A Novel Method for Sensitive Determination of Subclinical Left-Ventricular Systolic Dysfunction in Subjects With Obstructive Sleep Apnea

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BACKGROUND: This study was to evaluate the subclinical left-ventricular (LV) systolic dysfunction with 2-dimensional speckle-tracking echocardiography in subjects with obstructive sleep apnea (OSA) with normal left ventricular ejection fraction and without any confounding disease that can cause myocardial dysfunction. METHODS: Nineteen healthy individuals and 60 subjects with OSA were included in this study. According to the severity of disease, OSA subjects were examined in 3 groups: mild, moderate, and severe OSA. LV apical views (for longitudinal strain) and short-axis views (for circumferential strain) were acquired for evaluation. Three-layer longitudinal strain values and circumferential strain values were determined for each view, and averages of these were used in comparison with other groups. RESULTS: Three-layer longitudinal strain values of the subjects with OSA were lower than those of the healthy individuals, and these values were decreased along with the OSA severity. The difference was significant between severe OSA and all other groups. Three-layer circumferential strain values of the OSA subjects were lower than those of the healthy individuals, and the difference was significant between the control group and all other groups. The apnea hypopnea index was found to be correlated with the 3-layer longitudinal strain ($r = -0.74$, $P < .001$; $r = -0.72$, $P < .001$; $r = -0.69$, $P = <.001$). CONCLUSIONS: Three-layer longitudinal and circumferential LV systolic functions in OSA subjects with normal left ventricular ejection fraction are deteriorated in the subclinical stage. Two-dimensional speckle-tracking echocardiography can be used as an effective method in the determination of subclinical myocardial dysfunction in subjects with OSA. *Key words: obstructive sleep apnea; speckle-tracking echocardiography; myocardial strain.* [Respir Care 2016;61(3):366–375. © 2016 Daedalus Enterprises]

Introduction

Obstructive sleep apnea (OSA) is a syndrome characterized by repeated episodes of upper respiratory tract obstruction episodes during sleep and commonly a decrease in arterial oxygen saturation. Several investigations consistently showed that OSA may contribute to the impair-

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ment of left-ventricular (LV) systolic function, leading to heart failure.[1-3] Echocardiography is the most common imaging modality used today to assess LV myocardial function; the most widely used clinical tool to quantify LV systolic function is the ejection fraction. However, most patients with OSA without other cardiovascular disease usually exhibit normal LV ejection fraction; nevertheless, they still may have subclinical LV systolic dysfunction. This is due to the fact that the normal value of LV ejection fraction does not always indicate normal LV systolic function. On the other hand, diastolic function is often impaired in OSA.[4-6] Myocardial oxidative stress and ischemia are the main explanations for these disturbances.[7,8]

The LV wall is not homogenous and is composed of 3 layers of fibers. Opposite orientation of the myocardial fibers in the subendocardial and subepicardial layer is important for the equal redistribution of stress and strain in the heart.[9] Nonhomogenous deformation of the basal, middle, and apical ventricular segments provides coordinated LV contraction. However, coordination may be disturbed due to the decrease in arterial oxygen saturation and the high negative intrathoracic pressure imposed by OSA.[10]

Two-dimensional speckle tracking is a novel echocardiographic method for obtaining strain and strain rate measurements by tracking speckles in the ultrasonographic image in 2 dimensions. It is not based on tissue Doppler measurements; consequently, it is not angle-dependent.[11] This imaging technique discriminates between active and passive myocardial motion and enables the angle-independent quantification of myocardial deformation in 2 dimensions,[12]which allows analysis of myocardial deformation based on speckle tracking separately within each of 3 myocardial layers, defining the effect of OSA on deformation of the 3 layers. The evaluation of 3-layer longitudinal and circumferential strain of an OSA patient with 2-dimensional strain may be superior to conventional measurements and can be used in the clinic for early detection of LV systolic deterioration.

QUICK LOOK

Current knowledge

Two-dimensional speckle tracking is a novel echocardiographic method for obtaining strain and strain rate measurements by tracking speckles in the ultrasonographic image. This imaging technique discriminates between active and passive myocardial motion and enables the angle-independent quantification of myocardial deformation, which allows analysis of myocardial deformation based on speckle tracking separately within each of 3 myocardial layers, defining the effect of obstructive sleep apnea (OSA) on deformation of the 3 layers.

What this paper contributes to our knowledge

Longitudinal and circumferential strain in 3 myocardial layers were homogeneously decreased and correlated with the degree of OSA. The evaluation of 3-layer longitudinal and circumferential strain of a patient with OSA with 2-dimensional strain may be superior to conventional measurements and can be used in the clinic for early detection of left-ventricular systolic deterioration.

Methods

Study Population

In this study, subjects >18 y old with an OSA diagnosis who were examined in the Department of Respiratory Medicine, Zhongshan Hospital, Fudan University, between March 2014 and November 2014 were included in this study after conducting polysomnographies at the sleep laboratory. As a control group, we chose asymptomatic healthy individuals between 20 and 80 y old without cardiovascular diseases who visited the Department of Cardiology out-patient clinic in Zhongshan Hospital, Fudan University, for a cardiovascular check-up. The healthy group used in the study included subjects suitable for the study from the perspective of cardiac anatomy and functions, with no night snoring or daytime sleepiness, and with a low risk of OSA as judged by an evaluation. Exclusion criteria were as follows: history of documented myocardial infarction, myocarditis, dilated cardiomyopathy and/or HF, coronary artery disease diagnosed by clinical and coronary angiography, alcoholic cardiomyopathy, primary hypertrophic cardiomyopathy, prior myocardial revascularization, significant valvular heart disease, or electrocardiographic findings potentially associated with myocardial ischemia, such as grade III atrio-ventricular block.

Informed consent was obtained from every individual included in the study, and the local ethics committee of Zhongshan Hospital, Fudan University, approved the study. All individuals' blood pressures and anthropometric measures were recorded before echocardiography. Body mass index was derived from the anthropometric measures.

Polysomnography

Overnight fully attended polysomnography monitoring was performed with the Alice 4 Sleep System (Respironics, Murrysville, Pennsylvania) in the Referral Center for Sleep Medicine using standard recording techniques and a

precise protocol of polysomnography monitoring.[13] Surface electrodes were applied to perform a electroencephalogram, a chin electromyogram, an electrocardiogram, and electro-oculography. Air flow was monitored using an air pressure sensor placed at the nose and a thermistor placed at the nose and mouth, and oxygen saturation was recorded continuously with a pulse oximeter. Apnea was defined as the disappearance of air flow for >10 s; hypopnea was defined as a ≥50% decrease in air flow lasting for >10 s associated with arousal or a ≥3% decrease in oxygen saturation from the baseline level. The apnea-hypopnea index (AHI) was calculated as the total number of apnea and hypopnea episodes/h of sleep. According to the AHI, subjects were classified as having OSA when the obstructive component was >5 events/h. Severe OSA was classified as AHI of >30 events/h.[14]

Echocardiographic Measurements

Echocardiography was performed in the left lateral decubitus position with an ultrasound machine GE-Vingmed Vivid 9 system (GE-Vingmed Ultrasound AS, Horten, Norway) and 3S-RS (3.5-MHz) probe. Averages of 3 consecutive cycles were measured for all echocardiographic data. Images were obtained from parasternal and apical position using 2-dimensional echocardiographic techniques. Examinations were performed by 2 experienced cardiologists who were unaware of the groups of individuals. The LV ejection fraction was measured using the biplane Simpson method. The frame rate was ≥40 frames/s. Short-axis views at the basal, middle (papillary muscle level), and apical levels and apical 4-chamber, 3-chamber, and 2-chamber views were analyzed using the EchoPAC software package. Peak systolic myocardial strain was obtained in 3 myocardial layers from short-axis views (circumferential strain) and from apical views (longitudinal strain). The left ventricle was divided into 18 cardiac segments: 3 levels (basal, middle, and apical), each further divided into 6 segments (anterior, posterior, lateral, inferior, septal, and anteroseptal).

Statistical Analysis

SPSS 19.0 statistical analysis software (SPSS, Chicago, Illinois) was used to evaluate variables and test. Numeric variables are presented as mean ± SD values, and categorical variables are presented as rates. Three or more group comparisons were performed by a one-way analysis of variance test for normally distributed variables. The relationships between the LV parameter and AHI included in the study were analyzed using the Pearson correlation test. All of the hypotheses were constructed as 2-tailed, and an α critical value of .05 was accepted as significant.

Inter- and Intra-Observer Variability

Intra-observer variability was determined by the observer repeating the measurement of the peak global longitudinal strain in 20 random OSA or control subjects 2 weeks after the first measurement. Inter-observer variability was determined by another observer's measurement of those variables in the same database. Intra- and inter-observer variabilities were then calculated as the absolute difference between the 2 corresponding measurements as a percentage of their mean. Intra- and inter-observer reproducibilities were evaluated by means of the intraclass correlation coefficient.

Results

Clinical and Demographic Results

A total of 79 individuals enrolled into our study; 19 were healthy individuals, and 60 had OSA. Subjects with OSA were divided into 3 groups according to their AHI values: mild (n = 20), moderate (n = 16), and severe (n = 24). Controls were similar to subjects with mild OSA. Body weight and body mass index were greater in the severe and moderate OSA groups than that of the mild OSA and control groups. There was a significant decrease in mean S_{pO_2} and the lowest S_{pO_2} in severe OSA when compared with other groups ($P < .001$). The clinical and demographic data of the groups are presented in Table 1.

Echocardiographic Results

The M-mode, 2-dimensional, pulse-wave Doppler and tissue Doppler echocardiographic variables are presented in Table 2. Echocardiographic parameters other than interventricular septum diastolic thickness diameter and systolic pulmonary artery pressure were not different among groups; interventricular septum diastolic thickness diameter was greater in the severe (1.14 ± 0.19 vs 0.92 ± 0.11 cm, P = .01) and moderate (1.12 ± 0.16 vs 0.92 ± 0.11 cm, P = .02) OSA group than that of the control group. There was a significant difference between the severe OSA (32.8 ± 6.7 mm Hg vs 16.7 ± 6.2 mm Hg, P = <.001), moderate OSA (31.2 ± 5.6 mm Hg vs 16.7 ± 6.2 mm Hg, $P < .001$), and healthy control groups in systolic pulmonary artery pressure values.

Three-Myocardial Layer Longitudinal Strain in OSA and Control Subjects

Three-myocardial layer longitudinal strain in control subjects and subjects with OSA are depicted in Figure 1A and Figure 2A. There was a trend toward an epicardial-to-

Table 1. Clinical and Demographic Characteristics of Subjects With Obstructive Sleep Apnea and Healthy Controls

Variables	Controls (n = 19)	Mild OSA (n = 20)	Moderate OSA (n = 16)	Severe OSA (n = 24)
Age, median ± SD y	52.0 ± 10.8	53.9 ± 11.7	58.8 ± 10.7	49.7 ± 12.7
Female, %	36.8	20	18.8	8.3
Heart rate, median ± SD beats/min	67.7 ± 8.6	70.2 ± 9.7	68.5 ± 9.2	68.7 ± 8.9
Height, median ± SD cm	168.6 ± 5.2	167.5 ± 8.0	170.6 ± 6.6	169.3 ± 6.8
Body weight, median ± SD kg	63.7 ± 5.1	70.8 ± 12.9	76.7 ± 10.6*	80.5 ± 10.9†‡
Body mass index, median ± SD kg/m^2	24.2 ± 3.7	30.6 ± 9.7	35.0 ± 9.5*	38.9 ± 9.5†‡
Systolic blood pressure, median ± SD mm Hg	119.4 ± 8.6	116.6 ± 8.2	121.2 ± 8.7	118.1 ± 9.7
Diastolic blood pressure, median ± SD mm Hg	74.3 ± 7.1	71.6 ± 7.3	76.1 ± 6.9	72.6 ± 7.4
Smoking, %	39.2	43.6	41.7	48.5
Mean S_{pO_2}, median ± SD %	95.4 ± 1.7	96.0 ± 1.6	94.9 ± 1.2	91.3 ± 4.0†‡§
Lowest S_{pO_2}, median ± SD %	88.7 ± 3.1	83.1 ± 6.3	76.5 ± 10.9*	68.1 ± 12.0†‡§

* $P < .05$ between moderate OSA and controls.
† $P < .05$ between severe OSA and controls.
‡ $P < .05$ between severe and mild OSA.
§ $P < .05$ between severe and moderate OSA.
OSA = obstructive sleep apnea

Table 2. Left-Ventricular Echocardiographic Parameters of Subjects With Obstructive Sleep Apnea and Healthy Controls

Variables	Controls (n = 19)	Mild OSA (n = 20)	Moderate OSA (n = 16)	Severe OSA (n = 24)
LVEF, %	65.7 ± 3.3	66.1 ± 4.7	64.2 ± 3.4	63.8 ± 2.9
IVSD, cm	0.92 ± 0.11	0.97 ± 0.17	1.12 ± 0.16*	1.14 ± 0.19†
PWD, cm	0.91 ± 0.1	0.97 ± 0.15	1.09 ± 0.14	1.09 ± 0.15
LVEDD, cm	4.67 ± 0.37	4.71 ± 0.39	4.70 ± 0.41	4.72 ± 0.37
LVESD, cm	2.91 ± 0.36	2.97 ± 0.37	2.96 ± 0.47	3.09 ± 0.41
E/A	1.17 ± 0.21	1.07 ± 0.18	0.96 ± 0.24	1.02 ± 0.19
DT, ms	183.1 ± 20.6	178.6 ± 19.8	197.2 ± 23.7	183.1 ± 28.9
S, cm/s	11.1 ± 1.8	12.4 ± 2.1	10.2 ± 1.9	11.4 ± 1.2
SPAP, mm Hg	16.7 ± 6.2	18.2 ± 6.6	31.2 ± 5.6*	32.8 ± 6.7†

Values represent median ± SD.
* $P < .05$ between moderate OSA and controls.
† $P < .05$ between severe OSA and controls.
OSA = obstructive sleep apnea
LVEF = left-ventricular ejection fraction
IVSD = interventricular septum diastolic thickness diameter
PWD = posterior wall diastolic thickness diameter
LVEDD = left-ventricular end-diastolic diameter
LVESD = left-ventricular end-systolic diameter
E/A = ratio between early and late diastolic inflow velocities
DT = deceleration time
S = systolic annular myocardial velocity
SPAP = systolic pulmonary artery pressure

endocardial gradient: Endocardial strain was higher than middle and epicardial strain. Three-layer longitudinal strain in the severe OSA group was significantly lower than in the control, mild OSA, and moderate OSA groups, but there was no significant difference between any other groups (Fig. 3A). Nevertheless, at the basal, middle, and apical levels, 3-layer longitudinal strain in the severe OSA group was significantly lower than in the other 3 groups (Table 3).Three-layer strain at the apical level was higher than at the mid-ventricle, and strain at the mid-ventricle was higher than at the base (a basal-to-apical gradient).

Three-Myocardial Layer Circumferential Strain in Subjects With OSA and Control Subjects

Three-myocardial layer circumferential strain in control subjects and in subjects with OSA is depicted in Figure 1 (B, C, and D) and Figure 2 (B, C, and D). At each myocardial level, endocardial strain was highest, and epicardial strain was lowest (an epicardial-to-endocardial gradient). At all myocardial layers, apical strain was highest, and basal strain was lowest (a basal-to-apical gradient). Three-layer circumferential strain in

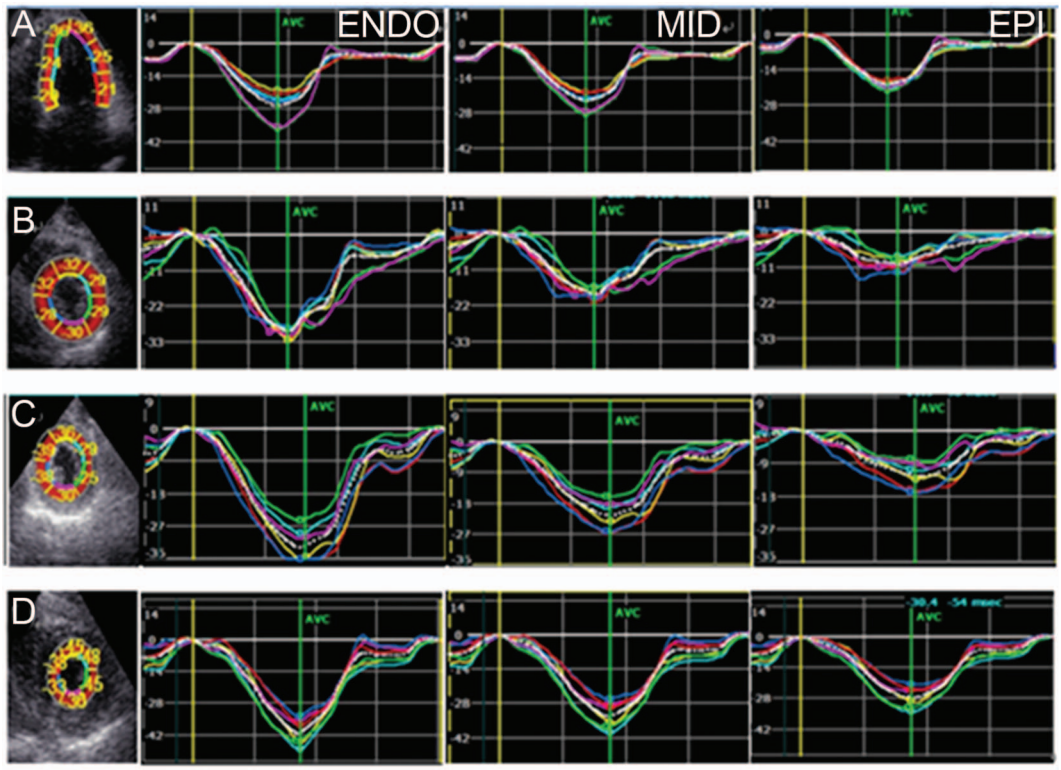

Fig. 1. Three-layer longitudinal and circumferential strain in a control subject. Shown are an apical 4-chamber view (A), short-axis view at the level of the mitral valve (B), short-axis view at the level of the papillary muscles (C), and short-axis view at the level of the apex (D). In the apical 4-chamber view (longitudinal strain) and short-axis views (circumferential strain), endocardial strain is highest, and epicardial strain is lowest (an epicardial-to-endocardial gradient). ENDO = endocardial strain; MID = mid-layer; EPI = epicardial strain.

the OSA group was significantly lower than in the control group. but there was no significant difference between any other groups (Fig. 3B). At the basal, middle, and apical levels, 3-layer longitudinal strain levels, except in the epicardial apical and middle segment in the severe OSA group, were significantly lower than in the 3 groups (Table 4).

Relationship Between Echocardiographic Parameters and AHI

In the correlation analysis, longitudinal and circumferential strain was found to be correlated with body mass index, interventricular septum diastolic thickness diameter, systolic pulmonary artery pressure, and AHI. Longitudinal strain of the OSA group was found to be well correlated with AHI (Fig. 4); however, linear regression analysis results indicated that only the AHI parameter has a contribution to endocardial longitudinal strain, which represents LV systolic function ($\beta = -0.59$, $P = .008$). Circumferential strain of the OSA group was found to be not correlated with AHI.

Reproducibility

Twenty subjects were randomly selected for the assessment of intra- and inter-observer variability in measurements of endocardial longitudinal strain. The intra- and inter-observer reproducibility of endocardial longitudinal strain was shown to be acceptable. The intra- and inter-observer variations were 6.2 and 7.4%, respectively, for endocardial longitudinal strain. The corresponding intra-class correlation coefficient was 0.91.

Discussion

Our results indicated that, beginning with the severe OSA group, 3-layer longitudinal LV functions decreases with increasing disease severity in OSA subjects despite the fact that the LV ejection fraction is not different between these subjects and healthy individuals. The decrease in longitudinal LV functions was correlated with the presence and severity of OSA. However, we found that the 3-layer circumferential LV functions decrease in all OSA subjects, and the decrease in circumferential LV functions did not correlate with the presence and severity of OSA.

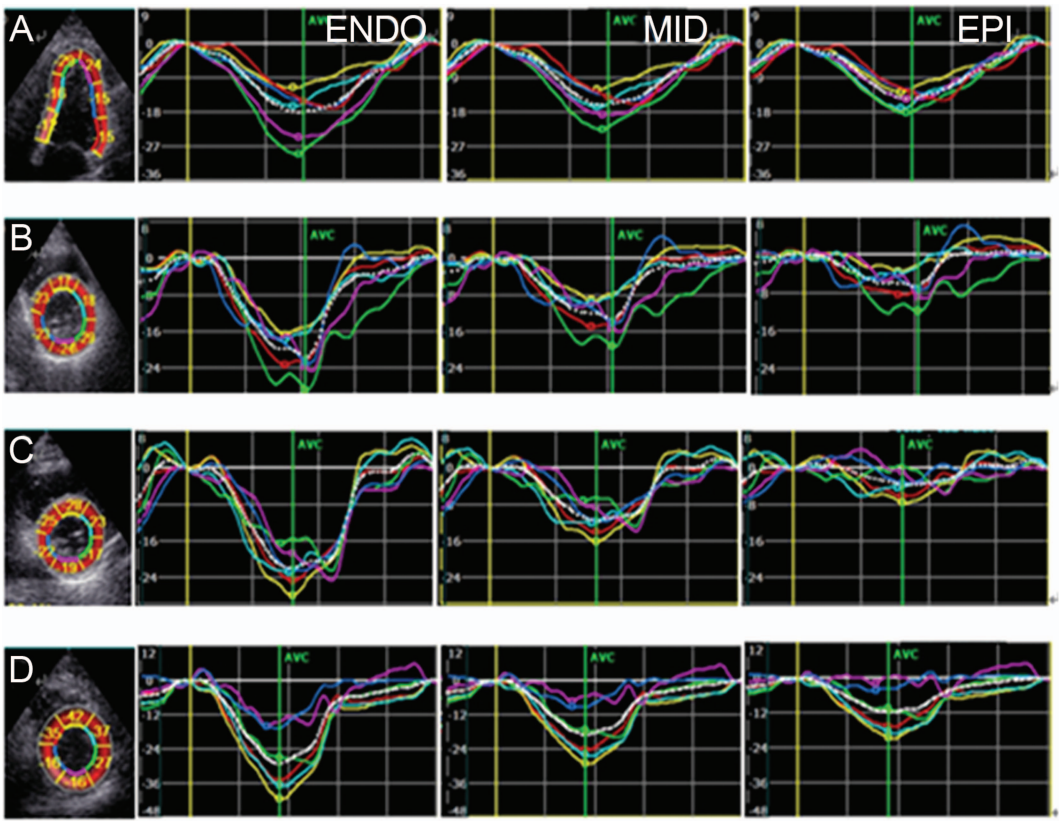

Fig. 2. Three-layer longitudinal and circumferential strain in a subject with severe obstructive sleep apnea (apnea-hypopnea index = 62.9). A: Apical 4-chamber view, longitudinal strain. B: Short-axis view at the level of the mitral valve, circumferential strain. C: Short-axis view at the level of the papillary muscles, circumferential strain. D: Apical short-axis view, circumferential strain. Compared with control subjects, these curves show reduced amplitude in most of the segments. ENDO = endocardial strain; MID = mid-layer; EPI = epicardial strain.

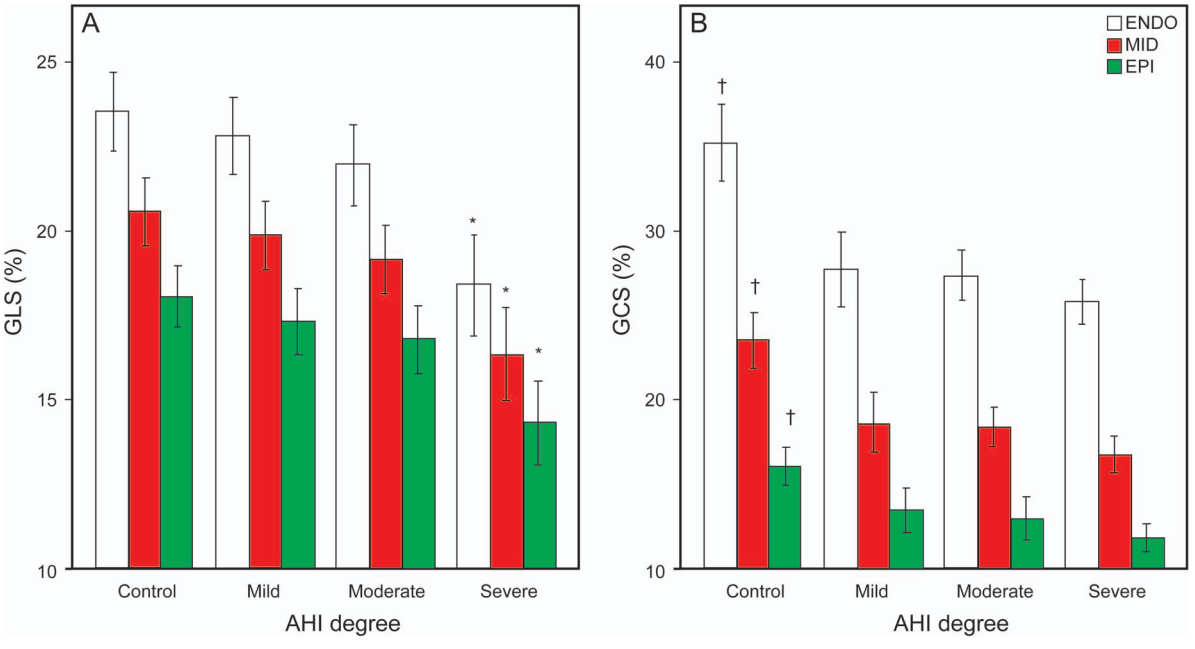

Fig. 3. Three-myocardial-layer longitudinal strain (A) and circumferential strain (B) in OSA subjects and controls. *, $P < .01$, between the severe group and the other 3 groups. †, $P < .01$ between control and the other 3 groups.

Table 3. Three-Layer Longitudinal Strain in Subjects With Obstructive Sleep Apnea and Control Subjects

	Base				Mid-Ventricle				Apex			
	Control	Mild	Moderate	Severe	Control	Mild	Moderate	Severe	Control	Mild	Moderate	Severe
Endocardial	−17.57 ± 2.48	−16.61 ± 2.30	−15.30 ± 2.55*	−13.6 ± 3.40†‡	−21.67 ± 2.53	−21.19 ± 2.79	−19.00 ± 3.67	−17.46 ± 5.75†‡	−33.36 ± 4.05	−32.74 ± 3.95	−30.78 ± 6.18	−26.45 ± 7.74†‡
Mid-layer	−17.16 ± 2.40	−16.34 ± 2.09	−14.88 ± 2.22*	−13.3 ± 3.24†‡	−19.71 ± 2.25	−19.50 ± 2.40	−17.36 ± 3.23	−15.83 ± 4.73†‡	−25.01 ± 3.08	−24.83 ± 3.71	−23.04 ± 4.80	−19.66 ± 5.67†‡
Epicardial	−16.74 ± 2.39	−16.01 ± 1.94	−14.50 ± 2.16*	−13.0 ± 3.09†‡	−18.08 ± 2.05	−18.01 ± 2.22	−15.86 ± 2.81	−14.54 ± 4.12†‡	−19.35 ± 2.46	−19.00 ± 3.17	−17.85 ± 4.00	−15.30 ± 4.09†‡

Values represent median ± SD.
* $P < .05$ between moderate OSA and controls.
† $P < .05$ between severe OSA and controls.
‡ $P < .05$ between severe and mild OSA.

Table 4. Three-Layer Circumferential Strain in Subjects With Obstructive Sleep Apnea and Control Subjects

	Base				Mid-Ventricle				Apex			
	Control	Mild	Moderate	Severe	Control	Mild	Moderate	Severe	Control	Mild	Moderate	Severe
Endocardial	−29.70 ± 5.43*†‡	−19.29 ± 5.27	−18.88 ± 5.45	−18.36 ± 4.14	−32.06 ± 4.34‡	−28.24 ± 6.04	−27.90 ± 4.94	−24.71 ± 5.29	−41.02 ± 7.60*†‡	−32.04 ± 6.28	−34.84 ± 6.81	−33.66 ± 6.22
Mid-layer	−19.72 ± 3.39*†‡	−14.64 ± 3.49	−13.82 ± 3.34	−13.70 ± 2.30	−21.25 ± 3.26†‡	−19.34 ± 3.82	−19.23 ± 2.28	−16.64 ± 3.98	−29.24 ± 5.34*†‡	−23.61 ± 4.38	−25.06 ± 5.29	−23.31 ± 4.41
Epicardial	−13.15 ± 2.19*†‡	−11.53 ± 3.32	−10.62 ± 3.08	−10.00 ± 2.94	−14.55 ± 2.25‡	−13.93 ± 2.25	−13.57 ± 1.63	−12.08 ± 3.25	−21.63 ± 4.47*†‡	−16.87 ± 3.88	−17.82 ± 3.84	−16.34 ± 4.07

* $P < .05$ between severe OSA and controls.
† $P < .05$ between moderate OSA and controls.
‡ $P < .05$ between mild OSA and controls.

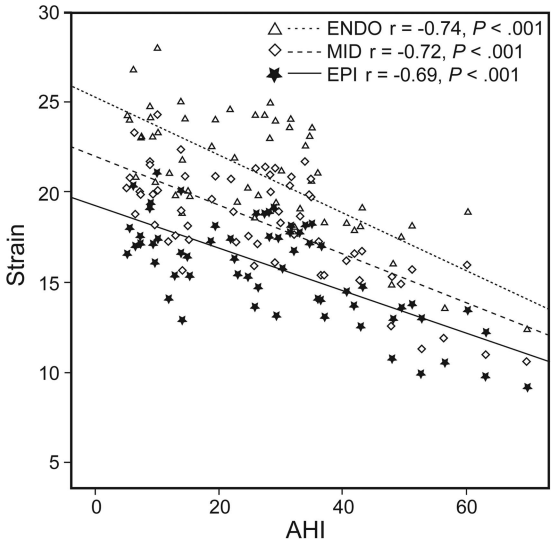

Fig. 4. Correlations between 3-layer longitudinal strain and apnea-hypopnea index. ENDO = endocardial strain; MID = mid-layer; EPI = epicardial strain.

In subjects with OSA, repetitive hypoxia due to sleep-induced apnea and/or hypopnea adversely affects the interaction between the myocardial oxygen demand and supply, resulting in the development of myocardial ischemia and subclinical LV myocardial dysfunction.[14,15] According to the previous data, there is a higher proportion of heart failure in subjects with OSA compared with the general population. However, most of them have normal ejection fraction despite signs of the heart failure.[16,17] Previous echocardiographic studies using tissue Doppler imaging and speckle-tracking imaging have demonstrated that OSA subjects may develop subclinical LV systolic and diastolic dysfunction.[1,18,19] Altekin et al[20] have also shown on a smaller group of subjects with OSA, using speckle-tracking echocardiography, that longitudinal and circumferential systolic LV dysfunction is detectable in addition to the diastolic dysfunction. In contrast to similar previous studies[19,21,22] evaluating the subclinical cardiac damage in subjects with OSA, our study is the first study to assess the 3-layer longitudinal and circumferential functions of the myocardium in subjects with OSA.

The LV wall is not homogenous and is composed of 3 layers of fibers. Opposite orientation of the myocardial fibers in the subendocardial and subepicardial layers is important for the equal redistribution of stress and strain in the heart.[9] The myocardial wall matures from a single-layered epithelium to a complex, multilayered structure.[23] Successive contraction and relaxation of the ventricular myocardial band produce several fundamental movements of the left ventricle: shortening, lengthening, thickening, thinning, etc.[24] Nonhomogenous deformation of the basal, middle, and apical ventricular segments provides coordinated LV contraction. In the present study, we found that in control subjects, longitudinal and circumferential strain was highest in the endocardium and apex and lowest in the epicardium and base, and in subjects with OSA, the strain was lower. This is in accordance with a previous study.[25]

LV systolic function is a complex coordinated action that involves longitudinal contraction, resulting in a shortening and twisting movement in the longitudinal axis, and circumferential contraction, resulting in shortening in the horizontal axis. The longitudinal LV mechanics, which are primarily governed by the subendocardial region, are the most vulnerable component of LV mechanics; therefore, this is the most sensitive part in a myocardial disease.[26] Repetitive hypoxia due to sleep-induced apnea may adversely affect the interaction between myocardial oxygen demand and supply, resulting in the development of relative myocardial ischemia and subclinical LV systolic dysfunction. This relative myocardial ischemia is different from the general myocardial ischemia that results from cardiovascular disease, such as coronary artery disease, myocardial infarction, and myocarditis. In most cases, LV systolic and diastolic dysfunction induced by OSA are temporary and functional, which could result in recovery when the hypoxia is improved or treated with CPAP therapy.[19] There are only a few published studies[19,21,22] that have used speckle-tracking echocardiography in the assessment of longitudinal and circumferential systolic myocardial functions in OSA subjects. In this study, we found that both longitudinal and circumferential strain in 3 myocardial layers were homogeneously decreased and correlated with the degree of OSA.

In our study, we investigated the correlation between OSA and subclinical systolic dysfunction by comparing 3-myocardial-layer longitudinal and circumferential strain values of OSA subjects. Longitudinal values decreased in the severe OSA group, and the values in the severe OSA group were lower than those in the healthy controls as well as the mild and moderate OSA groups. Strain values in basal, middle, and apical segments were also compared between the groups, and all segment values were decreased with increasing disease severity starting from the severe OSA group, the values for which were lower than for all other groups. Although no relationship was observed between OSA and circumferential strain values, the values in the OSA group were lower than those in the healthy controls. These negative effects of OSA on myocardial structure and functions have been attributed to various factors, especially hypoxia and hypercapnia, which ensue during sleeping; increased negative intrathoracic pressure during the arousal phase and hemodynamic changes, which occur during sympathetic system activation; and oxidative stress, systemic inflammation, endothelial dysfunction, ox-

ygen demand of the myocardium, and imbalances during presentation.[27,28]

There were limitations to our study, including the use of the Epworth and Berlin scales rather than the AHI in the selection of the control individuals. However, in daily clinical practice, we use these methods to select appropriate subjects for the polysomnography test. Although we excluded patients with diseases such as diabetes, coronary artery disease, and hypertension that may have adverse effects on myocardial function, the body mass index values of OSA subjects were higher than those of the healthy individuals.

Conclusions

OSA deteriorates LV systolic function, and the degree of LV longitudinal deterioration is proportionate with the disease severity. By evaluating LV longitudinal and circumferential functions with a 2-dimensional speckle-tracking echocardiography method, systolic dysfunction caused by OSA can be detected in the subclinical phase.

LV strain is not uniform over the left ventricle; it varies through myocardial layers and levels with circular and longitudinal inhomogeneity. The evaluation of 3-layer longitudinal and circumferential strain of OSA subjects with 2-dimensional strain proves to be superior to conventional measurements and can be used in the clinic.

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