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Atrial Electrical Remodeling and Sleep Disordered Breathing

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To the Editor

We read with interest the article from Bitter et al.[1] published in the last volume of JAFIB.

Introduction

This non-systematic review covers some of the most important physiopathological aspects of the link between sleep disordered breathing (SDB) and atrial fibrillation (AFib). We do agree with the authors on the role of hypertension, endothelial dysfunction and inflammation. These topics were, to our understanding and perspective, very well covered by the authors on this review.

However, despite that the authors mentioned atrial remodeling a couple of times during their review, we are not sure that this topic and specifically atrial electrical remodeling, was properly discussed and referenced.

The pathophysiology linking SDB to AF is multifactorial and may involve repetitive hypoxemia, increased sympathetic drive, fluctuations in intrathoracic pressure and systemic inflammation.[2] These physiologic changes may induce structural and electrical remodeling serving as a substrate to the development of AFib.

An indirect marker for such electrical remodeling is the prolongation of atrial conduction time, represented by increased maximum P-wave duration in the surface ECG. In a prior study, we showed that an increased P-wave duration has been associated with SDB.[3]Interatrial block (IAB), defined as a surface P-wave duration > 120 ms, was more prevalent in patients with moderate-severe SDB (34.7% SDB vs. 0% controls, p <0.001). P-wave dispersion, a measurement that was linked to development of new AFib, was also increased in patients with SDB (14.6±7.5 vs. 8.9±3.1, p<0.001). In linear regression, age and AHI (apnea/hypopnea index) > 25 were independent predictors of maximum P-wave duration (p=0.001 and p<0.001 respectively).[3]

Another non-invasive method to determine atrial electrical remodeling is the Signal-averaged P-wave (SAPW) duration. The SAPW duration represents the average of all P-wave durations in a given number of consecutive heartbeats. We recently postulated that SAPW would be useful to identify atrial electrical remodeling in patients with severe SDB and that treatment with C-PAP for 4-6 weeks may induce reverse atrial electrical remodeling.[4] The results of this study have shown that patients with severe SDB have a longer SAPW duration than controls (131.9 ± 10.4 vs 122.8 ± 10.5 ms; p = 0.04) and that a significant reduction of the SAPW duration occurs after treatment with C-PAP (131.9 ± 10.4 to 126.2 ± 8.8 ms; p < 0.001) (Figure 1).[4]

The shortening of SAPW duration and surface P-wave duration represents more rapid inter-atrial conduction and provides evidence for reverse atrial electrical remodeling. This may indicate an additional benefit of treating patients with C-PAP, as this evidence suggests that C-PAP may improve the anatomical and electrical substrate for AFib. Reverse atrial electrical remodeling is a concept in evolution, and several cardiovascular treatments may improve atrial dynamics.[5] It is of utmost importance, in the times of considering AFib ablation as first line therapy for recurrent AFib; to be familiarized with the impact of non-recognized/non-treated SDBs over the cardiac electrical system. Conventional treatment for SDB as C-PAP may also represent a benefit in terms of facilitating normal atrial conduction and reducing the risks associated with AFib.

Key Words:
Sleep disordered breathing; atrial fibrillation; atrial electrical remodelling

Disclosures:
None.

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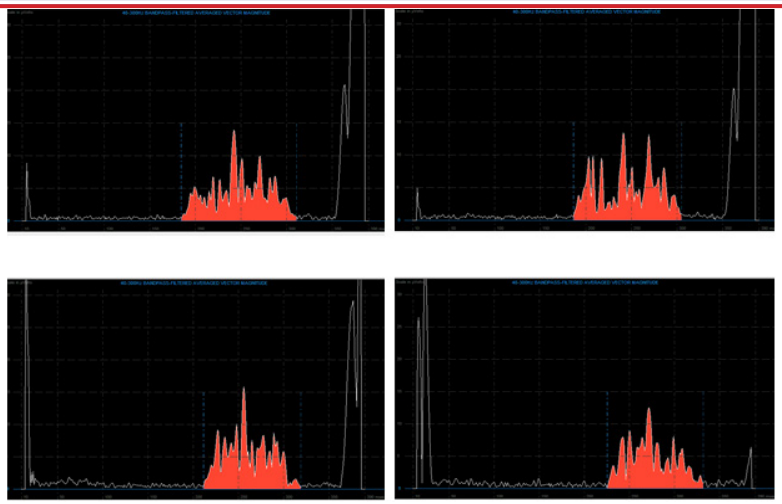

Figure 1: **Sample SAPW analyses for a SDB subject (upper panels) and a control subject (lower panels), at first (left) and second (right) recording. Note: x-axis = msec; dotted blue lines indicate duration; y-axis = µvolts. The area in red represents the average p wave of 100 beats. With permission, reference #4.**

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