Cardiopulmonary Response during Supine and Sitting Bicycle Exercises

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Abstract. The effect of body position on cardiopulmonary response was assessed by the following protocols:
1) Cardiopulmonary exercise testing with a bicycle ergometer (20 W/min) was performed on nine healthy men (mean age; 19.9 years) in a sitting and a supine position. Oxygen uptake, heart rate and blood pressure were measured during the test. Noradrenaline and Angiotensin II were analyzed at rest and after exercise.
2) Single-level exercise testing at 100 W was performed. The cardiac index was computed from the cardiac output, which was measured using the dye-dilution technique at rest and during exercise.
  The results were as follows:
1) Anaerobic threshold was lower in the supine than in the sitting.
2) Noradrenaline and Angiotensin II were slightly lower in the supine than in the sitting.
3) The cardiac index at rest was slightly greater in the supine, and that during 100 W exercise was the same in both positions.
  It is concluded that the blood flow to active muscle during 100 W exercise is lower in the supine than in the sitting. The etiology of lower anaerobic threshold in the supine is thought to be due to lowered blood flow to active muscle.
Key words: Cardiopulmonary response, Body position, Anaerobic threshold.

INTRODUCTION

Peak oxygen uptake and anaerobic threshold (AT), which are estimated by the cardiopulmonary exercise testing, are recognized as useful objective parameters of endurance exercise performance and exercise therapy[1]. Gas exchange AT is a particularly useful parameter because it is the point at which minute ventilation ($\dot{V}_E$) increases dispropor-

tionately relative to oxygen uptake ($\dot{V}O_2$) and the work level is 40 to 60 percent of maximum oxygen uptake ($\dot{V}O_{2\,max}$) in healthy sedentary subjects. Moreover, changes in AT with repeated noninvasive testing can be used to evaluate the progress of an underlying disease and the response to cardiovascular fitness therapy.

On the occasion of rehabilitation after myocardial infarction and others, treadmill (standing) and bicycle ergometer (sitting) exercises have widely

been used. While those exercises were done in upright positions, cardiac catheterization and scintigraphy were performed in supine positions. Recent report[2] offered some suggestion that using ergometer with a backrest and supine ergometer exercise are very useful for the physical fitness of disabled persons and aged people. Thus, it becomes very important to understand the difference in physiological response between supine and sitting exercises. The present study is, therefore, aimed at investigating the effect of body position on cardiopulmonary response during exercise.

METHODS

Subjects

The subjects for this study consisted of nine healthy Japanese men (mean age of 19.9 ± 1.5 years). Their height averaged 172.6 ± 4.5 cm, and their weight averaged 65.6 ± 8.9 kg. They had all regularly engaged in sports. The subjects were well acquainted with the experimental procedures before giving informed consent.

Cardiopulmonary exercise testing protocol

Exercise testing was performed using an electrically braked bicycle ergometer (Space cycle SSR, TS Health Systems, Co.) in both the supine and the sitting positions with a backrest. Initially, each subject had a rest period of 30 minutes. After a warming-up period of 3 minutes at 20 watts, the work rate was increased as a ramp protocol at a rate of 20 watts per minute until 180 watts. Pedal frequency was maintained near 50 cycle per minute.

The order of exercise was randomized. Heart rate (HR) and arrhythmia was continuously monitored from a 12-lead electrocardiogram (ML-5000, Fukuda Denshi Co.) during exercise. Blood pressure (BP) was measured by the cuff method at rest and at the end of each stage.

Respiratory measurements

Throughout the exercise, oxygen uptake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$) and minute ventilation ($\dot{V}_E$) were estimated from the resting state to the end of the exercise period using a Respiromonitor RM-300 (Minato Medical Co.) and a metabolic measurement cart equipped with an oxygen and a carbon dioxide gas analyzer MG-360 (Minato Medical Co.). The expired gas was sampled with the breath-by-breath method and measurements of $\dot{V}O_2$, $\dot{V}CO_2$ and V_E were computed on-line from ventilatory volumes and differences between inspired and expired gases every 10 second.

From these measurements, the ventilatory equivalent of oxygen ($\dot{V}_E/\dot{V}O_2$) and the ventilatory equivalent of carbon dioxide ($\dot{V}_E/\dot{V}CO_2$) were calculated. These calculations were made using a personal computer (NEC PC-9801). Anaerobic threshold (AT) was determined by the following conventional criteria: noting the $\dot{V}O_2$ at which 1) $\dot{V}_E/\dot{V}O_2$ systematically rose without an increase in $\dot{V}_E/\dot{V}CO_2$, 2) the gas exchange ratio started to increase steeply[1].

Biochemical measurements

A teflon catheter was inserted in a cubits vein of the participants. Blood samples ware obtained at resting control state and within the last minute at the end stage of exercise. Blood samples were immediately ice-cooled and plasma was separated by centrifugation for 10 minutes at 4 °C 3000 rpm. Plasma noradrenaline (NA) was analyzed with high performance liquid chromatography (HPLC) with electrochemical detection after extracted by alumina absorption. Angiotensin II (ANG II) was analyzed by the radioimmunoassay (RIA) method after solid liquid extraction.

Cardiac output measurements

On another day, a single level exercise testing was performed on all subjects to measure cardiac output (CO) in both the sitting and supine positions. A single-level exercise testing consisted of 6 minutes at 100 watts. The dye-dilution technique using ear-piece (NIHON KODEN, MLC-4200) was used for CO determination. Teflon catheter was inserted in a cubits vein. At the resting control state and the steady state during 100 watts exercise, indocyanin green (5 mg) was shot from the catheter. Cardiac index (CI) was computed by CO.

Statistical analysis

Values are expressed as mean $\pm$ SD. Differences were compared by means of paired Student t-tests. A p value of <0.05 was considered statistically significant.

RESULTS

Cardiopulmonary exercise testing

Figure 1 shows changes in HR and BP during each incremental exercise. HR and BP during exercise were lower tendency in the supine position compared to the sitting position, although not significantly. The mean values of HR at resting control state were 66.9 ± 6.7 bpm in the supine position and 73.0 ± 7.5 bpm in sitting position. The mean peak HR was 156.0 ± 15.3 bpm in the supine position, and 161.7 ± 14.6 bpm in sitting position. The mean values of BP (Systolic BP/Diastolic BP) at rest were 122.7 ± 8.5/75.3 ± 11.6 mmHg in the supine position and 139.4 ± 7.9/80.6 ± 9.7 mmHg in sitting position. The peak BP was 200.5 ± 10.7/88.7 ± 14.8 mmHg in the supine position, and 207.3 ± 10.8/86.7 ± 15.4 mmHg in sitting position. These were not significant.

Figure 2 shows the difference in $\dot{V}O_2$ and AT between two positions. No significant difference was observed in the mean value of $\dot{V}O_2$ at resting control state between the supine position (4.5 ± 0.7 ml/kg/min) and the sitting position (4.6 ± 0.6 ml/kg/min). The mean $\dot{V}O_2$ during warming up at 20 W in the supine position was significantly lower than that in the sitting position. The mean $\dot{V}O_2$ at the end of exercise (180 W) was not significantly lower in the supine position (33.7 ± 4.1 ml/kg/min) compared with the sitting position (35.8 ± 3.1 ml/kg/min). The mean AT was markedly lower in the supine position compared to the sitting position (18.3 ± 2.6 ml/kg/min in the supine position, 21.7 ± 1.9 ml/kg/min in the sitting position, p<0.01). The mean HR at AT in the supine position was not significantly lower than that in the sitting position (114.7 ± 9.4 bpm and 123.1 ± 7.8 bpm, respectively). The mean BP (Systolic BP/Diastolic BP) at AT in the supine position was slightly lower than that in the sitting position (160.9 ± 14.4/81.8 ± 14.4 mmHg and 172.3 ± 16.8/83.3 ± 9.5 mmHg, respectively). The mean value of Exercise time until AT was 163.7 ± 31.5 seconds in the supine position compared with 203.0 ± 33.8 seconds in the sitting position (p<0.05).

Table 1 shows changes of NA and ANG II in the supine and sitting positions during each incremental exercise. NA was lower tendency in the supine position compared with the sitting position both at the resting control state (274.6 ± 100.7 pg/ml vs 365.9 ± 139.6 pg/ml, p<0.07) and after exercise (1032.3 ± 581.1 pg/ml vs 1385.5 ± 653.3 pg/ml, p<0.1). ANG II also was lower tendency in the
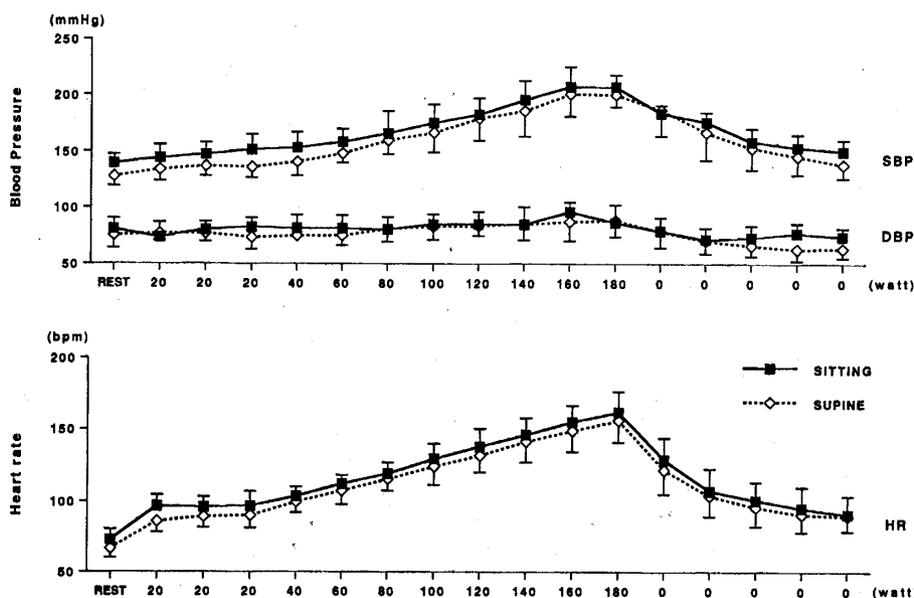

Fig.1. Responses of heart rate (HR) and systolic blood pressure (SBP), diastolic blood pressure (DBP) during exercise in supine (broken line) and sitting (full line) positions by the ramp method. HR and SBP, DBP are lower tendency in supine exercise than in sitting exercise, but not significantly. Values are means ± SD.
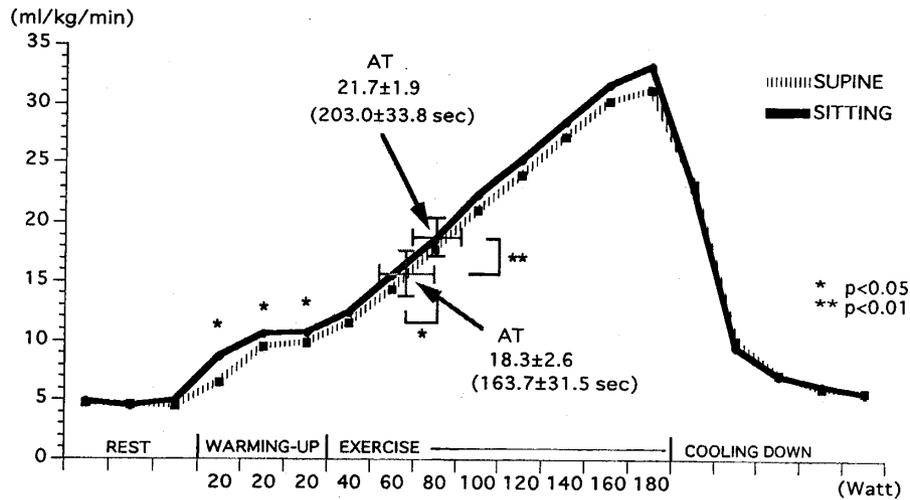
Fig. 2. Response of oxygen uptake ($\dot{V}O_2$) during exercise in supine (broken line) and in sitting (full line) positions by the ramp method. The arrows indicate Anaerobic Threshold (AT) in both positions. AT is significantly lower in supine position compared to sitting position. Exercise time is significantly shorter in supine position. Values are means ± SD. p=probability.

Table 1. Comparison of Noradrenaline and Angiotensin II at rest and after exercise testing (after ex) in the supine and sitting positions

		Supine	Sitting
Noradrenaline	rest	274.6 ± 100.7	365.9 ± 139.6
(pg/ml)	after ex	1032.3 ± 581.1	1385.5 ± 653.3
Angiotensin II	rest	8.0 ± 5.2[a]	14.9 ± 6.7[a]
(pg/ml)	after ex	11.4 ± 6.5	16.3 ± 5.1

Values are means ± SD. [a] means p<0.05.

Table 2. Comparison of oxygen uptake ($\dot{V}O_2$), Cardiac Index (CI) and arteriovenous oxygen difference ($_{AV}DO_2$) at rest and during 100 watts exercise (100 W ex) in both positions

		Supine	Sitting
$\dot{V}O_2$	rest	290.2 ± 41.1	287.7 ± 46.1
(ml/min)	100 W ex	1590.8 ± 70.1[b]	1667.3 ± 101.2[b]
CI	rest	3.34 ± 0.69[b]	2.55 ± 0.73[b]
(l/m2/min)	100 W ex	7.91 ± 1.76	7.63 ± 1.64
$_{AV}DO_2$	rest	5.0 ± 0.8[b]	6.7 ± 1.4[b]
(vol%)	100 W ex	11.8 ± 2.5[b]	12.9 ± 2.8[b]

Values are means ± SD. [b] means p<0.01.

supine position than the sitting position both before (8.0 ± 5.2 pg/ml vs 14.9 ± 6.7 pg/ml, p<0.05) and after exercise (11.4 ± 6.5 pg/ml vs 16.3 ± 5.1 pg/ml, p<0.1).

Single-level exercise testing

The mean VO_2 in the two positions were similar at the resting control state (290.2 ± 41.1 ml/min in the supine position and 287.7 ± 46.1 ml/min in the sitting position, NS), but the mean $\dot{V}O_2$ in the steady state of 100 W exercise is lower in the supine position compared with the sitting position (1590.8 ± 70.1 ml/min and 1667.3 ± 101.2 ml/min, respectively, p<0.01)

The CI computed by CO at the resting control state was significantly higher in the supine position (3.34 ± 0.69 l/m2/min) than in the sitting position

(2.55 ± 0.73 l/m²/min). However, the CI in the steady state of 100 W exercise was similar in both the supine and sitting positions, 7.91 ± 1.76 l/m²/min in the supine position and 7.63 ± 1.64 l/m²/min in the sitting position.

Consequently, the mean value of arteriovenous oxygen difference ($_{AV}DO_2$), calculated by Fick's formula, was significantly lower in the supine position compared with the sitting position at rest (5.0 ± 0.8 vol% and 6.7 ± 1.4 vol%, p<0.01) and the steady state of 100 W exercise (11.8 ± 2.5 vol% and 12.9 ± 2.8 vol%, p<0.01) (Table 2).

DISCUSSION

Previous studies have shown that lower maximal $\dot{V}O_2$, HR and exercise intensity were observed more during supine exercise than sitting exercise. This might be caused by mechanical efficiency[3] or unfamiliarity with the task and exercise of the small muscle mass in the supine position[4, 5].

In this study, the subjects were well acquainted with and practiced pedaling under supine condition before testing. In addition, using the ergometer with a backrest differs from the usual ergometer. We assumed that there was little effect of postural and stabilized muscle activity in our experiment. The difference in $\dot{V}O_2$ was therefore based on the change in cardiac output (stroke volume) under the condition that gravity is related.

There is general agreement that maximal SV increases markedly during the transition from rest to the early stage of exercise[6–8], within the anaerobic threshold[9]. The present study assumed that cardiac output would practically be the same in each position after arriving at maximal stroke volume because HR during exercise was similar in both positions. Regarding cardiac output, our measurements showed that the cardiac index computed by the cardiac output was higher in the supine position compared with the sitting position in the resting control state. This was similar in both positions during 100 W exercise. Thus, applying the Fick equation to the these results, these data indicate that the difference in $\dot{V}O_2$ between the supine and the sitting positions was prescribed by arteriovenous oxygen difference[4, 10, 11].

The maximal arteriovenous oxygen difference attained during exercise is influenced to some degree by one's capacity to divert a large portion of the cardiac output to working muscles[11]. The increase in the blood flow in working muscle during exercise is due to increased cardiac output. Moreover, the blood redistribution to working muscles from other areas is due to neural and hormonal vascular regulation and the local metabolic conditions of the working muscles[11]. In this study, the cardiac indexes showed a linear response in both positions at 100 W exercise so that the arteriovenous oxygen difference participated in $\dot{V}O_2$ may be due to the difference in blood redistribution to the working muscles. Concerning blood flow in working muscles, Folkow, et al[12] reported that a calf muscle's blood flow during rhythmic exercise in the "leg-down" position at a 60° angle was significantly higher than in the supine position. The results of our study also suggest that the blood flow to working muscles is larger in sitting exercise than supine exercise.

Additionally, the specific hormonal vascular regulation was recognized in this study so that we analyzed two hormones (noradrenaline and angiotensin II) having vasoconstrictor effects. The angiotensin II (ANG II) constricts arterioles by its direct effects, and raises vascular resistance indirectly through its actions on sympathetic nervous activity[13]. Previous studies have shown that NA and ANG II both begin to rise after incremental exercise, and a more rapid elevation in NA was shown after exercise intensity surpasses AT[8, 14–17]. In this study, lower NA and ANG II were observed during the supine position, which may be taken as a reflection of a lesser degree of activation of the sympathetic nervous system. This result suggests that a larger blood flow is distributed to active muscles in the sitting position because the peripheral vascular resistance in the non-active muscles and the abdominal cavity are raised.

It should also be noted in the present study that exercise time was shorter and $\dot{V}O_2$ at AT was lower in the supine exercise than in the sitting exercise. Prior studies have concluded that there is a strong correlation between AT and the mass of working muscles[18, 19] so that the working muscles in the supine exercise may not actually be much. As blood lactate concentration was not measured in this study, we could not refer to the relationship between the blood lactate concentration and NA, ANG II. However, Bevegard, et al[6] found that the blood lactate concentration was lower in the upright exercise than in supine exercise. Moreover, Eiken[20] found that the blood lactate concentration during supine leg exercise rose at a significantly lower work intensity in the lower body negative pressure (LBNP) condition. Eiken concluded that the primary limitation of work performance was set by the peripheral circulation in working muscles. The blood flow to working muscles in the supine exercise is therefore lower than in the sitting exercise, which indicates that the supine exercise could lead to a more rapidly increase in metabolic acidosis.

In conclusion, this study shows the difference in $\dot{V}O_2$ between supine and sitting positions and

lower NA and ANG II that were observed during the supine position contributed to a lower blood flow in working muscles. The major contribution of the present study is that the changes in blood distribution and lower blood flow to active muscles in the supine position were suspected the etiology of lowered AT.

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