Periodic breathing, which is a form of irregular breathing characterized by cyclic variation of ventilation at rest and exercise, has been a recognized feature of heart failure for almost 2 centuries. More recently, cyclic fluctuations in minute ventilation during exercise have been called exercise oscillatory ventilation or oscillatory breathing. During incremental exercise, when gas exchange parameters such as VO₂, VE and VCO₂ are plotted using a moving average, a systemic oscillatory pattern is evident both at rest and at low-level exercise. This finding is seen in some patients with left ventricular failure and has features similar to Cheyne-Stokes respiration. The oscillations have a period of approximately 1 min from peak to peak and are greatest at rest and during mild-to-moderate exercise. The rise in VO₂ begins first, followed by VCO₂, VE, and finally R.

Oscillatory breathing during exercise has emerged as a potent independent risk factor for adverse prognosis in heart failure patients that is additive to traditional echocardiographic and neurohumoral markers. The combined quantification of aerobic capacity (peak VO₂) and ventilatory efficacy (VE–VCO₂ slope) and oscillatory breathing appear to provide a more comprehensive insight into pathophysiology, disease severity, and prognosis. For the reasons stated above, cardiopulmonary exercise testing (CPX) is an important component in the prognostic and interventional assessment for cardiac patients.

In this issue of the Journal, Kato et al mentioned that the cycle length (CL) of oscillating VE significantly correlates negatively with peak VO₂ and positively with the VE–VCO₂ slope. Oscillatory breathing noted at rest sometimes becomes unclear or even disappears during high-intensity exercise. This would support the hypothesis that the circulation delay is an important component in the prognostic and interventional assessment for cardiac patients.

In a previous report, Koike et al defined oscillatory breathing and the apparent modifiability of oscillatory breathing during exercise, as reflected in the CPX indices, have a longer CL of oscillating VE. This finding supports the hypothesis by Murphy et al that oscillatory breathing is an important surrogate for hemodynamic impairment in patients with heart failure. Heart failure patients with exercise oscillatory ventilation (HF + EOV) demonstrated a greater degree of hemodynamic impairment at rest, as evidenced by higher resting right atrial pressure, pulmonary arterial pressure, and pulmonary capillary wedge pressure and lower cardiac index (CI) compared with heart failure patients without exercise oscillatory ventilation (HF – EOV). Exercise filling pressures were also greater in the HF + EOV group than in the HF – EOV group, and HF + EOV patients had 25% lower cumulative exercise CI compared with HF – EOV patients (P < 0.001). In multivariate analysis, exercise CI emerged as the leading predictor of EOV (odds ratio 1.39 for each 1.0 L · min⁻¹ · m⁻² decrement in CI, 95% confidence interval 1.14–1.70, P < 0.001). It is known that the oscillatory breathing noted at rest sometimes becomes unclear or even disappears during high-intensity exercise. This would support the hypothesis that the circulation delay is an important factor determining oscillatory breathing, because the circulation time becomes shorter with the increased intensity of exercise as the result of the increase in cardiac output.

Exercise oscillatory breathing is a noninvasive, easily recognizable and reproducible submaximal exercise parameter observed during CPX. The prognostic significance of exercise oscillatory breathing and the apparent modifiability of oscillatory breathing with heart failure interventions make oscillatory breathing emerging as a potent independent risk factor for adverse prognosis in heart failure patients, and has features similar to Cheyne-Stokes respiration. The oscillations have a period of approximately 1 min from peak to peak and are greatest at rest and during mild-to-moderate exercise. The rise in VO₂ begins first, followed by VCO₂, VE, and finally R.

The opinions expressed in this article are not necessarily those of the editors or of the Japanese Circulation Society. Received January 16, 2013; accepted January 16, 2013; released online January 30, 2013

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Oscillatory breathing a potentially attractive surrogate endpoint. Further work is needed to determine whether interventions such as cardiac resynchronization, intensification of neurohumoral agents, diuretics or emerging heart failure therapies will successfully attenuate exercise oscillatory breathing. Exercise training is also interesting intervention for chronic heart failure. Zurek et al first described that exercise training leads to a significant decrease of exercise oscillatory ventilation and improves ventilatory efficiency in patients with stable chronic heart failure. Further studies are also needed to determine the certain and valuable findings of exercise oscillatory breathing over serial measurements with interventions and to verify the hypothesis that exercise oscillatory breathing reversal will translate into improvement in heart failure outcomes.

Disclosure

The author has no conflict of interest to disclose.

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