Sensorineural Hearing Loss Due to Vertebrobasilar Artery Ischemia – Illustrative Case and Literature Review
Masafumi Ohki*
Department of Otolaryngology, Saitama Medical Center, Japan

Abstract
Acute sensorineural hearing loss is commonly caused by peripheral vestibulocochlear disorders such as sudden deafness, Meniere’s disease, and Ramsay Hunt syndrome, but is rarely due to infarction of the vertebrobasilar artery. In this report, a case of right anterior inferior cerebellar artery syndrome presenting with sudden deafness and vertigo is described in order to feature acute sensorineural hearing loss due to vertebrobasilar artery ischemia, and sensorineural hearing loss due to vertebrobasilar artery ischemia is reviewed and discussed. A 79-year-old man presented with right acute sensorineural hearing loss preceded by occasional, minute-long periods of dizziness without cranial neural symptoms other than vestibulocochlear symptoms. Magnetic resonance imaging (MRI) revealed infarction of the right anterior inferior cerebellar artery territory. The vertebrobasilar artery supplies the vestibulocochlear organ, brainstem, and cerebellum, whose abnormalities are related to vestibulocochlear symptoms. Vertigo is a major symptom associated with vertebrobasilar artery ischemia. Further, acute sensorineural hearing loss is caused by hypoperfusion of the vertebrobasilar artery. Vertigo and/or acute sensorineural hearing loss could be a prodrome of subsequent infarction of the vertebrobasilar artery territory. The artery most often responsible for acute sensorineural hearing loss is the anterior inferior cerebellar artery, whereas ischemia of the basilar artery, the posterior inferior cerebellar artery, and the superior cerebellar artery rarely cause acute sensorineural hearing loss. Patients with acute sensorineural hearing loss who are at a high risk of cerebrovascular disease must be examined with imaging tools such as MRI.

Keywords: Basilar artery; Hearing loss; Vestibular; AICA; Cerebellar; PICA

Illustrative Case
A 79-year-old man had experienced occasional, minute-long periods of dizziness for 8 months. He presented with sudden onset right hearing loss, vertigo, nausea, and vomiting. Pure-tone audiometry showed profound sensorineural hearing loss on the right (Figure 1A). He exhibited left-beating gaze and positional nystagmus with a counterclockwise component, and the stepping test revealed a deviation reaction toward the right side. The finger to nose, knee to shin, and hand pronation supination tests did not show cerebellar disturbance. He did not exhibit any cranial nerve symptoms other than vestibulocochlear symptoms. A routine brain computerized tomography scan, which was performed to rule out cerebral hemorrhage and extensive cerebral infarction, was normal. He was first diagnosed with right-sided sudden deafness. However, he demonstrated right-beating nystagmus when gazing rightward, left-beating nystagmus when gazing straight forward and leftward, and direction–change positional nystagmus 7 days later. Magnetic resonance imaging (MRI) of the brain was performed and a T1-weighted MRI showed high signal intensity in the right anterior inferior cerebellum, including part of the right tonsil (Figure 2). A T1-weighted MRI showed low signal intensity in the same area. An electronystagmogram revealed right-sided canal paresis of 83% by the caloric response and loss of visual suppression by left-sided water stimulation. He was diagnosed with right acute sensorineural hearing loss due to vertebrobasilar artery ischemia.

*Corresponding author: Masafumi Ohki, Department of Otolaryngology, Saitama Medical Center, Japan, Tel: +81-49-228-3685; Fax: +81-49-225-6312; E-mail: m-ohki@umin.ac.jp
Received March 04, 2013; Accepted May 08, 2013; Published May 15, 2013
Citation: Ohki M (2013) Sensorineural Hearing Loss Due to Vertebrobasilar Artery Ischemia—Illustrative Case and Literature Review. J Neurol Neurophysiol S8: 005. doi:10.4172/2155-9562.S8-005
Copyright: © 2013 Ohki M. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.
loss due to right AICA syndrome. An electrocardiogram revealed atrial fibrillation. He was treated with heparin sodium followed by ticlopidine hydrochloride and warfarin potassium. Pure-tone audiometry showed that his hearing levels on the right side had recovered to the same level as that on the opposite side 1 month later (Figure 1B). An MRI showed improvement of the ischemic area 2 months later.

**Review of the literature**

Previous cases presenting with sensorineural hearing loss without cranial nerve deficits in vertebrobasilar artery ischemia are shown in (Table 1) [4–29]. Sensorineural hearing loss due to AICA syndrome is usually ipsilateral (88%) [6–19], whereas sensorineural hearing loss due to ischemia in the territory of the PICA is also ipsilateral but very rare [20,30]. Although SCA syndrome does not usually lead to hearing loss, contralateral hearing loss has been rarely reported [23,31]. Basilar artery occlusion rarely results in hearing loss, and hearing loss is usually bilateral in these cases [4,5,24–29,32]. Hearing loss has been demonstrated in 0–31% of patients with vertebrobasilar artery insufficiency [33–40].

**Discussion**

**Vertebrobasilar artery anatomy**

The internal auditory artery, which supplies the cochlea, usually originates from the AICA and occasionally from the PICA ramifying from the rostral vertebral artery (VA) or the caudal basilar artery (BA) [41]. A major anastomosis between the AICA and the PICA is a common variant [42]. The AICA can arise from either side of the caudal basilar artery (BA) [33]. Many variations exist regarding from where the AICA and PICA arise from the vertebrobasilar artery, and the branches of the AICA have various multiple anastomoses from the lateral medullary artery. However, the inner ear artery is the final artery. The pontes consist of anterior, lateral, and posterior parts, and the BA, SCA, and the AICA irrigate these territories [43]. The anteromedial and anterolateral pontine arteries, which arise from the BA, supply the anterior part of the pons, whereas the lateral pontine artery, which arises from the BA and AICA branches, supplies the lateral pons. The lateral pontine artery territory contains the lateral lemniscus, the superior olive, and the lateral sides of the corticospinal tract. The AICA branches supply areas containing the superior vestibular nucleus and the lateral lemniscus. The posterior pons is irrigated by the SCA branch [43]. Typically, the AICA branches from the first or middle third of the BA and first passes the lateral pons, and then the rostralateral artery and the caudomedial artery. The internal auditory artery, which is a terminal artery, originates from the superior lateral branch of the AICA and perfuses the inner ear. The superior lateral branch sends branches out to the adjacent pons and extends to the flocculus and anterior inferior cerebellum. The inferior medial branch of the AICA perfuses the lateral pons and extends to the flocculus. The superior lateral branch and inferior medial branch often anastomose the PICA [44].

The internal auditory artery bifurcates the common cochlear artery and the anterior vestibular artery; subsequently, the common cochlear artery bifurcates the main cochlear artery and the vestibulocochlear artery, which is divided into the posterior vestibular artery and the cochlear ramus [8,41,45,46]. The common cochlear artery perfuses the apical three-fourths of the cochlea and the cochlear ramus perfuses the basal area on the final fourth of the cochlea. The anterior vestibular artery perfuses the utricle, which is the superior part of the saccule, and the anterior and horizontal semicircular canals. The inferior part of the saccule and the posterior semicircular canal are perfused by the posterior vestibular artery [8,45,46].

**Hearing loss originating from infarction of the AICA territory**

The AICA supplies the caudolateral pons, which includes the vestibular nucleus, cochlear nucleus, superior and inferior vestibular nerves, and the cochlear nerve. Infarction in the territory of the AICA is called AICA syndrome [47–53]. The major symptoms are vertigo,
sensorineural hearing loss, it very rarely results in retrocochlear hearing loss [23,31]. The impaired auditory pathway is presumably the lateral lemniscus. The auditory pathway projects from the cochlea to the auditory cortex via the cochlear nerve, cochlear nucleus, superior olivary nucleus, lateral lemniscus of the pons, and inferior colliculus. The main pathway crosses the brainstem at the level of the superior olivary nucleus, and then ascends toward the auditory cortex via the lateral lemniscus of the pons and inferior colliculus. Therefore, impairment of the lateral lemniscus leads to contralateral sensorineural hearing loss [23,31,65].

**Hearing loss due to basilar artery occlusion**

Occlusion of the basilar artery is a lethal condition, with a mortality rate ranging from 40% to 86% [22]. Vertigo, nausea, headache, and motor and oculomotor deficits are the common symptoms [66]. Basilar artery occlusion rarely results in hearing loss [4,5,25–29,32,66], and the reported cases of hearing loss due to basilar artery occlusion are generally bilateral [4,5,24–29]. Unilateral hearing loss due to basilar artery occlusion is very rare [29,32], and these patients generally have ischemic lesions within the cerebellum or the cerebellar artery territories, such as the AICA or PICA, as revealed by MRI [5,24–28]. However, 1 case with unilateral vestibulocochlear symptoms, including unilateral sensorineural hearing loss due to basilar artery occlusion, did not show ischemic lesions in the AICA territory, PICA territory, or cerebellum by T1-, T2-, and diffusion-weighted MRI [25]. In this case, MRI revealed an ischemic lesion only within the pontine artery territory [25]. In contrast to vertebrobasilar insufficiency (VBI), BA occlusion usually leads to more extensive ischemic lesions and causes bilateral hearing loss [4,5,24–26,28] or unilateral hearing loss [29]. Temporal bone histopathology in patients with occlusion of the VA or BA showed degenerative changes in the unilateral labyrinth and vestibulocochlear nerve [32]. Therefore, hearing loss associated with BA occlusion may be due to degenerative changes in the unilateral labyrinth and vestibulocochlear nerve. The brainstem area or the cerebellum usually is impaired because of BA occlusion. The auditory pathway projects from the cochlea to the auditory cortex via the cochlear nerve, and continues to the cochlear nucleus, superior olivary nucleus, and lateral lemniscus of the pons and inferior colliculus. Hypoperfusion in the territory of the BA may impair the central pathway in addition to causing peripheral sensorineural hearing loss.

**Hearing loss due to vertebrobasilar artery insufficiency**

Because the internal auditory artery is the final artery, the cochlea is considered susceptible to ischemia due to VBI and occlusive disease [8,32,67–70]. VBI is sometimes associated with hearing loss and/or tinnitus [33–40,62,71,72]. Subjective hearing loss has been reported in 0–31% of patients with VBI [33–40], whereas unilateral hearing loss occurs in 7% of patients and bilateral hearing loss is rare (0.5%) [34,40,71]. Yamasoba et al. reported that the symptoms associated with VBI include headaches, tinnitus, visual dysfunction, hearing loss, extremity weakness, unsteadiness, extremity numbness, drop attacks, dysarthria, loss of consciousness, facial weakness, and hoarseness. Cochlear symptoms such as hearing loss and/or tinnitus were found in 36% of VBI patients, with tinnitus and hearing loss reported in 30% and 21% of these patients, respectively [33]. Ischemic lesions associated with hearing loss involve, in order of descending frequency, the AICA territory, the PICA territory, and the brainstem territory [71]. Audiological examination indicated that the major involved site was the cochlea and that retrocochlear hearing loss was minor [33,34]. In the case of cochlear hearing loss, the auditory brainstem response was generally normal or the absolute latencies of
all waves were delayed, but the interpeak latencies of waves I-III-V were normal [33,34,62]. The interpeak latencies of waves I-III-V are prolonged in patients with retrocochlear hearing loss [33,34,62]. Retrocochlear hearing loss is associated with ischemic lesions that involve the central auditory pathway [34,57,62,71,73-77], and episodes of vertigo are the most frequent (62%), and often the initial (19–48%), symptom [36,78]. The caloric response is decreased or is absent in 20–86% [34,79]. Hypoperfusion to the vestibular labyrinth probably disturbs the vestibular function. The following cerebellar lobules have oculomotor functions: the vermis lobules VI and VII, the Crus I and II of the ansiform lobule, the hemisphere of the simplex lobule, dorsal paraflocculus, ventral paraflocculus, flocculus, uvula, nodulus, caudal dentate nucleus, lateral posterior interposed nucleus, and caudal pole of the fastigial nucleus [80–88]. The flocculus, uvula, and nodule are connected to the vestibular nuclei [82, 87–91], and the purkinje cells of the cerebellum project inhibitory fibers to the vestibular nuclei and cerebellar nuclei [82,87,91]. Disturbances in these areas sometimes cause vertigo and dizziness, which are similar symptoms to those of acute labyrinthine dysfunction.

**Features of prodromal vestibulocochlear symptoms**

Infarction in the territory of the AICA rarely presents with symptoms that are similar to those of pure peripheral vestibulocochlear disorders such as sudden deafness. The case presented in this study is similar to pure peripheral vestibulocochlear disorders because clear cranial nerve symptoms other than vestibulocochlear symptoms were

| Infarction of the AICA territory | Side of HL | Initial Symptoms | Risk Factors |
|---------------------------------|------------|-----------------|-------------|
| Biavati et al. [6]              | Ipsilateral| Recurrent vertigo|             |
| Deplanque et al. [7]            | Bilateral  | HL and vertigo  |             |
| Kim et al. [8]                  | Ipsilateral| Recurrent vertigo|             |
| Lee et al. [9]                  | Bilateral  | HL and vertigo  | DM          |
| Lee et al. [10]                 | Ipsilateral| HL and vertigo  |             |
| Lee et al. [11]                 | Ipsilateral| Recurrent tinnitus and HL |    |
| Lee et al. [12]                 | Bilateral  | Recurrent HL and vertigo | DM, HT |
| Lee et al. [13]                 | Ipsilateral| HL and vertigo  | HT          |
| Yi et al. [14]                  | Ipsilateral| HL and vertigo  | HT          |
| Murakami et al. [15]            | Ipsilateral| HL, tinnitus and vertigo | HT    |
| Son et al. [16]                 | Ipsilateral| HL and dizziness | DM, HT |
| Kim et al. [17]                 | Ipsilateral| HL and vertigo  | AF, old MI  |
| Lee et al. [18]                 | Ipsilateral| HL and vertigo  | HT          |
| Ikegami-Takada et al. [19]      | Ipsilateral| HL and vertigo  | HT, DM, Hypercholesterolemia |
| Ohki et al. [presented in this case] | Ipsilateral| Recurrent vertigo |             |
| Infarction of the PICA territory | Ipsilateral| HL and tinnitus |             |
| Kanzakin et al. [20]            | Ipsilateral| HL and tinnitus |             |
| Infarction of the AICA+PICA territory | Ipsilateral| Recurrent vertigo |         |
| Kido et al. [21]                | Ipsilateral| HL and vertigo  |             |
| Chiang et al. [22]              | Bilateral  | HL and vertigo  |             |
| Infarction of the SCA territory  | Contralateral| HL               | Hyperglycaemia, HT |
| Murakami et al. [23]            | Ipsilateral| HL, tinnitus and vertigo |             |
| Huang et al. [4]                | Bilateral  | HL, tinnitus and dizziness |             |
| Sunose H et al. [24]            | Bilateral  | HL, tinnitus and vertigo |             |
| Toyoda K et al. [25]            | Bilateral  | HL, dizziness    | HT          |
| Bilateral  | HL and vertigo  | HT, DM, Hypercholesterolemia |
| Bilateral  | HL and vertigo  |             |
| Sauvaget E et al. [5]           | Bilateral  | Vertigo         | Smoking     |
| Jung J et al. [26]              | Bilateral  | HL, tinnitus and recurrent vertigo | HT |
| Huang CC et al. [27]            | Bilateral  | Vertigo         |             |
| Bovo R et al. [28]              | Bilateral  | HL and vertigo  | HT, cerebrovascular disorders |
| Ohki M et al. [29]              | Ipsilateral| HL, tinnitus and vertigo |             |

**Table 1: Sensorineural hearing loss without cranial nerve deficits in vertebrobasilar artery ischemia.**

HL: Hearing Loss, DM: diabetes mellitus, HT: hypertension, MI: myocardial infarction, Af: atrial fibrillation
absent. However, acute sensorineural hearing loss in this case of AICA syndrome was preceded by episodes of recurrent minute-long dizziness. The feature of prodromal recurrent minute-long dizziness was different from the characteristics of typical peripheral vestibulococlear disorders such as Meniere’s disease, vestibular neuritis, and sudden deafness with vertigo because vertigo is characteristically hour- or day-long for Meniere’s disease and day- or week-long for vestibular neuritis and sudden deafness. The prodromal recurrent minute-long vertigo of vertebrobasilar artery ischemia is concomitant to the feature of TIA. Recurrent attacks of vertigo and/or dizziness and/or tinnitus preceded acute sensorineural hearing loss in 31% of previous reports, and vestibular symptoms such as vertigo or dizziness accompanied or preceded acute sensorineural hearing loss in 87% of previous reports (Table 1). Only 5 cases presented with auditory disturbance without vestibular symptoms. Further, previous reports showed that patients with vertebrobasilar artery ischemia sometimes demonstrate only inner ear dysfunction. The inner ear is susceptible to ischemia because the internal auditory artery, which is the artery of the inner ear, is the terminal artery. Therefore, hypoperfusion of the vertebrobasilar artery is one of the factors responsible for acute sensorineural hearing loss. Acute sensorineural hearing loss with vertigo and/or dizziness could be a prodrome of subsequent infarction of the vertebrobasilar artery territory.

Imaging such as T1-, T2-, or diffusion-weighted MRI or magnetic resonance angiography is suitable for detecting ischemic lesions. However, MRI is not a cost-effective method for examining all patients with acute sensorineural hearing loss because it has a low positive rate (2.4%) and is expensive [92]. Because recurrent vertigo is a major symptom in the prodromal stage of vertebrobasilar artery ischemia [53], patients with recurrent vertigo preceded by acute sensorineural hearing loss should be carefully monitored. Especially, patients with acute sensorineural hearing loss and recurrent vertigo who have risk factors of cerebrovascular disease such as hypertension, exposure to cigarette smoke, diabetes, atrial fibrillation, coronary artery disease, dyslipidemia, carotid artery stenosis, sickle cell disease, postmenopausal hormone therapy, poor diet, physical inactivity, and obesity [93] must be examined with MRI.

Conclusion

Acute sensorineural hearing loss could be a prodrome of subsequent critical infarction due to vertebrobasilar artery ischemia, such as AICA syndrome, PICA syndrome, or basilar artery occlusion. Acute sensorineural hearing loss associated with vertebrobasilar artery ischemia is usually accompanied by vertigo or is preceded by recurrent episodes of vertigo.

References

1. Huang CY, Yu YL (1985) Small cerebellar strokes may mimic labyrinthine lesions. J Neurol Neurosurg Psychiatry 48: 263-265.
2. Chang CF, Kuo YL, Chen SP, Wang MC, Liao WH, et al. (2013) Relationship between idiopathic sudden sensorineural hearing loss and subsequent stroke. Laryngoscope 123: 1011-1015.
3. Lin RJ, Krall R, Westerberg BD, Chadha NK, Chau JK (2012) Systematic review and meta-analysis of the risk factors for sudden sensorineural hearing loss in adults. Laryngoscope 122: 624-635.
4. Huang MH, Huang CC, Ryu SJ, Chu NS (1993) Sudden bilateral hearing impairment in vertebrobasilar occlusive disease. Stroke 24: 132-137.
5. Sauvaget E, Kici S, Petelle B, Kania R, Chabriat H, et al. (2004) Vertebrobasilar occlusive disorders presenting as sudden sensorineural hearing loss. Laryngoscope 114: 327-332.
6. Biavati MJ, Gross JD, Wilson WR, Dina TS (1994) Magnetic resonance imaging evidence of a focal pontine ischemia in sudden hearing loss and seventh nerve paralysis. Am J Otol 15: 260-263.
7. Deplanque D, Godefroy O, Guerouaou D, Laureau E, Desaully A (1998) Sudden bilateral deafness: lateral inferior pontine infarction. J Neurol Neurosurg Psychiatry 64: 817-818.
8. Kim JS, Lopez I, DiPatre PL, Liu F, Ishiyama A, et al. (1999) Internal auditory artery infarction: clinicopathologic correlation. Neurology 52: 40-44.
9. Lee H, Whitman GT, Lim JG, Lee SD, Park YC (2001) Bilateral sudden deafness as a prodrome of anterior inferior cerebellar artery infarction. Arch Neurol 58: 1287-1289.
10. Lee H, Sohn SI, Jung DK, Cho YW, Lim JG, et al. (2002) Sudden deafness and anterior inferior cerebellar artery infarction. Stroke 33: 2807-2812.
11. Lee H, Cho YW (2003) Auditory disturbance as a prodrome of anterior inferior cerebellar artery infarction. J Neurol Neurosurg Psychiatry 74: 1644-1648.
12. Lee H, Yi HA, Baloh RW (2003) Sudden bilateral simultaneous deafness with vertigo as a sole manifestation of vertebrobasilar insufficiency. J Neurol Neurosurg Psychiatry 74: 539-541.
13. Lee H, Ahn BH, Baloh RW (2004) Sudden deafness with vertigo as a sole manifestation of anterior inferior cerebellar artery infarction. J Neurol Sci 222: 105-107.
14. Yi HA, Lee SR, Lee H, Ahn BH, Park BR, et al. (2005) Sudden deafness as a sign of stroke with normal diffusion-weighted brain MRI. Acta Otolaryngol 125: 1119-1121.
15. Murakami T, Nakayasu H, Doi M, Fukada Y, Hayashi M, et al. (2006) Anterior inferior cerebellar artery infarction with sudden deafness and vertigo. J Clin Neurosci 13: 1051-1054.
16. Son EJ, Bang JH, Kang JG (2007) Anterior inferior cerebellar artery infarction presenting with sudden hearing loss and vertigo. Laryngoscope 117: 556-558.
17. Kim JS, Cho KH, Lee H (2009) Isolated labyrinthine infarction as a harbinger of anterior inferior cerebellar artery territory infarction with normal diffusion-weighted brain MRI. J Neurol Sci 278: 82-84.
18. Lee H, Kim HU, Koo JW, Kim JS (2009) Progression of acute cochleovestibulopathy into anterior inferior cerebellar artery infarction. J Neurol Sci 278: 119-122.
19. Ikekami-Takada T, Izumikawa M, Doi T, Takada Y, Tomoda K (2012) AICA syndrome with facial palsy following vertigo and acute sensorineural hearing loss. Auris Nasus Larynx 39: 244-248.
20. Kanzaki S, Suzuki T, Suzuki S, Suzuki N, Ogawa K (2013) Sudden onset hearing loss and vertigo just before posterior inferior cerebellar artery infarction (lateral medulla syndrome). Otol Neurotol 34: 66-7.
21. Kido T, Sekitani T, Okinaka Y, Tahara T, Hara H (1994) A case of cerebellar infarction occurred with the 8th cranial nerve symptoms. Auris Nasus Larynx 21: 111-117.
22. Chiang CI, Chou CH, Hsueh CJ, Cheng CA, Peng GS (2013) Acute bilateral hearing loss as a “worsening sign” in a patient with critical basilar artery stenosis. J Clin Neurosci 20: 177-179.
23. Murakami T, Ono Y, Akagi N, Oshima E, Hamakawa Y, et al. (2005) A case of superior cerebellar artery syndrome with contralateral hearing loss at onset. J Neurol Neurosurg Psychiatry 76: 1744-1745.
24. Sunohse H, Toshima M, Mitani S, Suzuki M, Yoshida F, et al. (2000) Sudden bilateral hearing loss and dizziness occurred with cerebellar infarction. Otolaryngol Head Neck Surg 122: 146-147.
25. Toyoda K, Hirano T, Kumai Y, Fuji K, Kiritoshi S, et al. (2002) Bilateral deafness as a prodromal symptom of basilar artery occlusion. J Neurol Sci 193: 147-150.
26. Jung J, Philippoue F, Truy E, Fischer C, Brousolle E, et al. (2004) Progressive deafness preceding a basilar artery thrombosis. Cerebrovasc Dis 17: 269-270.
27. Huang CC, Young YH (2005) Vertigo with rebound nystagmus as an initial manifestation in a patient with basilar artery occlusion. Eur Arch Otorhinolaryngol 262: 576-579.
28. Bovo R, Tortore R, Ciorda A, Berto A, Martini A (2007) Bilateral sudden profound hearing loss and vertigo as a unique manifestation of bilateral symmetric inferior pontine infarctions. Ann Otol Rhinol Laryngol 116: 407-410.
80. Ohki M, Kitazawa H, Hiramatsu T, Kaga K, Kitamura T, et al. (2009) Role of primate cerebellar hemisphere in voluntary eye movement control revealed by lesion effects. J Neurophysiol 101: 934-947.

81. Hiramatsu T, Ohki M, Kitazawa H, Xiong G, Kitamura T, et al. (2008) Role of primate cerebellar lobulus petrosus of paraflocculus in smooth pursuit eye movement control revealed by chemical lesion. Neurosci Res 60: 250-258.

82. Shutoh F, Ohki M, Kitazawa H, Itohara S, Nagao S (2006) Memory trace of motor learning shifts transsynaptically from cerebellar cortex to nuclei for consolidation. Neuroscience 139: 767-777.

83. Shutoh F, Katah A, Ohki M, Itohara S, Tonegawa S, et al. (2003) Role of protein kinase C family in the cerebellum-dependent adaptive learning of horizontal optokinetic response eye movements in mice. Eur J Neurosci 18: 134-142.

84. Kitazawa H, Xiong G, Hiramatsu T, Ohki M, Nagao S (2009) Difference of climbing fiber input sources between the primate oculomotor-related cerebellar vermis and hemisphere revealed by a retrograde tracing study. Neurosci Lett 462: 10-13.

85. Waespe W, Cohen B, Raphan T (1983) Role of the flocculus and paraflocculus in optokinetic nystagmus and visual-vestibular interactions: effects of lesions. Exp Brain Res 50: 9-33.

86. Aschoff JC, Cohen B (1973) Oculomotor deficiency after cerebellar cortical lesions. Adv Otorhinolaryngol 19: 222-249.

87. Voogd J, Schraa-Tam CK, van der Geest JN, De Zeeuw CI (2012) Visuomotor cerebellum in human and nonhuman primates. Cerebellum 11: 392-410.

88. Fernandez C, Fredrickson JM (1963) Experimental cerebellar lesions and their effect on vestibular function. Acta Otolaryngol Suppl 192: SUPPL 192:52+.

89. Nagao S (1992) Different roles of flocculus and ventral paraflocculus for oculomotor control in the primate. Neuroreport 3: 13-16.

90. Igarashi M, Miyata H, Alford BR, Wright WK (1973) Experimental cerebellar uvulonodular lesions in the squirrel monkey. Adv Otorhinolaryngol 19: 220-231.

91. Itô M, Shiida T, Yagi N, Yamamoto M (1974) Visual influence on rabbit horizontal vestibulo-ocular reflex presumably effected via the cerebellar flocculus. Brain Res 65: 170-174.

92. Wilson YL, Gandolfi MM, Ahn IE, Yu G, Huang TC, et al. (2010) Cost analysis of asymmetric sensorineural hearing loss investigations. Laryngoscope 120: 1832-1836.

93. Goldstein LB, Adams R, Alberts MJ, Appel LJ, Brass LM, et al. (2006) Primary prevention of ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council: cosponsored by the Atherosclerotic Peripheral Vascular Disease Interdisciplinary Working Group; Cardiovascular Nursing Council; Clinical Cardiology Council; Nutrition, Physical Activity, and Metabolism Council; and the Quality of Care and Outcomes Research Interdisciplinary Working Group: the American Academy of Neurology affirms the value of this guideline. Stroke 37: 1583-1633.