Auditory-pupillary responses in deaf subjects

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Abstract. Pupillary dilation in response to sound stimuli is well established and is generally considered to represent a startle reflex to sound. We believe that the auditory-pupillary response represents not only a simple startle reflex to sound stimuli but also represents a reaction to stimulation of other sense organs, such as otolith organs. Eight young healthy volunteers without a history of hearing and equilibrium problems and 12 subjects with bilateral deafness participated in this study. Computer pupillography was used to analyze the auditory-pupillary responses of both eyes in all subjects. We found that auditory-pupillary responses occurred even in subjects with bilateral deafness and that this response was comparable to those of normal subjects. We propose that the auditory-pupillary response also relates to vestibular function. Thus, assessing the auditory-pupillary response may be useful for evaluating the vestibulo-autonomic response in patients with peripheral disequilibrium.

Keywords: Auditory-pupillary response, vestibulo-autonomic response, bilateral deafness, vestibular function

1. Introduction

Pupillary dilation in response to sound stimuli is well established [19], and is generally considered to represent a startle reflex to sound, especially sounds related to emotions. This phenomenon is called the auditory-pupillary response (APR) [7]. APR mainly consist of biphasic response curves. Hirano et al. reported that application of a sympathetic blocking agent to an eye showing biphasic pupillary dilatation barely affected the first phase, but obviously inhibited the second phase of the APR [5]. They stated that the first phase of the response is caused by the inhibition of the parasympathetic nervous system, whereas the second phase is caused by the excitation of the sympathetic nervous system.

We believe that the APR represents not only a simple startle reflex to sound stimuli but may also represent a reaction to stimulation of other sense organs, such as otoliths. The present study was undertaken to clarify the relationship between the APR and vestibular and otolith organs.

2. Methods

Eight young healthy volunteers (4 males and 4 females; age range of 26-35 years; mean ± SD of 30 ± 4 years) without a history of hearing and equilibrium problems and 12 subjects with bilateral deafness (10 males and 2 females; age range of 23–72 years; mean ± SD of 51 ± 17 years) participated (Tables 1 and 2). All subjects underwent hearing tests (pure-tone audiogram), and 4 subjects underwent auditory brainstem response (ABR) testing. In addition, to clarify the relationship between APRs and vestibular function, especially otolith function, we performed caloric and vestibular-evoked myogenic response (VEMP) testing on the subjects.

Before examination, all subjects were placed in a dark room, where they rested for more than 10 minutes in a sitting position. During the actual test, the room was illuminated to 400 Lux. We chose 400 Lux be-
Table 1
Pupillary response data for normal subjects*

<table>
<thead>
<tr>
<th>Subject no.</th>
<th>Age</th>
<th>Sex</th>
<th>Basic pupillary diameter (mm)</th>
<th>M1 (msec)</th>
<th>D1 (msec)</th>
<th>PI 1</th>
<th>M2 (msec)</th>
<th>D2 (msec)</th>
<th>PI 2</th>
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<tr>
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<td>F</td>
<td>2.30</td>
<td>270</td>
<td>930</td>
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<td>↑</td>
<td>↑</td>
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<td>26</td>
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<td>750</td>
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<td>1070</td>
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<td>370</td>
<td>700</td>
<td>6.45</td>
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<td>1230</td>
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<td>4</td>
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<td>2.32</td>
<td>400</td>
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<td>1600</td>
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<td>M</td>
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<td>370</td>
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<td>370</td>
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<td>2.16</td>
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<td>2.97</td>
<td>770</td>
<td>1470</td>
<td>7.92</td>
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</table>

*Most normal subjects displayed biphasic pupillary dilation.
†Parameters were technically difficult to measure.

cause, at this intensity, the pupils remain physiologically composed, pupil dilation is negligible in the absence of stimuli, and pupil dilation is obvious with the presentation of sound stimuli [16]. Testing was performed between 2:00 PM and 5:00 PM because pupil size is stable during this period [16,18].

The auditory-pupillary responses of both eyes were assessed via computer pupillography. The testing was performed with a binocular infrared pupillograph designed and built at our institute (Fig. 1). After exposure to auditory stimuli, pupil size was measured with an infrared electronic ocular measurement device (NewOpt, Japan) and a data processor (NewOpt, Japan) in tandem with a portable computer (Windows XP).

Auditory stimuli consisted of impulsive stimuli or clicks. The clicks were rarefactive square waves (length: 0.1 ms; intensity: 100 dB nHL) generated by a Neuropack ∑ evoked-response measuring system (MEB-5500; Nippon Koden, Japan) and delivered through calibrated headphones (DR-531; ELEGA, Japan). For deaf patients with a hearing level greater than 100dB nHL, the intensity was set to less

Fig. 1. The measuring device. The device used to measure auditory-pupillary responses consists of an infrared electronic ocular measurement device (CCD camera), image manager, data control device, data processor, and Neuropack ∑. Sound stimulation and data recording were controlled by a portable computer. Each subject was fitted with goggles attached to a CCD camera.
Table 2
Pupillary response data for subjects with bilateral deafness*

<table>
<thead>
<tr>
<th>Subjects no.</th>
<th>Age</th>
<th>Sex</th>
<th>Cause of deafness</th>
<th>Caloric test†</th>
<th>ABR‡</th>
<th>Basic pupillary diameter (mm)</th>
<th>M1 (msec)</th>
<th>D1 (msec)</th>
<th>PI 1 (msec)</th>
<th>M2 (msec)</th>
<th>D2 (msec)</th>
<th>PI 2 (msec)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>23</td>
<td>M</td>
<td>progressive hearing loss since the age of 9 years (etiology unknown)</td>
<td>CP negative</td>
<td>Negative</td>
<td>††††††††††††††††††††††OUNTRY</td>
<td>2.68</td>
<td>††††</td>
<td>800</td>
<td>1230</td>
<td>4.48</td>
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</tr>
<tr>
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<td>30</td>
<td>M</td>
<td>progressive hearing loss since the age of 7 years (etiology unknown)</td>
<td>CP normal</td>
<td>Negative</td>
<td>††††††††††††††††††††††country</td>
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<td>††††</td>
<td>930</td>
<td>1300</td>
<td>2.94</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>33</td>
<td>M</td>
<td>after meningitis</td>
<td>CP irregular</td>
<td>–</td>
<td>††††††††††††††††††††††country</td>
<td>2.12</td>
<td>††††</td>
<td>970</td>
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<td></td>
</tr>
<tr>
<td>4</td>
<td>35</td>
<td>M</td>
<td>after meningitis</td>
<td>CP normal</td>
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<td>††††††††††††††††††††††country</td>
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<td>830</td>
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</tr>
<tr>
<td>5</td>
<td>47</td>
<td>F</td>
<td>bilateral sudden deafness (etiology unknown)</td>
<td>CP negative</td>
<td>–</td>
<td>2.96</td>
<td>270</td>
<td>670</td>
<td>2.70</td>
<td>1170</td>
<td>1600</td>
<td>2.70</td>
</tr>
<tr>
<td>6</td>
<td>52</td>
<td>F</td>
<td>after high fever of unknown origin</td>
<td>CP normal</td>
<td>–</td>
<td>2.30</td>
<td>100</td>
<td>470</td>
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<td>7</td>
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<td>F</td>
<td>after sepsis</td>
<td>normal negative</td>
<td>–</td>
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<td>CP normal</td>
<td>Negative</td>
<td>††††††††††††††††††††††country</td>
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<td>62</td>
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<td>–</td>
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<td>970</td>
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<td>M</td>
<td>after high fever of unknown origin</td>
<td>CP negative</td>
<td>–</td>
<td>2.22</td>
<td>200</td>
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<td>6.31</td>
<td>1530</td>
<td>2270</td>
<td>6.31</td>
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<tr>
<td>11</td>
<td>65</td>
<td>M</td>
<td>bilateral chronic otitis media</td>
<td>CP negative</td>
<td>–</td>
<td>2.00</td>
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<tr>
<td>12</td>
<td>68</td>
<td>M</td>
<td>bilateral Meniere’s disease</td>
<td>CP normal</td>
<td>–</td>
<td>2.44</td>
<td>370</td>
<td>630</td>
<td>1.64</td>
<td>1370</td>
<td>1630</td>
<td>1.64</td>
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</tbody>
</table>

*Most subjects with bilateral deafness displayed biphasic pupillary dilation. In the first phase, however, the latency of the subjects with bilateral deafness was significantly shorter than that of normal subjects.

†Parameters were technically difficult to measure.

CP – Canal palsy.

‡In the caloric test, if CP% was more than 25%, we diagnosed the subject as having CP. In the VEMP test, if responses were absent, we denoted them ‘negative.’ If responses were not absent but irregular, we denoted them ‘irregular.’ In the ABR test, if responses were absent, we denoted them ‘negative.’

than 90 dB nHL. Generally, APRs elicited by auditory and somatosensory stimulation disappear within 2.0 sec [15]. To avoid light reflex effects and trigger signal-associated contamination of the APR, we asked the subjects to open their eyes for 4 seconds before the delivery of each auditory stimulus. For subjects with bilateral deafness, we gently touched the patient’s arm or leg for 4 seconds before the delivery of each auditory stimulus to cue them to open their eyes. There was a possibility that this cue alerted the normal and deaf subjects. Therefore, we defined for both groups a pre-delivery waiting time of 4 sec. We recorded the diameter of both pupils for 3 seconds after the auditory stimulus was delivered. The subjects rested 10 seconds between each auditory stimulus delivery. This course was repeated 20 times. Before testing deaf subjects, we confirmed habituation to sound stimuli in some normal subjects and found that habituation develops after repeating the tests 3 or 4 times (Fig. 2). We reduced habituation and eye fatigue in the subjects by making them rest after every 3rd or 4th test stimulation. All responses were assessed according to the criteria listed below. We excluded responses that failed to meet the criteria, responses that could not be measured due to technical error, and responses in which a response curve could not be determined by habituation. The relevant responses were averaged and analyzed with a portable computer.
The response curves were not contaminated by irregular curves caused by nystagmus, blinking, or pupillary oscillations. We excluded pupillary-diameter data obtained immediately at the start of sound stimulation. In other words, response curves were preceded by a plateau.

2. Baseline pupillary diameter remained largely unchanged.

An example of the responses selected for analysis is shown in Fig. 3A.

This study was conducted in accordance with the Declaration of Helsinki. All procedures were carried out with the adequate understanding of the subjects and written consent and were approved by the review board of Tokyo Medical University (No. 802). We compared results obtained from the normal subjects and subjects with bilateral deafness. Calculations were performed with Stat Mate 3 software (Atoms, Japan). The Mann-Whitney U test was used for statistical analysis. $P < 0.05$ was considered significant.

3. Results

In the present study, APRs of the left and right eyes were nearly equal. Across all subjects, the above-
Fig. 3. A. Example of auditory-pupillary responses. Auditory stimulation elicited biphasic pupillary dilation. We recorded the diameters of both pupils and selected the most clear two recordings for analysis (in this example, the recording obtained from the left pupil was analyzed). B. Components of the auditory-pupillary response. Analysis involved averaging the response data with a portable computer. Once the data were averaged, we measured the basic pupillary diameter, latency of the initial mydriasis before the appearance of the first and second phase curves (M1 and M2), and dilation 1 (D1) and dilation 2 (D2). The amplitudes of the first and second phase curves were then converted into a pupillary index (PI) and analyzed.

In our comparison of normal subjects and subjects with bilateral deafness, the majority of subjects with bilateral deafness (8/12) exhibited biphasic pupillary dilation after auditory stimulation, while only 4 patients (4/12) exhibited monophasic pupillary dilation after auditory stimulation. The majority of normal subjects (5/8) also exhibited biphasic pupillary dilation, while only 3 subjects (3/8) exhibited monophasic pupillary dilation. In subjects with bilateral deafness, the latency of the first phase was significantly shorter ($P = 0.002$) and basic pupillary diameter was significantly wider ($P = 0.03$) than those in normal subjects. There was no significant difference, however, between the D1, D2, PI1, and PI2 values of the two groups.

When biphasic response curves of the auditory-pupillary response were obtained, it was difficult to measure the second phase, especially M2, because of physiological pupillary oscillations. Thus, in these cases, we did not measure D2 (Tables 1 and 2).

In normal subjects, the mean basic pupillary diameter was $2.15 \text{ mm} \pm 0.22$ (mean $\pm$ SD). All subjects responded to auditory stimulation; and in most subjects (5/8), auditory stimulation produced biphasic pupillary dilation. The mean latency of the initial mydriasis was $367 \text{ msec} \pm 51$, ranging from 300 to 430 msec ($n = 8$). The mean D1 was $706 \text{ msec} \pm 89$, ranging from 630 to 930 msec ($n = 8$, 14 eyes); and the mean D2 was $1455 \text{ msec} \pm 128$, ranging from 1230 to 1600 msec ($n = 5$, 6 eyes). The mean PI1 was $4.65 \pm 2.02$, ranging from 2.52 to 7.83 ($n = 8$, 14 eyes); and the mean PI2 was $4.10 \pm 2.75$, ranging from 0.84 to 7.92 ($n = 5$, 6 eyes) (Table 1).

On the other hand, in subjects with bilateral deafness, the mean basic pupillary diameter was $2.40 \text{ mm} \pm 0.31$ ($n = 12$, 16 eyes). All of these subjects responded to auditory stimulation; and in most of these patients (8/12), auditory stimulation produced biphasic pupillary dilation. The mean latency of the initial mydriasis was $236 \text{ msec} \pm 79$, ranging from 100 to 370 msec ($n = 10$, 13 eyes). The mean D1 was $749 \text{ msec} \pm 221$, ranging from 370 to 1130 msec ($n = 10$, 13 eyes); and the mean D2 was $1970 \text{ msec} \pm 339$, ranging from 1200 to 2270 msec ($n = 10$, 14 eyes). The mean PI1 was $5.12 \pm 3.67$ ($n = 10$, 13 eyes) and the mean PI2 was $4.69 \pm 2.58$ ($n = 10$, 14 eyes) (Table 2).
Fig. 4. Example of an auditory-pupillary response (APR) threshold in a bilaterally deaf subject. Bilaterally deaf subjects were subjected to 10db nHL-step decreases in sound pressure; response curves were recorded during each step and APR thresholds were examined. In this representative subject, responses were evoked by stimulation intensities of > 90db nHL but not by intensities of < 80db nHL. Black arrowheads point to the first phasic response curve; white arrowhead points to the second phasic response curve.

and 2 exhibited normal VEMP. In deaf subjects with bilaterally negative or irregular VEMP, the average PI 1 was 1.48, whereas in those with either unilateral negative or irregular VEMP or normal VEMP the average PI 1 was 4.7. Reduced vestibular function (as verified by caloric or VEMP testing) was characterized by a decreased PI 1.

4. Discussion

With respect to the latency of the initial mydriasis, Hirano et al. showed that the mean latency of the initial mydriasis in normal subjects ranges from 400 to 570 msec, and that D1 and D2 range from 700 to 1060 msec and from 1200 to 1710 msec, respectively [5]. The results of the present study were comparable to those of Hirano et al. [5]. Generally, the auditory-pupillary response is thought to be related to auditory function [15]. Surprisingly, in the present study, we found that APRs occurred even in subjects with bilateral deafness, indicating that the APRs may be related not only to auditory function but also to other functions, for example vestibular function. Other studies have also reported vestibular stimulation-related pupillary dilation [11,17]. Nishida et al. observed that rotation causes pupils to dilate in 80% of normal human subjects and normal rabbits [13]. However, rotation failed to cause pupil dilation in rabbits with bilateral inner-ear lesions [13]. On the basis of these findings, Nishida et al. concluded that pupillary responses are closely related to vestibular function [13]. These previous findings support our data.

In the present study, the average basic pupillary diameter of deaf subjects was significantly greater than that of normal subjects. This is possibly due to an autonomic imbalance that occurs in chronic vestibular dysfunction. Moreover, the latency of the first phase in deaf subjects was significantly shorter than that in normal subjects. The first phase of the response occurs as a result of inhibition of the parasympathetic nervous system [5]. Therefore, the latency of the first phase may be affected by chronic autonomic imbalance, especially in the parasympathetic nervous system.

There are several possible anatomical pathways involved in the auditory-pupillary response. Kubo et al. proposed the existence of vestibular axonal projections to the hypothalamus [6]. The existence of such a pathway was corroborated by Matsunaga et al., who observed that anterolateral hypothalamic stimulation increased the firing of vestibular units, whereas middle-medial hypothalamic stimulation mainly decreased the firing frequency of vestibular units [9]. Thus, pupillary dilation resulting from vestibular stimulation may occur through these pathways. According to Nakajima et al., inner ear disease-associated anisocorias occur due to a vestibular-sympathetic imbalance via the superior cervical sympathetic ganglion not due to a general autonomic imbalance [12]. They hypothesized that a cervical sympathetic nerve pathway reaches the dilator muscle of the pupils [12]. It thus appears that two vestibular nuclei-to-pupil pathways may exist: One pathway courses through the hypothalamus and the cervical sympathetic nerve and the other involves a direct pathway to the cervical sympathetic nerve (Fig. 5). The pathway mediating the auditory-pupillary response was generally thought to pass through the ascending reticular activating system and then through the hypothalamus [14]. Balaban indicated that in rabbits the vestibular nuclei may project to the Edinger-Westphal and anteromedian nuclei and proposed that these projections might be a substrate for vestibular influences on lens...
Fig. 5. Possible pathways mediating the auditory-pupillary response. Cells involved in the auditory-pupillary response mainly receive inputs from otolith organs. There are two pathways from the vestibular nuclei to the pupil: One pathway consists of vestibular projections to the cervical sympathetic nerve via the hypothalamus and the other pathway consists of direct vestibular projections to the cervical sympathetic nerve. In addition, vestibular nuclei may project to the Edinger-Westphal and anteromedian nuclei. With respect to the auditory-pupillary response, a possible pathway from the Edinger-Westphal and anteromedian nuclei to the pupil has yet to be identified. EW, Edinger-Westphal nucleus; AM, anteromedian nucleus; VTA, adjacent neurons in the ventral tegmental area.

accommodation, pupillary constriction, and regulation of intraocular circulation during postural changes and gravito-inertial challenges [1]. The auditory-pupillary response may have influenced the regulation of visual information during these changes. Moreover, Yates et al. reported that cells in the sympatho-regulatory region receive inputs from both otolith organs and semicircular canals, although inputs from the otolith organs predominate [20]. Of the vestibular end organs, it is the saccule that is most sensitive to sound [3,8,10,11]. Since the saccule lies just under the stapes footplate, it is in an ideal position to receive the full impact of a loud click delivered to the tympanic membrane. These previous findings led us to hypothesize that cells involved in the APRs mainly receive inputs from otolith organs, especially, the saccule.

Figure 4 shows an example of APR thresholds measured from a bilaterally deaf subject. In this subject, responses were evoked by stimulation intensities of > 90db nHL but not by intensities of < 80db nHL. The APR threshold in normal subjects was reported to be greater than 40dB nHL [5], suggesting that the APR threshold via the cochlea is over 40dB nHL. Colebatch et al. reported that the VEMP threshold is greater than 75dB SPL [2]. In the present study, the APR threshold was 90db nHL, which was closer to the VEMP threshold [2] than to the APR threshold via the cochlea. The smaller the VEMP, the smaller the APR (PI 1), suggesting that APRs could be evoked in ears with normal otolith function. Three subjects (nos. 3, 5, 10) with bilaterally absent VEMPs exhibited APRs. However, the APRs of two of the subjects (nos. 5, 10) were very weak. This means that an absent VEMP does not always equate with complete dysfunction. The APR of subject no. 3 was the same as that of normal subjects. Taken together, these results suggest that the pathway(s) underlying APRs receive inputs from otolith organs except the saccule. Moreover, APRs were present even in subjects with no ABR, indicating that APRs can be evoked by other types of stimulation, not only by auditory stimulation of the cochlea. Although previous studies suggest a correlation between the otolith organ and ABR thresholds [3,10], our study failed to find such a correlation between VEMPs and ABR thresholds. The next step is to closely compare APR threshold levels and ABRs in deaf subjects.

Generally, the pathway mediating the auditory-pupillary response related to auditory function was thought to pass through the ascending reticular activating system and then through the hypothalamus [15]. In bilateral deaf subjects, the reflex via this pathway can be less active than in normal subjects. Therefore in bilateral deaf subjects, other pathways related to vestibular function may be more active and may be different from the pathway related to auditory function. The possible anatomical pathways subserving the auditory-pupillary
response through the vestibular nuclei are shown in Fig. 5.

In summary, our study provides evidence that examining the auditory-pupillary response may be useful for evaluating the vestibulo-autonomic reflex. Therefore, we intend to use auditory-pupillary responses to examine the vestibulo-autonomic reflex of patients with peripheral disequilibrium, such as otolith dysfunction.

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References