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EFFECT OF WORK RATE ON CARDIORESPIRATORY RESPONSE TO RHYTHMIC-STATIC EXERCISE

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Influence of work rate (30 and 30 rpm) on exercise hyperpnoea, respiratory entrainment and cardiovascular system was studied in 9 healthy men performing rhythmic-static exercise (RSE). Respiratory frequency (f), tidal volume (VT), minute ventilation ($\dot{V}E$), heart rate (HR), stroke volume (SV), and cardiac output ($\dot{Q}$) were continuously measured. RSE was performed in upright position on a special motor-driven cycloergometer with an intensity of 40% $\dot{V}O_{2max}$ for 5 min. The subjects opposed the flywheel movement by pressing the pedal alternately with left and right leg. It was found that in both work rates respiratory frequency followed the rhythm of exercise. The increases in f (28v35 breaths/min. $p < 0.05$) were associated with decreased VT (1.3v1.0L, $p < 0.05$) but they did not influence $\dot{V}E$ which was 33 and 36 l/min (NS). Accelerations of f and $\dot{V}E$ were faster for 30 than 60 rpm reaching respective values of 2.70v0.75 breaths/min/s ($p < 0.05$), and 0.59v0.31 l/min/s ($p < 0.05$). Cardiac response and its kinetics were found to be similar for both exercise rhythms. It is concluded that breathing entrainment does not affect either ventilation or the cardiac response during the RSE exercise. Since changes in acceleration of ventilation were not accompanied by appropriate changes in cardiac output acceleration the cardiodynamic hypothesis of exercise hyperpnoea does not seem to be valid for rhythmic-static exercise.

K e y w o r d s: *rhythmic static exercise, hyperpnoea, respiratory entrainment, cardiovascular system.*

INTRODUCTION

Physical activity generally requires some combination of dynamic (isotonic) and static (isometric) exercises. However, static exercise differs from dynamic exercise not only in the type of muscle contraction but also in its physiological consequences such as blood supply to the working muscles, oxygen utilization, removal of metabolic end products, blood pressure response and time of performance. The problems with different response of the cardiorespiratory

system to dynamic and static work can partly be avoided by adopting a rhythmic-static exercise, which could be defined as an isometric contraction interrupted periodically by repeated relaxations (1).

Performance of any exercise requires increased activity of respiratory and cardiovascular system to match the increased metabolic demand (2). Krogh and Linghard (3) were the first to propose a hypothesis of „irradiation" of the command to exercise from the cerebral cortex to the cardiovascular and respiratory centres. Later, Dejours (4) postulated that the reflex from moving lims could be responsible for fast adaptation of the cardiorespiratory system to exercise. On the other hand, Wasserman et al. (5) have suggested that a sudden increase in minute ventilation ($\dot{V}E$) at the start of dynamic exercise might be linked to the concommitant increase in pulmonary blood flow caused by a sudden increase in cardiac output ($\dot{Q}$). The cardiodynamic hypothesis of Wasserman suggests some sequence of events in which an increase in cardiac output should precede, or be at last parallel, to the increase in minute ventilation. The aim of this study was, therefore, to verify the cardiodynamic hypothesis during rhythmic-static exercise.

Considering the cardiorespiratory function during exercise performed with some rhythm one should take into account the entrainment effect on the respiratory system. Entrainment of two periodic events implies an active interaction in which one oscillator captures the frequency of another (6). This definition indicates that in humans performing cycle exercise (dynamic or static, interrupted by some relaxation periods) the synchronization of the frequency of breathing with the frequency of exercise can occur. In fact, the spontaneous entrainment of breathing to the exercise cycle has been enlarged during rhythmic exercise (7, 8). Thus, the secondary aim of this study was to test whether the entrainment of respiratory system can affect the cardiovascular response to rhythmic-static exercise performed with the same relative intensity but with different rhythm.

METHODS

Nine healthy male subjects of a mean age 21.8 years, SD 0.7, height 169 cm, SD 6, and body mass 60.4 kg, SD 8.5 volunteered for this study. The subjects were university students and none of them had a history of circulatory or respiratory diseases. The subjects were instructed on experimental procedure and informed consent was obtained.

Experimental protocol consisted of a rhythmic-static exercise (RSE) performed on a cycle ergometer (Lode, Groningen) in the upright position. Two additional pre-tests were performed. In the first, the maximum aerobic capacity ($\dot{V}O_{2max}$) of the subjects was determined by the indirect method of Åstrand-Ryhming (9). On the next day the intensity of RSE was determined by adjusting the pedal torque to the level which required oxygen consumption ($\dot{V}O_2$) equal to 40% of $\dot{V}O_{2max}$. In the following two days the subjects performed the RSE with a rhythm of 60 and 30 rpm, respectively.

The RSE was performed using a specially modified cycle ergometer (10). The flywheel of the ergometer was driven by an electric motor. The pedal torque was individually adjusted for each

subject to the level corresponding to 40% $\dot{V}O_{2max}$, as already mentioned. The direction of pedal rotation was changed periodically, i.e. clockwise or anti-clockwisse, with a rhythm of 60 or 30 rpm. The subject was asked to oppose the flywheel movement by pressing the pedal alternately with left and right leg at an angle parallel to the horizontal plane for 5 min. The exercise started from the rest on an acoustic signal.

Stroke volume (SV), heart rate (HR) and cardiac output $\dot{Q}$) were continuously determined by an automated measuring system based on impedance cardiography. A detailed description of the system and evaluation of its accuracy has already been published (1).

Arterial blood pressure, systolic (SBP) and diastolic (DBP), were measured by the conventional cuff method in the left forearm of the subjects during each 4th min of the rest, exercise and recovery periods.

Respiratory frequency (f), tidal volume (VT) and minute ventilation ($\dot{V}E$) were measured automatically by a second computer system breath-by-breath (11). The subjects expired through a hotwire type pneumotachograph (RF-2, Minato, Tokyo) with a small dead space of 10 ml. Output signals from the pneumotachograph were integrated numerically and converted into a calibrated volume signal. The VT was determined by measurement of expired volume, whereas *f* was calculated by taking the reciprocal of two subsequent breathing cycles. The $\dot{V}E$ was then obtained multiplying *f* by VT.

The subject's $\dot{V}O_2$ was determined during the 4th min of RSE as well as during the rest before and after exercise, using the standard procedure of an open circuit system. Expired gas was collected to a Douglas bag and the composition of the gas was analyzed using a gas monitor (1H21A, NEC-Sanei, Tokyo).

Digital data of HR, SV, $\dot{Q}$, f, VT and $\dot{V}E$ were converted into calibrated analog signals and continuously recorded on an eight-channel chart recorder (Rectigraph 8k, NEC-Sanei, Tokyo). Since the cardiorespiratory variables generally exhibit an exponential time course during the onset of exercise a time constant was used to describe the kinetics of these variables. The time constant (τ) was defined as the time required to change from the control pre-exercise value to 63.2% of the new steady-state values obtained during exercise or recovery periods. To describe the rapidity of the cardiorespiratory responses a new parameter was introduced ($\Delta\tau/\tau$), defining ($\Delta\tau$) as a magnitude of each variable attained at τ. The coefficient of $\Delta\tau/\tau$ is a measure of acceleration at the beginning of exercise (or ON response), and negative acceleration (deceleration) for the recovery from exercise (or OFF response).

All data were calculated from individual chart records. The data, presented as means and SD, were analyzed by Student's paired t-test.

RESULTS

The mean $\dot{V}O_{2max}$ of the subjects was 2.92 1/min, SD 0.55. Their $\dot{V}O_{2max}$ uptake during the RSE was 1.20 1/min, SD 0.27 (41% $\dot{V}O_{2max}$) at 60 rpm, and 1.18 1/min, SD 0.18 (40% $\dot{V}O_{2max}$) at 30 rpm.

Ventilatory responses

Time courses of frequency of breathing (*f*), tidal volume (VT) and minute ventilation ($\dot{V}E$) during RSE are presented in *Fig. 1*. After a sudden increase a stabilization in *f* was observed at the level of 28 breaths/min for the first 2.5 min of both exercises. However, during RSE of 60 rpm a further increase in

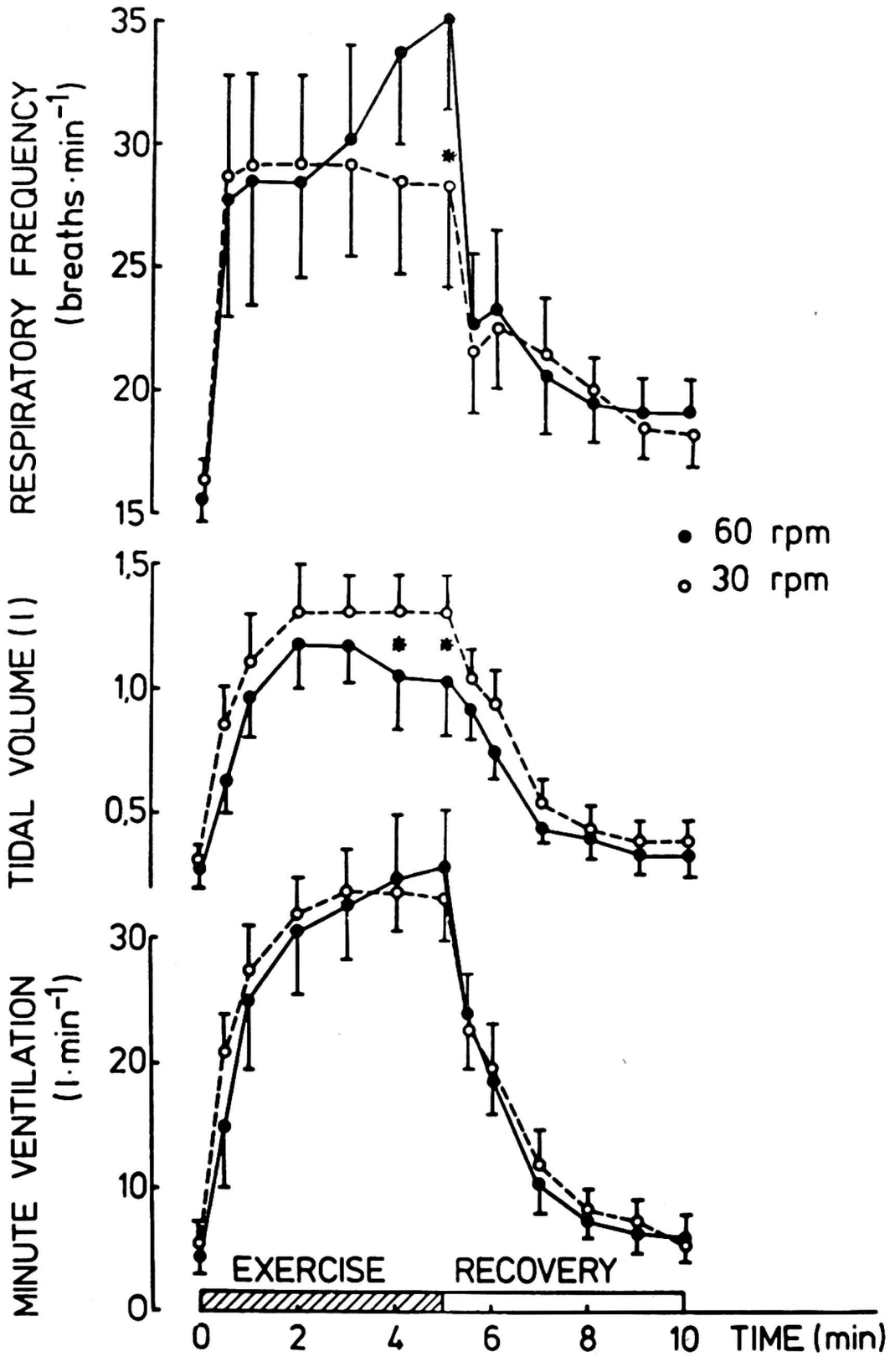

Fig. 1. Respiratory frequency, tidal volume, and minute ventilation during rhythmic-static exercise (RSE) performed at a rhythm of 30 and 60 rpm. Values are means and SD; * $p < 0.05$, paired t-test.

f appeared reaching 35 breaths/min at the end of exercise. The increment of f and its time constant were significantly greater at 60 than at 30 rpm (*Tab. 1*). However, acceleration of f for ON response at 60 rpm was over 3 times slower than that observed during exercise at 30 rpm.

Table 1. Kinetics of ventilatory variables determined at the beginning (ON) and the end (OFF) of the rhythmic-static exercise (RSE).

VARIABLE	RSE		ON			OFF		
	rpm		Δ	τ	Δτ/τ	Δ	τ	Δτ/τ
f	30	mean	13.7	10.4	2.70	11.7	16.1	0.88
		SD	9.0	7.2	1.70	6.5	10.4	0.98
	60	mean	20.4 *	39.2 *	0.75 *	17.2 *	28.1	1.07
		SD	11.9	23.2	0.35	7.8	42.4	1.00
VT	30	mean	1.01	48.8	0.02	0.95	68.6	0.012
		SD	0.55	24.7	0.01	0.56	24.7	0.013
	60	mean	0.80	57.2	0.02	0.73	81.2	0.008
		SD	0.58	40.1	0.02	0.56	32.0	0.011
$\dot{V}E$	30	mean	28.1	39.8	0.59	26.9	69.8	0.41
		SD	9.5	15.2	0.23	8.9	28.5	0.54
	60	mean	31.2	75.4 *	0.31 *	29.9	69.3	0.31
		SD	12.6	24.0	0.13	12.6	17.9	0.21

Δ increment or decrement in respiratory frequency *f* (breaths/min), tidal volume VT (1), and minute ventilation $V\dot{E}$ (1/min) for ON and OFF response; τ, time constant (s); Δτ/τ acceleration or deceleration in *f* (breaths/min·s), VT (1/s), $\dot{V}E$ (1/min·s). * $p < 0.05$, 30v60

The increase in *f* during the second half of RSE at 60 rpm was reflected in a significant decrease in VT. However, the interplay between *f* and VT did not influence the amplitude of $\dot{V}E$.

For ON response of $\dot{V}E$ time constant was greater and acceleration was slower at RSE 60 than 30 rpm (*Tab. 1*). Kinetics of the ventilatory variables for OFF response did not reveal significant differences between 60 and 30 rpm, except the amplitude of *f*, which was greater at 60 rpm.

CARDIAC RESPONSES

Time courses of heart rate (HR), stroke volume (SV) and cardiac output ($\dot{Q}$) during RSE at 30 and 60 rpm are presented in *Fig. 2*. HR increased more but SV and $\dot{Q}$ increased less at 30 than 60 rpm. These differences were, however, insignificant. No differences were found in the kinetics of the cardiac variables between the two work rates (*Tab. 2*).

The SBP increased during both exercise tests from 116 mmHg, SD 12 to 180 mm Hg, SD 32 ($p < 0.01$) at 30 rpm, and from 125 mmHg, SD 14 to

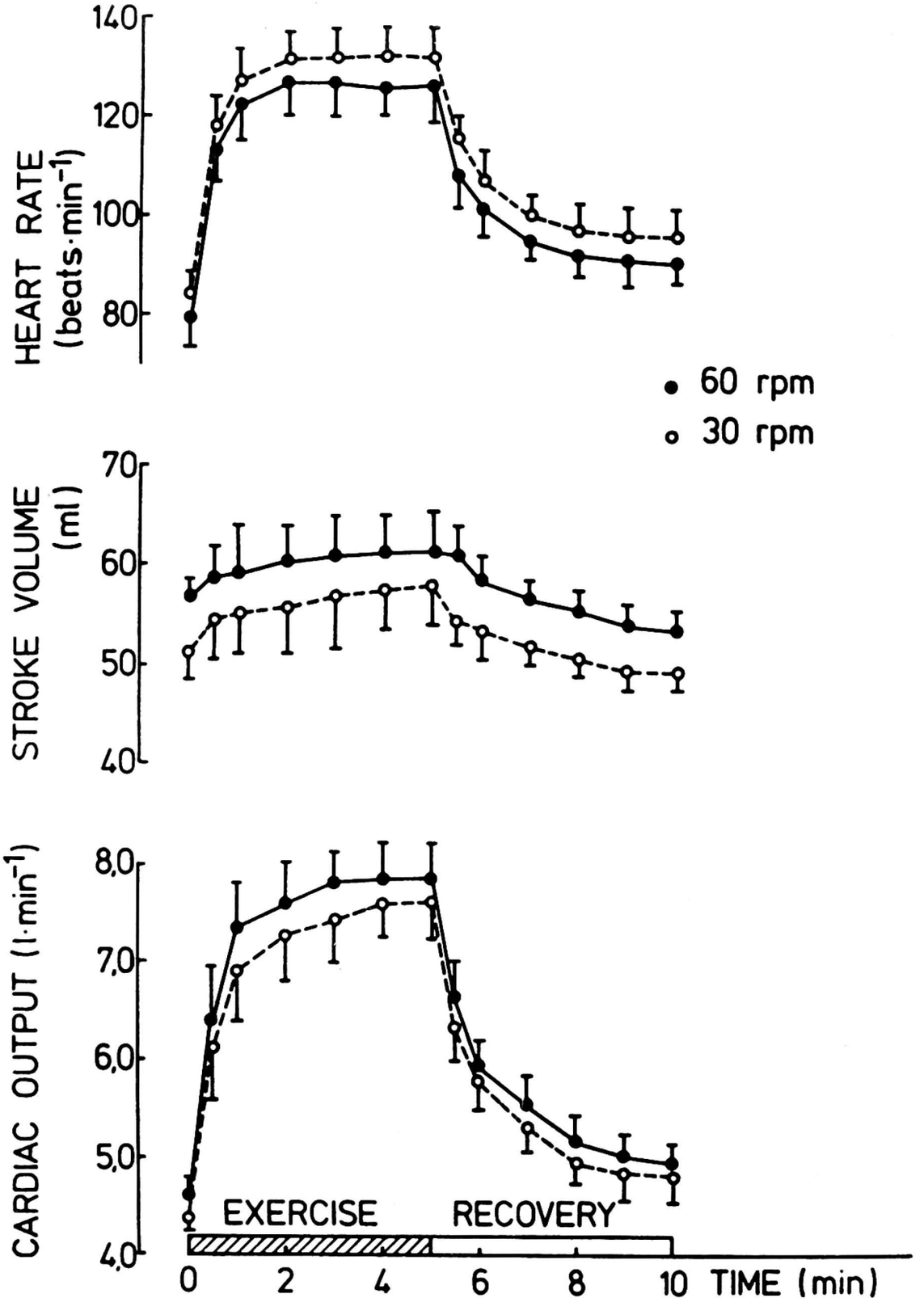

Fig. 2. Heart rate, stroke volume, and cardiac output during rhythmic-static exercise performed at a rhythm of 30 and 60 rpm. Values are means and SD.

197 mm Hg, SD 34 ($p < 0.01$) at 60 rpm. Also DBP increased during the exercise from 86 mmHg, SD 9 to 96 mmHg, SD 13 ($p < 0.05$), and from 78 mmHg, SD 8 to 95 mmHg, SD 20 ($p < 0.05$), respectively. The increases in SBP and DBP did not differ significantly in the experimental situations.

Table 2. Kinetics of cardiac output determined at the beginning (ON) and the end (OFF) of the rhytmnic-static exercise (RSE).

VARIABLE	RSE		ON			OFF		
	rpm		Δ	τ	Δτ/τ	Δ	τ	Δτ/τ
HR	30	mean	47.0	20.8	1.77	36.2	47.0	0.59
		SD	12.2	12.7	0.95	10.3	24.7	0.37
	60	mean	46.7	24.0	1.85	34.9	36.9	0.64
		SD	16.4	18.8	1.20	14.8	17.2	0.22
SV	30	mean	6.5	44.3	0.21	12.1	31.1	0.25
		SD	11.5	26.4	0.12	6.4	17.7	0.10
	60	mean	6.5	59.1	0.12	13.1	63.9	0.14
		SD	9.8	29.8	0.09	7.2	35.9	0.09
$\dot{Q}$	30	mean	3.3	56.1	0.05	2.8	51.7	0.04
		SD	0.6	45.6	0.03	0.5	27.4	0.02
	60	mean	3.3	57.0	0.04	2.9	63.8	0.04
		SD	0.9	25.6	0.02	0.9	38.6	0.03

Δ increment or decrement in heart rate HR (breaths/min), stroke volume SV (ml), and cardiac output $\dot{Q}$ (l/min) for ON and OFF response; τ, time constant (s); Δτ/τ acceleration or deceleration in HR (breaths/min·s), SV (ml/s), and $\dot{Q}$ (l/min·s).

DISCUSSION

The present study showed some evidence that acceleration of $\dot{V}E$ at the beginning of the rhythmic-static exercise was faster at 30 than 60 rpm, whereas the acceleration of $\dot{Q}$ was almost the same at both exercise rhythms. The results lead to the conclusion that the increase in VE is not related to the appropriate increase in $\dot{Q}$. It seems, therefore, that cardiodynamic hypothesis for exercise hyperpnoea cannot be considered as a valid at last for the rhythmic-static exercise. This opinion is in agreement with our previous study (1) and has been recently supported by the results of Ishida et al. (12) who observed that for passive leg movement the afferent drive from moving limbs could produce an increase in ventilation without any chang in $\dot{Q}$.

It was also found that breathing entrainment does not influence the cardiac response during rhythmic-static exercise. Furthermore, it had only an impact on tidal volume, with no effect on ventilation. It may be concluded, therefore, that within the range of moderate rhythmic-static exercise, the breathing entrainment plays only a little role in human exercise performance. However, during maximal exercise the coupling between breathing and the exercise rhythm might be of great importance because of an excessive work of respiratory muscles (13).

Majority of normal subjects presents some symptoms of respiratory entrainment while walking (6), cycling (13) or free running (14). The locomotive neural activity may be derived from central command, spinal generators, or peripheral feedback as a one of several inputs to the central respiratory pattern generator (10). According to Hill et al. (6) during walking these input signals are insufficient to capture the breathing pattern, whereas during running they could provide stimulus strong enough to do so. Entrainment has been reported to occur more readily in humans by the use of metronome for pacing, suggesting important involvement of cortical factors in this phenomenon. Our results confirm this opinion. The applied experimental protocol, in which the subjects opposed the pedal torque without leg movement, needed a high level of mental concentration and alertness of the subjects. This mental mobilization would supposedly increase the effect of cortical "irradiation" on the respiratory centres.

Dynamic parameters of cardiac variables did not reveal any significant differences between RSE of 30 and 60 rpm. The increase in cardiac output occured in both cases mainly on the expense of heart rate, since stroke volume increased very little. Almost parallel increases in arterial blood pressure suggest that total peripheral resistance was near the same during RSE of 30 and 60 rpm. It can be concluded, therefore, that intramuscular pressure developed during rhytmic-static exercise is not related to the frequency of muscle contractions. In fact, the ratio of time of muscle contraction to the muscle relaxation was the same (50 : 50) during both work rates. Thus, in the situation of static contraction with lack of leg movement and impaired venous return the available time for muscle relaxation could decide on the strain of the cardiovascular system.

It has been previously demonstrated that rhythmic-static exercise causes faster adaptation of cardiac function to steady-state than dynamic exercise (1). This finding has recently been confirmed by McCoy et al. (15) who presented data indicating that combined static effort (handgrip performed with an intensity of 30% maximum voluntary contraction force) with dynamic exercise (cycle ergometer exercise performed with an intensity of 50% $\dot{V}O_{2max}$) may enable faster achievement of cardiac steady-state than dynamic exercise alone. These results suggest that static or rhythmic-static exercise of moderate intensity could potentially enhance dynamic work or athletic performance.

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