Unilateral loss of peripheral vestibular function in patients: Degree of compensation and factors causing decompensation

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Unilateral surgical ablation of peripheral vestibular function has been suggested for the treatment of a number of diseases that involve vestibular dysfunction. The postoperative distressing symptoms usually subside with time, whereupon the patient is said to have clinically compensated. However, even in well-compensated patients, the initial symptoms may reappear—under certain conditions that are briefly discussed (decompensation)—and, in addition, vestibular gaze stabilization deficits, (apparently permanent) appear whenever moderately rapid head movements are imposed. Thus, surgical ablation of unilateral peripheral vestibular function should not be considered "a treatment of choice," and should be performed in only carefully selected cases.
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Unilateral ablation of peripheral vestibular function, by either labyrinthectomy or vestibular nerve section, is usually performed for intractable Meniere's disease,[1] but has also been suggested for a number of other conditions that cause vestibular dysfunction.[2,3] The immediate postoperative symptoms are postural instability, sensation of rotation, nausea, vomiting, and nystagmus with fast phases toward the healthy side. These symptoms, the severity of which is closely related to preoperative residual vestibular function, slowly diminish with time until they almost disappear, although certain signs may persist long after surgery. Spontaneous nystagmus, for instance, is not visible in light after a few days, but it may be recorded in darkness—occasionally for years.[4]

The gradual decrease of symptoms after unilateral loss of vestibular function is due to either recovery or compensation, or both. The term *recovery* is used to describe the restoration of peripheral function, which is of course impossible in the case of surgical ablation. *Compensation* is the central restoration of function in the permanent absence of peripheral vestibular function. Patients are said to have clinically "compensated" when the postoperative symptoms and signs disappear, and to have "decompensated" when some condition causes these symptoms to reappear.

Electrophysiological studies[5] have shown that subsequent to a unilateral peripheral vestibular lesion, second-order vestibular neurons tend to modulate their resting discharge and reach prelesion levels, perhaps through a rearrangement of the intervestibular commissural signals,[6,7] with a number of brainstem structures contributing to this process.[8] Presumably such neural rearrangements are responsible for postoperative improvement. This improvement may reach the point at which the patient feels almost normal and the physician is satisfied with the treatment result.

There are, however, a number of questions of clinical importance related to the process and state of compensation:

1. What conditions speed up or slow down the process of compensation?
2. What conditions induce decompensation; that is, the return of symptoms similar to those experienced immediately after the loss of peripheral vestibular function?
3. Are there any permanent deficits of vestibular function in the compensated state?

These inter-related questions are briefly discussed in this article.

THE RATE OF COMPENSATION

Several factors modulate the progress of compensation:

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First, in experimental labyrinthectomized animals, sensorimotor deprivation—such as lack of visual cues, or restriction of motion—greatly delays compensation.[9] Physical activity probably speeds up compensation in patients, and lack of it seems to delay it.*

Second, drugs are known to modulate the rate of compensation—at least in animals. Phenobarbital or chlorpromazine, for instance, are known to delay compensation in guinea pigs.[10] Diazepam, on the other hand, suppresses the acute effects of labyrinthectomy, but does not influence the compensation process in cats.[11] In contrast, other drugs, such as metamphetamine or caffeine, accelerate compensation in guinea pigs.[12] A recent study in cats[13] indicated that the most effective drugs in accelerating the process of compensation were amphetamines and trimethobenzamide in a group of five drugs tested (diazepam, dimenhydrinate, and scopolamine being the other three drugs).

Third, brainstem lesions may delay or even halt the process of compensation.[14]

REAPPEARANCE OF INITIAL SYMPTOMS (DECOMPENSATION)

Several situations may cause the reappearance of some initial postlesion symptoms:

First, in "compensated" patients we have observed that the immediate postlesion nystagmus may reappear after exposure to a number of moderately rapid head turns of velocities similar to those experienced during natural behavior. In some of these patients, the reappearance of such nystagmus was accompanied by mild nausea and unsteadiness. Such symptoms did not develop in normal subjects under the same experimental conditions.[15,17] Similar nystagmus, termed *head-shaking nystagmus* and induced in patients under comparable conditions, has been previously described as indicative of unilateral loss of vestibular function.[18] These observations suggest that natural high-velocity head movements may cause reappearance of postlesion symptoms, and that patients may "learn" to avoid such head movements so as to prevent this unpleasant experience.

Second, it is well known that lack of mental alertness greatly diminishes the quality and magnitude of vestibular response in normal subjects.[19] Patients, in the compensated state, frequently report that their postural stability deteriorates when they feel tired.* One might wonder whether fatigue and diminished mental alertness may cause diminished effects of active compensatory influences that may, in turn, disrupt how the patient relates to the environment during movement.

Third, drugs that cause reappearance of initial symptoms have been described in different species. For example, acetylcholine-agonists and -antagonists cause strong decompensation in frogs.[20] Ingestion of alcohol in hemilabyrinthectomized cats[21] causes similar symptoms. Also, patients with loss of unilateral vestibular function frequently report postural instability after ingestion of even small amounts of alcohol.*

Fourth, in compensated experimental animals, symptoms of vestibular decompensation reappear after different lesions in parts of the central nervous system, such as destruction of the fastigial nuclei,[22] transection of the spinal cord or hemispherectomy,[23] lesions of the inferior olive,[24] or interruption of the intervestibular commissural connections.[25]

VESTIBULAR FUNCTIONAL DEFICITS

Previous studies of patients with compensated unilateral loss of peripheral vestibular function have indicated that more than half of these *individual* patients exhibited vestibular function that was not significantly different from normal,[26] at least when examined during low-velocity (60°/s) sinusoidal head movements with frequencies in the mid-band range (0.5 to 2 Hz). Other studies have reported abnormalities in vestibulo-ocular behavior,[27,28] generally at lower frequencies (less than 0.1 Hz), but the functional implications of these observations were unclear.

We have recently examined patients with compensated unilateral loss of peripheral vestibular function during moderate-velocity (160°/s) head turns.[16] While comfortably secured to a rotating chair and wearing a helmet attached to the chair, each patient was asked to look at a point on the wall. All lights—including the target—were extinguished. Then, while the patient continued to try to "look" at the unseen target, the chair was quickly turned to a new position and stopped. The lights were turned on again and the point was re-fixated (if necessary). It was found that slow-phase eye movements, which tended to keep the eyes on the unseen target, became progressively less effective during more rapid head movements directed towards the side of the lesion. For moderate head velocities (160°/s) directed towards the affected side, slow-phase stabilization was about half of that seen in the opposite direction. A surprising finding was that these slow-phase eye movements were interspersed by compensatory quick-phase movements that supplemented deficient slow-phase movements, and thereby tended to keep the eyes on the unseen target. In all patients, however—although these quick phases were larger than normal (presumably to

*Personal observations (A.K.).

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supplement the deficient slow phases)—gaze stabilization due to both slow- and quick-phase eye movements was less effective than that seen in normal subjects. These results suggested that unilateral loss of peripheral vestibular function caused two gaze-stabilization deficits: (1) deficient slow-phase eye movements during rapid ipsilateral head movements and (2) deficient quick-phase eye movements that failed to completely supplement deficient slow-phase eye movements.

CONCLUSION

Long after unilateral loss of peripheral vestibular function, compensated patients continue to exhibit vestibular functional deficits during head movements well within the range of normal, everyday velocities. Furthermore, the postlesion distressing symptoms may reappear under certain conditions (drugs, fatigue, etc.) with fluctuating intensity. Such observations lead to the following conclusions:

1. Surgical ablation of unilateral vestibular function, although necessary in some cases, should not be regarded as the treatment of choice for vestibular disease, and should be performed only after other more conservative treatment modalities have clearly failed.
2. When ablation is performed, it is essential that the patient be mobilized and encouraged to resume normal activities as soon as possible in order to facilitate the process of compensation.

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