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Acquired Bilateral Peripheral Vestibular System Impairment: Rehabilitative Options and Potential Outcomes

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Abstract

Acquired bilateral vestibular impairment can be a devastating disorder that is most frequently the result of aminoglycoside-induced toxicity. The presenting complaints are typically oscillopsia and gait and balance disturbances. These patients can be excellent candidates for vestibular rehabilitation therapy that focuses on facilitating maximal use of any remaining vestibular function, improving gaze and postural stability through the use of visual and somatosensory cues, and improving home and workplace safety. The prognosis for recovery is determined by the extent of the loss and the presence of other progressive disorders that may affect vision or somatosensation, coexisting illnesses, and the patient's compliance with the therapy program. Two cases are presented to illustrate the salient aspects of vestibular rehabilitation for patients with acquired bilateral vestibular system loss, including factors affecting patient progress and final outcome.

Key Words: Imbalance, oscillopsia, rehabilitation, unsteadiness, vestibular

Abbreviations: COR = cervico-ocular reflex, DHI = Dizziness Handicap Inventory, ENG = electronystagmography, IV = intravenous, VOR = vestibulo-ocular reflex

Patients with acquired bilateral peripheral vestibular system impairments are seen frequently in a hospital-based balance function laboratory. Bilateral impairments occur most frequently following exposure to aminoglycoside antibiotics. As a group, patients with bilateral vestibular system impairments experience greater degrees of self-perceived balance handicap than do patients with unilateral vestibular loss (Jacobson and Calder, 2000), and their prognosis for complete recovery is poorer. Patients with bilateral impairments can be bothered by oscillopsia (i.e., the inability to focus clearly on a visual target with head motion) and gait and balance disturbances. They are particularly unsteady and at risk for falling when walking in darkness (e.g., going to the bathroom in the middle of the night) or in visually active environments (e.g., walking down the aisle of a supermarket). Further, these patients often are unsteady when walking on compressible or uneven support surfaces (e.g., thick carpeting).

The purpose of this report is to discuss the clinical presentation of bilateral peripheral vestibular impairment and rehabilitative options for this patient population. The discussion will include a brief overview of the theoretical basis for vestibular rehabilitation and factors to be considered in the vestibular rehabilitation evaluation, the design and implementation of a vestibular rehabilitation treatment plan, and prognostic variables affecting therapy progress. Two illustrative cases will be described. Both cases presented to our clinic with ototoxically induced vestibular loss. Although both patients have benefited from vestibular rehabilitation by improvements in their abilities to participate in normal activities of daily life, their therapy course and current status are quite different. Specifically, 1 year following the onset of vestibular rehabilitation, the first case continues to feel frustrated about his slow progress and oscillopsia. He remains on long-term disability. On the other hand, 1 year later, the second case reports that she is able to function in all of her

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normal premorbid activities, and although she notices some slight oscillopsia when moving her head rapidly, it is not interfering with her daily function.

CASE REPORTS

Case 1

The patient was a 52-year-old male referred to our clinic for vestibular rehabilitation from an outside otolaryngologist with a diagnosis of bilateral vestibular system paresis secondary to ototoxicity. He presented with a complicated medical history including perforation of the esophagus during a laparoscopic procedure for the treatment of peptic esophagitis to prevent reflux. He subsequently developed peritonitis, for which he was treated with intravenous (IV) administration of tobramycin, gentamicin, and vancomycin over the course of 2 months. He was hospitalized a second time for treatment of an abdominal abscess, a third time for a subdiaphragmatic abscess, and a fourth time for cellulitis of the neck. In total, the patient was hospitalized during most of the 6-month period immediately following the original outpatient surgical procedure. He received gentamicin alone and in combination with vancomycin and tobramycin during three of the hospital stays. Overall, 55 pharmacy entries for one of the three drugs were found in the patient's medical record. Additionally, following the first admission, the patient was discharged with IV gentamicin and vancomycin.

The patient was referred to our clinic 18 months following the first outpatient surgery. He had received physical therapy elsewhere 9 months following surgery. Despite that, he reported that 10 months following his original surgery, he fell and broke his clavicle, further complicating his overall recovery. His presenting complaints were unsteadiness, ataxic gait, and oscillopsia. He had been placed on long-term disability from his position as a tape editor for a local television station. He was unable to participate in any activities, including doing simple chores around the house. Furthermore, he appeared to be depressed, as he was tearful throughout many of the initial evaluation and therapy sessions.

Electronystagmography (ENG) was conducted at an outside laboratory. The report generated by the outside facility stated that the patient had a bilateral weakness on bithermal caloric testing. Additionally, no responses were obtained to ice water caloric irrigation of either ear.

Both rotational testing (Neurokinetics, Model 1010, Pittsburgh, PA) and dynamic posturography (EquiTest Version 5.07, NeuroCom International, Inc., Clackamas, OR) were completed at our facility. Rotational testing was completed at five chair frequencies ranging from 0.01 to 0.32 Hz (50 deg/sec peak velocity). The relevant dependent variables for rotational testing are gain, which is the amplitude relationship between chair (head) movement and the vestibulo-ocular reflex (VOR) phase, which is the timing relationship between chair movement and eye movement, and asymmetry, which is the amplitude relationship for eye movements in response to chair rotation to the right versus to the left. When VOR gain is significantly reduced (i.e., there is little or no nystagmus), phase and asymmetry are meaningless. In this patient, response gains were significantly reduced for all chair frequencies tested (Table 1). In this regard, the patient was unable to feel the chair moving at all frequencies except 0.32 Hz.

The sensory organization subtest portion of dynamic posturography revealed a multisensory pattern (Fig. 1). Specifically, the patient exhibited increased postural sway in conditions 2 and 3 (i.e., fixed support surface with absent

Table 1 Vestibulo-ocular Reflex Gain (%) at Each of the Five Chair Frequencies Tested for Cases 1 and 2

	Frequency (Hz)				
	0.01	*0.04*	*0.08*	*0.16*	*0.32*
Case 1	6	11	10	22	24
Case 2	5	7	13	27	28
Normal lower limit (mean − 2 SD)	14	34	45	45	45
Normal upper limit (mean + 2 SD)	46	100	100	101	101

Also shown are lower and upper normal limits (i.e., mean ±2 SD).

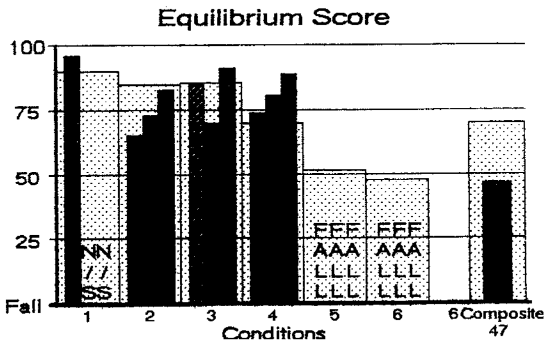

Figure 1 Sensory organization subtest results for case 1. Abnormal postural sway was noted in condition 2 (fixed support, absent vision) and condition 3 (fixed support, sway-referenced vision), and fall reactions were noted in condition 5 (sway-referenced support, absent vision) and condition 6 (sway-referenced support and vision).

vision and with sway-referenced vision, respectively), although the performance on the third trials was nearly normal and normal, respectively. Additionally, the patient fell on all trials for conditions 5 and 6 (i.e., sway-referenced support surface with absent vision and with sway-referenced vision, respectively). These findings suggested that the patient might have a tendency to emphasize visual cues in postural stability strategies. Motor control testing yielded normal responses, and no strength asymmetries were noted.

The patient also completed the Dizziness Handicap Inventory (DHI) (Jacobson and Newman, 1990). The DHI is a 25-item tool for the evaluation of self-perceived balance disability/handicap. The statements comprising the DHI may be answered with "yes" (scored as 4 points), "sometimes" (scored as 2 points), or "no" (scored as 0 points). Accordingly, a maximum score on the device is 100 points (i.e., representing maximal self-perceived balance disability/handicap). His total score was 70 points. The only "no" responses were on the items relating to having problems "getting into or out of bed," "turning over in bed" increasing the symptoms, and "fear of staying home alone." As we have noted previously (Jacobson and Calder, 2000), patients with complete bilateral vestibular loss tend not to report increased dizziness associated with head movement, as we would expect to see with unilateral vestibular disorders. Additionally, patients with bilateral impairments generally are not afraid to stay home alone, perhaps because they are most comfortable in a familiar environment.

At the initial therapy evaluation, the patient was able to perform a Romberg with his feet spread slightly, although increased postural sway was noted. The patient was unable to perform a sharpened Romberg with eyes open or eyes closed. Also, he was unable to stand on one leg without support. The patient demonstrated a wide-based gait when ambulating. He was unable to ambulate during head movements (e.g., during head turning or looking up and down). Also, he was unable to walk in a straight line.

The patient was given a home exercise program including activities designed to increase gaze stability (e.g., reading a book), postural stability (e.g., walking around the block), and general fitness (e.g., riding a stationary bicycle). He and his wife also were counseled, at length, regarding safety issues. Specifically, he was advised to install nightlights throughout the house so that he would have the advantage of visual information during ambulation at night. Further, he was instructed, for obvious reasons of safety, to avoid stepladders and stepstools. He was instructed to remove area rugs (i.e., uneven surfaces) and to be especially careful when walking on compressible or uneven surfaces in darkness or visually active environments. The patient was advised to return for follow-up at 6-week intervals.

Although the patient noted improvement over time, he continued to be frustrated about the speed of his recovery and his inability to do the things he could do previously. Within 7 months, the patient was able to ride a stationary bicycle continuously for 4 minutes, walk around the block in daylight unassisted, mow a portion of his lawn, drive short distances in his own neighborhood, and read a book using a book mark as a visual tracking guide. He was unable to do any of those activities at the start of therapy. Although he has reported a decrease in oscillopsia, it continues to be problematic for him. Lower limb weakness and stamina are improving slowly. At 1-year post initiation of therapy, he is able to ride the stationary bike continuously for 10 minutes, and he is able to function around the house. However, ambulating in darkness and in visually active environments continues to cause difficulty. For example, during a recent grocery shopping trip, he reported that he had to stop frequently in order to be able to read the labels of items on the shelves.

His post-rehabilitation DHI total score is 66 points (i.e., a reduction in DHI total score of 4 points). The 95 percent confidence interval for pre- and post-treatment changes in the DHI total score is 18 points. Improvement has been limited, in part, because of the duration and complexity of the illness and the extent of the loss. Additionally, the patient's compliance with treatment recommendations has been inconsistent.

Case 2

The patient was a 46-year-old female with a history of renal failure secondary to hypertension. At the time of the initial evaluation, she had been on hemodialysis three times weekly for a period of 2 months. Approximately 1 month prior to referral to our clinic, she was hospitalized for 4 days for an infection secondary to her hemodialysis catheter and was treated with IV gentamicin and quinine sulfate. She was discharged taking quinine sulfate, vancomycin, and gentamicin after dialysis for 2 weeks. Her primary complaints at the time of evaluation were oscillopsia and unsteadiness when walking. She reported that she felt drunk and that she was walking into walls. She also reported that she had fallen on a couple of occasions.

Audiometric testing revealed normal hearing sensitivity bilaterally. ENG testing revealed absent responses to bithermal caloric stimulation and absent responses to ice water irrigations bilaterally. Results of rotational testing showed abnormally low VOR gains at all chair frequencies (see Table 1). Her total score on the DHI was 64 points.

At the time of her therapy evaluation, she was able to perform a Romberg but was unable to perform a sharpened Romberg or stand on one foot without assistance. Her gait was slightly wide based and became unsteady during ambulation with head movements. With her eyes open, she was able to walk in a straight line but was unable to walk tandem without corrective steps or falls. She was given a home therapy program focusing on improving gaze control and postural stability. She was also encouraged to participate in general conditioning activities (e.g., walking or riding a stationary bike) on a daily basis. Finally, she was counseled extensively regarding safety issues.

When the patient returned for follow-up 1 month later, she reported that she felt much better. She reported improvement in her oscillopsia. Specifically, she reported no oscillopsia when walking, although she continued to notice some "bouncing" of the visual field when riding in a car. Her balance problem was much better, and she was able to walk without unsteadiness or falls. Further, she reported feeling much more secure.

Dynamic posturography was completed at the time of the first follow-up visit (Fig. 2). Her performance was consistent with a multisensory disorder. Specifically, she exhibited increased postural sway in condition 4, although her performance approached normal on trial 3 and fell on all trials in conditions 5 and 6. This pattern of performance suggests that the patient might have a tendency to emphasize somatosensory cues in maintaining postural stability. Motor control testing yielded normal results, and no strength asymmetries were noted. These findings suggested that the patient continued to be at risk for falling in situations in which she did not have access to accurate somatosensory cues (e.g., when walking on compressible or uneven support surfaces). It was expected that the patient's problems would be exaggerated when ambulating in darkness or in visually active environments. Although her original therapy plan included having her practice controlled swaying while standing on a compressible surface, she had been inconsistent in practicing that exercise. She was encouraged to practice exercise consistently to increase her ability to make use of visual cues when accurate somatosensory information was unavailable.

One month later, the patient reported further improvement. She had resumed many activities of daily living, including driving (e.g., to and from dialysis) and shopping. She continued to experience some unsteadiness when moving her head while walking. However, by her final therapy visit (14 weeks following the start of therapy), she reported that she had improved greatly.

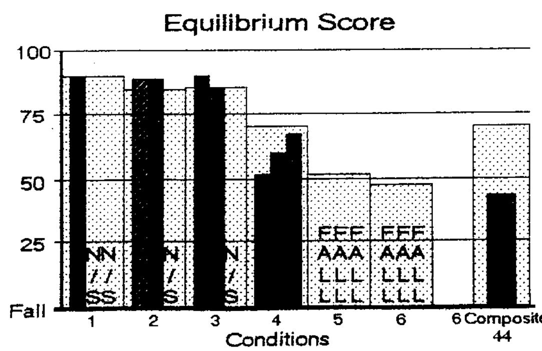

Figure 2 Sensory organization subtest results for case 2. Abnormal postural sway was noted in condition 4 (sway-referenced support, normal vision), and fall reactions were noted in condition 5 (sway-referenced support, absent vision) and condition 6 (sway-referenced support and vision).

Only on occasion did she notice oscillopsia. Additionally, she was able to move her head while walking without experiencing any vision problems or unsteadiness. She reported that she had worn shoes with heels a couple of days earlier, without incident. Finally, she reported that she was encouraged about the future. Her post-rehabilitation DHI total score was 24 points, suggesting a statistically significant improvement in self-perceived dizziness handicap (i.e., an improvement in DHI total score of 40 points).

COMMENT

The patients described in the present report had similar vestibulometric test results and baseline DHI scores, although their medical histories and treatment courses were different. The first patient suffered a long, complicated illness including several hospitalizations and multiple courses of ototoxic medications. For him, because of the seriousness of his overall medical condition and resulting muscle weakness and generalized fatigue, referral for vestibular rehabilitation occurred 1 year following the initial symptoms of his bilateral vestibular system paresis. To date, the patient has participated to some degree in vestibular rehabilitation for approximately 1 year. The patient continues to be disabled. The second patient experienced a single hospitalization for treatment of an infection and was referred for evaluation and treatment 1 month following the onset of her symptoms. In her case, vestibular rehabilitation occurred over a 3½-month period, and the patient was able to return to normal activities of daily life. Although both patients had profound vestibular system impairments, we contend that it was the difference in the patients' overall physical condition and their level of participation in the therapy program that led to the end result.

Characteristics of Bilateral Vestibular Loss

A complete or incomplete bilateral loss of peripheral vestibular function results in a reduction or loss of the VOR. Frequently, ototoxic agents cause the damage. Patients undergoing treatment with ototoxic medications often do not realize that they have a balance problem until they get out of their hospital bed and try to ambulate. Even then, physicians may assume that generalized weakness due to disease is the cause of the problem.

Normally, the VOR is responsible for enabling clear vision during head and body movement. With sudden loss of the VOR, the brain is unable to maintain gaze on a fixed point when the body is moving. Accordingly, patients with acquired bilateral loss of peripheral vestibular function have stereotypical complaints, including oscillopsia (i.e., a jiggling or bouncing of the visual field while the patient is in motion). The visual blurring that occurs may prevent patients from driving safely. Severe oscillopsia can result in motion sickness, further limiting the patient's ability to move around. In bilateral peripheral vestibular system impairment, quick movements of the head are associated with saccadic gaze readjustments instead of smooth compensatory eye movements, which impairs clear vision. Also, patients with complete bilateral vestibular loss may appear ataxic when walking as a result of the loss of the VOR. These patients rely on visual and proprioceptive information to maintain postural control during ambulation. At night, in darkness, patients with bilateral loss of vestibular system function are at particular risk for falls since they have only one functioning sense (proprioception) to help them remain upright. For these reasons, acquired bilateral vestibular system paresis can be a devastating disorder. However, in the presence of bilateral peripheral vestibular impairment and its associated disabilities, vestibular rehabilitation can improve stability of posture and gaze and quality of life.

Both of the patients in this report presented with primary complaints of unsteadiness and severe oscillopsia. In the first case, his balance problems were not fully recognized for over a year because of the nature of his illness and the duration of his hospital course and subsequent time of being bedridden. Once the balance impairments were identified, his physicians were insistent that he was merely weak. It was not until the problems persisted for several months that referral was made to an otolaryngologist, resulting in the documentation of the profound bilateral vestibular system deficit. Furthermore, once the vestibular deficit was identified, the patient's primary care physician was reluctant to refer for vestibular rehabilitation.

Although it is not clear that there is a prognostic relationship between the timing of the onset of vestibular rehabilitation and the magnitude of functional recovery, it is clear that this patient began therapy in a weakened state. His lower limb weakness and lack of stamina affected negatively his ability to practice the exercises he was given. He was unable to tolerate the general conditioning portion of his therapy program. As a result, his progress was slow, and he became frustrated.

The second case illustrates an ideal participation in and response to therapy. The second patient generally was in good physical condition (i.e., with the exception of her need for dialysis) and had been active up until the time of her hospitalization. Although she was unsteady at the onset of therapy, she did not have the muscle weakness or generalized fatigue that affected progress for the first patient. Consequently, she performed the exercises as prescribed and was determined to return to her premorbid activities.

Theoretical Basis for Vestibular Rehabilitation

Although they are considered to be reflexive, responses of even a partially functioning vestibular system can be modified. For example, research has shown that the magnitude of the VOR can be changed using an altered visual environment such as that produced by magnifying or miniaturizing lenses (Melville Jones, 1985). Additionally, repeated rotary stimuli can modify VOR timing (Baloh et al, 1982; Schmidt and Jeannerod, 1985). Finally, imagining an earth-fixed or a head-fixed visual target can affect VOR gain (Melville Jones and Berthoz, 1985).

Patients with bilateral vestibular loss rely initially on visual cues. However, over time, they tend to become more dependent on somatosensory cues. Accordingly, vestibular rehabilitation represents an attempt to assist patients in improving the extent that gaze stabilization during head movement and sensation of postural sway can be improved through optimization of these modalities. The additional loss or impairment of either visual or somatosensory senses (e.g., due to retinal degeneration or peripheral neuropathies, respectively) in addition to the vestibular system has a devastating effect on postural stability and limits the strategies that can be used in rehabilitation.

Although most of the support for the effectiveness of vestibular rehabilitation for patients with bilateral vestibular loss has come from anecdotal reports, in a blinded, controlled investigation, Krebs et al (1993) compared the results of a sham treatment (i.e., isometric exercises) with vestibular rehabilitation. The experimental program included vestibular adaptation exercises and exercises designed to develop compensatory strategies. Outpatient therapy occurred once weekly and home practice occurred once or twice daily. After 8 weeks of therapy, the investigators found that subjects were able to walk faster (i.e., during paced gait and stair climbing) and with greater stability than they were during baseline testing. They did not (predictably) observe changes in the vestibular function tests.

Vestibular Rehabilitation

Prior to designing a vestibular rehabilitation program, a thorough evaluation of the patient is completed by the therapist. For patients with bilateral vestibular disorders, it is particularly important to know how they are using sensory information in maintaining postural control. For that reason, dynamic posturography is an especially useful evaluation tool. For both of the cases presented, the data from dynamic posturography were used to help determine what activities the patient could do safely at home during therapy. For instance, since neither patient fell in condition 4 (sway-referenced support with normal vision), they were able to practice controlled postural sways while standing on a compressible surface with eyes open. Clinical tests of static and dynamic balance (e.g., Romberg, single leg, and tandem stance) and a qualitative assessment of gait (e.g., base speed, looking for veering with and without head movements, and during turning) were included in the evaluation. Both patients demonstrated the wide-based and ataxic gait seen typically in this population. These patients were very unsteady when asked to do anything else while walking (e.g., talk, turn or tilt head). Neither patient was able to turn without coming to a complete stop first.

Rehabilitation goals are determined for patients based on their specific problem areas. In general, home exercise programs are designed to address each of the goals. For patients with bilateral incomplete loss, the exercises are designed to facilitate maximal use of the vestibular input available to the patient and to help them learn to substitute other sensory inputs (e.g., vision and somatosensory inputs) for vestibular sensation. For patients with bilateral complete vestibular loss, the primary purpose of therapy is to facilitate the improved use of proprioceptive and visual information for stabilization of the eyes and body.

For instance, partial compensation for loss of the VOR may be to increase the effectiveness of the cervico-ocular reflex (COR). Like the VOR, the COR assists in producing compensatory eye movements during head movement (Herdman, 1994). Whereas in normal people, the COR complements the VOR and contributes only approximately 15 percent to the generation of compensatory eye movements, it may contribute up to 25 percent to the generation of the compensatory eye movements in patients having bilateral vestibular loss. An exercise that might be recommended to increase the COR (and that was given to both patients reported here) is having patients maintain visual fixation while turning their head. Interestingly, this exercise is designed to maximize use of the remaining VOR, so, for patients with some residual vestibular function, one exercise may serve both purposes.

Gaze stability during head movements may be improved by modifying saccade and pursuit system function. During head movement, patients with bilateral complete vestibular losses often use a combination of hypometric saccades and passive alignment of the eyes to move eyes to and with a visual target. Additionally, patients may use accurate saccadic eye movements during combined eye and head movements toward a target. These are followed by corrective saccades that pull the eyes back to the target as the head movement pulls the eyes off the target (Herdman, 1994). Part of the therapy process is to provide ample opportunities for patients to develop strategies for maintaining gaze stability. It is important to remember that neither vision nor proprioceptive inputs are able to fully compensate for the complete loss of the VOR. As a result, even under the best of circumstances, patients will continue to experience some visual blurring or jitter with rapid head movements.

Another component of a rehabilitation program is providing patients with opportunities to practice postural control in a variety of safe contexts. For example, patients may be asked to practice swaying from the ankles while standing on a compressible surface that is placed in the corner of a room (e.g., standing in a corner decreases the likelihood that a patient will free fall in an open space). Similarly, they may be asked to practice standing tandem or making a rapid 180-degree turn while standing in a corner. Each of those tasks might be unsafe to practice in an uncontrolled environment.

Finally, it is important to teach patients with profound bilateral peripheral vestibular deficits strategies to increase safety in their environments. Patients are advised to use nightlights whenever they ambulate in low light or darkness. Additionally, the use of an assistive device (e.g., cane, shopping cart, or another person) is encouraged, particularly when ambulating in visually active environments (e.g., aisle of a grocery store). Patients are encouraged to carry a small flashlight since the combination of a reduction of visual cues combined with reduced or inaccurate somatosensory cues increases the risk of falls in this population.

SUMMARY

Acquired bilateral vestibular paresis is a devastating disorder that is most frequently the result of aminoglycoside-induced toxicity, as was the situation with the illustrative cases presented here. The presenting complaints are typically oscillopsia and gait and balance disturbances. Patients with bilateral vestibular loss should be considered candidates for vestibular rehabilitation therapy. This therapy focuses on facilitating maximal use of any remaining vestibular function, improving gaze and postural stability through the use of visual and somatosensory cues, and improving home and workplace safety. It is important to remember, however, that functional recovery following bilateral vestibular loss is slower than it is for unilateral disorders, and progress can continue for a period of 2 years (Herdman, 1994). The prognosis for recovery is determined in part by the extent of the loss, and recovery is easily upset by a variety of patient variables, including the patient's compliance with the therapy program. In order to maintain improvements in function, patients should continue to implement some aspects of their vestibular rehabilitation program long term. Although patients may need to use an assistive device (e.g., a quad cane) in the early stages of recovery, under most circumstances, patients with bilateral vestibular paresis will be able to return to many normal activities of daily life. However, they will be at increased risk for falling when confronted with circumstances where low illumination or active visual environments are combined with reduced access to proprioceptive cues. Additionally, fatigue will continue to impact overall performance. For patients with complete bilateral loss and coexisting or progressive medical conditions that affect vision or proprioception, recovery may be limited, and the associated disability may be permanent.

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