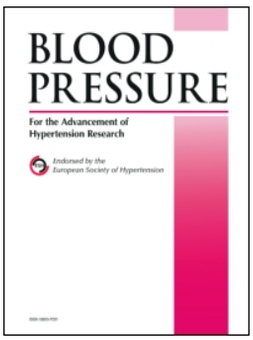

Blood Pressure

ISSN: 0803-7051 (Print) 1651-1999 (Online) Journal homepage: http://www.tandfonline.com/loi/iblo20

Blood pressure changes in patients with chronic heart failure undergoing slow breathing training

Tomasz Drozdz, Grzegorz Bilo, Dorota Debicka-Dabrowska, Marek Klocek, Gabriella Malfatto, Grzegorz Kielbasa, Katarzyna Styczkiewicz, Agnieszka Bednarek, Danuta Czarnecka, Gianfranco Parati & Kalina Kawecka-Jaszcz

To cite this article: Tomasz Drozdz, Grzegorz Bilo, Dorota Debicka-Dabrowska, Marek Klocek, Gabriella Malfatto, Grzegorz Kielbasa, Katarzyna Styczkiewicz, Agnieszka Bednarek, Danuta Czarnecka, Gianfranco Parati & Kalina Kawecka-Jaszcz (2015): Blood pressure changes in patients with chronic heart failure undergoing slow breathing training, Blood Pressure, DOI: 10.3109/08037051.2016.1099800

To link to this article: http://dx.doi.org/10.3109/08037051.2016.1099800

Published online: 29 Oct 2015.

BLOOD PRESSURE, 2015
HTTP://DX.DOI.ORG/10.3109/08037051.2016.1099800

Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

Blood pressure changes in patients with chronic heart failure undergoing slow breathing training

Tomasz Drozdz[a], Grzegorz Bilo[b], Dorota Debicka-Dabrowska[a], Marek Klocek[a], Gabriella Malfatto[b], Grzegorz Kielbasa[a], Katarzyna Styczkiewicz[a], Agnieszka Bednarek[a], Danuta Czarnecka[a], Gianfranco Parati[b,c] and Kalina Kawecka-Jaszcz[a]

[a]Department of Cardiology, Interventional Cardiology and Hypertension, Jagiellonian University Medical College, Krakow, Poland; [b]Department of Cardiovascular, Neural and Metabolic Sciences, San Luca Hospital, IRCCS Istituto Auxologico Italiano, Milan, Italy; [c]Department of Health Sciences, University of Milano-Bicocca, Milan, Italy

ABSTRACT

Background. Slow breathing training (SBT) has been proposed as a new non-pharmacological treatment able to induce favorable effects in patients with chronic heart failure (CHF). However, no information is available regarding its effects on orthostatic blood pressure (BP) changes in these patients, an issue of practical relevance given the reported BP-lowering effect of SBT. The aim of this study is to evaluate the influence of SBT on BP and whether SBT induces orthostatic hypotension (OH) or changes in quality of life (QoL) in CHF patients. *Methods*. The analysis was performed as part of an ongoing crossover open trial aimed at assessing the clinical effectiveness of SBT in treated patients with CHF. The patients underwent 10–12 weeks of SBT with the RESPeRATE device and 10–12 week follow-up under usual care. Patients were randomly divided into two groups: group I began with SBT, followed by usual care; group II began with usual care, followed by SBT. Patients undergoing SBT were asked to perform each day two separate 15 min sessions of device-guided SBT at a breathing frequency of 6 breaths/min. In all patients, before the enrollment and after each study phase, clinical data collection and BP measurements in sitting, supine and standing position were performed. OH was defined as a decrease of ≥ 20 mmHg in systolic blood pressure (SBP) or ≥ 10 mmHg in diastolic blood pressure (DBP) within 3 min of standing. QoL was assessed three times at the beginning, and after each phase of the study by the Minnesota Living with Heart Failure (MLHF) questionnaire. *Results*. Forty patients (two equal groups) completed the study, with the following baseline characteristics: 32 males/eight females, age 63.3 ± 13.4 years, 25 with ischemic CHF, 37 in New York Heart Association class II and three in class III, left ventricular ejection fraction 30.8 ± 6.7%, mean BP 138.7 ± 16.5/83.1 ± 11.5 mmHg, 23 with arterial hypertension and four with a history of stroke. There were no significant differences between the groups in clinical characteristics, SBP and DBP at rest, while seated and before and after standing up. OH prevalence was low and did not change during the study (10% vs 10%). No significant difference in average SBP and DBP changes secondary to body position were found when comparing the two study phases. Decrease in MLHF score was observed in group I during SBT ($p = 0.002$), but not in group II. *Conclusions*. Our data indicate that SBT is safe, does not affect the prevalence of OH in CHF patients and shows a non-significant tendency to improve QoL. These results should be confirmed in a larger sample of patients to support the safety of SBT and its possible benefits as a novel component of cardiorespiratory rehabilitation programs in CHF.

ARTICLE HISTORY
Received 3 August 2015
Accepted 15 September 2015
Published online 21 October 2015

KEYWORDS
Blood pressure, chronic heart failure, orthostatic hypotension, slow breathing training

Introduction

Despite unquestionable advances in treatment causing prolongation of survival, chronic heart failure (CHF) remains an important cause of death among patients with cardiovascular diseases. The prevalence of CHF increases with age and with longer lifetime (paradoxically due to better management of acute events) in patients with hypertension and coronary heart disease, diseases that constitute the most common causes of CHF [1–3]. The Framingham Study showed that more than 90% of participants who developed CHF had antecedent hypertension [4]. In recent years, the mortality of CHF patients has only slightly improved [5]. Moreover, it is estimated that in the future CHF treatment costs will increase considerably [6]. It is worth underlining that symptomatic heart failure negatively influences the quality of life (QoL) in patients by restricting various areas and domains of physical (fatigue, dyspnea, low physical capacity), psychological (emotional distress, depression) and social activities [7].

CONTACT Professor Kalina Kawecka-Jaszcz mckaweck@cyf-kr.edu.pl I Department of Cardiology, Interventional Cardiology and Hypertension, Collegium Medicum Jagiellonian University, 31-501 Kraków, ul. Kopernika 17, Poland

A continuing search for treatment modalities able to improve outcomes and QoL, and characterized by low nuisance for CHF patients, is therefore necessary. New non-pharmacological methods, including slow breathing training (SBT), may be of particular interest in this regard [8].

Slow breathing has a beneficial impact on the function of the cardiovascular system in CHF patients [9]. It has been shown that slow respiratory rate in CHF increases blood oxygen saturation at rest, improves respiratory exchange in the lungs, increases exercise tolerance by reducing the sensation of breathlessness and reduces excessive sympathetic activation, with a parallel increase in baroreceptor sensitivity reflecting parasympathetic modulation [10]. It has been demonstrated that slow and deep respiration suppresses steady-state sympathetic nerve activity in patients with high levels of resting sympathetic tone, as in heart failure [11].

To self-maintain a constant and low number of breaths per minute is a difficult task for a patient. Devices that give visual and audible signals to help patients to achieve a desired frequency of breathing may be used. One such device is the RESPeRATE (InterCure, Lod, Israel). So far, the device has been used as a non-pharmacological method for lowering high blood pressure (BP). Recommendations for the use of RESPeRATE have been recognized by the American Heart Association in the statement describing non-pharmacological methods for lowering BP [12].

An intervention potentially able to lower BP could lead to orthostatic hypotension (OH) in CHF patients, who commonly face low BP levels either spontaneously or owing to pharmacological treatment for their condition. Immediately after standing up, a gravitational displacement of blood occurs, with a decrease in venous return to the heart and reduction in left ventricular (LV) filling [13]. The result is a reduction in stroke volume and cardiac output, which could lead to hypotension, but which is commonly counteracted by the resulting reduction in arterial baroreceptor stimulation and by the consequent marked decrease in parasympathetic activity mediated by the vagus nerve, leading to a heart rate increase, and the increase in sympathetic activity, leading to peripheral vasoconstriction and to a greater LV contractility [14]. Patients with CHF demonstrate a dysfunction of this baroreflex-mediated mechanism [15], which may be one of the causes of the occurrence of OH in these individuals.

In healthy elderly people, the incidence of OH is estimated to be around 5–30% [16]. OH itself is associated with an increased risk of cardiovascular disease [17]. There is limited information, however, on the frequency of OH in patients with CHF. This is an important issue, because of the results of the Italian GISSI-HF study: in almost 7000 patients with CHF it was shown that low systolic blood pressure (SBP) increases the risk of early death [18].

The aim of this study was to evaluate the influence of SBT on BP and whether this recently proposed new type of non-pharmacological treatment induces OH and influences QoL in patients with CHF.

Materials and methods

Study design

The study is an ongoing crossover open trial where patients, in random order, undergo a 10–12 week period of SBT with the RESPeRATE device (InterCure, Lod, Israel) and a 10–12 week follow-up period under usual care without breathing exercises. Patients were randomly divided into two groups: group I started with SBT followed by usual care; group II started with usual care, followed by SBT (Figure 1). Patients randomized to SBT were asked to perform each day two separate 15 min sessions of device-guided SBT at a breathing frequency of 6 breaths/min. In all patients, before the enrollment and after each study phase, clinical data collection, BP measurements in supine and standing positions,

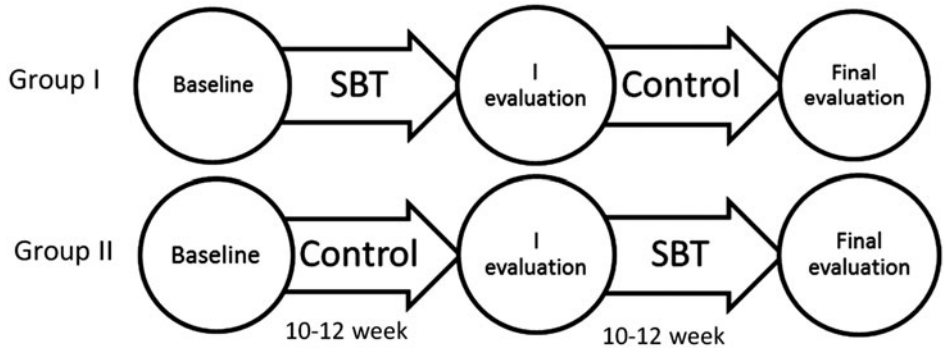

Figure 1. Study design. SBT, slow breathing training.

polysomnography, beat-to-beat BP monitoring, echocardiography, Six-Minute Walk Test, 24 h Holter monitoring, QoL assessment through the Minnesota Living with Heart Failure (MLHF) questionnaire and laboratory tests were performed. Optimized pharmacological treatment was kept constant during the study.

Study population

Patients aged >18 years with New York Heart Association (NYHA) class II–III CHF, with left ventricular ejection fraction (LVEF) < 40% (echocardiography) under stable clinical conditions, with no cardiovascular interventions in the past 3 months and who were stable on pharmacological treatment in the past 4 weeks were enrolled. Sinus rhythm presence was required and checked by 24 h Holter electrocardiogram monitoring. The patients had to be able to perform breathing exercises following instructions received during a previously completed supervised training session. Patients who had undergone heart transplantation, patients who were within 3 months since the end of traditional cardiac rehabilitation, and those with serious chronic obstructive pulmonary disease (forced expiratory volume in 1 second < 50%), ventricular arrhythmias or conduction abnormalities (ventricular tachycardia, ventricular fibrillation, atrioventricular block grade II and III) were excluded.

Blood pressure measurement

BP measurements were performed with a validated oscillometric device (OMRON M6 (OMRON Corporation, Kyoto, Japan)) at the brachial artery, while the patient was sitting, at room temperature, at a fixed time of day (between 08.00 and 11.00 h), having refrained from eating and smoking for at least 30 min, and after 10 min of rest. The measurements were performed twice with an interval of at least 5 min and mean values were calculated.

To diagnose OH, subjects were studied in the morning, in the supine position, in a quiet and dimly lit room after an overnight fast. Non-invasive BP measurements were taken with an arm cuff (OMRON M6) after 10 min rest in the supine position. OH was defined as a decrease of ≥20 mmHg in SBP or ≥10 mmHg in diastolic blood pressure (DBP) within 3 min of standing [13].

Slow breathing training (RESPeRATE device)

The system includes a belt-type respiration sensor connected to a computerized box that generates musical patterns through earphones. The device guides the user to slow their breathing, with the expiration phase being longer than the inspiration phase. After a "recognition" phase, during which the device learns the user's natural breathing patterns, it starts guiding the user to breathe more slowly in the following way: (i) the sensor monitors continuously breathing movements; (ii) the computerized box detects the breathing patterns and average inhale/exhale duration over the last four respiratory cycles; (iii) it synthesizes musical patterns with "inspiration" and "expiration" guiding tones with related but different durations (longer in expiration); and finally (iv) the user voluntarily and effortlessly synchronizes inhaling and exhaling movements with the guiding tones. As a result, breathing frequency is reduced from the initial spontaneous rate (usually 14–18 breaths/min) to a rate of 6–8 breaths/min, with prolonged exhalation [19].

Echocardiography

The same experienced operator, blinded to patients' allocation, performed the echocardiographic examinations of all patients (General Electric VIVID7 (GE Healthcare, Little Chalfont, United Kingdom), probe 2.5 MHz). The measurements, i.e. M-mode, 2D and Doppler method, were performed according to the standards required by the American Society of Echocardiography [20]. LVEF was measured with Simpson's technique based on the average from at least three registered cycle heart beats.

Minnesota Living with Heart Failure Questionnaire

Patients completed the Polish version of the 21-item MLHF questionnaire to assess disease-specific QoL. The questionnaire asks about how much the heart condition has affected the patient's life during the past month. Each item is rated on a Likert scale between 0 (no impairment) and 5 (very much impaired), resulting in a total score between 0 and 105 for total QoL. The MLHF also allows one to compute subscores for the physical (eight items, range 0–40) and emotional (five items, range 0–25) aspects of QoL [21]. Lower MLHF scores indicate better QoL.

Statistical analysis

All data were analyzed using the Statistica PL software (v. 10.0; StatSoft). Categorical variables are reported as percentages and continuous variables as means and standard deviation (SD). The chi-squared test was applied to all categorical variables. For continuous variables, analysis of variance (ANOVA) was used. A p value of less than 0.05 was considered to indicate statistical significance.

Results

Forty patients aged 23–88 years completed the study. Clinical characteristics of the enrolled patients are shown in Table I. Secondary forms of cardiomyopathy were the most prevalent causes of CHF: 25 patients (62.5%) had ischemic cardiomyopathy, two patients had a diagnosis of CHF as a result of myocarditis, in one patient heart failure had developed as a result of systemic disease (sarcoidosis) and 12 patients had primary dilated cardiomyopathy of unknown etiology. There were no significant differences in the studied parameters between the groups at baseline (Table I).

Before enrollment all patients had to be under constant pharmacotherapy for at least 4 weeks, according to the current European Society of Cardiology (ESC) guidelines [2], consisting of beta-blockers and angiotensin-converting enzyme inhibitors (ACEIs) (in four patients angiotensin receptor antagonists because of intolerance to ACEIs) at the maximum tolerated dose. The majority of patients used diuretics. In addition, all patients with LVEF below 35% received a mineralocorticoid receptor antagonist; one patient received ivabradine in accordance with the recommendations of the ESC. Three patients in the study required intensification of diuretic treatment owing to heart failure exacerbation (worsening of peripheral edema): two during the control period (groups I and II) and one during SBT.

There was a significant reduction in SBP and DBP between baseline and the final evaluation in group I. In group II the same trend was present, although it did not reach statistical significance (Table II). The prevalence of OH was low (10% at baseline, 12.5% at 12 weeks and 10% at final evaluation) and did not change between the phases of the study (Table III). The average changes in SBP/DBP after changing position were also not significant (Table IV).

A decrease in MLHF questionnaire score was observed in group I during SBT. In group II a similar decrease was present only after completion of the study. There were no differences between the groups in the MLHF score at any time during the study (Figure 2).

Discussion

A few previous reports have suggested that SBT may be a useful adjunct to standard treatment in patients with CHF. The acute effects of slow breathing in heart failure patients were assessed by Bernardi et al., who reported an improvement in blood oxygenation and exercise performance without significant changes in BP after 4 min of breathing at a rate of 6 breaths/min. In a small subgroup of participants who underwent 1 month of SBT, long-term benefits in terms of blood oxygenation and in objective and subjective indices of exercise

Table I. Baseline characteristics of the study groups.

Parameter	Group I ($n = 20$)	Group II ($n = 20$)	All ($n = 40$)	p
Age (years)	65.5 ± 11.6	61.2 ± 15.0	63.3 ± 13.4	0.57
Male gender (%)	15 (75)	17 (85)	32 (80)	0.46
NYHA class II/III (n)	18/2	19/1	37/3	0.50
Ejection fraction (%)	31.7 ± 6.7	29.9 ± 6.9	30.8 ± 6.7	0.41
SBP sitting (mmHg)	141.6 ± 14.5	135.9 ± 18.2	138.7 ± 16.5	0.42
DBP sitting (mmHg)	84.4 ± 11.6	81.9 ± 11.6	83.1 ± 11.5	0.53
SBP supine (mmHg)	122.0 ± 15.7	128.4 ± 19.5	125.4 ± 17.9	0.27
DBP supine (mmHg)	70.8 ± 9.5	74.5 ± 11.0	72.8 ± 10.3	0.56
SBP standing (mmHg)	119.2 ± 19.1	126.1 ± 16.0	122.8 ± 17.6	0.23
DBP standing (mmHg)	73.3 ± 11.1	77.3 ± 14.2	75.4 ± 12.8	0.34
Minnesota Quality of Life (points)	49.7 ± 10.0	48.7 ± 14.2	49.2 ± 12.2	0.79
Ischemic heart disease (%)	15 (75)	10 (50)	25 (62.5)	0.07
Hypertension (%)	12 (60)	11 (55)	23 (57.5)	1.0
Stroke (%)	2 (10)	2 (10)	4 (10)	0.50
Diabetes (%)	5 (25)	8 (40)	13 (32.5)	0.48

Data are shown as mean ± SD or n (%).
NYHA, New York Heart Association; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Table II. Changes in sitting blood pressure values (mmHg) among different study phases.

Group	Baseline SBP/DBP	At 12 weeks SBP/DBP	Final evaluation SBP/DBP	p baseline–12 weeks	p 12 weeks–final	p baseline–final
I ($n = 20$)	147.2/87.3	134.7/80.5	132/77.7	0.06/0.17	0.85/0.73	0.02/0.04
II ($n = 20$)	140.2/84.3	138.0/84.2	134.3/81.4	0.79/0.99	0.91/0.70	0.53/0.69
p I vs II	0.32/0.53	0.65/0.48	0.29/0.24			

SBP, systolic blood pressure; DBP, diastolic blood pressure.

performance were confirmed. Changes in BP after the SBT period were not reported in this study [10]. The information on the beneficial effects of SBT was further extended by a study by Parati et al. on 24 patients with CHF who underwent SBT using the same device as in the present report. These effects included reductions in NYHA class and pulmonary artery pressure, and improvement in ejection fraction, ventilatory variables and QoL [9].

The data on the effects of slow breathing and SBT on BP in patients with CHF are limited and inconsistent. During a slow breathing session, BP was unchanged in the study by Bernardi et al. [10]. Another paper from this group reported, however, a significant decrease in BP during a 4 min slow breathing session in CHF patients (but not in healthy controls) [22]. This discrepancy may be explained by impaired responses of the arterial baroreflex and chemoreflex occurring during slow breathing in CHF and in hypoxic patients [22,23]. Further insight into the possible mechanisms comes from a study by Bilo et al., performed in healthy volunteers exposed to high altitude (a condition in many regards similar to heart failure with hypoxemia). In this setting, a 15 min session of slow breathing induced a decrease in BP level, possibly due to the deactivation of peripheral chemoreceptors by increased blood oxygenation and consequent reduction of sympathetic drive [24].

In our study we found no changes in BP level after a period of SBT. This result is in line with the randomized trial by Ekman et al., in which 30 CHF patients underwent a 4 week period of SBT with the RESPeRATE device, while 35 CHF patients listened to music from a compact disc player (control group) [25]. In the study by Parati et al., only a non-significant reduction in BP was observed, mainly in patients with high baseline values [9].

Table III. Prevalence of orthostatic hypotension by group and study phase.

Group ($n = 20$)	Baseline	At 12 weeks	Final evaluation
I (SBT/usual care)	2 (10)	2 (10)	1 (5)
II (usual care/SBT)	2 (10)	3 (15)	3 (15)
p	0.52	0.66	0.32

Data are shown as *n* (%).
SBT, slow breathing training.

To our knowledge, this is the first study to assess the effects of SBT on dynamic regulation of BP during orthostatic challenge and its impact in terms of OH prevalence in patients with CHF. A beneficial effect of slow breathing on orthostatic tolerance was demonstrated in a randomized crossover study on healthy volunteers. The authors postulated that this improvement is mediated by the generation of negative intrathoracic pressure during slow and deep breathing, increasing right venous return and in turn LV stroke volume, with resulting beneficial effects on the cardiovascular and autonomic systems [26]. Another suggested mechanism of the positive influence of slow breathing on OH is the possibility that the generation of greater negative pressure in the thorax accelerates blood flow through capillary beds owing to a vacuum effect and to maximization of the pressure gradient.

Orthostatic intolerance is an important issue in CHF. In a meta-analysis by Ricci et al., compared with the absence of OH, the occurrence of OH was associated with a significantly increased risk of heart failure (relative risk 2.25, 95% confidence interval 1.52–3.33) [27]. An impairment in autonomic regulation is frequent in this population [28] and may be further exacerbated by the use of some of the medications commonly prescribed in CHF, such as diuretics or vasodilators [29]. In this context, our study did not reveal any negative influence of SBT in terms of orthostatic tolerance, indicating that this technique represents an additional, potentially valuable therapeutic approach devoid of negative effects on OH in CHF patients. In fact, one could hypothesize that many CHF patients for whom OH is an issue may benefit from SBT, should it be able to reduce the need for the use of diuretics or vasodilators to control symptoms. This possibility was beyond the scope of the present study but it appears to be of clinical interest and should be explored in future research.

Finally, we observed an improvement in the QoL during the course of the study, between baseline and final assessment, in line with that reported by Parati et al. [9]. However, the pattern of MLHF changes in the two groups (improvement was observed in both the SBT and usual care phases) suggests that it was not specifically due to SBT, but rather that direct contact of patients with the staff involved in the trial, performing check-ups,

Table IV. Changes in blood pressure in the two study groups and in the different study phases after standing up.

Group	Baseline SBP/DBP	At 12 weeks SBP/DBP	Final evaluation SBP/DBP	*p*
I	−2.6 ± 14.1/+2.4 ± 8.7	+0.3 ± 10.5/−1.3 ± 8.5	−0.4 ± 14.6/+4.3 ± 7.9	0.69/0.09
II	−2.3 ± 12.2/+2.8 ± 10.1	+0.3 ± 10.5/+0.7 ± 9.3	−4.35 ± 14.6/+2.5 ± 9.5	0.70/0.97

SBP, systolic blood pressure; DBP, diastolic blood pressure.

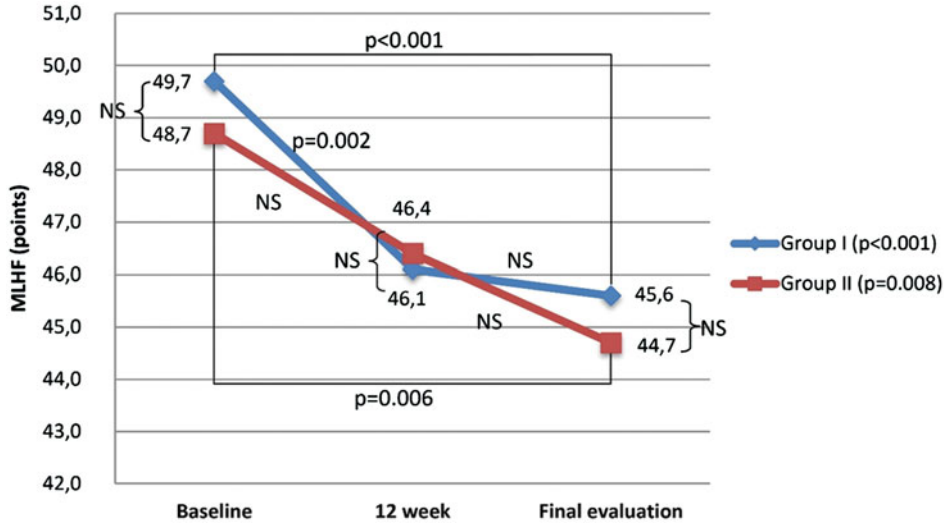

Figure 2. Results of the Minnesota Living with Heart Failure (MLHF) questionnaire according to group and study phase. NS, not significant.

increasing medical surveillance and showing interest in their problems, was probably the main cause of improved QoL. This emphasizes the need for appropriate control in studies on non-pharmacological interventions, where the effects of a close follow-up may be as important as those of the intervention itself, in particular when "soft" endpoints (such as QoL) are considered. However, it should be underlined that Ekman et al., in their well-controlled study on CHF patients, found that a positive effect of SBT on the QoL and severity of CHF symptoms was restricted to responding patients, i.e. those in whom SBT induced lasting changes in the breathing pattern [25]. In addition, owing to the crossover design of our study, we cannot completely exclude a carryover effect to explain the difference in QoL score between baseline and the end of the study.

Our study has several strengths, including having a sample size moderately larger than that of most previous studies [9,25], having a controlled design and using a device specifically designed for SBT (and approved by the US Food and Drug Administration for BP lowering), following a standardized exercise protocol. This device implements a series of features which make the exercise easier for the patient, including individualized acoustic guidance of breathing frequency, resulting in its gradual reduction, and visual feedback on the exercise performance. This is important as self-maintenance of a constantly low number of breaths by the patient is a difficult task and a rigid breathing pace could have negative effects on exercise performance.

There are also some limitations of our study which should be acknowledged. First, the BP measurement was performed using the traditional, non-continuous method. We therefore cannot completely exclude possible BP reductions between the scheduled measurements. Secondly, the number of subjects with OH was low and therefore the study may not be sufficiently powered to detect a small increase in OH prevalence. Nonetheless, the fact that the absolute values of orthostatic BP changes were virtually unchanged after SBT makes this possibility unlikely. Thirdly, for practical reasons we implemented a crossover rather than a parallel group design. Thus, we cannot exclude some carryover effect, as exemplified by the changes observed in the QoL. Finally, we cannot exclude the possible influence of coexisting morbidities (e.g. diabetes, hypertension) or that of concomitant drug therapy on OH, although drug treatment was kept stable during the study.

Conclusions

Our data indicate that SBT does not increase the prevalence of OH, thereby supporting it as a feasible and safe method which may be used in CHF patients. Moreover, our data do not seem to support a significant impact on QoL by this intervention; however, given the cross-sectional design of our study, this finding needs to be confirmed by longitudinal trials with a controlled parallel group design. Further data from controlled studies are still necessary to confirm the clinical benefits of SBT in CHF patients, before it may be proposed as a novel and useful component of cardiorespiratory rehabilitation programs in CHF.

Funding

This study has been funded by a grant from the National Science Centre (NSC) [no. UMO-2011/03/B/NZ5/00533].

Declaration of interest

No potential conflict of interest was reported by the authors.

References

1. Mosterd A, Hoes AW. Clinical epidemiology of heart failure. Heart. 2007;93:1137–46.
2. McMurray JJ, Adamopoulos S, Anker SD, et al. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. Eur Heart J. 2012;33:1787–847.
3. Kawecka-Jaszcz K, Kloch-Badełek M, Wojciechowska W. Hypertension as a risk factor for heart failure. Arterial Hypertens. 2011;15:275–82.
4. Levy D, Larson MG, Vasan RS, Kannel WB, et al. The progression from hypertension to congestive heart failure. JAMA. 1996;275:1557–62.
5. Chen J, Normand SL, Wang Y, Krumholz HM. National and regional trends in heart failure hospitalization and mortality rates for Medicare beneficiaries, 1998–2008. JAMA. 2011;306:1669–78.
6. Heidenreich PA, Albert NM, Allen LA, et al. Forecasting the impact of heart failure in the United States: a policy statement from the American Heart Association. Circ Heart Fail. 2013;6:606–19.
7. Klocek M, Czarnecka D. Quality of life in patients with chronic heart failure. In: Kawecka-Jaszcz K, Klocek M, Tobiasz-Adamczyk B, Bulpitt CJ, editors. Health-related quality of life in cardiovascular patients. Milan: Springer; 2013. p. 61–73.
8. Drozdz T, Debicka-Dabrowska D, Styczkiewicz K, et al. New non-pharmacological treatment methods in heart failure. Przegl Lek. 2014;71:441–6.
9. Parati G, Malfatto G, Boarin S, et al. Device-guided paced breathing in the home setting: effects on exercise capacity, pulmonary and ventricular function in patients with chronic heart failure: a pilot study. Circ Heart Fail. 2008;1:178–83.
10. Bernardi L, Spadacini G, Bellowon J, et al. Effect of breathing rate on oxygen saturation and exercise performance in chronic heart failure. Lancet. 1998;351:1308–11.
11. Harada D, Asanoi H, Takagawa J, et al. Slow and deep respiration suppresses steady-state sympathetic nerve activity in patients with chronic heart failure: from modeling to clinical application. Am J Physiol Heart Circ Physiol. 2014;307:H1159–68.
12. Brook RD, Appel LJ, Rubenfire M, et al. Beyond medications and diet: alternative approaches to lowering blood pressure: a scientific statement from the American Heart Association. Hypertension. 2013;61:1360–83.
13. Freeman R, Wieling W, Axelrod FB, et al. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Clin Auton Res. 2011;21:69–72.
14. Di Rienzo M, Parati G, Radaelli A, Castiglioni P. Baroreflex contribution to blood pressure and heart rate oscillations: time scales, time-variant characteristics and nonlinearities. Philos Trans A Math Phys Eng Sci. 2009;367:1301–18.
15. La Rovere MT, Pinna GD, Maestri R, Sleight P. Clinical value of baroreflex sensitivity. Neth Heart J. 2013;21:61–3.
16. Low PA. Prevalence of orthostatic hypotension. Clin Auton Res. 2008;18 Suppl 1:8–13.
17. Fan XH, Wang Y, Sun K, et al. Disorders of orthostatic blood pressure response are associated with cardiovascular disease and target organ damage in hypertensive patients. Am J Hypertens. 2010;23:829–37.
18. Barlera S, Tavazzi L, Franzosi MG, et al. Predictors of mortality in 6975 patients with chronic heart failure in the Gruppo Italiano per lo Studio della Streptochinasi nell'Infarto Miocardico-Heart Failure trial: proposal for a nomogram. Circ Heart Fail. 2013;6:31–9.
19. Gavish B. Device-guided breathing in the home setting: technology, performance and clinical outcomes. Biol Psychol. 2010;84:150–6.
20. Gottdiener JS, Bednarz J, Devereux R, et al. American Society of Echocardiography recommendations for use of echocardiography in clinical trials. J Am Soc Echocardiogr. 2004;17:1086–119.
21. Middel B, Bouma J, de Jongste M, et al. Psychometric properties of the Minnesota Living with Heart Failure Questionnaire (MLHF-Q). Clin Rehabil. 2001;15:489–500.
22. Bernardi L, Porta C, Spicuzza L, et al. Slow breathing increases arterial baroreflex sensitivity in patients with chronic heart failure. Circulation. 2002;105:143–5.
23. Spicuzza L, Gabutti A, Porta C, et al. Yoga and chemoreflex response to hypoxia and hypercapnia. Lancet. 2000;356:1495–6.
24. Bilo G, Revera M, Bussotti M, et al. Effects of slow deep breathing at high altitude on oxygen saturation, pulmonary and systemic hemodynamics. PLoS One. 2012;7:e49074.
25. Ekman I, Kjellström B, Falk K, et al. Impact of device-guided slow breathing on symptoms of chronic heart failure: a randomized, controlled feasibility study. Eur J Heart Fail. 2011;13:1000–5.
26. Lucas SJ, Lewis NC, Sikken EL, et al. Slow breathing as a means to improve orthostatic tolerance: a randomized sham-controlled trial. J Appl Physiol. 2013;115:202–11.
27. Ricci F, Fedorowski A, Radico F, Romanello M, Tatasciore A, Di Nicola M, et al. Cardiovascular morbidity and mortality related to orthostatic hypotension: a meta-analysis of prospective observational studies. Eur Heart J. 2015;36:1609–17.
28. Kishi T. Heart failure as an autonomic nervous system dysfunction. J Cardiol. 2012;59:117–22.
29. Poon IO, Braun U. High prevalence of orthostatic hypotension and its correlation with potentially causative medications among elderly veterans. J Clin Pharm Ther. 2005;30:173–8.