity relative to eye velocity) factors.

One can acquire some insight into the nature of one type of dynamic asymmetry, namely a unilateral loss of peripheral vestibular function, using a test method that employs discrete sinusoidal stimuli. Figure 3 shows the slow-phase eye velocity output in response to a sinusoidal input stimulus for two patients with a unilateral vestibular loss, in one case due to surgery for removal of a left acoustic neuroma and in the other case due to a right viral labyrinthitis. The two notable features of these plots are the difference in maximum velocity between the two halves of the cycle

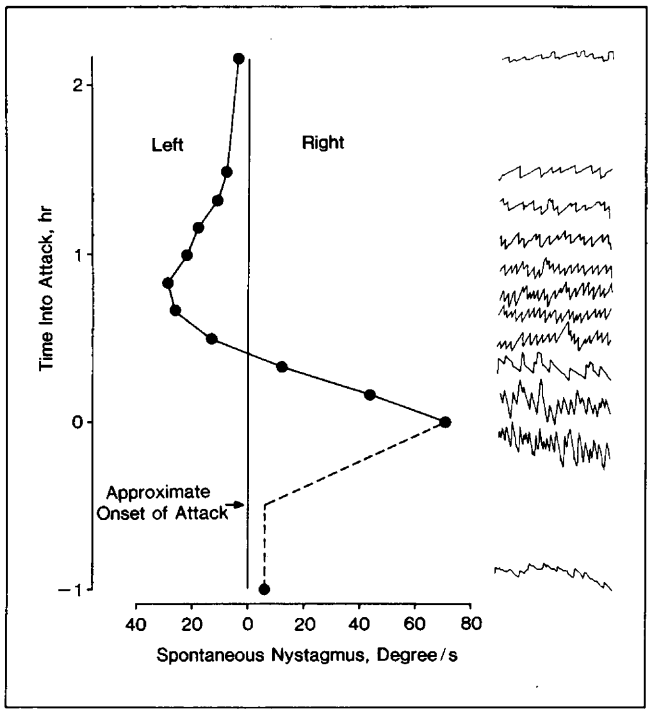

Fig 1.—Pattern of spontaneous nystagmus during attack of Meniere's disease involving left side. Initial recording in acute phase is designated as time zero. Dotted line indicates approximate time of onset of attack. Magnitude of slow-phase nystagmus velocity is plotted on x-axis. Right and left refer to nystagmus direction (ie, direction of fast phase). Actual sample nystagmus recordings for each point (calibrate not the same for each) are shown on right.

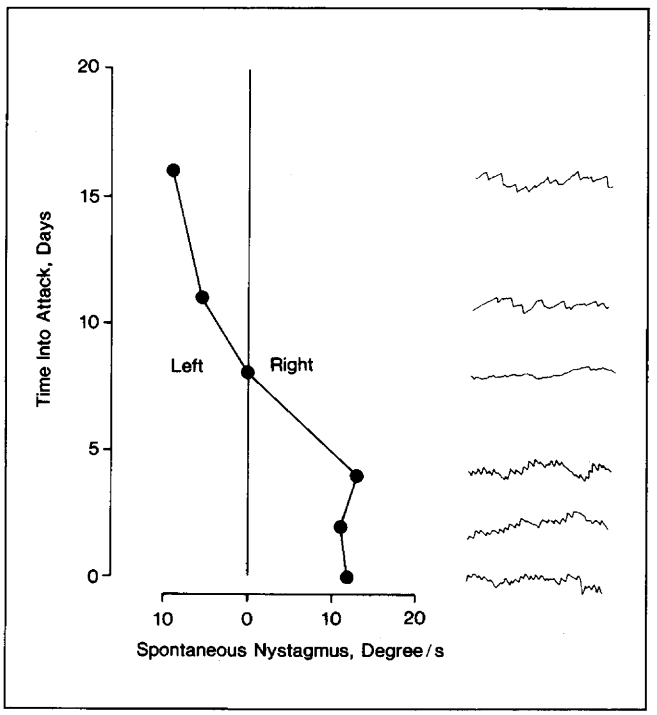

Fig 2.—Pattern of spontaneous nystagmus during attack of viral labyrinthitis involving left side. Figure details are same as those described for Fig 2.

and the discontinuity that occurs at the zero crossing points. It should be noted that both of these patients had a spontaneous nystagmus that is additive with the induced nystagmus (see later discussion). However, in the course of the data reduction procedure, the effect of the spontaneous nystagmus is removed and does not appear in the plots shown in Fig 3. For the patient with an acoustic neuroma, the maximum velocity is greater when the slow-phase eye velocity is directed toward the left, and for the patient with viral labyrinthitis when the slow-phase eye velocity is directed toward the right. This translates into a higher gain when the patient is rotated toward the unaffected side. From these findings, it is not difficult to appreciate why head movement could generate symptoms. As with static asymmetry, dynamic asymmetry can be observed in different stages with the acute stage representing that period when the patient is very sensitive to head movement and the chronic stage representing that period as the patient adjusts or adapts to the abnormality.

VISUAL INDUCED ASYMMETRY

In recent years, it has become apparent that sensory input originating in the peripheral visual system can converge on vestibular pathways.[9-11] At present, this convergence is thought to occur at the level of the vestibular nuclei. The results of such visual effects are manifest in the phenomena of circularvection and linearvection. For example, most people have experienced linearvection when sitting on a stationary train. If a train on an adjacent track starts to move and the movement is detected visually, then a distinct feeling develops that one's own stationary train is moving in the opposite direction. The counterpart in the circularvection world is the optokinetic phenomenon. The convergence of peripheral visual and vestibular information finds support in certain clinical observations associated with vestibular dysfunction. When vestibular dysfunction is present, one might anticipate that the patient's symptoms would be aggravated by excessive motion perceived in the peripheral visual field. It is certainly true that patients with an acute vestibular upset are more comfortable with eyes closed. It has also been our experience that patients with a vestibular disorder may find certain "motion-active" visual environments (eg, large busy department store or excessive movement on a television or movie screen) disturbing. The findings of Zee et al[12] provide some evidence for altered visual effects in association with a peripheral vestibular lesion. They showed that optokinetic responses such as optokinetic afternystagmus and aftersensation were deficient or absent in labyrinthine-defective humans.

COMMENT

The objective indicator of static vestibular asymmetry is a persistent

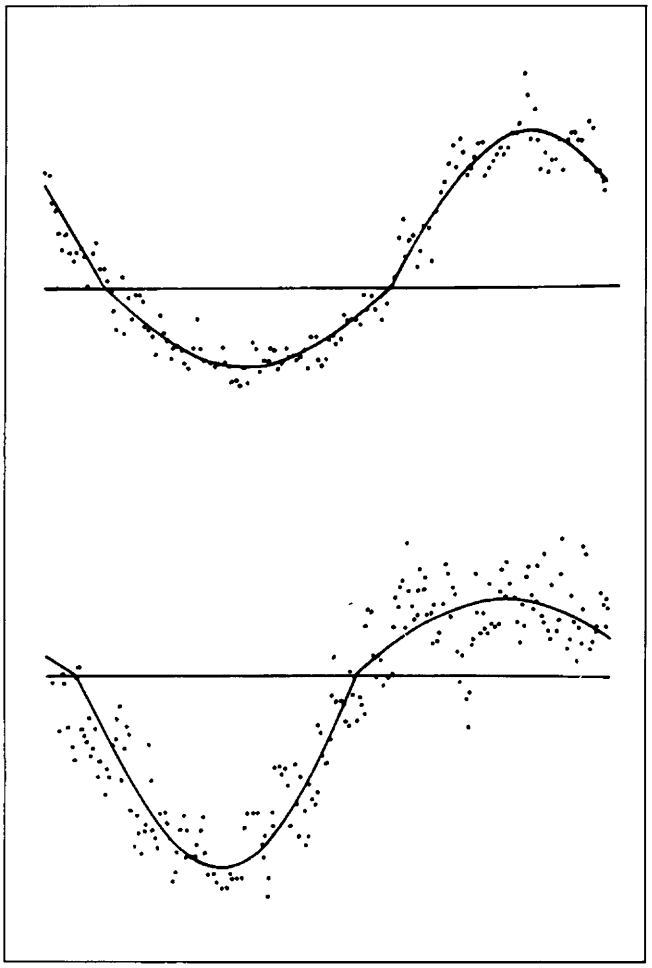

Fig 3.—Horizontal slow-phase eye velocity in response to 0.04-Hz sinusoidal rotational stimulus applied in plane of horizontal semicircular canals. Top trace, From patient following removal of left acoustic neuroma. Bottom trace, From patient with right viral labyrinthitis. One stimulus period (25 s) represented along x-axis. Horizontal line represents zero eye velocity. Positive velocity points represent slow-phase eye velocity to left (ie, nystagmus to right) and vice versa for negative velocity points.

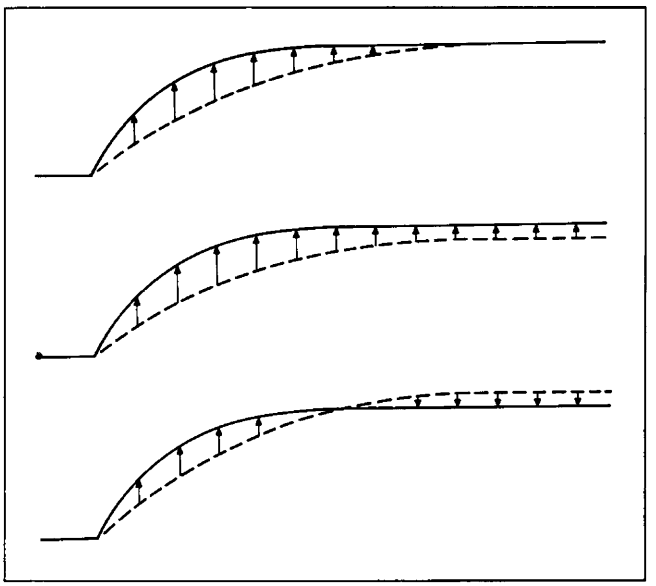

Fig 4.—Representation of theoretical concept to explain residual spontaneous nystagmus following adaptation to permanent peripheral vestibular asymmetry. See text for details.

spontaneous nystagmus that beats only in one direction depending on how the CNS perceives the direction of the asymmetry. In accordance with this concept, a spontaneous nystagmus exists irrespective of the presence or absence of any nystagmus secondary to an induced asymmetry. In fact, spontaneous nystagmus generated by static asymmetry would be additive with respect to a concomitant induced nystagmus. Often in the literature this distinction is not made. For example, based on clinical observation, Barber and Stockwell[13] defined *spontaneous nystagmus* as nystagmus that is direction fixed and beating with about the same intensity in all head positions when the eyes are closed. However, if the nystagmus changes in intensity or direction with position change, then they defined it as *positional nystagmus.* The implication is that spontaneous nystagmus and positional nystagmus cannot exist simultaneously.

If one accepts that spontaneous and positional nystagmus are generated independently and that these nystagmus types are additive, then one can readily explain the direction-fixed and direction-changing types of positional nystagmus. By definition, positional nystagmus is induced by the force of gravity and would be oppositely directed when the gravitational vector is applied in two directions of stimulation that are 180° out of phase. Most commonly, observations are made with the patient in the right and left lateral positions. When spontaneous nystagmus is present, it will add to the positional component that is in the same direction and subtract from the positional component that is in the opposite direction. In the latter case, if the spontaneous nystagmus is of lesser magnitude than the positional component, the positional component will predominate and the positional nystagmus will continue to appear as direction changing. However, if the spontaneous nystagmus is of greater magnitude than the positional component, the spontaneous nystagmus will predominate and the positional nystagmus will appear as direction fixed. The implication is that the direction-changing or direction-fixed feature of a persistent positional nystagmus is strictly a manifestation of the magnitude of a superimposed spontaneous nystagmus. Thus, it is not surprising that Baloh and Honrubia[14] made the comment that it is now generally accepted that direction-changing and direction-fixed stationary positional nystagmus are most common with peripheral vestibular disorders.

The additive effect can be observed with other forms of induced nystagmus. With hot and cold caloric tests, one frequently calculates a directional preponderance, and this reflects the

magnitude of a superimposed spontaneous nystagmus. In the case of sinusoidal rotational tests, Wolfe et al[15] used directional preponderance as a measure of system asymmetry. It is important to recognize, however, that in this particular instance, a difference in gain when rotating in opposite directions as well as spontaneous nystagmus can contribute to the directional preponderance. In certain situations, the differential gain effect and spontaneous nystagmus may augment (eg, acute stage of a unilateral hypoactivity). In other situations, these two effects may be oppositely directed (eg, recovery stage of a unilateral hypoactivity) with the resultant direction of preponderance dependent on the relative magnitude of the two effects. This latter situation may account for a finding reported by Olson and Wolfe.[16] They found that their group of patients with severe Meniere's disease (ie, patients who had had an attack within three months prior to testing) had a directional preponderance toward the diseased ear, and they attributed this to an irritation of the involved ear. Based on the findings of McClure et al,[6] it is more likely that the diseased ear was still hypoactive from a gain point of view and that the directional preponderance toward the involved ear was due to an overriding recovery nystagmus. Although Olson and Wolfe did not indicate the timing of their rotational tests, it is unlikely that they were done during the acute phase of a vertigo attack and more likely that they were done after or between attacks when the patient would be in a recovery phase. This group did indicate in another publication[17] that none of their patients with Meniere's disease were tested while in an acute phase.

To appreciate its importance, spontaneous nystagmus must be interpreted in the light of the clinical picture. For example, a complaint of severe vertigo accompanied by the finding of spontaneous nystagmus suggests an acute unilateral peripheral vestibular disorder. This is confirmed if one finds a hypoactive caloric response with spontaneous nystagmus directed toward the opposite side. If and when the peripheral vestibular system recovers, the patient may experience low-grade symptoms in the form of mild vertigo and instability. The persistence of such low-intensity symptoms following an acute vestibular upset can be of concern to both patient and physician and sometimes leads to extensive investigation. However, a recovery state is suspected if one finds a unilateral deficit with caloric tests and a spontaneous nystagmus beating toward the side with the reduced caloric response. In such situations, the symptoms will gradually subside as the recovery process reaches completion. In situations where the spontaneous nystagmus can be followed from the acute to recovery stage, such as the Meniere's attack illustrated in Fig 1, the direction of the nystagmus provides objective evidence of the side with active disease.

Residual spontaneous nystagmus following adaptation to a permanent peripheral vestibular asymmetry can be explained theoretically by the adequacy of the adaptation process. The concept is illustrated in Fig 4. In each graph, the solid line represents the magnitude of the static vestibular asymmetry and the dotted line represents the state of adaptation. The effective stimulus causing spontaneous nystagmus is the difference between the vestibular asymmetry and the level of adaptation, and in Fig 4 this is represented in magnitude and direction by the arrows. In the top graph of Fig 4, the adaptation process is ideal with a gain of one, and after sufficient time, adaptation completely compensates for the vestibular asymmetry. In the middle graph of Fig 4, the adaptation process has a gain of less than one, and it never completely compensates for the vestibular asymmetry. The result is a persistent low-intensity spontaneous nystagmus that beats in the same direction as that of the spontaneous nystagmus during the acute stage. In the lower graph of Fig 4, the adaptation process has a gain of more than one, and therefore it overcompensates for the vestibular asymmetry. This leaves a residual asymmetry that is in the opposite direction to that of the original asymmetry during the acute stage. The result is a persistent low-intensity spontaneous nystagmus that beats in the opposite direction to that of the spontaneous nystagmus during the acute stage. Fortunately, in most cases of unilateral vestibular loss, the adaptation process is able to reduce the magnitude of the asymmetry to a level that does not result in any bothersome residual symptoms. Occasionally, this is not the case and a patient with a unilateral loss can be left with persistent mild symptoms. In the case of a slowly progressive unilateral lesion, the adaptation process often cannot quite keep pace with the developing asymmetry. This gives rise to a persistent low-intensity spontaneous nystagmus that beats toward the unaffected side.

The dynamic asymmetry illustrated in Fig 3 is consistent with Ewald's second law. For the horizontal semicircular canals, Ewald's second law states that deflection of the cupula toward the utricle (utriculopetal) produces a greater change in the neuronal discharge rate than an equivalent deflection of the cupula away from the utricle (utriculofugal). Such a finding is readily demonstrable in single-unit studies in animals[18] but is not so obvious from the clinical tests that we use on patients. However, note in Fig 3 that rotation, which causes utriculopetal deflection of the cupula of the horizontal canal on the unaffected side, results in a higher-magnitude slow-phase velocity response. To state that this is evidence of Ewald's law assumes that with unilateral loss of function the unaffected side makes the largest contribution to the overall asymmetry. This is not an unreasonable assumption, at least in the early stages of a unilateral loss of function. The presence of a dynamic vestibular asymmetry introduces certain interesting clinical implications. Unlike static asymmetries over which the patient has no control, dynamic asymmetries can be avoided by keeping the head at rest. Often, the patient adapts quickly to a static asymmetry and is completely symptom free with the head at rest. However, the recurrence of symptoms with head movements can persist for days or weeks. Although the rate of

adaptation to dynamic asymmetry may be inherently slow, undoubtedly part of the reason for prolonged intolerance to head movements arises from the fact that the patient voluntarily restricts his activities, and this lack of exposure to the dynamic stimulus delays the adaptation process. Optimum time for adaptation is probably realized when the patient pursues a level of activity that produces mild symptoms.

In Fig 3, the asymmetrical gain and discontinuity at the zero crossing points indicate that the vestibulo-oculomotor system in this configuration is a nonlinear system. This has important practical implications in the analysis of rotational test results, since some workers use a linear system analysis approach in their rotational test procedure, and this could lead to erroneous results in disorders where there is system asymmetry.

An interesting concept encompasses the possibility that peripheral visual information does not converge solely in the vestibular nuclei but is also fed via the efferent vestibular system to the level of the vestibular hair cell. The information could then gain access to the central vestibular system via the normal afferent pathways and induce a peripheral vestibular asymmetry in the conventional manner. Klinke and Schmidt[19] were able to demonstrate a change in activity of afferent vestibular nerve fibers in goldfish in response to a moving pattern of stripes in the visual fields. However, in the monkey, Keller[20] did not detect any modification of afferent vestibular activity by full-field optokinetic stimulation. If convergence occurs at a peripheral level, then the occurrence of visually induced symptoms in the presence of a peripheral vestibular disorder is to be expected. The final word on the site of visual-vestibular convergence has probably not yet been written.

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