Journal of the Neurological Sciences xxx (2015) xxx–xxx

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Audiovestibular impairments associated with intracranial hypotension

Jae-Hwan Choi [a], Kee-Yong Cho [b], Seung-Yi Cha [b], Jae-Deuk Seo [c], Min-Ji Kim [b], Yu Ri Choi [b], Sung-Hee Kim [d], Ji-Soo Kim [d], Kwang-Dong Choi [b,*]

[a] Department of Neurology, Pusan National University School of Medicine, Research Institute for Convergence of Biomedical Science and Technology, Pusan National University Yangsan Hospital, Yangsan, Republic of Korea
[b] Department of Neurology, Pusan National University Hospital, Pusan National University School of Medicine and Biomedical Research Institute, Busan, Republic of Korea
[c] Department of Neurology, Bonhospital, Busan, Republic of Korea
[d] Department of Neurology, Seoul National University Bundang Hospital, Seongnam, Republic of Korea

ARTICLE INFO

Article history:
Received 28 December 2014
Received in revised form 4 June 2015
Accepted 1 July 2015
Available online xxxx

Keywords:
Intracranial hypotension
CSF hypovolemia
Audiovestibular impairment
Hearing loss
Dizziness
Nystagmus

ABSTRACT

Objective: To investigate the patterns and mechanisms of audiovestibular impairments associated with intracranial hypotension.
Methods: We had consecutively recruited 16 patients with intracranial hypotension at the Neurology Center of Pusan National University Hospital for two years. Spontaneous, gaze-evoked, and positional nystagmus were recorded using 3D video-oculography in all patients, and the majority of them also had pure tone audiometry and bithermal caloric tests.
Results: Of the 16 patients, five (31.3%) reported neuro-otological symptoms along with the orthostatic headache while laboratory evaluation demonstrated audiovestibular impairments in ten (62.5%). Oculographic analyses documented spontaneous and/or positional nystagmus in six patients (37.5%) including weak spontaneous vertical nystagmus with positional modulation (n = 4) and pure positional nystagmus (n = 2). One patient presented with recurrent spontaneous vertigo and tinnitus mimicking Meniere's disease, and showed unidirectional horizontal and torsional nystagmus with normal head impulse tests during the attacks. Bithermal caloric tests were normal in all nine patients tested. Audiometry showed unilateral (n = 6) or bilateral (n = 1) sensorineural hearing loss in seven (53.8%) of the 13 patients tested.
Conclusions: Intracranial hypotension frequently induces audiovestibular impairments. In addition to endolymphatic hydrops and irritation of the vestibulocochlear nerve, compression or traction of the brainstem or cerebellum due to loss of CSF buoyancy may be considered as a mechanism of frequent spontaneous or positional vertical nystagmus in patients with intracranial hypotension.

1. Introduction

Intracranial hypotension is characterized by orthostatic headache due to low CSF volume caused by either spontaneous or post-traumatic dural laceration [1–5]. The headache is typically provoked by sitting or standing, and relieved by lying flat. Associated symptoms include nausea, vomiting, pain and tight feeling in the neck, diplopia, and blurred vision. A study of 30 consecutive patients with intracranial hypotension has described neurotological symptoms including dizziness (30%), tinnitus (20%), aural fullness (20%), and hearing loss (3%) [1], while another reported auditory symptoms in approximately 70% of the patients [6]. Earlier reports have also documented unilateral or bilateral low-frequency sensorineural hearing loss on audiometry with or without vertigo, likewise in Meniere's disease (MD) [6–17]. However, previous studies have mostly focused on auditory dysfunction, and no study has attempted objective documentation of vestibular dysfunction in intracranial hypotension.

To determine the patterns and mechanisms of audiovestibular impairments associated with intracranial hypotension, we performed neurotological evaluation in 16 consecutive patients.

2. Materials and methods

2.1. Subjects

We had consecutively recruited 16 patients with intracranial hypotension according to the International Classification of Headache Disorders criteria for headaches attributed to spontaneous (or idiopathic) low CSF pressure at the Neurology Center of Pusan National University Hospital for two years from November 2011 to November 2013 [18]. All patients had orthostatic headache, and brain MRIs showed strong contrast enhancements and thickening of the pachymeninges in ten (62.5%), compatible with the typical findings of intracranial

* Corresponding author at: Department of Neurology, College of Medicine, Pusan National University, Pusan National University Hospital, 1-10 Ami-dong, Seo-gu, Busan, 602-739, Republic of Korea.
E-mail address: kdchoi@medimail.co.kr (K.-D. Choi).

http://dx.doi.org/10.1016/j.jns.2015.07.002
0022-510X/

hypotension. Lumbar CSF studies were also performed in 13 patients. Eleven patients had spontaneous intracranial hypotension while the remaining five developed the symptoms just after lumbar puncture for spinal anesthesia due to operation (n = 3, rectal cancer, hemorrhoids, and penile curvature respectively), or chiropractic manipulation for chronic lower back pain (n = 2). The duration of intracranial hypotension ranged from 1 to 21 days (6.1 ± 5.2 days). The patients included seven men and nine women with the ages ranging from 23 to 64 years (mean ± SD = 41.9 ± 10.1 years).

All the experiments followed the tenets of the Declaration of Helsinki and were approved by the Institutional Review Board of Pusan National University Hospital. Informed contents were obtained after the nature and possible consequence of this study had been explained to the participants.

2.2. Neuro-otological evaluation

All patients had full bedside neurological and neuro-otological evaluation by the authors. Eye movements were recorded binocularly at a sampling rate of 60 Hz using 3D video-oculography (SensoMotoric Instruments, Teltow, Germany). Spontaneous nystagmus (SN) was recorded both with and without fixation. Gaze-evoked nystagmus was induced with horizontal (±30°) and vertical (±20°) target displacements. For positional nystagmus (PN), patients bent down, straightened, and turned their heads to either side while supine. Patients were also subjected to the straight head hanging. The presence of spontaneous and positional nystagmus in the patients was defined only when the mean slow phase velocity (SPV) of the spontaneous nystagmus and maximal SPV of the positional nystagmus exceeded the values (mean + 2SD) observed in normal controls (horizontal SN ≥ 1.1°/s; vertical SN ≥ 1.8°/s; torsional SN > 0°/s; horizontal PN ≥ 2.6°/s; positional upbeat nystagmus ≥ 3.8°/s, positional downbeat nystagmus ≥ 0°/s; torsional PN ≥ 0.9°/s). In patients with spontaneous nystagmus, the SPV of positional nystagmus was determined by subtracting the SPV of spontaneous nystagmus.

Out of 16 patients, 13 received pure tone audiometry in sitting position, and nine had bithermal caloric tests in supine position with the head inclined at 30°. The remaining patients refused to receive the tests due to profound orthostatic headache. The caloric stimuli comprised alternative irrigation for 25 s with about 250 ml of cold and hot water (30 °C and 44 °C). Caloric paresis was defined by a response difference of 25% or more between the ears using the Jongkees' formula.

Medications that could potentially affect the vestibular system were not allowed during the study.

2.3. Treatment strategy and follow-up evaluation

All patients received conservative treatment (n = 6) or epidural blood patch (n = 10). After resolution of orthostatic headache with the treatments, the patients with abnormal eye movements previously had a follow-up recording of eye movements.

3. Results

3.1. Clinical characteristics

Detailed demographic and clinical profiles of the patients are described in Table 1.

Five (5/16, 31.3%) had neuro-otological symptoms along with the orthostatic headache. Two patients (patients 1 and 3) developed both orthostatic dizziness and tinnitus, and the remaining three had orthostatic dizziness (patient 6) or orthostatic tinnitus (patients 9 and 10). Two patients (patients 1 and 3) reported tinnitus in both ears while another two (patients 9 and 10) noticed tinnitus only in one ear. One patient (patient 1) without a previous history of medical or neuro-otological diseases presented with recurrent spontaneous vertigo, earfullness, and tinnitus in either ear along with the orthostatic dizziness, tinnitus, and headache. The attacks lasted for several hours and occurred 1–2 times per day, which mimicked MD.

3.2. Neuro-otological evaluation

Six (6/16, 37.5%) patients showed spontaneous and/or positional nystagmus (Table 2). Three patients (patients 1, 2, and 6) developed weak spontaneous downbeat nystagmus without visual fixation in sitting position. In patient 1, the downbeat nystagmus did not change after taking supine position, but changed into upbeat nystagmus during straight head hanging (Fig. 1A). Patient 2 showed slight attenuation of downbeat nystagmus while supine, but no change during straight head hanging. In patient 6, downbeat nystagmus increased significantly and accompanied counter-clockwise torsional and left-beating components in supine and straight head hanging positions (Fig. 1B and Video 1). Patient 3 disclosed weak spontaneous upbeat nystagmus without visual fixation in sitting position, which was exaggerated in supine and straight head hanging positions (Fig. 1C). Counter-clockwise torsional and left-beating components were also accompanied in the supine position, and left-beating component was associated in the straight head hanging position. The remaining two patients exhibited isolated positional nystagmus including pure upbeat (patient 4) or mainly upbeat nystagmus mixed with right-beating and clockwise torsional components (patient 5) in the straight head hanging position. In all six patients, positional nystagmus lasted more than one minute. On resuming sitting position, reversal of the positional nystagmus occurred in two patients (patients 1 and 5).

During two attacks of vertigo in patient 1, we observed unidirectional horizontal and torsional nystagmus beating to the right or left without visual fixation (Video 2), but bedside horizontal head impulse tests were always normal.

Bithermal caloric tests were normal in all nine patients tested, and seven (54%) of the 13 patients with audiometry showed unilateral low-frequency sensorineural hearing loss (SNHL) (n = 6) or bilateral low- and high-frequencies SNHL (n = 1). The degree of hearing loss was mild in all the patients.

3.3. Follow-up evaluation of the audiovestibular dysfunction

All patients had resolution of orthostatic headache immediately or within several days after epidural blood patch or conservative treatments. Six patients also showed the disappearance of spontaneous and positional nystagmus immediate or within several days after resolution of orthostatic headache. In four patients (patients 1, 2, 5, and 6), spontaneous and positional nystagmus disappeared within several days ranging from 1 to 7 days (mean = 4 days) after epidural blood patch. In patient 1, recurrent attacks of vertigo and auditory symptoms improved after diuretics treatment for three months.

Four (patients 1, 3, 5, and 10) of the seven patients received follow-up evaluation of auditory function at different periods. Three (patients 1, 3, and 10) of them showed resolution of hearing loss at three months, 20 days, and four months respectively after the resolution of orthostatic headache. However, hearing loss did not improve at two weeks after the resolution of orthostatic headache in patient 5.

4. Discussion

The present study demonstrated that approximately 30% of patients with intracranial hypotension report neuro-otological symptoms including dizziness, earfullness, and tinnitus along with orthostatic headache, while laboratory evaluation discloses audiovestibular impairments in 63%. Unilateral or bilateral SNHL was found in about half of the patients, and 38% exhibited spontaneous or positional nystagmus. These results support earlier findings of frequent audiovestibular impairments among the patients with intracranial hypotension [1,6]. Of interest, one

Please cite this article as: J.-H. Choi, et al., Audiovestibular impairments associated with intracranial hypotension, J Neurol Sci (2015), http://dx.doi.org/10.1016/j.jns.2015.07.002

Table 1
Demographic and clinical features of the patients.

Patient no.	Sex/age	Duration (days)	Associated symptoms	CSF pressure (mm H_2O)	Brain MRI	Treatment	Etiology
Patients with vestibular dysfunction (n = 6)							
1	F/52	3	Orthostatic dizziness and B tinnitus, recurrent spontaneous vertigo, earfullness, and tinnitus	35	Pachymeningitis	Epidural blood patch	Spontaneous
2	F/41	21	Orthostatic dizziness	50	Normal	Epidural blood patch	Spontaneous
3	F/43	2	Orthostatic vertigo and B tinnitus	ND	Pachymeningitis	Conservative	Spontaneous
4	F/37	11	–	55	Pachymeningitis	Conservative	Spontaneous
5	F/37	7	–	53	Pachymeningitis	Epidural blood patch	Spontaneous
6	M/44	1	Orthostatic dizziness	ND	Pachymeningitis	Epidural blood patch	Iatrogenic
Patients without vestibular dysfunction (n = 10)							
7	M/30	3	–	20	Normal	Conservative	Spontaneous
8	M/23	7	–	ND	Pachymeningitis	Conservative	Iatrogenic
9	F/36	12	Orthostatic R tinnitus	20	Pachymeningitis	Conservative	Spontaneous
10	M/34	7	Orthostatic R tinnitus	70	Pachymeningitis	Epidural blood patch	Iatrogenic
11	M/39	4	–	50	Normal	Epidural blood patch	Spontaneous
12	F/47	1	–	32	Normal	Epidural blood patch	Spontaneous
13	M/38	3	–	50	Normal	Epidural blood patch	Iatrogenic
14	F/51	4	–	UM	Pachymeningitis	Epidural blood patch	Spontaneous
15	M/54	3	–	35	Normal	Conservative	Iatrogenic
16	F/64	8	–	67	Pachymeningitis	Epidural blood patch	Spontaneous

M = male; F = female; R = right; B = bilateral; ND = not done; UM = unmeasurable.

patient also suffered from recurrent spontaneous vertigo, earfullness, and tinnitus along with orthostatic dizziness, tinnitus, and headache. Our patients mostly had unilateral low-frequency SNHL while one showed bilateral low- and high-frequencies SNHL. These are consistent with the findings of earlier case reports on intracranial hypotension, which described unilateral or bilateral low-frequency SNHL with occasional progression to downsloping mild-to-profound SNHL [6–17]. In contrast, the caloric tests were normal in all. The discrepancy between the audiometry and caloric tests may be ascribed to the position during the tests. The audiometry was carried out in sitting position where CSF leakage was more aggravated, whereas the caloric test was performed in supine position.

Several mechanisms have been proposed for the neurotological dysfunction in intracranial hypotension, which have converged on irritation or hypofunction of the peripheral labyrinth or vestibulocochlear nerve [7–9]. Under normal condition, there is a balance of pressure between the CSF and perilymph via the cochlear aqueduct (if permeable) [19]. An experimental study proved that intracranial pressure changes are transmitted to the inner ear by passive liquid transfer; rapid if the cochlear canal is permeable, and slow when it is nonfunctional [7]. Therefore, the reduction of the perilymph due to CSF hypovolemia would result in compensatory endolymphatic hydrops that will in turn generate vertigo and auditory dysfunction mimicking MD. The lower frequency SNHL observed in most of our patients and in earlier reports supports this assumption. In a patient with intracranial hypotension, venous engorgement was once found on MRIs in both internal acoustic canals [8]. Thus, the irritation of the vestibular and cochlear nerves may be an alternative mechanism. Stretching of the vestibulocochlear nerve due to brain sagging may be another possibility [9]. However, this can hardly explain isolated auditory symptoms without vertigo and other cranial or cervical nerve dysfunctions in some patients.

Remarkably, our study found spontaneous and/or positional nystagmus in 38% of patients with intracranial hypotension. Patients showed weak spontaneous vertical nystagmus with prominent positional modulation or pure positional nystagmus. To our knowledge, this is the first description of objective vestibular dysfunction in intracranial hypotension. It is of interest that the vertical nystagmus is conspicuous during supine or head hanging positions when the intracranial pressure (ICP) may be likely closest to normal. The patterns of spontaneous and positional nystagmus suggest brainstem or cerebellar dysfunction since spontaneous vertical nystagmus has mostly been reported in brainstem or cerebellar lesions [20], and various central patterns of positional nystagmus have largely been described in patients with lesions dorsolateral to the fourth ventricle or in the cerebellar vermis [21]. In patients with intracranial hypotension, compression or traction along

Table 2
Neuro-otological evaluation of the patients.

Patient no.	Spontaneous nystagmus (°/s)	Supine position (°/s)	Straight head hanging position (°/s)	Bithermal caloric tests	Pure tone audiometry
1	D(2.3)	D(2.2)	U(3.3)	Normal	L low tone SNHL
2	D(3.2)	D(2.2)	D(3.2)	ND	Normal
3	U(3.0)	U(8.2), CW(3.2)	U(24), L(5.3)	ND	L low tone SNHL
4	–	–	U(3.8)	Normal	Normal
5	–	U(3.7), R(1.3), CW(1.0)	U(3.9), R(1.7)	Normal	R low tone SNHL
6	D(3.5)	D(15), CCW(5.8), L(1.9)	D(16.6), CCW(6.1), L(2.4)	Normal	Normal
7	–		–	ND	ND
8	–		–	ND	Normal
9	–		–	Normal	R low tone SNHL
10	–		–	Normal	B low- and high tone SNHL
11	–		–	Normal	Normal
12	–		–	Normal	R low tone SNHL
13	–		–	ND	ND
14	–		–	Normal	Normal
15	–		–	ND	L low tone SNHL
16	–		–	ND	ND

D = downbeat; U = upbeat; R = right; L = left; D = downward; NC = not changed compared to the spontaneous nystagmus; ND = not done; SNHL = sensori-neural hearing loss.

Please cite this article as: J.-H. Choi, et al., Audiovestibular impairments associated with intracranial hypotension, J Neurol Sci (2015), http://dx.doi.org/10.1016/j.jns.2015.07.002

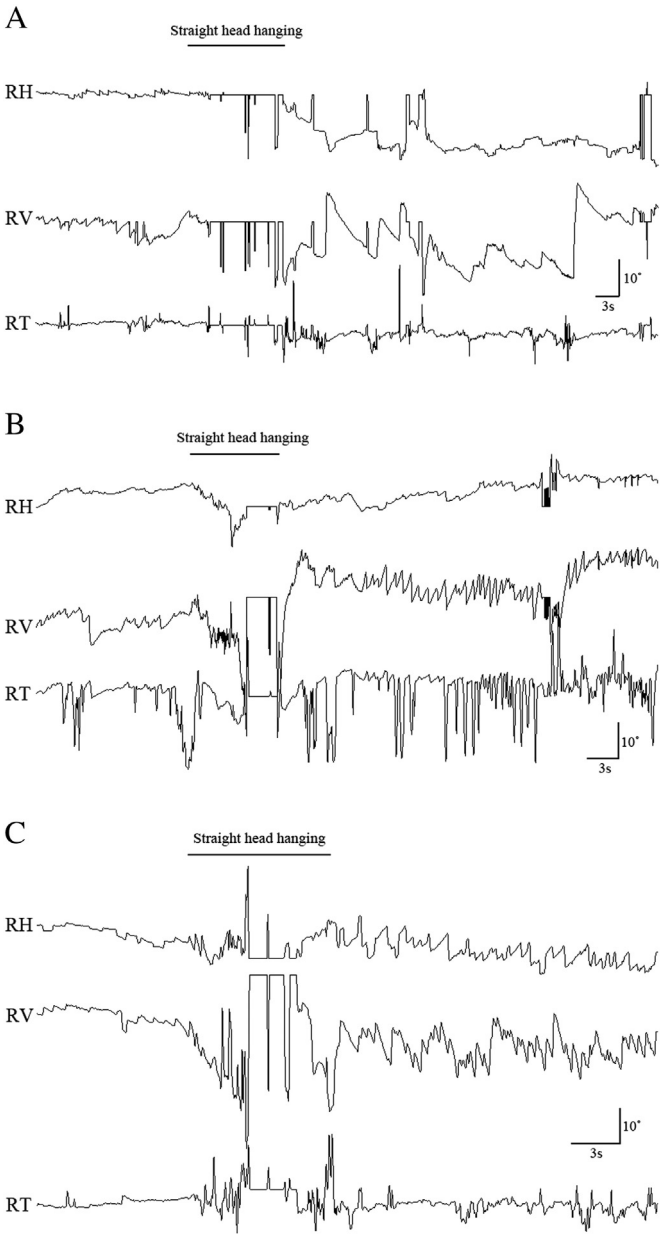

Fig. 1. Oculographic analyses in four patients with intracranial hypotension. (A) Patient 1 shows weak spontaneous downbeat nystagmus in sitting position, which changes into upbeat in straight head hanging position. (B) In straight head hanging position, the weak spontaneous downbeat nystagmus while sitting increases significantly, and counter-clockwise torsional and left-beating components occurs in patient 6. (C) In patient 3, spontaneous upbeat nystagmus while sitting becomes stronger and left-beating component newly occurs in straight head hanging position. RH = horizontal recording of the right eye movement; RV = vertical recording of the right eye movement; RT = torsional recording of the right eye movement. Upward deflection indicates rightward, upward, and clockwise (from the patient's perspective) eye motion in each trace.

the cranial floor of the brainstem or cerebellum due to a loss of CSF buoyancy may produce audiovestibular impairments, which may be the most prominent during supine or head hanging position [22–24]. However, pure vertical nystagmus without other ocular motor abnormalities suggestive of brainstem or cerebellar dysfunction can also be explained by the irritation or hypofunction of bilateral peripheral vestibular structures since both ears are subjected to the same effect of any change in intracranial pressure.

Concurrent benign paroxysmal positional vertigo (BPPV) may be considered as the mechanism of positional nystagmus in our patients. Indeed, three patients developed vertical nystagmus mixed with horizontal and/or torsional components during positional tests, which mimicked vertical canal BPPV. However, the concurrent BPPV is contradicted by accompanying spontaneous vertical nystagmus, persistent positional nystagmus without severe vertigo during the positional tests, no reversal of the positional nystagmus on resuming sitting position, and immediate disappearance of the spontaneous and positional nystagmus after resolution of orthostatic headache.

We also observed unidirectional horizontal and torsional nystagmus beating to either side with normal head impulse tests during the attacks of vertigo in a patient with MD-like symptoms. Although unidirectional nystagmus with normal head impulse tests may suggest central vestibular dysfunction [25,26], accompanying auditory symptoms and the variable direction of horizontal and torsional nystagmus during attacks favor secondary endolymphatic hydrops. Indeed, the vestibulo-ocular reflex during higher frequency stimulation such as head impulse can be preserved in MD since this disorder preferentially affects the lower frequency sensitivity of the canals [27]. It is of interest that the symptoms in this patient had persisted for several months even after the resolution of orthostatic headache, which suggests that more longer period may be required for recovery of imbalance between the CSF and endolymph.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.jns.2015.07.002.

Conflicts of interest

We have no disclosure of any competing interest.

Acknowledgments

This study was supported by the Research Institute for Convergence of biomedical science and technology Grant (30-2013-006), Pusan National University Yangsan Hospital.

References

[1] S.J. Chung, J.S. Kim, M.C. Lee, Syndrome of cerebral spinal fluid hypovolemia: clinical and imaging features and outcome, Neurology 55 (2000) 1321–1327.
[2] B. Mokri, Spontaneous low pressure, low CSF volume headaches: spontaneous CSF leaks, Headache 53 (2013) 1034–1053.
[3] J. Hoffmann, P.J. Goadsby, Update on intracranial hypertension and hypotension, Curr. Opin. Neurol. 26 (2013) 240–247.
[4] F. Amoozegar, D. Guglielmin, W. Hu, D. Chan, W.J. Becker, Spontaneous intracranial hypotension: recommendations for management, Can. J. Neurol. Sci. 40 (2013) 144–157.
[5] R. Lahoria, L. Allport, D. Glenn, L. Masters, R. Shnier, M. Davies, et al., Spontaneous low pressure headache — a review and illustrative patient, J. Clin. Neurosci. 19 (2012) 1076–1079.
[6] E. Ferrante, C. Regna-Gladin, A. Citterio, I. Arpino, Spontaneous intracranial hypotension syndrome with hearing loss and pachymeningeal enhancement in the internal acoustic canal: neuroimaging correlations, J. Craniofac. Surg. 21 (2010) 1660–1661.
[7] A. Walsted, G. Salomon, J. Thomsen, M. Tos, Hearing decrease after loss of cerebrospinal fluid. A new hydrops model? Acta Otolaryngol. 111 (1991) 468–476.
[8] H. Isildak, S. Albayram, H. Isildak, Spontaneous intracranial hypotension syndrome accompanied by bilateral hearing loss and venous engorgement in the internal acoustic canal and positional change of audiography, J. Craniofac. Surg. 21 (2010) 165–167.
[9] S.J. Chung, J.H. Ahn, J.H. Lee, J.H. Im, M.C. Lee, Orthostatic hypoacusis in a patient with CSF hypovolemia, Cephalalgia 26 (2006) 613–616.
[10] Y. Taguchi, H. Fushiki, S. Takashima, K. Tanaka, Intracranial hypotension with positional hearing loss, Intern. Med. 52 (2013) 1541–1542.
[11] M. Arai, T. Takada, M. Nozue, Orthostatic tinnitus: an ontological presentation of spontaneous intracranial hypotension, Auris Nasus Larynx 30 (2003) 85–87.
[12] J.R. Couch, Spontaneous intracranial hypotension: the syndrome and its complications, Curr. Treat. Options Neurol. 10 (2008) 3–11.
[13] S. Chen, M. Hagiwara, P.C. Roehm, Spontaneous intracranial hypotension presenting with severe sensorineural hearing loss and headache, Otol. Neurotol. 33 (2012) e65–e66.
[14] N. Fontaine, A. Charpiot, A. Gentine, A case of spontaneous intracranial hypotension: from Meniere-like syndrome to cerebral involvement, Eur. Ann. Otorhinolaryngol. Head Neck Dis. 129 (2012) 153–156.
[15] R.S. Miller, T.A. Tami, M. Pensak, Spontaneous intracranial hypotension mimicking Meniere's disease, Otolaryngol. Head Neck Surg. 135 (2006) 655–656.

Please cite this article as: J.-H. Choi, et al., Audiovestibular impairments associated with intracranial hypotension, J Neurol Sci (2015), http://dx.doi.org/10.1016/j.jns.2015.07.002

[16] F. Portier, C. de Minteguiaga, E. Racy, P.T. Huy, P. Herman, Spontaneous intracranial hypotension: a rare cause of labyrinthine hydrops, Ann. Otol. Rhinol. Laryngol. 111 (2002) 817–820.
[17] M. Taki, F. Nin, T. Hasegawa, H. Sakaguchi, S. Yamamoto, T. Suzuki, et al., Case report: two cases of hearing impairment due to intracranial hypotension, Auris Nasus Larynx 36 (2009) 345–348.
[18] Headache classification subcommittee of the international headache society, The International Classification of Headache Disorder, 2nd edition, Cephalalgia, 24, 2004. 9–160.
[19] R.R. Ciuman, Communication routes between intracranial spaces and inner ear: function, pathophysiologic importance and relations with inner ear diseases, Am. J. Otolaryngol. 30 (2009) 193–202.
[20] C. Pierrot-Deseilligny, D. Milea, Vertical nystagmus: clinical facts and hypotheses, Brain 128 (2005) 1237–1246.
[21] U. Büttner, C. Helmchen, T. Brandt, Diagnostic criteria for central versus peripheral positioning nystagmus and vertigo: a review, Acta Otolaryngol. 119 (1999) 1–5.
[22] J.L. Atkinson, B.G. Weinshenker, G.M. Miller, D.G. Piepgras, B. Mokri, Acquired Chiari I malformation secondary to spontaneous spinal cerebrospinal fluid leakage and chronic intracranial hypotension syndrome in seven cases, J. Neurosurg. 88 (1998) 237–242.
[23] C. Samii, E. Möbius, W. Weber, H.W. Heienbrok, P. Berlit, Pseudo Chiari type I malformation secondary to cerebrospinal fluid leakage, J. Neurol. 246 (1999) 162–164.
[24] S.E. Kasner, J. Rosenfeld, R.E. Farber, Spontaneous intracranial hypotension: headache with a reversible Arnold–Chiari malformation, Headache 35 (1995) 557–559.
[25] J.C. Kattah, A.V. Talkad, D.Z. Wang, Y.H. Hsieh, D.E. Newman-Toker, HINTS to diagnose stroke in the acute vestibular syndrome: three-step bedside oculomotor examination more sensitive than early MRI diffusion-weighted imaging, Stroke 40 (2009) 3504–3510.
[26] D.E. Newman-Toker, K.A. Kerber, Y.H. Hsieh, J.H. Pula, R. Omron, A.S. Saber Tehrani, et al., HINTS outperforms ABCD2 to screen for stroke in acute continuous vertigo and dizziness, Acad. Emerg. Med. 20 (2013) 986–996.
[27] H.J. Park, A.A. Migliaccio, C.C. Della Santina, L.B. Minor, J.P. Carey, Search-coil head-thrust and caloric tests in Meniere's disease, Acta Otolaryngol. 125 (2005) 852–857.

Please cite this article as: J.-H. Choi, et al., Audiovestibular impairments associated with intracranial hypotension, J Neurol Sci (2015), http://dx.doi.org/10.1016/j.jns.2015.07.002