COCHLEAR AFFECTION IN VESTIBULAR NEURITIS

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SUMMARY:

49 cases diagnosed with unilateral vestibular neuritis were examined using pure tone (PT) audiometry at both conventional (250 Hz-8 KHz) and high frequencies (10- 20 KHz). The affected ears with vestibular neuritis (VN) were compared with the unaffected ear and with a group of normal volunteers. The difference in PT threshold between both ears in patients with VN and the normal group was calculated. Results: In patients with VN, the mean PT threshold differences between the affected and unaffected ears showed no significant difference when compared to the mean PT threshold differences in conventional audiometric frequencies. On the other hand, the mean PT threshold differences between the affected and unaffected ears in patients with VN showed significant difference when compared to the mean PT threshold differences in high audiometric frequencies. Conclusion: there is no isolated vestibular lesion. The causative factors for vestibular pathology may affect the cochlear function.

INTRODUCTION & RATIONALE:

Acute, spontaneous, unilateral vestibular lesion called acute vestibular neuritis (VN) or neuronitis is a common vestibular disease and is considered the second most common cause of vertigo. Typically, there is sudden onset of vertigo with no associated hearing loss or central nervous system disorders. Some call it labyrinthitis or neurolabyrinthitis but generally these terms are used when there is associated cochlear affection. The multiplicity of terms implies uncertainty of the pathology

so some authors prefer the term acute unilateral peripheral vestibulopathy [7].

Clinically, VN can be presented with three different patterns of vestibular abnormalities. 1) Total VN pattern: both the superior and inferior vestibular nerve functions are affected (3 and 8) 2) Superior vestibular nerve pattern: posterior: SCC function (2), and saccular function (5), appears preserved. This might be the most common pattern, possibly because the bony canal of the superior vestibular nerve is longer than that of the inferior nerve (6). 3) Inferior vestibular nerve pattern: there is absent VOR on impulsive testing only from the posterior SCC and abnormal VEMP of saccular origin (2 and 9).

Wemmo and Pyykko, (1982) reported associated CNS abnormalities in one third of their patients of VN in the form of occulomotor abnormalities. Also Aantaa and Virolainen, (1979) showed that 50% of their patients of VN develop minor hearing loss after 3 years although these minor changes in hearing loss falls within the normal variations Bergeius and Borg, (1983) showed abnormal elevated acoustic reflex threshold in about 50% in the patients of VN.

In 1986, Rahko et al. revealed that in most cases (17 of 21 patients) of their study of VN using high frequency audiometry up to 15,000 Hz, there are no isolated vestibular lesions, but most cases also involve an auditory end organ lesion. The rationale of the present study is that the vestibular system and the cochlea are constructed in the same configuration and have common blood supply. So, it seems unlikely that for example a viral insult caused damage to the vestibular apparatus without affecting even minimally the nearby closely related cochlea. Accordingly, the purpose of the present study is to evaluate cochlear integrity using high frequency audiometry up to 20,000 Hz in patients with unilateral VN.

METHODOLOGY

This study was conducted at Audiology unit, ENT department of Mansoura University.

Study group:

49 cases aged from 20 to 39 years including 22 female and 27

male diagnosed with unilateral VN using otoscopy, basic audiological evaluations including audiometry at conventional PT frequencies (250Hz to 8000 Hz) and immitancemetry and videonystagmography.

Diagnosis of VN was based on the history typical of VN that was single episode of rotational vertigo lasting several hours up to few days managed with vestibular suppressants and referred for audio-vestibular investigations. The clinical finding for diagnosis of VN include normal otoscopic examination, normal pure tone audiometry (250 Hz to 8 KHz) and VNG finding suggestive of unilateral peripheral vestibular lesion including spontaneous nystagmus with fast phase beating towards healthy ear suppressed by visual fixation and unilateral caloric weakness more than 20% on the affected side.

After establishment of diagnosis of VN, the PT hearing threshold for all patients in study groups was obtained at frequencies of 10, 12.5, 14, 16, 18, and 20 KHz using HF audiometer (Interacoustic clinical audiometer AC 40 software version: 1.48) and HAD 200 high frequency headphones. The audiometer and HAD 200 headphone were calibrated using IEC60318 with type 1 adaptor coupler. The maximum output of the audiometer was 100, 90, 80, 60, 30, 15 dBHL at 10, 12.5, 14, 16, 18, 20 KHz respectively. The PT threshold in the affected (Ears with VN) and unaffected ears was compared for conventional and high PT frequencies.

Control group:

The PT threshold of 14 normal volunteers served as control group was obtained at conventional and high frequency audiometric frequencies.

All cases in both groups were selected with no systemic diseases as DM and hypertension without history of ototoxic drugs intake. Patients with hepatitis B or C or received drugs for hepatitis were excluded from the study because of potential auditory hazards of anti-hepatitis virus drugs.

RESULTS

49 patients in study group had a mean age of 30.2 years and 14 normal volunteers had a mean age of 28.6 years. In study group patients, the right ear was affected with VN in

28 patients and the left ear in 21 patients.

As showed in table 1 and figure 1, there were no statistically significant differences in mean PT threshold difference between the control and study groups at conventional PT audiometric frequencies.

Results in table 2 and figure 2 showed highly significant difference in mean PT threshold between the control and study groups in high frequency audiometry. In control group, the mean difference remained similar to conventional audiometric frequencies in contrast to study group where the mean difference differed from conventional audiometric frequencies.

At 18 and 20 KHz, the PT threshold of the affected ears in 9 and 31 patients respectively exceeded the maximum of the audiometer and the difference between both ears can not be calculated.

Tables 1: showed mean PT threshold differences at conventional audiometric frequencies in control (Right and left ear) and study groups (affected and unaffected ears):

PT frequency in Hz	Control group		Study group		P value
	Mean	SD	Mean	SD	
250	5.3	1.6	5	1.4	0.49
500	4.8	1.3	5.1	1.62	0.52
1000	3.9	1.26	3.7	1.3	0.57
2000	4.6	1.42	5.2	1.49	0.18
4000	4.4	1.43	5.4	1.8	0.06
8000	5.8	1.87	6.6	2.1	0.2

Table 2: showed Mean PT threshold difference and SD in high audiometric frequencies in both control (right and left ears) and study groups (affected and unaffected ears):

PT frequency	Control group		Study group		P value
	Mean	SD	Mean	SD	
10 KHz	6.3	1.96	10.9	2.84	**<0.001***
12.5 KHz	4.3	1.78	14.8	4.6	**<0.001***
14 KHz	5.9	1.92	20.9	5.8	**<0.001***
16 KHz	6.6	2.06	13.7	4.2	<0.001*
18 KHz	5.8	1.69	18.3	5.8	<0.001*
20 KHz	6.5	2.2	11.2	3.4	<0.001*

Figure 1: illustrates the mean PT threshold differences at conventional audiometric frequencies in control (Right and left ear) and study groups (affected and unaffected ears):

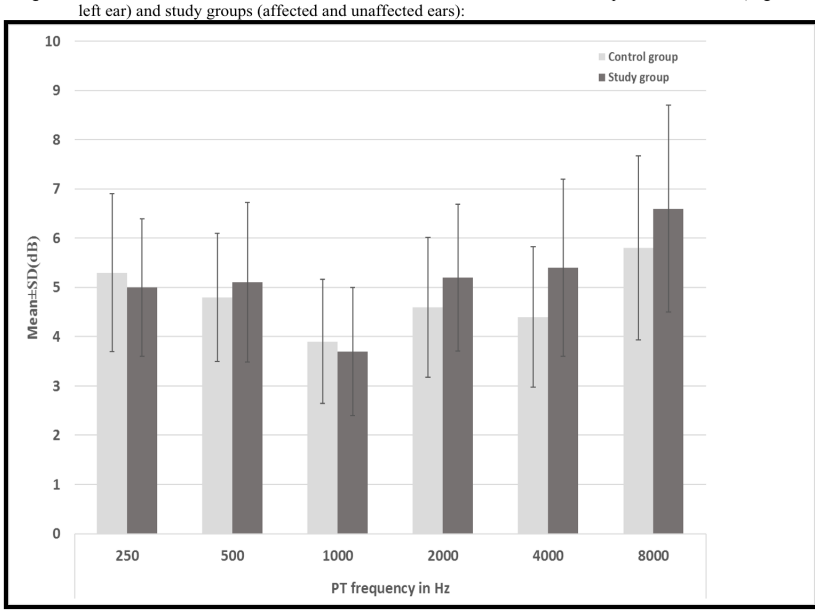
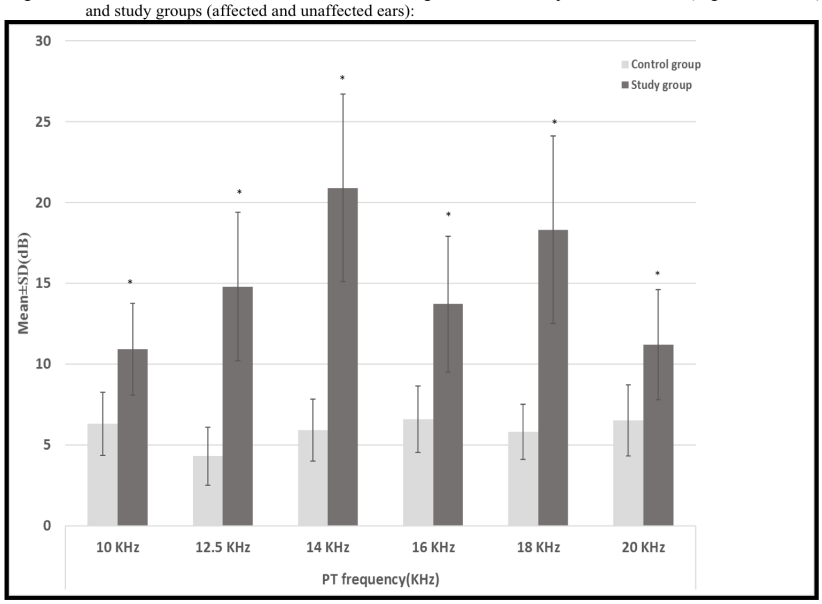


Figure 2: illustrates the mean PT threshold differences at high audiometric frequencies in control (Right and left ear) and study groups (affected and unaffected ears):



DISCUSSION

In the present study, although the patients were selected with unilateral VN and the unaffected ear can be used as a control ear, a control group of normal volunteers was selected in the study to ensure that measurements settings of high frequency audiometry is not biased calibrations, placement of headphones or other environmental variables.

Tables 1& 2 showed that there were limited mean PT threshold differences between both ears in control group either in conventional or high audiometric frequencies not exceeding 10 dBHL. Study group patients also showed a similar mean PT threshold differences for only conventional audiometric frequencies between affected (VN ears) and unaffected ears. On the other hand, 41 of 49 patients in the study group patient showed mean PT threshold differences exceeding 10 dBHL. Tables 2 showed statistically significant difference between the control and study groups in high frequency PT threshold. The mean differences for high frequency PT thresholds in the study group patients ranging from 10.9 to 20.9 dBHL.

As mentioned earlier that VN can be presented in three different forms of clinical presentation and some authors reported CNS abnormalities and others reported subsequent hearing loss, it seems that the cause for VN may be not selective and is not limited only to one site of vestibular system. The audio-vestibular finding in VN may be related to the degree of the damage to the audio-vestibular system. In other words, the pathology of VN may be so mild that only affect a restricted site in the vestibular system or a single branch of the vestibular nerve, more severe and expand to affect both branches of the vestibular nerve or expand to the nearby cochlea and presented clinically as labyrinthitis with sudden hearing loss as discovered by conventional PT audiometry.

The high frequency region in the cochlea or the basal region is the more vulnerable region to early affection and this can be noticed clearly in the process of presbyacusis and toxic effect of ototoxic drugs. In the present study, the same principle may be considered and applied. The differences in PT high frequency threshold in most patients exceeded 10 dBHL and the differences increased as a function of frequency up to 14 KHz. It may be assumed that the causative factor of VN may affect cochlear function and vulnerability of affection increases as the frequency increases.

The results in this study matched with the previous results of Rahko et al. (1986) and suggested that in most cases of vestibular neuritis, there is no isolated vestibular lesion.

Conclusion

There is no single isolated vestibular lesion in VN. The associated impairment of cochlear integrity may be minimal and can not be detected by conventional audiometry or significant and presented clinically as sudden hearing loss.

Criticism:

The diagnosis of VN did not consider different patterns of clinical presentations of VN either superior, inferior or total VN. The present study depended in unilateral caloric weakness exceeding 20%, so all cases of VN in study group may be total or superior vestibular nerve patterns. We recommend another research that considers different types and the associated cochlear impair-

ment in high frequency audiometry with each type of VN.

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