Advanced International Journal for Research (AIJFR)
E-ISSN: 3048-7641 ● Website: www.aijfr.com ● Email: editor@aijfr.com

Distortion Product Otoacoustic Emission evaluation as a diagnostic tool to identify the prevalence of cochlear/noncochlear types in Cerebellopontine Angle tumors

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Abstract:
Cerebellopontine Angle tumors disrupt the auditory system through different mechanisms. Acoustic tumors seems to affect cochlear function more predominantly. Nonacoustic tumors seems to affect neural mechanisms more. Distortion Product Otoacoustic Emission is a wonderful tool to study this aspect. The present study conducted in the Indian context is the first of its kind considering the large number of CPA tumor patients evaluated.

Key words: Cerebellopontine Angle tumors, Acoustic tumor, Nonacoustic tumor, DPOAE.

1. Introduction:

The Cerebellopontine Angle (CPA) is one of the most common sites of intracranial tumors and approximately 10% of them originate in the CPA. The Hearing Loss (HL) associated with CPA tumors can be attributed to dysfunction in either the cochlea or retrocochlear structures (Auditory Nerve-AN and central auditory pathway) or both. Acoustic tumors are those which arise from VIIIth Cranial Nerve and Nonacoustic tumors are those tumors which arise from Vth, VIIth, IXth, Xth, XIIth Cranial Nerves and other tumors such as Meningiomas, Epidermoid, arachnoid cyst, primary melanoma, hemangioma, lipoma dermoid and teratoma. The cochlear dysfunction has been theorized to arise from any one or a combination of the following pathophysiological mechanisms: vascular compromise, hydrops, degradation of inner ear fluids/tumor toxicity, deafferentation and loss of efferent nerve tuning (Telischi, Roth, Stagner, Lonsbury-Martin & Balkany, 1995). With Otoacoustic Emission (OAE) evaluation, it is possible to assess the cochlear status, in particular the Outer Hair Cell (OHC) dysfunction. Studies have reported the relative frequency of occurrence of cochlear/noncochlear/mixed pattern of OAE in acoustic and nonacoustic CPA tumors. Mobley et al (2002) compared the frequencies of cochlear and noncochlear patterns of Distortion Product (DP) OAE in nonacoustic tumors of the CPA/Internal Auditory Meatus (IAM) with that of Vestibular Schwannoma (VS). They reported cochlear pattern in 58% and noncochlear pattern in 42% in

their study group of VS patients. Acoustic tumors differed from nonacoustic tumors in their propensity to cause cochlear dysfunction. The preponderance of acoustic tumors to cause cochlear dysfunction have also been reported by studies of Telischi et al (1995), Prasher et al (1995), Oeken (1996), Ferber-Viart, Colleaux, Laoust, Dubreuil and Duclaux (1998), Telischi (2000) and Odabasi, Telischi, Gomez-Marin, Stagner and martin (2002).

2. Method:

The clinical group comprised of 98 patients with CPA tumor. Inclusion criteria comprised of a) Confirmed CPA tumor patients as evidenced by neuroradiological findings on Magnetic Resonance Imaging or Computed Tomography; (b) Patients aged between 15-55 years; (c) Patients giving signed informed consent. Histopathological findings categorised the tumors into acoustic and nonacoustic types. The study was undertaken at National Institute of Mental Health and Neuro Sciences (NIMHANS), Bangalore, India. The NIMHANS ethics committee had approved the protocol for the study.

Puretone audiometry: Grason Stadler Incorporates (GSI) 61, dual channel clinical-diagnostic audiometer with TDH-50 P earphones was used for measuring the puretone thresholds. Puretone audiometry was undertaken in a sound treated audiometric room (two room situation) adhering to the standard protocols including calibration of the audiometer as per American National Standards Institute 1996 standards.

OAE: The OAE evaluations were carried out using an Otoacoustic Emission Analyser DP Echoport plus TE+DPOAE Screener/analyser (Otodynamics Ltd. ILO292, V2.0. The testing was carried out in a quiet room. The DPOAEs were measured by presenting two puretone stimuli (f1 and f2) simultaneously to the ear. The f2/f1 ratio was maintained at 1.2. The intensity of both stimuli was kept at moderate levels, i.e., stimulus settings of 65 decibel Sound Pressure Level (dB SPL) (L1) and 60 dB SPL (L2) for f1 and f2 respectively. Two points per octave DPOAE resolution was utilised to obtain the DP gram. Two thousand stimulus repetitions were recorded in DPOAE evaluation. Even though due to intermodulation distortion responses occur from different frequency regions, emissions from 2f1-f2 are believed to be the largest and are commonly utilized in OAE measurements. In the present study, OAEs and corresponding noise floor were measured at 1 kilo Hertz (kHz), 1.5 kHz, 2 kHz, 3 kHz, 4 kHz and 6 kHz. At each of these frequencies, the difference in amplitude between OAEs and noise floor were estimated.

The puretone thresholds and DPOAEs of tumor ears were compared. The DPOAE results were divided according to a subjectively based, qualitative analysis into cochlear, noncochlear and mixed categories by comparing the emission amplitude (the difference in amplitude between DPOAEs and corresponding noise floor) to the related hearing levels at the same test frequencies (i.e; 1, 2, 4 and 6 kilo Hertz). When diminished DPOAE levels were associated with impaired hearing (i.e; puretone threshold > 25 decibel), the ears were categorised as cochlear. In contrast, when DPOAE levels were within the normal range or were better than expected based on the accompanying behavioral HL, the ears were designated as noncochlear. A combination of cochlear and noncochlear effects manifested at different areas of the measured frequency spectrum was categorised as mixed group. The prevalence of cochlear/noncochlear/mixed types was separately estimated in acoustic and nonacoustic tumor types and statistical significance between them was determined.

Chi-square test was employed to determine the statistical significance between cochlear/noncochlear/mixed types in acoustic and nonacoustic tumor types. The mean difference between variables was considered statistically significant at $P<0.05$.

3. Results

Out of 98 CPA tumor patients, 61 were categorised in the acoustic and 37 in the nonacoustic groups. The patients were divided into cochlear, noncochlear and mixed DPOAE pattern groups based on the findings observed in tumor ears. Cochlear patterns were seen in 54 (89%) acoustic patients and 16 (43%) nonacoustic patients, whereas noncochlear patters emissions were noted in 6 (10%) acoustic patients and 12 (32%) nonacoustic patients. Mixed patterns were seen in 1 (1%) acoustic patient and 9 (25%) nonacoustic patients. The difference in the distribution of cochlear, noncochlear and mixed patterns between acoustic and nonacoustic patients was statistically significant ($p<0.001$; chi-square value=24.63).

4. Discussion:

Recording of DPOAE can be considered as an effective means to measure cochlear OHC function. Puretone audiometric and DPOAE findings can be used to distinguish the relative frequency of cochlear versus noncochlear (neural) versus mixed (cochlear and noncochlear) dysfunction in patients with CPA tumors. Various authors have reported the preponderance of cochlear pattern of DPOAE in acoustic tumor patients. Telischi et al (1995) noted 59% cochlear, 30% noncochlear and 11% mixed pattern in their study group. The authors opined that the acoustic tumors may cause Hearing Impairment (HI) according to two types of influence that act at different levels of the peripheral auditory system. The tumor's cochlear effect is considered to be most likely caused by an indirectly mediated compromise of the Organ of Corti's vascular supply whereas; the direct pressure of the tumor on the VIIIth Cranial Nerve (CN) is believed to be responsible for the noncochlear effects. Oeken (1996) classified 54% cochlear and 29% noncochlear pattern in the patient group. Telischi (2000) classified 57% of the tumor ears in the cochlear group, 41% in the noncochlear group and 2% in the mixed group. Odabasi et al (2002) classified 60.5% of patients to the cochlear group.

Similar to the findings noted above, in the present study the acoustic group (n=61) had 89% cochlear pattern, 10% noncochlear pattern and 1% mixed pattern DPOAE in the tumor ears. Hence, the predominance of cochlear pattern of DPOAE in the acoustic tumor ears was obvious. In contrast, the nonacoustic group had 43% patients with cochlear pattern, 32% with noncochlear pattern and 25% with mixed pattern of DPOAE. The difference in DPOAE patterns between the acoustic and the nonacoustic groups were statistically significant, suggesting that the acoustic and the nonacoustic CPA tumors may cause hearing loss via different mechanisms in some cases, i.e., hearing loss in the acoustic tumors occur more commonly by affecting cochlear function through vascular compromise, inner ear toxicity or reduced efferent control of cochlear mechanical tuning. The same aspect may not be applicable with respect to nonacoustic tumors. Nonacoustic tumors seem to affect neural mechanisms more frequently.

In a similar study, Mobley et al (2002) compared the frequencies of cochlear and noncochlear patterns of DPOAE in patients with nonacoustic tumors of the CPA/IAM with those in patients with acoustic tumors. They noted that in the nonacoustic group, 42% patients had a cochlear pattern and 58% had a noncochlear pattern, whereas, in the acoustic group, 58% patients had a cochlear pattern and 42% had a noncochlear

pattern. The differences between the nonacoustic and acoustic groups were statistically significant in their study. The authors concluded that the acoustic tumors appeared to differ from nonacoustic tumors of the CPA in their propensity to cause sensory versus neural HL.

Based on the findings obtained in the present study, it seems that the acoustic and the nonacoustic tumors of the CPA share some of the pathophysiological mechanisms with regard to HL. However, the acoustic tumors may exert a negative effect on the cochlea more frequently than the nonacoustic types.

5. Conclusion:

The preponderance of cochlear pattern of DPOAEs in acoustic tumors was clearly evident when compared with nonacoustic tumors.

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