

Brainstem encoding of speech in normal-hearing individuals with absent acoustic reflex

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The acoustic reflex test is an important tool for identifying auditory disorder from the middle ear to the superior olivary complex. Absence of acoustic reflexes is the early sign of many auditory disorders. Absence of acoustic reflex with normal hearing sensitivity may be an early sign of auditory neuropathy with poor encoding of speech at initial stage. Speech auditory brainstem response was recorded with /da/ (40 ms) stimuli in two groups of patients. The control group contained normal-hearing participants with presence of acoustic reflex, whereas the experimental group contained normal-hearing participants with absent acoustic reflexes. The peak latency, amplitude, and F0 and F1 mean amplitude were analyzed in both groups. MANOVA showed no significant difference in any parameter between the control and experimental group. Results of the current study showed that absence of acoustic reflexes in normal-hearing patients without auditory complaint is not sufficient by itself to diagnose the existence of auditory neuropathy. This study also highlighted that normal-hearing patients with absence of acoustic reflex have similar brainstem encoding of speech as that of patients with acoustic reflex.

Keywords:

acoustic reflex, auditory neuropathy, BioMark, brainstem response, speech auditory brainstem response

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Background

Loud sounds cause involuntary contraction of the stapedius muscle, called acoustic reflex. It involves either one or both middle ear muscles. It can be recorded ipsilaterally and contralaterally. If it is being recorded ipsilaterally, then recording should be done on the same side of the stimulus. In contralateral recording, the recording should be done on the opposite side of the sound stimulus [1]. The acoustic reflex is usually measured at 500, 1000, 2000, and 4000 Hz. At present, acoustic reflex is one of the important tests in audiology, as it helps in the assessment of afferent (sensorial) and efferent (motor) systems. These are the part of acoustic reflex pathway.

Recording of the acoustic reflex allows the professional to assess the middle ear up to the superior olivary complex. The acoustic reflex has many functions, such as enhancement of localization ability and sense of sound direction with the help of binaural interaction; it helps in improving auditory attention for continuous sounds; it improves speech intelligibility by separating hearing signals from background noise; it helps to know the variation in intensity level above threshold; it helps in reducing noise produced during chewing and movement of the mandible during speech; it participates in vocalization; and it helps in understanding speech through frequency selectivity and improves speech discrimination at higher intensities [2–7].

The normal value of the acoustic reflex threshold varies from 70 to 90 dBHL [8–9]. In clinical practice, if the acoustic reflex is absent even above 110 dBHL, it indicates an absence of the acoustic reflex [10]. In clinical practice we see patients without auditory complaints and with hearing threshold within the normal limit correlating with speech audiometry showing absent acoustic reflex responses at values above those expected on an A-type tympanogram. A study by Pinotti *et al.* [12] showed that 10% of normal-hearing individuals had absent acoustic reflex, without any ear complaints. These findings wherein absence of acoustic reflex occurs alone, with normal pure tone audiometry and speech audiometry, normal tympanometry, and presence of otoacoustic emission within normal patterns have aroused interest in research in these areas. Absence of AR under the above-mentioned conditions may be an indication of auditory neuropathy at initial stage. Disorder in the auditory nerve in auditory neuropathy may be the reason for absent acoustic reflex. In subjects with auditory neuropathies, because of lack of synchronous firing, the auditory nerve may not be able to process sound, which is required for the

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activation of middle ear muscles to activate the acoustic reflex [11]. One of the most important objective tests that help in diagnosing auditory neuropathy is auditory brainstem response (ABR) audiometry. Several studies have been conducted on such cases using ABR, which suggest the presence of an abnormal synchronization or absence of peak in the waveform. The study by Pinotti *et al.* [12] revealed that ABR audiometry using click stimuli in seven patients with no auditory complaints, normal hearing, and A-type tympanogram with absence of acoustic reflex showed the presence of waves I, III, and V, with absolute latency values and interpeak latencies within the normal standard, which suggests that absence of stapedial reflexes alone in normal-hearing patients, without any auditory complaint, is not sufficient by itself to diagnose the existence of auditory neuropathy. The studies revealed poor processing to speech sound in case of auditory neuropathy [13–16]. Speech ABR in patients with no auditory complaints, with normal hearing, and with an A-type tympanogram with the absence of acoustic reflex has not been documented yet. Such a study would give insight into the processing capability of speech sound in patients with auditory neuropathy. There is a need to study brainstem encoding of speech sound in individuals with absent acoustic reflex with normal pure tone audiometry correlating well with speech audiometry, as well as normal tympanometry and presence of otoacoustic emission.

Materials and methods

Participants

The study consisted of 24 age-matched individuals (48 ears) aged 18–26 years who were divided into two groups. Group I comprised 12 healthy adults (24 ears) (seven males and five females) having A-type tympanogram with presence of reflex, and group II consisted of 12 adults (24 ears) (seven males and five females) having A-type tympanogram with absence of acoustic reflex.

Inclusion criteria

Patients aged 18–26 years with no otological and auditory complaints, normal hearing sensitivity below 15 dBHL for octaves from 250 to 8000 Hz, correlated SRT with PTA, and presence of otoacoustic emission were eligible for the study. The above audiological testing was carried out by experienced audiologists. All participants had normal otological and otoscopic examination findings, with no middle ear pathology, evaluated by an experienced otolaryngologist. All had 'A' type tympanogram but the only difference between group I and group II was the presence of ipsilateral and contralateral reflexes in

both ears in group I, which was absent in group II. All participants were well informed about the study and written consent was taken before the study.

Instrumentation

An AC-40 Interacoustics (Drejervaenget 8, DK-5610 Assens) dual-channel, clinical audiometer was used for measuring pure tone thresholds. Live speech and tones were presented through earphones (Telephonics, TDH-39, 815 Broad Hollow Road, Farmingdale, New York 11735, USA) mounted in supra-aural cushions (MX-51/AR). Calibration of the audiometer was performed according to the American National Standard Institute (ANSI). (2004). American National Standard specification for Audiometer, ANSIS3.6-2004. New York: ANSI. GSI Tymstar Immittance Audiometry (Grason-Stadler, 7625 Golden Triangle Drive, Suite F, Eden Prairie MN 55344) was used with a middle ear analyzer using 226 Hz probe frequency. Ipsilateral and contralateral reflexes for both ears were assessed at 500, 1000, 2000, and 4000 Hz. ECHO Scan screening MAICO OAE instrument (MAICO Diagnostics, 10393 West 70St, Eden Prairie, MN 55344) was used for Distortion product evoked otoacoustic emissions elicited using an 80 dB SPL peak equivalent (peak) level click, present in five successive trials, where L1-65 dB and L2-55 dB was set and a signal-to-noise ratio of at least 6 dB with a reproducibility score of at least 70% , in frequency band of 2 to 5 kHz. Biologic Navigator Pro was used for recording speech ABR.

Procedure

Pure tone thresholds were obtained using the modified version of the Hughson and Westlake procedure to establish normal hearing sensitivity. Air conduction thresholds were obtained for octave frequencies between 250 Hz and 8 kHz, whereas bone conduction thresholds were measured at octave frequencies between 250 Hz and 4 kHz. SRT and SIS were obtained using spondee words and phonetically balanced words, respectively. These tests were administered in participants' native language using live monitored speech. To rule out any middle ear pathology, immittance audiometry and reflexometry were carried out. Immittance audiometry was performed using a 226 Hz probe tone, whereas reflexometry included obtaining ipsilateral and contralateral acoustic reflexes at 500, 1000, 2000, and 4000 Hz with the same 226 Hz probe tone. Biologic Navigator pro (version 7) (Natus Medical Incorporated, One Bio-logic Plaza, Mundelein, IL 60060) was used to record click-evoked ABR to check the integrity of the neural pathway at the levels of the brainstem before measuring the speech-evoked ABR. Speech-evoked ABR was recorded for all participants with speech stimuli /da/ of 40 ms duration produced using a KLATT synthesizer (Klatt, 1980) [17]. Silver chloride electrodes

were used to record the responses. Recording was done monaurally and ipsilaterally with electrodes at the vertex (noninverting), at the ipsilateral mastoid (Inverting), and at the contralateral mastoid (ground). The absolute electrode impedance and interelectrode impedance were maintained below 5 and 2 k Ω , respectively. At least two recordings of 3000 sweeps to rarefaction polarity at a rate of 10.9/s were collected. The responses were amplified 100 000 times and weighted. The sum of the two recordings was taken for analysis. Time window of 64 ms, including 10 ms prestimulus time, was used. The responses were band pass-filtered online between 100 and 3000 Hz. The stimuli were presented through Etymotic ER-3A (Etymotic Research, Inc. 61 Martin Lane Elk Grove Village, IL 60007) earphones and the intensity level was 80 dB SPL (Table 1).

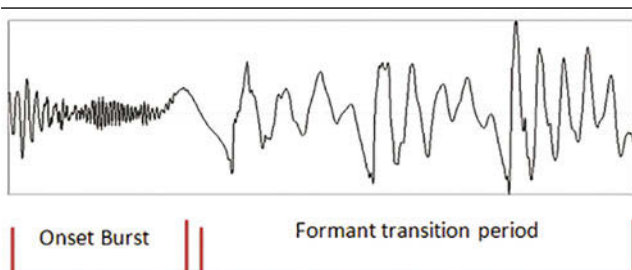
Test environment

All audiological evaluations were carried out in sound-treated rooms (ANSI S3.1, 1999). Pure tone audiometry and speech audiometry were carried out in a two-room audiological setup, whereas immittance audiometry and click-evoked and speech-evoked ABR were carried out in a single-suit audiological setup.

Stimulus and recording parameters

The speech stimulus /da/ was used to record speech-evoked ABR, which is a synthesized speech syllable of 40 ms produced using the KLATT synthesizer. This stimulus has fast temporal information and broad spectral characteristic of stop consonants. This stimulus also has spectrally reaching formant transition between vowel and consonants. The voicing for this stimulus begins at 5 ms, with onset noise burst during the first 10 ms. The fundamental frequency of /da/ stimulus is from 103 to 125 Hz, increasing in a linear manner. The first formant of /da/ (F1) increases from 220 to 720 Hz, whereas the second formant (F2) of /da/ decreases from 1700 to 1240 Hz over the duration of the stimulus. The third formant (F3) falls slightly from 2580 to 2500 Hz, whereas the fourth (F4) and fifth formants (F5) remain the same at 3600 and 4500 Hz, respectively. Figure 1

Figure 1



Time domain waveform of /da/ stimulus.

shows the time domain waveform and Fig. 2 shows the spectral waveform of /da/ stimulus used in the present study.

Measurement of fundamental frequency (F0) and first formant frequency (F1)

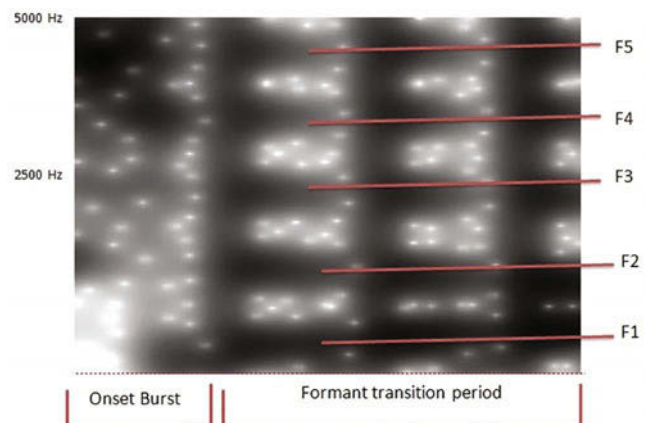
Frequency following response has energy mainly at its harmonics and at fundamental frequency. We performed Fast Fourier analysis of waveform from 11.5 to 46.5 ms. We measured activity occurring between 103 and 121 Hz, which corresponds to fundamental frequency (F0) of speech, and between 220 and 279 Hz, which corresponds to first formant frequency (F1). The subject response should be above noise floor to enable the analysis. This was ascertained by comparing the spectral magnitude from prestimulus condition to poststimulus condition – that is, response. If the magnitude of the fundamental frequency (F0) or first formant frequency (F1) was greater than or equal to 1, response was considered to be present.

Table 1 Stimulus and acquisition parameters for click and speech-evoked auditory brainstem response

Stimulus	Click-evoked ABR	Speech-evoked ABR
Duration	100 μ s	40 ms
Intensity	80 dBnHL	80 dB SPL
Polarity	Rarefaction	Rarefaction
Repetition rate	11.1/s	10.9/s
Number of sweeps	1500	3000
Analysis time	10 ms	64 ms
Pre stimulus time	2 ms	10 ms
Filter setting	100–3000	100–3000
Electrode placement	Noninverting: vertex Inverting: test ear Ground: opposite ear	Non inverting: vertex Inverting: test ear Ground: opposite ear
Transducers	Insert earphone (ER-3A)	Insert earphone (ER-3A)

ABR, auditory brainstem response

Figure 2



Spectral waveform of /da/ stimulus.

Results

The present study aimed to evaluate and compare the speech-evoked brainstem responses between groups I and II. Mean, SD, and significance values of the data in each group were calculated. The data obtained were subjected to statistical analysis using SPSS software (Version 17, Chicago, USA). Independent *t*-test was applied to find out the statistical difference, if any.

The response measures that were considered for analysis were V, A, D, E, F, and O peaks. These measures were divided into transient peaks (V, A, and O) and sustained peaks (D, E, and F). The mean latencies of the transient peaks V, A, and O and of the sustained peaks D, E, and F in both groups were obtained. The mean latencies and amplitude of response peaks obtained from group I were compared with the response measures obtained from group II. Multivariate analysis of variance (MANOVA) was used to find out the level of significance. Descriptive statistics were used to analyze the results (Tables 2 and 3, Graph 1).

MANOVA was used for within-group and between-group comparisons for the latency measures of waveforms V, A, D, E, F, and O. The results revealed that there were no significant differences between the two groups for latencies of wave V [$F(1, 47) = 1.44$; $P = 0.24$; partial $\eta^2 = 0.025$], wave A [$F(1,47) = 3.01$; $P = 0.09$; partial eta squared = 0.05], wave D [$F(1,47) = 0.22$; $P = 0.66$; partial $\eta^2 = 0.004$], wave E [$F(1,47) = 1.25$; $P = 0.26$; partial $\eta^2 = 0.03$], wave F [$F(1, 47) = 0.29$; $P = 0.67$; partial $\eta^2 = 0.003$], and wave O [$F(1,47) = 1.49$; $P = 0.30$; partial $\eta^2 = 0.028$].

Measurement of amplitude of F0 and F1 indicated that the mean amplitude of F0 was greater than the mean amplitude of F1 (Table 4).

MANOVA was carried out compare control and experimental groups for F0 and F1 amplitude. The test revealed no significant difference for F0 amplitude [$F(1,47) = 3.62$; $P = 0.09$; partial $\eta^2 = 0.103$] and F1

amplitude [$F(1,47 = 2.28$; $P = 0.98$; partial $\eta^2 = 0.01$] (Figs 3–5 and Graph 2).

Discussion

Results of the current study showed no significant difference between control and experimental groups in terms of the latency and amplitude measures of speech ABR. The result of the current study showed that there was no significant difference in the amplitude

Table 2 The mean and SD of the latency of different peaks of speech-evoked auditory brainstem response of the two groups

Peaks of SABR	Control (AR present)		Experimental (AR absent)	
	Mean (ms)	SD (ms)	Mean (ms)	SD (ms)
V	6.23	0.23	6.25	0.22
A	7.69	0.26	7.71	0.21
D	22.97	0.84	22.92	0.89
E	31.52	0.85	31.63	0.72
F	39.91	1.03	39.24	0.98
O	44.25	0.35	44.28	0.38

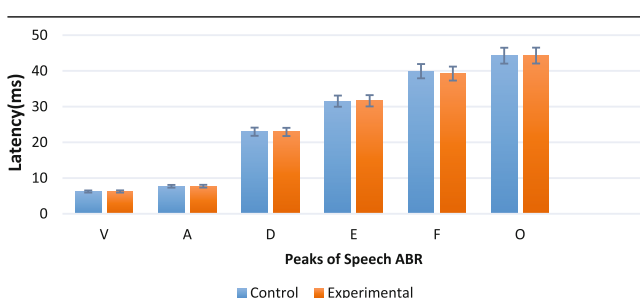
Table 3 The mean and SD of the amplitude (μV) of different peaks of speech-evoked auditory brainstem response of the two groups

Peaks of SABR	Control (AR present)		Experimental (AR absent)	
	Mean (μV)	SD (μV)	Mean (μV)	SD (μV)
V	0.19	0.12	0.20	0.13
A	-0.22	0.09	-0.21	0.10
D	-0.36	0.11	-0.32	0.09
E	-0.34	0.08	-0.35	0.10
F	-0.29	0.04	-0.28	0.07
O	-0.18	0.03	-0.19	0.06

Table 4 The values of the amplitude of the fundamental and first formant frequency

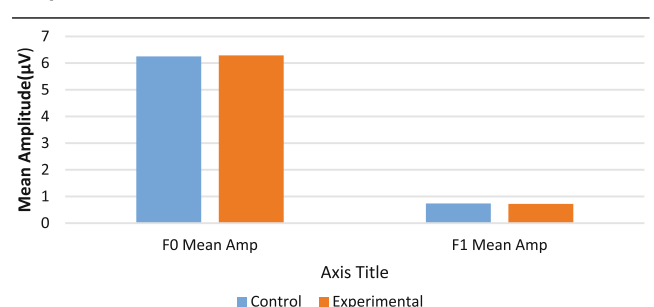
Formant Frequency	Control (AR present)		Experimental (AR absent)	
	Mean	SD	Mean	SD
F0	6.25	2.10	6.29	2.50
F1	0.74	0.20	0.72	0.26

Graph 1



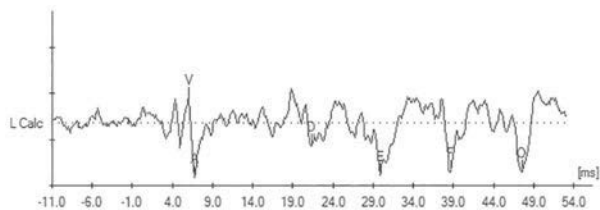
Latency of different peaks of speech auditory brainstem response in the control (AR present) and experimental (AR absent) group.

Graph 2



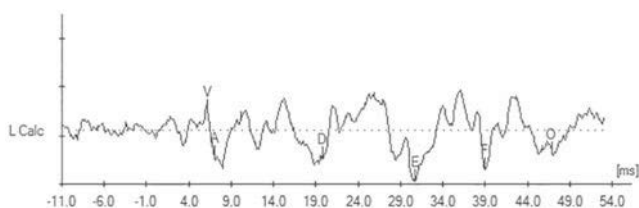
Graph of F0 and F1 mean amplitude of the control (AR present) and experimental (AR absent) group.

Figure 3



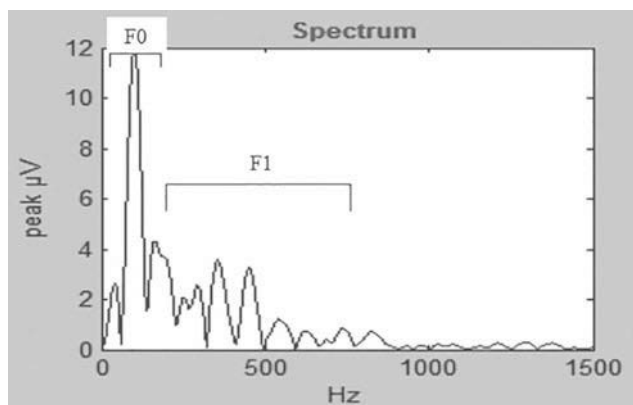
Sample waveform of the control group (acoustic reflex present).
Waveform of speech auditory brainstem response of the two groups.

Figure 4



Sample waveform of the experimental group (acoustic reflex absent).
Waveform of speech auditory brainstem response of the two groups.

Figure 5



Analysis of F0 and F1. Response indicates that only the fundamental frequency and first formant frequency (F0 = 103–121 Hz; F1 = 220–720 Hz) were measurable.

of fundamental frequency (F0) between the control and experimental group. Similar findings were seen for amplitude of first formant frequency (F1) also. There was no significant difference in the coding of F1 between control and experimental groups. Result of the current study also showed that normal-hearing subjects with absence of acoustic reflex have similar brainstem encoding of speech as that of patients with acoustic reflex. In a study by Berlin *et al.* [18] of 136 patients with auditory neuropathy, none showed normal reflexes at any frequency. Reflexes were either absent or observed above 100 dBHL, which was not congruent with their normal optoacoustic emissions.

Pinotti *et al.* [12] also reported that ABR audiometry using click stimuli in seven patients with no auditory complaints, normal hearing, and an A-type tympanogram with the absence of acoustic reflex showed presence of waves I, III, and V, with absolute latency values and interpeak latencies within the normal standards, which suggests that the absence of stapedial reflexes in normal-hearing patients without auditory complaint is not sufficient by itself to diagnose the existence of auditory neuropathy.

The finding of the current study also suggests that normal-hearing subjects with absence of acoustic reflex, without any auditory complaint, have similar encoding of speech at the brainstem level, and absence of acoustic reflex in normal-hearing patients without auditory complaint is not sufficient by itself to diagnose the existence of auditory neuropathy.

Conclusion

The present study showed that brainstem encoding of speech is similar in patients with absent acoustic reflex without any auditory complaint when compared with normal-hearing subjects with acoustic reflex.

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Conflicts of interest

There are no conflicts of interest.

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