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RESEARCH ARTICLE

ASSESSMENT OF HIDDEN HEARING LOSS IN NOISE EXPOSED WORKERS

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Abstract

Aim: To evaluate hidden hearing loss (HHL) in industrial workers using the extended high frequency audiometry(EHFA) and ABR wave I amplitude during routine follow-up visits to the audiology clinic to identify and managehidden hearing loss as early as possible.

Materials and Methods: This study is comprised of two groups: study group which consisted of 50 adultindustrial workersattending the audio-vestibular medicine clinic for their routine follow-up visits and control group which consisted of 20 adults with bilateral normal peripheral hearing in the frequency range of 250-8000 Hz, including 3 and 6 kHz without any history of occupational noise exposure. Both groups were age and sex-matched.

Results: Results showed statistically significant differences between patient and control groups as regards thresholds of EHFA at 12 and 16 KHz. In addition, there was statistically significant reduction in ABR wave I amplitudes between the two groups.

Conclusion: Combining extended high-frequency audiometry and ABR amplitude measurements as two methods for early detection of audibility declines upon exposure to noise as well as an early warning of hair cell damage and loss is advised.

Introduction:-

The most frequent preventable hearing impairment and the second most common cause of hearing loss after presbycusis is noise-induced hearing loss (NILH) (Mehrparvar et al., 2014). A permanent threshold shift (PTS) can occur after exposure to high intensity sounds, resulting from loss of cochlear sensory hair cells, causing an irreversible increase in hearing thresholds. On the other hand, it's well known that temporary thresholds shift is caused by an acoustic exposure that causes reversible damage to hair cell stereocilia and cochlear nerve endings. Recent studies claimed that TTS might end up affecting the synapses between the inner hair cells and fibers of the cochlear nerve without damaging the hair cell or causing any change in hearing thresholds (Liberman, 2016; Lobarinas et al., 2017). This selective synaptopathy following exposure to loud noises was named hidden hearing loss. It's believed that Cochlear synaptopathy may present with tinnitus, hyperacusis, or a reduction in the ability to understand speech despite a normal audiogram (Kujawa& Liberman2009).

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NIHL can be prevented but is permanent and irreversible (Mehrparvar et al., 2014). Pure tone audiometry at frequencies ranging from 250 to 8000 Hz is a typical procedure for determining NIHL (Schmuziger et al. 2007). Otoacoustic emissions and EHFA are two other suggested methodsin the literature for the early detection of NIHL. Yeend et al. are the authors who initially coined the phrase hidden hearing loss (HHL). Despite having normal hearing thresholds, HHL describes a reduction in speech perception in noisy environments. According to recent research, the lesion may be caused by harm to the outer hair cells or synaptic damage to the inner hair cells' nerve fibres (Taylor, 2019; Bharadwaj et al., 2019).

OHCs damage can exist without a rise in hearing thresholds, according to several studies (Bharadwaj et al., 2019; Smith et al., 2019; Yeend et al., 2017). Also, exposure to loud noise generated synaptopathy without any impact on hearing thresholds (Bharadwaj et al., 2019; Taylor, 2019; Yeend et al., 2017).

To identify HHL, a standard audiometric examination is insufficient. HHL can be evaluated by OAE, electrocochleography, auditory brainstem response (ABR), and EHFA (Barbee et al., 2018; Tepe et al., 2017).

Although EHFA cannot predict speech-in-noise performance, poor speech-in-noise performance correlates substantially with EHFA thresholds. By lowering the number of hair cells not specified in standard audiometry and providing a mechanism to identify people with HHL, EHFA may aid identification (Le Prell, 2018).

According to animal research, Kujawa and Liberman (2009) hypothesized that speech in noise challenges are caused by noise-induced cochlear synaptopathy, which results in decreased sound encoding.

Recent studies have pointed out the occurrence of cochlear synaptopathy with normal hearing thresholds using ABR measurements. In several studies, ABR wave I/wave V ratios anddiminished auditory brainstem response (ABR) have been linked to noise exposure (Stamper & Johnson 2015; Bramhall et al. 2017; Grose et al. 2017). A correlation between ABR wave I amplitude and frequent or ongoing tinnitus in young veterans of the military has also been shown (Bramhall et al., 2018).

But there hasn't been enough evidence to prove a connection between noise exposure, synapse loss, and deficiencies in suprathreshold sound processing in people with clinically normal audiograms.

This study aims to evaluate HHL in industrial workers using the EHFA and ABR wave I amplitude during routine follow-up visits to the audiology clinic to identify and manage HHL as early as possible.

Subjects And Methods:-

Subjects were classified into two groups:

Study group:

Consisted of 50 adult industrial workers attending the audio-vestibular medicine clinic for their routine follow-up visits. All subjects had bilateral normal peripheral hearing in the frequency range of 250-8000 Hz, including 3 and 6 kHz. This was defined as a hearing threshold level equal to or below 25 dBHL and bilateral normal middle ear function. The exclusion criteria were those older than 50 years of age, presence of hearing loss, disorders of the middle ear, history of psychological or neurological problems, history of neck problems and a history of previous ototoxic medication.

Control group:

consisted of 20 adults with bilateral normal peripheral hearing in the frequency range of 250-8000 Hz, including 3 and 6 kHz without history of occupational noise exposure. All subjects had bilateral normal middle ear function. They were age and sex-matched to the study group.

Methods:-

Informed consent was taken from each patient. All subjects were subjected to the following:

1. Complete history taking and otoscopic examination
2. Impedance testing: tympanometry and ipsilateral acoustic reflexes
3. Pure tone audiometry and speech audiometry were done using AC40 by interacoustics audiometer. Air conduction was done for the frequency range of 250-8000 Hz. Frequencies of 3 and 6 kHz were also tested to

exclude audiograms with minor dips at those frequencies. Bone conduction audiometry was done for the frequency range of 500-4000 Hz.

4. EHFA Thresholds of 12 and 16 kHz
5. ABR was recorded ipsilaterally with the positive recording electrode on the forehead, the reference electrode on the ipsilateral mastoid and the ground electrode on the contralateral mastoid. Rarefaction click stimuli were presented at 80 dBnHL, at a rate of 21.1/sec, through TDH-49p headphones. A total number of 1000 sweeps were obtained, and a low pass filter with a cut-off frequency of 3000 Hz and a high pass filter with a cut-off frequency of 100 Hz were used. ABR waveforms were analyzed, and wave I amplitudes were compared.

Statistical analysis:

Data was analyzed by IBM® SPSS® v.28. Descriptive statistics were done for quantitative data as median, minimum and maximum of the range and the mean ± SD (standard deviation) for the quantitative data. Inferential analyses were done for non-parametric quantitative variables using the Mann-Whitney method. Correlation coefficients were calculated using Spearman's correlation analysis. p values not more than 0.05 were considered statistically significant.

Results:-

EHFA thresholds between groups

The study recruited 50 patients and 20 controls.

Table 1:- Comparison between patients and controls as regards the PTA results.

Frequency		Control (n=20)					Patient (n=50)					p-value
		Mean	SD	median	Min	Max	Mean	SD	median	Min	Max	
Right	8Khz	16.00	5.76	15	5	25	19.60	4.61	20	10	25	0.016
	12 KHz	30.75	5.45	30	20	40	42.00	11.16	40	30	70	<0.001
	16 KHz	43.50	6.30	45	35	55	53.20	9.68	55	25	75	<0.001
Left	8 KHz	16.00	5.03	15	5	25	18.30	5.40	20	5	25	0.107
	12 KHZ	32.25	6.38	30	25	45	39.30	13.59	35	20	75	0.037
	16 KHz	43.00	6.16	40	35	55	54.50	10.46	55	30	75	<0.001

Mann-Whitney U test was conducted to compare controls and patients as regards the EHFA results. There were statistically-significant differences between patients and controls with regard to PTA ($p < 0.05$) in RT and LT ears at all tested frequencies except at 8 KHz in left ears.

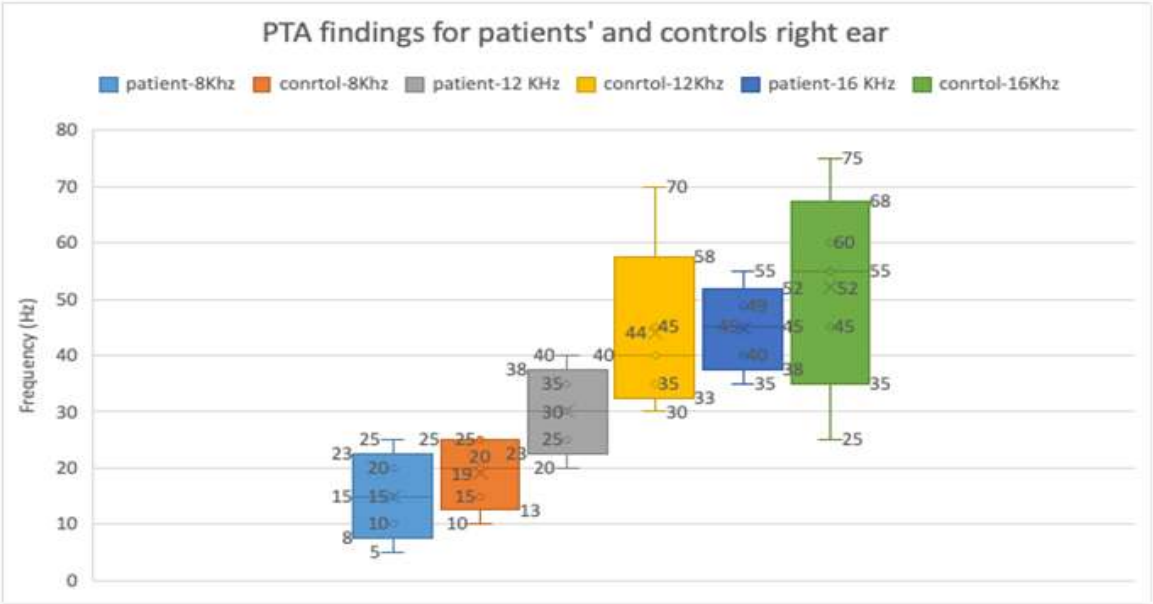

Figure 1:- Box-plot illustrating the PTA results for the right ear of both patients and controls at 8 KHz, 12 KHz and 16 KHz.

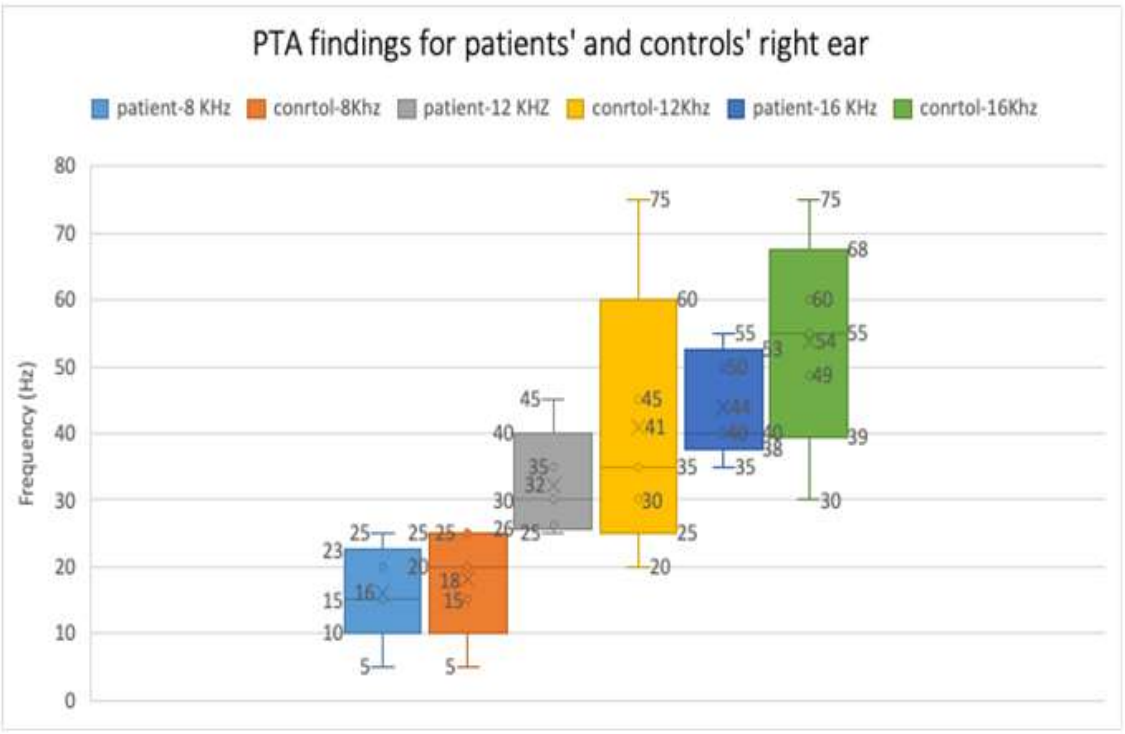

Figure 2:- Box-plot illustrating the PTA results for the left ear of both patients and controls at 8 KHz, 12 KHz and 16 KHz.

Table 2:- Comparison between patients and controls as regards waveI amplitude.

Wave I amplitude	Control					Patient					p-value
	Mean	SD	median	Min	Max	Mean	SD	median	Min	Max	
Right ear	0.252	0.032	0.25	0.19	0.30	0.16	0.036	0.165	0.10	0.24	<0.001
Left ear	0.254	0.034	0.26	0.21	0.35	0.17	0.03	0.17	0.12	0.23	<0.001

Mann-Whitney U test was used to compare the wave I amplitude between patients and controls. There were statistically significant differences between both groups for both right and left ears; with the patients sowing less median amplitude (0.165, 0.17) than the controls (0.25, 0.26) for right and left ear respectively.

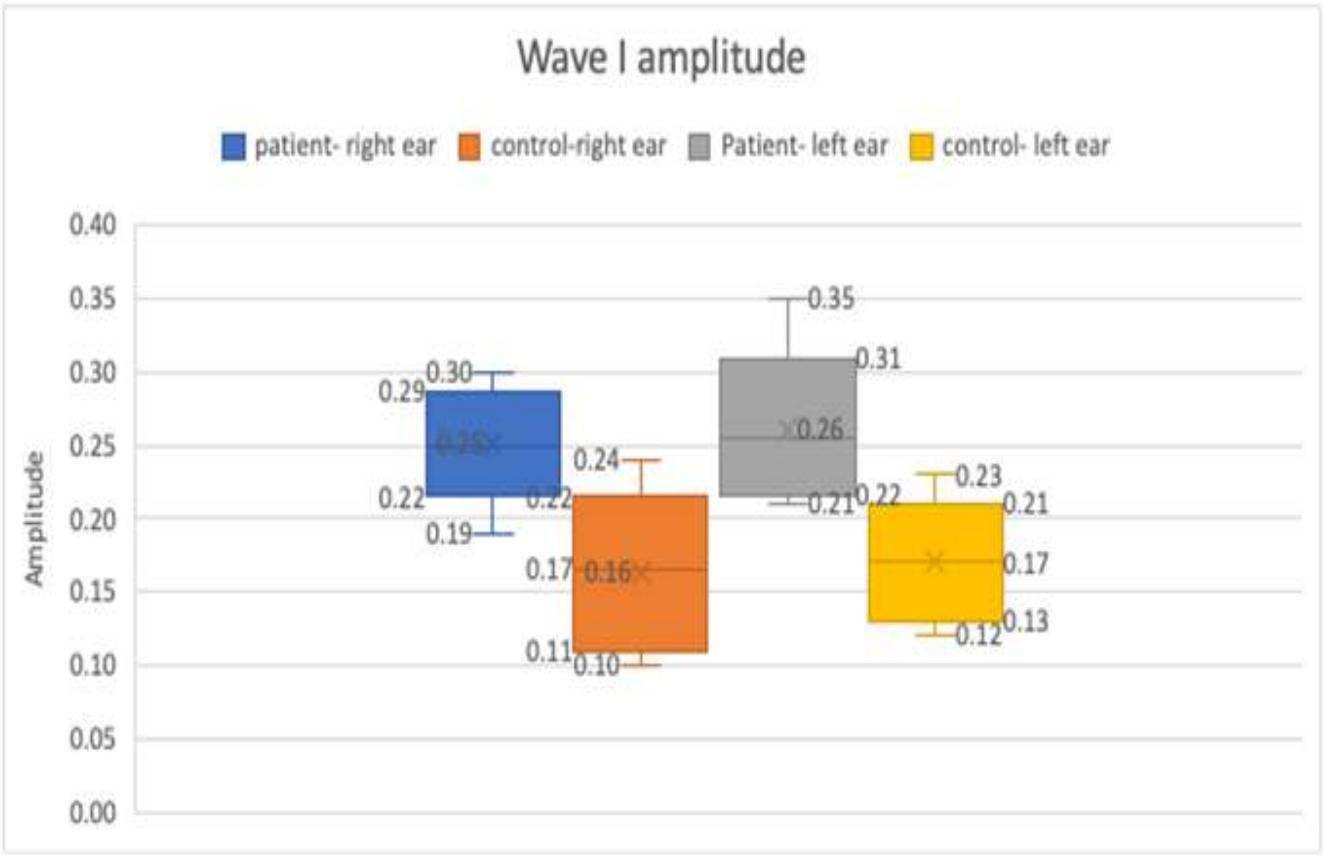

Figure 3:- Box plot illustrating wave I amplitude for both patients and controls (right and left ears).

Age and EHFA thresholds

Table 3:- Correlation between age and EHFA results.

	Frequency	Spearman's rho	p-value
Right	8Khz	0.355	0.011
	12 KHz	0.605	<.001
	16 KHz	0.54	<.001
Left	8 KHz	0.456	0.012
	12 KHZ	0.455	<.001
	16 KHz	0.612	<.001

Spearman's correlation showed statistically significant positive correlation between age and EHFA findings especially with 12 and 16 KHz.

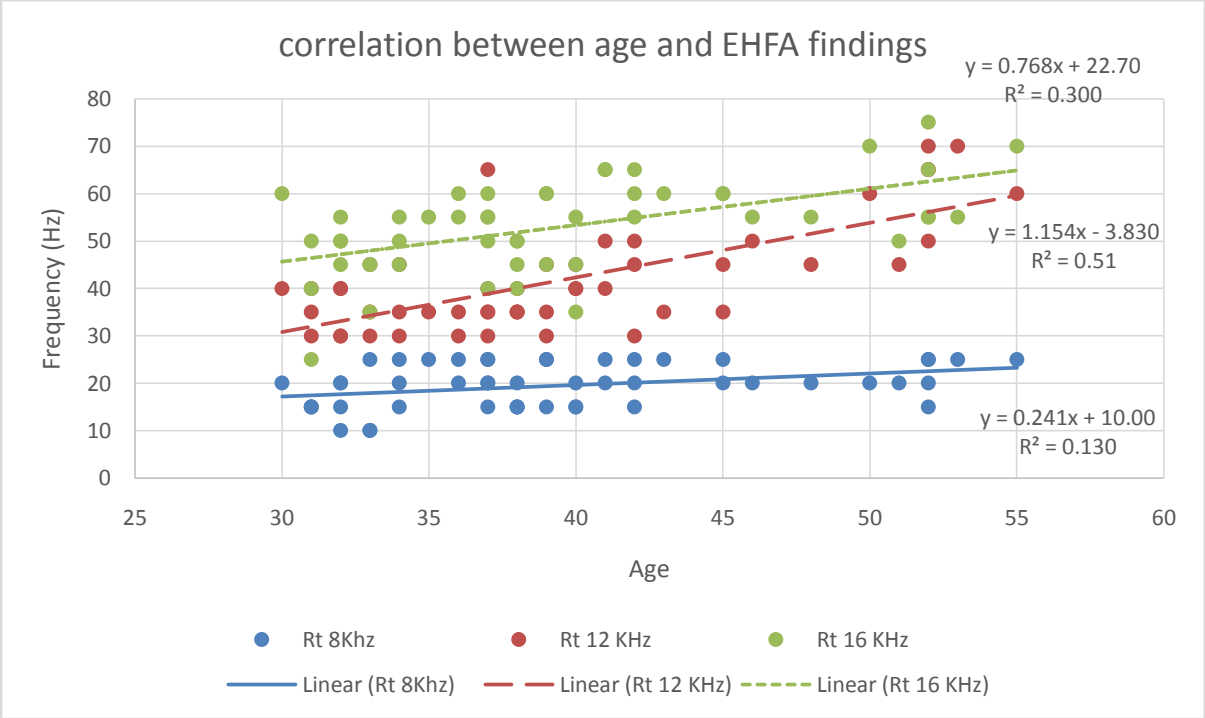

Figure 4:- Scatterplot demonstrating the correlation between age and EHFA findings for the right ear.

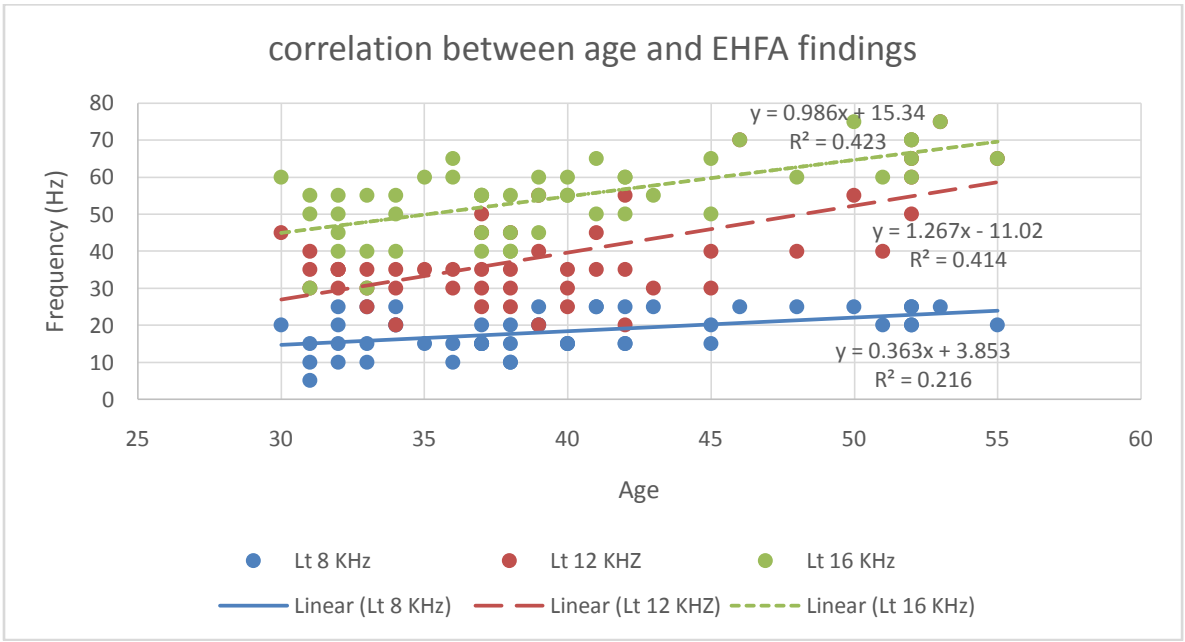

Figure 5:- Scatter plot demonstrating the correlation between age and EHFA findings for the left ear.

Table 4:- Correlation between age and wave I amplitude.

Wave I Amplitude	Spearman's rho	p-value
Right	-0.619	<.001
Left	-0.585	<.001

Using Spearman's correlation, there is statistically significant relation between age and wave I amplitude (p-value < 0.05).

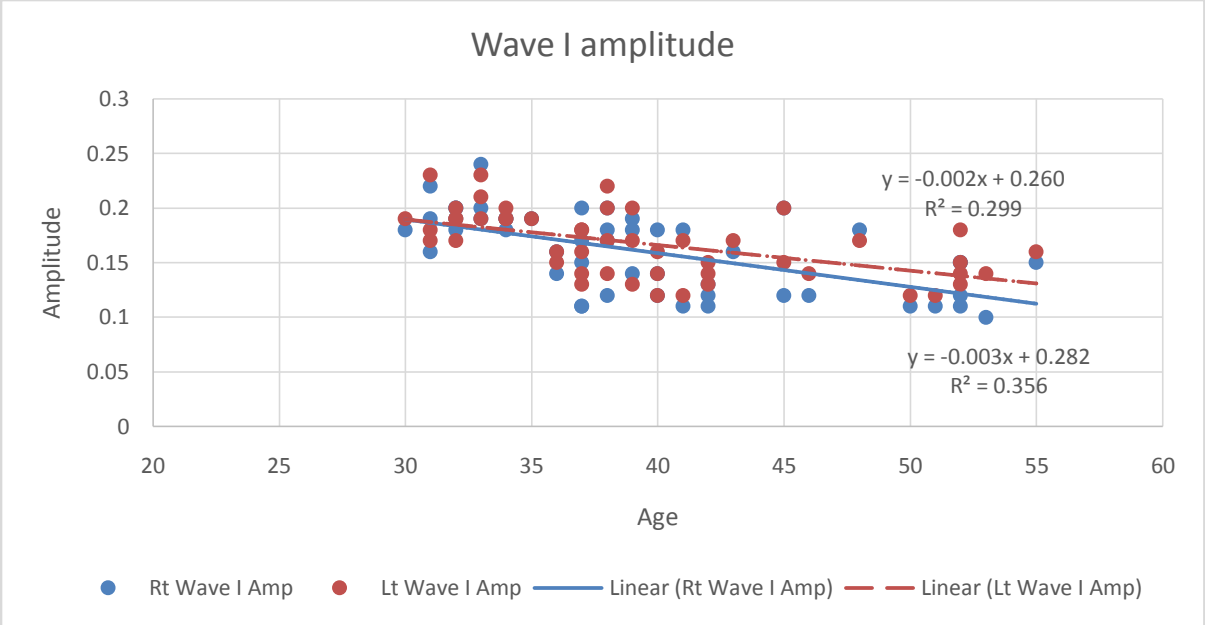

Figure 6:- Scatter plot demonstrating the correlation between age and wave I amplitude for right and left ears.

Duration of noise exposure and EHFA, wave I amplitude

Table 5:- Correlation between duration of noise exposure and EHFA findings.

	Frequency	Spearman's rho	p-value
Right	8 Khz	0.279	0.049
	12 KHz	0.624	<0.001
	16 KHz	0.527	<0.001
Left	8 KHz	0.405	0.004
	12 KHZ	0.475	<0.001
	16 KHz	0.630	<0.001

Spearman's correlation showed statistically significant positive correlation between duration and EHFA findings (p-value <0.05).

There is weak positive relation between duration of noise exposure and patients' thresholds at 8 KHz. Meanwhile; there is strong positive correlation between duration of noise exposure and patients' thresholds at 12 kHz and 16 kHz.

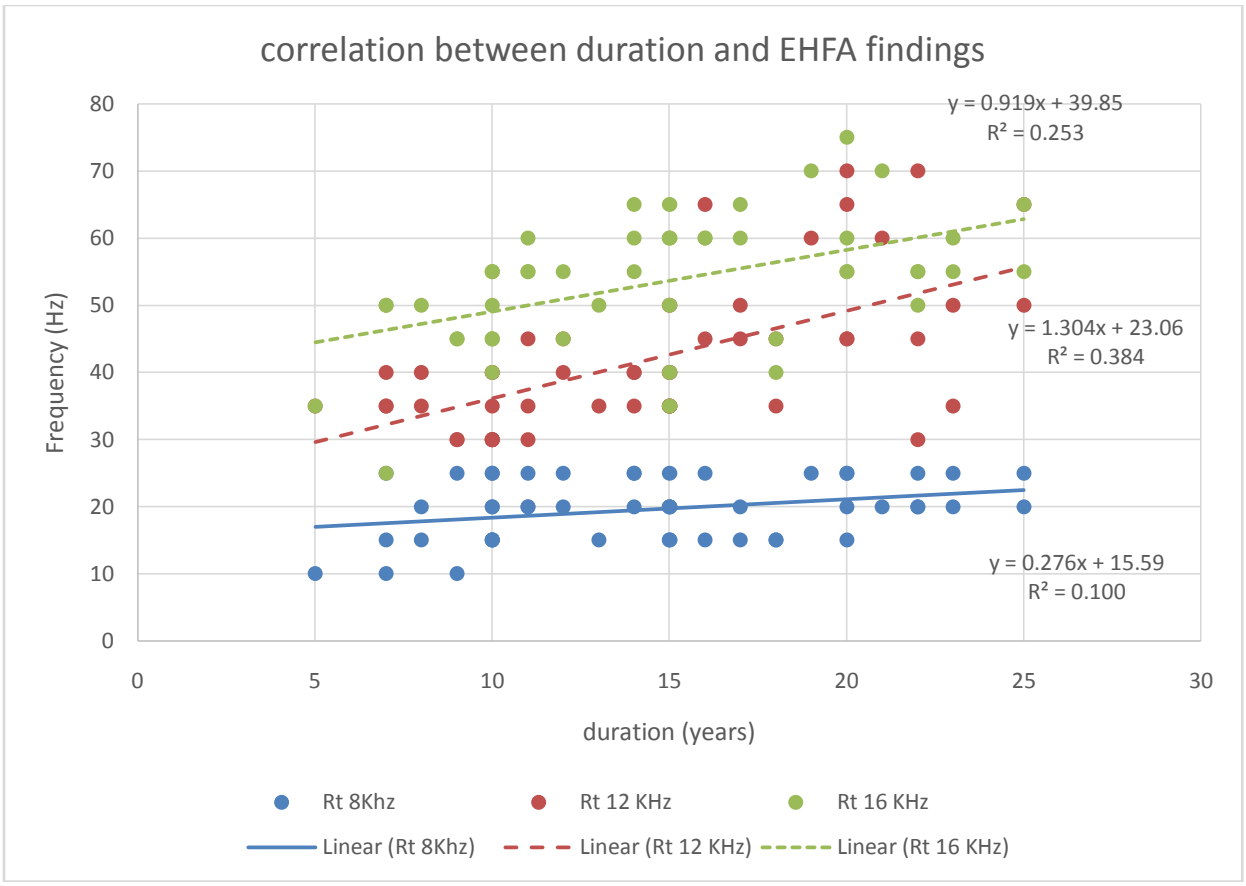

Figure 7:- Scatter plot demonstrating the correlation between duration of noise exposure and EHFA for the right ear.

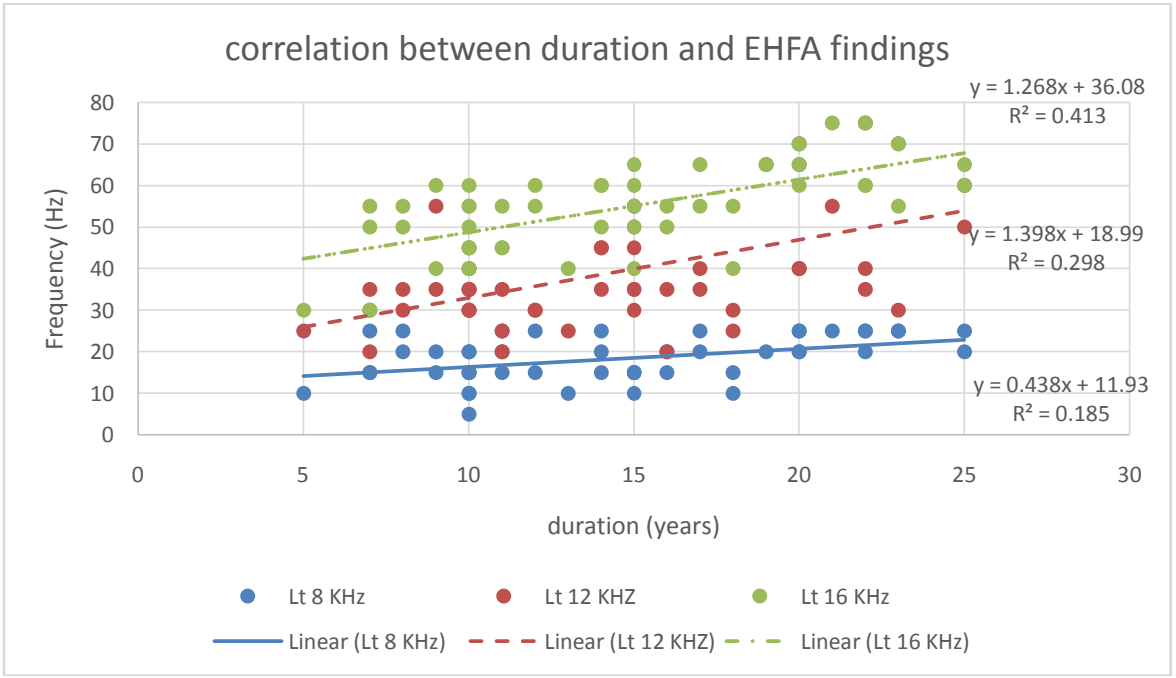

Figure 8:- Scatter plot demonstrating the correlation between duration of noise exposure and EHFA findings for the left ear.

Table 6:- Correlation between duration of noise exposure and wave I amplitude.

Wave I Amplitude	Spearman's rho	p-value
Right	-0.718	<0.001
Left	-0.685	<0.001

Spearman's correlation showed statistically significant (p-value <0.05) strong negative relation between duration and wave I amplitude for right and left ear.

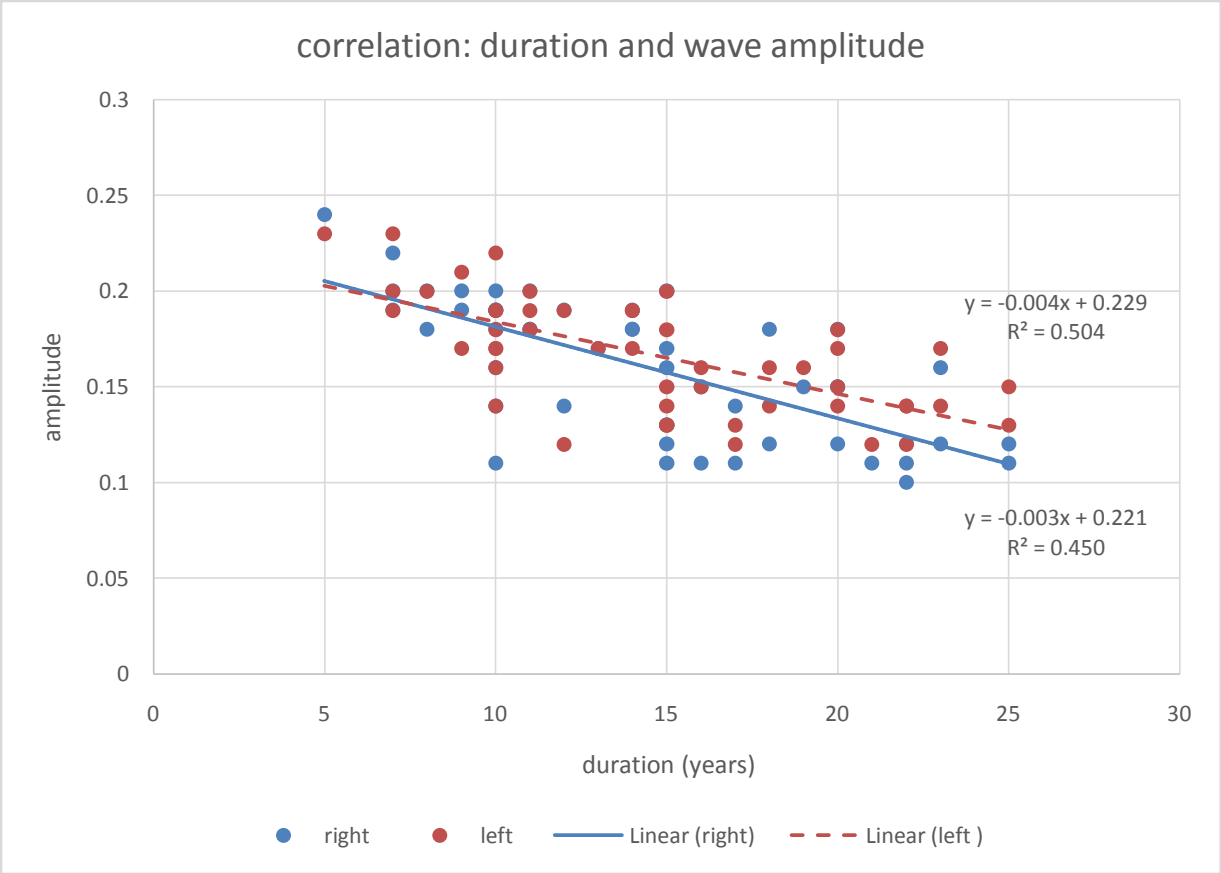

Figure 9:- Scatter plot demonstrating the correlation between noise exposure duration and wave I amplitude for right and left ears.

Amplitude of waveI and EHFA thresholds

Table 7:- Correlation between wave I amplitude and EHFA findings at 8 kHz, 12 kHz and 16 kHz for the right ear.

EHFA frequency	Spearman's rho	p-value
8Khz	-0.251	0.078
12 KHz	-0.498	<.001
16 KHz	-0.422	0.002

Spearman's correlation showed statistically significant negative correlation between wave I amplitude and findings (p-value <0.05), except at frequency 8 kHz.

There moderate negative relation at frequency of 12 and 16 kHz (ρ = -0.498 and -0.422 respectively).

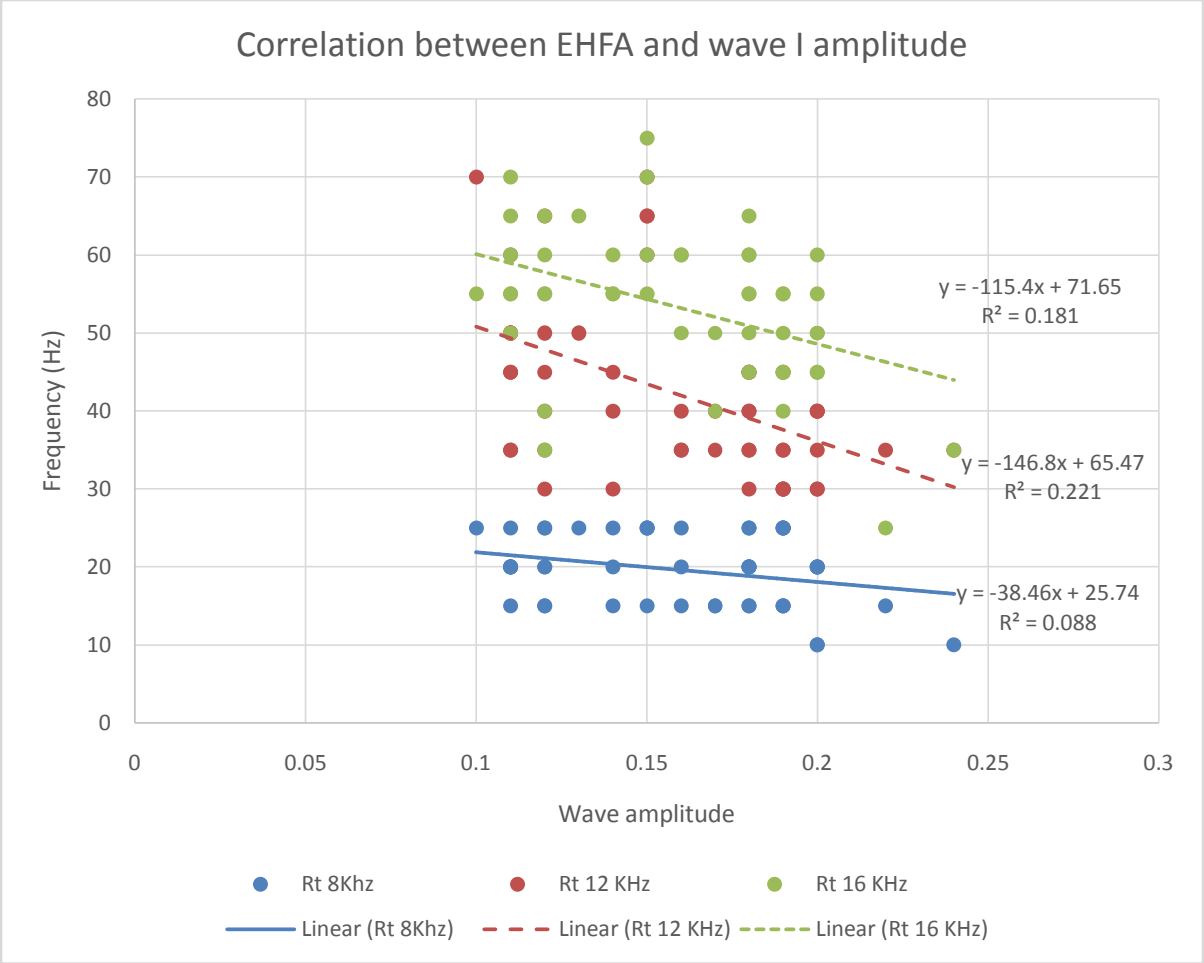

Figure 10:- scatter plot demonstrating the correlation between wave amplitude and EHFA findings for the right ear

Table 8:- Correlation between wave I amplitude and EHFA findings at 8 kHz, 12 kHz and 16 kHz for the left ear.

EHFA frequency	Spearman's rho	p-value
8 KHz	-0.118	0.414
12 KHZ	-0.378	0.007
16 KHz	-0.611	<.001

Spearman's correlation showed statistically significant negative correlation between wave I amplitude and EHFA findings (p-value <0.05), except at frequency 8 kHz.

There weak (ρ= -0.378) and strong (ρ= -0.611) negative relation at frequency of 12 and 16 kHz respectively.

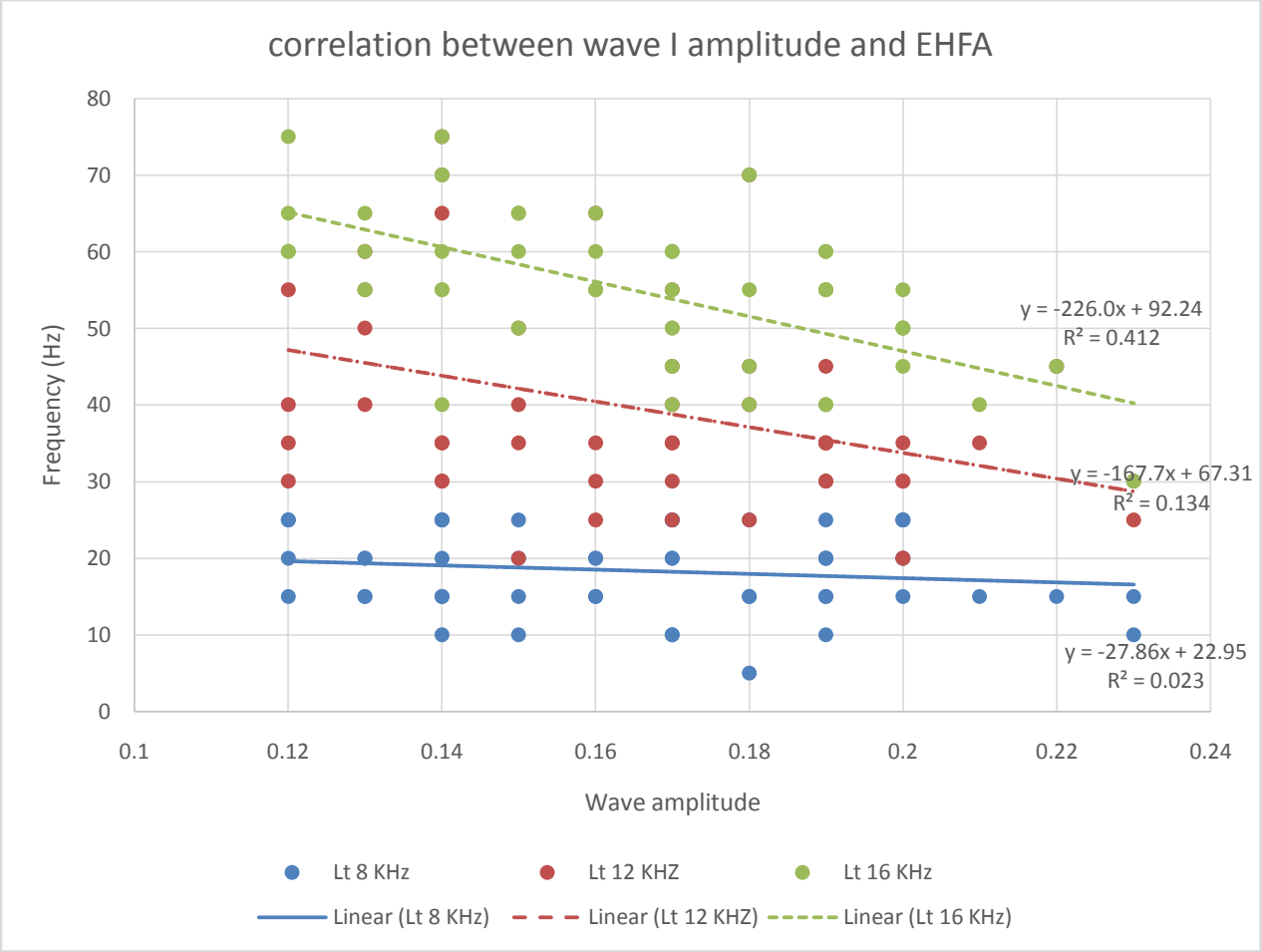

Figure 11:- scatter plot demonstrating the correlation between waveI amplitude and EHFTA findings for the left ear.

Discussion:-

As per the World Health Organization reports, noise exposure is counted as the primary global cause of preventable hearing loss (WHO, 1997). Different forms of noise exposure exist including environmental, occupational at work and recreational noise exposure. The sensory hair cells in the cochlea and the connections between hair cells and nerve cells are only a couple of the many areas of the auditory system that can be damaged by noise exposure (synaptopathy). Injury of the auditory nerve cells can cause tinnitus and hyperacusis to the nosie exposed individuals (Eggermont, 2017).

Pure tone audiometry is a common method for assessing hearing. It calculates the threshold for quiet sounds by measuring the threshold for single-frequency tones up to 8 kHz (British Society of Audiology, 2011). This method examines the capacity to perceive quiet noises. Up until recently, it was thought that the main cause of hearing loss was the damage of the sensory hair cells in the cochlea (Borg et al. 1995). The reported research, however, suggests that fundamental damage to different areas of the brain might come before hair cell loss and might not be detectable by pure tone audiometry. Consequently, concealed hearing loss is a common term for cochlear synaptopathy with perceptual consequences (Eggermont, 2017).

Significant noise exposure may result in temporary threshold changes by irreversibly damaging the connections between inner hair cells and auditory nerve fibres (Kujawa& Liberman,2009). Acoustic trauma has been shown to be especially harmful to auditory nerve fibres with low spontaneous rates and high thresholds; significant synapse loss was seen above 4 kHz (Song et al., 2016). Sound coding requires auditory nerve fibres with low spontaneous rates and high thresholds, especially when there is background noise (Shi et al., 2016).

Reduced peak I amplitude in the compound action potential (CAP) and coding errors in auditory brainstem responses are two reported findings of noise-induced cochlear synaptopathy (NICS) (Song et al., 2016).

In our study, we compared controls and patients as regards the EHFA results. There were statistically-significant differences between patients and controls in RT and LT ears at all tested frequencies. Also, we found positive relation between age and duration of noise exposure and EHFA thresholds in both ears.

Our findings were supported by Fillipo et al., who demonstrated early indicators of hearing loss in workers through high-frequency audiological assessment. In China, Wang et al. looked into the application of EHFA in the early identification of NIHL. In this study, 500 to 8000 Hz pure tone audiometry and EHFA were used to assess one thousand workers exposed to noise. Subjects exposed to noise exhibited minor changes in the typical frequency range compared to the control group, however the EHF hearing thresholds were elevated. Hence, they concluded that change in EHF thresholds happens sooner than the typical frequency range in ears that have been exposed to noise. No responses to highest output and elevated thresholds in EHF can be utilized for early detection of NIHL while assessing noise exposed individuals (Wang et al., 2000).

We compared the wave I amplitude between patients and controls. Both ears showed statistically significant differences regarding the values of wave I amplitudes between the two groups, with the patients exhibiting lower amplitudes than the controls. There was negative correlation between age and duration of noise exposure and wave I amplitude. Our work also showed statistically significant negative correlation between wave I amplitude and EHFA findings. These findings are in accordance with several studies (Barbee et al., 2018; Bharadwaj et al., 2019).

In numerous studies, changes wave I amplitude were assessed and compared with histological results, and it was found that wave I amplitude is a trustworthy indication of synaptic damage following noise exposure (Kujawa& Liberman, 2009; Wang et al., 2000; Lobarinas et al., 2017; Bramhall et al., 2017).

According to Lee et al., noise exposure may result in temporary threshold changes and irreversible insult to ribbon synapses. The results of the ABR recovery threshold using paired click stimuli are related to both the histological analysis and peak I amplitude.

On the other hand, several studies, especially those that focused on testing younger individuals (those under the age of 35), failed to prove neither the anticipated diminished wave I amplitude with noise exposure nor the elevated high frequency thresholds (Fulbright et al., 2017; Grinn et al., 2017; Prendergast et al., 2017).

Tinnitus and hyperacusis, two of the most annoying sensory distortions typically associated with SNHL, could potentially be caused by cochlear synaptopathy. This might be the outcome of compensatory plasticity, whereby neural inputs from the periphery are reduced but synaptic gain in core auditory circuits is increased. Tinnitus could be generated by decreased afferent output from a damaged cochlea which consequently results in low input to auditory centers thus increase in the central gain (Shaheen et al., 2015).

Conclusion:-

In clinical and occupational settings, pure tone audiometry analyses the impact of noise, ototoxic drugs, and other factors on hearing. establishing and confirming standards for threshold measurement techniques. Individual sensitivity is compared to population sensitivity norms based on these measurements, and occupational exposure restrictions have been influenced by threshold-based noise risk calculations. As per our results, combining extended high-frequency audiometry and ABR amplitude measurements, should be helpful as a precursor to impending audibility declines as well as an early warning of hair cell damage and loss.If threshold elevation and OHC loss in humans are preceded by synaptic loss, clinical decision-making and occupational health monitoring procedures would need to be updated in order to preserve hearing function and identify early deterioration.

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