

# Spontaneous Otoacoustic Emissions in Neonates and Effect of Contralateral White Noise Stimulation

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**Abstract:** Neonates younger than the age of 12 weeks (10 full-term, 20 preterm) had an audiological assessment consisting of brainstem audiometry, tympanometry, transiently evoked otoacoustic emissions, and spontaneous otoacoustic emissions with contralateral white noise stimulation. Results from brainstem audiometry, tympanometry, and transiently evoked otoacoustic emissions suggested normal middle ear function and normal cochlear function. All full-term neonates had multiple spontaneous otoacoustic emissions, and contralateral white noise stimulation resulted in enhancement of emissions in 80%, whereas in 20% the emissions were suppressed. In preterm neonates, spontaneous otoacoustic emissions were present in 55%. These emissions were mostly solitary and, in 64%, showed suppression with contralateral white noise stimulation. Embryological data taken into consideration suggest that the pattern of spontaneous otoacoustic emissions in preterm neonates is more likely related to immaturity of the central auditory pathway rather than the cochlea.

**Key Words:** central auditory pathway; cochlear maturity; contralateral white noise stimulation; neonates; spontaneous otoacoustic emissions

Recent publications concerned with the maturity of the auditory system in neonates and with incorporating otoacoustic emissions in their assessment conclude that the cochlea still is undergoing maturation after birth [1–4]. In contrast to these interpretations are embryological data, which suggest that maturity of the cochlea is achieved well before birth [5–10]. This discrepancy has encouraged us to collect our own data using brainstem audiometry and otoacoustic emissions in neonates referred to our clinic for a routine audiological assessment.

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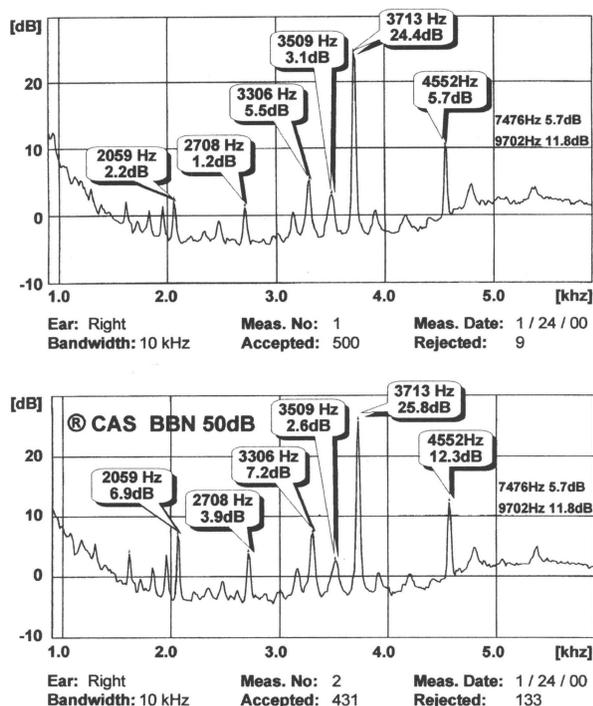
Portions of this study were presented at the Twenty-Seventh Congress of the Neurootological and Equilibriometric Society, March 23–26, 2000, Bad Kissingen, Germany.

## METHOD

Over a period of 10 months, we examined 30 neonates (60 ears), all younger than 12 weeks of age and referred to our clinic for an audiological assessment. Of them, 10 neonates were full-term, and 20 neonates were preterm, with prematurity ranging from 6 to 8 weeks. The male-female ratio in both groups was 2:3. The test battery consisted of brainstem audiometry (Biologic Navigator, Chicago, IL); tympanometry (GSI 34, Grason Stadler, Littleton, MA); transiently evoked otoacoustic emissions (TEOAEs; 17 neonates); spontaneous otoacoustic emissions (SOAEs); and SOAEs with contralateral white noise stimulation at the intensity of 50 dB (Madsen Celesta, Madsen Electronics, Denmark). The tests were performed during natural sleep.

## RESULTS

All neonates had normal brainstem audiograms, except one who had a type B tympanogram suggesting middle ear fluid in one ear. This was consistent with normal



**Figure 1.** Multiple spontaneous otoacoustic emissions recorded in a full-term neonate at 12 weeks of age. Enhancement of emissions occurs with contralateral white noise stimulation.

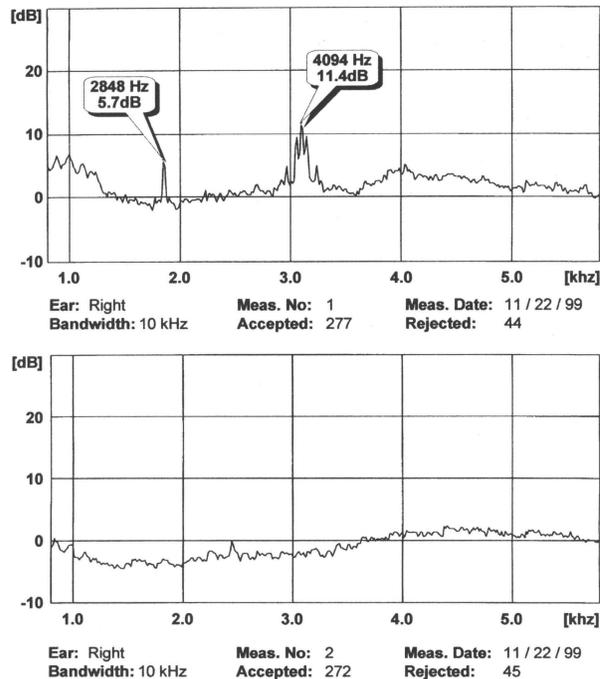
hearing levels, at least in the higher speech frequencies, and possible adequacy for normal speech and language development.

TEOAEs were present in 17 neonates (9 full-term, 8 preterm) and indicated normal cochlear function. All 10 full-term neonates had multiple SOAEs. With contralateral white noise stimulation, 8 showed enhancement of SOAEs (Fig. 1). Occasionally, additional emissions could be seen, and a frequency shift was observed. Two of the full-term neonates showed suppression with contralateral white noise stimulation. One of them was treated with gentamicin after birth, and one had a family history of hearing losses.

Of 20 preterm neonates, 11 (55%) had SOAEs, but these were not multiple but rather solitary (Fig. 2). Within this group, four (all female neonates) had enhancement of SOAEs with contralateral white noise stimulation, and 7 showed suppression with contralateral white noise stimulation.

## DISCUSSION

A general assumption is that the cochlea is the origin of otoacoustic emissions and that most likely the outer hair cells are their generator. Whether and how outer hair cells generate emissions still is a matter of debate and



**Figure 2.** Spontaneous otoacoustic emissions recorded in a 6-weeks' preterm neonate at the age of 12 weeks. Emissions are rather solitary and are suppressed with contralateral white noise stimulation.

speculation. Researchers agree that SOAEs are observed more often in the right ear, are prevalent in female subjects, have different frequency ranges in neonates and adults, and are multiple in neonates [1, 2, 11, 12]. SOAEs also have a higher intensity level in neonates and young children, and this level gradually decreases with age, as demonstrated in longitudinal studies [13].

SOAEs are measured infrequently in neonates. This tendency appears to be related to a poor response in persons in this age group. In a comparison study, 62.5% of subjects demonstrated SOAEs, as compared to 89.7% who demonstrated TEOAEs (14).

Generally, TEOAEs are regarded as a suitable cochlear test and can be helpful in the differential diagnosis of hearing losses. Their detection in the presence of a sensorineural hearing loss still would indicate normal outer hair cell function [15]. In neonates, TEOAEs have been used successfully in the diagnosis of auditory neuropathy [16–18], in which their presence indicates normal cochlear function, and abnormal brainstem audiometry suggests retrocochlear pathology.

Normal cochlear function at birth is not surprising when embryological data are taken into consideration. Electron-microscopical studies of the cochlea, and particularly of the organ of Corti with the onset of auditory function in mind, confirm that the peripheral auditory

organ is ready for sending afferent impulses at nearly 24 weeks of embryonic age [6]. Hair cells begin to develop between 10 and 11 gestational weeks, and the membranous labyrinth is developed fully in the early fetal period lasting from day 75 to day 80 of gestation [5].

A recent study concludes that functional maturity of the outer hair cells is complete at least at 33 weeks but that functional maturity of the medial olivocochlear system possibly is complete just before full-term birth [10]. Very early in the embryonic life (at 11 weeks of gestation), auditory receptors begin maturation and establish connections with the peripheral and the central nervous system [7]. At 22 weeks' gestation, inner hair cells and outer hair cells have an adult stereociliary pattern. Although maturation is achieved in the last trimester of pregnancy [8], complete cochlear maturation still is suspected to occur at 28 weeks [9].

Thus, expected maturity of the inner ear occurs early. This is confirmed by our findings wherein normal results from brainstem audiometry and those supported by TEOAEs point toward normal cochlear function in both full-term and preterm neonates. In the past, the pattern of SOAEs in neonates has been interpreted to indicate the developmental status of the cochlea. A change of pattern in premature neonates is assumed automatically to be due to lack of maturity of the cochlea rather than lack of maturity of the central auditory pathway [2, 4].

Our findings require the consideration of a different interpretation. One also must take into account the fact that the pattern of emissions with contralateral stimulation could affect outer hair cell function via the olivocochlear bundle [19]. The olivocochlear bundle might not have reached maturity, although indications suggest that its maturity has been reached at birth [20].

Our results would suggest that SOAEs that are suppressed with contralateral white noise stimulation and absent SOAEs are not due to immaturity of the inner ear but rather to immaturity of the central auditory pathway. This central immaturity might involve the medial olivocochlear bundle. We believe that a mature auditory system after birth would exhibit multiple SOAEs and that these emissions would be enhanced with contralateral white noise stimulation. A deviation from this pattern after birth would suggest immaturity of the central auditory pathway.

## CONCLUSION

In the neonate, absence of SOAEs and suppression with contralateral white noise stimulation in the presence of normal brainstem audiometry and presence of TEOAEs would indicate immaturity of the central auditory pathway.

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