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On the physiological location of otoacoustic emissions

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Abstract

During recent years, much attention has been paid otoacoustic emissions in the clinical audiological practice. The received view locates their origin in the cochlea, more precisely in the outer hair cells. It is, however, still uncertain if there is an interaction between the ears regarding otoacoustic emissions. Earlier findings suggest an interaction at the level of the olivocochlear bundle. The aim of this pilot study was to find out if there is any interaction between the two cochleae in the case of otoacoustic emissions. Five subjects with normal hearing participated. Recordings were made of spontaneous otoacoustic emissions during the presentation of contralateral stimuli at three different frequencies (500, 1000 and 2000 Hz). In general, contralateral stimulation did not provoke otoacoustic emissions. It was concluded that otoacoustic emissions could be part of the fine-tuning mechanism in the cochlea. The frequency resolution, e.g. for speech, depends on very fast modulation of the incoming signal. Due to the neural distance, this modulation would lag behind, if otoacoustic emissions in one ear would effect the opposite one.

1. Introduction

1.1 Otoacoustic emissions

Otoacoustic emissions (OAE) are acoustic energy produced by the cochlea (most probably by the movement of the outer haircells). This energy can be dependent on a stimulus, e.g. transiently evoked otoacoustic emissions (TEOAE) or spontaneous (SOAE). In the latter case, sounds that are recordable are emitted from the unstimulated cochlea. In a population with normal cochlear function, spontaneous otoacoustic emissions are present in about one-third. If one ear displays spontaneous otoacoustic emissions, the probability that also the other cochlea shows spontaneous otoacoustic emissions is doubled. This has been taken as evidence that spontaneous otoacoustic emissions could be an innate feature (Probst et al. 1991).

1.2 Previous evidence for cochlear location of otoacoustic emissions

Evoked otoacoustic emissions (EOAE) may be suppressed electrically at the level of the fourth ventricle or by a broadband contralateral stimulation (Maison et al., 2001). The latter results in a delayed suppression of evoked otoacoustic emissions, which takes some tens of milliseconds to reach a significant level (e.g. Hill et al., 1997 and Maison et al.,

2001). This effect is reduced or completely removed after vestibular neurectomy (for example in severe cases of monaural Mb. Ménière) due to the removal of the efferent auditory neurones at the same time as the vestibular neurones (Giraud et al., 1995). This finding points to an intracochlear phenomenon.

Transiently evoked otoacoustic emissions are evoked by a stimulus no longer than 2 ms and consisting of a broadband frequency click. The registration of the transiently evoked otoacoustic emissions is made during an interval of about 20 ms, during which the acoustic response from the cochlea declines. Different frequencies require different latencies for measurement, since high frequencies are placed at the base of the cochlea and low frequencies at the helicotrema. The latency for 1000 Hz stimulus is about 10-16 ms (Probst et al., 1991). This also suggest a cochlear location of the otoacoustic emissions (Pickles, 1988).

2 Method

The subjects were five males at the ages of 23, 24, 26, 27 and 28 years. The informants underwent a standard audiological examination and were judged to have normal hearing.

The examination was conducted in the following way. To start with, the ear status of the informants was checked with an otoscope. Next, ordinary pure tone audiometry was performed using air- and boneconducted stimuli to examine if the subjects had normal hearing and also to obtain the reference values required for the calculation of the contralateral stimulation. The equipment used was a Grason-Stadler audiometer (GSI 16), earphones of type Telephonics THD-50P and boneconductor Radioear B-71.

Furthermore, spontaneous otoacoustic emissions were measured for the right ear. This procedure was then repeated with stimulation at 500, 1000 and 2000 Hz in the opposite ear in the order mentioned. The reason for choosing these frequencies was partly their coherence with the pure tone average, which is used in the clinical evaluation of hearing, and partly to limit the scope of the test. The same procedure was performed with the left ear as the registration ear. The levels of stimulation were selected by adding 40 dB to the best air- and boneconduction thresholds at the frequencies chosen. This was done to avoid crossover interference at the probe microphone. A Capella OAE-equipment together with a Celesta-probe (Madsen Electronics) were used for recording the emissions. The contralateral stimulation was presented with the audiometer and earphones mentioned earlier. All measurements took place in a soundproofed room used in clinical audiological measurements (ISO 8253, 1989).

3 Results

3.1 Results

In general, there was no influence on the spontaneous otoacoustic emissions from the contralateral stimulation. Tables 1 and 2, show the emissions recorded in each of the subjects. Three of the subjects (*Male* 23, 24 and 28) do not show any spontaneous otoacoustic emissions at all, neither with nor without contralateral stimulus. The other two males (*Male* 26 and 27) each display a number of spontaneous otoacoustic emissions in both ears in the interval between 534 and 2072 Hz. For the *Male* 26, there is not effect of the different stimuli presented for registrations in either the left or the right ear. No differences were found between the unevoked and the evoked spontaneous otoacoustic emissions, with one exception. When the recording was in the left ear with 500 Hz as contralateral stimulation, there was a slight increase in frequency, 13 Hz, in the *emission* (2059 Hz), which prevails in the other measurements. *Male* 27 has emissions from both left and right ear with and without contralateral stimulation. *Male* 27 shows more variation

Table 1. Presence and frequency (Hz) of spontaneous otoacoustic emissions. Registration in the right ear.

Informant	Stimulus			
	None	Left 500 Hz	Left 1000 Hz	Left 2000 Hz
Male 26	1538	1538	1538	1538
Male 24	None	None	None	None
Male 27	534 724	534 724 1004	546 1004	546
Male 28	None	None	None	None
Male 23	None	None	None	None

Table 2. Presence and frequency (Hz) of spontaneous otoacoustic emissions. Registration in the left ear.

Informant	Stimulus			
	None	Left 500 Hz	Left 1000 Hz	Left 2000 Hz
Male 26	1297 1525 1805 2059	1297 1525 1805 2072	1297 1525 1805 2059	1297 1525 1805 2059
Male 24	None	None	None	None
Male 27	559 775	559 775	559 775	559 788
Male 28	None	None	None	None
Male 23	None	None	None	None

in frequency than *Male 26*. *Emission (534 Hz)* and *emission (724 Hz)* were recorded in the right ear without contralateral stimulation. These emissions are also present with stimulation at 500 Hz, but *emission (534 Hz)* disappears during stimulation at 1000 and 2000 Hz, although a new emission, *emission (546 Hz)*, appears. *Emission (724 Hz)* disappears totally with stimulation at these frequencies. Another emission appears, *emission (1004 Hz)*, which only exists during contralateral stimulation at 500 and 1000 Hz. When the registration was made in the left ear, no deviations were noted except during stimulation at 2000 Hz. *Emission (775 Hz)* disappears and a new one, *emission (788 Hz)*, appears.

3.2 Source of errors

The different types of anomalies in the results can all be accounted for reasonably well. In the case of *Male 26*, there was a difference in the frequency recorded during contralateral stimulation at 500 Hz with registration in the right ear. This deviation only occurs in this case. Its reasonable to believe this is due to an equipment malfunction. Concerning the results of *Male 27*, who exhibits great variation, an overall explanation may account for all these deviations. Since all recorded emissions were located in the low-frequency register, thus in the high-level noise register, it is likely that the S/N-ratio has been crucial. When a signal varies at the threshold of what is an acceptable S/N-ratio for the equipment, whether

it is the emission, the noise, or both that is fluctuating in amplitude, the emission will, depending on this ratio, sometimes be recorded and sometimes not. Thus, there is a reasonable explanation for these deviations considering the S/N-ratio and operations of the equipment used.

4 Conclusions

According to the results of the present study, there is no connection between registration of emissions and contralateral stimulation, i.e., no correlation between spontaneous otoacoustic emissions and contralateral influence. This suggests a cochlear location of otoacoustic emissions. It seems reasonable to propose that otoacoustic emissions are located in the peripheral part of the eighth cranial nerve. They seem to perform an instant modulation of frequency in the basilar membrane, an important mechanism in the signal processing of the ear. Since directional hearing and noise reduction depends on interaural differences, it is hard to believe that there should be a process working against these differences, which would be the extension of an interaural influence. The tendency in this material seems to be consistent, although a larger survey is required to fully examine the relationship.

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