SUBCLINICAL PERMANENT HEARING DISORDERS IN PATIENTS WITH SLEEP APNEA

ALTERAZIONI UDITIVE SUBCLINICHE PERMANENTI NEI PAZIENTI CON APNEE NEL SONNO

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Parole chiave: ipossia, sindrome delle apnee ostruttive nel sonno, otoemissioni acustiche

Abstract

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 reduction in oxygen saturation in arterial blood (SaO2) and therefore hypoxia. The 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 the hearing of patients with severe OSAS and to prevent the early damage may become 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 frequency of 3 kHz.

Finally the TEOAE in the right ear appear to be pathological at 2 kHz frequency in 9 out of 20 OSAS patients in the right ear and in 10 patients OSAS out of 20 at a frequency of 3kHz. OSAS patients are pathological TEOAE in the left ear at frequencies of 2 and 3 kHz.

**Discussion and 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.

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**Abstract**

**Introduzione:** La sindrome da apnea ostruttiva del sonno è caratterizzata dall’ostruzione parziale o completa delle vie aeree superiori e che colpisce il 5% della popolazione adulta. Episodi ripetuti di ostruzione parziale o totale delle vie aeree superiori sono responsabili della riduzione della saturazione dell’ossigeno nel sangue arterioso (SaO2) e quindi dell’ipossia L’ ipossia è legata ad un danno delle strutture cocleari: stria vascolare, sinapsi afferente, cellule ciliate interne ma soprattutto cellule ciliate esterne del giro basale che sembrano essere le più vulnerabili; tali danni sono responsabili di ipoacusia neurosensoriale.

**Obiettivi:** Determinare la correlazione tra danno cocleare e ipossia attraverso le emissioni otoacustiche (TEOAE e DPOAE), valutare l’influenza dell’ipossia sull’ udito dei pazienti con OSAS grave ed agire sui danni precoci per prevenire quelli permanenti.

**Metodi:** 20 pazienti con Sindrome da apnea ostruttiva del sonno di grado severo (AHI> 30 eventi / ora di sonno) e 20 soggetti sani, non russatori e clinicamente non-OSAS, sono stati sottoposti ad esame polisonnografico domiciliare, otoemissioni acustiche evocate transitorie (TEOAE) e prodotti di distorsione (DPOAE).

**Risultati:** 10 pazienti OSAS hanno DPOAE patologiche all’orecchio destro alla frequenza 3 e 4 kHz: all’orecchio sinistro le DPOAE sono patologiche solamente alla frequenza 3 kHz.

Le TEOAE sono patologiche all’orecchio destro alla frequenza 2 kHz in 9 pazienti OSAS e alla frequenza 3 kHz in 10 pazienti OSAS, sempre nel orecchio destro. 8 pazienti OSAS hanno TEOAE patologiche all’orecchio sinistro alle frequenze 2 e 3 kHz.

**Discussione e Conclusioni:** Le DPOAE e le TEOAE sono predittive di un danno cocleare conseguente a ipossia, prima di una perdita uditiva neurosensoriale misurabile con esami audiologici di routine.
Subclinical permanent hearing disorders in patients with sleep apnea

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 which occur per hour of sleep (this is known as the Apnea Hypopnea Index or AHI). An AHI of less than 5 is considered normal. An AHI of between 5 and 15 is considered mild; an AHI of 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. Hypoxia in the ear 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 ciliate cells and mechanical-structural features of the basilar membrane with the transformation of mechanical energy into sound energy (8, 9, 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, inside of this is housed 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, 14, 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 these becoming permanent.

Materials and methods
Our case study comprised people seen at the ENT clinic of the University of Roma 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 using a portable home monitoring system.

The group of OSAS patients, consisted of 7 males and 13 females, their ages were 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 patients with OSAS differed significantly statistically from those of the healthy control group within the examined polysomnographic parameters.

Table 1: Polysomnographic characteristics of the two groups of subjects

<table>
<thead>
<tr>
<th></th>
<th>OSAS Group</th>
<th>Control Group</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>

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). Patients with OSAS also have significant sleep fragmentation and a marked hypoxemia, indicated by their average oxygen saturation.

16 out of 20 OSAS patients 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 the 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 over that of 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 shows that the OSAS patients (10 out of 20) compared to the control group (2 out of 20) have right ear DPOAE pathologies with not only a frequency of 3kHz, but also of 4 kHz: left ear DPOAE pathologies instead, are only of 3kHz frequency.

Table 2: DPOAE in OSAS group

<table>
<thead>
<tr>
<th>DPOAE</th>
<th>OSAS Group</th>
<th>Ear Concerned</th>
<th>Frequency Concerned (kHz)</th>
<th>$X^2$</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10/20</td>
<td>right</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0,006</td>
</tr>
<tr>
<td></td>
<td>6/20</td>
<td>right</td>
<td>4.0</td>
<td>4,329</td>
<td>&lt;0,037</td>
</tr>
<tr>
<td></td>
<td>10/20</td>
<td>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>DPOAE</th>
<th>Control Group</th>
<th>Ear Concerned</th>
<th>Frequency Concerned (kHz)</th>
<th>$X^2$</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2/20</td>
<td>right</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0,006</td>
</tr>
<tr>
<td></td>
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<td>right</td>
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Subclinical permanent hearing disorders in patients with sleep apnea

Table 4: percentage of falls in the frequency of the DPOAE of the right ear and the left ear

![Graph showing percentage of falls in the frequency of DPOAE for right and left ears.]  

Finally, 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 patients OSAS out of 20 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.

Table 5: TEOAE in OSAS group

<table>
<thead>
<tr>
<th>TEOAE</th>
<th></th>
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<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>OSAS</td>
<td>Ear</td>
<td>Frequency</td>
<td>X²</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>Group</td>
<td>Concerned</td>
<td>Concerned</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(kHz)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9/20</td>
<td>right</td>
<td>2.0</td>
<td>8,533</td>
<td>&lt;0,003</td>
<td></td>
</tr>
<tr>
<td>10/20</td>
<td>right</td>
<td>3.0</td>
<td>7,619</td>
<td>&lt;0,006</td>
<td></td>
</tr>
<tr>
<td>2/20</td>
<td>right</td>
<td>4.0</td>
<td>7,025</td>
<td>&lt;0,008</td>
<td></td>
</tr>
<tr>
<td>8/20</td>
<td>left</td>
<td>2.0</td>
<td>7,025</td>
<td>&lt;0,008</td>
<td></td>
</tr>
<tr>
<td>8/20</td>
<td>Left</td>
<td>3.0</td>
<td>4,800</td>
<td>&lt;0,028</td>
<td></td>
</tr>
</tbody>
</table>

Table 6: TEOAE in Control group

<table>
<thead>
<tr>
<th>TEOAE</th>
<th></th>
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<tbody>
<tr>
<td>Control</td>
<td>Ear</td>
<td>Frequency</td>
<td>X²</td>
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<td></td>
</tr>
</tbody>
</table>
Table 7: Percentage of falls in the frequency of TEOAE in the right ear and the left ear.

![Graphs showing percentage of falls in the frequency of TEOAE](image)

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

**Discussion and Conclusions**

From the preliminary data in our possession we can say that DPOAE and TEOAE are predictive of cochlear damage resulting from hypoxia before that a sensorineural hearing loss is measured by routine PTA.

Obstructive sleep apnea are a common pathological condition, debilitating and often unrecognized, which can affect both adults and pediatric patients.

And it is necessary that OSAS would be diagnosed promptly and, when necessary, promptly treated in order to significantly improve the quality of life of patients and any comorbidities, and thus prevent car accidents and work related to excessive daytime sleepiness.

If every patient with OSAS would be readily identified, he will also cause a major reduction in medical costs. Often difficulty in diagnosing OSAS is 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.
Subclinical permanent hearing disorders in patients with sleep apnea

References

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