Analysis of Echocardiographic Alterations Observed in Sleep Apnea–Hypopnea Syndrome and How They Are Influenced by Hypertension

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Introduction and objectives. Sleep apnea-hypopnea syndrome (SAHS) is associated with significant effects on the heart, which can be assessed using noninvasive methods such as transthoracic echocardiography. However, it is not clear whether these effects are due to the condition itself or are influenced by associated factors, such as high blood pressure (HBP). The objective of this study was to investigate the echocardiographic alterations observed in SAHS patients and how they are affected by the presence of concomitant HBP.

Methods. The study involved 103 consecutive patients (49 with HBP and 54 without) with SAHS and an indication for continuous positive airway pressure treatment and 24 controls matched for age and body mass index. Doppler echocardiography was performed in a blinded manner. Both morphology (ie, wall thickness, and diameters) and function (ie, ejection fraction, peak E and A wave velocities, mitral deceleration time, and Tei index) were assessed. Results were compared using ANOVA and Bonferroni's test.

Results. Hypertensive patients had larger morphological changes characteristic of left ventricular hypertrophy (ie, increased septal and posterior wall thicknesses) than nonhypertensive patients, who in turn had larger changes than controls (septal thickness: HBP-SAHS, 12 [2] mm; non-HBP SAHS, 11 [2] mm, and controls, 9.5 [5] mm; 1 vs 2, *P*=.038; 1 vs 3, *P*=.0001, 2 vs 3, *P*=.034) (posterior wall thickness: HBP-SAHS, 11 [2] mm; non-HBP SAHS, 10 [1] mm, and controls, 9 [1.5] mm; 1 vs 2, *P*=.5; 1 vs 3, *P*=.0001; 2 vs 3, *P*=.001). In addition, there were also greater changes in ventricular filling patterns on the left (HBP-SAHS, 92%; non-HBP SAHS, 72%, controls, 29%; *P*=.0001) and on the right (HBP-SAHS, 72%; non-HBP SAHS, 58%; controls, 25%; *P*=.001). There was a trend towards a larger left ventricular Tei index (HBP-SAHS, 0.56 [0.2]; non-HBP SAHS, 0.54 [0.12]; controls, 0.5 [0.1]; 1 vs 2, *P*=.8; 1 vs 3, *P*=.09; 2 vs 3, *P*=.7).

Conclusions. From the time of diagnosis, SAHS was associated with left ventricular hypertrophy and impaired biventricular filling, even in the absence of concomitant HBP. The abnormalities observed were more severe when HBP was present.

Key words: *Echocardiography. Systemic hypertension. Sleep. Hypertrophy.*

Análisis de las alteraciones ecocardiográficas del síndrome de apneas e hipopneas del sueño y su modificación con la presencia de hipertensión arterial

Introducción y objetivos. El síndrome de apneas e hipopneas del sueño (SAHS) conlleva importantes repercusiones cardiacas que se pueden evaluar mediante métodos no invasivos como la ecocardiografía transtorácica; sin embargo, está por dilucidar si se deben a la propia enfermedad o a la influencia de factores concomitantes como la hipertensión arterial (HTA). El objetivo de este estudio es analizar las alteraciones ecocardiográficas en pacientes con SAHS y si se modifican en caso de HTA concomitante.

Métodos. Estudiamos a 103 pacientes consecutivos diagnosticados de SAHS e indicación de presión positiva continua en la vía aérea (HTA, 49 pacientes; sin HTA, 54) frente a 24 controles ajustados por edad e índice de masa corporal. Realizamos ecocardiograma-Doppler por un observador para el que la asignación estaba enmascarada. Analizamos variables morfológicas (grosor de paredes y diámetros) y funcionales (fracción de eyección, velocidad máxima de ondas E y A, tiempo de deceleración mitral e índice Tei). Se compararon los resultados mediante ANOVA y test de Bonferroni.

Resultados. Los pacientes hipertensos tuvieron más alteraciones morfológicas tipo hipertrofia ventricular izquierda (mayor grosor de septo y pared posterior) que los no hipertensos, y éstos más que los controles. Grosor del septo: SAHS-HTA (1), 12 ± 2; SAHS sin HTA (2), 11 ± 2, y controles (3), 9,5 ± 5 mm (1 frente a 2, p = 0,038; 1 frente a 3, p = 0,0001, y 2 frente a 3, p = 0,034). Pared posterior: SAHS-HTA, 11 ± 2; SAHS sin HTA, 10 ± 1, y controles, 9 ± 1,5 mm (1 frente a 2, p = 0,5; 1 frente a 3,

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p = 0,0001, y 2 frente a 3, p = 0,001). También hubo más alteraciones en el patrón de llenado ventricular izquierdo (SAHS-HTA, 92%; SAHS sin HTA, 72%, y controles, 29%; p = 0,0001) y derecho (SAHS-HTA, 72%; SAHS sin HTA, 58%, y controles, 25%; p = 0,001). Los valores del índice Tei del VI tuvieron tendencia a incrementarse (SAHS-HTA, 0,56 ± 0,2; SAHS sin HTA, 0,54 ± 0,12, y controles, 0,5 ± 0,1; 1 frente a 2, p = 0,8; 1 frente a 3, p = 0,09; 2 frente a 3, p = 0,7).

Conclusiones. El SAHS presenta signos de hipertrofia ventricular izquierda y alteración del llenado biventricular aun en ausencia de HTA concomitante, y desde el momento de su diagnóstico. Las alteraciones detectadas son mayores cuando se asocia HTA.

Palabras clave: *Ecocardiografía. Hipertensión arterial sistémica. Sueño. Hipertrofia.*

ABBREVIATIONS

AHI: apnea–hypopnea index
BMI: body mass index
CPAP: continuous positive airway pressure
HBP: high blood pressure
SAHS: sleep apnea–hypopnea syndrome

INTRODUCTION

Sleep apnea–hypopnea syndrome (SAHS) is an emerging disorder that affects 4% to 6% of middle-aged men and 2% to 4% of middle-aged women, with a higher prevalence with increasing age.[1,2] The condition is defined as a set of symptoms that include excessive drowsiness, cognitive–behavioral disturbances, and respiratory, cardiac, metabolic, or inflammatory disturbances secondary to repeated episodes of upper airway obstruction during sleep.[3] SAHS is an independent risk factor for high blood pressure (HBP)[4,5] and has been shown to have other important cardiovascular consequences.[6] Cardiac involvement can be detected by echocardiography.[7-9] However, it is not clear if this condition is due to the disease itself or to other concomitant factors, mainly HBP, because the vast majority of studies excluded patients with HBP[10] or did not adjust for this variable.[9,11]

It is not uncommon for patients to receive treatment for SAHS-related cardiovascular problems or for SAHS to be underdiagnosed,[12] a situation that implies a high utilization of health care resources.[13,14] Knowing the cardiovascular impact from the moment of diagnosis and whether or not it is the result of the syndrome itself could provide greater insights into the pathophysiology of the condition and a more appropriate therapeutic approach.

The purpose of the present study was to investigate by echocardiography the cardiac alterations of patients with SAHS at the time of diagnosis and to analyze the influence of diurnal HBP in these patients.

METHODS

Study Population

We assessed 110 consecutive patients referred to a pulmonology outpatient clinic for suspected SAHS. The symptoms used to guide the SAHS diagnosis (snoring, witnessed apneas, excessive daytime drowsiness) were assessed on a severity scale, in order to establish the clinical probability of SAHS. Patients were included if they had SAHS and an indication for continuous positive airway pressure (CPAP) treatment, defined as an apnea–hypopnea index (AHI) ≥30 or ≥10 when there was pathological drowsiness, cardiovascular risk factors, or previous cardiovascular disease.[3]

We considered a patient with HBP to be one who was on drug therapy or had blood pressure values >140/90 mm Hg in 3 morning measurements performed by the nursing staff at their health centers.[15] The SAHS study population was divided into 2 groups: HBP-SAHS and non-HBP SAHS.

The control group included 32 consecutive individuals referred to the sleep clinic with no cardiovascular history and similar body mass index (BMI) and age. All had a low probability of SAHS when the described protocol was applied, and all underwent nocturnal polygraphy. Patients with pathological findings on polygraphy study or suspected HBP were excluded.[15]

Exclusion Criteria for Both Groups

The exclusion criteria for the study were: failure to obtain patient consent, atrial fibrillation, bradyarrhythmia (<60 bpm) or tachyarrhythmia (>100 bpm), known or echocardiography-detected cardiac disease, and poor acoustic window.

Nocturnal Study

Screening for SAHS was performed using an EMBLETTA® (ResMed, Spain) unit that had been validated against conventional polysomnography.[16] Nasal flow was recorded with a pressure transducer, and O_2 saturation and heart rate were measured with a digital pulse oximeter. Snoring and the number of apneas according to patient position were detected by a body position sensor, and chest and abdominal movements with an elastic thoracoabdominal band via a piezoelectric sensor. All studies were reviewed manually by the same pulmonologist.

Obstructive apnea was considered to be absence or >90% reduction in the breathing signal that lasted more than 10 s in a respiratory stress situation detected by the

thoracoabdominal bands. Central apnea was absence or >90% reduction of the breathing signal for more than 10 s in the absence of respiratory effort detected by the thoracoabdominal bands. Mixed apnea was considered to exist when the respiratory event usually started with a central component and ended with an obstructive component.[3] Hypopnea was defined as a discernible decrease (>30% and <90%) in the width of the respiratory signal that lasted more than 10 s, detected by thermistors, nasal cannula, or pneumotachograph, which was accompanied by desaturation (≥3%) and/or arousal in the electroencephalogram of the polysomnography record.[3] We considered AHI to be the number of respiratory events (apneas or hypopneas) per recording hour in bed. The study was considered valid once the patient acknowledged that s/he had slept almost normally for at least 3 h, and not valid if the recording was less than 4 h or the sensors had turned off. We defined SAHS as AHI ≥10 with pathological daytime hypersomnia (Epworth >10 points).

When the polygraphy was considered negative for the SAHS diagnosis, but the clinical symptoms were clearly indicative of the diagnosis, the patient was referred to a reference sleep clinic for conventional polysomnography.[3]

Doppler Echocardiography

Doppler echocardiography was performed within the first 2 weeks following the SAHS diagnosis and before CPAP treatment, as well as among all controls. The tests were carried out using an HP Sonos 5500® system with 2.5-MHz transducer (Philips, Eindhoven, The Netherlands). The other examinations were performed by the same echocardiography specialist, who was unaware of which group the patient belonged to.

According to the established guidelines,[17] the morphological measurements were taken in M mode with respect to a parasternal long-axis view. The ejection fraction was calculated by the Teicholz method. The left diastolic function parameters were obtained by pulsed Doppler between the mitral valve tips in a 4-chamber apical view. The aortic flow was obtained in the aortic valve plane. The tricuspid and pulmonary flows were analyzed in the parasternal cross-sectional view. The right ventricular isovolumic relaxation time was calculated by subtracting the time between QRS and pulmonary closure from the time between QRS and tricuspid opening. The Doppler measurements were taken at a sweep speed of 100 mm/s. Each individual value was the mean of three measurements.

Doppler Echocardiography Variables Analyzed

– Left ventricular hypertrophy (LVH) parameters: interventricular septal thickness (IVST) and posterior wall thickness (PWT). Left ventricular mass calculated by Penn method[18]

– Left chamber size: left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), and left atrium (LA)

– Right chamber size: right ventricular end-diastolic diameter (RVEDD)

– Systolic function parameters: left and right ventricular ejection fraction

– Diastolic function parameters: peak flow velocity through the atrioventricular valves (E and A waves); mitral deceleration time; E/A ratio; percentage of patients in whom the echocardiography specialist found an abnormal ventricular filling pattern, with the normal pattern defined as E>A and abnormal pattern defined as manifest relaxation alteration (E<A) or a pseudonormal pattern (E>A initially, but E<A after Valsalva maneuver)

– Myocardial performance parameters: Tei index for both ventricles, calculated using established standards[19,20] (Figure 1)

Reproducibility

The intraobserver variability was calculated from the 3 values obtained in each measurement. The variability was 1.8% for the morphological variables, 0.9% for Doppler variables of the left ventricle, and 1.2% for Doppler variables of the right ventricle, with an index of agreement κ>0.8 in all cases.

Statistical Analysis

The statistical analysis consisted of a descriptive and comparative study between categorical or continuous variables, taking their normal or non-normal distribution into account, using the χ^2 test or ANOVA with the Bonferroni post-hoc test. The numeric variables are expressed as mean (standard deviation), and the categorical variables, as percentages. A *P* value less than .05 was considered significant. SPSS 12.0 (SPSS Inc.) was used for the statistical analysis.

RESULTS

Of the 110 patients assessed, 7 were excluded (3 with atrial fibrillation, 1 with moderate aortic valve regurgitation, 1 with poor acoustic window, and 2 who refused participation). Among 32 controls, 8 were excluded (6 cases with elevated systolic and/or diastolic blood pressure values, and 2 with poor acoustic window). The final study population consisted of 103 patients (HBP, 49; non-HBP, 54), and 24 controls.

Clinical Profile of the Study Population

The clinical characteristics were similar between the SAHS population and control group, except for a higher percentage of smokers and former smokers and, obviously, higher AHI values among the SAHS population (Table 1).

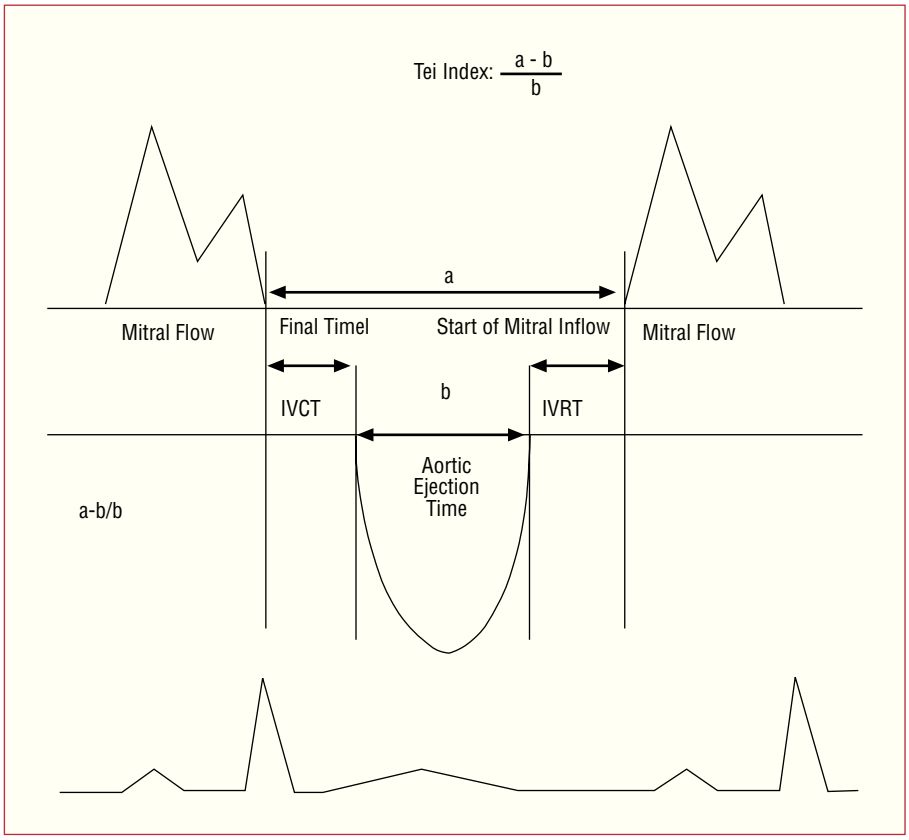

Figure 1. Left ventricular Tei index. The calculation is performed using the formula: a–b/b(IVCT+IVRT/ET). ET indicates aortic ejection time; IVCT, isovolumic contraction time; IVRT, isovolumic relaxation time.

Morphological and Systolic Function Variables

There were significant differences in the LVH parameters and patients with HBP-SAHS had greater left ventricular septum and posterior wall thicknesses than those with non-HBP SAHS, and non-HBP SAHS patients had higher values than the controls. There were no differences between groups in the other measurements (Table 2).

Functional Variables

A significantly abnormal ventricular filling pattern (both left and right) was found in the SAHS group and an even more severely altered pattern in the HBP-SAHS patients. In the LV, this was due to an increase in peak A-wave velocity, which showed a tendency to be higher in non-HBP SAHS patients compared to the controls and even higher in HBP-SAHS patients compared to the other 2 groups. Additionally, the peak E-wave velocity tended to decrease and was lower in the HBP-SAHS group. In the right ventricle, the tricuspid A-wave pattern was similar and no E-wave abnormalities were observed.

Although the differences are not significant, left ventricular myocardial performance (Tei index) was higher in HBP-SAHS patients than in the other groups, whereas right ventricular myocardial performance was similar (Table 3).

DISCUSSION

Recent studies show that patients with SAHS have a higher prevalence of HBP than the general population, even after adjusting for various confounding variables (eg, BMI, age, sex).[4,21] In addition, AHI has been shown to be an independent predictive factor of persistent HBP in patients with SAHS.[22] The explanation appears to be the cyclical increases in blood pressure that occur with nonobstructive respiratory events during the night, in which the following are implicated: central and peripheral chemoreceptors, pulmonary afferent baroreceptors, hypoxia and hypercapnia, increase in intrathoracic negative pressure, and transitory arousals. This induces a series of autonomic, hemodynamic, and humoral changes that produce a pressor effect when the apneas cease. The Wisconsin studies[5] and the Sleep Heart Health Study[4,23] have shown that the association between SAHS and HBP follows an increasing dose-response pattern according to SAHS severity. Furthermore, SAHS is known to be related to congestive heart failure and ischemic heart disease,[24] and is probably itself a risk factor capable

TABLE 1. **Clinical Characteristics of the SAHS and Control Groups**

	HBP-SAHS	Non-HBP SAHS	Control	*P*
Patients, n	49	54	24	
Age, mean (SD), y	56 (7)	52 (12)	53 (11)	.1
Male sex, %	71	79	63	.2
BMI, mean (SD)	33 (6)	31 (4)	30 (5)	.1
AHI, mean (SD)	39 (16)	41 (18)	7 (2)	.006
Smoking, %				<.001
Smokers	26	40	13	
Nonsmokers	51	24	79	
Ex-smokers	23	36	8	
Alcohol use, %	16	19	0	.08
Hyponotics and sedatives, %	6	26	0	.001
Duration, median (25th-75th percentiles), y	3.5 (2-6.5)	5 (2-5.75)	—	.6[a]

AHI indicates apnea–hypopnea index; BMI, body mass index; SAHS, sleep apnea–hypopnea syndrome.
[a]Comparing the HPB-SAHS group to the non-HBP SAHS group.

TABLE 2. **Echocardiographic (Morphological) Variables**

	HBP-SAHS (1) (n=49)	Non-HBP SAHS (2) (n=54)	Control (3) (n=24)	*P*
Left atrium, mm	38 (6)	36.5 (6)	35 (5)	1 versus 2=.3 1 versus 3=.07 2 versus 3=.9
Interventricular septum, mm	12 (2)	11 (2)	9.5 (2)	1 versus 2=.038 1 versus 3=.0001 2 versus 3=.034
Posterior wall, mm	11 (2)	10 (1)	9 (1.5)	1 versus 2=.5 1 versus 3=.0001 2 versus 3=.001
LV end-diastolic diameter, mm	47 (6)	47 (5)	47 (4)	1 versus 2=1 1 versus 3=1 2 versus 3=1
LV end-systolic diameter, mm	27 (6)	28 (5)	26 (5)	1 versus 2=.9 1 versus 3=.9 2 versus 3=.9
LV mass, g	254.5 (80.5)	224.6 (50)	186.5 (51.9)	1 versus 2=.058 1 versus 3=.0001 2 versus 3=.049
LV ejection fraction, %	79 (10)	78 (9)	81 (6)	1 versus 2=.9 1 versus 3=.9 2 versus 3=.6
RV end-diastolic diameter, mm	24 (5)	22 (5)	23 (4)	1 versus 2=.2 1 versus 3=.9 2 versus 3=.9
RV hypertrophy	4%	2%	0%	.5
Normal RV function	100%	100%	100%	1

HBP indicates high blood pressure; LV, left ventricle; RV, right ventricle; SAHS, sleep apnea–hypopnea syndrome.

of producing a wide array of cardiovascular diseases[25] which, in the long-term, are the factors that condition the prognosis.[26,27] It remains to be seen if intermittent nocturnal hypoxia and the other pathophysiological mechanisms of SAHS have a direct impact on the heart, such that any echocardiographic alterations would be due to the syndrome itself and not to other factors such as obesity or HBP.[28]

In this study, we proposed to investigate these cardiac abnormalities from the time of SAHS diagnosis and determine whether they are due to concomitant HBP. To do this, we compared these patients to a control group of people of similar BMI and age, but without SAHS. Until now, most studies conducted to analyze the myocardial repercussions have excluded hypertensive patients,[10] and those studies which did include them failed to adjust for this variable.[9,11]

TABLE 3. **Echocardiographic (Functional) Variables**

	HBP-SAHS (1) (n=49)	Non-HBP SAHS (2) (n=54)	Controls (3) (n=24)	*P*
Mitral peak E-wave velocity, cm/s	65 (19)	75 (20)	72 (16)	1 versus 2=.046 1 versus 3=.5 2 versus 3=.9
EDT, ms	204 (64)	200 (45)	177 (39)	1 versus 2=.9 1 versus 3=.1 2 versus 3=.2
Mitral peak A-wave velocity, cm/s	83 (13)	71 (20)	62 (10)	1 versus 2=.001 1 versus 3=.0001 2 versus 3=.06
Mitral E/A	0.78 (0.2)	1.11 (0.4)	1.19 (0.3)	1 versus 2=<.0001 1 versus 3=<.0001 2 versus 3=.9
Patients with abnormal LV filling, %	92	72	29	.0001
LV IVRT, ms	105 (14)	102 (18)	107 (15)	1 versus 2=.9 1 versus 3=.9 2 versus 3=.6
LV TEI	0.56 (0.2)	0.54 (0.12)	0.5 (0.1)	1 versus 2=.8 1 versus 3=.09 2 versus 3=.7
Tricuspid peak E-wave velocity, cm/s	54 (10)	55 (11)	56 (12)	1 versus 2=.9 1 versus 3=.9 2 versus 3=.9
Tricuspid peak A-wave velocity, cm/s	63 (17)	53 (14)	48 (10)	1 versus 2=.004 1 versus 3=.0001 2 versus 3=.5
Tricuspid E/A	0.9 (0.2)	1.09 (0.3)	1.19 (0.2)	1 versus 2=.003 1 versus 3=<.0001 2 versus 3=.49
Patients with abnormal RV filling, %	72	58	25	.001
RV IVRT, ms	74 (28)	65 (26)	80 (34)	1 versus 2=.2 1 versus 3=.8 2 versus 3=.09
RV TEI	0.47 (0.2)	0.43 (0.3)	0.44 (0.14)	1 versus 2=.8 1 versus 3=.9 2 versus 3=.9
Tricuspid regurgitation	4%	11%	12%	.3

EDT indicates E-wave deceleration time; HBP, high blood pressure; IVRT, isovolumic relaxation time; LV, left ventricle; RV, right ventricle.

Similar to the results described by other authors with respect to the magnitude of the alterations,[8,29] we found that non-HBP SAHS patients had LVH, but these changes were greater if the patient had HBP. This finding could be due to the sympathetic overstimulation of the myocardium that occurs in this syndrome and to the short, but repeated, postloading increments that occur during the apneas, since CPAP treatment could solve these aspects with an effect similar to that of beta-blockers in patients with heart failure.[24]

However, LVH was not the only finding. Our study indicates that cardiac function abnormalities can occur in SAHS and may even be present from the time of diagnosis. These alterations are mainly related to the diastolic function of both ventricles, but also to a trend toward worsening of left ventricular myocardial performance. Our findings for systolic function, however, were normal in all groups and contradicted those of other studies.[7,9,11] Alchanatis et al[7] reported 62% of mild-to-moderate systolic dysfunction with isotopic ventriculography in a population of 29 patients. This difference could be explained by an inclusion bias resulting from inadequate screening for heart disease, because the study's SAHS population had blood pressure values in the range of HBP (137 (7)/91 (11) mm Hg) even though HBP and antihypertensive therapy were listed as exclusion criteria. Curiously, the previous echocardiograms and electrocardiograms were found to be normal in a population with such a high percentage of mild-to-moderate systolic dysfunction. Laaban et al[11] analyzed 169 patients and found that the prevalence of systolic dysfunction was 7.7%, although there may have been a similar problem because these authors arbitrarily defined high blood pressure as systolic pressure >160

mm Hg and/or diastolic pressure >95 mm Hg. In addition, even though patients who had presented clinical symptoms of heart failure (not in the 2 months prior to inclusion) were eligible for enrollment, the heart disease screening process looked at patients only if systolic dysfunction had been observed in the ventriculography and focused only on ischemic and valvular heart disease. Shivalkar et al[9] found statistically significant differences in systolic function among a population of 43 patients with SAHS and another of 40 control subjects. However, as we found in our study, both were within normal limits (SAHS, 62% [9%] vs controls, 68% [6%]; *P*=.012). This difference may have been influenced by the fact that the control group had significantly lower BMI values (SAHS, 31.6 [5.4]; controls, 26.4 [2.3]).

Our results showed no differences between groups in terms of right ventricular size and function. Other authors describe significant right ventricular dilation with respect to the controls,[9] possibly due to higher pulmonary systolic pressure in the SAHS group. However, we did not observe pulmonary hypertension in any of our patients.

Our patients with SAHS had abnormal diastolic function of both ventricles, regardless of whether they had HBP, although this does aggravate it. These findings are not clearly delineated in the literature, since most series only assessed systolic function by isotopic techniques.[7,11] Some authors assume that when left atrium size is normal, increased ventricular wall thickness should not have caused significant distensibility abnormalities.[8] Although no differences between the groups were observed in our study in terms of the anteroposterior diameter of the left atrium, it is likely that volume calculations would have shown some difference, since it is well known that left ventricular diastolic function abnormalities produce left atrial enlargement.[30] Other authors, such as Shivalkar et al,[9] took the measurements needed for characterization using pulsed and tissue Doppler[31] and found biventricular diastolic dysfunction in SAHS patients compared to a control group (without stratifying their population according to blood pressure); however, BMI and blood pressure were significantly lower in the controls than the patients, and the discussion was focused on the systolic function results. The additional use of tissue Doppler imaging made it possible to detect left ventricular diastolic function changes consisting of decreases in protodiastolic velocity and increases in the end-diastolic velocity of the mitral annulus in obese SAHS patients in whom the transmitral flow parameters were not significantly different from those observed in the controls.[32] Alchanatis et al[10] compared a sample of SAHS patients to a control group with no differences in age and BMI and found left ventricular diastolic dysfunction with preserved systolic function, as observed in our study. However, these authors also did not look at the role of HBP in their SAHS group. Dursunoglu et al[33] did take HBP into account as a confounding factor when analyzing a population of non-HBP subjects in whom they found right ventricular diastolic dysfunction at baseline that improved after 6 months of CPAP treatment. However, the study only included 18 patients and did not have a control group.

Myocardial performance has not been as well described in these patients. The Tei index is a measure related to ejection fraction, E/A ratio, systolic volume, peripheral resistances, and ventricular mass, and is independent of heart rate and blood pressure.[34] This parameter does not require normalization, and its pathological significance is interpreted as higher values are reached. In our series, there was a nonsignificant trend in SAHS patients to present a higher index than the controls, which was even higher in the HBP-SAHS group. Healthy ventricle has a long ejection time, whereas pathological ventricle is characterized by progressive shortening of the ejection time as the disease progresses.[35] Therefore, regardless of the presence of HBP, SAHS appears to generate chronic stress in the myocardium that leads to a deterioration in myocardial performance. These alterations in the Tei index may be more obvious in patients with more severe SAHS or who have a longer history of untreated SAHS.

Study Limitations

Tissue Doppler imaging would have provided greater capacity for the early detection of abnormalities of diastolic function, along with a more precise Tei index.[29,36] However, this had not been implemented by our team at the time we started our study. Similar to other authors,[37] we used the Valsalva maneuver to distinguish between normal and pseudonormal filling. Nonetheless, this method has not been sufficiently validated and probably lacks adequate standardization. Moreover, whereas the percentage of control subjects in whom the echocardiography specialist found an abnormal ventricular filling pattern was similar (after adjusting for obesity and the predominance of men) to the results published by European epidemiological studies,[38] the patients (of similar age to the controls and therefore comparable) had significantly higher percentage of individuals with alterations (HBP, 92%; without HBP, 72%; controls, 29%; *P*=.0001). The use of tissue Doppler imaging would probably have increased the percentage of patients observed with diastolic dysfunction in each group, but the differences are so large that we do not feel that the final result would have been affected. The SAHS diagnosis by polysomnography would give more weight to our results. However, the waiting lists are long and we felt that the polygraphy was a useful diagnostic alternative in patients with a high clinical suspicion of SAHS,[3,39,40] and our diagnostic protocol and screening criteria are similar to those used in other studies.[41] The 24h blood pressure monitoring would have been able to classify the patients with HBP more accurately because it would have detected those with nocturnal nondipper pattern,[42] but our purpose was focused on determining if patients with

non-HBP SAHS detected by the usual methods already showed cardiac involvement. A multivariate analysis would have allowed us to adjust the results to parameters such as smoking habit, and alcohol and sedative use; however, this would have required a higher number of subjects.

CONCLUSIONS

We detected a series of morphological, functional, and cardiac performance abnormalities characterized by increased ventricular wall thickness and impaired biventricular filling that worsened in the presence of diurnal HBP, but were also present in non-HBP SAHS.

We conclude that cardiac abnormalities detectable on echocardiography occur in SAHS and are characterized by increased left ventricular wall thickness and impaired ventricular filling unrelated to the presence of diurnal HBP. It remains to be seen whether these abnormalities will disappear with continuous CPAP treatment and how long this will take.

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