

Deafness associated with vertebrobasilar insufficiency

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Abstract

The incidence of auditory disturbances in vertebrobasilar insufficiency (VBI) is considered much rarer than vestibular disturbances, but no routine audiometric test results for VBI patients have been published. To determine the incidence of deafness associated with VBI and the sites predominantly involved in deafness, we studied 70 vertiginous patients with VBI using a routine audiological examination and magnetic resonance imaging (MRI). MRI detected a lacunar infarct involving the cochlear nuclei in one patient, but lacunae did not involve central auditory structures in the other patients. Twenty-five patients experienced tinnitus, deafness, or both, during vertigo episodes. Audiological examinations confirmed the development of unilateral deafness in 15 (21%) patients, involving the cochlea in 14 and cochlear nuclei in one. These findings indicate that hearing loss occurs in approximately one-fifth of VBI patients, much less frequently than vestibular disturbances, and that deafness associated with VBI mainly involves the cochlea. © 2001 Elsevier Science B.V. All rights reserved.

Keywords: Lacunar infarct; Deafness; Cochlear nuclei; Cochlea; Auditory brainstem response vertebrobasilar insufficiency

1. Introduction

Vertebrobasilar insufficiency (VBI) presents a complex varied clinical picture, vertigo being the most common symptom. Most early reports [1,2] excluded patients from the VBI category who had only vertigo unaccompanied by other neurologic deficits, whereas recent studies show that isolated episodes of vertigo often precede other VBI symptoms [3,4]. Because vestibular pathways are widely distributed in the hindbrain and the vestibular endorgan is supplied by vertebrobasilar circulation, in VBI the vestibular system may be affected at any level. This may explain why vertigo is most frequently manifested in VBI.

Auditory systems from the cochlea to central auditory connection in the brainstem also are supplied by vertebrobasilar circulation; therefore, deafness may occur in association with decreased blood flow, occlusive disease in the vertebrobasilar system, or both [1–3,5–12]. The incidence of auditory disturbances in VBI, however, is much

rarer than that of vestibular disturbances. Subjective loss of hearing has been reported in 0–24% of patients with VBI [1–3,5–8]. This is interesting because of anatomical proximity of the peripheral and central auditory and vestibular pathways, lack of collaterals to the labyrinthine artery (LA), and limited cochlear tolerance for ischemia reported in experimental and human studies [9–14]. Khurana et al. [15] suggested that unilateral or incomplete bilateral hearing loss might go unnoticed by patients and physicians and they recommended audiometric reevaluation of patients with VBI. Are auditory structures, including the cochlea, less susceptible to ischemia within vertebrobasilar circulation as compared to vestibular structures, or do some auditory disturbances, such as mild hearing loss, go unnoticed by patients and physicians? Another question is which parts of the auditory pathways are commonly involved in VBI. The cochlea, cochlear nerve, and central auditory connection in the brainstem are all supplied by vertebrobasilar circulation; therefore, auditory function can be compromised at any level in VBI.

In our study of the incidence of auditory disturbances in VBI, we routinely examined 70 patients with vertigo associated with VBI using pure-tone audiometry and auditory brainstem response (ABR). We also used auditory test batteries and magnetic resonance imaging (MRI) to inves-

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tigate whether deafness in VBI involves the cochlea or retrocochlear pathways.

2. Patients and methods

Records kept since 1991 at the neuro-otology clinic at Kameda General Hospital were reviewed. Seventy patients who fulfilled the following criteria were selected for study: (1) those referred for consultation because of vertigo, (2) those who had accompanying symptoms and signs suggestive of VBI, (3) those who had symptoms not related to definite etiologies, such as Meniere's disease [16], acoustic neuroma, and migraine-related vestibulopathy [17], and (4) those for whom pure-tone audiometry could be performed during episodes or within a few days after an episode. Because patients with VBI may have both vestibular and auditory symptoms as do patients with Meniere's disease, we used an operational definition of Meniere's disease proposed by Japanese Society for Equilibrium Research [16] to exclude Meniere's disease. Meniere's disease was diagnosed when patients had two or more definitive spontaneous episodes of vertigo several minutes or longer, repeated episodes of hearing loss commonly accompanied with aural fullness, and no evidence of other diseases causing vestibular and/or auditory symptoms. We also examined patients with electrocochleogram and glycerol test when Meniere's disease was suspected. The diagnosis of VBI was based on the patient's detailed clinical history and results of a neurological examination and MRI. In order to be accepted into the study with a diagnosis of VBI, repeated episodes of transient clinical symptoms referable to the vertebrobasilar arterial territory were required, such as transient visual disturbance, transient dysarthria, transient ataxia, drop attacks, or transient extremity weakness and numbness. The study group consisted of 35 men and 35 women, aged from 51 to 82 years (mean: 68 years). Thirteen of the 70 patients had hypertension, eight diabetes mellitus, seven hyperlipidemia, and five ischemic cardiovascular diseases.

Each patient's history, including prior medical records and tests, was obtained. All the patients had completed a standardized dizziness questionnaire, neuro-otological examinations, pure-tone audiometry, ABR and MRI. An experienced neurologist evaluated the neurologic findings for all the patients. When hearing thresholds differed by 10 dB or more at two or more frequencies between the right and left ears, hearing was regarded to be asymmetrical. The pure tone average (PTA) was obtained by averaging hearing thresholds at octave intervals from 500 to 4000 Hz. A PTA greater than 20 dB was regarded as indicative of hearing loss. Hearing loss was judged to occur in association with vertigo when patients were aware of such loss during vertigo episodes and when pure-tone audiometry showed an increased hearing threshold in the symptomatic ear. Additional auditory testing of those patients

who developed hearing loss included speech discrimination tests, Bekesy's audiometry, Fowler's alternate binaural loudness balance (ABLB) test, and stapedial reflex tests.

ABR testing was done inside an electrically shielded, sound-attenuating room. Data was recorded through silver disk electrodes, the differential active electrode being placed on the test ear mastoid and the reference electrode on the forehead. The opposite mastoid was grounded. The acoustic stimuli were alternating polarity clicks (0.1-ms rectangular pulse) with an intensity of 90 dB HL. The contralateral ear was masked with white noise of 70 dB HL. Two thousand sweeps were averaged with bandpass filters (150–3000 Hz) with a 6 dB/octave roll-off. Latencies of wave I, III, and V and the interpeak latencies I–III, III–V, and I–V were measured and compared with institutional normative data. An interpeak latency longer than the mean + two standard deviations was regarded as an indication of retrocochlear abnormalities. The amplitude ratio V/I was calculated and considered pathological if the value was less than one. Otological examinations, including inspection of the eardrum and evaluation of the cranial nerve functions, were performed. Vestibular laboratory testing that included electronystagmography (ENG), was performed to assess pathologic nystagmus (changes in fixation, gaze, and head position), vestibulo-ocular reflex function (caloric testing), and visual-ocular control (eye-tracking test, saccade test and optokinetic nystagmus test).

MRI was done with a superconducting 1.5-T MR system at a slice thickness of 4 mm, as reported elsewhere [4, 12]. Proton-density images (TR/TE = 3000 ms/15 ms) and T1-weighted (TR/TE = 600 ms/15 ms) and T2-weighted (TR/TE = 3000 ms/90 ms) images were obtained on both the axial and coronal planes. Two experienced radiologists who were blinded to the patients' clinical findings confirmed the presence of a lacunar infarct when a small distinct spot with a high-intensity signal on the T2-weighted images was found as an iso- to low-intensity spot on the T1-weighted images. Informed consent was obtained from all the participants after the nature of the procedures had been fully explained.

3. Results

3.1. Clinical features

The symptoms reported in association with vertigo are given in Table 1, the most frequent one being headache. Twenty-five patients (36%) had auditory symptoms during episodes of vertigo: hearing loss in four, tinnitus in 10, and both in 11. No patient was aware of bilateral hearing loss, although temporary bilateral tinnitus was noted by two. Twenty-two patients had previously experienced one or more isolated episodes of vertigo, 16 an attack(s) of ver-

Table 1
Associated symptoms in 70 vertiginous

Patients with vertebrobasilar insufficiency	
Symptoms	Number of patients
Headache	24
Tinnitus	21
Visual dysfunction	18
Hearing loss	15
Extremity weakness	12
Unsteadiness—incoordination	9
Extremity numbness	7
Drop attacks	5
Dysarthria	5
Loss of consciousness	4
Facial weakness	2
Hoarseness	1

tigo accompanied by other neurologic symptoms, and 14 a transient attack(s) of neurologic symptoms other than vertigo. The remaining 18 patients had not had any previous attacks, but had experienced repeated episode(s) during follow-up observations.

3.2. Magnetic resonance imaging findings

Almost all the patients ($n = 68$) had lacunae in the brainstem. No large infarcts were present in the hindbrain. Schematic drawings of the brainstem lacunae locations are

shown in Fig. 1. In the midbrain, the substantia nigra usually was involved. Lacunae in the pons were common in the central or medial region of the ventral portion. The medulla was involved in two patients; the cochlear nuclei in one and a site just anterior to the dorsal vagal nucleus in the other. No lesion involved the lateral lemniscus or trapezoid, nor the superior olivary or inferior colliculus nucleus.

3.3. Auditory and neuro-otological findings

Pure-tone audiometry detected the presence of unilateral sensorineural hearing loss ($PTA > 20$ dB) in the symptomatic ear of the 15 patients who were aware of hearing loss. In contrast, normal hearing or symmetrical slight high-tone hearing loss within normal limits for their ages was found in 10 patients who experienced only tinnitus during episodes. Hearing loss therefore is considered to have been manifested in 15 (21%) patients during VBI attacks. Hearing impairment was nearly total or profound ($PTA \geq 70$ dB) in five patients, moderate ($70 \text{ dB} > PTA \geq 40$ dB) in four, and mild ($40 \text{ dB} > PTA > 20$ dB) in six. Hearing loss was more frequent in patients with lacunae in the lower brainstem than in those with lacunae in the upper brainstem ($p < 0.05$, Table 2).

Of the 15 patients who developed hearing loss, central auditory system abnormalities, such as tone decay and

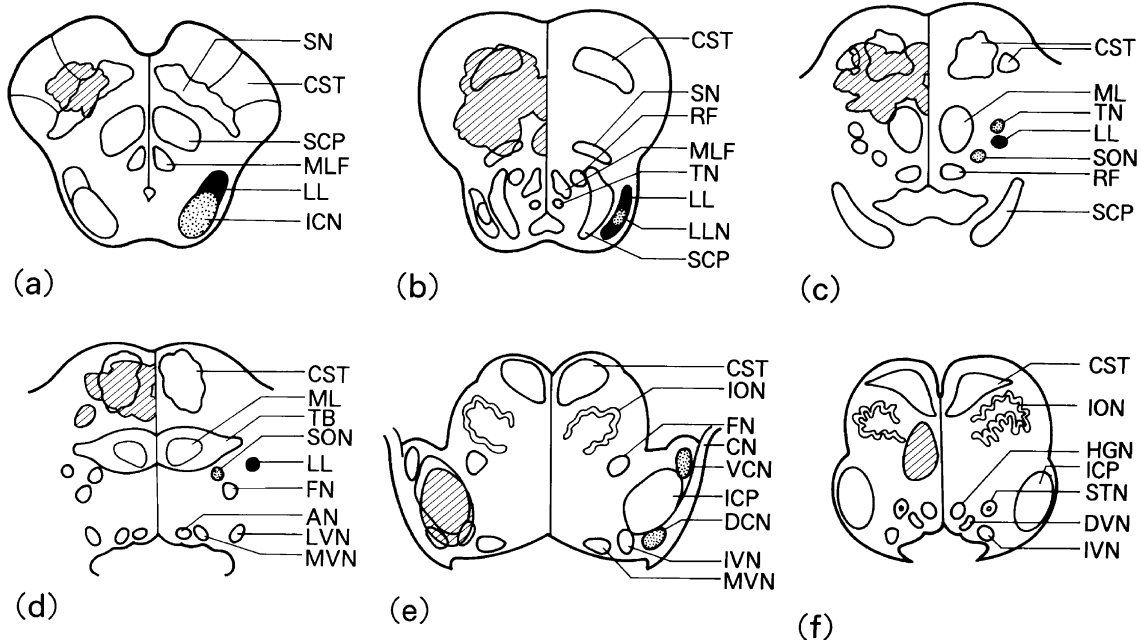


Fig. 1. Schematic drawings of brainstem lacunae locations. The region involved is plotted as an area shaded with oblique lines, all on the left side, without regard to the involved sides. (a) inferior mesencephalic level ($N = 24$); (b) upper pons level ($N = 38$); (c) mid pons level ($N = 50$); (d) lower pons level ($N = 18$); (e) pontomedullary junction level ($N = 1$); (f) upper medullary level ($N = 1$). CN: cochlear nerve; DCN: dorsal cochlear nucleus; ICN: inferior collicular nucleus; LL: lateral lemniscus; LLN: lateral lemniscus nucleus; SN: substantia nigra; SON: superior olivary nucleus; TB: trapezoid body; TN: trapezoid nucleus; VCN: ventral cochlear nucleus.

Table 2

Percentages of hearing loss, abnormal visual-ocular control and canal paresis. The upper brainstem includes the midbrain and upper pons; the lower brainstem the mid to lower pons and medulla. PT: pure-tone audiometry; ETT: eye tracking test; SAC: saccade test; OKN: optokinetic nystagmus test.

Paresis in 70 vertiginous patients with vertebrobasilar insufficiency

Location of lacunae	Incidence of abnormality (%)				
	PT	ETT	SAC	OKN	Caloric
Upper brainstem (<i>N</i> = 28)	11	21	7	11	39
Lower brainstem (<i>N</i> = 19)	31	63	32	37	63
Lower to upper brainstem (<i>N</i> = 23)	26	70	35	57	52
Total (<i>N</i> = 70)	21	51	23	33	50

poor speech recognition, were detected only in one, who had a lacunar infarct involving the cochlear nuclei (case 1, discussed later). Hearing loss was considered to involve the cochlea in 14 patients based on findings of the Bekesy's audiometry, ABLB, and stapedial reflex tests. In these patients, speech recognition scores corresponded well with the increased hearing thresholds, and no tone decay was found. The ABR examination detected no abnormal wave-

forms indicative of retrocochlear lesions, such as prolonged interpeak latencies or a decreased wave V/I ratio. Pure-tone audiometry showed that hearing was symmetrical in the 55 patients who did not develop hearing loss. Eighteen patients had normal hearing, 35 symmetrical high-tone loss within normal limits for their age, and two had symmetrical mid-to-high tone loss, probably due to chronic noise exposure. ABR detected no apparent abnormalities in 50 of the patients, and in five a slight delay in all the waves due to hearing loss at high frequencies. Interpeak latencies and wave V/I ratios were all within the normal range.

Otological physical examinations did not find either abnormality in the eardrum nor cranial nerve palsy. Vestibular examinations showed that the eye-tracking test was the most sensitive for detecting central vestibular abnormalities caused by brainstem lacunae, as reported previously [4]. The incidence of abnormal visual-ocular control was higher when lacunae were present in the lower rather than upper brainstem. Canal paresis was present in half the patients, the incidence being high when lacunae involved the lower rather than the upper brainstem (Table 2). Patients who developed unilateral hearing loss during VBI episodes commonly had a decreased caloric response in the involved ear.

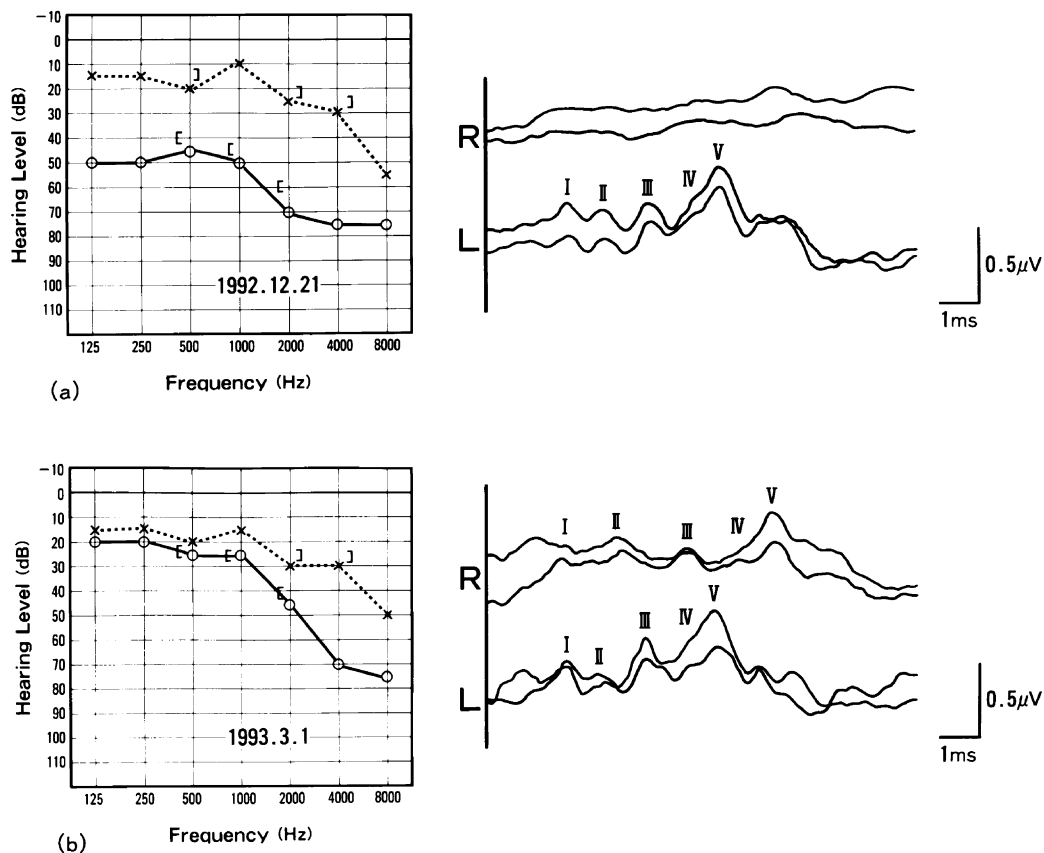


Fig. 2. Auditory findings in Case 1. (a) Moderate sensorineural hearing loss and absence of response on ABR testing were found at the patient's first visit. (b) Partial recovery of hearing and prolonged interpeak latencies between waves I–III and I–V occurred 2 months after the onset of these symptoms.

4. Representative cases

4.1. Case 1. Patient with retrocochlear-type hearing loss

A 51-year-old man under treatment for diabetes mellitus was admitted to the hospital because of a 4-day history of hearing loss and tinnitus in the right ear, vertigo, double vision, and dysarthria. The Romberg sign, ataxic gait, right-sided dysmetria, hypoesthesia in the right external ear canal, and clockwise rotatory spontaneous nystagmus were all positive. Pure-tone audiometry detected moderate sensorineural hearing loss in the right ear, in which Bekesy's audiometry showed tone decay. The recruitment phenomenon was absent on ABLB, and no stapedial reflex was elicited. Speech discrimination scores were 25% on the right and 90% on the left. In the ABR examination, no responses were evoked by stimulation on the right, whereas there were normal waveform responses on the left (Fig. 2). An ENG examination showed saccadic pursuit and dysmetria, and a decrease in caloric response on the right. MRI showed a lacunar infarct involving the right cochlear nuclei, as well as another in the central portion of the left mid pons (Fig. 3).

Hearing impairment gradually lessened, as did his other neurologic symptoms. Pure-tone audiometry performed 2 months later showed partial improvement of hearing on the

right side. ABR detected waves on the right side, which had prolonged interpeak latencies between waves I–III and I–V (Fig. 2). During the next 2 years, he had two additional episodes, in which he experienced tinnitus but no hearing loss. Pure-tone audiometry, performed 2 years later, detected no hearing deterioration in either ear.

4.2. Case 2. Patient with cochlear-type hearing loss

A 74-year-old under treatment for hypertension visited our outpatient clinic complaining of rotatory vertigo and occipital headache, which had occurred three times over a 2-week period, but which subsided within several hours of each attack. During the first and second episodes, she also had experienced transient visual dysfunction. Pure-tone audiometry performed 6 months earlier had shown slight symmetrical high-tone hearing loss, probably due to aging. During the last episode, she became aware of persistent tinnitus on the right, and pure-tone audiometry showed deterioration of hearing in the right ear. Results of stapedial reflex testing and Bekesy's audiometry indicated that her hearing loss involved the cochlea. Speech discrimination scores were 70% on the right and 80% on the left. Normal waveform responses were evoked bilaterally on ABR testing (Fig. 4). An ENG examination showed spon-

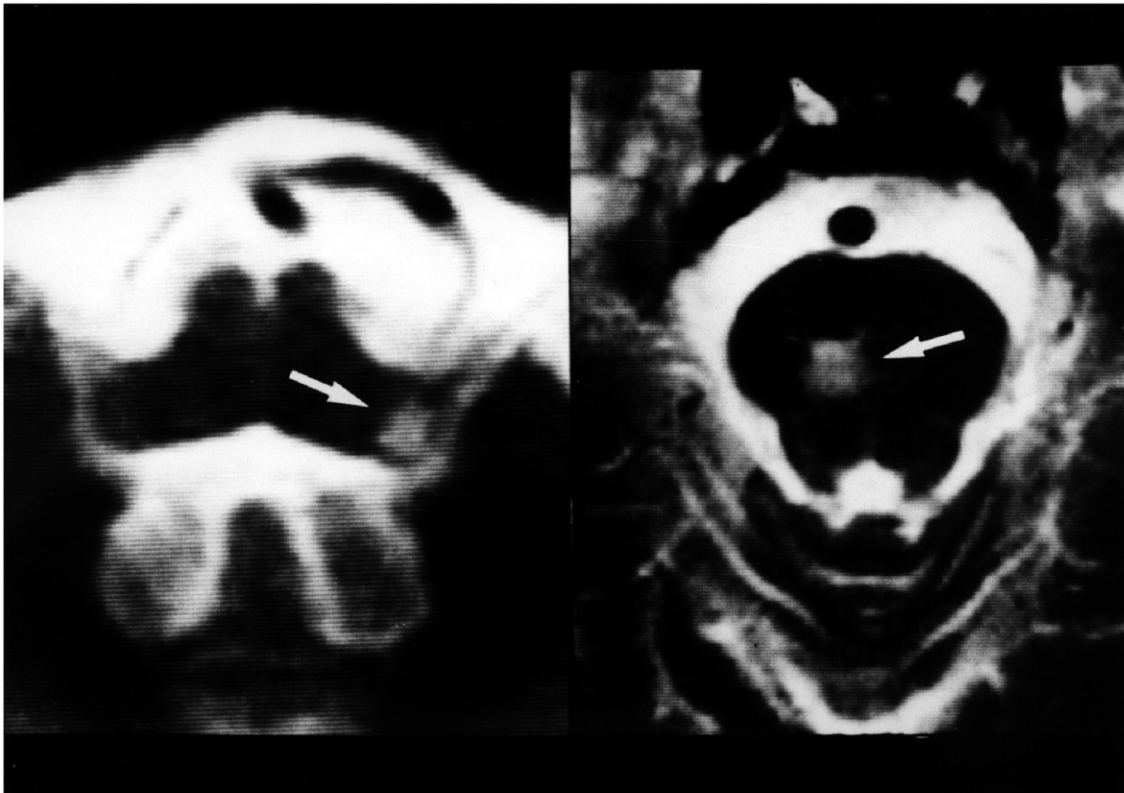


Fig. 3. Magnetic resonance images (T2-weighted images) in Case 1 showing lacunae involving the right cochlear nuclei (left) and central portion of the left mid pons (right).

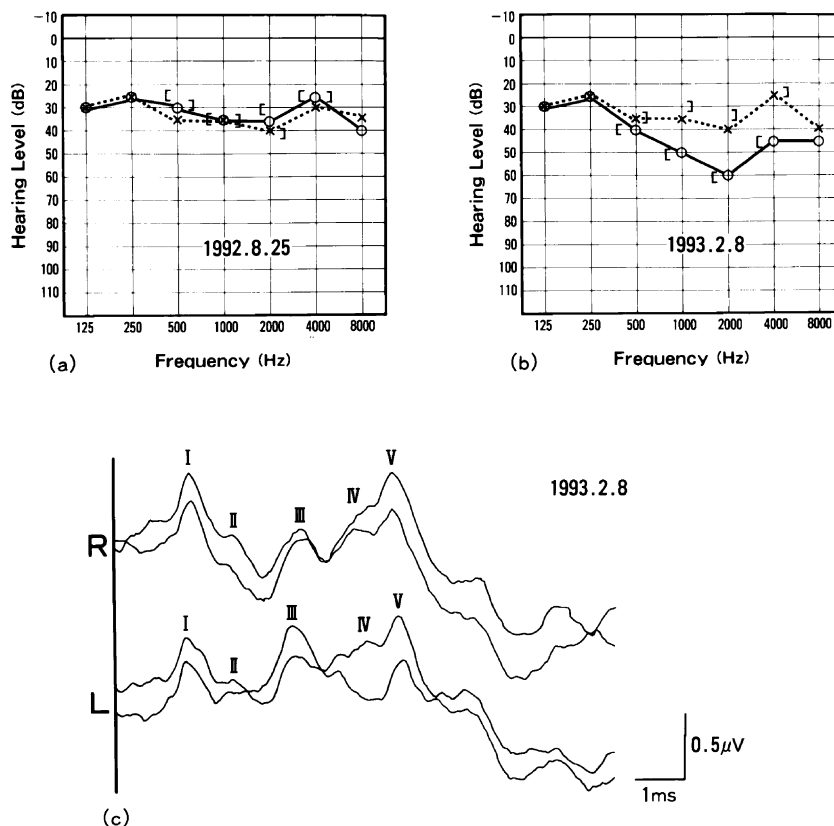


Fig. 4. Auditory findings in Case 2. (a) Mild sensorineural hearing loss in the bilateral ears was diagnosed 6 months before the onset of other symptoms. (b) Deterioration of hearing in the right ear occurred during an episode of vertigo. (c) Normal waveform responses were evoked on ABR testing.

taneous nystagmus to the right, saccadic pursuit, and a decrease in caloric response on the right. MRI detected a lacunar infarct in the central region of the ventral portion of the left mid pons. None of the auditory structures in the brainstem was involved.

5. Discussion

This study showed that 25 (36%) of 70 patients with vertigo associated with VBI were aware of hearing loss, tinnitus, or both, during the episodes. Routine pure-tone audiometric examinations showed that unilateral hearing loss occurred during these episodes in 15 (21%) patients. Of the 55 patients who did not report hearing loss during episodes, normal hearing was present in 18, symmetrical high-tone loss within normal limits for their ages in 35, and symmetrical mid-to-high tone loss due to chronic noise exposure in two. Overall, there was hearing impairment in 52 patients, but hearing loss developed during episodes of VBI in only 15 patients (21%). It is possible that hearing loss in these patients was incidental in this aging population, but they did notice hearing loss during episodes of VBI. Thus, we consider that the hearing loss in the 15 patients was associated with VBI. This incidence of deafness does not differ much from incidences reported

(0–24%) in previous studies [1–3,5–8], in which no routine audiological examination was performed. Unilateral or incomplete bilateral hearing loss therefore is not likely to frequently go unnoticed by patients and physicians. Indeed, deafness is considered to occur only occasionally in VBI.

Audiological examinations, including ABR, showed that the cochlea was involved in 14 of the 15 patients who developed hearing loss. Retrocochlear hearing loss was found in only one patient, in whom a lacunar infarct directly involved the cochlear nuclei. MRI showed no obvious signs of involvement of central auditory structures in the brainstem of the other patients. No central auditory abnormalities were detected in any other patients; therefore, unless lacunae directly involve central auditory structures, retrocochlear deafness may not occur. Pathologic changes in ABR have been observed during surgical and thrombotic occlusion of the basilar artery (BA) in patients with severe neurologic deficits [18,19]. None of our patients had such deficits. Faught and Oh [20] reported that patients with small midline pontine infarcts rarely show a pathologic ABR, whereas those with Horner's sign commonly have an abnormal ABR because of the proximity of the sympathetic tracts to the auditory pathways. In our study, lacunar infarcts commonly involved the central or medial portion of the ventral pons or substantia nigra in the midbrain, far from the auditory pathways. Horner's

sign was not present in our patients. These findings may explain why only one patient had abnormal ABR waveforms indicative of retrocochlear lesions.

Hearing loss was more frequent when the lacunae involved the lower rather than the upper brainstem. Similarly, a decrease in caloric response, indicative of peripheral vestibular system damage, predominated in patients with lower brainstem lacunae. The labyrinthine artery (LA) usually originates from the anterior inferior cerebellar artery (AICA) and occasionally from the posterior inferior cerebellar artery (PICA) that arises from a rostral vertebral artery (VA) or caudal BA. The AICA may arise on either side of BA, anywhere from the caudal part to the middle of the pons [21]. Lacunae may result from occlusion of the small penetrating branches that arise proximally from major cerebral arteries, including the BA and VA [22]. The presence of lacunae in the lower brainstem suggests atherosclerotic changes in the caudal BA or rostral VA. These lesions may more readily cause circulatory disturbance in the LA, resulting in inner ear ischemia. This may explain why deafness and canal paresis were more frequent in the patients with lacunae in the lower rather than in the upper brainstem.

The LA has two branches: a large common cochlear artery that supplies the cochlea and the inferior part of the vestibular labyrinth, and a small anterior vestibular artery that supplies the superior part of the vestibular labyrinth. Anastomoses exist between these two arteries at the level of the inferior but not the superior labyrinth. There is no collateral circulation to the membranous labyrinth from the otic capsule [22]. The superior vestibular labyrinth therefore is considered more susceptible to ischemia than the cochlea because of the small diameter of the anterior vestibular artery and the lack of collaterals [3]. Because deafness in VBI commonly involves the cochlea, this may also explain the rarer incidence of deafness compared to vestibular disturbance in VBI.

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