Case Report

Inferior vestibular neuritis in a fighter pilot: A case report

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Pilot spatial disorientation is a leading factor contributing to many fatal flying accidents. Spatial orientation is the product of integrative inputs from the proprioceptive, vestibular, and visual systems. Vestibular neuritis (VN) can lead to sudden pilot incapacitation in flight. VN is commonly diagnosed by demonstration of unilateral vestibular failure, as unilateral loss of caloric response. As this test reflects the function of the superior part of the vestibular nerve only, cases of pure inferior nerve neuritis will be lost. This paper describes a fighter pilot with symptoms suggestive of VN but with normal caloric test results. Further test showed unilateral loss of vestibular evoked myogenic potential. We believe that the pilot suffered from pure inferior nerve vestibular neuritis. VEMP plays a major role in the diagnosis of inferior nerve vestibular neuritis in pilots. Aeromedical concerns are also discussed.

Key words Vestibular neuritis, Vestibular evoked myogenic potential (VEMP), Spatial orientation, Case report

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 (23%) of these accidents [1]. 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 [2].

Spatial orientation is the product of integrative inputs from the proprioceptive, vestibular, and visual systems. The importance of normal function of all organs involved in orientation is emphasized in the aviation medical literature as being a prerequisite for flying. Sudden spontaneous unilateral loss of vestibular function with preserved hearing and no signs of brain stem dysfunction is generally attributed to viral infection and called acute vestibular neuritis (aVN). aVN can lead to sudden pilot incapacitation in flight. In routine clinical practice, vestibular integrity is tested by caloric irrigation and the horizontal head impulse tests. Both of these procedures test the horizontal semicircular canal. Cases of selective loss of inferior vestibular nerve function, i.e. inferior vestibular neuritis, will not be identified as such if vestibular tests are restricted to caloric tests. Patients who present with above described symptoms but normal caloric test results are commonly assumed to suffer from a lesion of the central nervous system [3]. The introduction of the vestibular evoked myogenic potential (VEMP) testing allows examination of saccular function, innervated by the inferior branch of the vestibular nerve.

We report one case of probable "inferior vestibular neuritis".

Case report

A previously healthy 45-year-old male, Chinese na-

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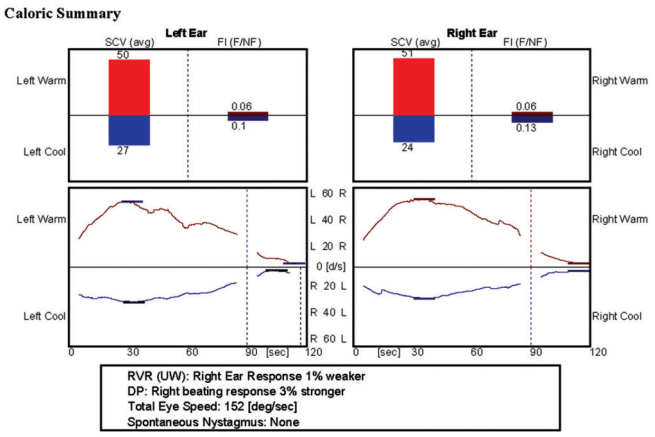

Fig.1 Alternate binaural bithermal caloric test show symmetrical responses on both sides.

val command pilot with close to 2000 logged flying hours presented with rotatory vertigo accompanied with nausea and vomiting. The symptoms had started five days earlier as lasting vertigo episodes of about twenty minutes. Clinical examination showed BP 130 / 80 and heart rate 90 beats / min. Otoscopy was normal. No spontaneous nystagmus in darkness. Neurological examination was unremarkable. One day later the patient reported minimal improvement, with continuing nausea and vertigo. Dix–Hallpike maneuver did not evoke nystagmus or accentuation of his vertigo. Horizontal head impulse, headshaking, vibration–induced nystagmus test with Frenzel goggles and subjective visual vertigo tests [4] were within normal limits. Hyperventilation testing was negative. ENG showed no spontaneous nystagmus. Caloric tests were normal (Fig.1). VEMP showed a normal amplitude response on the right, and loss of response on the left (Fig.2). Audiogram was normal. CT scan and MRI were normal.

He was temporarily grounded with symptomatic treatments, followed by a program of vestibular exercise.

On follow up at three months, his symptoms had disappeared completely. ENG showed no spontaneous nystagmus and normal caloric responses. Hallpikes maneuvre did not evoke nystagmus. A repeated VEMP test showed normal symmetrical responses (Fig.3). A recommendation of waiver for return to flying status in two–seat aircraft was issued, and he returned to flying status to fly with copilot six months after his episode of acute vertigo. A follow–up vestibular function evaluation 1 yr later showed no change in his condition, and he continued on flying status on a follow–up at 2 years. **Discussion**

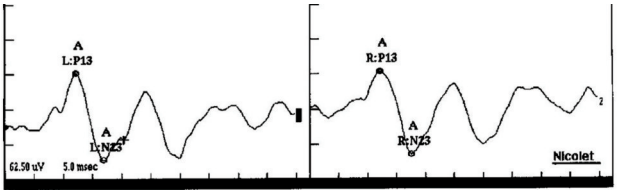

Fig.3 VEMP showed symmetrical responses on both sides on follow up at three months later

The symptoms in this case are suggestive of acute vestibulopathy or vestibular neuritis (VN). A common feature of vestibular neuritis is selective damage to the superior part of the vestibular labyrinth (horizontal and anterior semicircular canals and utricle) supplied by the superior division of the vestibular nerve, sparing the inferior part (posterior semicircular canal and saccule) supplied by the inferior division. Selective inflammation of the superior division of the vestibular nerve or anatomical differences in the bony canals of the two divisions, which make the inferior nerve less susceptible to entrapment during inflammatory swelling, may explain this difference in relative vulnerability. Hypofunction of the lateral semicircular canal, as shown by a loss or decrease of caloric response, is a common sign of vestibular neuritis [4–6]. VN that selectively affect the inferior nerve (iVN) is difficult to diagnose, owing to the lack of clinical tests for posterior canal or saccular function. It is possible that iVN is often wrongly classified as minor stroke or, more likely, remains undiagnosed. Although head impulse test with scleral coil technique can identi-

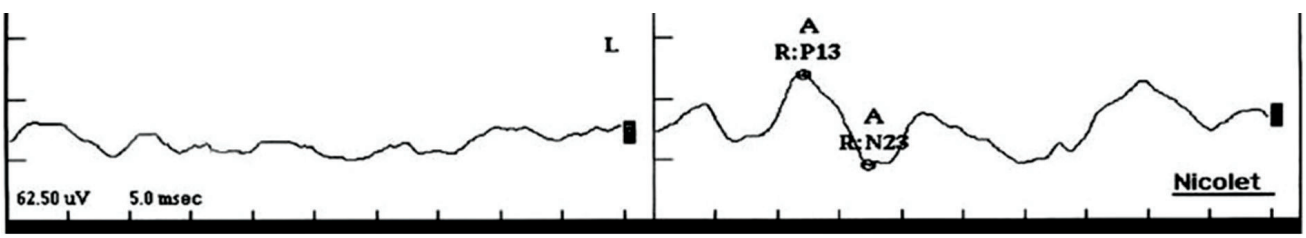

Fig.2 VEMP showed a normal amplitude response on the right, and loss of response on the left

fy loss of function of the posterior canal, this advanced technique is out of reach for most hospitals that treat acute vertigo patients. VEMP is a test of saccular function. It can be easily performed with routine equipment for evoked potentials.

Monstad et al has presented three patients with probable inferior nerve VN[7]. We believe that our fighter pilot patient also suffered from inferior nerve VN(iVN). Cerebrovascular lesions can not be completely ruled out in these cases. Even patients with symptoms and signs of classical vestibular neuritis may be suffering from CNS lesions, mainly vascular, in up to 15 % of the cases [8]. VEMP is an ipsilateral reflex, and cerebrovascular mechanisms for bilateral loss of VEMP are unlikely in cases with no other clinical findings indicative of a brainstem disorder. No lesions were found in the brainstem in our patient. Diffusion-weighted MRI was not available and this might reduce the sensitivity of MRI in the diagnosis of pontine lacunar strokes. Absent or reduced unilateral VEMP has been described in patients with MRI signs of lower brainstem lesions, whether cerebrovascular or inflammatory [9]. However, multiple sclerosis patients might suffer from loss of VEMP without obvious lesions in the brainstem that explain this finding [10], and this might be the case in cerebrovascular accidents as well. Loss of VEMP will probably demand a pontine or medullary lesion [8]. Our patient had no other findings or symptoms that suggest such CNS lesions. The symptoms, as well as the symptomatic recovery on follow up are suggestive of a peripheral vestibular lesion. VEMP is extremely sensitive to conductive hearing loss, in which cases air-conducted stimuli were attenuated and not sufficiently loud to elicit VEMP. Our patient had normal audiogram, and no middle ear pathology. Under these circumstances, VEMP is a fairly robust test of saccular function.

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.

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. 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 [1]. 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 [12]. 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 [12]. 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.

On follow up at three months later, our fighter pilit patients' symptoms had The complete disappearance of symptoms, normal caloric responses and VEMP at the 3 month follow up suggest a complete restoration of peripheral vestibular function. Since vestibular neuronitis is usually restricted to one attack, the recommendation was made for the patient to fly with a copilot.

However, 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 techniques for testing higher frequencies, by

the patient actively generating head movements, such as vestibular autorotation test (VAT), 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 [13].

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 [14]. It is likely that many patients have high-frequency vestibular-ocular reflex deficiency. The combination of head shaking test, smooth harmonic acceleration (SHA) test, caloric test and vibration induced nystagmus test [15] may detect these patients and will play an increasingly important role in comprehensive assessment of vestibular function for pilots.

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

Vestibular testing is currently limited by technical difficulties involved in simulating the linear and angular accelerations, sensory deprivation, and sensory conflicts experienced while flying an aircraft. The known contribution of spatial disorientation to serious flying accidents appears to justify the development of laboratory equipment and the research efforts required for such elaborate testing of the vestibular system.

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References

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 Kirkham WR, Collins WE, Grape PM, Simpson JM, Wallace TF. Spatial disorientation in general aviation accidents. Aviat Space Environ Med, 1978; 49:1080-1086.

3 Lee H, Sohn SI, Cho W, et al. Cerebellar infarction pres- enting as isolated vertigo. Neurology 2006; 67: 1178-1183.

4 Aw ST, Fetter M, Cremer PD, Karlberg M, Halmagyi GM: Individual semicircular canal function in superior and inferior vestibular neuritis. Neurology, 2001; 57(5): 768-774.

5 Ochi K, Ohashi T, Watanabe S. Vestibular-evoked myogenicpotential in patients with unilateral vestibular neuritis: abnormal VEMP and its recovery. J Laryngol Otol, 2003;117(2):104-108.

6 Goebel JA, O'Mara W, Gianoli G: Anatomical considerati- ons in vestibular neuritis. Otol Neurotol, 2001; 22(4):512-518.

7 Monstad P, Økstad S, Mygland A. Inferior vestibular neuritis: 3 cases with clinical features of acute vestibular neuritis, normal calorics but indications of saccular failure. BMC Neurol, 2006, 6:45.

8 Thomke F, Hopf HC: Pontine lesions mimicking acute peripheral vestibulopathy. J Neurol Neurosurg Psychiatry, 1999, 66 (3):340-349.

9 Itoh A, Kim YS, Yoshioka K, et al. Clinical study of vesti- bular evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. Acta Otolaryngol, 2001, 116-119.

10 Alpini D, Pugnetti L, Caputo D, et al. Vestibular evoked myogenic potentials in multiple sclerosis: Clinical and imaging correlations. Multiple sclerosis, 2004, 10: 316-321.

11 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.

12 Guerraz M, Yardley L, Bertholon P, et al. Visual vertigo: symptom assessment, spatial orientation and postural control. Brain, 2001, 124: 1646 - 1656.

13 O'Leary DP, Davis LL. High-frequency autorotational testing of the vestibulo-ocular reflex. Neurol Clin, 1990, 8: 297-312.

14 Hain TC, Fetter M, Zee DS. Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. Am J Otolaryngol, 1987, 8: 36-47.

15 Ohki M, Murofushi T, Nakahara H, et a1. Vibration—induced nystagmus in patients with vestibular disorders. Otolaryngol Head Neck Surg, 2003, 129: 255-258.

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