Effects of the Continuous Positive Airway Pressure on the Airways of Patients with Chronic Heart Failure

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Abstract

Background: Heart failure can present with asymptomatic dysfunction at decompensation, with limitations and decrease in the productive capacity. The Continuous Positive Airway Pressure (CPAP) is a non-pharmacological means to decrease afterload.

Objective: To analyze the effects of CPAP (10 cmH_2O), for 30 days in patients with chronic heart failure.

Methods: We assessed 10 patients with heart failure (6 males, 4 females) of several etiologies, with a mean age of 54 ± 14 years, with a BMI of 21 ± 0.04 kg/m². The therapy was applied for 60 min., 5 times a week for 30 days, during the daytime. The echocardiogram and the ergospirometry were analyzed, before and 30 days after the therapy.

Results: There was a 19.59% increase in the left ventricular ejection fraction (LVEF): 23.9 ± 8.91 *vs* 27.65 ± 9.56%; p = 0.045. At the ergospirometry, the exercise time (ET) showed a significant increase from 547 ± 151.319 *vs* 700 ± 293.990 sec., p = 0.02; oxygen consumption (VO_2) was 9.59 ± 6.1 *vs* 4.51 ± 2.67 ml.kg^{-1}.min.$^{-1}$, p = 0.01, whereas the carbon dioxide production (VCO_2) at rest (9.85 ± 4.38 *vs* 6.44 ± 2.88 ml.kg^{-1}.min.$^{-1}$, p = 0.03) decreased.

Conclusion: The CPAP resulted in an increase in the LVEF and ET, decreased the oxygen consumption and the carbon dioxide production at rest. (Arq Bras Cardiol. 2010; [online]. ahead print, PP.0-0)

Key words: Continuous positive airway pressure; heart failure; echocardiography; oxygen consumption.

Introduction

Heart failure (HF) is a syndrome of which presentation can vary from the acute to the slowly progressive form, from an asymptomatic dysfunction to a state of intense decompensation, resulting in the onset of functional limitations that have an unfavorable effect on the patients' functional capacity. This syndrome has high hospitalization and medical attention costs, in addition to resulting in limitations to the quality of life, adding a production deficit. Due to the increasing number of cases, it became mandatory to create units specialized in HF, with positive results on the patient's functional capacity as well as cost reduction, such as a decrease in the number of rehospitalizations[1-3].

According to the II Guideline for the diagnosis and treatment of chronic heart failure (CHF), the treatment principles are based not only on the pharmacological management, but also on the non-pharmacological treatment, the surgical interventions and the use of pacemakers[2]. The continuous positive airway pressure (CPAP) is included among the non-pharmacological treatments. The daily clinical practice shows that the use of CPAP decreases dyspnea during exercise and has favorable results on left ventricular function[4].

However, there has been scarce scientific evidence of these benefits. This study was designed with the objective of scientifically demonstrating the results of the use of CPAP on CHF.

Patients and methods

The sample consisted of patients with a clinical-laboratory diagnosis of CHF, who were treated at the Heart Failure Outpatient Clinic of the Service of Cardiology of *Hospital Universitário Clementino Fraga Filho* (HUCFF) - UFRJ. This is a prospective, longitudinal case study with intervention, of which sample consisted of 10 patients, who having met the inclusion criteria, were submitted to CPAP therapy (10 cmH_2O) for 60 minutes, 5 times a week for a month and who were monitored before, during and after the procedure. The patients were assessed at the start of the treatment and reassessed 30 days later by echocardiogram and ergospirometry. The inclusion criteria were: patients with a clinical-laboratory diagnosis of CHF, ejection fraction < 40%, optimized medical treatment for at and at least three months of treatment duration. The exclusion criteria were: history of prior myocardial infarction

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within a 3-month period, unstable angina, clinical evidence of significant tricuspid regurgitation, history of rheumatic valvular disease or primary structural valvular abnormalities detected at the two-dimensional echocardiogram, primary obstructive pulmonary disease defined by spirometry and lastly, to be undergoing respiratory physical therapy.

After reading and signing the Informed Consent Free and Informed Consent Form (FICF), which had been approved by the Research Ethics Committee (REC) of HUCFF-UFRJ, protocol# 086/06 - CEP., the patients were submitted to the transthoracic two-dimensional echocardiography with color Doppler (ECTT) (SIEMENS - SONOLINE G60S, USA), calculated through the Simpson's method for left ventricular ejection fraction (LVEF) estimate. The examination was carried out using the conventional approach, by a single observer before and after the CPAP therapy. During the analysis of the echocardiogram after the CPAP therapy, the observer was blinded to the results of the pre-therapy examination. With the objective of acquiring more reliable measurements, the left-ventricular outflow tract diameter was obtained.

The patients were submitted to a physical therapy assessment (physical examination, weight, height, body mass index (BMI), pulmonary function test. Weight and height were measured using a Fillizola scale made in Brazil. The BMI was obtained by dividing the weight by the squared height (weight/height^2). The spirometry was carried out with the objective of excluding pneumopathies, with an excluir foi realizada EasyOne™ Model 2001 equipment, according to the guidelines of the American Thoracic Society and European Respiratory Society (ATS/ERS)[5], with patients in a comfortable sitting position. The cardiopulmonary stress test with direct oxygen measurement (ergospirometry) was carried out on a treadmill feito (ECAFIX EG700.2 - BR), using the Naughton protocol and the analysis of expired gases was carried out in a VO_{2000} equipment (Inbrasport - BR). The electrocardiographic monitoring was carried out using the electrocardiogram equipment (Cardio Control - BR). All patients were previously advised before undergoing the test and the ergospirometry measurements were extracted with medical follow-up at the Laboratory of Exercise Physiology (LABOFISE) of the School of Physical Education and Sports of *Universidade Federal do Rio de Janeiro* (EEFD-UFRJ).

The treatment with CPAP was carried out with the positive pressure device (REMstar™ PLUS Systema CPAP (Respironics INC™ EUA), in the continuous mode, with a face mask. During this procedure, the blood pressure (BP) was indirectly monitored (Tycos sphygmomanometer - USA), as well as the heart rate (HR) (Polar Sport Tester™, Finland) and the respiratory rate (RR), which was assessed as the number of respiratory incursions per minute. The patients were in the sitting position with their backs comfortably supported and the feet were placed on the floor. The equipment was calibrated before measurements were obtained.

Statistical analysis

The statistical analysis was carried out with the software program SPSS for Windows release 15.0 and the variables with normal distribution were analyzed through paired Student's *t* test. For the variables that were not considered as having a normal distribution, the Wilcoxon's test was used. The level of significance was set at 5%.

Results

The study was carried out with 10 (ten) patients from the HF Outpatient Clinic of HUCFF-UFRJ, with a diagnosis of chronic heart failure (CHF), with the following etiologies: hypertensive (50%), alcoholic (20%), idiopathic (20%) and peripartoum (10%), with a mean age of 54 ± 14 years, with a slight predominance of the male sex (6) in comparison to the female sex (4). The patients presented a mean BMI value of 21 ± 0.04 kg/m^2. Table 1 shows the demographic data of the patients, the functional class, the CHF etiology and medications being used (Table 1).

Analysis of the echocardiographic variables

The left ventricular ejection fraction (LVEF) values (Table 2) showed a significant increase of 19.69% after one month of treatment with CPAP (10 cmH_2O) for 60 min/day, with p = 0.045 (Figure 1). The other variables did not show a statistically significant difference.

Analysis of the ergospirometric variables

At the assessment of the functional capacity (Table 3), the treatment with CPAP for 30 days resulted in an increase in

Table 1 - **Patients' characteristics (n = 10)**

Variables	Patients (n = 10)
Age (years)	54 ± 14 yrs
Sex	male = 6 / female = 4
Weight (kg)	69 ± 17 kg
Height (cm)	162 ± 11 cm
Functional class (NYHA)	
I	3
II	7
Etiology	
Idiopathic	2
Hypertensive	5
Alcoholic	2
Peripartoum	1
Time of diagnosis	5 ± 1.83 yrs
Medications	
ACEI	6
Beta-Blocker	7
Diuretics	10
Digitalis	8
Anti-arrhythmic	1
Anti-hypertensive	7

The age, weight and height variables correspond to the means and standard deviations (SD) of the 10 patients; ACEI - angiotensin-converting enzyme inhibitor.

Table 2 - **Echocardiographic variables**

Variables	Pre-treatment	Post-treatment	p Value
Ao (cm)	3.3 ± 0.5	3.26 ± 33	0.17
LA (cm)	4.45 ± 1.04	4.4 ± 1.08	0.33
EDV (cm)	7.93 ± 1.16	7.95 ± 1.13	0.44
ESV (cm)	6.88 ± 0.08	6.83 ± 0.82	0.28
LVEF (%)	23.9 ± 8.91	27.65 ± 9.56	0.045*

The values correspond to the means and SD of the 10 patients; Ao - aorta; LA - left atrium; EDV - end-diastolic volume; ESV - end-systolic volume; LVEF - left ventricular ejection fraction; (*) statistically significant difference.

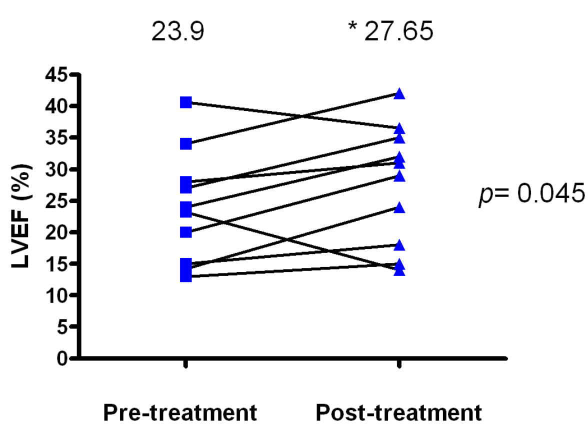

Figure 1 - *Left ventricular ejection fraction (LVEF) in the pre and post-treatment moments. The values correspond to the means and SD of the 10 patients; (*) significant difference (p = 0.045).*

Table 3 - **Ergospirometric variables**

Variables	Pre-treatment	Post-treatment	p Value
ET - sec.	547.0 ± 151.3	700.3 ± 294.0	0.02*
LV (STPD) rest (l.min.$^{-1}$)	16.19 ± 15.02	12.47 ± 3.78	0.09
LV (STPD) peak (l.min.$^{-1}$)	34.17 ± 11.2	33.90 ± 7.79	0.29
VO_2 rest (ml.kg^{-1}.min.$^{-1}$)	9.59 ± 4.51	6.1 ± 2.67	0.01*
VO_2 peak (ml.kg^{-1}.min.$^{-1}$)	18.73 ± 7.34	17.08 ± 2.32	0.25
VCO_2 rest (ml.kg^{-1}.min.$^{-1}$)	9.85 ± 4.38	6.44 ± 2.88	0.03*
VCO_2 peak (ml.kg^{-1}.min.$^{-1}$)	21.96 ± 10.9	20.79 ± 5.24	0.30
LV/VO_2	30.56 ± 11.46	28.57 ± 4.65	0.40
LV/VCO_2	30.85 ± 6.12	30.00 ± 2.50	0.27
Borg	15 ± 2.51	16 ± 3.09	0.23
R	0.93 ± 0.22	0.95 ± 0.13	0.41
SAP (mmHg)	133 ± 24.97	136.6 ± 27.73	0.24
DAP (mmHg)	79 ± 15.95	84 ± 10.75	0.21
HR (bpm)	135.3 ± 18.94	136.9 ± 15.95	0.38

The values correspond to the means and SD of the 10 patients; ET - exercise time in seconds; LV (STPD) rest - pulmonary ventilation in l.min.$^{-1}$ at rest; LV (STPD) peak - pulmonary ventilation in l.min.$^{-1}$, at the exercise peak; VO_2 rest - oxygen consumption in ml.kg.min.$^{-1}$, at rest; VO_2 peak - oxygen consumption in ml.kg. min.$^{-1}$, at the exercise peak; VCO_2 rest - carbon dioxide production in ml.kg^{-1}.min.$^{-1}$, at rest; VCO_2 peak - carbon dioxide production in ml.kg^{-1}.min.$^{-1}$, at the exercise peak; LV/VO_2 - respiratory equivalent of VO_2, LV/VCO_2 - respiratory equivalent of VCO_2; Borg - the rating of perceived exertion using Borg's scale; R - gas exchange ratio; SAP - systolic arterial pressure in mmHg; PAD - diastolic arterial pressure in mmHg; HR - heart rate in bpm. (*)statistical differences : Tex- sec. (p = 0.02), VO_2 rest (p = 0.01), VCO_2 rest (p = 0.03).

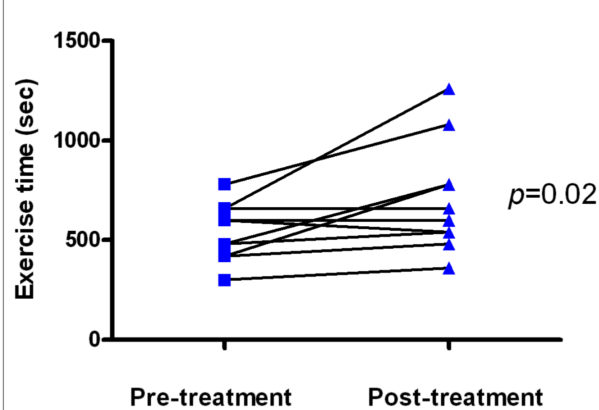

Figure 2 - *Exercise time at the pre and post-treatment moments. The values correspond to the means and SD of the 10 patients; (*) significant difference: p = 0.02.*

the time of exercise of 547 ± 151.3 *vs* 700 ± 293.9 sec with $p = 0.02$ (Figure 2). At rest, there was a significant decrease in the following variables: VO_2 (9.59 ± 4.51 *vs* 6.1 ± 2.67 ml.kg^{-1}.min^{-1}; $p = 0.01$) (Figure 3) and VCO_2 (9.85 ± 4.38 *vs* 6.44 ± 2.88 ml.kg^{-1}.min^{-1}; $p = 0.03$) (Figure 4).

Discussion

The present study assessesevaluates the use of CPAP in patients with clinically compensated CHF through echocardiography and ergospirometry (functional capacity), identifying the physiological and functional outcomes of this procedure.

After a one-month treatment, the patients started to presentshowed significant variations in left ventricular ejection fraction (LVEF), with a relative increase of 19.69%. This result is supported by the finding of the study by Mallone et al (1991)[6], who obtained an increase of 32% in LVEF when using CPAP in patients with dilated cardiomyopathy and sleep apnea for 4 weeks, during the night[7-9]. Our study also confirms the findings by Kaneko et al[10] who, after a one-month nocturnal CPAP treatment, observed a relative increase of 35% in LVEF in patients with HF and Obstructive Sleep Apnea (OSA). Apneia Mansfield et al[11] and Egea et al[12] obtained an increase of 14 % and 11%, respectively, in the LVEF of patients with CHF and sleep apnea after CPAP therapy. In these studies, the LVEF increase was more pronounced in patients with LVEF > 30%. According to the authors, patients with lower

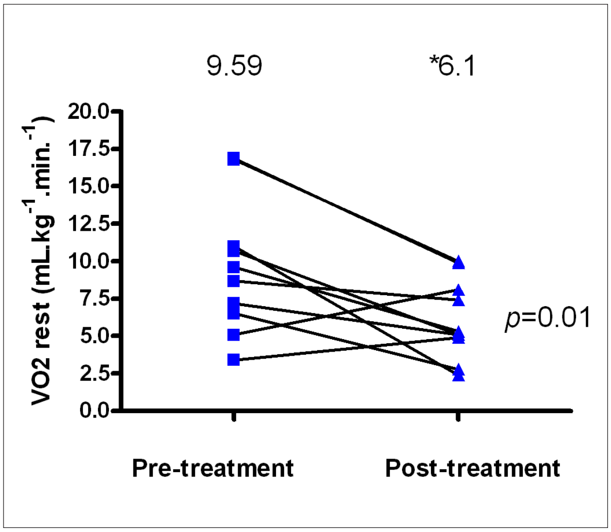

Figure 3 - Oxygen consumption at rest at the pre and post-treatment moments. The values correspond to the means and SD of the 10 patients; (*) significant difference p = 0.01.

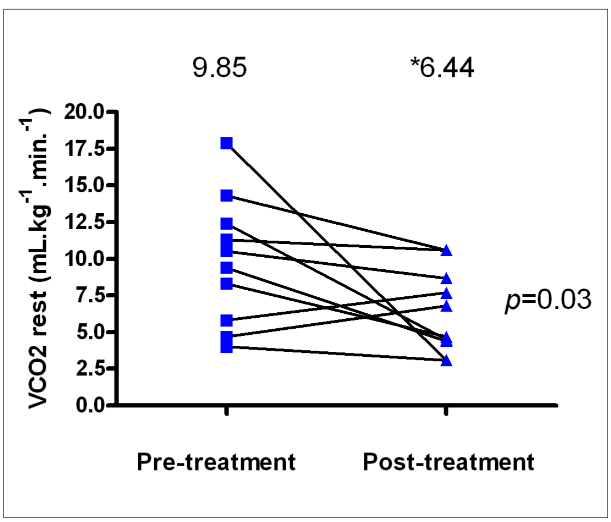

Figure 4 - Carbon dioxide consumption at the pre and post-treatment moments. The values correspond to the means and SD of the 10 patients; (*) significant difference p = 0.03.

LVEF have a lower cardiac contractile reserve and therefore, are incapable of increasing their systolic function. In all aforementioned studies, the patients were NYHA classes II to IV. The differentiated increase in LVEF that was observed in our study can be justified by the fact that the analyzed patients were NYHA classes I and II, in addition to the fact that they were stable and receiving adequate medication.

It has been well postulated that the CPAP therapy has its effect on cardiac function by increasing the intrathoracic pressure and reducing preload and afterload[13], decreasing mitral regurgitation, the blood concentrationlevels of ANP and improving LVEF[14] and decreasing the workload and respiratory exertion[15]. In addition to decrease in the left ventricular (LV) afterload, Metha et al[16] observed a decrease in the right ventricular afterload after using CPAP for 30 minutes in patients with dilated cardiomyopathy. The increase in left ventricular function in patients with CHF after a short-term therapy with CPAP has been attributed to the increase in intrathoracic pressure and the decrease in the LV transmural pressure[11].

The choice of levels of the positive-end expiratory pressure (PEEP) used in the present study (10 cmH_2O) was based on results of literature[8,14,15], supported by the fact that PEEP values of 5 cmH_2O do not present significant effects, whereas the hemodynamic effects (decrease in venous return and increase in cardiac output) were only observed when PEEP values were ≥ 10 cmH_2O[17,18].

In addition to the ejection fraction, another variable that showed improvement was the exercise time (ET), which is directly associated with the functional capacity alterations. This is an important assessment, as the walking test can be used to monitor the mortality of patients with CHF[19]. The use of CPAP results in an increase in the distance walked by patients with stable CHF, even in situations of acute effect (30 minutes) and with low PEEP pressures (3.4 to 6 cmH_2O)[20]. The measurement of peak oxygen uptake at exertion (VO_2 peak) is the most important procedure for functional assessment and prognosis stratification in CHF, in addition to being used as a cutoff for heart transplantation (14 ml.kg^{-1}.min^{-1}). VO_2 peak values < 10 or > 18 ml.kg^{-1}.min^{-1}, demonstrate, equally, a bad prognosis[21].

The patients in the present study presented a decrease in VO_2 peak values at the end of the treatment with CPAP, but such decrease was not statistically significant. This fact might be associated with the small sample size, considering that before the treatment with CPAP, 4 patients (40%) presented VO_2 peak values < 14 ml.kg^{-1}.min^{-1}, whereas after the CPAP therapy (one month) only one patient (10%) presented peak values < 14 ml.kg^{-1}.min^{-1}, which expresses a non-negligible result, as it represents an improvement in the response of the studied group.

The pharmacological therapy with beta-blockers in these patients is consensual[22] and it influences the VO_2 peak values. The correct use of the medicationsoptimizes drugs therapy prescribed by assistant physician directly interferes with the obtained results, considering that its adequate use decreases mortality in patients with LV failure. This fact was confirmed by O'Neill et al[23] after the analysis 2,105 patients that underwent a cardiopulmonary test, divided in two groups: beta-blocker use (n = 909) and non-beta-blocker use (n = 1,196). The patients treated with beta-blocker presented a high survival rate, with functional capacity that exceeded the VO_2 peak value of 10 ml.kg^{-1}.min^{-1}.

In our study, 70% of the patients had received a prescription forwere taking beta-blockers; however, as the correct intake of medication was not directly inspected, we cannot affirm that the patients used the medication correctly.

The use of CPAP brought benefits to the studied patients, as they presented a significant decrease in VO_2 at rest in relation to VO_2 during exercise.

This decrease in oxygen consumption was also observed when VO_2 was estimated in metabolic equivalents (METs)[24]. The values calculated pre and post-treatment with CPAP were, respectively, 2.74 to 1.74 METs at rest and 5.35 to 4.88 METs at exercise peak.

The presence of dyspnea and fatigue after small exertion, as well as at rest, is an important clinical sign. At the ergospirometry, the assessment of the ventilatory parameters presented during the exercise is represented by the ventilatory index (VI) that analyzes the progression, as well as the stratification of HF[25,26].

The VI values at the exercise peak and at rest, assessed before treatment, presented a difference of 0.95, without statistical significance. There was difference of 0.77 at the post-treatment period (p = 0.02). We can affirm that there is a tendency towards risk reduction in this group after CPAP therapy, based on the results by Jankowska et al[27], who demonstrated that the prognosis in patients with CHF with a high VI is significantly worse.

Chronotropic incompetence is defined as the incapacity of the heart to achieve 80% of the predicted heart rate (HR) according to Astrand's formula[28]. The HR values obtained during and after the cardiopulmonary test are excellent tools to perform the prognostic analysis of mortality in routine clinical practice[29-,30], in addition to being a marker of reduced parasympathetic activity (chronotropic incompetence), as well as a predictor of mortality[31].

The heart rate values at the exercise peak as well as at the one-minute heart rate recovery in the pre-treatment period presented a difference of 19.5 bpm (135 ± 18.94 to 115.8 ± 19,1bpm) and in the post-treatment period, 18.9 bpm (136.9 ± 15,95 to 118 ± 17.93 bpm), with no statistical difference, which suggests that these patients presented reduced parasympathetic activity.

Bilsel et al[32] stated that the chronotropic incompetence is common in these patients and that the chronotropic index (CI) is an important prognostic marker in healthy individuals. In our study, CI values in the pre and post-treatment periods were 0.40 and 0.33. That indicates chronotropic incompetence, considering that normal values are ≤ 0,80 [31].

The hemodynamic responses during exercise can determine the prognosis of patients with CHF, allowing the risk stratification to be determined (high, medium or low cardiovascular risk), as well as their functional capacity[33].

The results observed between the VO_2 peak x SBP peak (circulatory power), although not statistically significant, were higher than the ones observed in the post-treatment period (2,535.10 x 2,362.76 $mmHg.mlO_2.min^{-1}.kg^{-2}$), with a difference of 172.34. This fact has clinical relevance, as possui Cohen-Solal et al[34], when assessing the circulatory power of 28 patients who died (2,567 ± 984 $mmHg.mlO_2.min^{-1}.kg^{-2}$), 32 patients who underwent a heart transplant (2,402 ± 843 $mmHg.mlO_2.min^{-1}.kg^{-2}$) and 115 patients that survived (3,573 ± 1,273 $mmHg.mlO_2.min^{-1}.kg^{-2}$), concluded that patients with low circulatory power present a particularly poor prognosis. In the present study, the CPAP therapy caused a decrease in the circulatory power. When compared to the aforementioned values, our patients presented values close to those of patients that underwent a heart transplant.

The blood pressure, heart rate and respiratory rate were monitored during the CPAP therapy and there was no significant difference when values at rest were compared to values after the end of the therapy.

The results of the present study demonstrated that the CPAP therapy can be a good therapeutic therapeutic resourceoption for patients with compensated chronic heart failure treated at the outpatient clinic; the therapy adds benefits to the pharmacological treatment and becomes an excellent method to prevent complications. The CPAP therapy in these patients, in addition to increasing the LVEF, resulted in the improvement of functional capacity, through an increase in exercise time. None of the patients presented clinical-hemodynamic complications or adverse reactions to the technique during the treatment, which demonstrates the therapy safety.

Conclusion

The use of CPAP (10 cmH_2O) daily for 60 min. during 1 month, increased the LVEF in the studied patients and decreased VO_2 and VCO_2 at rest. This protocol led to an increase in exercise time, improving exercise tolerance and could become an important methodology in the treatment of patients with stable CHF treated at the outpatient clinic.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

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