Assessment of auditory dysfunction as an extra-articular manifestation in rheumatoid arthritis using brainstem auditory-evoked potential

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Abstract

Objectives
The paper aims to study brainstem auditory-evoked potential parameter changes in rheumatoid arthritis (RA) patients, and their relation to the duration of illness, degree of disease activity, functional stage of the disease, and intensity of pain.

Materials and methods
Control (group 1) comprised 25 healthy female patients of age 50–60 years, while the study group (group 2) comprised 25 female patients with RA of more than 5 years. Proven cases of RA (according to american college of rheumatology (ACR) 2010 criteria) underwent brainstem auditory-evoked potentials. The recording was carried out by using RMS EMG EPMK2. In the RA group, the following parameters were collected: number of painful and swollen joints, intensity of pain in the joints of patients evaluated by a visual analog scale (VAS) of 0–100 mm, speed sedimentation rate, a disease activity index, and functional disability of patients.

Results
In this study, we found in the right ear, a highly significant difference in mean peak latency of wave I between groups 1 and 2 with \( P < 0.01 \); also, there was a significant difference in the mean peak latency of waves IV and V between groups 1 and 2 with \( P < 0.05 \). As regards the mean interpeak latency from I to III, there were highly significant differences between groups 1 and 2 with \( P < 0.01 \). For the left ear, there was a highly significant difference in mean peak latencies of waves I and V between groups 1 and 2 with \( P < 0.01 \), while there was a significant difference in mean peak latency of wave II between groups 1 and 2 with \( P < 0.05 \). The mean differences in interpeak latencies of waves I and II and III–V between groups 1 and 2 were highly significant \( P < 0.00 \). Estimation of the correlation coefficients for the parameters of brainstem auditory-evoked potential, and the indices of disease activity, showed statistically significant correlations with the functional class and intensity of pain by the VAS. Also, there was a significant correlation between the absolute peak latency of wave I and interpeak latency of waves I–III and disease duration with \( P < 0.05 \).

Conclusion
The authors conclude that RA causes delayed latencies and alteration of the waves of brainstem auditory-evoked potentials due to hearing affection.

Keywords: Autoimmune inner-ear disease, brainstem auditory-evoked potential, rheumatoid arthritis
distribution [1]. The disease onset is most frequent during the fourth and fifth decades of life, with 80% of patients developing the disease between 35 and 50 years of age [2].

Many organ systems could be affected in RA patients, including the auditory system. Sensorineural hearing loss (SNHL), conductive hearing loss (CHL), or mixed type has been recorded in RA patients. Currently, the pathogenesis of hearing disability in RA is not distinctly recognized [3]. RA can involve the ependymal bone and incudostapedial joints altering the ossicular mechanics in response to static air-pressure modifications. These joints are true diarthroses and therefore subject to the same rheumatic lesions as any other articulation in the body and could make CHL [4].

In addition, RA can produce an autoimmune response to the sensitive cells of the inner ear; the inflammatory cells can enter the cochlea from the circulatory system through the spiral modiolar vein leading to SNHL. SNHL is a collection of common auditory disorders resulting from dysfunction of the inner ear, auditory nerve, or the auditory-processing pathway in the central nervous system [5].

Brainstem auditory-evoked potential (BAEP) is a sensitive and specific method to diagnose retrocochlear hearing loss, demyelination, and other diseases of brain stem, so it is important to be used for early detection of auditory defects associated with RA patients. BAEP are potentials recorded from the ear and vertex in response to a brief auditory stimulus to assess the conduction through the auditory pathway up to the midbrain [6]. Sound is converted into an electrical impulse and passes from the cochlea to the auditory cortex. Any type of hearing alteration, such as conductive or sensorineural, for instance, results in changes on record of this potential. RA is thought to affect the auditory system through different mechanisms causing these different types of Hearing defects (7).

According to the literature, the alterations occur on BAEP record in cases of CHL, where an increase of latency values of waves I, III, and V with normal interpeaks I–III, III–V, and I–V can occur [8]. Also, in other studies, SNHLs in high frequencies of cochlear origin affect the morphology of BAEP waves and retro-cochlear dysfunction [9].

The BAEP is formed by seven waves. I, III, and V are the most visible ones. In relation to the place of origin of these waves, the most accepted classification nowadays is I—distal portion at the brainstem of the hearing nerve, II—proximal portion at the brainstem of the hearing nerve, III—cochlear nucleus, IV—superior olivary complex, V—lateral lemniscus, VI—inferior colliculus, and VII—medial geniculate body [10].

Severe hearing impairment in RA patients is significantly associated with having a hearing-related disability and self-reported communication difficulties [11]. Social and psychological well-being in those patients may be profoundly affected. The auditory system manifestations and complications of RA disease may be prevented or reduced with early detection and effective treatment [12].

**Aim**

The paper aims to study BAEP changes in RA patients, and their relation to the duration of illness, degree of disease activity, functional disability, and intensity of pain.

**Patients and Methods**

The study protocol was approved by the Ethnic Committee of GONI. Our prospective study included 50 patients selected from an outpatient clinic in Mataria Teaching Hospital. Our participants were divided into two groups: one group that included 25 RA patients was diagnosed on clinical and radiological basis according to ACR/EULAR 2010 classification criteria [13], with mean age of 50.58 ± 0.93 years and disease duration of more than 5 years, and the second group included 25 healthy individuals with mean age of 49.41 ± 1.08 years as the control group. Patients with history of the intake of drugs with known ototoxicity, patients with history of ear discharge and deafness, renal diseases, hepatic diseases, chronic respiratory diseases, diabetes mellitus, uncontrolled hypertension, and pregnant or lactating mothers were excluded.

Full history and clinical examination were done. In the RA group, the following parameters were collected: number of tender and swollen joints, intensity of joint pain evaluated by a visual analog scale (VAS) of 0–100 mm, a disease activity index using Disease Activity Score (DAS28) calculated from number of swollen joints (out of 28) and number of tender joints (out of 28), and number of swollen joints (out of 28), and number of tender joints, erythrocyte sedimentation rate. The score may range from 0 to 9.3, where a DAS28 score less than or equal to 3.2 is mild disease activity; greater than or equal to 3.2 and less than or equal to 5.1 is moderate disease activity; and greater than or equal to 5.1 is severe disease activity [14]. Functional activity of patients was assessed using Health Assessment Questionnaire (HAQ) score [15].

Otological examination in the outpatient clinic in Mataria Teaching Hospital included examination of preauricular region, ear pinna, postauricular region, external acoustic canal, and tympanic membrane.

Both studied groups underwent the study of BAEP.

**Brainstem auditory-evoked potential test (BAEP)**

It was conducted using the Intelligent Hearing System (Smart EP windows USB version 3.91) with insert earphones ER-3A. Responses were collected with silver chloride electrodes and were differentially recorded from Cz (active) to the ipsilateral mastoid (reference), with a common grounding electrode placed on the forehead. The patients were instructed to lie comfortably in the supine position and relax to promote a passive recording condition. No other stimulus was used during the tests. Frequency following response recording: the frequency following response was evoked by 1024 sweeps and 60 dBHL tone bursts at 500 Hz using rarefraction polarity, with a 5000-µs rise and fall time in a trapezoidal envelope and a 15 000-µs duration at a rate of 5.1 per sec. The response
was filtered between 30 and 3000 Hz in a graduated analysis window of 50 msec. The response cycles obtained for the study group were compared with those obtained for the control group. Auditory brainstem response recording: the auditory brainstem response was evoked by 1024 clicks with a 100-μs duration using a rarefaction polarity and a rate of 37.1 clicks per sec. A high-pass filter of 100 Hz and a low-pass filter of 3000 Hz were used on a 12.5-msec graduated window of analysis. Waveforms were obtained in both ears at 70 dBHL, and waves I, III, and V were identified. The absolute latencies of waves I, III, and V and interpeak latencies I–III, III–V, and I–V were obtained and compared between the study and control groups.

**Statistical analysis**

All tabulated data were expressed as mean ± SD. Comparisons between patients and control groups were done by using Student’s t test. For all statistical tests, significance was done using the correlation coefficient (r) test in which significance is defined as the level of probability “P” value of < 0.05. Computations were done using an SPSS statistical program version 12 and graphs were assessed using Microsoft excel XP version.

**RESULTS**

We studied 25 healthy females as a control group and 25 rheumatoid patient groups (23 were females and 2 were males) with mean disease duration of 11.2 ± 3.6. Both groups were with mean age of 54.1 ± 2.4 and 54.0 ± 3.1 years, respectively, with no statistically significant difference (P = 0.091).

The results of tests of functional ability of the patients (RA group) using the HAQ showed average values of 2.85 ± 0.06, 4.18 ± 0.85, and 2.2 ± 3.08, respectively [Table 5, Fig. 4].

In our study, we found that the frequency of altered BEAP is 45 ears (90%) of RA patients studied with delayed latency of wave I in 22 (44%), of wave II in 8 (16%), of wave III in 8 (16%), of wave IV in 7 (14%), and of wave V in 9 (16%). It decreased the amplitude of wave Ia in 24 (48%) and of wave Va in 45 (90%) of RA patients. It also increased interpeak latency between III and V in 25 (50%) and in 10 (20%) of RA patients. There is a statistically significant difference in the frequencies of all BEAP parameters between control and RA groups with P > 0.05.

In our study, we found in the right ear, a highly significant difference in peak latency of wave I in groups 1 and 2 with (P < 0.01), as seen in Table 1. Also, there was a significant difference in the peak latency of waves IV and V of groups 1 and 2 with (P < 0.05). While for the rest of the waves, it was insignificant with (P > 0.05). There were significant differences in the amplitudes of waves I—al and V—aV in groups 1 and 2, with P < 0.05 [Table 2].

<table>
<thead>
<tr>
<th>Wave</th>
<th>Control group n=25</th>
<th>Patients’ group n=25</th>
<th>P</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave I</td>
<td>1.38±0.39</td>
<td>1.82±0.28</td>
<td>0.000</td>
<td>HS</td>
</tr>
<tr>
<td>Range</td>
<td>1-1.88</td>
<td>1.5-2.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave II</td>
<td>2.85±0.06</td>
<td>2.77±0.23</td>
<td>0.089</td>
<td>NS</td>
</tr>
<tr>
<td>Range</td>
<td>2.8-3</td>
<td>2.2-3.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave III</td>
<td>3.92±0.11</td>
<td>4.00±0.49</td>
<td>0.449</td>
<td>NS</td>
</tr>
<tr>
<td>Range</td>
<td>3.8-4.08</td>
<td>3.5-2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave IV</td>
<td>5.11±0.06</td>
<td>5.30±0.44</td>
<td>0.030</td>
<td>S</td>
</tr>
<tr>
<td>Range</td>
<td>5-5.1</td>
<td>4.72-6.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave V</td>
<td>5.75±0.04</td>
<td>6.06±0.63</td>
<td>0.018</td>
<td>S</td>
</tr>
<tr>
<td>Range</td>
<td>5.7-5.8</td>
<td>5.43-6.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amplitude I—La (μv)</td>
<td>0.28±0.14</td>
<td>0.18±0.03</td>
<td>0.04</td>
<td>S</td>
</tr>
<tr>
<td>Range</td>
<td>0.14-0.42</td>
<td>0.15-0.21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amplitude V—Va (μv)</td>
<td>0.47±0.2</td>
<td>0.32±0.23</td>
<td>0.03</td>
<td>S</td>
</tr>
<tr>
<td>Range</td>
<td>0.27-0.67</td>
<td>0.09-0.55</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 1: Values of the estimates of disease activity in the rheumatoid arthritis group**

<table>
<thead>
<tr>
<th>Indices of disease activity</th>
<th>Mean±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease duration (y)</td>
<td>11.2±3.6</td>
</tr>
<tr>
<td>Disease activity (DAS28)</td>
<td>4.18±0.85</td>
</tr>
<tr>
<td>HAQ</td>
<td>2.82±0.56</td>
</tr>
<tr>
<td>Intensity of pain (VAS) mm</td>
<td>46.5±11.92</td>
</tr>
</tbody>
</table>

DAS28, Disease Activity Score; HAQ, Health Assessment Questionnaire; VAS, visual analog scale.

<table>
<thead>
<tr>
<th>RT ear latency</th>
<th>Control group n=25</th>
<th>Patients’ group n=25</th>
<th>P</th>
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<tr>
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<td></td>
<td></td>
</tr>
<tr>
<td>Wave III</td>
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<td>4.00±0.49</td>
<td>0.449</td>
<td>NS</td>
</tr>
<tr>
<td>Range</td>
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<td>3.5-2</td>
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<td>S</td>
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<td>Range</td>
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</table>

P<0.05: nonsignificant (NS). P<0.05: significant (S). P<0.01: highly significant (HS). *Independent t-test.

The mean differences in interpeak latencies of waves (I–III) of groups 1 and 2 were highly significant (P < 0.00) (Table 3, Figs 1 and 2).

In the left ear, there was a highly significant difference in mean peak latencies of waves I and V of groups 1 and 2 with (P < 0.01), while there was a significant difference in mean peak latency of wave II of groups 1 and 2 with (P < 0.05), but for rest of the waves, there was an insignificant difference in the mean peak latencies with (P > 0.05). There were insignificant differences in the amplitudes of all waves I—al and V—aV in groups 1 and 2 with P > 0.05 (Table 4, Fig. 3).

The mean differences in interpeak latencies of waves (I–III) and (III–V) of groups 1 and 2 were highly significant (P < 0.00) (Table 5, Fig. 4).

There is a significant positive correlation between disease duration and peak latency of wave I and interpeak latency...
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of waves I–III with \((r = 0.47^* \text{ and } -0.65^*\), respectively, and \(P > 0.05\)). Also, there are statistical significant correlations between BEAP-studied parameters and DAS28, functional activity (HAQ), and pain severity (VAS), with \(P > 0.05\), as shown in Table 6.

**DISCUSSION**

RA is a systemic inflammatory autoimmune disease of unknown etiology that affects \(~ 1\%\) of the worldwide population. Females are more commonly affected than males, usually between 30 and 50 years of age [16]. RA is characterized by the presence of chronic symmetric polyarthritis of mainly the small joints that leads to progressive irreversible damage of articular and periarticular structures, deformities, and functional impairment, and inversely affect the quality of life [17]. RA may affect many-body systems, including heart, lung, skin, and eye. In this regard, the possibility of auditory system
contention in RA has been one of the domains with great concern. Raut et al.[18] studied 35 patients with RA with 35 age- and sex-matched controls to assess the relation between RA and hearing disability; the finding revealed a considerable hearing disability at 500 Hz, 1.0 kHz, and 2.0 kHz in patients with RA.

After exposure of the inner ear in RA patients to either local or systemic immunization of the antigen, this will generate immune responses through the inflammatory cells entering the scala tympani through the spiral modiolar vein and causing labyrinthitis. The ensuing labyrinthitis results in physiologic dysfunction, loss of sensory cells, and ultimately fibrosis and osteoneogenesis within the cochlea [19].

The incudomalleolar and incudostapedial joints are synovial in type. These joints can be affected by rheumatoid changes. Autoimmune ear injury can involve the auricle, external auditory canal, middle ear, and the inner ear. Inner-ear involvement is termed autoimmune inner-ear disease (AIED) [20].

McCabe defined AIED as a rapidly progressive (weeks to months) bilateral SNHL that responds to the administration of immunosuppressive agents [21].

The aim of our study is to study the role of BAEP in early detection of the sub clinical cases of auditory dysfunction in RA patients and the correlation between BAEP parameters and disease duration and activity.

In our study, we found that the frequency of altered BEAP is detected in 45 ears (90%) of RA patients studied with delayed latency of wave I in 22 (44%), of wave II in 8 (16%), of wave III in 8 (16%), of wave IV in 7 (14%), and of wave V in 9 (16%). It decreased the amplitude of wave Ia in 24 (48%) and of wave Va in 45 (90%) of RA patients. It increased the interpeak latency between III and V in 25 (50%), and in 10 (20%) of RA patients. There is a statistically significant difference in the frequencies of all BEAP parameters between control and RA groups with \( P > 0.05 \).

Our results were in agreement with those of other studies where they confirmed that SNHL has been reported as the

\[
\begin{array}{|c|c|c|c|}
\hline
\text{LT ear latency} & \text{Control group} & \text{Patients’ group} & \text{\( P \)} & \text{Sig.} \\
\hline
\text{Wave I} & 1.3±0.39 & 1.92±0.25 & 0.00 & \text{HS} \\
\text{Range} & 1.88 & 1.45-2.2 & & \\
\text{Wave II} & 2.86±0.06 & 3.01±0.30 & 0.01 & \text{S} \\
\text{Range} & 2.8 & 2.4-3.5 & & \\
\text{Wave III} & 3.92±0.11 & 3.99±0.37 & 0.38 & \text{NS} \\
\text{Range} & 3.8-4.08 & 3.55-5.08 & & \\
\text{Wave IV} & 5.11±0.06 & 5.24±0.41 & 0.11 & \text{NS} \\
\text{Range} & 5.2 & 4.72-6.1 & & \\
\text{Wave V} & 5.8±0.04 & 6.10±0.50 & 0.00 & \text{HS} \\
\text{Range} & 5.7-5.8 & 5.33-6.60 & & \\
\text{Amplitude I—Ia (μv)} & 0.29±0.11 & 0.25±0.10 & 0.06 & \text{NS} \\
\text{Range} & 0.18-0.40 & 0.15-0.35 & & \\
\text{Amplitude V—Va (μv)} & 0.43±0.23 & 0.39±0.23 & 0.23 & \text{NS} \\
\text{Range} & 0.20-0.66 & 0.17-0.62 & & \\
\hline
\end{array}
\]

\( P>0.05: \text{nonsignificant (NS). } P<0.05: \text{significant (S). } P<0.01: \text{highly significant (HS). } *\text{Independent t-test.} \)

\[
\begin{array}{|c|c|c|c|c|}
\hline
\text{LT ear interpeak latency} & \text{Control group} & \text{Patients’ group} & \text{Test value} & \text{\( P \)} & \text{Sig.} \\
\hline
\text{I-III} & 2.62±0.53 & 2.05±0.38 & 4.366 & 0.000 & \text{HS} \\
\text{Range} & 1.9-3.87 & 1.55-3 & & \\
\text{III-V} & 1.83±0.14 & 2.11±0.39 & −3.487 & 0.001 & \text{HS} \\
\text{Range} & 1.6-2 & 1.4-2.8 & & \\
\text{I-V} & 4.37±0.40 & 4.11±0.71 & 1.623 & 0.111 & \text{NS} \\
\text{Range} & 3.87-4.8 & 1.68-5.45 & & \\
\hline
\end{array}
\]

\( P>0.05: \text{nonsignificant (NS). } P<0.05: \text{significant (S). } P<0.01: \text{highly significant (HS). } *\text{Independent t-test.} \)

\[
\begin{array}{|c|c|c|c|c|c|c|c|c|}
\hline
\text{Indices of disease activity} & \text{Wave I} & \text{r} & \text{\( P \)} & \text{Wave III} & \text{r} & \text{\( P \)} & \text{Wave V} & \text{r} & \text{\( P \)} & \text{I-III} & \text{r} & \text{\( P \)} & \text{III-V} & \text{r} & \text{\( P \)} & \text{I-V} & \text{r} & \text{\( P \)} \\
\hline
\text{Duration} & 0.47* & 0.01 & −0.25 & 0.21 & −0.16 & 0.42 & −0.65* & 0.0 & 0.03 & 0.8 & −0.3 & 0.07 \\
\text{DAS28} & 0.44 & 0.02 & 0.47 & 0.01 & −0.38 & 0.05 & 0.031 & 0.88 & −0.38 & 0.05 & 0.47 & 0.01 \\
\text{HAQ} & −0.42 & 0.02 & −0.231 & 0.26 & −0.16 & 0.43 & 0.47 & 0.01 & 0.03 & 0.88 & −0.6* & 0.0 \\
\text{VAS} & −0.38 & 0.05 & 0.44 & 0.02 & −0.38 & 0.05 & −0.14 & 0.48 & 0.42 & 0.02 & −0.6* & 0.0 \\
\hline
\end{array}
\]

0.05+significant. BAEP, brainstem auditory-evoked potential; DAS28, Disease Activity Score; HAQ, Health Assessment Questionnaire; VAS, visual analog scale.

Table 4: Comparison between rheumatoid arthritis patients and controls according to the latencies and amplitudes of left (LT) ear

Table 5: Comparison between rheumatoid arthritis patients and controls according to interpeak latencies of left (LT) ear

Table 6: Correlation of disease duration, disease activity index, functional activity, and VAS scores with the BAEP studied parameters
most common hearing impairment in RA, with a prevalence of ~12–80%, following by CHL and mixed hearing loss [22]. Özcan et al. [23] investigated hearing- and middle-ear functions in 37 patients with RA and 35 controls to study the prevalence and the nature of hearing loss in RA. The results of this study showed a higher prevalence of an abnormal tympanogram in RA patients. In 1980, Reiter et al. [24] measured the middle-ear immittance in RA patients. In their study, immittance data disclosed abnormal discoveries in 59% of the patients. The discrepancies between these findings may be due to differences in the mean age and the number of samples.

In our study of the right ear, we observed significant delay in absolute peak latency of waves I, II, and V of BAEP with \( P < 0.00 \), and in the left ear, we observed significant delay in absolute peak latency of waves I, IV, and V when RA patients were compared with controls, which is in agreement with the study of Shelja et al. [25], who showed that in the right ear, the difference in the absolute peak latency of wave III of groups 1 and 2 was significant, while for the rest of the waves, it was insignificant. In the left ear, the difference in absolute peak latency of waves I, IV, and V of groups 1 and 2 was significant, while for the rest of the waves, it was insignificant \( (P > 0.05) \). The differences in interpeak latencies (I–III, III–V, and I–V) were insignificant \( (P > 0.05) \), when controls were compared with RA patients.

Our findings were confirmed by other authors, where auditory brainstem responses (ABRs) were recorded using 105-dB click stimulation, they found a statistically significant increase in the wave I latency of ABRs in RA patients compared with controls. They evaluated the prevalence and features of hearing impairment in 28 RA patients [26].

In our study of the right ear, there were significant differences in the amplitudes of waves I—aI and V—aV in RA patients compared with controls, with \( P < 0.05 \). The mean differences in the interpeak latencies of waves (I–III) between both groups were statistically significant. In the left ear, there were insignificant differences in the amplitudes of all waves I—aI and V—aV between both groups with \( P > 0.05 \). The mean differences in the interpeak latencies of waves (I–III) and (III–V) between both groups were highly significant \( (P < 0.00) \).

Similar results were observed in other studies where they found that in the right ear, there was no significant \( (P > 0.05) \) change in interpeak latencies I–III, III–V, and I–V when controls were compared with RA patients. There was no significant \( (P > 0.05) \) change in the amplitude of I—la and V—aVa when controls were compared with RA patients. In the left ear, there was a significant \( (P < 0.05) \) change in the amplitude of I—la the in left ear when controls were compared with RA patients. There was an insignificant \( (P > 0.05) \) change in the amplitude of V—aVa when controls were compared with RA patients [25].

In our study, there was a significant correlation between the disease duration of RA patients and hearing affection, which is similar to the study of Ozturk et al. [3], where they determine a positive significant correlation between disease duration and mean hearing threshold value in the left-ear studies.

Controversially, Arslan et al. in 2011 [27] examined the relation between the incidence of hearing loss and duration of disease in 44 RA patients with mean age of 47.2 ± 11.2. The results of this study showed no relation between the incidence of hearing loss and duration of disease.

Also, in 2016, the authors investigated hearing in RA population. They carried out their study on fifty-three patients with RA and 71 patients with an indigenous condition of health who were matched for age and sex. In their study, no correlation was found between those diagnosed with SNHL using PTA and the duration of the disease [20]. The discrepancies between these findings may be due to differences in the mean age and the number of samples.

In our study, there are statistical significant correlations between BEAP-studied parameters and disease-activity score DAS28, functional activity (HAQ), and pain severity (VAS), with \( P > 0.05 \).

A significant association, especially at high frequencies, between hearing impairment and disease activity, was detected in a study by Yildirim et al. [28], where the audiometric results of 62 patients with active disease were compared with 26 patients in remission. DAS28-C-Reactive Protein (DAS28-CR) was measured based on a count of 28 tender joints, 28 swollen joints, patients’ global assessment, and laboratory results of CRP, which is widely used in clinics.

**Conclusion**

In conclusion, the present study showed that the frequency of hearing affection in the RA group was significantly more than the control group. RA causes increased latencies of the waves of BAEP. Therefore, the disease could directly interfere in neurotransmission of the auditory pathway or indirectly through altering certain processes that modulate brainstem auditory activity. Accordingly, audiological assessment should be considered in routine evaluation of patients with RA, to prevent hearing-related handicap.

**Conflicts of interest**

None.

**References**

Ismaiel, et al.: Assessment of auditory dysfunction


