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

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Background—Regular slow breathing is known to improve autonomic cardiac regulation and reduce chemoreflex sensitivity in heart failure. We explored the acceptability and usefulness of a device for paced slow breathing at the home setting.

Methods and Results—In this open pilot study, 24 patients with chronic heart failure (61% males, mean age, 64±9 years; New York Heart Association class, 2.81±0.01) were randomized to a control group receiving conventional treatment (n=12) or to a group receiving conventional treatment and device-guided paced breathing (n=12). Groups were comparable for age, therapies, and clinical characteristics. They were evaluated at baseline and again after 10 weeks by Doppler echocardiography, pulmonary function, cardiopulmonary stress test, and quality of life (Minnesota Quality of Life questionnaire). The treatment group was instructed to use the equipment for 18 minutes twice daily. The device is a computerized box connected to a belt-type respiration sensor and to headphones; it generates musical tones (based on the user’s breathing rate and inspiration ratio), which guide the user to progressively and effortlessly slow his or her breathing rate <10 breaths/min. The treatment group showed high compliance to the device (90% of the prescribed sessions were completed). Blinded analysis of data demonstrated increased ejection fraction and decreased estimated pulmonary pressure in the echocardiograms of the treated group versus controls and favorable changes in New York Heart Association class, VE/VCO2, FEV1, and a quality of life measure, as well (all P<0.05).

Conclusions—This pilot investigation demonstrates that device-guided paced breathing at home is feasible and results in an improvement in clinically relevant parameters for patients with heart failure and systolic dysfunction. (Circ Heart Fail. 2008;1:178-183.)

Key Words: heart failure ■ nervous system, autonomic ■ respiration ■ rehabilitation

Respiratory abnormalities (from shallow breathing to Cheyne-Stokes periodicity) are very frequent in advanced chronic heart failure, particularly during sleep, being the rule in patients in transplant’s list, and becoming less common or absent when patients improve their functional status.1–4 Their presence, particularly when extended during daytime, is an indicator for poor prognosis.5 It has been postulated that respiratory abnormalities express a severe form of derangement of central neural control of both respiratory and cardiovascular functions in chronic heart failure.2–5 Spectral and cross-spectral analyses of heart rate and respiratory variability performed in patients have shown that most of the so-called very low frequency heart rate oscillations in the heart rate spectrum can be accounted for an abnormal breathing pattern.2,6 Moreover, if patients are trained to breathe regularly, spectral abnormalities disappear2–9 and exercise tolerance improves.8 However, whether correction of such abnormal pattern of respiration might improve the patients’ prognosis is still a matter of debate. Mancini et al showed a favorable response to a period of specific training of respiratory muscles in advanced heart failure,10 but other groups denied that respiratory interventions, such as nasal continuous positive airway pressure or inspiratory muscle training, could induce any significant benefit.11–13

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Results of interventions aimed at modifying alterations in breathing patterns differ; one of the underlying reasons for this may be the difficulty in setting clear protocols for respiratory exercises, which should be easy to perform by patients and overcome the limitations of the traditional...
muscle strengthening training\textsuperscript{10} or of the use of mechanical devices,\textsuperscript{12,14,15} In fact, these interventions may require significant effort of respiratory muscles and induce undesirable fatigue in the patient. The current pilot study aimed at providing additional information on this issue. In particular, we tested the possibility that a commercial electronic device, which guides interactively the subject through musical tones toward an effortlessly slowing in respiratory rate (below 10 breaths/min), would improve the functional and clinical status of patients with moderate to severe chronic heart failure, even in the absence of detectable breathing abnormalities during waking hours. Preliminary data have been reported in abstract form.\textsuperscript{16}

**Methods**

**Patients and Controls**

This pilot randomized study included 24 consecutive patients (18 males, 6 females; mean age $\pm$ SD, 64$\pm$9 years; range, 45 to 75) fulfilling the inclusion and exclusion criteria after a 6-week conventional rehabilitation training period, ie, the standard treatment approach in our Heart Failure Clinic for patients who remain in New York Heart Association (NYHA) class II to III despite optimal treatment in the past 6 months (ie, diuretics, $\beta$-blockers, ACE-inhibitors, or angiotensin II receptor blockers, all given at the maximal tolerated dose according to current international guidelines\textsuperscript{17}). Inclusion criteria were left ventricular ejection fraction $<40\%$, NYHA class II to III, and peakVO\textsubscript{2} $\leq 16$ mL/($\text{kg}\cdot\text{min}$) at cardiopulmonary stress test. We excluded patients with significant rhythm disturbances (atrial flutter/fibrillation, AV block, frequent ventricular arrhythmias, implantation of pacemaker), recent cardiac surgery, severe chronic obstructive lung disease (FEV\textsubscript{1}$<50\%$ predicted), abnormal breathing patterns, significant arterial oxygen desaturation during wakefulness, and uncompensated diabetes mellitus.

The study was approved by the ethics committee of our institution. Patients were included only if, after being informed on the nature and purpose of our study, they gave their written informed consent.

**Study Design**

Patients were randomized to a control group in which optimized pharmacological treatment was continued (n=12), or to a treatment group (n=12) in which, in addition to optimized standard therapy, patients were instructed to use a respiratory device (RESPeRATE, InterCure Ltd, Lod, Israel) that has proven useful for the nonpharmacological management of hypertension.\textsuperscript{18} After recruitment and baseline evaluation, patients were randomized to the control or to the slow paced breathing group. At the end of the treatment period (10 weeks), all data files downloaded from the RESPeRATE device on a weekly basis were pooled in a single file on a laptop PC for further analysis. In all patients, data derived from a physical examination were obtained during the visits performed at baseline and at the end of the 10-week study period. Echocardiography, cardiopulmonary stress test, and Minnesota Quality of Life Test were carried out at enrollment and after the 10-week follow-up. Investigators responsible for collection and analysis of echocardiography, cardiopulmonary test, and quality of life data were unaware of the individual patient allocation throughout the whole duration of the study. Patients assigned to the training group received detailed instructions about use of the breathing device. They also received an oscilometric device to be used for daily blood pressure measurements at home (Omron Healthcare, Kyoto, Japan). Patients were asked to perform, every day, 2 separate 18-minute sessions of paced breathing guided by the RESPeRATE device. During the first 3 minutes, the device measured the spontaneous breathing rate of the patient in order to set the rhythm of the music on it. In the following 15 minutes, the instrument helps the patient to slow down his or her breathing rate effortlessly in an individualized way to the range of less than 10 breaths/min. This range has been selected because it has been shown to induce favorable changes in autonomic cardiac control in normal subjects and in heart failure patients as well.\textsuperscript{8} The RESPeRATE device includes a battery-operated hand-held computerized box attached to headphones and to a belt-type respiration sensor to be mounted, even on clothes, to the chest or upper abdomen. The device provides a guide to effortless breathing in a closed loop way. First, the individual respiratory pattern is monitored, then inspiration and expiration times are calculated in real time and a 2-tone melody is created, with a high tone for inhalation and a low tone for exhalation: the user easily learns to synchronize his or her breathing with this musical pattern, whose rhythm is progressively reduced by increasing the duration of the exhalation tone. Thus, patients are gradually driven to breathe at a slow rate, usually $<10$ breaths/min, by prolonging the expiration phase. The device automatically stores individuals’ performance data from each session, and it is programmed to stop after 15 minutes of use. At each periodic visit, information on patients’ daily usage of the device, stored in its memory, was downloaded as well as blood pressure values measured at home with the Omron device.

**Echocardiography**

The same very experienced operator (G.B.), blinded to patients’ allocation, performed the echocardiography examinations of all patients. The quality of our cardiac ultrasound laboratory is certified according to the high standards required by the American Society of Echocardiography.\textsuperscript{19} In previous assessments, the reproducibility of routine measures (such as ejection fraction or pulmonary artery systolic pressure) was high, with a between-test variability of $<5\%$. We used a Sequoia C505 equipment (Siemens, Mountain View, Calif) with a 2.5 to 4.0 MHz (V4c) probe, with the patient in the left semilateral position. Variables assessed were left ventricular volumes and diameters, left ventricular ejection fraction with the Simpson’s technique, diastolic function through the analysis of transmitral and pulmonary veins flow. The presence and degree of mitral regurgitation were also evaluated; systolic pulmonary artery pressure (PAP) was indirectly estimated through Doppler evaluation of transtricuspidal flow.

**Cardiopulmonary Stress Test**

All patients, who had already undergone a rehabilitation period including 2 cardiopulmonary evaluations, were acquainted with the procedure. Indeed, the reproducibility of cardiopulmonary stress test parameters, repeatedly obtained at the time of the initial physical rehabilitation period, was high, with a variability of measured parameters between tests performed in the same condition $<10\%$.\textsuperscript{20} The examination was performed with Sensor Medics 2900 Pulmonary Function Test System (Sensor Medics, Loma Linda, Calif). The equipment allowed the preliminary assessment of pulmonary function, with the measure of forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV\textsubscript{1}); results were expressed as percent of the theoretical values, predicted according to age, sex, and body habits. Stress test was performed on a bicycle with a ramp protocol of 10-W increase every minute preceded by a 2-minute warm-up period. The equipment provided breath-by-breath evaluation of oxygen consumption (VO\textsubscript{2}), carbon dioxide production (VCO\textsubscript{2}), ventilation/min (V\textsubscript{T}, L/min), tidal volume (VT), and respiratory rate. Anaerobic threshold was measured with the V-slope method as described by Wasserman et al.\textsuperscript{21} Peak VO\textsubscript{2} (pVO\textsubscript{2}) was defined as the highest VO\textsubscript{2} obtained (usually, it corresponded to the highest workload reached) corrected for the body weight (mL/($\text{kg}\cdot\text{min}$)). Submaximal exercise capacity was evaluated as the O\textsubscript{2} consumption at the anaerobic threshold (VO\textsubscript{2}AT), whereas ventilatory efficiency was considered as the ratio between ventilation (V\textsubscript{T}, L/min) and CO\textsubscript{2} production (VCO\textsubscript{2}, L/min) at peak exercise. Finally, O\textsubscript{2} pulse at peak exercise (pO\textsubscript{2}, mL) was calculated as the ratio between VO\textsubscript{2} (mL/min) and heart rate (min$^{-1}$); because pO\textsubscript{2} corresponds to the product between stroke volume and arterial-venous O\textsubscript{2} concentration difference, it can be used as an index of stroke volume when peripheral O\textsubscript{2} extraction is normal.\textsuperscript{21}
Quality of Life Assessment

The Minnesota Quality of Life Questionnaire was designed for patients affected by chronic heart failure to appraise their quality of life in a quantitative way, through calculation of a score based on replies to a series of standardized questions. The sum of all scores provides a value related with the patient’s quality of life as a whole. In detail, the various questions investigate several aspects of the patient’s life, such as symptoms, difficulties in daily activities, patient’s perception of himself or herself and of his or her own disease. A low Minnesota Quality of Life Questionnaire score corresponds to less symptoms, less anxiety, and depression, ie, to a better quality of life; a high score, instead, indicates a worse quality of life, and it seems significantly related with a poorer survival.22,23

Statistical Analysis

Data are reported as mean±SD in the text, Figure, and tables. For continuous variables inside the groups, comparisons within groups (ie, before and after intervention) were performed by 2-tailed paired Student t test, even if the design of our study implied a “superiority comparison” (usual care versus intervention + usual care).24 To test for differences of the means between the 2 groups (ie, treated patients and controls), both at baseline and at the end of the study, we used 1-way ANOVA with subsequent Bonferroni test when allowed by the F value. To compare dichotomous variables, we used the χ² test. A probability value ≤0.05 was taken as the minimum level of statistical significance.

Results

Patients’ clinical characteristics are summarized in Table 1. The device was accepted by patients, and compliance to the treatment was high, as shown by analysis of the data downloaded from the device (90% of the prescribed sessions were completed). The effects of 10 weeks of home-based paced breathing, compared with the control group, on the study variables are shown in Table 2 and are described in detail.

Blood Pressure

In the treated group patients, who were all normotensive at baseline, a slight reduction in systolic blood pressure was induced by the regular use of the device (Table 2), whereas in the control group, no changes were observed. The change in systolic blood pressure in treated patients was significantly related to the initial value: the higher the baseline systolic pressure, the greater its reduction (R=0.68, P<0.001). No change in diastolic blood pressure was observed in both groups.

Table 1. Baseline Characteristics of the 2 Groups of Patients

<table>
<thead>
<tr>
<th></th>
<th>Treated</th>
<th>Controls</th>
<th>P (ANOVA or χ² Test)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>12</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Age (range)</td>
<td>64±9</td>
<td>62.8±10</td>
<td>0.38</td>
</tr>
<tr>
<td>Males/females</td>
<td>9/3</td>
<td>9/3</td>
<td>0.32</td>
</tr>
<tr>
<td>Ischemic/nonischemic</td>
<td>9/3</td>
<td>8/4</td>
<td>0.29</td>
</tr>
<tr>
<td>NYHA</td>
<td>2.84±0.02</td>
<td>2.72±0.03</td>
<td>0.89</td>
</tr>
<tr>
<td>EF%</td>
<td>32.5±6.3</td>
<td>33.2±4.4</td>
<td>0.35</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>121±17</td>
<td>111±11</td>
<td>0.19</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>81±12</td>
<td>77±9</td>
<td>0.25</td>
</tr>
<tr>
<td>PAP, mm Hg</td>
<td>49±17</td>
<td>45±13</td>
<td>0.21</td>
</tr>
<tr>
<td>Maximal workload (W)</td>
<td>74±25</td>
<td>75±21</td>
<td>0.44</td>
</tr>
<tr>
<td>Peak VO₂, mL/(kg-min)</td>
<td>12.2±3.4</td>
<td>13.4±4.4</td>
<td>0.19</td>
</tr>
<tr>
<td>ATVO₂, mL/(kg-min)</td>
<td>8.8±2.6</td>
<td>9.3±5.6</td>
<td>0.28</td>
</tr>
<tr>
<td>O₂ pulse, mL/beat</td>
<td>8.5±2.4</td>
<td>9.1±3.2</td>
<td>0.36</td>
</tr>
<tr>
<td>Peak VEVO₂</td>
<td>39.9±5.6</td>
<td>41.3±11.4</td>
<td>0.43</td>
</tr>
<tr>
<td>FEV₁, %</td>
<td>71±14</td>
<td>72±11</td>
<td>0.48</td>
</tr>
<tr>
<td>β-Blockers, %</td>
<td>58</td>
<td>60</td>
<td>0.45</td>
</tr>
<tr>
<td>ACE inhibitors, %</td>
<td>75</td>
<td>80</td>
<td>0.36</td>
</tr>
<tr>
<td>AT-2 inhibitors, %</td>
<td>33</td>
<td>20</td>
<td>0.21</td>
</tr>
<tr>
<td>Digitalis, %</td>
<td>8.3</td>
<td>11</td>
<td>0.35</td>
</tr>
<tr>
<td>Diuretics, %</td>
<td>91.6</td>
<td>88</td>
<td>0.37</td>
</tr>
</tbody>
</table>

EF% indicates left ventricular ejection fraction; SBP, systolic blood pressure; DBP, diastolic blood pressure; PAP, pulmonary artery pressure; ATVO₂%, VO₂ at anaerobic threshold; VEVO₂, ratio between ventilation and CO₂ flow; FEV₁%, forced expiratory volume at 1-second expiration.
Table 2. Effects of Device Guided Breathing on symptoms and Functional Assessments

<table>
<thead>
<tr>
<th></th>
<th>NYHA</th>
<th>EF%</th>
<th>SBP mm Hg</th>
<th>DBP mm Hg</th>
<th>PAP mm Hg</th>
<th>Maximal Workload, W</th>
<th>Pulmonary Artery Pressure (mm Hg)</th>
<th>O2 Pulse, mL/beat</th>
<th>VEVO2, mL/beat</th>
<th>FEV1%</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Treated (n=12)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>2.84±0.02</td>
<td>32±6</td>
<td>121±17</td>
<td>81±12</td>
<td>49±17</td>
<td>74±25</td>
<td>12.2±3.4</td>
<td>8.8±2.6</td>
<td>8.5±2.4</td>
<td>41±6</td>
<td>71±14</td>
</tr>
<tr>
<td>Home-based paced breathing</td>
<td>1.78±0.02†</td>
<td>39±9†</td>
<td>112±15*</td>
<td>76±11</td>
<td>38±9†</td>
<td>85±20†</td>
<td>14.1±3.2†</td>
<td>10.4±2.0†</td>
<td>9.3±2.9*</td>
<td>37±5†</td>
<td>78±16†</td>
</tr>
<tr>
<td>Controls (n=12)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>2.72±0.03</td>
<td>33±4</td>
<td>111±11</td>
<td>77±9</td>
<td>45±13</td>
<td>75±21</td>
<td>13.4±4.4</td>
<td>9.3±5.6</td>
<td>9.1±3.2</td>
<td>41±11</td>
<td>72±11</td>
</tr>
<tr>
<td>After 10 weeks</td>
<td>2.78±0.02†</td>
<td>32±5†</td>
<td>110±10</td>
<td>77±14</td>
<td>46±15†</td>
<td>72±18†</td>
<td>13.6±3.9</td>
<td>9.0±6.4†</td>
<td>9.6±4.9</td>
<td>43±9†</td>
<td>71±13†</td>
</tr>
</tbody>
</table>

* P<0.05 vs Baseline (within group, 2-tailed paired t test).
† P<0.05 between groups after 10 weeks (ANOVA).

EF% indicates left ventricular ejection fraction; SBP, systolic blood pressure; DBP, diastolic blood pressure; PAP, pulmonary artery pressure; ATVO2%, VO2 at anaerobic threshold; VEVO2, ratio between ventilation and CO2 flow; FEV1%, forced expiratory volume at 1-second expiration.

NYHA Class
There was a reduction in NYHA class in treated patients, from 2.84±0.02 to 1.78±0.02, that was not present in control patients, in whom it remained stable over time (ANOVA, P<0.05). No relationship was found between initial NYHA class and its changes after paced breathing. Most patients described a reduction in dyspnea (rather than a reduction in dyspnea class and its changes after paced breathing). Most patients observed significant improvement in several variables compared with changes in the control patients. The amount of change in ejection fraction was not related to the initial value; on the other hand, patients with higher baseline PAP showed a larger change after using the RESPeRATE device (Figure). No significant variation in these variables over the same time interval was observed in control patients.

Echocardiography
Results for blinded analysis of echocardiography variables are also shown in Table 2. In trained patients, we observed an improvement in ejection fraction, which increased from 32±6% to 39±9%, and a reduction in estimated PAP from 49±17 mm Hg to 38±9 mm Hg, both changes significant compared with changes observed in control patients. The amount of change in ejection fraction was not related to the initial value; on the other hand, patients with higher baseline PAP showed a larger change after using the RESPeRATE device (Figure). No significant variation in these variables over the same time interval was observed in control patients.

Cardiopulmonary Stress Test
After the period of paced breathing, at blinded analysis, we observed significant improvement in several variables compared to changes in the control group, including maximal workload (from 74±25 to 85±20 W), O2 pulse (from 8.5±2.4 to 9.3±2.9 mL/beat), VEVO2 (from 41±6 to 37±5). Moreover, a slight improvement in pulmonary function was observed (FEV1 from 71±14% to 78±16%) compared with changes in the control patients (ANOVA, P<0.05 versus trained group). Other variables, including peak VO2 and VO2AT, did not show significant change between groups, although improved slightly in the treated group.

Minnesota Quality of Life Test
Because there were few missing answers in the questionnaires filled in by our patients, we took advantage of a statistical method (based on Statistical Package for the Social Sciences) that allowed interpolation of missing values by considering the average of the answers provided by the whole patients group to a given item. Application of this method was possible because missing values were only a small number (less then 10%) as compared with the total number of answers provided for each item. In the respiratory training group the Minnesota Quality of Life Questionnaire score obtained after the period of home-based paced breathing was significantly lower than that obtained before treatment: the average score was reduced from 41.4±18.5 to 31.0±18.0; the between-group change was significantly better than control (Table 2). There was not a clear trend toward a reduction in any specific item; rather, there was a uniform reduction in all the items covered by the test.

Discussion
This open pilot study in patients with moderate chronic heart failure demonstrates that a short period of home-based, device-guided slow breathing improved NYHA class, some variables of exercise ventilation, pulmonary and left ventricular function, in association with a slight but significant recovering of aerobic capacity. Results were obtained through a user-friendly interactive device, applied for the first time in heart failure. Indeed, RESPeRATE has been recently approved by the FDA for the nonpharmacological treatment of arterial hypertension, because it has been shown to significantly reduce blood pressure after 2 months of regular use,18,25–27 and as a matter of fact, the device slightly reduced systolic blood pressure also in patients in the current study. An endurance training of the respiratory muscles in heart failure has been successfully used by Mancini et al.,10 who reported an increase in peak oxygen consumption, and by others using a mechanical device to strengthen inspiratory muscles and diaphragm.15 However, a randomized study using a mechanical device failed to show favorable results.13 A possible explanation for the discrepancy among data from different studies may derive from the fact that respiratory training, as generally perceived, is an intervention for increasing fitness or endurance; thus, it may be considered a special case of physical training. Obviously such training involves an effort of respiratory muscles, eg, breathing under resistive load or deep abdominal breathing: this may be fatiguing to heart failure patients, who experience an intrinsic muscular weakness.28 In designing the current study in a set of patients with low exercise tolerance, we tested an effortless breathing exercise that gradually slowed breathing rate, rather than focusing on enhancement of muscle strength and power. Slowing of breathing rate in heart failure patients had been already tested by Bernardi et al28: they demonstrated that...
1-hour of continuous or split daily home-based practice improved \( \text{O}_2 \) saturation and aerobic capacity at cardiopulmonary exercise test. The fact that we observed significant favorable results with 15-minute effortless breathing sessions coached by an enjoyable musical guide may be of practical relevance in everyday practice. A final difference with previous reports is that our patients did not display any severe breathing abnormality or blood oxygen desaturation during wakefulness. Yet, regular and slow breathing exercise determined favorable results even in this population with less advanced disease, which represents the majority of patients referred to heart failure clinics.

Rehabilitation interventions based on traditional physical exercise require at least 3 to 6 months to induce significant changes in exercise capability, autonomic tone, and survival in patients with heart failure. Remarkably, in our study, after 10 weeks of paced breathing, an increase in left ventricular ejection fraction, a reduction in pulmonary pressure, an improvement in some ventilatory measures during exercise and a reduction in lung airways obstruction were already observed. These effects might have resulted from an improvement in ventilation mechanics or from a modulation of regulatory cardiopulmonary reflexes. The analysis of autonomic modulation was not the purpose of this preliminary analysis. However, data obtained in chronic heart failure patients and in other clinical settings show that slow breathing, regardless of the method used for its implementation, significantly increased baroreflex sensitivity. Overall, we believe that a better function of the whole heart-lung complex was induced, reflected by the finding that patients’ improvement in NYHA class was frequently attributed to a reduction of dyspnea.

In conclusion, respiratory training, aimed at slowing breathing rate, is feasible and can be successfully used in the home-based rehabilitation in patients with heart failure, leading to clinical improvement. A limitation of this pilot study was that many of the parameters that were favorably modified could have been affected by patients’ knowledge of the use of the device. Thus, future larger, randomized, multicenter studies will need to incorporate a blinding algorithm to rigorously assess the value of such device, combined with more traditional rehabilitation protocols, in long-term maintenance programs.

Acknowledgments
We thank Ada Spiezia and Alice Giolo, RN, for their valuable support during patients’ training and follow-up to monitor patients’ compliance with exercise and adequate performance of breathing exercises.

Disclosures
Drs Gavish and Alter belong to the Intercure Company Ltd, producing the RESP@RATE device.

References
CLINICAL PERSPECTIVE

In heart failure, slow breathing improves autonomic cardiac regulation and reduces chemoreflex sensitivity. In this open pilot study, we explored the acceptability and usefulness of a device for paced slow breathing in 24 patients with chronic heart failure (61% males; 64±9 years; NYHA, 2.81±0.01), randomized to a control group receiving conventional treatment (n=12) or to a group receiving conventional treatment and device-guided paced breathing (n=12). Groups were comparable for age, therapy, and clinical and instrumental characteristics. At baseline and after 10 weeks, they underwent Doppler echocardiography, pulmonary function evaluation, and the cardiopulmonary stress test (CPT) and received the Minnesota Quality of Life questionnaire. Patients in the treatment group learned to use, for 15 minutes twice daily, a computerized box connected to a belt-type respiration sensor and to headphones; the device generates musical tones based on the user’s breathing rate and inspiration ratio; they guide the user to progressively slow the breathing rate to below 10 breaths per minute. The treatment group showed high compliance to the device. Blinded echocardiographic analysis demonstrated increased ejection fraction and decreased estimated pulmonary pressure in the treated group versus controls (P<0.05). Open-label analysis demonstrated favorable changes in NYHA class, Vs/VCO₂, FEV₁, and a quality-of-life measure. Thus, device-guided paced breathing at home is feasible and results in an improvement in clinically relevant parameters in patients with heart failure and systolic dysfunction.
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