

# Relation Between Obstructive Sleep Apnea and Duration of QRS Complex

Fatih Aygün<sup>1</sup>, Harun Karamanlı<sup>2</sup>

<sup>1</sup> Department of Cardiovascular Surgery, Başkent University, Konya Medical and Research Center, Konya, Turkey

<sup>2</sup> Department of Pulmonology, Faculty of Medicine Mevlana University, Konya, Turkey



## ABSTRACT

**Introduction:** To investigate the relation between severity of obstructive sleep apnea (OSA) and widened QRS complex on electrocardiogram.

**Patients and Methods:** The present study comprised 85 subjects diagnosed with OSA at our clinic between March 2013 and September 2014. PSG reports were reviewed by a self-proclaimed expert physician. Whilst the patients with severe sleep apnea disease were assigned to Group 1, patients with mild-moderate sleep apnea disease were assigned to Group 2.

**Results:** The age of the patients participated in the study ranged between 20 years (y) (minimum-min) and 85 years (maximum-max) (mean  $\pm$  standard deviation  $48.5 \pm 13.2$  y). Of these subjects, 51 (60%) were male and 34 (40%) were female. Mean duration of QRS complex was  $0.112 \pm 0.016$  seconds (sec) in Group I consisted of patients with severe OSA of which Polysomnography (PSG) records and scoring were done using ALICE 6 system, whereas it was  $0.080 \pm 0.017$  sec in Group 2.

**Conclusion:** It was observed that duration of QRS complex was longer in the patients with severe OSA as compared to those with mild-moderate OSA. The result was statistically significant ( $p < 0.01$ ).

**Key Words:** Monitoring sleep; electrocardiogram; obstructive sleep apnea

## Obstrüktif Uyku Apne Şiddeti ile QRS Süresi Arasındaki İlişkinin Araştırılması

### ÖZET

**Giriş:** Bu çalışmada, obstrüktif uyku apnesi (OSA) şiddeti ile elektrokardiyogramdaki QRS süresinin genişlemesi arasındaki ilişki araştırılmıştır.

**Hastalar ve Yöntem:** Bu çalışma; kliniğimizde Mart 2013 ve Eylül 2014 tarihleri arasında, OSA tanısı almış 85 kişiyi içermektedir. PSG raporları alanında uzman hekim tarafından değerlendirildi. Şiddetli uyku apne hastalığı olan bireyler Grup 1 olarak adlandırılırken, orta, hafif uyku apne hastalığı olan bireyler Grup 2 olarak belirlendi.

**Bulgular:** Çalışmaya katılan tüm bireylerin yaş dağılımı; minimum(min) 20 yıl (y), maksimum(max) 85 yıldır (ortalama  $\pm$  standart sapma  $48.5 \pm 13.2$  y). Bu kişilerin 51 (%60)'i erkek, 34 (%40)'ü kadındır. Polisomnografi (PSG) kayıtları ve skorlaması ALICE 6 system kullanılarak oluşturulan, şiddetli OSA'sı olan bireyleri içeren Grup 1'de QRS süresi  $0.112 \pm 0.016$  saniye iken Grup 2' de  $0,080 \pm 0,017$  saniye olduğu görüldü.

**Sonuç:** Şiddetli OSA'sı olan bireylerin, hafif ve orta şiddette OSA'sı olan bireylere göre QRS sürelerinin yüksek olduğu görüldü. Sonucun istatistiksel olarak anlamlı olduğu tespit edildi ( $p < 0.01$ ).

**Anahtar Kelimeler:** Uyku monitorizasyonu; elektrokardiyogram; obstrüktif uyku apnesi

## INTRODUCTION

Obstructive sleep apnea (OSA) is frequently encountered among obese people. It is defined as recurrent closure of the upper airway while respiratory effort is ongoing during sleep. Apnea leads to decrease in negative intrathoracic pressure and in arterial oxygen saturation. Cardiac arrhythmias and conduction alterations are common in the people with OSA<sup>(1-9)</sup>.

OSA is a breathing disorder during sleep with a prevalence rate of 10-15% in males and 4-7% in females<sup>(10)</sup>. OSA involves many systems, particularly cardiovascular system. Although the association of OSA with cardiovascular events such as hypertension, congestive heart failure and sudden death is known, etiopathological mechanism of the association with many of these remains unclear<sup>(11-15)</sup>.

### Yazışma Adresi

Fatih Aygün

E-posta: fatihaygun@ttmail.com

Geliş Tarihi: 21.08.2014

Kabul Tarihi: 04.11.2014

@ Copyright 2015 by Koşuyolu Heart Journal.  
Available on-line at  
www.kosuyolukalpdergisi.com

Many studies have demonstrated association between OSA and widened QRS complex on electrocardiogram (ECG)<sup>(16-20)</sup>. There are studies suggesting that duration of QRS complex is an independent predictor of sudden death in case that OSA is accompanied by hypertension or heart failure<sup>(21-23)</sup>.

The present study investigated the relation between severity of OSA and widened QRS complex.

## PATIENTS and METHODS

### Clinical Characteristics of Patients

The study comprises 85 subjects diagnosed with OSA at our clinic between March 2013 and September 2014. Data were retrospectively collected and approval of the ethical committee was obtained.

All the subjects were investigated in terms of age, gender, smoking status, hypertension, diabetes mellitus, hyperlipidemia, family history of coronary artery disease, congestive heart failure and atrial fibrillation.

Height (human bascule, NAN TARTI Co, Turkey) and weight (TANITA Body Composition Analyzer, TANITA Corporation, Japan) of the participants were measured, and their body mass indexes (BMI) were calculated. A BMI lower than 25 kilograms (kg)/square meter (m<sup>2</sup>) (BMI < 25 kg/m<sup>2</sup>) was considered normal weight, between 25 kg/m<sup>2</sup> and 30 kg/m<sup>2</sup> (25 kg/m<sup>2</sup> ≤ BMI < 30 kg/m<sup>2</sup>) was considered overweight, and 30 kg/m<sup>2</sup> and over (30 kg/m<sup>2</sup> ≤ BMI) was considered obese.

### Electrocardiographic Evaluation

Resting ECG was obtained from electronic recordings. Heart rate and duration of QRS complex were obtained from computed ECG monitoring (ALICE 6 Analysis program, PHILIPS). Duration of QRS complex during sleep was measured in seconds (sec).

### Polysomnographic Evaluation

At baseline, all subjects were evaluated by a self-proclaimed expert physician in terms of sleep apnea disease. Patients that were considered available for polysomnography (PSG) were referred to the sleep laboratory. Four-channel EEG (C3-A2, C4-A1, O1-A2, O1-A1), two-channel electrooculography, electromyogram of the mandible and the left and right tibialis anterior, body position, oro-nasal air flow, thoracic and abdominal respiratory movements, ECG, respiratory sounds, oxygen saturation, continuous positive airway pressure (CPAP) and synchronous video were recorded as the parameters. Above-mentioned parameters, as well as PSG, were measured in each subject during night sleep. Records were evaluated according to the 2007 International Classification of Sleep Disorders. PSG recording and scoring were done using ALICE 6 system. OSA was considered moderate-mild if apnea-hypopnea index was between 5 and 30 and severe if apnea-hypopnea index was higher than 30.

### Groups

The study comprised a total of 85 subjects. PSG reports were evaluated by a self-proclaimed expert physician. Whilst the subjects with severe sleep apnea disease were assigned to Group 1, the subjects with moderate-mild sleep apnea disease were assigned to Group 2.

### Statistical Analysis

Statistical analysis were done using SPSS program (SPSS Inc., Chicago, IL, USA). Statistical significance of nonparametric data between the groups was analyzed by Chi-Square Test and Fisher's Exact Test (because observed values were below the expected values). Whilst the parametric data were represented as minimum, maximum and mean ± standard deviation, statistical significance of parametric data between the groups was analyzed by independent student t-test. The result was considered statistically significant if two-tailed p value was smaller than 0.05 (p < 0.05) (Table 1).

## RESULTS

### Subject Characteristics

Age distribution of all study participants ranged between 20 years (y) (minimum-min) and 85 years (maximum-max) (mean ± standard deviation 48.5 ± 13.2 y). Of these subjects, 51 (60%) were male and 34 (40%) were female. The number of smokers was 37 (43.5%) and there were 7 (8.2%) subjects with diabetes mellitus type 2, 15 (17.6%) subjects with dyslipidemia and 27 (31.8%) subjects with hypertension (HT). With regard to BMI, there were 11 (12.9%) normal weight subjects, 40 (47.1%) overweight subjects and 34 (40%) obese subjects. Eleven (12.9%) patients had coronary artery disease, no (0%) patient had congestive heart failure and no (0%) patient had atrial fibrillation. Duration of QRS complex was between 0.053 sec (min) and max 0.132 sec (mean ± standard deviation 0.093 ± 0.023 sec).

### Group Characteristics

In the males of Group I, mean duration of QRS complex was 0.111 ± 0.016 sec and mean age was 47.7 ± 14.2 y. There were 15 smokers (57.7%), 9 obese subjects (34.6%), 3 (11.5%) patients with diabetes mellitus, 4 (15.4%) patients with dyslipidemia, 5 (19.2%) patients with hypertension, 4 (15.4%) patients with coronary artery disease, no (0%) patient with congestive heart failure, and no (0%) patient with atrial fibrillation.

In the females of Group I, mean duration of QRS complex was 0.114 ± 0.016 sec and mean age was 57 ± 7.7 y. There was 3 (30%) smoker, 7 (70%) obese subjects, 1 (10%) subject with diabetes mellitus, 3 (30%) subjects with dyslipidemia, 4 (40%) subjects with hypertension, 1 (10%) subject with coronary artery disease, no (0%) subject with congestive heart failure, and no (0%) subject with atrial fibrillation.

In the males of Group 2, mean duration of QRS complex was 0.079 ± 0.018 sec and mean age was 45.6 ± 13.7 y. There were

**Table 1. Demographical characteristics of patients**

	Group 1 (n= 36)	Group 2 (n= 49)	p value
Age ( $\pm$ SD)	50.3 $\pm$ 13.3	47.2 $\pm$ 13.1	0.291 <sup>T</sup>
Gender (male)	26 (72.2%)	25 (51%)	0.049 <sup>C</sup>
Smoking	18 (50%)	19 (72.2%)	0.302 <sup>C</sup>
Body weight			
Normal	0 (0%)	11 (22.4%)	
Over weight	20 (55.6%)	20 (40.8%)	*0.473 <sup>C</sup>
Obesity	*16 (44.4%)	*18 (36.7%)	
BMI	30.3 $\pm$ 3.6	29.2 $\pm$ 5.4	0.299
Diabetes mellitus type 2	4 (11.1%)	3 (6.1%)	0.450 <sup>F</sup>
Dyslipidemia	7 (19.4%)	8 (16.3%)	0.709 <sup>C</sup>
Hypertension	9 (25%)	18 (36.7%)	0.251 <sup>C</sup>
Coronary artery disease	5 (13.9%)	6 (12.2%)	0.823 <sup>C</sup>
Congestive heart failure	0 (0%)	0 (0%)	-
Atrial fibrillation	0 (0%)	0 (0%)	-
QRS complex (sec)	0.112 $\pm$ 0.016	0.080 $\pm$ 0.017	<0.001 <sup>T</sup>

\* P value was calculated according to obesity

<sup>T</sup>: P value was presented as a result of Student t-test

<sup>C</sup>: P value was presented as a result of Chi-Square Test

<sup>F</sup>: P value was presented as a result of Fisher's Exact Test

BMI: Body Mass Index, SD: Standart deviation, Sec: Second

13 (52%) smokers, 8 (32%) obese subjects, 2 (8%) patients with diabetes mellitus, 4 (16%) patients with dyslipidemia, 8 (32%) patients with hypertension, 4 (16%) patient with coronary artery disease, no (0%) patient with congestive heart failure, and no (0%) patient with atrial fibrillation.

In the females of Group 2, mean duration of QRS complex was 0.080  $\pm$  0.016 sec and mean age was 48.8  $\pm$  12.5 y. There were 6 (25%) smokers, 10 (41.7%) obese subjects, 1 (4.2%) patient with diabetes mellitus, 4 (16.7%) patients with dyslipidemia, 10 (41.7) patients with hypertension, 2 (8.3%) patients with coronary artery disease, no (0%) patient with congestive heart failure, and no (0%) patient with atrial fibrillation.

Areas, in which duration of QRS complex on ECG were accumulated according to age and BMI, are illustrated in Figure 1 and 2.

## DISCUSSION

In the present study, duration of QRS complex was found longer in Group 1 consisted of patients with severe OAS as compared to Group 2 consisted of patients with moderate-mild OAS. The difference between Group 1 and Group 2 was found statistically significant ( $p < 0.05$ ). Statistical data between the groups are demonstrated in Table 1.

Obstructive sleep apnea (OSA) is called as recurrent closure of upper airway while respiratory effort is ongoing during sleep. Apnea is accompanied by decrease in negative

intrathoracic pressure and arterial oxygen saturation. Cardiac arrhythmias and alterations in cardiac conduction are frequently encountered in the subjects with OSA<sup>(1-9)</sup>.

OSA accompanied by widened QRS complex is seen in the presence of certain cardiologic conditions including hypertension, congestive heart failure, ventricular arrhythmia, and enhanced left ventricle mass<sup>(1,12,13)</sup>. In OSA, breathing occurs against closed upper airway. It is characterized by increased negative intrathoracic pressure, which results in increased left ventricle transmural pressure and afterload<sup>(24)</sup>.

Drager et al. stated that OSA alters cardiac structure and enhances left ventricle mass<sup>(16)</sup>. Many studies appear to support this statement<sup>(17-19)</sup>. Enhanced left ventricle mass leads to electrocardiographic alterations. Normal duration of QRS complex is between 0.04 and 0.08 sec. Ott et al. expressed that a QRS complex of 120 milliseconds (msec) and longer is an independent risk factor for sudden death<sup>(22)</sup>.

Many studies have demonstrated that OSA is associated with left ventricle hypertrophy, which is characterized by widened QRS complex on ECG<sup>(16-20)</sup>. There are studies revealing that duration of QRS complex is an independent predictor of sudden death in case OSA is accompanied by hypertension or heart failure<sup>(21-23)</sup>.

It has been found that a QRS complex longer than 120 msec is independent predictor of mortality and sudden death<sup>(21,22)</sup>. In the present study, although mean duration of QRS complex was shorter than 120 msec (0.111  $\pm$  .016 sec) in Group 1 consisted of patients with severe OSA, it was higher than mean

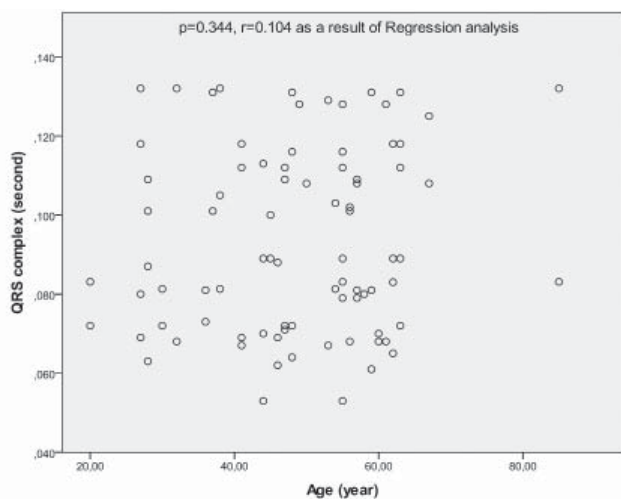


Figure 1. QRS complex duration according to age.

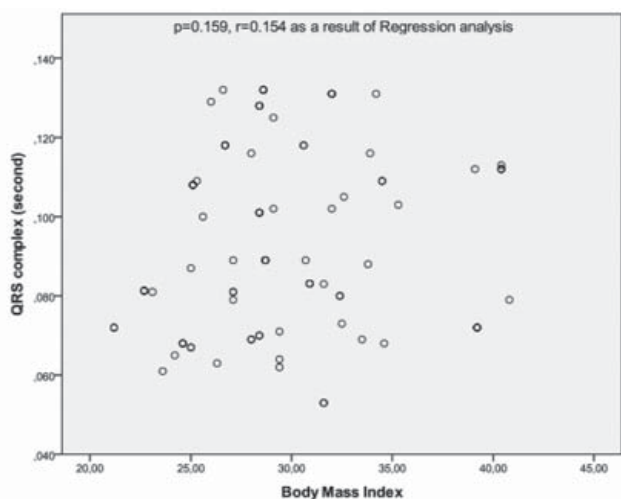


Figure 2. QRS complex duration according to BMI.

duration of QRS complex in Group 2 consisted of patients with moderate and mild OSA.

In conclusion, based on the results of the present study, it can be said that duration of QRS complex is longer in the subjects with severe OSA as compared to those with mild-moderate OSA. We think that subjects with severe OSA should be informed about cardiovascular problems due to widened QRS complex. We believe that our results need to be supported by larger-scale studies.

#### Study Limitations

Study participants are the Caucasians living in the same geographical region. Number of the subjects in the groups is adequate for statistical analysis but inadequate to adapt the results to the population. They have been followed for short time. Therefore, larger epidemiological studies are needed to verify the results of the present study.

#### CONFLICT of INTEREST

The authors reported no conflict of interest related to this article.

#### ACKNOWLEDGMENTS

We thank Assoc. Prof. Ismail Keskin\*, PhD for his contributions to the evaluation of results and statistical analysis.

\*Selçuk University, Zootechnics Division, Department of Biometry and Genetics, Konya, TURKEY.

#### AUTORSHIP CONTRIBUTIONS

Concept/Design: FA

Analysis/Interpretation: FA

Data acquisition: HK

Writing: FA

Critical revision: FA, HK

Final approval: FA, HK

#### REFERENCES

- Guilleminault C, Connolly SJ, Winkle RA. Cardiac arrhythmia and conduction disturbances during sleep in 400 patients with sleep apnea syndrome. *Am J Cardiol* 1983;52:490-4.
- Shephard JW, Garrison MW, Grither DA, Dolan GF. Relationship of ventricular ectopy to oxyhemoglobin desaturation in patients with obstructive sleep apnea. *Chest* 1985;88:335-40.
- Andreas S, Hajak G, von Breska B, Rütger E, Kreuzer H. Changes in heart rate during obstructive sleep apnoea. *Eur Respir J* 1992;5:853-7.
- Koehler U, Dübler H, Glaremin T, Junkermann H, Lübbers C, Ploch T, et al. Nocturnal myocardial ischemia and cardiac arrhythmia in patients with sleep apnea with and without coronary heart disease. *Klin Wochenschr* 1991;69:474-82.
- Boudoulas H, Schmidt HS, Clark RW, Geleris P, Schaaf SF, Lewis RP. Anthropometric characteristics, cardiac abnormalities and adrenergic activity in patients with primary disorders of sleep. *J Med* 1983;14:223-38.
- Miller WP. Cardiac arrhythmias and conduction disturbances in the sleep apnea syndrome. Prevalence and significance. *Am J Med* 1982; 73: 317-21.
- Tilkian AG, Guilleminault C, Schroeder JS, Lehrman KL, Simmons FB, Dement WC. Sleep-induced apnea syndrome: prevalence of cardiac arrhythmias and their reversal after tracheostomy. *Am J Med* 1977;63:348-58.
- Bolm-Audorff U, Köhler U, Becker E, Fuchs E, Peter JH, v Wichert P. Nächtliche Herzrhythmusstörungen bei Schlafapnoe Syndrom. *Dtsch Med Wochenschr* 1984;109:853-6.
- He J, Kryger MH, Zorick FJ, Conway W, Roth T. Mortality and apnea index in obstructive sleep apnea. Experience in 385 male patients. *Chest* 1988;94:9-14.
- Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230-5.
- Malhotra A, White DP. Obstructive sleep apnoea. *Lancet* 2002;360:237-45.
- Palomaki H, Partinen M, Juvela S, Kaste M. Snoring as a risk factor for sleep-related brain infarction. *Stroke* 1989;20:1311-5.
- Shahar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study. *Am J Respir Crit Care Med* 2001; 163:19-25.

15. Gami AS, Somers VK. Implications of obstructive sleep apnea for atrial fibrillation and sudden cardiac death. *J Cardiovasc Electrophysiol* 2008;19:997-1003.
16. Drager L, Bortolotto LA, Figueiredo LA, Caldin B, Krieger E, Lorenzi-Filho E. Obstructive sleep apnea, hypertension, and their interaction on arterial stiffness and heart remodeling. *Chest* 2007;131:1379-86.
17. Noda A, Okada T, Yasuma F, Nakashima N, Yokota M. Cardiac hypertrophy in obstructive sleep apnea syndrome. *Chest* 1995;107:1538-44.
18. Shivalkar B, Van de Heyning C, Kerremans M, Rinkevich D, Verbraecken J, De Backer W, et al. Obstructive sleep apnea syndrome: more insights on structural and functional cardiac alterations, and the effects of treatment with continuous positive airway pressure. *J Am Coll Cardiol* 2006;47:1433-9.
19. Usui K, Parker JD, Newton GE, Floras JS, Ryan CM, Bradley TD. Left ventricular structural adaptations to obstructive sleep apnea in dilated cardiomyopathy. *Am J Respir Crit Care Med* 2006;173:1170-5.
20. Dhingra R, Ho Nam, B, Benjamin EJ, Wang TJ, Larson MG, D'Agustino RB Sr, et al. Cross-sectional relations of electrocardiographic QRS duration to left ventricular dimensions: the Framingham Heart Study. *J Am Coll Cardiol* 2005;45:685-9.
21. Morin DP, Oikarinen L, Viitasalo M, Troivonen L, Nieminen MS, Kiildsen SE, et al. QRS duration predicts sudden cardiac death in hypertensive patients undergoing intensive medical therapy: the LIFE study. *Eur Heart J* 2009;30:2908-14.
22. Ott P, Marcus FI. Electrocardiographic markers of sudden death. *Cardiol Clin* 2006;24:453-69.
23. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22:667-89.
24. Somers VK, White DP, Amin R, Abraham WT, Costa F, Culebras A, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. *J Am Coll Cardiol* 2008;52:686-717.