Brief Rapid Communications

Slow Breathing Increases Arterial Baroreflex Sensitivity in Patients With Chronic Heart Failure

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Background—It is well established that a depressed baroreflex sensitivity may adversely influence the prognosis in patients with chronic heart failure (CHF) and in those with previous myocardial infarction.

Methods and Results—We tested whether a slow breathing rate (6 breaths/min) could modify the baroreflex sensitivity in 81 patients with stable (2 weeks) CHF (age, 58±1 years; NYHA classes I [6 patients], II [33], III [27], and IV [15]) and in 21 controls. Slow breathing induced highly significant increases in baroreflex sensitivity, both in controls (from 9.4±0.7 to 13.8±1.0 ms/mm Hg, P<0.0025) and in CHF patients (from 5.0±0.3 to 6.1±0.5 ms/mm Hg, P<0.0025), which correlated with the value obtained during spontaneous breathing (r=+0.202, P=0.047). In addition, systolic and diastolic blood pressure decreased in CHF patients (systolic, from 117±3 to 110±4 mm Hg, P=0.009; diastolic, from 62±1 to 59±1 mm Hg, P=0.02).

Conclusions—These data suggest that in patients with CHF, slow breathing, in addition to improving oxygen saturation and exercise tolerance as has been previously shown, may be beneficial by increasing baroreflex sensitivity.

Key Words: baroreflex ■ heart failure ■ heart rate ■ blood pressure ■ respiration

The protective role of a preserved arterial baroreflex in patients with chronic heart failure (CHF) or with previous myocardial infarction is now well established,1,2 and in recent years much attention has been paid to those drugs (eg, scopolamine, pirenzepine, ACE inhibitors3,4) and to those interventions (eg, physical exercise5) that are able to increase the vagal tone or the baroreflex sensitivity. A slow rate of breathing (in the range of 6 breaths/min) has several favorable effects on the cardiorespiratory system in patients with CHF: It increases resting oxygen saturation, improves ventilation/perfusion mismatching, and improves exercise tolerance by reducing the sensation of dyspnea;6 it also reduces chemoreflex activation7 and muscle nerve sympathetic activity.8 Whether slow breathing has any effect on arterial baroreflex sensitivity in heart failure, however, is still unknown.

The aim of this study, therefore, was to assess whether the arterial baroreflex can be enhanced by a slow rate of breathing (6 breaths/min) in healthy subjects and in patients with CHF. This may have practical implications because this breathing pattern can be easily learned by patients with CHF.6

Methods

We studied 81 patients with stable CHF (no changes in their signs and symptoms within the 2 weeks before examination) and 21 healthy controls. The protocol of the study was approved by local ethics committees, and all subjects gave informed consent to participate in the study. Exclusion criteria were the presence of atrial fibrillation, pulmonary diseases, or a smoking history in the previous 2 years. Clinical data for CHF patients and controls are shown in the Table. Recordings of ECG, respiration (Respitrace), and blood pressure (Pilot model, Colin Tonometry) were obtained during 5 minutes of spontaneous breathing, 4 minutes of controlled breathing at 15 breaths/min (ie, similar to the spontaneous breathing rate; for the purpose of verifying the effect of simple regularization of breathing rate), and 4 minutes of controlled breathing at 6 breaths/min.

Arterial baroreflex sensitivity was measured by spectral analysis using the “α-angle” method.5 Briefly, the gain of the arterial baroreflex was obtained by dividing the amount of fluctuation in the RR interval by the fluctuations of systolic blood pressure at the same frequency (respiration-synchronous and slow, nonrespiratory oscillations during spontaneous and controlled breathing at 15 breaths/min; 6 breaths/min unique oscillatory component during slow breathing). A mathematical function (squared coherence) was used to prove that fluctuations in the RR interval are in fact related to similar fluctuations in blood pressure. This approach gives results comparable to those obtained with the Oxford phenylephrine test.

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Results

During spontaneous breathing (breathing rates were 16.2±0.5 and 13.5±1.1 breaths/min in CHF patients and controls, respectively [P=0.021]), heart rate variability and baroreflex sensitivity were depressed in CHF patients compared with the controls (the SD of RR intervals was 23.5±2.3 versus 41.9±2.8 ms, respectively, P=0.0002). Breathing at 6 breaths/min, compared with spontaneous breathing, slightly increased overall spontaneous fluctuations in RR interval, reduced fluctuations in blood pressure, and significantly increased the baroreflex sensitivity in both CHF patients (from 5.0±0.3 to 6.1±0.5 ms/mm Hg, P<0.0025) and controls (from 9.4±0.7 to 13.8±1.0 ms/mm Hg, P<0.0025) (Figure 1). Subjects with milder CHF tended to have greater baroreflex sensitivity. The overall intergroup differences, however, were not significant (baroreflex sensitivity during spontaneous breathing: NYHA class I: 6.8±1.4; II: 5.2±0.5; III: 5.0±0.7; and IV: 3.9±0.5 ms/mm Hg; during breathing at 6 breaths/min: NYHA class I: 9.3±1.9; II: 6.0±0.6; III: 6.2±0.9; and IV: 4.8±1.2 ms/mm Hg). The increase in baroreflex sensitivity in CHF patients during breathing at 6 breaths/min remained lower compared with the increase observed in controls, and it correlated positively with the value obtained during spontaneous breathing (r=+0.202, P=0.047). The effect of controlling the breathing rate at a frequency similar to that of spontaneous breathing (15 breaths/min) did not induce significant changes in baroreflex sensitivity (Figure 1). The slow breathing rate in the CHF group also produced an increase in mean RR interval of 20 ms and a decrease in both systolic and diastolic blood pressure (systolic, from 117±3 to 110±4 mm Hg, P=0.009; diastolic, from 62±1 to 59±1 mm Hg, P=0.02) (Figure 2). No changes were observed during controlled breathing at 15 breaths/min compared with spontaneous breathing.

Discussion

Baroreflex sensitivity can be enhanced significantly by slow breathing, both in health and in the presence of CHF. This seems to occur through a relative increase in vagal activity and a reduction in sympathetic activity, as could be argued by the small reduction in heart rate observed during slow breathing and by the reduction in both systolic and diastolic blood pressures. The increase in tidal volume, which compensates for the reduced breathing rate in order to maintain minute ventilation,6,8 could be responsible for these autonomic changes through a reduction in sympathetic activity8 or via the Hering-Breuer reflex. In fact, sympathetic activity was found to increase with faster breathing rates and to decrease...
with higher tidal volumes in CHF. The increase in baroreflex sensitivity depended on the slow breathing rate and not on the regularization obtained by controlling the breathing, inasmuch as this effect was not evident when breathing was controlled at a frequency (15 breaths/min) similar to the spontaneous rate. The reduction in blood pressures observed during slower and deeper respiration (Figure 2) confirms that this finding is the consequence of a reduced afterload, rather than worsening of pump function. This more favorable sympathovagal balance also may be linked to a reduction of chemoreflex overactivity due to the reciprocal influences of these 2 reflexes. The chemoreflex actually is reduced by the slow breathing, thus adding another favorable effect on CHF.

In accordance with previous reports, the baroreflex sensitivity in patients with CHF under basal conditions was lower than that of controls, and the extent of the increase observed during slow breathing was smaller in the patients with CHF than in the controls. Interestingly, subjects with lower values at baseline tend to have smaller changes, whereas those with higher values show proportionally greater changes (as evidenced by the correlation between resting values and the increase induced by slow breathing); this probably is connected to the fact that reflex sensitivity shows a slightly skewed distribution, but it indicates that even a small increase with respect to a low initial value is clinically important.

It is noteworthy that this improvement in baroreflex sensitivity was obtained by simply modifying the breathing pattern without administration of any drug; yet the extent of the increase in baroreflex sensitivity that we observed was similar to that obtained with captopril in patients with CHF. It remains to be assessed whether these changes persist after resuming normal respiration. However, the slow breathing pattern is well tolerated by the patients; carbon dioxide is maintained within resting values, and the chemoreflex activity is not stimulated by this breathing rate. Because it does not stimulate ventilation, which may be deleterious in subjects who already have a tendency to hyperventilate, this pattern could be maintained as spontaneous and could be learned by appropriate training. Slow breathing has been found to improve resting oxygen saturation, and, possibly because of an enhanced mobilization of respiratory muscles and diaphragm, it may improve exercise capacity through a delayed onset of dyspnea and fatigue.

In conclusion, we have described a new, simple, and inexpensive method to increase the baroreflex sensitivity and vagal activity in patients with heart failure, which also increases oxygen saturation, improves the ventilation efficiency and the exercise tolerance (as previously described), and reduces sympathetic overactivity. Practicing slow and deep breathing thus can be beneficial in heart failure or in other diseases (eg, coronary disease) in which impaired baroreflex sensitivity may have adverse prognostic value.

References
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