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Analysis of the relationship between changes in the auditory brainstem response and prognosis in patients with sudden hearing loss

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Main Article

Associate Professor D-K Kim takes responsibility for the integrity of the content of the paper

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Abstract

Objective. To analyse how the auditory brainstem response changes in patients with sudden sensorineural hearing loss.
Method. Data were collected via retrospective medical chart review.
Results. Forty-three patients were included in this study. The mean latency of auditory brainstem response wave 1 was significantly longer for the affected side than for the unaffected side ($p = 0.003$). The mean latency of auditory brainstem response wave 1 was significantly shorter, and the mean amplitude of auditory brainstem response wave 1 was significantly larger, in the good response group compared to the poor response group. In forward conditional logistic regression analysis, auditory brainstem response wave 1 latency was an independent predictor of a good response (odds ratio = 34.37, 95 per cent confidence interval = 1.56–757.15, $p = 0.025$).
Conclusion. In patients with sudden sensorineural hearing loss, the latency of wave 1 of the auditory brainstem response was significantly increased and was related to prognosis.

Introduction

Idiopathic sudden sensorineural hearing loss (SNHL) has an annual worldwide incidence of approximately 5–20 per 100 000 individuals.[1] Sudden SNHL is a medical emergency and remains clinically challenging.[2] Sudden SNHL is defined as SNHL of 30 dB or more at three consecutive frequencies over 3 days or fewer.[3] The aetiology of sudden SNHL remains unknown; it is difficult to obtain inner-ear tissue. Therefore, it is unclear whether sudden SNHL lesions are localised to the cochlea or are retrocochlear in nature.

Previous studies have shown that the prognosis of sudden SNHL is better when the otoacoustic emissions (OAEs) are larger.[4,5] From these reports, we can infer that less damage to cochlear hair cells is related to an improved prognosis for sudden SNHL patients. However, assuming that retrocochlear lesions such as cochlear neuritis occur in sudden SNHL, little is known about whether changes in audiological results are associated with these retrocochlear lesions, or whether the changes are related to the prognosis.

This study aimed to analyse how auditory brainstem responses (ABRs) change in patients with sudden SNHL, and whether these changes are related to the prognosis, assuming retrocochlear lesions occur in sudden SNHL.

Materials and methods

The data of patients diagnosed with unilateral sudden SNHL at our hospital between February 2016 and February 2018, and whose ABR waveform measured 90 dB HL, were collected via retrospective medical chart review. A diagnosis of sudden SNHL was made when SNHL of at least 30 dB was present at three sequential frequencies, over 3 days or fewer, with no identifiable cause of hearing loss. We assessed each patient's hearing by pure tone audiometry and/or speech audiometry. Magnetic resonance imaging and ABR tests were performed to exclude vestibular schwannoma.

All patients were treated with high-dose systemic steroids. After initial oral methylprednisolone (48 mg/day for 5 days), the dose was tapered over an additional 5 days. If this treatment was ineffective, four additional intra-tympanic steroid injections were administered. Of our 43 patients, 33 received intra-tympanic steroid injections. A 0.3–0.5 ml dose of dexamethasone (dexamethasone sodium phosphate, 5 mg/ml; Il Sung Pharm, Seoul, Korea) was injected into the middle-ear cavity at 2–5-day intervals. After four injections, treatment was terminated regardless of symptom status.

In this study, patients with profound hearing loss, in whom ABR waveforms could not be measured at 90 dB HL, were excluded. Patients with identifiable causes of hearing loss, such as Ménière's disease, vestibular schwannoma or eardrum abnormalities, were excluded too. Patients with acute low-frequency hearing loss were excluded as well, because they may have had a disease aetiology other than sudden SNHL.[6] Finally, we also excluded patients who visited our hospital more than 30 days after the onset of illness

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Table 1. Demographic and clinical characteristics of sudden SNHL patients*

Clinical characteristic	Value
Sex (males:females (*n*))	22:21
Side of hearing loss (right:left (*n*))	16:27
Age (mean ± SD; years)	57.5 ± 14.1
Diabetes mellitus (*n* (%))	15 (34.9)
High BP (*n* (%))	10 (23.3)
Hearing level (mean ± SD; dB HL)	
– Affected ear	72.2 ± 17.8
– Unaffected ear	21.4 ± 12.4
Final PTA (mean ± SD; dB HL)†	49.7 ± 24.8
Final gain of AC threshold (mean ± SD; dB HL)	22.4 ± 24.9

*$n = 43$. †At 0.5, 1, 2 and 4 kHz. SNHL = sensorineural hearing loss; SD = standard deviation; BP = blood pressure; PTA = pure tone average; AC = air conduction

or who were not followed up for more than 3 months after finishing treatment. In total, 43 patients were included in the study.

If damage to the auditory nerve occurs in sudden SNHL patients, as our study assumes, this may affect the amplitude and latency of wave 1 of the ABR, which originates from the cochlear nerve.[7] We measured the amplitude and latency of ABR wave 1, and the amplitude of wave 5, at 90 dB HL, in both ears. The latency of wave 5 was not measured because it is affected by the change in latency of wave 1. We then compared the parameters of affected and unaffected ears, and those of patients exhibiting good and poor hearing recovery after treatment.

Statistical analysis

The chi-square test was used to compare categorical variables, and the independent *t*-test was applied for the analysis of continuous variables. For all statistical analyses, we used SPSS software (version 20.0; SPSS, Chicago, Illinois, USA). *P*-values of less than 0.05 were considered statistically significant.

Ethics statement

The study protocol was reviewed and approved by the Institutional Review Board of Catholic University Hospital (Institutional Review Board number: DC19RESI0015). The requirement for informed consent was waived by the board.

Results

In total, 43 patients (22 men and 21 women), with a mean age of 57.5 ± 14.1 years, were included in this study (Table 1). The right ear was involved in 16 cases and the left ear in 27.

The initial mean pure tone averages (at 0.5, 1, 2 and 4 kHz) of the affected and unaffected ears were 72.2 ± 17.8 dB HL and 21.4 ± 12.4 dB HL, respectively. The mean final pure tone average was 49.7 ± 24.8 dB HL and the increase in the air conduction threshold after treatment was 22.4 ± 24.9 dB HL.

The mean latency of ABR wave 1 was significantly longer in the affected ear than in the unaffected ear (1.65 ± 0.27 *vs* 1.49 ± 0.18 ms; $p = 0.003$), but the mean amplitudes of wave 1 (0.12 ± 0.07 *vs* 0.20 ± 0.28 μV) and wave 5 (0.20 ± 0.12 *vs* 0.33 ± 0.95 μV) did not differ between the groups.

The 43 patients were divided into a response group ($n = 21$) and a non-response group ($n = 22$) based on a 15 dB hearing improvement cut-off. The mean latency of ABR wave 1 was significantly shorter (1.50 ± 0.20 *vs* 1.77 ± 0.26 ms), and the mean amplitude of ABR wave 1 was significantly larger (0.15 ± 0.08 *vs* 0.10 ± 0.06 μV), in the response group than in the non-response group (Table 2). The initial mean pure tone average of the unaffected ear was significantly better in the response group than in the non-response group. The mean age of the response group was lower than that of the non-response group, although not significantly.

In order to determine which factors independently increased the probability of a response to treatment, we conducted forward conditional logistic regression analysis. This revealed that ABR wave 1 latency was an independent predictor of response to treatment (odds ratio = 34.37, 95 per cent confidence interval = 1.56–757.15, $p = 0.025$). Age, mean amplitude of ABR wave 1, and initial mean pure tone average of the unaffected ear did not predict the response to treatment. Bivariate correlation analysis between the latency of ABR wave 1 and the pure tone average showed a significant negative relationship ($r = -0.349$, $p = 0.037$; Figure 1).

Discussion

Between 32 per cent and 65 per cent of sudden SNHL cases recover spontaneously.[8,9] Treatment options are myriad, and include systemic and topical steroids, antiviral agents, rheology agents, diuretics, hyperbaric oxygen treatment, other medications, middle-ear surgery for fistula repair, and observation alone. There is no standard treatment protocol for sudden hearing loss because of a lack of definitive knowledge regarding its causes.[10] The prognosis for recovery is dependent on a number of factors, including the patient's age, presence of vertigo at onset, degree of hearing loss, audiometric configuration, and time between onset of hearing loss and treatment.[8,11,12]

- In patients with sudden hearing loss, auditory brainstem response (ABR) wave 1 latency in the affected ear increased significantly
- The ABR wave 1 latency was an independent predictor of prognosis
- As ABR wave 1 originates from the cochlear nerve, sudden hearing loss may be associated with a retrocochlear lesion

Our results showed that the mean latency of ABR wave 1 was significantly longer on the affected side compared to the unaffected side in the patients with sudden SNHL, and the degree of change in ABR wave 1 was significantly correlated with the prognosis.

Few papers have studied ABR changes in patients with sudden SNHL, but several recent studies have reported results similar to ours. Lin *et al.*[13] observed ABR changes in 102 sudden SNHL patients. The mean latencies of ABR waves 1, 3, and 5 were significantly increased on the affected side, and the latency of wave 1 was significantly correlated with hearing outcome. Another study investigating audiovestibular function in patients with severe-to-profound sudden SNHL demonstrated that both the presence of ABR and vestibular-evoked myogenic potential waveforms were significantly correlated with better hearing outcomes in the group with severe sudden SNHL.[14] A recent study examining whether ABR and OAE are related

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Table 2. Comparison between response and non-response groups

Parameter	Response group*	Non-response group†	*P*-value
Age (mean ± SD; years)	53.3 ± 15.1	61.5 ± 12.1	0.055
Hypertension (yes:no (*n*))	6:15	9:13	0.526
Diabetes mellitus (yes:no (*n*))	6:15	4:18	0.488
Affected side (mean ± SD)			
– Initial PTA (dB HL)	75.4 ± 19.0	69.0 ± 16.5	0.246
– ABR wave 1 amplitude (μV)	0.15 ± 0.08	0.10 ± 0.06	0.024‡
– ABR wave 5 amplitude (μV)	0.18 ± 0.06	0.22 ± 0.15	0.246
– ABR wave 1 latency (ms)	1.50 ± 0.20	1.77 ± 0.26	0.002‡
– Speech discrimination score (%)	38.4 ± 37.1	31.6 ± 32.3	0.536
Unaffected side (mean ± SD)			
– Initial PTA (dB HL)	15.2 ± 8.6	27.3 ± 12.7	0.001‡
– ABR wave 1 amplitude (μV)	0.18 ± 0.09	0.22 ± 0.39	0.643
– ABR wave 5 amplitude (μV)	0.18 ± 0.08	0.47 ± 0.32	0.323
– ABR wave 1 latency (ms)	1.47 ± 0.18	1.50 ± 0.19	0.695
– Speech discrimination score (%)	97.8 ± 3.3	93.4 ± 9.31	0.049‡

*$n = 21$; †$n = 22$. ‡$P < 0.05$, *t*-test and chi-square test. SD = standard deviation; PTA = pure tone average; ABR = auditory brainstem response

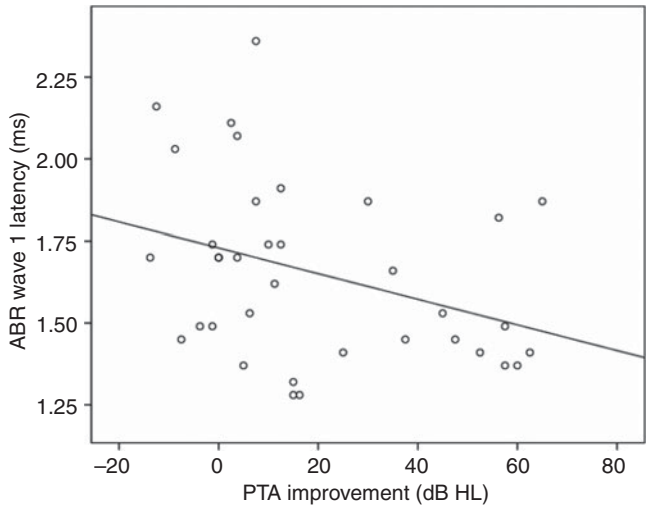

Fig. 1. Correlation between auditory brainstem response (ABR) wave 1 latency (on the affected side) and pure tone average (PTA) improvement (r = −0.349; $p = 0.037$).

to the prognosis of sudden SNHL concluded that the presence of the ABR waveform, but not OAE, was correlated with the prognosis of sudden SNHL.[15]

We hypothesised that retrocochlear lesions are present in patients with sudden SNHL, and that ABR wave 1 can reflect such lesions. It is not possible to obtain inner-ear tissue from patients with sudden SNHL, which makes it difficult to confirm our assumptions. Several studies have analysed the cochlea of sudden SNHL patients post-mortem.[16,17] They reported that hair cells and supporting cells in the organ of Corti[17] and apical ganglion[16] were significantly less abundant in the cochlea of patients with sudden SNHL. However, the time lag between the onset of sudden SNHL and the observation of the cochlea in these studies makes it impossible to infer causality. Additionally, these studies examined only changes in the cochlea, and did not account for whether retrocochlear lesions, including those of the auditory nerve, were involved.

Identifying the location of a lesion in sudden SNHL could guide new treatments and improve cure rates. However, it is difficult to determine whether more than one lesion is present, and differences in lesions among patients cannot be accounted for. If patients are grouped by various audiometry markers, such as OAE or ABR, and the hearing outcomes and responses to steroid treatment in each group are then measured, individualised and optimised treatment plans could be achieved in future.

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Competing interests. None declared

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