P wave dispersion and severity of obstructive sleep apnea syndrome

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Abstract Background: OSA is associated with an increased prevalence of coronary artery disease, heart failure and rhythm disturbance. Also, P-wave dispersion (Pd) reflects inhomogeneous atrial depolarization secondary to insults such as chronically elevated atrial pressure, ischemia, or metabolic stress that promote atrial structure remodeling and provide a substrate for atrial fibrillation. We aimed to investigate Pd in patients with OSA and to determine if there is any relationship with severity of the disease.

Patients and methods: This study was conducted in Chest & Cardiology Departments, Assuit University Hospital, Egypt on 40 OSA patients (29 males and 11 females), and 20 healthy controls. We excluded patients with COPD and any diagnosed cardiac disease. For every patient, we did a polysomnography and ECG.

Results: Pd was significantly more in OSA (98.50 ± 4.77 m/s) than controls (72.00 ± 3.37 m/s) (p value <0.05). Pd in severe, moderate and mild OSA were 111.43 ± 5.62 m/s, 95.00 ± 7.83 m/s and 65.71 ± 8.41 m/s, respectively with a significant positive correlation with severity of OSA. Multiple linear regression shows that systolic blood pressure and BMI are independently associated with Pd (β = 0.56, p = 0.00) (β = 0.27, p = 0.05).

Conclusion: Pd is increased and correlated with severity of OSA. Systolic blood pressure and BMI are independent risk factors for Pd. Follow up of patients to detect clinical implications is recommended.

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Introduction
Obstructive sleep apnea (OSA) is an important diagnosis for physicians to be considered because of its strong association with some of the most debilitating medical conditions, including hypertension, cardiovascular disease, coronary ar-
tery disease, insulin-resistant diabetes, depression, and sleepiness-related accidents [1].

It is characterized by repetitive upper airway collapse resulting in drops of oxygen saturation and sleep fragmentation with cortical arousals and activation of the autonomic nervous system. For a long time, OSA was recognized as a potential cardiovascular risk factor [2].

The relationship between OSA and cardiovascular disease has been attributed to many mechanisms such as increased sympathetic activity, oxidative stress, inflammation, metabolic disturbance and endothelial dysfunction [3].

P wave prolongation is an intermediate step in the accumulation of insults ultimately leading of AF [4].

The prolongation of intra-atrial and inter-atrial conduction time and the inhomogeneous propagation of sinus impulses are well known electro physiological characteristics of the atrium prone to fibrillate and have been evaluated using a simple electrocardiographic (ECG) marker which is P-wave dispersion (Pd) [5,6].

P wave dispersion is defined as the difference between the maximum and minimum duration of P waves [7].

We aimed to investigate Pd in patients with OSA and to determine if there is any relationship with severity of the disease.

Patients and method

Our cross-sectional study was conducted at Cardiology and Chest Diseases Departments, Assuit University Hospital, Egypt.

Inclusion criteria

Patients were diagnosed according to International Classification of Sleep Disorders published by the American Academy of Sleep, which stated that, diagnosis of obstructive sleep apnea could be made if the respiratory disturbance index (RDI) is ≥15, independent of occurrence of symptoms, or whenever an RDI > 5 is associated with any of the following: [8]

1. Sleep attacks, excessive daytime sleepiness (EDS), unrefreshing sleep, fatigue or insomnia
2. Awakenings with a choking sensation; or
3. Witnessed heavy snoring and/or breathing pauses referred by the partner (The patient may be unaware of this symptom – usually the bed partner is extremely aware of this).

Exclusion criteria

- Patients with COPD
- Pacemaker implantation, permanent or paroxysmal atrial fibrillation
- Pericarditis, valvular heart disease, pulmonary emboli
- Cardiomyopathies, pulmonary hypertension, abnormal serum electrolyte values, receiving any ant arrhythmic drugs, and
- Coronary artery disease

All patients were subjected to the following:

1. Full clinical history taking and clinical examination
2. ECG

All standard 12-lead ECGs were obtained simultaneously using a recorder set at 50 mm/s paper speed and 2 mV/cm standardization in a comfortable supine position. For standardization, ECG was taken between 10 and 11 a.m. During ECG recordings all patients breathed freely and did not speak. ECGs were numbered and presented to the analyzing investigators without name and date information. P-wave duration was measured manually in all simultaneously recorded 12 leads of the surface ECG. The onset of the P wave was defined as the point of first visible upward departure from baseline for positive waveform, and as the point of first downward departure from baseline for negative waveforms. The return to the baseline was considered to be the end of the P wave. The difference between the P_max and the P_min was calculated and defined as P wave dispersion (Fig. 1) [9].
3. Sleep study

**Polysomnography (PSG)**

It was done overnight at Assiut chest department sleep laboratory, with monitoring of oxygen saturation, heart rate, sleep stage by electroencephalography, nasal airflow, oral airflow, jaw muscle tone by electromyography, sleep position, and chest and abdominal movement.

The Apnea–Hypopnea Index (AHI) was calculated as the number of apneas and hypopneas per hour of sleep.

- Patients with AHI (5–15) were considered to have mild OSA.
- AHI (15–30) were considered to have moderate OSA.
- AHI (>30) were considered to have severe OSA.

**Statistical analysis**

Data were analyzed by statistical package for the social sciences (SPSS, version 16.0).

Continuous variables were expressed as Mean ± SD, while categorical variables were displayed in numbers and percentages.

Continuous variables were compared among the groups of patients with one-way ANOVA test for normally distributed data, while chi-square tests were performed for nominal variables.

Correlations between all variables were calculated with the Pearson correlation coefficient. Multiple linear regression analysis was utilized to identify the factors related to P wave dispersion. $P < 0.05$ was considered significant.

**Results**

We recruited 40 obstructive sleep apnea patients with a mean age ± SD of 48.68 ± 10.61 years and 20 healthy controls from March 2012 to April 2013. Baseline clinical characteristics of the study population are shown in Table 1.

Among our patients, 21 (52.5%) patients had severe obstructive sleep apnea, 12 (30%) patients had moderate OSA and 7 (17.5%) patients had mild OSA.

P wave maximum was higher in OSA patients than controls 173.50 ± 5.61 m/s versus 145.00 ± 5.60 m/s, ($p$ value <0.05).

P wave minimum in OSA patients was 75.00 ± 5.30 m/s and in controls was 73.00 ± 5.85 m/s ($p$ value >0.05).

P wave dispersion in OSA patients was higher than control persons 98.50 ± 4.77 m/s versus 72.00 ± 3.37 m/s, ($p$ value <0.05) (Table 2).

There was a significant positive correlation between P wave dispersion and disease variables with $r$ values 0.43, 0.64 and 0.64, respectively (Table 3).

Among risk factors, multiple linear regression analysis showed that: systolic blood pressure and BMI are independently associated with Pd ($\beta = 0.56, p = 0.00$) ($\beta = 0.47, p = 0.05$) (Table 4).

### Table 2 P-wave measurements and level of OSA.

<table>
<thead>
<tr>
<th>P-wave</th>
<th>Level of OSA</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild (Mean ± SD)</td>
<td>Moderate (Mean ± SD)</td>
</tr>
<tr>
<td>P-wave minimum</td>
<td>88.57 ± 12.99</td>
<td>78.33 ± 10.86</td>
</tr>
<tr>
<td>P-wave maximum</td>
<td>154.29 ± 11.31</td>
<td>173.33 ± 10.25</td>
</tr>
<tr>
<td>P-wave dispersion</td>
<td>65.71 ± 8.41</td>
<td>95.00 ± 7.83</td>
</tr>
</tbody>
</table>

### Table 3 Correlation between P wave dispersion and disease variables.

<table>
<thead>
<tr>
<th>Disease variables</th>
<th>Correlation ($r$)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI</td>
<td>0.43</td>
<td>0.006</td>
</tr>
<tr>
<td>BMI</td>
<td>0.64</td>
<td>0.000</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>0.64</td>
<td>0.000</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>0.20</td>
<td>0.116</td>
</tr>
<tr>
<td>Age</td>
<td>0.18</td>
<td>0.17</td>
</tr>
</tbody>
</table>

AHI, apnea hyponea index; BMI, body mass index.

### Table 4 Multiple linear regression analysis (P-wave dispersion).

<table>
<thead>
<tr>
<th></th>
<th>Unstandardized coefficients</th>
<th>Standardized coefficients</th>
<th>T</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
<td></td>
</tr>
<tr>
<td>(Constant)</td>
<td>−56.488</td>
<td>38.542</td>
<td>−1.466</td>
<td>0.152</td>
</tr>
<tr>
<td>Age</td>
<td>−0.365</td>
<td>0.310</td>
<td>−0.128</td>
<td>−1.179</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>1.343</td>
<td>0.285</td>
<td>0.568</td>
<td>4.719</td>
</tr>
<tr>
<td>Diastolic BP</td>
<td>−0.718</td>
<td>0.360</td>
<td>−0.221</td>
<td>−1.993</td>
</tr>
<tr>
<td>AHI</td>
<td>0.180</td>
<td>0.133</td>
<td>0.167</td>
<td>1.362</td>
</tr>
<tr>
<td>BMI</td>
<td>1.690</td>
<td>0.814</td>
<td>0.272</td>
<td>2.077</td>
</tr>
</tbody>
</table>
Discussion

In our study, P wave maximum was higher in OSA patients than controls. Moreover, P wave dispersion was higher in severe and moderate than mild grade of obstructive sleep apnea. There was a significant positive correlation between P wave dispersion and AHI.

Factors related to OSA, such as repetitive hypoxemia, autonomic nervous system imbalance, systemic inflammation, fluctuations in intrathoracic hemodynamic, and diastolic dysfunction may lead to prolongation of intra-atrial and inter-atrial conduction time and provoke inhomogeneous propagation of sinus impulses reflected as increased P_{max} and P wave dispersion [5]. The Sleep Heart Health Study has reported a fourfold increase in the prevalence of atrial fibrillation in subjects with an AHI > 30 [10].

We found a significant positive correlation between P wave dispersion and body mass index. This is because obesity is considered a major risk factor for the development and progression of OSA [11]. Moreover, obesity may worsen OSA because of fat deposition at specific sites. Fat deposition in the tissues surrounding the upper airway appears to result in a smaller lumen and increased collapsibility of the upper airway predisposing to apnea [12,13].

There was a significant positive correlation between P wave dispersion and Systolic and diastolic blood pressure this may be explained by Left ventricular diastolic dysfunction in hypertensive patients which may result in an increase in left ventricular end-diastolic (LVED) pressure and in left atrial dimensions. The increase in left atrial dimensions as a result of rising intra-atrial pressure changes the geometry of atrial fibrils; this, in combination with inhomogeneous fibrosis of the left atrial wall, interrupts the conduction of sinus impulses, which induce P wave dispersion [14–17].

To conclude, Pd is increased and correlated with severity of OSA. Systolic blood pressure and BMI are independent risk factors for Pd. Follow up of patients to detect clinical implications is recommended.

Study limitations

The small number of patients and the manual measurement of P-wave instead of computer-assisted measurement are the main drawbacks of our study. Follow up of the patients in order to evaluate the significance of Pd may be a topic for future studies.

Conflict of interest

None declared.

References