VIA MEDICA
ORIGINAL ARTICLE
Cardiology Journal
2011, Vol. 18, No. 2, pp. 171–175

ISSN 1897–5593

# Interatrial block in patients with obstructive sleep apnea

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## Abstract

**Background:** *Obstructive sleep apnea (OSA) is a common disorder that affects 5% of the adult North American population. It is associated with atrial arrhythmias and stroke. The mechanisms of this association remain unclear. The aim to the study was to identify the factors associated with interatrial block (IAB) among patients with OSA.*

**Methods:** *Patients referred for polysomnography were studied. Sleep apnea severity (apnea-hypopnea index [AHI]) was measured in each subject. 12-lead ECGs were scanned and amplified (× 10); P-wave duration and dispersion were measured using a semi-automatic caliper. IAB was defined as a P-wave duration ≥ 120 ms.*

**Results:** *Data from 180 consecutive patients was examined. Moderate-severe OSA (mean AHI = 56.2 ± 27.9) was present in 144 (OSA group). The remaining 36 had mild or no OSA (mean AHI = 5.6 ± 3.6) and were used as controls. Age distribution between the groups did not differ and there were more males in the OSA group (69.4%* vs *47.2%, p = 0.01). Obesity (78.5%* vs *39.4%, p < 0.001) and hypertension (51.4%* vs *27.8%, p < 0.01) were more prevalent in the OSA group. IAB was more prevalent in patients with moderate-severe OSA (34.7% OSA* vs *0% controls, p < 0.001). In linear regression, age and AHI > 30 were independent predictors of maximum P-wave duration (p = 0.001 and p < 0.001, respectively). P-wave dispersion was significantly higher in the severe OSA group (14.6 ± 7.5 for OSA, 8.9 ± ± 3.1 controls, p < 0.001).*

**Conclusions:** *Older age and moderate-severe OSA are predictors of IAB. P-wave dispersion is increased in patients with moderate-severe OSA. This may partly explain the high prevalence of atrial arrhythmias in patients with OSA.* (Cardiol J 2011; 18, 2: 171–175)

**Key words: interatrial block, obstructive sleep apnea**

## Introduction

Obstructive sleep apnea syndrome (OSA) is a common breathing disorder that affects 5% of the North American adult population, with men being affected almost twice as much as women [1]. The condition has well defined associations with increased cardiovascular morbidity and mortality, arrhythmia, daytime hypersomnolence, motor vehicle accidents and neurocognitive dysfunction; but despite this it is grossly under diagnosed [2–6]. Atrial fibrillation (AF) is strongly associated with

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Received: 09.08.2010 Accepted: 07.10.2010

OSA [7]. In addition, patients with untreated OSA are at higher risk for AF recurrence after electrical cardioversion [8].

Interatrial block (IAB) is prevalent in the population at large, in particular in those over 65 years of age [9–12]. Abnormal atrial depolarization is referred to as IAB when the P-wave is ≥ 110 ms which signifies excessive time for sinus impulses to conduct from the right atrium (RA) to the left atrium (LA) [13]. LA conduction that results in (delayed) LA activation in IAB is thought to principally occur in the region of the atrioventricular node, where a caudocranial ('upward') deflection of the preceding RA-descending sinus impulses then occurs [14]. The conduction delay between the RA and LA is depicted on the electrocardiogram (ECG) as prolonged, often bifid ('notched') P-waves with distinctive RA and LA components [15–18].

Several pathophysiological mechanisms have been involved in the link between OSA and AF, such as increased sympathetic tone, hypertension, obesity (a co-morbidity that is frequent for both conditions) and increased pulmonary pressure [19, 20]. However, the prevalence of IAB, an accepted risk factor for AF, in patients with OSA, has only been reported in a small study [21].

Our hypothesis is that IAB is commoner among patients with OSA than in those with less severe, or no, OSA. We designed this study to determine the association between severity of OSA and P-wave prolongation, and to compare P-wave dispersion between patients with and without severe OSA.

## Methods

We analyzed 180 patients referred for overnight polysomnography study at the Sleep Laboratory at Kingston General Hospital. Demographics and the results of the polysomnographic study collected included: age, gender, obesity, systemic hypertension, coronary artery disease (CAD), diabetes mellitus (DM), heart failure, apnea/hypopnea index (AHI), and maximum oxygen desaturation. Apnea was defined as a cessation of the airflow with $O_2$ saturation reduction > 4%. Consent was not required as the analysis was done retrospectively. We obtained Internal Review Board approval from our institution.

### Polysomnography

Standard overnight polysomnography was performed on all patients. This included four EEG channels (two central: C3–A2, C4–A1, and two occipital: O2–A1, O1–A2), two EOG channels, submental EMG, finger pulse oximetry, modified lead II ECG, thoracic and abdominal movement (piezoelectric bands), right and left anterior tibialis EMG, diaphragmatic surface EMG, and snore vibration sensor. Airflow was measured with both a nasal cannula pressure transducer and oral thermistor. The recording duration was seven hours or the patient's usual time in bed. OSA was considered moderate-severe when AHI ≥ 25 episodes per hour, and considered not present when AHI was < 5 episodes per hour.

### ECG measurements

The 12-lead ECGs were recorded at 25.0 mm/s, 10 mm/mV and 150 Hz; and scanned at 300 DPI. For measuring intervals we used a semi-automatic caliper (Iconico, New York, USA) amplifying the ECG ten times. ECGs were measured by two investigators. In case of disagreement, the ECG was blinded and interpreted by an expert electrophysiologist (AB, DR, CS). The onset of the P-wave was defined as the point of initial upward or downward deflection calculated from baseline. The P-wave offset was the returning point of the waveform to baseline.

IAB was defined as a P-wave duration ≥ 120 ms to facilitate ECG measurement. P-wave dispersion was calculated as max. P-wave–min. P-wave. Patients with implantable devices and without readable ECGs were excluded from the analysis.

### Statistical analysis

Data was entered into an Excel spreadsheet designed for this study and imported into SPSS Version 17.0 for Windows (2008) for statistical analysis. Frequencies and proportions were generated for all categorical data, and means, medians and standard deviations were calculated for the continuous data. These descriptive analyses provided the prevalence of IAB in the sample. Demographic and clinical characteristics of those with moderate-severe OSA were compared to those with no OSA using $\chi^2$ tests (categorical data) and t-tests (continuous data). The relationship between maximum P-wave duration and age was assessed by means of Pearson correlation. Linear regression modeling was used to assess the association between OSA status and maximum P-wave duration while controlling for age.

## Results

Demographic and clinical characteristics, and results of the polysomnography, are shown in Table 1. Moderate-severe OSA (mean AHI 56.2 ± ± 27.9) was present in 144 patients (OSA group).

**Table 1.** Demographic and clinical characteristics and polysomnography results.

| | Moderate-severe obstructive sleep apnea (AHI > 25) | Control group | P |
|---|---|---|---|
| Age (years) | 56.7 ± 12.6 | 56.4 ± 12.4 | 0.90 |
| Gender (male, %) | 69.4 | 47.2 | 0.01 |
| Hypertension (%) | 51.4 | 27.8 | < 0.01 |
| Obesity (%) | 78.5 | 39.4 | < 0.001 |
| Heart failure (%) | 17.6 | 5.6 | 0.12 |
| Coronary artery disease (%) | 30.1 | 16.7 | 0.11 |
| Diabetes mellitus (%) | 30.8 | 22.2 | 0.31 |
| Left atrium dimension [mm] | 40.8 ± 7 | 34.6 ± 4.8 | 0.038 |
| Apnea/hypopnea index (AHI) | 56.2 ± 27.9 | 5.6 ± 3.6 | < 0.001 |
| Maximum desaturation (%) | 79.8 | 88.4 | < 0.001 |

**Table 2.** Interatrial block and P-wave dispersion in patients with and without obstructive sleep apnea (OSA).

| | Moderate-severe OSA (AHI > 25) | Control group | P |
|---|---|---|---|
| Interatrial block (%) | 34.7 | 0 | < 0.001 |
| P-wave dispersion [ms] | 14.6 ± 7.5 | 8.9 ± 3.1 | < 0.001 |

The remaining 36 patients had mild or no OSA (mean AHI = 5.6 ± 3.6) and were used as controls (control group). No patients were taking antiarrhythmic drugs. Age distribution between the two groups did not differ (56.7 ± 12.6 years for OSA, 56.4 ± 12.4 years for controls, p = 0.9), and there were more males in the OSA group (69.4% *vs* 47.2%, p = 0.01). Obesity and hypertension were more prevalent in the OSA group (78.5% *vs* 39.4%, p < 0.001 and 51.4% *vs* 27.8%, p < 0.01, respectively). CAD, DM and heart failure tended to be more prevalent in patients with OSA also, but the differences did not reach statistical significance. LA dimensions measured by two-dimensional echocardiogram (antero-posterior) showed a larger diameter in the OSA group (40.8 ± 7 mm *vs* 34.6 ± 4.8 mm; p = 0.03).

The prevalence of IAB in patients with and without moderate-severe OSA can be seen in Table 2.

IAB was more prevalent in patients with moderate-severe OSA (34.7% OSA *vs* 0% controls, p < 0.001). In linear regression, age and AHI > 25 were independent predictors of max. P-wave duration (p = 0.001 and p < 0.001, respectively). CAD, arterial hypertension, obesity and minimum saturation did not predict IAB in the linear regression model. The P-wave was prolonged by 1.8 ms for each decade of age, and by 7.6 ms for the OSA group compared to the control group. Max. P-wave duration correlated with the severity of OSA (Fig. 1, r = = 0.195, p = 0.009). P-wave dispersion was significantly higher in the moderate-severe OSA group (14.6 ± 7.5 *vs* 8.9 ± 3.1, p < 0.001).

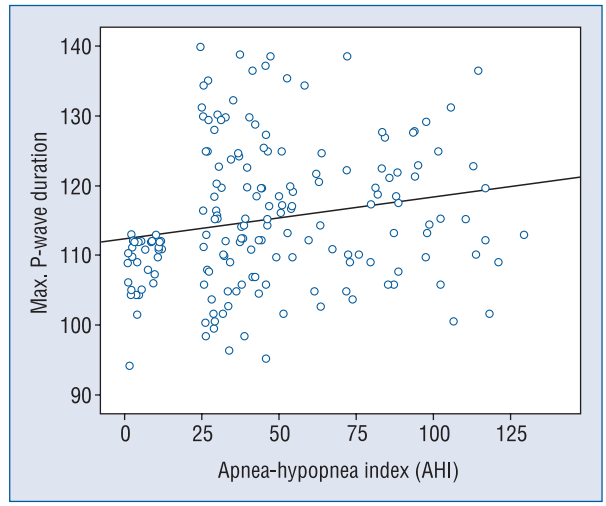


**Figure 1.** Correlation between obstructive sleep apnea and max. P-wave duration. Correlation coefficient is 0.195, p = 0.009.

## Discussion

The main findings of our study were that IAB (clinically defined as P-wave duration > 120 ms) was highly prevalent in an unselected population with moderate-severe OSA; that IAB was significantly more frequent in patients with moderate-severe OSA than in patients with no or mild OSA; and that P-wave dispersion was significantly increased in patients with moderate-severe OSA. The severity of OSA, determined by an AHI > 25, correlated with the longer P-wave duration. This may indicate the presence of atrial electrical remodeling in patients with moderate-severe OSA. However, one should be cautious in interpreting these results because when the AHI is between 0 and 15, all P-wave durations were normal (below 120 ms), and in patients with an AHI > than 25, about one third presented a P-wave longer than 120 ms. Whereas the correlation coefficient between OSA and P-wave duration may statistically be correct, in view of the large variations in P-wave duration, in individual cases the correlation could be clinically less relevant.

A previous study [21] found similar results in a smaller population and using manual calipers. In that study, the OSA was less severe than in our study, but still showed that patients with AHI > 30 had an increased P-wave duration and dispersion in comparison to no OSA. Surprisingly, in the Can et al. study [21] the LA dimension between the three populations (mild, moderate and severe OSA) did not differ; indicating that the electrical atrial remodeling could be an independent phenomenon not necessarily related to structural atrial remodeling. Our findings showed that the LA dimension was increased in the group with moderate-severe OSA, making the presumption of electrical atrial remodeling as an independent phenomenon less likely.

The association between OSA and AF has been previously reported. A sub-study of the Sleep Health Heart Health Study [22] showed a four-fold increased AF prevalence in patients with severe OSA using the ECG recordings of the polysomnography, which could underestimate the 'true' prevalence of AF in this population [23]. Gami et al. [7] demonstrated, using a validated tool for the screening of OSA (Berlin Questionnaire), a strong association between AF and OSA. Our group showed that the presence of intermittent interatrial block after a successful cardioversion could be a predictor of early AF recurrence [24]. Despite this well-demonstrated association, the mechanisms that link the two conditions remain somewhat unclear [25, 26]. However, some observations can be made. OSA produces an autonomic nervous system imbalance with increased sympathetic tone [19, 20]. Systemic and pulmonary hypertension are common and both may impact on atrial stretching, inducing structural atrial remodeling and loss of normal atrial architecture [27]. In addition, intermittent chronic hypoxemia, hypercapnia and increased plasmatic catecholamine levels may represent a direct proarrhythmogenic injury to the atrial tissue.

Increased P-wave duration in the surface ECG may be a manifestation of atrial electrical remodeling. In our study, P-wave duration in patients with moderate-severe OSA was significantly longer than in patients with no OSA. Additionally, the severity of the OSA correlated positively with a prolongation of the P-wave duration. AHI > 25 and older age were independent predictors of maximum P-wave duration.

Not surprisingly, P-wave dispersion was also increased in patients with moderate-severe OSA.

Is the atrial electrical remodeling induced by OSA the explanation for the frequent association between OSA and AF?

Both IAB and increased P-wave dispersion have been found to be predictors of AF in different clinical scenarios than OSA [10, 17, 18]. It is possible that OSA-related IAB could be a manifestation of atrial electrical remodeling that facilitates AF development, maintenance and/or recurrence.

### Limitations of the study

Although IAB is considered a risk factor for AF, the lack of follow-up in this study does not allow evaluating the clinical relevance of increased P-wave dispersion and, specifically, whether the presence of IAB confers a higher risk of developing AF in this population.

## Conclusions

Interatrial block is significantly more prevalent in patients with moderate-severe OSA than in controls. Age and severity of OSA are independent predictors of maximum P-wave duration. P-wave dispersion is increased in patients with moderate--severe OSA. Further studies are needed to confirm whether IAB in patients with OSA is associated with AF development or recurrence.

## Acknowledgements

The authors have no disclosure or conflict of interest to declare.

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