Effects of Three Month Nasal Continuous Positive Airway Pressure Treatment on Electrocardiographic, Echocardiographic and Overnight Polysomnographic Parameters in Newly Diagnosed Moderate/Severe Obstructive Sleep Apnea Patients

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SUMMARY

The objective of the study was to determine the effects of nasal continuous positive airway pressure (nCPAP) therapy on left ventricular (LV) function and electrocardiographic parameters in newly diagnosed moderate/severe obstructive sleep apnea (OSA) patients without cardiovascular comorbidities and medical treatments. We examined 44 patients who underwent overnight polysomnography together with 24-hour Holter electrocardiography, cardiopulmonary exercise testing including heart rate recovery at 1 minute (HRR-1), echocardiography, surface electrocardiography, and those who were diagnosed with moderate/severe OSA apnea—hypopnea index ≥ 15. After 3 months of nCPAP treatment, the above-mentioned examinations were repeated. Forty-four patients completed the treatment period. Twelve weeks on effective nCPAP induced a significant increase in the mitral E/A ratio ($P = 0.001$), as well as reductions in isovolumic relaxation time ($P = 0.001$) and mitral deceleration time (DT) ($P = 0.002$). There were no significant differences in LV ejection fraction, LV mass index, and pulsed wave Doppler parameters. Mean heart rate was 79.2 ± 12.5 pulses/minute, maximum P-wave duration 117.5 ± 8.6 msec, P-wave dispersion (PWd) 54.6 ± 10.2 msec, corrected QT interval (QTc) 436.5 ± 40.5 msec, and QT dispersion (QTd) 46.3 ± 7.1 msec, which significantly decreased to 70.4 ± 9.6 pulses/minute ($P < 0.001$), 111.5 ± 8.7 msec ($P < 0.001$), 51.6 ± 8.9 msec ($P < 0.001$), 418.4 ± 31.2 msec ($P < 0.001$), and 33.8 ± 3.4 msec ($P < 0.001$), respectively. Exercise capacity at baseline determined as 10.5 ± 2.2 metabolic equivalents (METS) and HRR-1 (20.6 ± 11.7 bpm) significantly increased (12.1 ±1.5 METS and 27.4 ± 8.6 bpm). There was no significant difference in aortic root parameters. Three-month nCPAP therapy significantly increased LV shortening fraction, with no effect on systolic function or aortic root diameters and a positive effect on heart rate, PWd, HRR-1, QTc and QTd time following nCPAP therapy. (Int Heart J 2015; 56: 94-99)

Key words: Apnea-hypopnea index, Heart rate recovery time, Left ventricular function, P-wave dispersion, QT corrected interval

The prevalence of OSA is increasing, presently affecting at least 5% of the adult population[1,2] and is associated with several cardiovascular disorders and increased morbidity/mortality.[3] The physiologic links between obstructive airway events and cardiac disorders are multifactorial and are summarized in a consensus document from the American College of Cardiology and American Heart Association[4] as well as in several recent review articles.[5,6] Currently, nCPAP is a standard treatment in OSA.[7-9] The effects of nCPAP treatment on LV systolic[10,11] and diastolic function[12] as well as arrhythmic electrocardiographic parameters[13-16] were investigated in a number of studies. However, the effect of nCPAP treatment on moderate/severe OSA patients has not been fully investigated because of coexisting factors affecting these parameters such as obesity, hypertension, coronary artery disease, and rhythm disorders.

In the present study, we attempted to evaluate the effects of nCPAP treatment on electrocardiographic, echocardiographic, and overnight polysomnographic parameters in newly diagnosed well selected moderate/severe OSA patients without cardiovascular comorbidities and medical treatments.

METHODS

A total of 1112 patients who had undergone overnight polysomnography at our institute from November 2009 to March 2012 were eligible for inclusion in the present study. Among these patients, those who met the following conditions that might have had an influence on the results of echocardio-

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graphic and electrocardiographic parameters were excluded; chronic atrial fibrillation, second or third atrioventricular block, all bundle branch blocks, a permanent pacemaker, symptoms or signs of congestive heart failure, pericarditis, valvular heart disease, pulmonary emboli, abnormal thyroid function, cardiomyopathies, pulmonary hypertension, abnormal serum electrolyte values, neurological disease, use of digitalis, use of antiarrhythmic agents, use of beta-blockers, or use of calcium antagonists affecting heart rate (HR), including verapamil and diltiazem. Finally, from 654 patients who were clinically suspected of an OSA (apnea-hypopnea index, AHI ≥ 5) diagnosis; 46 patients without our exclusion parameters were prospectively analyzed using overnight polysomnography and cardiopulmonary exercise testing, including HRR-1, echocardiography, 24-hour Holter electrocardiography, surface electrocardiogram, and measurement of several metabolic parameters. Two patients were non-compliant with continuous positive airway pressure (CPAP) and discharged from the study group. Following 3 months of CPAP treatment, the above-mentioned examinations were repeated. During follow-up, we did not make any changes in the patients' medical treatment. The study protocol was approved by the ethics committee at the listed institutions, and informed consent was obtained from each patient prior to the study.

Polysomnography: All study participants underwent polysomnography at a sleep laboratory using a computerized polysomnography device (Compumedics, E series, 44 channel, Australia). Sixteen channels were used to document the following parameters: four channel electroencephalogram, electrooculogram, submental and leg electromyogram, electrocardiogram, nasal airflow using nasal pressure cannula, airflow at the nose and mouth (thermistors), chest and abdominal respiratory movement, oxygen saturation (pulse oximetry), snoring microphone, and body position. All studies were interpreted by a sleep specialist who was blinded to participant characteristics. Apnea was defined as the absence of airflow for 10 sec, and hypopnea was defined as a discernible reduction of airflow associated with a reduction in oxygen saturation by 4% from baseline. The AHI was defined as the average number of apneic and hypopneic events per sleep hour. Sleep staging was performed according to American Academy of Sleep Medicine criteria.[17] Moderate/severe OSA was defined as an AHI ≥ 15/ AHI ≥ 30.

Echocardiography: Transthoracic echocardiography was performed with a Vivid 4 system (GE Vingmed, Norway) using a 3.5 MHz transducer, according to the method of the American Society of Echocardiography.[18] The echocardiograms were reviewed and were excluded from the analysis if they had inadequate two-dimensional images. Imaging was performed by one experienced echocardiographer, who was unaware of which group the subjects belonged to.

LV end-diastolic and end-systolic diameters were obtained by M-mode measurements during two-dimensional echocardiography, according to the method of the American Society of Echocardiography.[19] The LV mass index was calculated according to Devereux, *et al*.[20] Systolic function was assessed by left ventricular shortening fraction (LVSF) and left ventricular ejection fraction (LVEF).[21]

LV diastolic function was assessed with both two-dimensional and Doppler echocardiography in accordance with the American Society of Echocardiography method. The mitral inflow pattern of the sample volume between the leaflet tips was recorded using the pulsed-wave Doppler technique. The following variables were measured: peak flow velocity in early diastole (E wave), peak velocity at atrial contraction (A wave), isovolumic relaxation time (IVRT), and mitral deceleration time (DT). The modified Bernoulli equation for tricuspid regurgitation was used to calculate estimated systolic pulmonary artery pressure.[22] Measurements were made in 3 cardiac cycles, and the average was calculated for 3 consecutive analyses.

Aortic root (Ao) diameter was measured according to the method of Stefanadis, *et al*.[23] Aortic diameters were measured with a caliper in systole and diastole as the distance between the trailing edge of the anterior aortic wall and the leading edge of the posterior aortic wall. Aortic systolic diameter (AoS) was measured at full opening of the aortic valve, and aortic diastolic diameter (AoD) was measured at the QRS peak. Three consecutive beats were measured and averaged. The aortic strain, expressed as the percentage change of the Ao, was calculated as follows: %Ao = 100 × (AoS – AoD)/ AoD. Aortic stiffness index = (SBP/DBP)/(AoS – AoD)/AoD, where DBP is diastolic blood pressure and SBP is systolic blood pressure.

All Doppler and echocardiographic recordings were stored on optical disks and were analyzed off-line by a single experienced investigator blinded to patient status and treatment.

Electrocardiographic analyses: All subjects underwent a 12-lead electrocardiography recording following a 20-min resting period in a supine position at a paper speed of 50 mm/sec and 2 mV/cm. The P-wave duration was measured manually in all simultaneously recorded 12 leads of the surface electrocardiography by two of the investigators unaware of the study hypothesis. The mean values for the three complexes recorded in each lead were calculated. The onset of the P-wave was defined as the point of the first visible upward departure from baseline for positive waveforms, and as the point of the first downward departure from baseline for negative waveforms. Return to baseline was considered to be the end of the P-wave. Maximum P-wave duration (Pmax) measured in any of the 12 leads of the surface electrocardiography was used as the longest atrial conduction time. The difference between Pmax and minimum P-wave duration (Pmin) was calculated and defined as P-wave dispersion (PWd = Pmax – Pmin).

The QT intervals were manually measured from the onset of the QRS to the end of the T wave, which was defined as the return to T-P isoelectric baseline, using a tangential method. Only monophasic well-defined T waves were accepted for measurement. When U waves were present, the QT was measured to the nadir of the curve between the T and U waves, with the aid of a tangent. If the end of the T wave was not reliably determined and the T waves were isoelectric or of low amplitude, then the lead was not included in the analyses. The measurements were obtained from precordial leads. The measurements were performed manually by an experienced observer blinded to the clinical data of the patients. Bazett's formula (corrected QT interval, $QTc = QT/\sqrt{RR}$) was used to obtain HR-corrected values of QT intervals.[24]

Holter electrocardiography: Together with overnight polysomnography, 24-hour Holter electrocardiography was performed using a digital recorder with 7 channels (DMS 300-7

Holter Recorder; MTM Multitechmed GmbH; Germany). Polysomnography and Holter electrocardiography were time-synchronized. Mean HRs during a 24-hour cycle of wakefulness and sleep were calculated. Based on the electroencephalogram, a sleep period was defined as a period from the start of sleep to the end of sleep.

Cardiopulmonary exercise test: Participants underwent cardiopulmonary exercise testing on a treadmill ergometer (Q-4500 Quinton, San Diego, CA, USA). Prior to testing, the tightness of the face mask was adjusted manually to avoid leakage. To allow familiarization with the test mode, the protocol started with a warm-up period of 3 minutes at a constant workload (speed 1.6 km/hour, inclination 6%), followed by an individualized ramp protocol. The ramp protocol was selected according to the patient's subjective estimate of physical fitness from a set of 12 ramp protocols for exercise capacities ranging from 4 to 17 METS (as estimated from speed and grade of the treadmill) to achieve a test duration of approximately 10 minutes (ramp portion of the test).[25] During the tests, subjects were verbally encouraged to exercise until exhaustion. A 12-lead electrocardiography was recorded continuously, and blood pressure (by indirect arm-cuff sphygmomanometry) was assessed every 2 minutes. For assessment of the chronotropic response, peak HR was expressed as the percent of age-predicted HR, where age-predicted HR was defined as '220 – age' in years. In addition, the percentage of HR reserve used was calculated, and it was defined as (peak HR - HR at rest) / (age-predicted HR - HR at rest) × 100.[26] Following the achievement of peak workload, participants spent at least 2 minutes in a cool-down period on the treadmill at baseline workload (speed 1.6 km/hour, inclination 6%), and HRR-1 data were obtained. HRR-1 was defined as the difference between HR at peak exercise and after 1 minute of recovery.[27]

Statistical analyses: Data are presented as the mean ± standard deviation (SD). Categorical variables are presented as numbers and percentages. The Shapiro-Wilk test was used to evaluate whether the distribution of variables was normal. Paired *t*-test analyses were used to compare the variable at baseline and following nCPAP therapy. A two-tailed $P < 0.05$ was considered significant. SPSS 18.0 software was used for statistical analyses (Version 18, SPSS Inc., Chicago, IL, USA).

Table I. Baseline Characteristics of the Study Population

Variable	
Patients*, *n*	44
Age, years	58.7 ± 12.8
Male/Female, *n*	12/32
Diabetes mellitus, *n* (%)	13 (29.5)
Smoking, *n* (%)	25 (56.8)
Dyslipidemia, *n* (%)	14 (31.8)
Body mass index, kg/m^2	28.5 ± 5.3

Data are presented as mean ± SD or number (*n*) (%). *Patients with apnea-hypopnea index ≥ 15.

Table II. Overnight Polysomnographic Findings and Blood Pressures of the Study Group Before and After CPAP Treatment

	Before CPAP	After CPAP	*P*
Apnea-hypopnea index	44.2 ± 13.9	37.8 ± 16.7	< 0.001
Epworth sleepness scale	8.5 ± 5.1	5.8 ± 2.9	< 0.001
Mean O_{2night} saturation, %	89.2 ± 3.2	91.9 ± 2.4	< 0.001
Mean O_{2day} saturation, %	95.5 ± 2.5	96.9 ± 1.4	0.003
Minimum O_2 saturation, %	78.9 ± 7.2	79.6 ± 5.7	< 0.001
Total sleep time, hours	6.4 ± 0.4	6.1 ± 0.8	< 0.001
Systolic blood pressure, mmHg	133.5 ± 6.1	122.4 ± 9.3	0.002
Diastolic blood pressure, mmHg	86.4 ± 3.3	78.8 ± 10.4	0.001

Data are presented as the mean ± SD or (%).

Table III. Electrocardiographic Parameters of the Patients With Obstructive Sleep Apnea Before and After CPAP Treatment

	Before CPAP	After CPAP	*P*
Heart rate (pulse/minute)	79.2 ± 12.5	70.4 ± 9.6	< 0.001
Pmax, ms	117.5 ± 8.6	111.5 ± 8.7	< 0.001
PWd, ms	54.6 ± 10.2	51.6 ± 8.9	< 0.001
Pmin, ms	46.1 ± 8.4	49.0 ± 7.8	0.064
QTc, ms	436.5 ± 40.5	418.4 ± 31.2	< 0.001
QTd, ms	46.3 ± 7.1	33.8 ± 3.4	< 0.001
HRR-1, bpm	20.6 ± 11.7	27.4 ± 8.6	< 0.001
Exercise capacity, METS	10.5 ± 2.2	12.1 ± 1.5	0.001

HRR-1 indicates heart rate recovery at 1 minute; METS, metabolic equivalents; Pmax, maximum p-wave duration; Pmin, minimum p-wave duration; PWd, P-wave dispersion; QTc, corrected QT interval; and QTd, QT dispersion. Data are presented as mean ± SD.

RESULTS

Forty-four of 46 patients were compliant with nCPAP therapy, which was defined as ≥ 3.5 hours of nightly use. At the end of the study protocol, the mean daily CPAP usage of 44 patients was 6.1 ± 1.3 hours per night. There were 12 male patients and 32 female patients. None of the compliant patients used alcohol, and 56.8% of them were smokers. Thirteen of 44 patients were diabetic and 14 of the 44 patients were dyslipidemic. The basic characteristics of the compliant OSA patients are shown in Table I. The mean age of the patients was 58.7 ± 12.8 and the mean body mass index was 28.5 ± 5.3 kg/m^2.

Overnight polysomnographic parameters and blood pressure: Baseline overnight polysomnographic findings and blood pressure values of the study group are shown in Table II. The mean AHI and Epworth sleepiness scale were 44.2 ± 13.9 per hour and 8.5 ± 5.1 per hour, respectively. Following 3 months of nCPAP treatment, AHI (37.8 ± 16.7 per hour, $P < 0.001$) and the Epworth sleepiness scale (5.8 ± 2.9 per hour, $P < 0.001$) decreased significantly.

Mean O_{2night}, O_{2day}, and minimum O_2 saturation were 89.2 ± 3.2, 95.5 ± 2.5, and 78.9 ± 7.2 respectively. Following treatment, O_2 saturation (night, daytime and minimum) (91.9 ± 2.4, 96.9 ± 1.4, 79.6 ± 5.7) increased significantly.

We also observed a significant increase in systolic (133.5 ± 6.1 versus 122.4 ± 9.3 mmHg, $P = 0.002$) and diastolic (86.4 ± 3.3 versus 78.8 ± 10.4 mmHg, $P = 0.001$) blood pressure following 12 weeks of nCPAP treatment.

Electrocardiographic parameters: Electrocardiographic parameters of the study group are shown in Table III. The mean HR at baseline (70.2 ± 12.5 pulses/minute) significantly decreased following nCPAP therapy (70.4 ± 9.6 pulses/minute) ($P < 0.001$). Pmax at baseline (117.5 ± 8.6 msec) significantly decreased following nCPAP therapy (111.5 ± 8.7 msec) ($P < 0.001$). There was no significant change in mean Pmin follow-

Table IV. Left Ventricular Systolic, Diastolic and Aortic Root Parameters of the Study Group Before and After CPAP Treatment

	Before CPAP	After CPAP	*P*
LVEF, %	69.5 ± 5	68.7 ± 5.3	0.883
LV mass index, g/m^2	135 ± 36	133 ± 29	0.113
LVEDD, mm	42.1 ± 5.3	40.8 ± 4.2	< 0.001
LVESD, mm	28.9 ± 4.8	27.1 ± 3.5	< 0.001
Left atrium, mm	33.2 ± 4.1	32.1 ± 3.6	< 0.001
IVS, mm	1.2 ± 0.2	1.1 ± 0.1	< 0.001
PW, mm	1.0 ± 0.1	1.2 ± 0.3	0.537
Mitral E, cm/s	0.9 ± 0.2	1.1 ± 0.2	< 0.001
Mitral A, cm/s	0.9 ± 0.2	0.8 ± 0.1	0.056
Peak E/A ratio	1.0 ± 0.2	1.4 ± 0.1	0.001
IVRT, ms	81 ± 18	69 ± 19	0.001
IVCT, ms	47 ± 15	44 ± 17	0.121
DT, ms	212 ± 31	195 ± 26	0.002
Estimated sPaP, mmHg,	22.1 ± 8.4	15.8 ± 4.8	< 0.001
Ao systolic diameter, mm	32.9 ± 3.7	30.3 ± 3.3	0.342
Ao diastolic diameter, mm	30.3 ± 2.5	29.7 ± 2.1	0.435

Data are presented as the mean ± SD. A indicates late diastolic peak flow velocity; Ao, aortic root; DT, mitral deceleration time; E, early diastolic peak flow velocity; IVCT, isovolumic contraction time; IVRT, isovolumic relaxation time; IVS, interventricular septum; LVEDD, left ventricular end diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end systolic diameter; PW, posterior wall; and sPAP, systolic pulmonary artery pressure.

ing nCPAP therapy. PWd at baseline (54.6 ± 10.2 msec) significantly decreased following 3 months of nCPAP therapy (51.6 ± 8.9 msec, $P < 0.001$). The QTc (436.5 ± 40.5 msec) and QTd (46.3 ± 7.1 msec) at baseline significantly decreased following nCPAP treatment (418.4 ± 31.2 msec, $P < 0.001$ and 33.8 ± 3.4 msec, $P < 0.001$). The exercise capacity at baseline determined as 10.5 ± 2.2 METS and HRR1-1 (20.6 ± 11.7 bpm) significantly increased (12.1 ± 1.5 METS and 27.4 ± 8.6 bpm) following 3 months of nCPAP therapy.

Echocardiographic parameters: At baseline, mean LVEF of the study group was 69.5 ± 5.0 (%). Following treatment, no significant difference existed for LVEF ($P = 0.883$), LV mass index (g/m^2) ($P = 0.113$), or posterior wall diastolic thickness ($P = 0.537$). Following 12 weeks of nCPAP treatment, a significant increase in the E/A ratio (1.0 ± 0.2 versus 1.4 ± 0.1, $P = 0.001$) and a significant reduction in IVRT (81 ± 18 versus 69 ± 19, $P = 0.001$) and DT (212 ± 31 versus 195 ± 26, $P = 0.002$) were observed.

AoS and AoD at baseline were 32.9 ± 3.7 mm and 30.3 ± 2.5 mm. There were no significant differences following 3 months of nCPAP treatment in the aortic root parameters (30.3 ± 3.3 mm, $P = 0.342$ and 29.7 ± 2.1 mm, $P = 0.435$). Estimated systolic pulmonary artery pressure decreased significantly following nCPAP treatment (22.1 ± 8.4 mmHg versus 15.8 ± 4.8 mmHg, $P < 0.001$).

Discussion

This study investigated the effects of nCPAP therapy on LV function and electrocardiographic parameters in moderate/severe OSA patients. In our last two trials with this well selected group, we demonstrated that HR, PWd, HRR-1, and QTc are correlated with the severity of obstructive sleep apnea syndrome (OSAS)[28] and also we published the association between OSA and aortic root parameters and LV functions.[29] The main findings of the present study are that abnormalities in diastolic function, arrhythmic electrocardiographic parameters such as PWd, QTc, QTd, and HRR-1 and deteriorated overnight polysomnographic parameters could be reversible, at least in part, with nCPAP therapy.

Previous studies have demonstrated a positive association between elevated HR at rest and adverse cardiovascular events in both the general population and the population with cardiovascular disease.[30-36] Few studies have investigated the influence of severity of OSA on HR in patients with OSA. Sumi, *et al*[16] reported that mean HR during 24 hours correlated positively with AHI in 62 patients with OSA and that mean HR in the daytime (06:00-22:00 hour) and night-time (22:00-06:00 hour) were significantly reduced following 3 or 4 days of nCPAP treatment in those patients. However, their study included patients using beta-blockers, which can affect mean HR. Therefore, it is unclear whether mean HR is independently associated with the severity of OSA in patients with OSA. In addition, the long-term effects of nCPAP therapy on mean HR remain unclear. In our study, we found a significant decrease in mean HR in moderate/severe OSA patients following 3 months of nCPAP therapy. Our study group is well selected without cardiovascular comorbidities and medical treatment, which can affect heart rate and nCPAP therapy is required for longer periods than the above-mentioned trial.

Increased activation of the sympathetic nervous system due to nocturnal hypopnea and apnea episodes and arousal reactions, respectively, is a pathophysiological hallmark of OSAS,[37-39] which has been shown to be improved following therapy with CPAP ventilation.[40] HRR-1 following exercise termination is a simple and readily available measure of vagal tone that has been found to be of high prognostic value in patients referred for exercise testing for evaluation of coronary artery disease,[27] in patients with established severe coronary artery disease,[41] and in patients with congestive heart failure.[42] Maeder, *et al*[15] concluded that the severity of OSAS expressed as higher AHI is independently associated with lower HRR-1. The mean AHI was similar in both severe OSAS groups (57.8 ± 22.5 versus 50 ± 11). In our moderate/severe OSAS patients, we found a significant improvement in HRR-1 following 12 weeks of nCPAP treatment as in previous studies.

The prolongation of intra-atrial and inter-atrial conduction time and the inhomogeneous propagation of sinus impulses are well-known electrophysiological characteristics of the atrium prone to fibrillate and have been evaluated using two simple electrocardiography markers, Pmax and PWd.[43-45] Increases in the P-wave duration and PWd have been used as predictors of atrial fibrillation development in various clinical settings.[44-49] Can, *et al* found that PWd was greater in patients with OSA than patients without OSA and is associated with severity of the disease.[13] To the best of our knowledge, the effects of nCPAP treatment on P-wave duration and PWd have not been evaluated in patients with OSA. In our study, we observed a significant decrease in Pmax and PWd following 3 months of nCPAP therapy and there were no significant changes in mean Pmin following nCPAP therapy.

QTd reflects inhomogeneity of repolarization. Delayed cardiac repolarization leading to prolongation of the QT interval is a well-characterized precursor of arrhythmias. The QTd is increased in patients with a prior myocardial infarction, who

have a susceptibility to ventricular tachyarrhythmias, most obviously by re-entry mechanisms.[50] Roche, *et al*[51] reported a significant alteration in the relationship between QT length and HR during CPAP treatment, but they did not find any changes in static repolarization parameters (QT, RT, QTc, and RTc). On the other hand, Yamashita, *et al*[52] observed more severe autonomic nervous dysfunction and increased QTd in hypertensive patients with OSA during episodes of apneas and hypopneas compared to those without OSA, and suggested that OSA might be associated with future development of cardiac events. Also, in our study we observed a significant decrease in QTc and QTd after nCPAP treatment in moderate/severe OSA patients.

Changes in echocardiographic diastolic parameters following nCPAP therapy have been reported in several studies, some with different patient selection criteria.[53-55] The effects of cardioactive drugs, hypertension, obesity, or possible disease that affect diastolic function could have affected the results in some of the studies.[56-59] Arias, *et al* investigated the effects of nCPAP therapy on LV diastolic function and concluded that chronic application of nCPAP could avoid the progression of diastolic abnormalities, and indeed, it might reserve these alterations, at least in the initial stages prior to severe structural changes developing.[12] In our study, we observed a significant improvement in diastolic function following 12 weeks of nCPAP therapy in moderate/severe untreated OSA patients with/without diastolic dysfunction.

The positive effect of nCPAP treatment on LV systolic function in heart failure patients has been well reported in previous studies.[60,61] At baseline, the mean LVEF of our study group was normal. Following treatment, we did not observe any significant difference for LVEF ($P = 0.883$). We can conclude that nCPAP treatment has a positive effect on deteriorated LV function, but no effect on patients with normal LV function.

Conclusion: We demonstrated that in patients with moderate/severe OSA, 12 weeks of nCPAP therapy significantly increased LV diastolic function and had no effect on systolic function and aortic root diameters. A positive effect on HR, PWd, HRR-1, QTc, and QTd was found following effective nCPAP therapy.

Disclosure

The authors declare that they have no conflict of interests.

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