



Original article

Effect of physical training on ventilatory patterns during exercise in patients with heart disease



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ABSTRACT

Background: Exercise training is known to improve the shortness of breath experienced by patients with heart disease when the ventilatory pattern becomes abnormal during exercise. However, the precise relationship between breathing patterns and the effect of exercise training has not been elucidated to date. We evaluated the relationship between the effect of exercise training on exercise tolerance and the amelioration of the ventilatory response during exercise in such patients.

Methods and results: Patients with heart disease ($n = 170$) underwent cardiopulmonary exercise testing twice (pre- and postexercise training for 3–6 months). They were divided into the exercise training group (Group E, $n = 123$) and control group (Group C, $n = 47$). Regression line relating tidal volume to respiratory rate (TV–RR slope) during a ramp protocol below the inflection point was regarded as an indicator of rapid ventilation. Tidal volume after the inflection point was regarded as an indicator of shallow ventilation (TV at plateau). The TV–RR slope and TV at plateau improved after exercise training from 94.8 ± 45.9 to 129.9 ± 69.5 ($p < 0.001$) and from 1473.6 ± 321.9 mL to 1673.2 ± 355.1 mL ($p < 0.001$), respectively, in Group E. In contrast, no improvement was evident in Group C. In total, %anaerobic threshold (%AT) [AT improving ratio = $(\text{post-AT} - \text{pre-AT})/\text{pre-AT} \times 100$] was positively correlated with both %TV–RR slope [TV–RR slope improving ratio = $(\text{post-TV-RR slope} - \text{pre-TV-RR slope})/\text{pre-TV-RR slope} \times 100$] ($r = 0.60$) and %TV at plateau [TV at plateau improving ratio = $(\text{post-TV at plateau} - \text{pre-TV at plateau})/\text{pre-TV at plateau} \times 100$] ($r = 0.51$).

Conclusion: Exercise training improved the rapidness and depth of breathing during exercise. Therefore, improvement of abnormal ventilatory patterns is correlated with exercise tolerance.

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Introduction

Exertional dyspnea and working muscle fatigue are major symptoms limiting daily activities in patients with chronic heart failure (CHF); they can be assessed objectively using incremental exercise testing with metabolic gas exchange measurements [1].

Normally, below the moderate exercise intensity during incremental work, increase in minute ventilation (VE) mainly depends on increase of tidal volume (TV). However, respiratory rate (RR) also increases simultaneously to some degree. This ventilation pattern can be elucidated by plotting TV and RR during

cardiopulmonary exercise testing using a ramp protocol [2]. Therefore, slope of TV–RR relation is not always vertical.

The steepness of this line is an indicator of rapidness of ventilation. After this point, subjects cannot breathe any deeper, and any increase in VE becomes RR-dependent. This breaking point is known as the inflection point. Any value of TV after the inflection point is an indicator of shallow ventilation. Accordingly, it is possible to quantify the rapidness and shallowness of ventilation using this plot.

During incremental exercise, patients with CHF adopt a breathing pattern that differs substantially from that of normal subjects at all levels of muscular work. In general, these patients breathe with a relatively smaller TV and a greater RR at any given VE, resulting in a characteristic downward shift of the TV–RR slope in comparison with that of normal subjects [2–6].

The effect of physical training on the ventilatory response to exercise in patients with heart disease has not yet been elucidated.

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Therefore, the aim of this study was to assess the effect of exercise training on the ventilatory pattern and the relationship between the effect of exercise training and the amelioration of the ventilatory response during exercise in these patients.

Methods

In total, 170 patients with heart disease were enrolled in this study. They were divided into two groups according to the frequency of exercise training. Group E (the exercise training group) comprised patients who participated in exercise training more than once per week, and Group C (the control group) comprised those who did not perform exercise training after enrollment in the study.

Patient characteristics and basal disease are shown in Table 1 and no differences were evident between the two groups.

Ethical approval for the study was granted by the hospital committee, and all subjects provided informed consent.

Exercise testing

Exercise tolerance was measured by a cardiopulmonary exercise test (CPX) using an upright, calibrated cycle ergometer (CPE2000, MedGraphics Co., St. Paul, MN, USA) 2–4 h after eating a light meal. The test began with 4 min of rest and 4 min of warm-up at 20 W, followed by a continuously increasing work rate of 1 W every 6 s until exhaustion. The work rate increase levels were chosen on the basis of ability of the subjects to complete an exercise program lasting between 8 and 15 min [7].

The anaerobic threshold (AT) would be determined by the V-slope method [8]. The second CPX was performed 3–6 months after the first test in the same manner.

Table 1
Patients' characteristics.

	Group E	Group C	
N	123	47	n.s.
Age (years)	63.3 ± 11.5	66.7 ± 11.7	n.s.
Height (cm)	164.3 ± 7.1	163.6 ± 7.6	n.s.
Weight (kg)	65.8 ± 11.1	63.5 ± 10.8	n.s.
Sex (M/F)	109/14	38/9	n.s.
LVEF (%)	55.7 ± 15.3	52.3 ± 17.1	n.s.
Basal disease			n.s.
IHD	92	30	
HF	31	17	
NYHA			
I	4	3	
II	13	9	
III	14	5	
Medication			
Diuretics	24	13	
BB	56	27	
ARB/ACEI	85	38	
Anaerobic threshold (mL/min/kg)	11.6 ± 2.8	13.1 ± 2.8	n.s.
VE vs. $\dot{V}CO_2$ slope	31.6 ± 5.8	30.9 ± 8.0	n.s.
Peak $\dot{V}O_2/HR$ (mL/beat)	8.8 ± 2.4	9.3 ± 2.6	n.s.

Group E, exercise training; Group C, control; LVEF, left ventricular ejection fraction; IHD, ischemic heart disease (angina pectoris, myocardial infarction) who underwent intervention before entry and had no ischemic change in cardiopulmonary exercise test after intervention.

HF, heart failure due to dilated cardiomyopathy, ischemic cardiomyopathy, hypertensive heart disease or tachycardia induced cardiomyopathy; NYHA, New York Heart Association; BB, beta-blockers; ARBs, angiotensin receptor blockers; ACEIs, angiotensin-converting enzyme inhibitors; VE, minute ventilation; $\dot{V}CO_2$, carbon dioxide production; $\dot{V}O_2$, oxygen uptake; HR, heart rate; n.s., not significant.

Gas exchange measurements

Oxygen uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and $\dot{V}E$ were measured on a breath-by-breath basis using an aeromonitor (MINATO 280E, Minato Science Co. Ltd., Osaka, Japan). The slope of the $\dot{V}E$ – $\dot{V}CO_2$ relation was calculated by linear regression analysis using the values of $\dot{V}E$ and $\dot{V}CO_2$. Because the relationship of $\dot{V}E$ and $\dot{V}CO_2$ during the period of incremental exercise alters above the respiratory compensation point, the slope of $\dot{V}E$ – $\dot{V}CO_2$ was calculated below the respiratory compensation point. The dead space is likely to be the same during the first and second CPX study, because the same type and size of face mask were used in both studies.

TV–RR relationship was calculated using a graph of TV as a function of RR (Fig. 1). The TV–RR relationship increased linearly during exercise until moderate work intensity when the linearity broke down abruptly and was followed by a horizontal line, implying RR augmentation without an increase in TV. Usually, this inflection point occurs between AT and the respiratory compensation point. We determined the earlier portion below the inflection point to evaluate the rapidness of breathing and measured the TV–RR slope by linear regression analysis as an index of the TV–RR relationship. The results showed the highest value of TV during exercise (TV at plateau) as an index of the depth of breathing [2]. Although no study has shown the normal range of the TV–RR slope, our preliminary data demonstrate that class A in the Weber–Janicki classification is equal to 90 of the TV–RR slope [9,10].

Exercise training

Exercise training was performed at the intensity of the lactic acidosis threshold which was decided using the V-slope method [8]. Patients performed supervised exercise training for 30 min/day for 3–6 months.

Data analysis

All data are expressed as mean ± standard deviation. Differences between pre- and postparameters were assessed by the paired *t*-test.

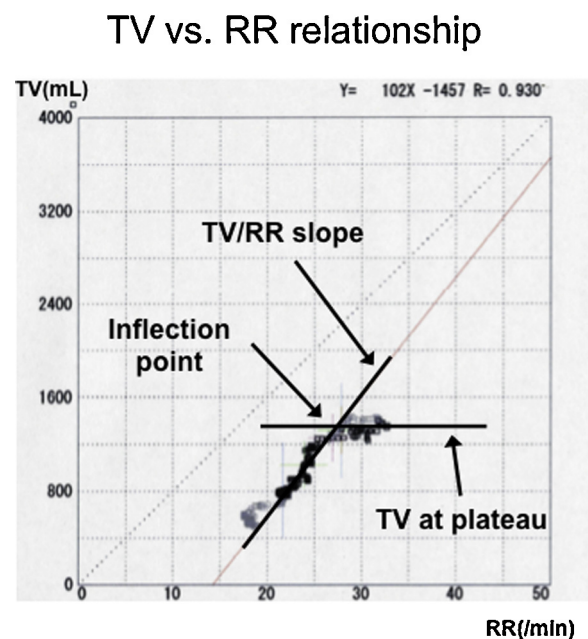


Fig. 1. Example of a tidal volume to respiratory rate (TV–RR) relationship. The patient (58 years) had dilated cardiomyopathy (ejection fraction = 41%). His exercise tolerance was preserved (anaerobic threshold = 11.3 mL/min/kg).

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