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Exercise-induced Changes in R Wave Amplitude and Heart Rate in Normal Subjects

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Abstract: An intermittent exercise protocol on a treadmill was used to examine six healthy subjects, and a steady protocol was applied to three of the subjects before and after short-term training. The peak blood velocity in the common carotid artery increased by 73.1% during the intermittent protocol and recovered to resting level within 3 minutes, while the heart rate (HR) remained high even 5 minutes after exercise. R wave amplitude (RWA) increased significantly from 1.40 ± 0.39 mV at rest to 1.59 ± 0.33 mV ($P < .05$) immediately after the start of walking, and decreased gradually to 1.46 ± 0.36 mV ($P < .05$) during 3 minutes of walking. Thus, it decreased significantly to 1.31 ± 0.40 mV ($P < .01$) during the interphase from exercise to rest, and increased again during recovery or rest periods in the intermittent protocol. The results suggest that an increase in the venous return per heart beat at the start of walking induces the increase in RWA, and that its abrupt decrease at the end of walking induces the decrease in RWA. Subjects with a higher HR response and recovery slopes have smaller abrupt changes in RWA at the interphases between rest and walking. The gradual decrease in RWA during walking may be related to a gradual increase in HR and a gradual decrease in systemic peripheral resistance, and the gradual increase in RWA after walking may be related to a gradual decrease in HR and a gradual increase in systemic peripheral resistance. **Key words:** blood pressure, carotid blood velocity, peripheral resistance, treadmill testing, venous return.

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Materials and Methods

Subjects and Exercise Protocols

Studies were made on six healthy men with a mean age of 28.7 ± 1.73 years, a mean body mass of 63.0 ± 4.8 kg, and a mean height of 168.8 ± 6.2 cm. We explained the experimental details to them

prior to the investigation and obtained their consent prior to participation.

Two exercise protocols were used for the experiments. Protocol 1 was an intermittent protocol. Subjects were asked to take a 5-minute rest and a 5-minute recovery period standing on a sloped treadmill before and after four periods of 3-minute walking at a speed of 1.34 m/s on the treadmill at 15% grade with 1-minute rest in between (ie, 9.5 METs; 1 MET = VO_{2max} of 3.5 mL/kg/min). Protocol 2 was a steady protocol consisting of an initial 5-minute rest, walking for 20 minutes at a speed of 0.89 m/s on the treadmill at 25% grade (ie, 9.4 METs), and finally standing on the treadmill for a 5-minute recovery after the exercise. The controller gave an audible sign to subjects before starting the treadmill. The treadmill reached the speed of 0.89 or 1.34 m/s within 2 seconds. Subjects were asked to perform 2 minutes of walking on the treadmill twice to familiarize themselves with the protocols.

Experiments were conducted indoors, and the room temperature was 27°–29°C. Protocol 1 tests were conducted 2 days prior to protocol 2 tests. All subjects participated in the protocol 1 test. Three of the subjects followed a short-term (1 week) training protocol, which included 4 days of jogging for 6.0 km at a speed of 3.0 m/s (11.2 METs, 33 minutes each day), followed by 3 days of swimming for 1.0 km at a speed of 0.54 m/s (< 8.0 METs, 31 minutes each day). Results obtained for the protocol 2 tests before and after training were compared. Protocol 2 tests for each subject were conducted at the same time in the afternoon of the day before and the day after the training.

Since Simonson made the first measurement of electrocardiographic (ECG) response to treadmill testing in 1953, the ECG in treadmill exercise has become a part of routine clinical examination. Many investigators suggested that the analysis of changes in R wave amplitude (RWA) during exercise could provide additional information.[1–6] One study suggested that an exercise-induced decrease in the RWA is an adverse prognostic factor,[7] while another suggested the converse.[8] An increase in left ventricular (LV) end-diastolic volume would cause an increase in RWA.[9–12] Manaoach et al., using animal models, have linked changes in RWA with intracardiac volume changes.[13] Despite these findings, the relation between RWA and LV chamber size remains controversial.[14,15]

In contrast, Doppler ultrasound is a very important technique, not only for diagnosing cardiovascular diseases,[16] but also for making physiologic measurements.[17,18] We developed a telemetry system for measuring simultaneously both carotid blood velocity and the ECG, and presented some data on the blood velocity and the ECG measured during physical exercise and breath-hold diving.[19,20]

The purpose of this study is to reveal the mechanism of changes in RWA during exercise. The intermittent protocol was used to examine the changes in RWA, heart rate (HR), and blood velocity in the common carotid artery. Using the time-continuous recording method, the dynamic changes in RWA were analyzed synchronously with the HR in each subject. In this study, we found that the RWA changes abruptly at interphases from rest to exercise and from exercise to recovery, and that changes in RWA deviated greatly among subjects and were strongly related to the HR response. To examine the relation between changes in the RWA and the HR response, the steady protocol was applied to three subjects before and after short-term training.

Telemetry System

The telemetry system has been reported previously.[21] A brief description follows.

Recently, the 12-lead system was favored by many investigators in ECG measurement[22,23]; however, single-lead systems like CM5 and V_5 were also widely adopted,[11,24,25] and many results of RWA were presented using CM5 and V_5 lead systems.[26,27] Therefore, the simple lead system of CM5 was adopted in our telemetry system. We use two chest electrodes, E1 and E2 (VL-00-S, MEDICOTEST, Denmark), to detect the ECG signal. An additional electrode, E3, attached to the chest at an appropriate position was connected to the ground of the preamplifier.[28] Skin was cleaned with 70% ethanol and disposable electrodes with cardiac cream were attached to the chest. Adhesive foam pads (8 cm square, Nihon Kohden, Tokyo, Japan) were used instead of surgical sheets to cover the electrodes. After amplification, the ECG signal was frequency-modulated to a subcarrier of 7 kHz. Meanwhile, blood velocity in the common carotid artery was measured by Doppler ultrasound. In accordance with our previous study, a 2 MHz continuous-wave ultrasound signal was propagated to the common carotid artery, and the reflected signals from blood cells and surrounding tissues were received by an ultrasonic probe 30 × 50 × 15 mm^3 in size and 15 g in weight.[20] The ultrasonic probe was attached to the left side of the neck and fixed with a strap wound around the neck. The exact attachment position was between the fourth and fifth cervical vertebrae. The incident angle of the ultrasonic beam to the common carotid artery was 45°, and the intersectional position of the ultrasonic beam and the common carotid artery was about the seventh cervical vertebra.

The FM ECG signals were multiplexed by the discriminated Doppler signals and then transmitted by

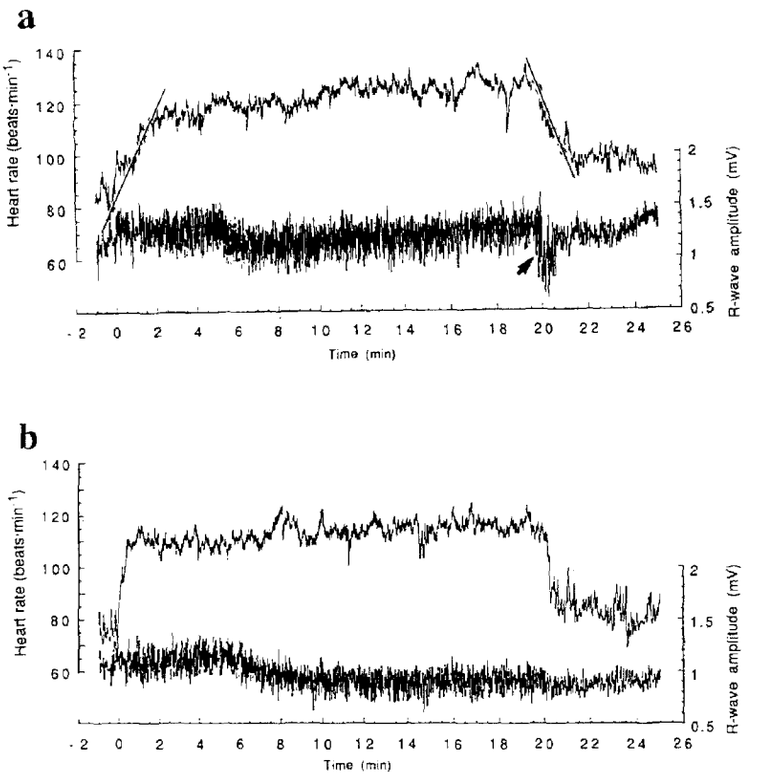

Fig. 1. Changes in the heart rate (HR) and the R wave amplitude of subject 3 during protocol 2. (a) Before the short-term training. (b) After the training. The HR response and HR recovery slopes are drawn in a. The HR response slopes are 17.0 and 62.2 beats/min^2 and the HR recovery slopes are 23.0 and 85.0 beats/min^2 in a and b, respectively. Arrow indicates sudden decrease of R wave amplitude at the stop of walking.

a 250 MHz FM transmitter (TD7001, Micro Com, Tokyo, Japan). The signals received by an FM module (RD7001, Micro Com) were stored on the audio track of video tapes synchronized with the subjects behavior on the video track recorded by a video camera (CCD-TR45, Sony, Tokyo, Japan). The Doppler signal in the audio band (200 Hz–3.4 kHz) was also used as the monitoring signal.

The ECG signal was calibrated by four calibration pulses of 1, 2, 3, and 4 mV to the cardiac electrodes.

Blood pressure of the left brachial artery was measured automatically at 1-minute intervals by an infrasound blood pressure monitor (model 4000, Yagishita Electrical, Tokyo, Japan).

Data Processing

Analog signals reproduced from the video tapes were separated into ECG FM signals and Doppler signals by two filters. After FM demodulation, the ECG signal was A/D converted (Analog ProII, Canopus, Tokyo, Japan) with a sampling frequency of 333 Hz and inputted into a personal computer (PC9801VX, NEC, Tokyo, Japan). The Doppler signals were fed into the computer at 10 kHz sampling frequency simultaneously with the ECG. Data were later transferred to a workstation (HP9000 835 SRX, Hewlett-Packard, McMinnville, OR) for further processing.

The Doppler signals were processed with a fast Fourier transformation (FFT) of 256 points with a Hanning window to show the distribution of blood flow velocity. The peak blood velocities in the systolic phase were obtained from the FFT processed data.

The R wave was detected from the ECG data on the workstation. The HR was then calculated by using the reciprocal of the RR interval. The average value of the late half cycle of the diastole was used as the baseline to compute the RWA. The measured RWA showed large oscillations at high frequency (Fig. 1). R wave amplitude data at critical phases in protocol 1 were averaged over 15 beats to minimize the respiratory variations.

Two physical parameters, HR response slope and HR recovery slope, were defined, respectively, as the rate of increase in the HR at the beginning of exercise and its rate of decrease after exercise for estimating the cardiac response ability. For protocol 2, the HR response slope and the HR recovery slope ([beats/min]/min=beats/min^2) were as follows:

(1) HR response slope = $[(HR_{max}-20)-HR_{rest}]/[t_{(HRmax-20)}-t_0]$

HR recovery slope = $[HR_{stop}-(HR_{rest}+20)]/[t_{(HRrest+20)}-t_{stop}]$

where HR_{max} in beats/min^{-1} is the maximal HR

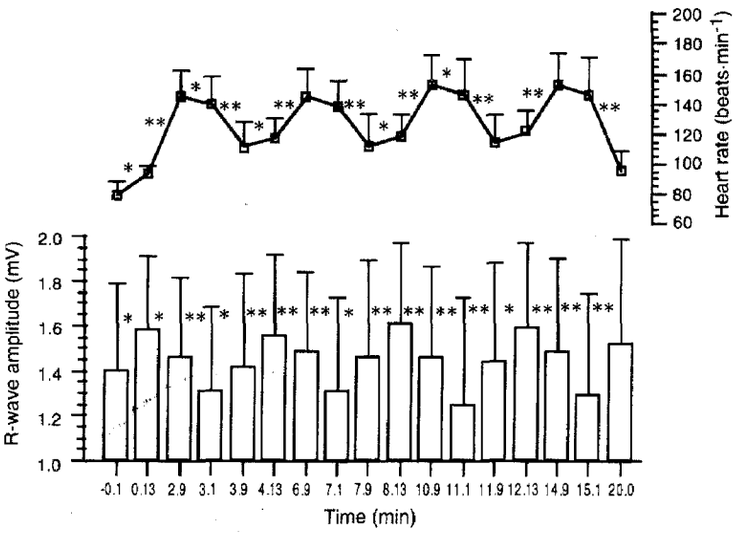

Fig. 2. Mean R wave amplitude (RWA) and heart rate (HR) with SD of the subjects at critical phases during exercise in protocol 1. Statistical comparisons for RWA and HR at rest and during exercise were examined by one-way analysis of variance ($P < .05$ or $P < .01$). The changes of RWA and HR between phases were examined by paired Student's t-test. $^*P < f.05$. $^{**}P < .01$.

attained during the exercise, HR_{rest} is the resting HR, HR_{stop} is the HR at the stop of exercise, $t_{(HRmax - 20)}$ is the time when the HR reaches $HR_{max} - 20$, t_0 is the starting time of exercise, $t_{(HRtest + 20)}$ is the time when the HR decreases to $HR_{rest} + 20$ during the recovery, and t_{stop} is the stopping time of exercise.

Data shown in Figures 2 and 3 are mean and SD values of various parameters of cardiovascular activity of the six subjects and were analyzed by a Macintosh computer using Statview (Abacus Concepts, Berkeley, CA) and SPSS (Chicago, IL) software packages. Comparisons between the values before each test and the values during or after each test were examined statistically with a one-way analysis of variance, and comparisons between two different phases were examined with Student's t-test. Results were considered significant at the 95% confidence level ($P < .05$).

Results

Changes in Heart Rate and R Wave Amplitude

An example showing changes in HR and RWA in protocol 1 is illustrated in Figure 4. The HR increased from 85 beats/min at rest to 152 beats/min at the end of the first 3-minute walking period and to a maximum of 178 beats/min during the last 3-minute walking period. The RWA increased from 1.10 mV at rest to 1.49 mV immediately after the start of walking, but decreased gradually during walking. It abruptly decreased further from 1.35 to 1.05 mV when the exercise was stopped. The maximal range of RWA change was 0.75 mV (ie, from 0.75 to 1.50 mV) between 11.0 and 13.0 minutes.

The mean RWA of the six subjects increased significantly from 1.40 ± 0.39 mV at rest to 1.59 ± 0.33 mV ($P < .05$) immediately after the start of walking,

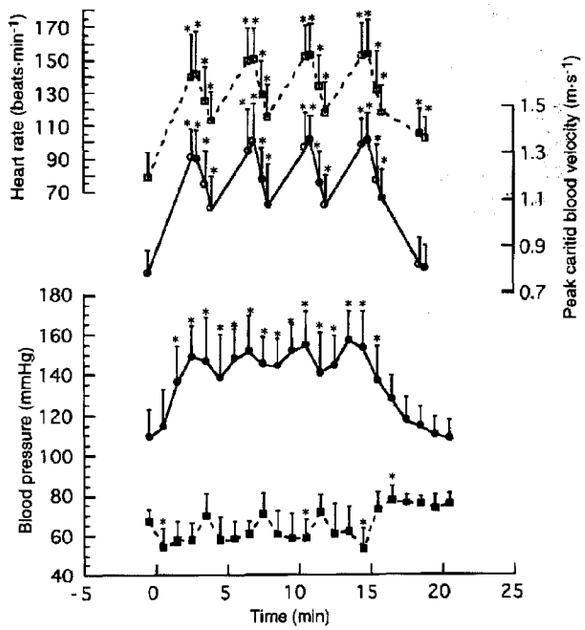

Fig. 3. Means and SDs of the blood velocity in the common carotid artery (○), heart rate (□), systolic pressure (●), and diastolic pressure (■) of the six subjects in protocol 1. $^*P < .05$ compared with the resting values (one-way analysis of variance).

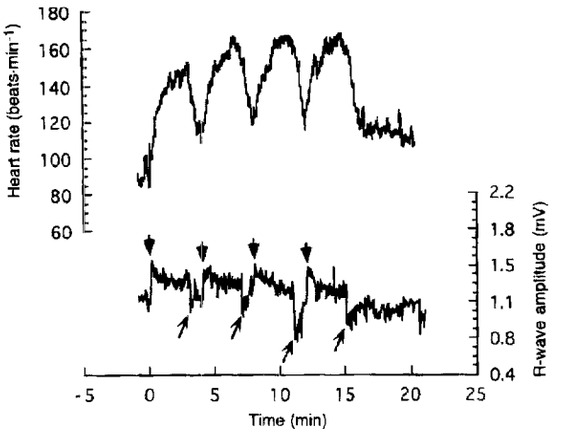

Fig. 4. Changes in the heart rate and R wave amplitude (RWA) of subject 3 during protocol 1, where the upper curve is the heart rate and the lower one is the RWA. Arrows indicate sudden increases of RWA at the start or restart of walking. Arrows indicate sudden decreases in the RWA at the cessation of walking. Walking of protocol 1 started at 0.0 minutes and stopped at 15.0 minutes. Negative value means time before the start of walking.

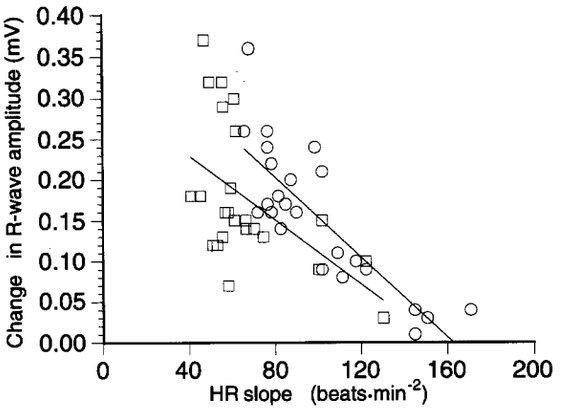

Fig. 5. Correlations between changes in R wave amplitude and heart rate (HR) slopes. Circles show results for all subjects at the starting and three restarting phases of protocol 1; squares show results for all subjects at the three intermittent stopping and stopping phases of protocol 1. Simple regressions are ○: $y = 0.40 - 0.0025x$, $R = .83$, $P < .001$; □: $y = 0.31 - 0.0020x$, $R = .53$, $P < .001$, where x is HR response slope or HR recovery slope (beats/min^2), y is change in R wave amplitude (mV) (comparing with the values of previous phases), and R is the correlation coefficient.

and decreased significantly from 1.46 ± 0.36 mV before the sudden stop to 1.31 ± 0.40 mV ($P < .01$) after the sudden stop of the first 3-minute walking period in protocol 1. The changes in RWA were reproducible at the second to fourth 3-minute intermittent walking periods ($P < .05$). The mean RWA values of the subjects at critical phases in protocol 1 are shown with the synchronous HR in Figure 2. The RWA decreased gradually from 1.59 ± 0.33 mV at 0.13 minutes to 1.46 ± 0.36 mV at 2.9 minutes during walking (the change was significant, $P < .05$), while the HR increased significantly from 91.9 ± 5.7 to 144.1 ± 17.6 beats/min ($P < .01$). After the decrease of RWA at the sudden stop, however, RWA shows a significant increase from 1.31 ± 0.38 at 3.1 minutes to 1.42 ± 0.42 mV at 3.9 minutes ($P < .05$) during the intermittent rest period, while the HR shows a continuous decrease from 138.4 ± 18.1 to 110.3 ± 15.7 beats/min ($P < .01$) during the same period. The RWA was 1.51 ± 0.48 mV at 20.0 minutes in protocol 1, still being significantly higher than that at rest ($P < .05$).

Increases in RWA at the start and intermittent restart phases and decreases in RWA at stop phases were observed for all subjects. Figure 5 shows correlations between changes in RWA and HR slopes. Deviations of change in RWA at the interphases were large, as shown in Figure 5. Subjects with large HR response and HR recovery slopes had smaller changes in RWA. Changes in RWA became smaller for each of the subjects when the HR response and HR recovery slopes became larger after the short-term training.

Figure 1 shows a typical example in which change in RWA at the phase when the subject stops walking in protocol 2 becomes smaller after the training. There was no significant difference in the maximal values of HR before and after the short-term training. After the training, the HR recovered to 80.0 beats/min (ie, the resting level), within 2.5 minutes, in contrast to a level of 95.0 beats/min at 5 minutes after walking, before the training.

Carotid Blood Velocity, ECG, and Blood Pressures

Means and SDs of the peak blood velocity in the common carotid artery (CPBV), HR, and blood pressures of the six subjects are shown in Figure 3, where the same trend of changes in the HR and CPBV during the exercise was observed. The CPBV increased from 0.78 ± 0.10 m/s before exercise to 1.35 ± 0.11 m/s (73.1% increase) at 14.9 minutes ($P < .05$) and completely returned to the resting level of 0.80 ± 0.10 m/s at 18.9 minutes (ie, 3.9 minutes after walking) when the HR was 103.0 ± 12.7 beats/min, still being significantly higher than the resting level of 78.7 ± 15.6 beats/min ($P < .05$). Unlike the HR, the systolic blood pressure (P_S) increased gradually after the start of walking. The P increased from 109.3 ± 13.3 mmHg at rest to 114.1 ± 18.8 mmHg (not significant) at 0.5 minute and to

136.3 ± 17.7 mmHg ($P < .05$) at 1.5 minutes during walking. Meanwhile, the diastolic blood pressure (P_D) decreased from 67.4 ± 5.5 mmHg at rest to 54.4 ± 8.8 mmHg ($P < .05$, t-test) at 0.5 minute during walking. The P_S showed significantly higher values during walking and intermittent rests than its resting value and returned to the resting value at about 4 minutes after walking, while the P_D showed significantly lower values during walking, but recovered even within 1-minute intermittent rests. The P_D, however, increased to 77.4 ± 7.5 mmHg after walking, being significantly higher than the resting value ($P < .05$) (Fig. 3).

Heart Response

As shown in Figure 1, the HR response slope increased from 17.0 beats/min^2 before training to 62.2 beats/min^2 after training, and the HR recovery slope increased from 23.0 to 85.0 beats/min^2. The HR response slopes of the other two subjects increased from 20.0 and 45.2 beats/min to 24.4 and 55.7 beats/min^2, respectively. The HR recovery slopes were 13.8 and 23.4 beats/min^2 before and 18.6 and 34.5 beats/min^2 after training, respectively.

The HR response and HR recovery slopes in protocol 1 as shown in Figure 5 were greater than those in protocol 2.

Discussion

The sudden onset of foot exercise in an upright position is accompanied by an increase in venous return by femoral veins, whereas a sudden stop is accompanied by a decrease. The significant increase in RWA at the starting and restarting walking phases in protocol 1 may be due to the sudden increase in venous return, whereas the significant decrease in RWA at the sudden stop phases may be due to the sudden decrease in venous return. A significant increase in RWA by 57.1% ($P < .01$) was observed when subjects submerged themselves in water in an upright position, which might be caused by an increase in venous return due to the increased water pressure that acted on the lower part of the body (He et al., unpublished observation). On the other hand, Lange et al. reported that heart size increased from 658.8 ± 46.7 mL in air to 839.1 ± 65.2 mL during water immersion in the same upright position.[29]

The short-term training protocol was used to confirm the relation between the abrupt changes in RWA and the HR responses. We obtained the same result from three subjects that changes in RWA became smaller and the HR response and HR recovery slopes became larger after the short-term training. This was the reason why we stopped enlarging the sample size in the protocol 2 test.

Changes in RWA from the same subject show different patterns in Figures 1 and 4 due to the different intensities and duration of the exercise. For low-intensity exercise (protocol 2), the RWA increased slightly within a few minutes from the start of exercise and then decreased (Fig. 4). Low-intensity exercise may be accompanied by a lower degree of muscle excitation and induce a smaller increase in venous return compared with protocol 1 at the start phase. Changes in RWA at the start and stop phases of exercise depended largely on the HR response and HR recovery slopes (Fig. 5). Larger increases and decreases in RWA occurred when the HR response and HR recovery slopes were less steep.

Changes in the cardiac response to exercise were significant even after only 1 week of training of normal previously untrained subjects. The HR response and the HR recovery slopes increased dramatically. Before training, the RWAs initially showed a large decrease, followed by an increase over 5 minutes during recovery. After training, the RWAs remained almost unchanged at the interphase between exercise and recovery, and the fluctuation of the RWAs during exercise became smaller. The sudden stop of walking was accompanied by a decrease in venous return. Since the HR recovery slope increased after training, changes in venous return per heart beat would become smaller and the RWA remained unchanged at the stop phase as a consequence.

Venous return per heart beat is analogous to LV volume at end-diastole. The results obtained here agree with the theoretic results of Brody, and the findings of Rudy et al., Daniels et al., and Feldman et al.[9–11,30]

Decreases in the diastolic peak (D) and end-diastolic foot (d) of carotid blood velocity with a simultaneous decrease in diastolic blood pressure have been observed in a steady treadmill protocol.[21] D and d in this study decreased during exercise and recovered after exercise. In reference to the significant decrease in the diastolic blood pressure, the systemic peripheral resistance (peripheral resistance of the whole body) should decrease during the exercise. On the other hand, marked increases in d were observed during breath-holding both in air and in water, which we believe is due to the vasoconstriction of the peripheral vessels (ie, increase in the systemic peripheral resistance).[31,32] Therefore, the lower the systemic peripheral resistance, the smaller the diastolic blood velocity would be and vice versa.

Organ blood flows were measured in dogs during moderate exercise by Fixler and colleagues using radioactive microspheres (reviewed by Froelicher).[33] Diaphragmatic flow increased by 500%, flow to the exercising gastrocnemius muscle by 224%, and flow to the cardiac muscle by 109% during moderate exercise. The decrease in the systemic peripheral resistance would cause an increase in blood volume in peripheral vessels and, hence, blood volume in the veins, heart, and aorta may decrease.

After abrupt increases of RWA at the start and restart of walking in protocol 1, it showed gradual decreases during walking (Figs. 2, 4). One explanation may be a decrease in the venous return per heart beat caused by gradual increases in the HR during walking. Another possibility was that the venous return decreased due to the decreases in the systemic peripheral resistance. Likewise, the gradual increase in RWA after its abrupt decrease during the recovery could be explained by the gradual decrease in HR and the recovery (increase) of the systemic peripheral resistance.

In conclusion, the HR response and recovery slopes were influenced by training, becoming much steeper in all subjects as a result. Results in this study suggest that the increase in venous return per heart beat induces the increase in the RWA and vice versa. Subjects with higher HR response and HR recovery slopes have smaller abrupt changes in RWA soon after the start and end of exercise. The gradual changes in RWA during exercise and in the recovery phase may be explained by the gradual changes in HR and in the systemic peripheral resistance.

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