QRS Complex Patterns in Patients with Obstructive Sleep Apnea

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Abstract. *We evaluated QRS complex morphology in patients with obstructive sleep apnea (OSA) that could be potentially linked to arrhythmias. The study population consisted of 199 consecutive patients examined in the sleep laboratory, divided into quartiles according to the apnea/hypopnea index (AHI):Group Q1: AHI 0.7-10.9 #/h; Group Q2: AHI 11.0-32.3 #/h; Group Q3: AHI 32.3-63.0 #/h; Group Q4: AHI 65.1-133 #/h. Resting 12-lead ECG was recorded, the QRS parameters analyzed included QRS amplitude in individual leads, QRS spatial vector magnitude (QRSmax), electrical axis (EA), ECG criteria for left ventricular hypertrophy (ECG-LVH) and right ventricular hypertrophy (ECG-RVH). There were no significant differences in QRSmax values between the groups. The occurrence of ECG-LVH was low (total 7%), on the other hand 96 % of patients showed signs of ECG-RVH. The values of EA were significantly shifted gradually to the left (Q1: 40.1±19.8°; Q2: 34.5±18.0°; Q3: 27.6±15.3°; Q4: 31.6±16.2°). Additionally, QRS morphology showed a variety of intraventricular conduction defects, including QRS notching, non-specific conduction defects and patterns of left/ or right bundle branch blocks. Conclusions: The OSA patients displayed significant changes in QRS complex morphology suggestive of depolarization sequence deterioration indicative of considerable electrical remodeling that could be potentially linked to occurrence of arrhythmias.*

Keywords: sleep apnea, QRS complex, right ventricular hypertrophy, left ventricular hypertrophy, right bundle branch block

1. Introduction

Obstructive sleep apnea (OSA) is a clinical condition characterized by repeated disruptions of breathing during sleep, leading to repetitive episodes of hypoxia and reoxygenation. OSA is associated with multiple disorders and co-morbidities, such as obesity, hypertension, insulin resistance/ metabolic syndrome, chronic obstructive pulmonary disease [1]. These clinical conditions themselves result in a complex impairment of myocardium that is reflected in the QRS morphology changes as a result of altered sequence of depolarization. The increased occurrence of arrhythmias and the prolongation of QRS complex have been described previously [2, 3].

In this study we analyzed the morphology of the QRS complex in relation to the degree of apnea/ hypopnea index (AHI) as a measure of intermittently reduced oxygen supply. We were interested if hypoxia has additional effect on the sequence of depolarization observed in the given cardiac pathology.

2. Subject and Methods

Study population
The study subjects were recruited within the Project APVV- 0134 -11 Effects of Hypoxia on Molecular Pathways related to Increased Cardiovascular Risk in Patients with Sleep Apnea and their Reversal by Therapy (HICART).

The total number of 199 OSA patients was divided into quartiles according to the apnea/hypopnea index:

- Group Q1 (n = 50): AHI 0.7-10.9 #/h, aged 24 to 80 years, mean 50.9 years;
- Group Q2 (n = 50): AHI 11.0-32.3 #/h; aged 23 to 80 years, mean 51.3 years;
- Group Q3 (n = 50): AHI 32.3-63.0 #/h, aged 29 to 78 years, mean 57.9 years;
- Group Q4 (n = 49): AHI 65.1-133.8 #/h, aged 31 to 70 years, mean 54.8 years.

The basic characteristics of the study population are presented in Table 1.

Table 1. Baseline characteristics of the study population.

	n	Age yrs mean SD	Gender M/F (%)	sBP mmHg mean SD	dBP mmHg mean SD	Hypertension n (%)	BMI kg/m^2 mean SD
Q1	50	50.9 11.9	22/28 (44/56)	121.9 16.1	79.5 9.6	26 (52)	28.7 4.8
Q2	50	51.3 12.3	34/17 (66/34)	126.2 16.6	82.5 10.6	26 (52)	30.3 5.0
Q3	50	57.9 9.8	40/10 (80/20)	137.3 16.5	86.7 9.2	37 (74)	32.8 4.1
Q4	49	54.8 12.8	43/6 (87/13)	139.6 24.2	88.2 14.8	42 (84)	38.8 6.1

All participants underwent diagnostic overnight polysomnography (Alice 4, Respironics Inc., Murrysville, Pennsylvania, USA). All records were scored manually following the AASM 2007 rules [4]. Apnea was identified as a drop in airflow of >90% from the baseline excursion for ≥10 seconds; hypopnea was defined as a reduction in airflow of at least 50% of baseline for ≥10 seconds accompanied either by a decrease in hemoglobin saturation for ≥3%, an EEG-recorded arousal, or both. The apnea-hypopnea index (AHI) was defined as the number of apnea and hypopnea episodes per hour of sleep.

The standard 12-lead ECG was recorded using a Marquette Centra electrocardiograph. The amplitudes of the QRS waves in individual leads were measured manually.

The following ECG parameters were analyzed:

- The maximum spatial vector magnitude (QRSmax) estimated as:

$$QRS_{max} = \sqrt{V2^2 + aVF^2 + V5^2} \quad (1)$$

where V2 the maximum QRS deflection in lead V2
aVF the maximum QRS deflection in lead aVF
V5 the maximum QRS deflection in lead V5.

- The electrical QRS axis (EA) was calculated using the formula:

$$EA = arc\tan\left(\frac{2*aVF}{I+\sqrt{3}}\right) \quad (2)$$

where aVF the maximum QRS deflection in lead aVF
I the maximum QRS deflection in lead I.

ECG-LVH criteria:

- The Sokolow-Lyon index calculated as a sum of V1 and RV5 or V6;
- The Cornell voltage calculated as a sum of RaVL and SV3;
- The Gubner criterion, calculated as a sum of RI and SIII;

ECG-RVH criteria:

- The Sokolow-Lyon criterion for right ventricular hypertrophy, calculated as a sum of RV1 and SV5(or V6);
- Butler-Leggett formula, calculated as sum of R (or R') V1 (or V2) and S I (or V6) minus SV1.

The occurrence of complete or incomplete RBBB with and without LAD was also evaluated.

3. Results

The values of QRSmax were low, and there were no significant differences in QRSmax values between the groups. The values of EA were significantly shifted gradually to the left with increasing values of AHI (Figure 1).

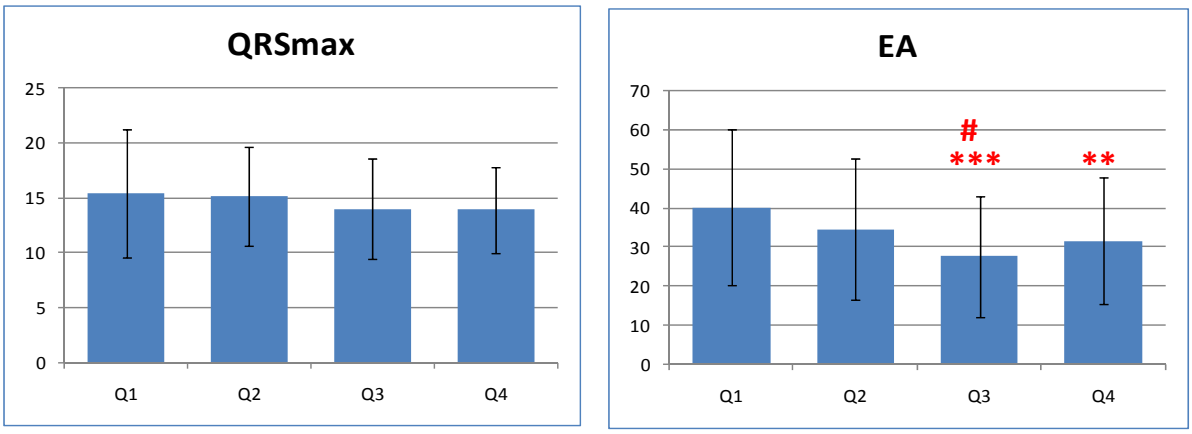

Figure 1. **Values of maximum** QRS spatial vector magnitude and electrical axis. Values presented as means, the error bars represent standard deviations. **: p<0.01 with respect to Q1, ***: p<0.001 with respect to Q1. #: p<0.05 between Q2 and Q3.

The occurrence of ECG-LVH was low (total 7%). On the other hand, nearly all patients (96%) showed signs of ECG-RVH by BL criteria and total 17% by SLI-RVH criteria with maximum (7%) in the Q4 group. The values of ECG criteria for right ventricular hypertrophy are presented in Table 2.

Table 2. Values of ECG criteria for right ventricular hypertrophy. SL-RVH: Sokolow-Lyon criterion for right ventricular hypertrophy, BL: Butler-Leggett formula. *Significant as compared to Q1 and Q3.

	SL [mm] mean (SD)	BL [mm] mean (SD)
Q1	7.3 (3.8)	13.4 (4.0)
Q2	8.0 (3.9)	14.2 (4.9)
Q3	7.5 (3.7)	13.3 (4.3)
Q4	9.2 (3.9)*	14.0 (5.2)

The QRS patterns of complete or incomplete RBBB occurred in 12.5% of patients (in combination with left axis deviation 10.5%), there were no significant differences among the groups.

4. Discussion

Regarding the QRS patterns, we observed low QRSmax values in all quartiles, the ECG-LVH criteria did not exceed partition values. On the other hand, the ECG-RVH signs and a slight but significant shift of electrical axis to the left were present, pronounced in quartiles with increasing AHI.

The low QRS values are traditionally attributed to the effect of obesity; however there were no differences between the groups in spite of significantly increased BMI values in higher quartiles. Although a great proportion of patients were hypertensive (in Q4 group 84%), none of the patients had ECG-LVH signs. As mentioned above, the absence of increased QRS voltage is attributed to the effect of obesity. However, the effect of obesity did not affect the ECG-RVH criteria – it cannot be assumed that the effect of obesity works selectively for ECG-LVH criteria.

ECG-RVH signs were present in almost all patients. Theoretically it could be attributed to pulmonary hypertension and/or concomitant chronic obstructive pulmonary disease. However, we observed a gradual shift of the electrical axis to the left and not to the right as it would be expected in RVH.

5. Conclusions

Obstructive sleep apnea represents a clinical condition with a variety of comorbidities, such as systemic and pulmonary hypertension, obesity, metabolic syndrome, chronic obstructive pulmonary disease, etc., each of them with possible considerable impairment of myocardium and an additional effect of intermittent hypoxia. We observed non-typical QRS changes that are suspected of intraventricular conduction defects. This assumption is consistent with the published evidence of cardiac arrhythmias and conduction disturbances documented in OSA patients.

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