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Auditory Brain Stem Response in Noise Induced Permanent Hearing Loss

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Abstract

Noise exposure usually leads to hearing loss, most severe in the range of 4 to 8 KHz, that seems highly probable due to cochlear lesion which could be explored by the measurement of auditory brain-stem response (ABR). There is general agreement that the sensory-neural hearing loss is due to the degeneration of the outer and inner hair cells. This study included twenty male individuals, chosen from textile factories, age ranged between 25 and 45 years duration of exposure to noise is from five years up to thirty years. In addition ten normal subjects were included in the study as a control group. Both the study and control groups were submitted to full history, full ear, nose and throat examination and audiological examination (pure tone and speech audiometry- emmittancemetry and evoked response audiometry).

The objectives of the study are :

1. To prove the existence of the cochlear affection in noise induced hearing loss by means of ABR.
2. to study the effect of noise exposure on speech discrimination ability.
3. To study the latency intensity function curve in the noise induced sensory-neural hearing loss as compared to a normal subject.
4. To study the effect of the period of noise exposure on the hearing threshold level.

Introduction

SUBJECTIVELY, noise is an unpleasant sound if compared to speech and music. But objectively, noise is a complex sound having little or no periodicity, the waveform not being repeated at any calculably regular interval or time, such sound when it is of moderate or high intensity and long standing duration, will produce that unpleasant sensation to be called noise. Noise is formed of mixture of waves caused by a very large number of pure tones of different frequencies, not harmonically related [1].

Chronic noise-induced auditory damage is characterized by sensory-neural hearing loss affecting the high frequencies. There is general agreement that it is probably due to a degeneration of the outer and inner hair cells [2, 3, 4].

The retro-cochlear involvement observed by other authors is still uncertain [5, 6, 7].

The effect of noise on hearing is a major public health and environmental problem in our society. Noise is reported to be the source of many undesirable effects on mental and physical health [8]. The only well established effect of noise on health is that of noise-induced hearing loss [9].

Noise is classified as steady or non steady [10]. The steady sounds are those which are continuous and remain relatively constant in intensity for a long period of time, and the non steady sounds are further differentiated into fluctuating, intermittent, and impulsive sounds [11].

The physical parameters of noise depend on the noise spectrum, exposure intensity, exposure duration and time varying noise levels. [12] Noise damage is in the 4 KHz region (actually 3 to 6 KHz). It is felt to center around processes in the inner ear rather than the more peripheral portions of the ear, e.g. the region of the organ of corti about 8 to 10 mm from the basal end (which corresponds to the 4 KHz region of the audiogram). [13] The pillar cells in the basal portion of the organ of corti are rather sensitive to noise exposure at least as sensitive as the hair cells. [14] Intense low frequency stimuli have been observed to cause damage in the base of the cochlea in some cases commensurate with that occurring in the region related to the frequency of the exposure frequency [15,16]. The intense sound exposure can alter the oxygen tension in the cochlea, so implying some sound-induced changes in the cochlear blood supply [17]. The oxygen is very important to metabolic processes, such as oxidative phosphorylation, the process by which cells derive their energy. If the oxygen supply is reduced due to abnormal constriction and/or atrophy of blood vessels, normal metabolic processes would be expected to break down [18]. But still this vascular theory is debatable, as ischemia first damages nerve fibres and inner hair cells, in contrast to noise-which injures the outer hair cells initially. The otherwise normal process of degeneration in the cochlear vascular system which occurs throughout life may be accelerated

by noise exposure and other adverse condition [3].

In fact, the primary effect of noise is on the hair cells with the observed changes in the blood vessels of the cochlea being secondary [19]. With very high levels of exposure, typically more than 130 dB sound pressure level (SPL) for brief duration, the first effects on the structures of the hearing organ are likely to be mechanical in nature [20].

The early effect of noise seems to take place at the cochlear level, while the degeneration of central auditory connections can become evident later, so later there will be abundant evidence of neuronal cell loss and atrophy of the spiral ganglion, in the axonal endings of the ascending cochlear nerve fibres, in the ventral cochlear nucleus, in the adjacent regions of the antroventral and dorsal cochlear ones, in the superior olivary complex, lateral lemniscal nuclei and ventral nucleus of the inferior colliculus [21].

It is also suggested that the location of central degeneration following partial destruction of the organ of corti corresponds to the tonotopic organization of the cochlear nerve. In summary, the central lesions seen to follow and reflect peripheral changes [22].

Histopathological changes in human cochlea exposed to noise were studied from a collection of temporal bones obtained at autopsy. These changes were correlated with available audiograms and occupation-

al history recorded of the same subject. The evaluation of cochlear lesions was based on the study of cytochleograms (it is a curve indicating the percentages of hair cells present), in terms of an "ideal pattern" of three outer rows and a single inner row, this is done millimeter by millimeter throughout the entire length of the basilar membrane [3].

It is known that the different kinds of noise can lead to different damage patterns, which may be caused by different kinds of mechanical events in the cochlea even at correspondingly equivalent sound levels [23, 24, 25]. The temporal characteristics, the critical intensity and especially the individual susceptibility to damage can influence the severity of the noise-induced trauma [26].

Bohne [27] traced the progression of degeneration of the organ of corti, including some of the fine structural changes following traumatic noise exposure:

1. *Within the first hour after exposure :*

The only signs of damage are found as a slight swelling and displacement of outer hair cells about 4 mm from the basal end of the hearing organ. There are detectable changes in the cellular contents particularly increased infoldings of endoplasmic reticulum, few outer hair cells are actually missing, and all inner hair cells are present.

2. *One and half hours after exposure :*

The outer hair cells show increased swelling, further alterations in the endoplasmic reticulum and fusing of the stereocilia.

3. *Two hours post exposure :*

Outer hair cells over about a 1 mm segment of the basilar membrane are lost apparently because of rupturing from continued swelling of the cell bodies, small holes may be seen in the reticular lamina, the surface of the organ of corti, in places once occupied by hair cells.

4. *Beyond two hours :*

Both supporting and inner hair cells begin to show signs of damage. And by 14 days essentially, 1 mm of the organ of corti is missing, the final stage of permanent damage appears to take about 2 months to develop [28].

Material and Methods

This study was conducted in Hearing and Speech institute at Embaba. The study included 20 male individuals (20 ears) chosen from textile factories, age ranged between 25 to 45 years, duration of exposure to noise was for more than 5 years, 8 hours / day, 6 days per week. They were not exposed to head trauma, or ototoxic drugs. In addition 10 normal subjects (10 ears) were included in the study as a control group, they had no history of ear diseases for at least 2 months before examination, and no history of upper respiratory tract infection at time of examination.

Both groups (study and control) were subjected to full E. N. T. examination,

audiometric, tympanometric and examination by the evoked response audiometry (ERA).

Pure tone audiometry was performed in sound treated room, the audiometer was Madsen Model OB 822. The air conduction threshold for frequencies 500 to 4000 Hz reported.

Tympanometry was performed using immittancemeter (Amplaid Model 720). Immittancemetry, tympanometry and acoustic reflex thresholds were estimated.

Evoked response audiometry (ERA) was performed using Madsen Model 2250, and it was done with these parameters:

1. *Stimulus parameters :*

- a - Type of the stimulus: Click rarification of 0.25 msec duration.
- b - Intensity : 90, 70, 50 and 30 dB hearing level (HL) which is delivered through headphone TDH 39.
- c - Repetition rate of 20 click / second.
- d - Number of sweeps is 1000 sweeps.

2. *Recording parameters :*

- a - Pass and filter 150 - 1500 Hz.
- b - Sweep time : 10 msec.

As regards electrode location in the ABR, three electrodes were used, two of which were connected to the preamplifier inputs with the other electrode serving as

ground [29]. The active (positive) electrode was placed on the vertex, where a small area of skin was shaved prior to vertex placement, and the reference (negative) electrode was adherent to an assumed inactive (neural) site which was the ipsilateral earlobe with the contralateral counterpart earlobe reserved for the ground electrode.

The electrode site was cleaned with acetone solution to remove dirt and natural oils, and the electrode was affixed to the patient with conductive electrode gel as reported by Newton and Barratt [30].

Results

This study included 20 subjects (20 ears), all were males. Age ranged between 25 to 50 years, with a mean of 34 years.

The mean duration of noise exposure was 20 ± 3.2 years, with a range between 7 to 30 years. Results of the present study were interpreted in the following manner :

1. Estimation of the mean and standard deviation (S.D.) of different parameters of pure tone audiometry (PTA), tympanometry and auditory brainstem evoked response (ABR), as obtained from tables (1, 2, 3 and 4).
2. Comparing the mean of the control group and study group as regard statistical significance by using "t" test (tables 1,2, 3 and 4).
3. Correlation between period of noise exposure and hearing loss (figure 1).

Table (1): Mean air Conduction Threshold and Standard Deviation (S.D) of both Control and Patient Groups at Different Frequencies and "t" Test and Statistical Significance of Comparing both groups.

Freq. in Hz.	250		500		1000		2000		4000		8000	
Group	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.
Mean PTA	15.5	18.5	12.5	19	12.5	21	13	27.7	15	44.2	15.5	36
SD	2.8	4	2.6	4.1	2.6	2.5	2.5	6.7	2.3	5.4	3.6	6.4
t	4.3		9.4		11.7		19.1		41.0		24.7	
p significance	< 0.01		< 0.01		< 0.01		< 0.01		< 0.01		< 0.01	
	Sig.		Sig.		Sig.		Sig.		Sig.		Sig.	

Cont. = Control

Pat. = Patient

Table (2): Mean and Standard Deviation of Speech Reception Threshold (STR) and Word Discrimination Score (WDS) of both Control and Patient Groups and "t" Test and Statistical Significance of Comparing both Groups.

	Speech Reception Threshold (SRT)		Word Discrimination Score (W.D.S.)	
	Control	Patient	Control	Patient
Mean	12	20	100%	92.6%
SD	2.5	4.2	0	3.5
<i>t</i>		11.6		19.6
<i>p</i>		< 0.01		< 0.01
significance		Sig.		Sig.

Table (3): Mean Air Conduction Threshold and Standard deviation (AR) of both Control and Patient Groups at Different Frequencies and "t" Test and Statistical Significance of Comparing both Groups.

Freq. in Hz.	Compliance		500		1000		2000		4000	
	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.
PTA	0.7	0.7	88.5	4.2	94	101	91.5	97.7	93.5	102.2
SD	0.2	0.2	4.1	4.2	3.1	5.9	4.1	5.4	4.7	5.4
<i>t</i>	0.22		15.19		8.9		7.5			10.1
<i>p</i>	< 0.05		< 0.01		< 0.01		< 0.01			< 0.01
significance	Non. Sig.		Sig.		Sig.		Sig.			Sig.

Cont. = Control

Pat. = Patient

Table (4): The Mean and Standard Deviation (SD) of Absolute Wave Latency of Waves I, III and V and Interwave Latency I - V and "t" test results and Statistical Significance of Comparing both Groups.

	Wave I		Wave III				Wave V				I	III	III	V	I	V
	at 90 dB		at 90 dB		at 70 dB		at 90 dB		at 70 dB		at 90 dB	at 70 dB		at 70 dB		
	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.	Cont.	Pat.
Mean	1.7	1.8	3.7	3.9	3.8	4.2	5.7	5.8	5.7	6.2	2.05	2.02	1.9	1.9	3.9	3.9
SD	0.07	0.1	0.1	0.2	0.1	0.2	0.2	0.1	0.2	0.2	0.1	0.2	0.1	0.1	0.2	0.1
t	1.3		0.8		0.8		0.7		2.4		0.2		0.06		0.1	
p	> 0.05		> 0.05		< 0.05		> 0.05		< 0.05		> 0.05		> 0.05		> 0.05	
significance	Non. Sig.		Non. Sig.		Sig.		Non. Sig.		Sig.		Non. Sig.		Non. Sig.		Non. Sig.	

Cont. = Control

Pat. = Patient

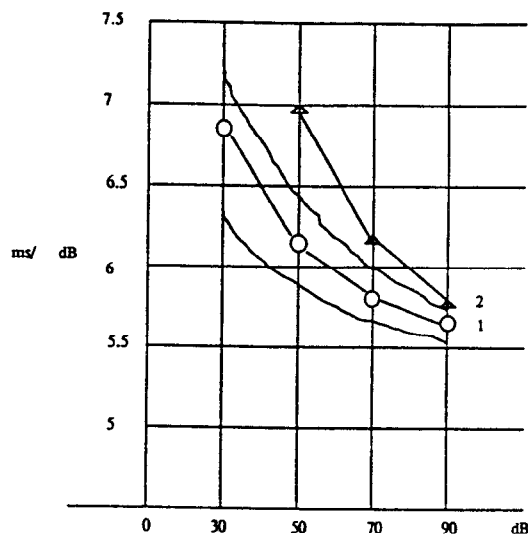


Fig. 1

- The latency intensity function curve.

1. The reference curve.

Wave V latency at 90 dB = 5.7 & SD = 0.22.

Wave V latency at 70 dB = 5.79 & SD = 0.24.

Wave V latency at 50 dB = 6.18 & SD = 0.298.

Wave V latency at 30 dB = 6.82 & SD = 0.265.

2. The cochlear curve.

Wave V latency at 90 dB = 5.84

Wave V latency at 70 dB = 6.25

Wave V latency at 50 dB = 6.90

Table (1) shows the mean and standard deviation (S.D.) of pure tone threshold for both the control and patients group. The average air conduction at different frequencies (250, 500, 1000, 2000, 4000, and 8000 Hz) was 15.5, 18.5 - 12.5, 19 - 12.5, 21 - 31, 27.7 - 15, 44.2 and 15.5, 36 db at

the previously mentioned frequencies for both groups respectively. Comparison between the two groups was done using "t" test and it shows a statistically significant result at all frequencies.

Table (2) shows the mean and standard deviation (S.D.) of speech reception threshold (SRT) for both the control and patient groups respectively (12, 20 - 100%, 92.6%). Statistical analysis using "t" test shows that there is statistically significant results on comparing both means of SRT and WDS in both the control and patient groups ($t = 11.6, 19.6$ respectively and $p < 0.01$).

Table (3) shows the mean and standard deviation (S.D.) of acoustic reflex threshold, compliance of both the control and patient groups.

The compliance was 0.7 ± 0.2 in both groups, and it shows no statistical significant results. The acoustic reflex at different frequencies (500, 1000, 2000 and 4000 Hz) was 88.5, 100.5 - 94, 101 - 91.5, 97.7 and 93.5, 102.2 db. at the previously mentioned frequencies for both groups respectively. Comparison between the two groups was done using "t" test and it shows statistically significant results at all frequencies.

Table (4) shows the mean and standard deviation (S.D.) of absolute latencies in the auditory brain-stem evoked response (ABR) in waves (I, III, V at 90 db.) for both the control and patient groups.

It was 1.7, 1.8 - 3.7, 3.9 and 5.7, 5.8 millisecond (ms) for both groups respectively. There was no statistically significant results by using "t" test on comparing both groups.

As regards the absolute latencies of wave III and wave V, test at 70 db for both

control and patient groups, (table 4) shows that it was 3.8, 4.2 millisecond (ms) for wave III at 70 db for the control and patient groups and this parameter was statistically significant.

As regards absolute latencies of wave V at 70 db. it was 5.7, 6.2 millisecond (ms)

Table (5): The Pure Tones for every Noise Exposed with the Period of Exposure to Noise for Years and Showing "r"* for each Frequency

Code No.	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz	8000 Hz	Period of exposure to noise in years
1	15	15	20	25	45	35	17
2	15	20	20	30	40	35	15
3	15	15	20	25	40	30	10
4	20	20	20	25	50	40	18
5	25	20	15	25	45	25	11
6	20	15	15	20	40	30	13
7	15	15	15	20	45	350	15
8	15	15	15	25	45	40	13
9	25	25	30	50	60	50	20
10	15	20	20	25	45	35	15
11	20	15	15	25	40	35	9
12	15	15	20	25	45	35	12
13	20	20	25	30	40	35	10
14	15	20	25	30	45	40	12
15	20	20	25	30	50	45	16
16	15	20	20	25	40	30	7
17	20	20	25	30	45	40	11
18	25	25	30	35	40	35	9
19	25	30	30	35	50	45	14
20	15	15	15	20	35	25	6
*r	0.14	0.22	0.24	0.40	0.80	0.67	

*r = Correlation Coefficient

for both control and patient groups and it was also statistically significant.

Table (4) shows the mean and standard deviation (S.D.) of the interwave interval (I, III, III - V and I-V) at 90 db for both control and patient groups, and it was 2.0, 2.0 - 1.9, 1.9 and 3.9, 3.9 millisecond (ms) for both group respectively, and it was statistically insignificant.

Table (5) shows the hearing threshold at different frequencies (250 to 8000 Hz) with the period of exposure worker. The correlation coefficient (r) is at 250 Hz = 0.14, at 500 Hz = 0.22, at 1000 Hz = 0.24, at 2000 Hz = 0.40, at 4000 Hz = 0.80 and at 8000 Hz = 0.67.

Figure (1) shows the latency intensity curve, for both the control and patient groups. At high intensities the two curves, control (reference) and patient curves are similar approximately, and as the intensity decreases, there was diverging of the study subjects curve from the reference curve.

Discussion

This work aimed to study the effect of noise exposure on speech discrimination ability, latency intensity function curve in the noise-induced sensory-neural hearing loss and to study the effect of the period of noise exposure on the hearing threshold level. The study included 20 subjects (20 ears) exposed to noise, all were males. They had a complaint of hearing impairment and tinnitus. Their age ranged between 25 to 45 years, older subjects were

excluded to avoid the effect of presbycusis, in addition, subjects who received ototoxic drugs, with history of diabetes, history of renal and hepatic diseases were also excluded.

The study included 10 normal subjects (10 ears) as a control group. Pure tone audiometry was performed, the average bone conduction of noise exposed subjects and control subjects at frequencies 500 to 4000 Hz were reported.

From table (1) we found the following :

1. All frequencies for 250 to 8000 Hz (in the audiogram) gave statistically significant differences in hearing threshold levels where the hearing sensitivity was less in the noise exposed workers than the control group. These differences showed the following characteristics:

- a - They were more prominent for high frequencies (2000, 4000 and 8000 Hz) than low and moderate frequencies (250, 500 % 1000 Hz), so starting to increase from 2000 Hz and tries to return again at 8000 Hz, but still more than normal.

- b - Maximal affection of hearing threshold was at 4000 Hz, where the significance ($T = 41.08$) being the most noise sensitive frequency [31].

2. Also from table (2), we found that in the speech reception threshold (SRT) there was significance in the difference between the control and the study groups, it was better in control group, as $T = 11.64$.

3. In the same table (2), as regards the word discrimination score (WDS), there was statistical significant difference between the control and the study groups ($T = 19.6$).

In our work, there were 4 workers of the noise exposed individuals complained from difficulties in understanding speech, and this represented 20% of the total number of the study group. This unexpected poorer discrimination was suggestive of their sensory-neural hearing loss, which was of neural affection rather than pure cochlear lesion [3,4].

From table (3), we found that:

1. The compliance of the middle ear was not affected in the noise exposed subjects, when compared with those of the control group (no significant difference) $T = 0.22$, because the lesion was a cochlear lesion and the middle ear was not affected from the noise exposure.

2. Although the stapedial muscle reflex thresholds were within normal for both control and study groups, still there were significant differences at all frequencies tested (at 500 Hz $t = 15.19$, at 1000 Hz $t = 8.92$, at 2000 Hz $t = 7.56$ and at 4000 Hz $t = 10.13$) these normal values of acoustic reflex thresholds were considered as evidence of cochlear site of the lesion due to the recruitment phenomenon.

Also, it was found that there was a subjective feeling of tinnitus which was the major complaint of the noise exposed

individuals. These were 8 workers from the study group (20 subjects) so this represented about 40% of the total number that complained from tinnitus. It is noticed that the tinnitus was the earliest symptom in the noise-induced hearing loss in individuals, which was followed by difficulty in understanding speech, then hearing deterioration followed at last.

From table (4) ABR absolute latencies showed the following:

1. Wave I at 90 dB: It was slightly prolonged in latencies of the study group than the control group, but this difference failed to reach a significant level ($T = 1.34$) and $p < 0.05$. Some Workers [32] proved that wave I is not shifted or prolonged except at low intensities.

2. Wave III at dB: Although the absolute latency of this wave had longer latency in the study group, yet the difference was statistically insignificant ($T = 0.81$) and $p < 0.05$.

3. Wave V at 90 dB: The same results of the above waves I & III were found also for wave V at this intensity level, (90 dB), the longer latency of the study group still showed no statistical significance ($T = 0.78$) and $p < 0.05$. Picton et al [33] stated that wave V latency was not affected at high intensities.

4. Wave III at 70 dB: Its latency showed a statistical significance between the control and the study groups, as the latency of wave III of the noise exposed

subjects was longer than that of the normal subjects ($T = 2.23$) and $p < 0.05$. Drift et al [34] stated that at cochlear hearing loss, waves III & V are longer than the normal subjects.

5. Wave V at 70 dB: Its latency showed a statistically significant difference between the control and study groups ($T = 2.41$) and $p < 0.05$.

6. Wave V at 50 dB: Its latency was longer in the study group, except in one subject of the study group, where wave V was absent, and its audiogram was showing hearing loss up to 60 dB level at 4000 Hz, ($T = 3.173$) and $p < 0.01$. At cochlear hearing loss wave V was not be affected except at low intensities [35].

7. Wave V at 30 dB : Wave V was not present in all the study group (absent from all the noise exposed workers), at the same time it was present at 30 dB in all the control group subjects. This finding is in agreement with the hearing sensitivity of both group individuals [36] as ABR was used for hearing assessment.

From the above we found that:

- At high intensity levels e.g. 90 dB, the affection of the wave V was minimal or absent (insignificant).

- While at intensity 70 dB, there was greater affection of the Wave V latency [35], in the moderate intensities as 50 dB, the prolongation in the latency of

wave V was highly significant [35]. And this was due to the recruitment of the cochlear phenomenon.

From table (4): ABR interwave intervals showed the following :

1. For the interwave interval (IWI) I-III there was no significant difference between the IWI I - III of the control and the study groups ($T = 0.20$) and $p > 0.05$.

2. As regards the interwave interval (IWI) III - V there was no statistical significance between that of the control subjects and the study group, the significance test $T = 0.06$ and $p > 0.05$.

3. Accordingly interwave interval (IWI) I-V there was no statistical significant difference between the I-V IWI in the control and that of the study groups, as the $T = 0.181$ $p > 0.05$.

Figure (1) showed the latency intensity function curve of wave V for both the control and study subjects. The curve obtained from the normal subjects was drawn as a reference curve for the study group. The curve representing the latency values of wave V of the study group was considered as typical response for the cochlear hearing loss, where the latency at high intensity level (90 dB) was at normal value, while as the intensity decreased the latency prolonged till the response disappeared at the hearing thresholds. This was due to the recruitment phenomenon which characterised the cochlear hearing loss, [34].

The correlation coefficient (r):

As regard the values of "t" were:

0.20 to 0.40 = low correlation.

0.40 to 0.60 = moderate correlation.

0.60 to 0.80 = high correlation.

0.80 to 1.00 = extra-high correlation.

So from table (5), we found that, as the period of exposure was increased as the hearing sensitivity was decreased at high frequencies (2000, 4000 and 8000 Hz) but most prominent at 4000 Hz as "t" at 250 Hz = 0.14 (no correlation), "t" at 500 Hz = 0.22 (low correlation), "t" 1000 = 0.24 (low correlation), "t" at 4000 Hz = 0.80 (extra-high correlation) and "t" at 8000 Hz = 0.67 (high correlation).

From our work we noticed that there was no previous audiograms or any records for the workers to compare them with the audiograms or the ABR tracings done in this work. The audiograms must be done as one of the pre-employment examinations, to be kept as a document to show the effect of noise on hearing and in evaluation of the degree of the workers disability due to noise.

Although the noise level in engines in most departments were more than 95 dB, many countries as Canada and France consider 85 dB as a critical level above which hearing protection must be seriously considered [33].

Conclusion:

The aim of this work was to study the effect of continuous industrial noise on hearing of the workers by using the auditory brain-stem response (ABR). The total number of twenty noise exposed workers were chosen from textile factories, and ten not exposed subjects as control group.

All of the two groups were males, their ages ranged from 25 up to 50 years old. All of them were subjected to a sheet for occupational deafness. Clinical otological examination was carried out at Hearing and Speech institute at Embaba. All the subjects had clinically normal ears, with intact mobile tympanic membranes, with no history of ear diseases, no head trauma or ototoxic drug administration. All of the control and noise exposed workers were subjected to audiometric tests, speech tests, tympanometric tests and brain-stem response audiometry tests.

The study revealed that:

1. There was no pre-employment audiograms or follow up audiograms.
2. The 4000 Hz frequency was the first and the most frequency to be affected followed by 3000 and 2000 Hz respectively.
3. The average hearing losses at 2,3 and 4 KHz must be taken as the level of hearing loss instead of the all frequencies from 250 to 8000 Hz, as the noise affect the high frequencies, most prominent at 4 KHz.

4. The wave V disappeared at low intensities as regard the degree of hearing loss at frequencies 2 and 4 KHz, which was related to the period of exposure to noise.

5. The latency intensity curve was used to confirm the cochlear hearing loss, and to differentiate it from the conductive and retro-cochlear hearing loss.

6. The Interwave intervals in the ABR were not affected in the cochlear hearing loss, but only there was a delay in the absolute latency of wave V at low intensities.

7. As the period of exposure to noise was increased, the hearing sensitivity was diminished.

8. Lastly, the protection from noise was very important to keep the hearing sensitivity not affected.

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