Subclinical permanent hearing disorders in patients with sleep apnea

Mario Fabiani
Valerio Fabiani
Andrea Rea

Department of Sense Organs, “Sapienza” University of Rome, Italy

Corresponding Author:
Mario Fabiani
Otolaryngological Clinic, Policlinico Umberto I
00161 Rome, Italy
E-mail: mario.fabiani@uniroma1.it

Summary

Background: the obstructive sleep apnea syndrome is characterized by partial or complete obstruction of the upper airway and it affects 5% of the adult population. Repeated episodes of partial or total obstruction of the upper airway are responsible for the decrease in oxygen saturation in arterial blood (SaO2) and therefore hypoxia. Hypoxia is linked to a damage of the cochlear structures: vascular stria, afferent synapse, inner hair cells but also outer hair cells of the basal turn which appear to be the most vulnerable. Such damages are responsible for sensorineural hearing loss. Objectives: aim of the study is to determine the correlation between cochlear damage and hypoxia through the Otoacoustic Emissions (TEOAE and DPOAE), to assess the influence of hypoxia on hearing in patients with severe OSAS and to prevent the early damage from becoming permanent. Methods: 20 patients with severe obstructive sleep apnea syndrome (AHI> 30 events/hour of sleep) and 20 healthy subjects, non-snorers and clinically non-OSAS, underwent domiciliary polysomnographic examination, Transient Evoked Otoacoustic Emissions (TEOAE) and Distortion Product Otoacoustic Emissions (DPOAE). Results: compared to the control group (2 out of 20) OSAS patients (10 out of 20) have right ear DPOAE pathologies with a frequency of 3kHz,4 kHz: left ear DPOAE pathologies have only a 3 kHz frequency. Finally, right ear TEOAE appears to be pathological at 2 kHz frequency in 9 out of 20 OSAS patients and in 10 out of 20 OSAS patients (always right ear) at a frequency of 3kHz. 8 OSAS patients have left ear pathological TEOAE at 2 and 3 kHz frequencies. Conclusions: the DPOAE and TEOAE are predictive of cochlear damage resulting from hypoxia before a sensorineural hearing loss can be measured by routine audiological tests.

KEY WORDS: hypoxia, obstructive sleep apnea syndrome, otoacoustic emissions.

Background

Obstructive Sleep Apnea Syndrome (OSAS) is a respiratory disturbance of sleep characterized by repeated episodes of partial or complete clogging of the upper respiratory tract that takes place during the inspiratory phase (1). It is a common and often overlooked pathology that affects 2% and 4% of middle aged men and women and more than 42% of subjects aged 65 or over (2, 3). The reduction of air flow in the upper respiratory tract is called hypopnea, while cessation of the air flow is called apnea. In order to determine the severity of apnea, an index has been established which relates to the number of episodes occurring per hour of sleep (known as the Apnea Hypopnea Index or AHI). An AHI less than 5 is considered normal. An AHI between 5 and 15 is considered mild; an AHI 15 to 30 is moderate and anything beyond 30 is deemed severe (4, 5). Repetitive, complete or partial clogging of the upper respiratory tract, that characterizes OSAS in sleep, is responsible for a reduction in oxygen saturation in arterial blood (SaO2) and therefore hypoxia. Ear hypoxia is linked to damage of cochlear structures, vascular streaks, afferent synapses, internal ciliated cells but above all it is external ciliated cells of the basal turn which seem most vulnerable: this damage is responsible for sensorineural hearing loss (6, 7). Acoustic otoemissions are sounds recordings which actively issue from the human cochlea. They are usually taken from the contractile activity of outer ciliated cells and mechanical-structural features of the basilar membrane with the transformation of mechanical energy into sound energy (8-10).

The acoustic otoemissions are present either spontaneously (SOAE: Spontaneous Otoacoustic Emissions), after stimulation by sound TEOAE (created by transitory stimuli) or through DPOAE stimuli (created as a result of distortion) (11, 12). The method for detecting acoustic evoked otoemissions is to send stimulus applied via the external ear canal through a special probe, housing a miniature microphone and an escape tube in order to avoid excessive acoustic coupling between microphone and speaker. The otoemissions captured by the microphone are sent to a computerized device that routinely filters certain artifacts (13-15).
Objectives of the study

1. To determine the existent correlation between damage to the cochlear and hypoxia through otoacoustic emission testing;
2. To evaluate the influence of hypoxia on patients’ hearing with severe apnea (AHI > 30 events per hour of sleep) in order to identify any changes not detectable through standard audiometric examinations;
3. To act on damages in order to prevent them from becoming permanent.

Materials and methods

Our case study included people visited at the ENT clinic of the University of Rome from February 2009 to June 2009 because of their heavy snoring and disturbed sleep patterns over a number of years.

Inclusion criteria:
• AHI > 30 events/hour of sleep
• Aged between 40 and 70
• No clinical signs of hypoacusis.

Exclusion criteria:
• Otologic illness or disease
• ENT surgery
• Chronic exposure to noise
• A different pathology to OSAS
• Diabetes mellitus
• Cardio-vascular pathology
• Dyslipidemia.

In this case study, 20 patients were identified as suffering from severe obstructive sleep apnea (AHI > 30 events/per hour of sleep) by polysomnography, performed via a portable home monitoring system. The group of OSAS patients consisted of 7 males and 13 females aged between 40 and 70 years (mean age = 55.05). The group of OSAS patients was compared with a control group consisting of 20 healthy subjects who did not snore and who clinically were non-OSAS. The group comprised 8 males and 12 females aged between 40 and 70 years (mean age = 55.6).

All patients underwent:
• a polysomnographic examination at home which determined the presence of apnea/ hypopnea, snoring, heart rate and arterial oxygen saturation
• Pure Tone Audiometry (PTA)
• TEOAE
• DPOAE.

Analysis of variance showed that OSAS patients had a statistically significant difference from the healthy control group within the examined polysomnographic parameters (Tab. 1).

In OSAS patients the mean AHI was 47.44 (range 32.2 - 77.5), while in the control subjects it was 2.32 (range 0-4.3). OSAS patients also have significant sleep fragmentation and a marked hypoxemia, indicated by their average oxygen saturation.

16 out of 20 OSAS patients with pathological TEOAE and DPOAE, as compared to the control group, showed a significant reduction in the amplitude of the DP-gram especially for frequencies between 2 and 4 kHz. In addition, there was a positive correlation between AHI, TEOAE and DPOAE; in particular an increase in AHI results in an increase of cochlear damage to both the left and right ears. There is also a negative correlation between SaO2, DPOAE and TEOAE; the decrease of oxygen saturation increases cochlear damage to the right ear with respect to the left.

PTA was within normal values in both ears of all patients.

Statistical analysis

To better investigate the nature of this relationship, a χ² analysis was performed to check the frequencies at which the OSAS group and control groups differed in DPOAE and TEOAE pathologies.

Results

The data show that OSAS patients (10 out of 20) compared to the control group (2 out of 20) have right ear DPOAE pathologies with not only a 3kHz frequency, but also a 4 kHz frequency; left ear DPOAE pathologies, instead, are only a 3kHz frequency (Tabs 2-4).

Table 1 - Polysomnographic characteristics of the two groups of subjects.

<table>
<thead>
<tr>
<th></th>
<th>OSAS Group Average (DS)</th>
<th>Control Group Average (DS)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI</td>
<td>47.44 (14.3)</td>
<td>2.32 (1.21)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>SaO2%</td>
<td>93.6 (2.52)</td>
<td>98.97 (0.617)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>DPOAE</th>
<th>Ear Concerned</th>
<th>Frequency Concerned (kHz)</th>
<th>χ²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>OSAS Group</td>
<td>10/20 right</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0.006</td>
</tr>
<tr>
<td></td>
<td>6/20 right</td>
<td>4.0</td>
<td>4,329</td>
<td>&lt;0.037</td>
</tr>
<tr>
<td></td>
<td>10/20 left</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0.006</td>
</tr>
</tbody>
</table>

Table 3 - DPOAE in Control group.

<table>
<thead>
<tr>
<th>Control Group</th>
<th>DPOAE</th>
<th>Ear Concerned</th>
<th>Frequency Concerned (kHz)</th>
<th>χ²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/20 right</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0.006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/20 right</td>
<td>4.0</td>
<td>4,329</td>
<td>&lt;0.037</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2/20 left</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0.006</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Finally the TEOAE were found to be significantly different between the experimental group and the control group: 9 out of 20 OSAS patients appear to be pathological at 2 kHz frequency in the right ear and 10 out of 20 OSAS patients at a frequency of 3 kHz, always in the right ear. 8 OSAS patients are pathological TEOAE in the left ear at frequencies of 2 and 3 kHz (Tabs 5-7).

This indicates that frequencies of 3, 2 and 4 kHz DPOAE and TEOAE are what separates OSAS patients from the control group.

**Discussion and Conclusions**

From the preliminary data in our possession we can say that DPOAE and TEOAE can predict a cochlear damage resulting from hypoxia before a sensorineural hearing loss is measured by routine PTA. Obstructive sleep apnea is a common pathological condition, debilitating and often unrecognized, which can affect both adults and paediatric patients.

Furthermore, a prompt and early OSAS diagnosis is recommended as well as, when necessary, a prompt treatment in order to significantly improve the life quality of patients and any comorbidities, thus preventing car accidents and work related to excessive daytime sleepiness.

If OSAS patients were all promptly identified, medical costs would consequently be minimized. Difficulty in diagnosing OSAS is often due to the fact that many doctors are still unable to recognize this disease and that signs and symptoms are not specific. These conditions lead, also, in most cases to a late diagnosis.

**References**