Duration of the Hemodynamic Effects of 6 Weeks Repeated Moderate Aerobic Exercise after Its Cessation

Midori Tanaka[1][a], Motoaki Sugawara[1], Yasuo Ogasawara[2], Tadafumi Izumi[1], Kiyomi Niki[3] and Fumihiko Kajiya[2]

[1]*Faculty of Health Care Sciences, Himeji Dokkyo University, Himeji, Japan*
[2]*Department of Medical Engineering, Kawasaki University of Medical Welfare, Kurashiki, Japan*
[3]*Biomedical Engineering Department, Tokyo City University, Tokyo, Japan*

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Abstract: **Purpose:** We investigated changes in hemodynamic parameters at one-week intervals for 6 weeks after the cessation of the aerobic exercise. **Methods:** A total of 20 young healthy volunteers were recruited. Using ultrasonic diagnostic equipment, we measured wave intensity (WI) in the carotid artery. The maximum value of WI during a cardiac cycle (W_1) increases with an increase in Peak dP/dt. The exercise sessions were 30 min in duration on the bicycle ergometer. Subjects were asked to make an effort to maintain the heart rate during the session at anaerobic threshold (AT) point determined during the pre-exercise cardiopulmonary exercise testing (CPET). Exercise sessions were conducted three times per week over a period of 6 weeks, and then stopped. After the cessation of the exercise sessions, WI was measured during CPET at one-week intervals for six weeks. **Results:** The work rate (WR) at AT point and at RC point, V_{O2} at RC point increased and remained increased significantly up to 2 weeks after the cessation of the exercise. Oxygen consumption (V_{O2}) at AT point also increased, but remained increased significantly only up to 1 week after. RC point W_1 increased and remained increased significantly up to 1 week after.

1 INTRODUCTION

Aerobic exercise has been reported to be one of the most effective methods to keep one's health (Racil et al., 2016) (Ozaki et al., 2015) and to help prevent cardiovascular disease (Pantelic et al., 2013). Li J et al. (Li and Siegrist, 2012) reported that high level of leisure time physical activity had a beneficial effect on cardiovascular health by reducing the overall risk of incident coronary heart disease (CHD) and stroke among men and women by 20% to 30%, while moderate level of occupational physical activity might reduce 10% to 20% risk of cardiovascular disease. However, it is not well documented how long the effects of exercise continue after its cessation. We investigated changes in hemodynamic parameters at one-week intervals for six weeks after the cessation of the aerobic exercise.

2 METHOD

2.1 Noninvasive Measurements of Wave Intensity

Wave intensity (WI) is a novel hemodynamic index, which is defined as

$$WI = (dP/dt)\cdot(dU/dt) \quad (1)$$

at any site of the circulation, where dP/dt and dU/dt are the derivatives of blood pressure and velocity with respect to time, respectively. WI was obtained from the carotid artery using a color Doppler system for blood velocity measurement combined with an echo-tracking system for detecting vessel diameter changes. The vessel diameter changes were automatically converted to pressure waveforms by calibrating its peak and minimum values by systolic and diastolic brachial blood pressures. The WI shows two sharp positive peaks (Niki et al., 2002). The first

[a] https://orcid.org/0000-0002-1730-5594

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peak (W_1) appears in early phase of LV ejection (Figure. 1). The magnitude of W_1 significantly correlates with the maximum rate of LV pressure rise which is a conventional index of contractility (r = 0.74, P < 0.001)(Ohte et al., 2003).

2.2 Cardiopulmonary Exercise Testing (CPET)

Graded cardiopulmonary exercise test (CPET) was performed by using a bicycle ergometer combined with a computerized breath-by-breath expiration gas analyzer system. The system measured tidal volume, oxygen concentration and carbon dioxide concentration, and automatically displayed oxygen consumption (V_{O2}), carbon dioxide production (V_{CO2}), minute ventilation (V_E), V_E/V_{O2}, V_E/V_{CO2}, partial pressure of end-tidal carbon dioxide ($PETCO_2$), partial pressure of end-tidal oxygen ($PETO_2$), anaerobic threshold (AT), and respiratory compensation point (RC).

2.3 Subjects

We studied 20 healthy young volunteers (9 men, 11 women, mean age 20.6 ± 0.5 years, age range 19 – 23 years). We obtained informed consent from all the subjects. The study was approved by the Himeji Dokkyo University Research Ethics Committee and conformed with guidelines of the Declaration of Helsinki.

2.4 Experimental Protocol

The exercise sessions were 30 min in duration on the bicycle ergometer. Subjects were asked to make an effort to maintain the heart rate during the session at anaerobic threshold (AT) point determined during the pre- exercise CPET.

Bicycle work rate (WR) was quantified in Watts (W). The initial work rate was 20 W and increased by 20 W every 1 minutes until limitation. The limitation criteria for the establishment of peak Vo_2 included a plateau in the Vo_2 with increasing WR, attainment of HR to 158 bpm ((220 bpm − age) x 0.8) and attainment of fatigue to the maximum, or impossibility of continuing exercise. Electrocardiogram was continuously monitored. The blood pressure was measured approximately every 30 s.

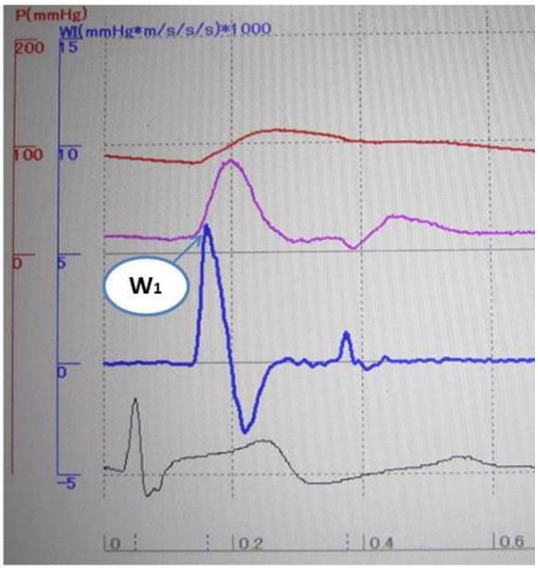

Figure 1: Representative recordings of wave intensity obtained from the carotid artery of a normal subject. Blood pressure waveform, blood velocity waveform, wave intensity waveform, and ECG are shown in order from the top. The first peak of wave intensity (W1) appears in early ejection phase.

Exercise sessions were conducted three times per week over a period of 6 weeks, and then stopped. After the cessation of the exercise sessions, measurements were performed during CPET at one-week intervals for six weeks in the same manner as the exercise session. We measured W_1, Vo_2, and Vco_2 during CPET, and analyzed at AT point and RC point.

2.5 Statistical Analysis

The obtained data were presented as mean ± SD. Repeated measures ANOVA, followed by Bonferroni test when necessary, was used to evaluate the changes after the cessation of the exercise. A value of $p < 0.05$ was considered statistically significant.

Table 1: Baseline data of the subjects.

Height [cm]	Weight [kg]	BMI [kg/m^2]	HR [bpm]	W_1 [mmHg*m/s^3]	Ps [mmHg]	Pd [mmHg]
161±6	55±9	21±2	70±9	9883±4865	112±9	72±7

HR : heart rate, W_1 : the first peak of wave intensity, Ps : systolic pressure, Pd : diastolic pressure.

3 RESULTS

After 6-weeks exercise training, Ps, Pd and HR at AT and RC point did not change significantly.

However, WR at AT point increased and remained increased significantly up to after 2 weeks. Vo_2 at AT point also increased, but remained increased only up to 1 week after (Figure. 2).

W1 at RC point increased and remained increased significantly up to after 1 week. WR and Vo_2 at RC point increased and remained increased significantly up to 2 weeks after (Figure. 3).

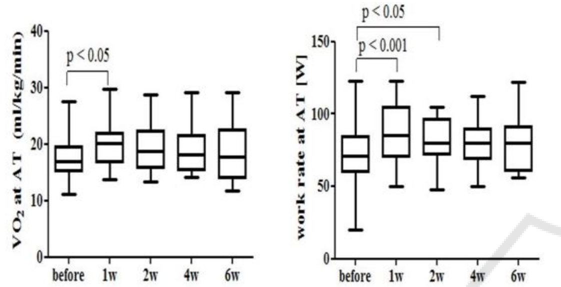

Figure 2: Changes in indices at anaerobic threshold (AT) point after the cessation of the 6 weeks exercise. Values are compared with those before the commencement of the exercise. mean ± SD.

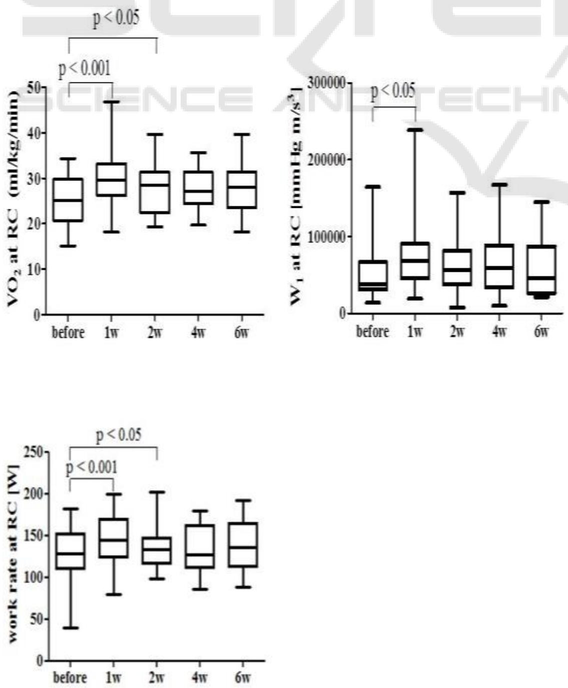

Figure 3: Changes in indices at Respiratory compensatory (RC) point after the cessation of the 6 weeks exercise. Values are compared with those before the commencement of the exercise. mean ± SD.

4 DISCUSSION

The anaerobic threshold (AT) is an index used for estimating exercise capacity. During the initial aerobic phase of CPET, which lasts until 50 – 60% of Vo_2 max is reached, minute ventilation (VE) increases linearly with Vo_2. This indicates that aerobically produced Co_2 in the muscles blood lactate levels do not change substantially during this phase, since muscle lactic acid production is minimal (Takano, 2000).

In this study of 6 weeks exercise, Vo_2 at AT point increased and remained increased significantly up to 1 week after the cessation of the exercise. WR at AT point also increased and remained increased up to 2 weeks after the cessation of the exercise. RC point is an indicator that exercise intensity has reached the physiological maximum level to start compensation for acidosis by increasing Co_2 excretion. Therefore, RC point ascent indicates increase in exercise tolerance. In our study, RC point WR, and RC point Vo_2 increased by the 6 weeks exercise and remained increased significantly up to 2 weeks after the cessation of the exercise. RC point W_1(cardiac contractility index) also increased and remained increased 1 week after.

5 CONCLUSIONS

In healthy young subjects, W_1, WR and Vo_2 were increased by intermittent, moderate-intensity aerobic exercise for 6 weeks. The effects of the exercise continued up to 2 weeks after its cessation.

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