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Vestibular Neuronitis in Pilots: Follow-up Results and Implications for Flight Safety

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Objectives: **To report our experience over the past 12 years with the evaluation and follow-up of pilots with vestibular neuronitis and to discuss points relevant to flight safety and the resumption of flying duties.** ***Study Design:*** **A retrospective, consecutive case series.** ***Methods:*** **Eighteen military pilots with vestibular neuronitis were examined and followed up. A complete otoneurological workup was performed, including both physical examination and laboratory evaluation. The latter included electro-oculography (EOG) and a rotatory chair test using the smooth harmonic acceleration protocol.** ***Results:*** **The mean patient age was 35 ± 6 years (range, 23 to 42 y), and the average follow-up period was 20.5 ± 12.8 months (mean ± standard deviation [SD]; (range, 11 to 48 mo). Electro-oculography caloric test on presentation documented significant unilateral hypofunction in all patients. Thirteen of the 18 patients (72%) had abnormal smooth harmonic acceleration test results. None of the pilots reported any symptoms on follow-up. However, five (28%) had positive otoneurological examination findings, and eight (44%) still had significant caloric lateralization (>25%). The average caloric hypofunction was reduced from 67.8% ± 29.3% at onset to 40% ± 16% (mean ± SD, *P* <.05, paired *t* test). Seven of the patients (39%) had additional electro-oculography findings beyond caloric hypofunction. These included spontaneous, positional, and positioning nystagmus. Smooth harmonic acceleration disease on follow-up was documented in eight patients (44%), five of whom had canal paresis. Eleven patients (61%) demonstrated residual vestibular damage on follow-up. In 6 of these 11 cases (55%), the laboratory evaluation revealed vestibular deficits otherwise undiagnosed by the bedside test battery.** ***Conclusions:*** **The vestibular system plays a central role in orientation awareness and is often challenged by flying conditions. The finding that approximately 60% of pilots who have had vestibular neuronitis continue to show signs of vestibular malfunction, despite apparent clinical recovery, emphasizes the need for a complete vestibular evaluation, including specific bedside testing and laboratory examinations, before flying duties can be resumed.** ***Key Words:*** **Vestibular function tests, electronystagmography, rotatory chair, vestibular neuronitis, vestibular compensation.**

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INTRODUCTION

Safety is a major consideration when flying military, commercial, or private aircraft. By virtue of its size, weight, and the amount of fuel carried, an aircraft is a potentially hazardous object. An accident involving the loss of an aircraft has serious consequences for passengers, the general public, and the environment under the flight path. Pilot spatial disorientation is a leading factor contributing to many fatal flying accidents. U.S. Air Force statistics of major aircraft accidents from 1980 to 1989 reported 356 mishaps caused by operator error, spatial disorientation accounting for 81 of these (23%).[1] A recent report from Canada has described a similar role for spatial disorientation as a cause of such accidents.[2] Spatial disorientation is not restricted to military aviation alone. Of the 4012 fatal general aviation accidents occurring between 1970 and 1975, 627 (15.6%) involved spatial disorientation. Ninety percent of mishaps in which disorientation was a cause or factor were fatal,[3] and the reason no evasive action was taken by the pilots in most cases was that they failed to recognize the disorientation episode which led to the accident.[2]

Spatial orientation is the product of integrative inputs from the proprioceptive, vestibular, and visual systems. Flying presents a continuous challenge toward this critical function, even for the completely healthy pilot. Motion and acceleration are encountered in three vectors and to degrees that are foreign to the human vestibular apparatus, and visual cues are sometimes missing or distorted. Pilots are exposed to many types of visual and vestibular illusions endangering flight safety. These are related to the lack of peripheral visual cues, linear and angular vection misperceptions, and somatogyral and somatogravic illusions, in which the input from the semicircular canals and otolith organs secondary to prolonged,

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high acceleration leads to misjudgment of rotation and altitude.[1,4]

The importance of normal function of all organs involved in orientation is mentioned in the aviation medical literature as being a prerequisite for flying. In cases of acute vertigo, full evaluation is recommended to ascertain the cause of the vertiginous attack and to determine the probability of recurrence that might lead to sudden pilot incapacitation.[5,6] According to current aeromedical guidelines, a patient with viral involvement of the vestibular system, either viral labyrinthitis or vestibular neuronitis, can return to flying duties following symptomatological recovery and central compensation, which might take 4 to 6 weeks.[5–7] Acute peripheral vestibulopathy is characterized by dysfunction of both static and dynamic components of the vestibular reflexes. Whereas the static symptoms and signs generally disappear within a few days, deficiencies of at least some of the dynamic vestibular functions have been reported even years after apparent clinical recovery.[8,9] These may be found by specific bedside examinations and sometimes only by vestibular laboratory evaluation specifically addressing dynamic vestibular responses. The purpose of the present study was to describe our experience with military pilots with acute peripheral vestibulopathies and to discuss vestibular findings obtained on follow-up as they pertain to the patient's return to flying duties.

MATERIALS AND METHODS

Eighteen male military pilots with vestibular neuronitis were evaluated and followed up at the Israel Naval Medical Institute, Haifa, between the years 1990 and 2001. Vestibular neuronitis was diagnosed clinically by the acute appearance of vertigo and dysequilibrium accompanied by spontaneous nystagmus and vegetative signs of nausea or vomiting, without hearing impairment or other neurological deficits.[10] When first examined, all patients had canal paresis greater than 25% on the electro-oculography (EOG) alternate bithermal water caloric test. The clinical presentation and caloric findings matched the accepted description of unilateral vestibular neuronitis.[8,10] Before the vestibular neuronitis episode, all patients were performing full flying duties, and no patient had a previous history of otoneurological problems. The average follow-up period was 20.5 ± 12.8 months (mean ± standard deviation [SD]; range, 11–48 mo). The mean patient age at the last follow-up examination was 35 ± 6 years (mean ± SD; range, 23–42 y).

A thorough otoneurological workup, including physical examination and laboratory vestibular evaluation, was performed on presentation and at the follow-up examinations. The bedside examination included a general head and neck examination, pneumatic otoscopy, tuning-fork test, and tests for pathological nystagmus, including spontaneous, gaze-evoked, positional and positioning nystagmus using the Dix-Hallpike maneuver, with the patient wearing Frenzel glasses to avoid pursuit system cancellation of the nystagmus. When no spontaneous nystagmus was found, the patient was tested for the appearance of post–head-shake nystagmus in response to 20 seconds of vigorous, active horizontal head shaking without visual fixation,[11] and for the presence of re-fixation saccades secondary to 20° head impulse.[12] Postural stability was examined by Romberg's test, past pointing, and tandem walking and stepping tests. Bedside visual-vestibular interaction was examined by the dynamic visual acuity test. For this test, the patient shook his head rapidly in the horizontal plane while reading Snellen's visual acuity chart at a standard distance. A drop in visual acuity of more than one line on Snellen's chart was considered abnormal.[13]

The vestibular laboratory evaluation included EOG, in which eye movements were measured during the saccade, gaze, and positional tests; the Dix-Hallpike maneuver; and alternate bithermal caloric tests. In addition, the response of the vestibulo-ocular reflex to various sinusoidal angular accelerations was examined by the smooth harmonic acceleration (SHA) test, which calculates the gain, phase, and asymmetry of the reflex in response to a series of yaw-axis angular accelerations.[14]

Statistical analysis was carried out using SAS software (SAS Institute, Inc., Cary, NC) on an IBM personal computer. The Student paired *t* test was used to compare caloric test averages on presentation and follow-up.

RESULTS

The patients presented with typical symptoms of vestibular neuronitis, including acute vertigo, spontaneous nystagmus, dysequilibrium, and accompanying vegetative signs. The EOG caloric test on presentation documented significant unilateral hypofunction in all patients. Average canal paresis was 67.8% ± 29.3% (mean ± SD). Thirteen of the 18 patients (72%) had abnormal SHA test results, manifesting as increased phase lead, low gain, and asymmetry.

None of the pilots reported symptoms on follow-up evaluation. However, five (28%) had positive otoneurological bedside examination findings, including low-grade spontaneous nystagmus when wearing Frenzel glasses, post–head-shake nystagmus, corrective saccades on the head impulse test, or reduced dynamic visual acuity. Eight of the 18 pilots (44%) still had significant caloric lateralization on their last follow-up examination. The group average caloric hypofunction was reduced from 67.8% ± 29.3% at onset to 40% ± 16% (mean ± SD; $P < .05$, paired t-test). Seven of the patients (39%) had additional EOG findings beyond caloric hypofunction, including spontaneous, positional, and positioning nystagmus. All five patients who had positive bedside findings on follow-up were in this group. These patients also had canal paresis and abnormal SHA findings. Follow-up SHA disease was documented in eight patients (44%), but only five of these also had canal paresis on the EOG. Eleven patients (61%) had residual vestibular damage on follow-up. In 6 of these 11 cases (55%), laboratory evaluation revealed vestibular deficits otherwise undiagnosed by the bedside test battery.

DISCUSSION

Vestibular neuronitis is a common clinical syndrome characterized by the acute onset of prolonged severe rotatory vertigo, which is associated with spontaneous nystagmus, a reduced or absent caloric response in one ear, postural imbalance, and nausea and vomiting, without cochlear or neurological findings.[10]

Vestibular neuronitis is considered to have a benign course. The static rotatory vertigo and dysequilibrium, present even when the patient is completely at rest, subside in most cases within a few days, and a gradual return to daily activities is the rule. However, it has been shown that there is generally incomplete restoration of peripheral function, and clinical recovery is achieved by proprioceptive and

visual substitution for the unilateral vestibular deficit, combined with central vestibular compensation of the imbalance in vestibular tone.[9] New behavioral strategies, such as increasing the number of blinks and saccades during head movement and restricting head perturbations during locomotion, with greater reliance on the optokinetic and pursuit systems to match eye and target velocities, also contribute to the satisfactory maintenance of balance and the absence of vertigo.[15,16]

Although vestibular neuronitis is usually restricted to one attack, several studies have reported continuous or episodic vertigo or unsteadiness in a significant number of patients (43%–53%).[17,18] No recurrent attacks or symptomatic residua were reported among our patient population. Possible explanations may be the relatively young age of our patient group and the fact that they were physically fit and maintained a high level of activity, all factors known to enhance vestibular compensation.[15,17,19] A further important trait inherent in our patients was their high motivation to resume flying duties, which might have led to their "playing down" any symptoms or even dissimulation on their part. Despite the absence of complaints on follow-up examination, the rate of positive findings on vestibular evaluation (canal paresis in 8 patients; positive SHA findings in 8 patients; and spontaneous, positional, or positioning nystagmus in the EOG recording in 7 of the 18 patients) was similar to that reported by other groups.[20–22] The decrease in the number of patients with canal paresis on follow-up and in the magnitude of canal hypofunction, and recovery of the vestibulo-ocular reflex time constant, as reflected by normalization of the phase parameter in the SHA test in some of the patients, match previous reports.[17,20–25] Three of the patients had positive SHA findings on follow-up examination, despite a normal caloric test response. The caloric stimulus produces low-frequency endolymph accelerations in the range of 0.002 to 0.004 Hz.[26] The SHA protocol we used includes a series of accelerations in the higher frequency range of 0.01 to 0.16 Hz. Thus, vestibulo-ocular reflex deficits not revealed by EOG caloric testing might be revealed by the SHA protocol.[27] Our findings confirm the results of a previous study, which failed to find a correlation between caloric test results and the vestibulo-ocular response to rotation on follow-up examination after acute peripheral vestibulopathy,[25] and reports of a prolonged deficient dynamic response to acceleration at high frequencies after peripheral vestibular deafferentation.[9,16,25,28]

Spatial orientation and postural control depend on the integration of information from the proprioceptive, vestibular, and visual sensory systems. Despite overlapping of the optimal frequency ranges for each of these systems, the integration of these signals requires correct weighting of the inputs.[29] Conflict between the senses requires adjustment of the integration process to determine the correct orientation in space and the appropriate motor response. Flying presents a challenge to spatial orientation, even when the dynamic responses of the vestibular system are normal, because visual and proprioceptive cues may often differ from vestibular information. Distorted proprioception secondary to extreme linear acceleration and deceleration requires greater reliance on visual and vestibular cues to maintain orientation.[30] The interaction between vestibular and visual inputs for the maintenance of spatial orientation is particularly apparent in the case of moving visual scenes. In normal subjects, the perceived visual and postural vertical are displaced in the direction of the moving visual field, accompanied by a sensation of body rotation in the opposite direction.[31] In subjects with peripheral vestibular deficits, enhanced effects of visual stimuli on perception of the vertical and on postural control were documented.[32,33] It has been suggested that the detectability of visual distance cues diminishes in the case of moving visual fields, and reliance on nonvisual information alone is then required to control posture. When the vestibular inputs are missing or distorted, the ability to counteract the effects of the disorienting visual stimuli decreases. Vestibular-deficient subjects often have visual vertigo, in which symptoms are provoked or aggravated by increased visual motion.[34] This vertigo is explained as being due to difficulty in resolving contradictory information between visual and vestibular inputs in the face of the increased visual dependence found in these subjects.[33] Recent animal data suggest that the deficient processing of spatial information associated with peripheral vestibular insults results from an irreversible decrease in the expression of neuronal nitric oxide synthase in the hippocampus.[35]

We suggest that in pilots with residual vestibular deficiency, the equilibrium and orientation responses to the moving visual stimuli encountered during flying will be inadequate and will contribute to disorientation. Additional potential sources of dizziness and loss of balance are the long visual distances and reorientation to gravity commonly experienced in the cockpit[29,36] and the increasing use of night-vision devices to improve navigation in low-light environments.[37] Another threat to flying safety is the association described between peripheral vestibular dysfunction and space and motion phobia. Panic attacks were elicited in vestibular-deficient patients by excessive vestibular stimulation, the absence of visual fixation cues, and unusual movements of the visual surround and self.[38] Stressful situations and hyperventilation might also in themselves exacerbate vestibular symptoms.[39] The link between vestibular deficiency and anxiety reactions might be explained by the presence of concomitant neural circuits. These include monoaminergic inputs to the vestibular system, which may mediate the effects of anxiety on the vestibular system, and the parabrachial nucleus network, which may mediate emotional responses to vestibular dysfunction.[40] A pilot subject to panic attacks or acute vestibular symptoms obviously presents a serious risk to the safe completion of any flight mission. Reported disorientation incidents are sometimes characterized by awareness of misperception combined with an inability on the part of the pilot to take the necessary action to regain control of the aircraft (type III spatial disorientation).[1] An anxiety reaction exacerbated by a vestibular deficiency might contribute to such events.

The potentially grave outcome of loss of control due to deficient vestibular function is not sufficiently addressed in current flight disposition guidelines. These guidelines suggest that resolution of symptoms and clinical compen-

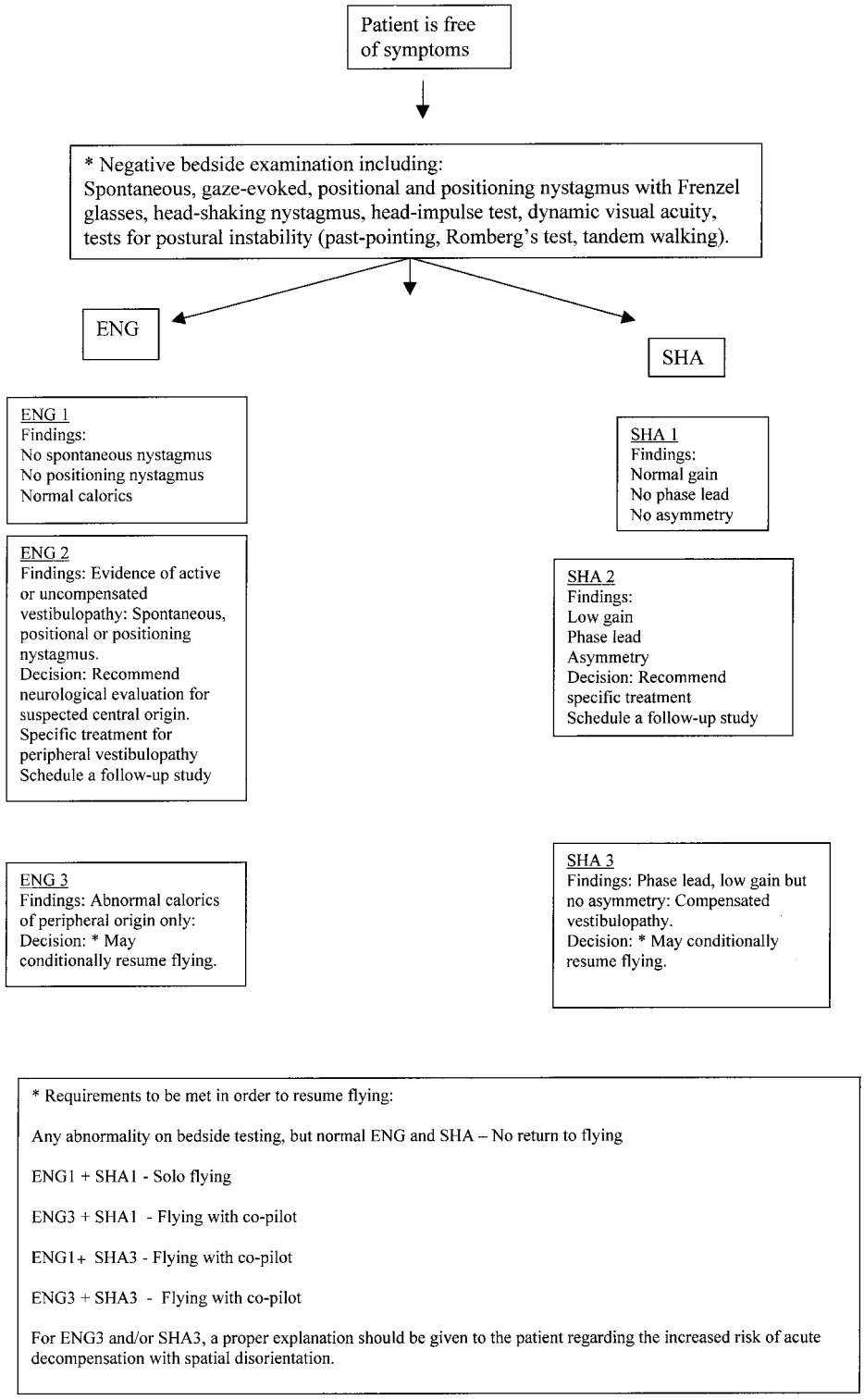

Fig. 1. Decision-making algorithm for patient's return to flying activities.

sation are satisfactory criteria for the resumption of full flying duties.[5–7,41]

We could find only a single case study describing the influence of vestibular neuronitis residua on spatial awareness in a student naval pilot, who had recurrent disorientation episodes during reduced visual conditions.[42] Although the patient failed a dynamic visual acuity test and the vestibular laboratory evaluation demonstrated bilateral vestibular dysfunction, he still passed a standard Romberg test, which is the only U.S. Navy aviation screening test for vestibular function.

The absence of vestibular symptomatology, with normal ocular movements, posture, and gait under terrestrial conditions, cannot be considered sufficient grounds for the

resumption of flying duties. Sensory substitution and vestibular compensation are context specific, depending on the accumulation of direction-specific information delivered through various sensors, and involve changes in the relative weighting of the various senses contributing to orientation.[15] Thus, acute decompensation is a continuous threat when the adapted vestibular system is exposed to unusual acceleration patterns, often in combination with insufficient ambient visual cues. Pilot fatigue and transmeridian flights interfering with circadian rhythms might further contribute to such decompensation.[43]

The evaluation of vestibular fitness for the high demands of flying is further complicated by the limitations of currently available vestibular laboratory tests. The properties of the vestibular reflexes tested in response to low-frequency accelerations by caloric stimulation and the rotating chair differ greatly from the level of performance required during the extreme acceleration and velocities encountered in flight. The commercially available technique for testing higher frequencies, by the patient actively generating head movements,[44] has inherent limitations secondary to the involvement of neck afferent input and the subject's ability to use predictive mechanisms to generate compensatory eye movements.[45] The instruments used to test dynamic vestibular responses under high-frequency, randomized accelerations are expensive research tools, capable of producing acceleration stimuli only in the range of daily locomotion and not for the prolonged, intensive stimuli experienced during flight.[46] The bedside tests of post–head-shake nystagmus and eye movements in response to head thrust, which might reveal dysfunction during vestibular system saturation and an inability to avoid retinal slip in response to high accelerations, provide only a partial solution to the lack of appropriate laboratory tests.[11,12,25] It is likely that many patients with vestibular symptoms have otolith function deficits. At present, most vestibular tests measure the dynamic function of the horizontal semicircular canal, whereas tests of other vestibular sensory regions are not yet available to the practicing otoneurologist.

Taking into account the high rate of long-term vestibular deficits and the limitations of currently available vestibular evaluation, on the one hand, and the potential hazard to flying safety, on the other, the physician's decision regarding the pilot's return to flying duties after an episode of vestibular neuronitis is not easy. We recommend that full solo flying privileges may be granted to the asymptomatic pilot only when apparent recovery is confirmed by bedside examination and the laboratory tests. We think that any abnormality revealed by the bedside examination reflects the vestibular system's inability to perform optimally, even under terrestrial conditions. Thus, a pilot may not resume flying responsibilities when minor abnormality is detected on bedside testing, even if subsequent EOG and SHA tests are normal. Flying with a copilot might be considered when a compensated residual vestibular deficiency is diagnosed by normal bedside examination, with no pathological nystagmus on the EOG and no asymmetry in the rotary chair test (Fig. 1). The suggested approach, whose purpose is to perform vestibular evaluation with dynamic loading of both low- and higher-acceleration frequencies, might expose vestibular deficiencies not reflected by the patient's complaints. However, current diagnostic capabilities are still limited because of hardware restrictions. These make it impossible to examine the patient while simulating real flight conditions, with the accompanying vestibular demands related to extreme linear and angular accelerations, and the conflict between the sensory systems that convey information on spatial orientation.

CONCLUSION

Vestibular deficiencies were found in the follow-up examinations of 11 of 18 pilots (61%) with vestibular neuronitis, despite the absence of symptoms. Follow-up bedside otoneurological testing revealed abnormal vestibular responses in only five patients, whereas disease was found by laboratory evaluation in a further six patients. These results emphasize the need to perform careful, comprehensive vestibular evaluation when considering the resumption of flying duties after an episode of vestibular neuronitis. Vestibular testing is currently limited by the technical difficulties involved in simulating the linear and angular accelerations, sensory deprivation, and sensory conflict experienced while flying an aircraft. The known contribution of spatial disorientation to serious flying accidents would appear to justify the development of laboratory equipment and the research effort required for such elaborate testing of the vestibular system.

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BIBLIOGRAPHY

1. Gillingham KK, Previc FH. Spatial orientation in flight. In: DeHart RL, ed. *Fundamentals of Aerospace Medicine,* 2nd ed. Baltimore: Williams & Wilkins, 1996:309–397.
2. Cheung B, Money K, Wright H, Bateman W. Spatial disorientation-implicated accidents in Canadian Forces, 1982–92. *Aviat Space Environ Med* 1995;66:579–585.
3. Kirkham WR, Collins WE, Grape PM, Simpson JM, Wallace TF. Spatial disorientation in general aviation accidents. *Aviat Space Environ Med* 1978;49:1080–1086.
4. Benson AJ. Spatial disorientation: common illusions. In: Ernsting J, Nicholson AN, Rainford DJ, eds. *Aviation Medicine,* 3rd ed. Oxford: Butterworth Heinemann, 1999:437–454.
5. Yarrington CT Jr, Hanna HH. Otolaryngology in aerospace medicine. In: DeHart RL, ed. *Fundamentals of Aerospace Medicine,* 2nd ed. Baltimore: Williams & Wilkins, 1996: 567–592.
6. Rayman RB. *Clinical Aviation Medicine,* 2nd ed. Philadelphia: Lea & Febiger, 1990:102–116.
7. O'Reilly BJ. Otorhinolaryngology. In: Ernsting J, Nicholson AN, Rainford DJ, eds. *Aviation Medicine,* 3rd ed. Oxford: Butterworth Heinemann, 1999:319–336.
8. Strupp M, Arbusow V. Acute vestibulopathy. *Curr Opin Neurol* 2001;14:11–20.
9. Curthoys IS, Halmagyi GM. Clinical changes in vestibular function with time after unilateral vestibular loss. In: Herdman SJ, ed. *Vestibular Rehabilitation,* 2nd ed. Philadelphia: FA Davis, 2000:172–194.
10. Dix MR, Hallpike CS. The pathology, symptomatology and

diagnosis of certain common disorders of the vestibular system. *Ann Otol Rhinol Laryngol* 1952;61:987–1016.
11. Hain TC, Fetter M, Zee DS. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. *Am J Otolaryngol* 1987;8:36–47.
12. Halmagyi GM, Curthoys IS. A clinical sign of canal paresis. *Arch Neurol* 1988;45:737–739.
13. Longridge NS, Mallinson AI. The dynamic illegible E (DIE) test: a simple technique for assessing the ability of the vestibulo-ocular reflex to overcome vestibular pathology. *J Otolaryngol* 1987;16:97–103.
14. Stockwell CW, Bojrab DI. Interpretation and usefulness of rotational testing. In: Jacobson GP, Newman CW, Kartush JM, eds. *Handbook of Balance Function Testing.* St. Louis: Mosby–Year Book, 1993:249–258.
15. Curthoys IS, Halmagyi GM. Vestibular compensation: a review of the oculomotor, neural, and clinical consequences of unilateral vestibular loss. *J Vestib Res* 1995;5:67–107.
16. Vidal PP, de Waele C, Vibert N, Muhlethaler M. Vestibular compensation revisited. *Otolaryngol Head Neck Surg* 1998; 119:34–42.
17. Bergenius J, Perols O. Vestibular neuritis: a follow-up study. *Acta Otolaryngol (Stockh)* 1999;119:895–899.
18. Imate Y, Sekitani T. Vestibular compensation in vestibular neuronitis: long-term follow-up evaluation. *Acta Otolaryngol (Stockh)* 1993;113:463–465.
19. Baloh RW, Honrubia V. *Clinical Neurophysiology of the Vestibular System,* 2nd ed. Philadelphia: FA Davis, 1990: 44–87.
20. Aantaa E, Virolainen E. Vestibular neuronitis: a follow-up study. *Acta Otorhinolaryngol Belg* 1979;33:401–404.
21. Okinaka Y, Sekitani T, Okazaki H, Miura M, Tahara T. Progress of caloric response of vestibular neuronitis. *Acta Otolaryngol (Stockh)* 1993;113(Suppl 503):18–22.
22. Matsuo T, Sekitani T. Vestibular neuronitis: neurotological findings and progress. *ORL J Otorhinolaryngol Relat Spec* 1985;47:199–206.
23. Taborelli G, Melagrana A, D'Agostino R, Tarantino V, Calevo MG. Vestibular neuronitis in children: study of medium and long term follow-up. *Int J Pediatr Otorhinolaryngol* 2000;54:117–121.
24. Brantberg K, Magnusson M. The dynamics of the vestibulo-ocular reflex in patients with vestibular neuritis. *Am J Otolaryngol* 1990;11:345–351.
25. Allum JHJ, Ledin T. Recovery of vestibulo-ocular reflex-function in subjects with an acute unilateral peripheral vestibular deficit. *J Vestib Res* 1999;9:135–144.
26. Shepard NT, Telian SA. *Practical Management of the Balance Disorder Patient*. San Diego: Singular Publishing Group, 1996:109–128.
27. Paydarfar JA, Goebel JA. Integrated clinical and laboratory vestibular evaluation. *Curr Opin Otolaryngol Head Neck Surg* 2000;8:363–368.
28. Allum JHJ, Yamane M, Pfaltz CR. Long-term modifications of vertical and horizontal vestibulo-ocular reflex dynamics in man, I: after acute unilateral peripheral vestibular paralysis. *Acta Otolaryngol (Stockh)* 1988;105:328–337.
29. Redfern MS, Yardley L, Bronstein AM. Visual influences on balance. *J Anxiety Disord* 2001;15:81–94.
30. Borger LL, Whitney SL, Redfern MS, Furman JM. The influence of dynamic visual environments on postural sway in the elderly. *J Vestib Res* 1999;9:197–205.
31. Dichgans J, Held R, Young LR, Brandt T. Moving visual scenes influence the apparent direction of gravity. *Science* 1972;178:1217–1219.
32. Bronstein AM, Yardley L, Moore AP, Cleeves L. Visually and posturally mediated tilt illusion in Parkinson's disease and in labyrinthine defective subjects. *Neurology* 1996;47: 651–656.
33. Redfern MS, Furman JM. Postural sway of patients with vestibular disorders during optic flow. *J Vestib Res* 1994; 4:221–230.
34. Guerraz M, Yardley L, Bertholon P, et al. Visual vertigo: symptom assessment, spatial orientation and postural control. *Brain* 2001;124:1646–1656.
35. Zheng Y, Horii A, Appleton I, Darlington CL, Smith PF. Damage to the vestibular inner ear causes long-term changes in neuronal nitric oxide synthase expression in the rat hippocampus. *Neuroscience* 2001;105:1–5.
36. Brandt T, Arnold F, Bles W, Kapteyn TS. The mechanism of physiological height vertigo, I: theoretical approach and psychophysics. *Acta Otolaryngol (Stockh)* 1980;89: 513–523.
37. Cho AA, Clark JB, Rupert AH. Visually triggered migraine headaches affect spatial orientation and balance in a helicopter pilot. *Aviat Space Environ Med* 1995;66:353–358.
38. Jacob RG, Lilienfeld SO, Furman JMR, Durrant JD, Turner SM. Panic disorder with vestibular dysfunction: further clinical observations and description of space and motion phobic stimuli. *J Anxiety Disord* 1989;3:117–130.
39. Bance ML, O'Driscoll M, Patel N, Ramsden RT. Vestibular disease unmasked by hyperventilation. *Laryngoscope* 1998;108:610–614.
40. Balaban CD, Thayer JF. Neurological bases for balance-anxiety links. *J Anxiety Disord* 2001;15:53–79.
41. Waiver guide topics. Naval Aerospace Medical Institute. Available at: http://www.nomi.med.navy.mil/Nami/WaiverGuideTopics/ent.htm. Accessed December 25, 2001.
42. Clark JB, Rupert AH. Spatial disorientation and dysfunction of orientation/equilibrium reflexes: aeromedical evaluation and considerations. *Aviat Space Environ Med* 1992;63: 914–918.
43. Katsarkas A, Segal BN. Unilateral loss of peripheral vestibular function in patients: degree of compensation and factors causing decompensation. *Otolaryngol Head Neck Surg* 1988;98:45–47.
44. O'Leary DP, Davis LL. High-frequency autorotational testing of the vestibulo-ocular reflex. *Neurol Clin* 1990;8:297–312.
45. Leigh RJ, Brandt T. A reevaluation of the vestibulo-ocular reflex: new ideas of its purpose, properties, neural substrate, and disorders. *Neurology* 1993;43:1288–1295.
46. Kaplan DM, Marais J, Ogawa T, Kraus M, Rutka JA, Bance ML. Does high-frequency pseudo-random rotational chair testing increase the diagnostic yield of the ENG caloric test in detecting bilateral vestibular loss in the dizzy patient? *Laryngoscope* 2001;111:959–963.