



## Research Article

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# Influence of exercise capacity on cardiocomotor coupling during walking in young people

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## Abstract

**Objective:** The specific mechanisms of coupling between cardiac and locomotor rhythms remain unclear. We speculated that a decline in the function of cardiovascular and/or locomotor systems would increase the coupling of these systems to respond to the energy demand during exercise and increase efficiency. We studied mechanisms of cardiocomotor coupling and the exercise capacity in young healthy people. **Methods:** Twenty young, healthy people performed cardiopulmonary exercise testing using a bicycle ergometer to determine cardiopulmonary function. Further, the subjects underwent a treadmill walk test to record cardiocomotor coupling strength on separate days. The R2 value is an index of cardiocomotor coupling strength calculated from the step and corresponding R-R intervals during incremental speed walking. We divided subjects into two groups according to the strength of the R2 value (high and low R2 groups) and compared resting cardiac variables, autonomic indices, and cardiopulmonary exercise testing data between the two groups. **Results:** Peak oxygen uptake, oxygen pulse, and minute ventilation volume were significantly lower in the high R2 group than in the low R2 group. The high R2 group had smaller subjects with lower habitual exercise. No significant differences in other indices were observed between both groups. **Conclusions:** Our findings indicate that exercise capacity affect the dynamic interaction between cardiovascular and locomotor systems during exercise in young people.

**Keywords:** Heart rate, Exercise, Cardiopulmonary, Treadmill, Walk.

## INTRODUCTION

In humans and other mammals, the heartbeat interacts with other biological and external rhythms during rhythmic exercise [1]. Coupling between cardiac and locomotor rhythms (cardiocomotor coupling) has been observed during rhythmic exercise, such as walking, running, and cycling [2-4]. Previous studies have shown that cardiocomotor coupling could increase the efficacy of blood circulation to contracting muscles and reduce myocardial stress during exercise [5-7]. However, specific mechanisms involved in cardiocomotor coupling remain unclear. Novak et al. examined cardiocomotor coupling in elderly subjects compared with that in younger subjects during treadmill walking at incremental speeds [8]. They assumed that attenuations in respiratory sinus arrhythmia (RSA) and vagally mediated arterial baroreceptor reflex influenced coupling strength with aging. However, we reported that the coupling strength index varied, even in young, healthy subjects [9]. We hypothesized that the difference in exercise capacity for each subject would cause variations in the coupling strength between subjects. Therefore, we postulated that a decline in function of the cardiovascular and/or locomotor systems would increase the coupling of these systems to respond to the energy demand of exercise, leading to greater efficiency. We believe that measurement of coupling offers a new means to assess the function of cardiovascular and/or locomotor systems and the dynamic interactions between them.

We hypothesized that the exercise capacity of each subject impacts the dynamic interactions between the motor and circulatory systems. Therefore, we studied the relationships among cardiocomotor coupling during walking and cardiopulmonary function and cardiac variables in healthy young people.

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## MATERIALS AND METHODS

None of the participants had a history of cardiovascular disease, smoking, or any medication. Each subject provided informed consent after a verbal explanation of the purpose of the study and the experimental procedures. The experimental protocol was approved by the Ethics Committee of the Seirei Christopher University (approval number, 11070). The study was conducted following the principles of the Declaration of Helsinki. All subjects were prohibited from exercising rigorously and consuming alcohol and caffeine beverages for 24 h before the protocol. Eating and drinking were also prohibited for 3 h before the protocol.

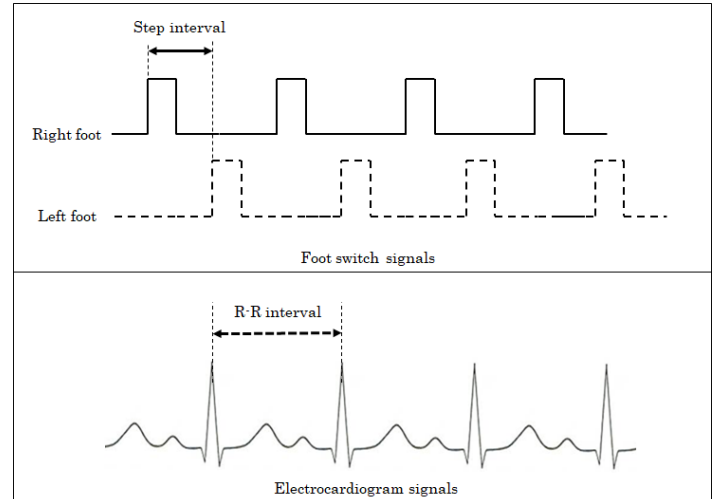
To investigate the index of the strength of cardiocomotor coupling, subjects underwent a treadmill walk test. To investigate the cardiopulmonary function, they also underwent cardiopulmonary exercise testing using a bicycle ergometer within three days. The bicycle ergometer was used for safety and quantification of exercise load.

Cardiopulmonary exercise testing consisted of a 5-min rest on the bicycle ergometer (AEROBIKE 75XLIII, Konami Sports Club Co., Ltd., Japan) followed by a 4-min warm-up at a 20-Watt load, and full exercise with a linear increase in load by 1 Watt every 3 s. Pedaling frequency was monitored during the exercise period. The subjects were instructed to pedal at 50 rpm. The incremental exercise period was continued until the subject was unable to pedal at more than 45 rpm. Oxygen uptake ( $\text{VO}_2$ ), carbon dioxide production ( $\text{VCO}_2$ ), and minute ventilation volume (VE) were measured breath-by-breath throughout the exercise testing using an expired gas analyzer (AE-310S, Minato Medical Science Co., Ltd., Japan) and calculated on a personal computer. Subjects' heart rate (HR) and blood pressure were continuously monitored during the exercise testing using a 12-lead electrocardiogram and arm-cuff blood pressure monitor (EC-12S Resting and stress test system with built-in blood pressure monitor, Labtech Ltd., Hungary). Peak  $\text{VO}_2$ /weight was calculated as an index of exercise capacity. The peak oxygen pulse was obtained by dividing the peak  $\text{VO}_2$  during the highest work load by the corresponding HR to calculate cardiac pump function during exercise. Peak VE was obtained as an index of ventilation capacity.

Subjects participated in a treadmill walk test to examine cardiocomotor coupling during walking according to a previous study [8]. To determine self-paced NWS, the subjects walked for 1 min on a floor. The subjects stood for 5 min after sitting on a chair for 3 min. The treadmill (Autorunner AR-200, Minato Medical Science Co., Ltd., Japan) speed started at 1.3 km/h and was increased by 0.3 km/h every 30 s until NWS was achieved then maintained for 6 min. R-R intervals (RRI) were measured continuously using a heartbeat sensor (FA-DL-330, 4Assist, Inc, Japan) placed on the chest. Step intervals were also measured using foot switch sensors (FA-DL-250, 4Assist, Inc, Japan) placed on both heels. Signals from these sensors were digitized at a sampling frequency of 1 kHz using a personal computer-based system (Chart 5 for Windows, ADInstruments, Australia) equipped with an analog-to-digital converter (ML880 PowerLab 16/30, ADInstruments, Australia) through an analog interface (FA-DL-720, 4Assist, Inc, Japan) (Fig. 1).

In each subject, the coefficient of determination ( $R^2$ ) was calculated from the step intervals and corresponding RRI for all walking speeds. In this study, we defined the value of  $R^2$  as the strength of cardiocomotor coupling during walking. The  $R^2$  value is maximal for strong coupling between the cardiac and locomotor rhythms and is minimal for weak or no coupling. We analyzed heart rate variability (HRV) as autonomic nerve activity from RRI data at rest in the treadmill protocol. HR at rest, standard deviation of all normal to normal RRI (SDNN), and root mean square of successive RRI differences (RMSSD) were analyzed as domain measures of HRV. The very low frequency

(VLF; direct current-0.04 Hz), low frequency (LF; 0.04–0.15 Hz), and high frequency (HF; 0.15–0.40 Hz) components of HRV were analyzed by applying a fast Fourier transform by the Welch method. The LF-to-HF ratio (LF/HF) was calculated as level of cardiac sympathetic tone. The normalized units [absolute power  $\times 100$ /(total power-VLF power)] of HF (nuHF) were also calculated to measure level of vagal nerve tone. We analyzed HR and %HR at NWS in the treadmill protocol. %HR was obtained by dividing the HR at NWS by the peak HR in the cardiopulmonary exercise testing to calculate relative exercise intensity at NWS.



**Figure 1:** Electrocardiogram and foot switch signals for the right and left foot, step interval and R-R interval

The upper waves are foot switch signals. The solid line shows right foot signal, and the dotted line shows left foot signal. Elevation of the wave indicates heel contact. The waves at the bottom of the panel are electrocardiogram signals. The sharply elevated wave is the R wave.

**Table 1:** Subject characteristics

Variable	Mean $\pm$ SD
n	20
Male / female	14 / 6
Age (year)	22.6 $\pm$ 2.4
Height (cm)	165.4 $\pm$ 7.6
Weight (kg)	57.5 $\pm$ 7.6
Body mass index ( $\text{kg}/\text{m}^2$ )	21.0 $\pm$ 2.5
Normal walking speed (km/h)	4.28 $\pm$ 0.43
Exercise habits (with/without)	11/9

## Statistical Analysis

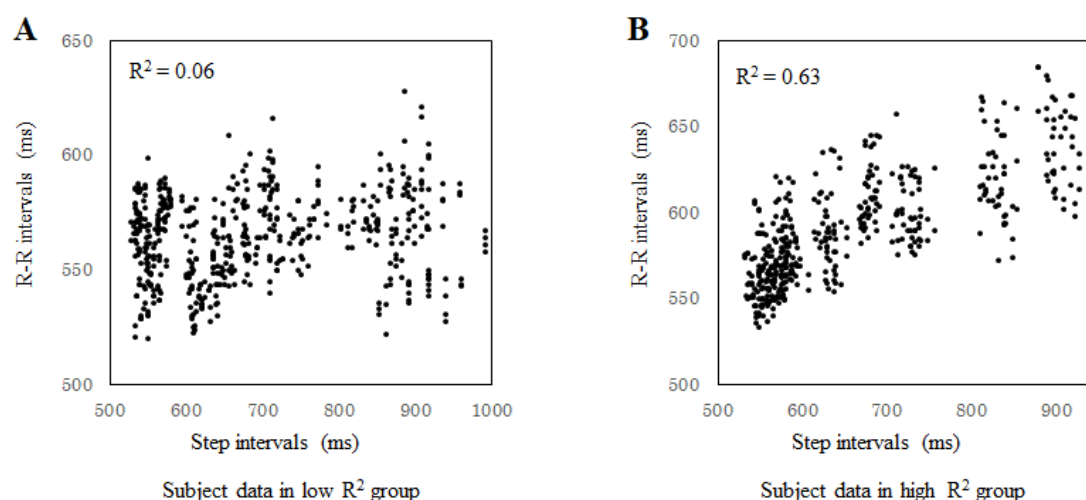
Values are shown as mean  $\pm$  standard deviation. We divided subjects into two groups according to  $R^2$  value (low  $R^2$  and high  $R^2$ ). Cardiopulmonary exercise testing and treadmill protocol data and resting cardiac variables and autonomic nerve indexes were compared between the two groups using the student's *t* test. Habitual exercise status (habitual exercise  $\geq 3$  times/week  $\geq 30$  min/session) was compared between two groups using the chi-squared distribution. Data were analyzed using the SPSS 19 (SPSS Japan, Inc., Japan). A *p*-value less than 0.05 was considered statistically significant.

## RESULTS

The mean value and standard deviation of  $R^2$  was 0.32  $\pm$  0.17 (range: 0.06–0.63), and the coefficient of variance was 0.54 (Fig. 2). All female

subjects comprised the high R<sup>2</sup> group. Results of comparison of the physiological characteristics between both groups are presented in Table 2. Peak VO<sub>2</sub>/weight, peak O<sub>2</sub> pulse, peak VE, and peak pedaling load were significantly lower in the high R<sup>2</sup> group. Therefore, the high

R<sup>2</sup> group comprised with smaller subjects with habitual exercise (X<sup>2</sup> value = 5.05, p < 0.05). There were no significant differences in the other indices between both groups.



**Figure 2:** The relationship between R-R intervals and step intervals of representative subjects in low and high R<sup>2</sup> groups

Each point corresponds to a step interval and the corresponding R-R interval for incremental walking speeds. The relationship between R-R intervals and step intervals were clearer in the high R<sup>2</sup> group subjects (B) than in the low R<sup>2</sup> group subjects (A).

**Table 2:** Comparison of physiological characteristics between groups divided by strength of cardiocomotor coupling

	Low R <sup>2</sup> group	High R <sup>2</sup> group	
R <sup>2</sup> value	0.18 ± 0.10	0.45 ± 0.12	*
Exercise habits (with/without)	8 / 2	3/7	*
Cardiopulmonary exercise testing data			
Peak oxygen uptake (ml/min/kg)	35.2 ± 5.8	27.8 ± 4.6	*
Peak oxygen pulse (ml/beat)	12.7 ± 2.6	9.2 ± 2.4	*
Peak heart rate (beat/min)	167.8 ± 10.7	168.2 ± 6.9	
Peak ventilation volume (l/min)	66.5 ± 13.7	49.1 ± 11.6	*
Peak pedaling load (watt)	186.5 ± 25.8	147.8 ± 31.6	*
Treadmill protocol data			
Normal walking speed (mile/h)	2.70 ± 0.25	2.62 ± 0.30	
Heart rate at NWS (beat/min)	97.3 ± 10.7	95.5 ± 12.5	
%Heart rate at NWS (%)	58.1 ± 6.5	56.8 ± 7.0	
Resting cardiac variables and autonomic indexes			
Systolic blood pressure (mmHg)	107.8 ± 8.36	102.3 ± 12.5	
Diastolic blood pressure (mmHg)	67.3 ± 8.56	65.8 ± 7.86	
Heart rate (beat/min)	73.4 ± 8.4	69.2 ± 11.4	
LF (ms <sup>2</sup> )	930 ± 407	567 ± 481	
HF (ms <sup>2</sup> )	2135 ± 2343	1619 ± 1469	
LF/HF	0.97 ± 0.73	0.68 ± 0.69	
SDNN (ms)	60.7 ± 22.3	57.0 ± 21.0	
RMSSD (ms)	48.6 ± 26.2	55.5 ± 24.6	
nLF (%)	41.5 ± 19.2	30.7 ± 19.1	
nHF (%)	55.6 ± 19.8	62.0 ± 19.8	

\* P < 0.05 compared with the low R<sup>2</sup> group

R<sup>2</sup> value is index of strength of cardiocomotor coupling calculated from the step intervals and corresponding R-R intervals during incremental speed walking.

## DISCUSSION

The study results indicate that the exercise capacity of each subject impacts the dynamic interactions between motor and circulatory systems. Indices of exercise capacity, cardiac pump function, and ventilation capacity were significantly lower in the high  $R^2$  group. In addition, this group comprised subjects who had lower habitual exercise. These results suggested that young people with a low cardiopulmonary function and sedentary life have stronger coupling between cardiac and locomotor rhythms during exercise.

Novak *et al.* (2007) quantified cardio-locomotor coupling using the same method as in the present study and reported that the  $R^2$  value was  $0.15 \pm 0.16$  in the young group [8]. The  $R^2$  value in our study is high in comparison to their results. The reason for this is unknown, but could be attributed to differences in subject characteristics. These different results suggest that aging is not central to strong coupling between cardiac and locomotor rhythms.

There are two possible hypotheses to explain the physiological mechanisms responsible for cardiac activity from locomotor rhythms during coupling and/or synchronization [10]. The first hypothesis is that ascending signals from intramuscular mechanoreceptors modulate heartbeat intervals [11]. The second hypothesis is that the stretching stimulation caused by diastolic volumetric loading in the atrium shortens the heartbeat interval without modifying the autonomic nervous activity. This phenomenon is known as mechano-electric feedback, as the intrinsic property within the myocardium [12]. These two factors would increase SBP and shorten RRI in response to skeletal muscle mechanoreceptors and/or mechano-electric feedback in the atrial wall. Novak *et al.* (2007) speculated that attenuations in RSA and vagally mediated arterial baroreceptor reflex with aging increased the coupling strength [8]. They also suggested that the baroreceptor reflex inhibited the SBP elevation from beat-to-beat, prolonged RRI, and increased RRI variability [13, 14]. In addition, RSA increased RRI variability [15]. Thus, Novak *et al.* concluded that with the attenuation of autonomic feedback mechanisms, such as baroreceptor reflex and RSA with aging, the effect of muscle contraction may exert a stronger effect on the heartbeat [8].

Although the present study included only young subjects, the  $R^2$  value varied. Kaushal and Taylor (2002) reported that the neural baroreceptor reflex component was more strongly related to RSA in older but not in younger subjects [16]. Therefore, we should focus on RSA and baroreceptor reflex separately. Firstly, in the present study, indices of RSA and vagal nerve tone at rest were not different between the two groups. On the other hand, Niizeki *et al.* (1993) observed that the incidence of coupling between cardiac and locomotor rhythms during walking was associated with blunted RRI variability [3]. In addition, we reported that the standard deviation of HR during walking was significantly lower at inducing coupling between the two rhythms than walking at self-cadence [9]. However, we did not measure the parasympathetic nerve activity during exercise. The strength of coupling could be affected from RSA not at rest but at exercise.

Secondly, Komine *et al.* (2009) reported that arterial baroreceptor reflex, a neural component, and exercise tolerance were greater in exercise-trained than sedentary young men [17]. They suggested that endurance exercise training in young men enhances arterial baroreceptor reflex not owing to changes in vessel wall compliance of the carotid artery but owing to changes in the neural component of the baroreceptor reflex arc. In the present study, the high  $R^2$  group was more sedentary and had lower exercise tolerance. These results could explain the decrease in the neural component of the baroreceptor reflex in the high  $R^2$  group. Our results indicated that the neural component of the baroreceptor reflex was partially responsible for the coupling between cardiac and locomotor rhythms.

Our results could not reveal the other factors, such as afferent signal from the mechanoreceptor of skeletal muscle, mechano-electric feedback in sinus node, and venous return controlled by skeletal muscle and respiratory pump function, for the occurrence of coupling between cardiac and locomotor rhythms. Further research should investigate the mechanisms involved in coupling.

Numerous studies [5-7] have shown that cardio-locomotor coupling could enhance the efficacy of blood circulation to contracting muscles and reduce myocardial stress during exercise. Our findings support the theory that people with low fitness levels have a tendency for strong dynamic interactions between locomotor and cardiovascular systems during exercise. Our findings and those of previous studies implicate that cardio-locomotor coupling/synchronization could be a compensatory mechanism for exercise maintenance. This phenomenon could be used as an evaluation and intervention method for training and rehabilitation.

Our study has five limitations: (1) Arterial baroreceptor reflex and autonomic nerve activity were not measured during walking. It was unclear what affected the coupling strength. (2) The respiratory signal was not recorded during walking. Respiration could indirectly influence the occurrence of coupling because RSA modulates heartbeat through neural effects [18] and ventilation modulates venous return through the movement of the diaphragm [19]. (3) There was a partiality regarding gender in the result. Novak *et al.* (2007) reported that the cardio-locomotor coupling was associated with age, but not with sex, body mass index, NWS, or the rate of perceived exertion during walking [8]. Therefore, this partiality could not be due to the gender bias but due to the difference of fitness level and baroreceptor reflex responsiveness [20]. (4) We adopted associations between step and RRI as the methods for estimating the coupling strength to obtain spontaneous coupling. However, the high correlations do not imply that the heart and step rates are synchronized. (5) Further studies on patients with other conditions, such as those with cardiovascular diseases, are necessary to confirm our results because our study included only healthy young subjects.

## CONCLUSION

Our findings suggest that exercise capacity affects the dynamic interaction between cardiovascular and locomotor systems during exercise. Cardio-locomotor coupling could be employed to maintain load-bearing exercise, especially in people with low fitness levels.

## Conflicts of interest

The authors declare no conflicts of interest.

## Authors' Contribution

All authors equally contributed to designing the study. Takeuchi and Yokoyama collected data. Takeuchi analyzed the data and wrote the manuscript.

## Acknowledgements

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