Effect of change in blood volume in skin plus active muscle on heart rate drift during submaximal exercise

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Abstract

The purpose of the present study was to examine the effect of change in blood volume in skin plus active muscle on heart rate drift during moderate exercise and heavy exercise for 30 min. Total hemoglobin concentration (Total Hb) in the vastus lateralis muscle plus its skin was determined by near-infrared spectroscopy. Total Hb significantly increased and remained stable from 20 min in moderate exercise and from 10 min in heavy exercise. Heart rate (HR) rapidly increased until 3 min and showed a steady state in moderate exercise. HR at 30 min was significantly higher than that at 3 min in moderate exercise. HR rapidly increased until 3 min and then gradually but significantly increased in heavy exercise. Increase in total Hb was not significantly related with HR after 3 min of exercise when HR was around 120 beats per min in moderate exercise. Increase in total Hb was significantly related with HR from 3 min to 10 min in the heavy exercise (correlation coefficients ranged from 0.959 to 0.702). It is concluded that an increase in the blood volume in skin plus active muscle is not simply associated with HR drift.

Keywords: heart rate, muscle and heart pumping, total Hb, near-infrared spectroscopy.
Cardiovascular (CV) drift during submaximal exercise for 60-120 min is a well-known phenomenon. It is characterized by progressive declines in stroke volume (SV) and pulmonary and systemic mean arterial pressures and a parallel increase in heart rate (HR), whereas cardiac output is maintained at an almost constant level (2, 16). CV drift might be due to alterations in cardiac and vascular functions. The long-held and presently prevailing concept, summarized by Rowell (16), is that CV drift is due to a progressive increase in cutaneous blood flow as body temperature rises. The rise in cutaneous blood flow is thought to lead to an increase in cutaneous venous volume, thus reducing ventricular filling pressure, end-diastolic volume and SV during submaximal exercise (16). Indeed, right atrial pressure progressively decreases during prolonged exercise (13).

In previous studies, cutaneous blood flow was determined rather than cutaneous blood volume, which affects ventricular filling pressure. Furthermore, it is thought that a change in cutaneous blood volume is quantitatively insufficient to affect venous return to the heart since the ratio of cutaneous blood volume to total blood volume is small. Recently Hayashi et al., (7) determined change in blood volume in skin plus active muscle during prolonged exercise using near-infrared spectrometry (NIRS), and the results suggested that an increase in blood volume in skin plus active muscle induces a decrease in SV, resulting in an increase in HR. However, there are points of criticism regarding that study. One is that a long exercise period of 60 min was used. Their data showed that blood volume in skin plus active muscle increased after the start of exercise and that the rate of increase became lower, whereas HR increased at a constant rate for 60 min. It has also been shown that cutaneous blood flow, directly or indirectly determined, remains fairly stable after 20-30 min of exercise (5, 8, 13).
Therefore, it is likely that HR drift is associated with factors other than changes in cutaneous blood volume and in blood volume in skin plus active muscle, at least after 20-30 min. Another point is that the effect of muscle pumping was not taken into consideration in their study.

Hydrostatic pressure from the legs to the heart increases when posture is changed from the supine position to upright position. Then blood distribution in the body shifts to the legs and consequently blood is pooled in the legs. This pooling increases hydrostatic pressure in the veins in the legs. However, since the degree of increase in hydrostatic pressure is insufficient, venous return is reduced. Consequently, SV is reduced despite activation of sympathetic nervous tone by postural change (10).

Exercise in the upright position results in marked blood volume shifts from legs and abdominal organs to the heart and lungs (4) probably due to muscle pumping. It is known that muscle pumping increases venous return during leg exercise in an upright position. This could result in an increase in SV (Gauer and Thron 1965). Recent studies have indicated that there is evidence of a muscle pump effect in humans during upright cycle exercise at light intensity (15, 17) but not during high-intensity exercise (11). Therefore, HR drift might occur due to a small muscle pump effect in high-intensity exercise but might be inhibited in light-intensity exercise by effective muscle pumping if local blood volume in skin plus active muscle is increased during prolonged exercise.

The purpose of the present study was to examine the effect of change in blood volume in skin plus active muscle on heart rate drift during moderate exercise and heavy exercise for 30 min.

**Materials and Methods**
Subjects

Eight healthy trained males (track and field athletes) with a mean ± SD age of 19 ± 0.8 years, a mean body weight of 65 ± 5.6 kg, a mean height of 171 ± 3.6 cm, a mean ventilatory threshold (VT) of 1.59 ± 0.32 and a mean $\dot{V}O_2$ peak of 2.98 ± 0.34 l/min participated in this study. After the objective and procedure of the experiment and the risks associated with the experiment had been explained, written consent to participate in the study was obtained from each subject. This study was approved by the local ethics committee.

Experimental protocol

An aerobic exercise ergometer (Nihon Kohden Co., Tokyo) was used. On the first day, each subject performed incremental-load exercise after a 5-min rest period to determine his peak $\dot{V}O_2$. After cycling at a work rate of 20 watts for 4 min, the work rate was increased by 30 watts per min until the subject could no longer maintain a rotation rate of 60 rpm. On different days, two constant-load exercises were performed for 30 min after previous exercise at 45 watts for 4 min. One was moderate exercise performed at an exercise intensity at 80% of the VT and the other was heavy exercise performed at $\text{VT} + (\dot{V}O_2\text{peak} - \text{VT})/2$. Two subjects stopped the heavy exercise at 20 min and 25 min due to exhaustion.

Measurements

$\dot{V}O_2$ was measured breath-by-breath using a respiratory gas analyzer (MMC 4400tc: Sensor Medics). The flow volumes of inspiration and expiration were determined using a turbine flow meter. The flow signals were integrated electrically for
each breath and converted to ventilation per minute. The respiratory meter was calibrated using a 2-liter syringe. The results of measurement using this instrument were linear with ventilation in the range of 6-220 l/min. The O₂ and CO₂ concentrations were analyzed using a zirconium sensor and infrared absorption analyzer, respectively. The data of VO₂ were outputted every 15 seconds.

In the present study, the NIRS signal was from the vastus lateralis muscle plus its skin every 15 sec. Oxygenated hemoglobin (HbO₂) and total hemoglobin (Total Hb) were determined using a NIRS device (HEO200N, Omuron, Japan). The NIRS probe consisted of a light source and an optical detector, with a distance of 3.0 cm between the light source and detector. Dual-wavelength light (760 and 850 nm) emitted from the light source penetrates tissue, where it is either absorbed or scattered, and some of the scattered light returns to the optical detector. The depth of penetration of the radiation is about 1.5 cm (MacCully and Hamaoka 2000). The method for determining HbO₂ and total Hb is described elsewhere (19).

Heart rate (HR) was measured by a telemeter for a cardiograph (ZB-810P Nihonkoden Co., Tokyo) and the data were received by Life Scope 6 (OEC-6510, Nihonkoden Co., Tokyo).

_Determination_

VT was determined by the V-slope method (1). VO₂ was plotted against CO₂ output. Two straight lines were drawn at portions in low and high values. The intersection of the two straight lines was defined as VT. VO₂peak was the maximal value for 15 sec obtained in ILE.
Statistical analysis

Data are expressed as means ± standard deviation (SD). A repeated measures analysis of variance (ANOVA) was used to show significant differences in \( \dot{V}_O_2 \), HR and total Hb. If significant F ratios were obtained, the means were compared by using Tukey-Kramer’s post-hoc test. Comparisons were made among values at 3, 10 and 20 min in heavy exercise and among values at 3, 10, 20 and 30 min in moderate exercise. The strength of the relationship between HR and total Hb was obtained by Pearson’s correlation. The level of significance was set at P<0.05.

Results

Figure 1 shows \( \dot{V}_O_2 \) kinetics in heavy exercise and moderate exercise. \( \dot{V}_O_2 \) increased rapidly in both heavy exercise and moderate exercise. After 3 min of heavy exercise and moderate exercise, the F ratio did not show a significant level.

Figure 2 shows HR in moderate exercise and heavy exercise. HR in moderate exercise showed a rapid increase followed by a gradual increase in one subject and followed by a steady state in the other subjects. Therefore, HR for moderate exercise was averaged except for the gradual increase. There was no significant difference in average HR from 3 min (110 ± 8.3 beats/min) to 20 min (116 ± 8.1 beats/min), but average HR at 30 min (118 ± 7.8 beats/min) was significantly higher than that at 3 min. Average HR in heavy exercise showed a rapid increase for the first 3 min and then a gradual but significant increase. HR was 155 ±14.1 beats /min at 3 min and 172 ±11.4 beats/min at 30 min. The drift was about 10%.

Average total Hb in heavy exercise and that in moderate exercise are shown in Figure 3. Since the same work rate was loaded in pre-exercise, it is thought that total Hb
decreased at the same rate in moderate exercise and heavy exercise. However, total Hb reached almost the same level at the end of the two exercises despite different work rates in heavy exercise and moderate exercise. Total Hb started to increase after the start of moderate exercise and heavy exercise. In moderate exercise, total Hb significantly increased from 3 min to 20 min, but there was no significant difference between total Hb at 20 min and that at 30 min. In heavy exercise, total Hb significantly increased from 3 min to 10 min, but there was no significant difference between total Hb at 10 min and that at 20 min. Thus, there were two phases in both moderate exercise and heavy exercise. That is, total Hb rapidly increased (increasing phase) and its rate of increase changed to a steady state (stable phase). This occurred from 20 min in moderate exercise and from 10 min in heavy exercise.

Relationships between HR and total Hb obtained from 3 to 20 min in moderate exercise are individually shown in Figure 4. In moderate exercise, three types can be separated from the relationship between HR drift and total Hb as follows. In one subject, HR increased in response to the increase in total Hb and the relation had a high correlation coefficient (Table 1: r=0.779). In that subject, HR at 3 min was about 120 beats/min and soon increased to 140 beats/min. Then HR increased to 160 beats/min at 30 min. In four subjects, HR did not greatly increase despite an increase in total Hb, although significant correlation coefficients were found. In three subjects, HR did not increase and significant correlation coefficients were not found. The steady state in HR was maintained at around 120 beats/min.

Relationships between HR and total Hb obtained from 3 min to 10 min in heavy exercise are individually shown in Figure 5. There were significant linear relationships between total Hb and HR in the increasing phase of total Hb after 3 min in
heavy exercise. The correlation coefficients ranged from 0.959 to 0.702 (Table 1).

**Discussion**

HR rapidly increased until 3 min after the onset of moderate and heavy exercises. Administration of atropine, which blocks the parasympathetic impulse, results in an increase in HR in light exercise, and administration of propranalol, which blocks the activity of adrenergic β-receptors in the heart, results in a decrease in HR in exercise at higher intensity (3). Analysis of HR variability has suggested a contribution of parasympathetic nerves in light exercise and a contribution of sympathetic nerves in exercise at higher intensity (18). Therefore, it could be inferred that an increase in HR until 3 min after the start of moderate exercise is due to inhibition of parasympathetic nervous tone and that in heavy exercise the effect of activation of sympathetic nervous tone is added to the increase of HR. Thus, the autonomic nervous system should work for the sudden great requirement of blood flow into active muscle at the onset of exercise.

HR drift is a phenomenon that occurs after the onset of exercise. In heavy exercise, HR drift was found in relation to an increase in total Hb. The starting point of HR drift was defined to be at 3 min in the present study. This comes from the results of \( \dot{V}O_2 \) kinetics. \( \dot{V}O_2 \) after 3 min of heavy exercise showed a steady state, suggesting a steady state of cardiac output. HR level during drift in the present study (10%) was the same as that found in pervious studies (2, 13). SV was not measured in the present study, but it has been reported that HR and SV changed by 10% from 10 min to 30 min of exercise (2). This suggests that HR drift obtained in the present study reflects a decrease in SV.
One explanation for HR drift is an increase in cutaneous blood volume (16). During constant-load exercise, skin temperature increases and cutaneous blood volume is increased to release heat from the body to the environment. In the present study, we used NIRS to determine the blood volume that included blood volume in skin and in active muscle. We observed that blood volume increased until 20 min after the onset of moderate exercise and until 10 min after the onset of heavy exercise. These increases in local blood volume can have a greater effect on venous return than the effect of cutaneous blood volume alone. Kimura et al. (9) examined whether total Hb in skin and inactive muscle during moderate exercise affects HR drift for 30 min using NIRS, and they showed that total Hb, which is related to deep temperature in inactive muscle, is related to HR drift 3 min after the onset of moderate exercise. They suggested that blood volume in skin and inactive muscle can affect HR drift. Thus, it is likely that blood volume in inactive muscle as well as in active muscle affects HR drift.

However, in the case of active muscle, the effect of muscle pumping must be taken into consideration. Muscle pump frequency in this study was 120 beats/min, which was the pedaling rate. In the present study, when HR was around 120 beats/min, there was little HR drift from 3 min to 20 min of moderate exercise despite the increase in total Hb. This is a new finding. Muscle pumping provides venous return to the right ventricle at the same frequency. As a result, venous return is maintained even if blood volume is increased as in moderate exercise. On the other hand, in heavy exercise, HR is faster than the rate of muscle pumping. In this case, muscle pumping is not effective during the interval between heart beats. Since venous return is dependent on blood pressure in active muscle during relaxation between muscle contractions and since the pressure would not be sufficiently high due to the compliance reduced by an
increase in temperature during prolonged exercise, venous return would be reduced, resulting in a decrease in SV and HR drift.

It has been reported that total Hb determined by NIRS increased for the first 20-30 min of exercise and remained fairly stable for the next 30 min (7). In the present study, this stable phase appeared after 20 min in the moderate exercise and appeared after 10 min in the heavy exercise. This early stable phase that appeared in heavy exercise was not expected because vasodilatation is associated with a continuous increase in body temperature. However, this early stable phase may occur due to an increase in sympathetic nerve tone. During prolonged exercise, vasodilatation in cutaneous blood vessels and in active muscle may occur with an increase in body temperature and metabolites, and consequently arterial blood pressure can be reduced (2, 7, 13). In this case, sympathetic nerve tone is increased to maintain blood pressure (14). This may result in vasoconstriction not only in arteries but also in veins. The activation of sympathetic nerve tone may also induce an increase in HR. Thus, these reactions might be associated with reorganization of circulation in the middle of exercise for the preceding vasodilatation due to rise of body temperature and metabolites produced by exercise.

It is concluded that an increase in the blood volume in skin plus active muscle is not simply associated with HR drift.
REFERENCES


Table 1. Correlation coefficients obtained between heart rate and total hemoglobin from 3 min to 20 min in moderate and from 3 min to 10 min in heavy exercises.

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<th>Moderate exercise</th>
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Fig. 1. Oxygen uptake at rest for 5 min, during pre-exercise for 4 min and during moderate and heavy exercises for 30 min. Open squares show results in moderate exercise, and closed squares show results in heavy exercise.

Fig. 2. Heart rate at rest for 5 min, during pre-exercise for 4 min and during moderate and heavy exercises for 30 min. Open squares show results in moderate exercise, and closed squares show results in heavy exercise.

Fig. 3. Total hemoglobin (Total Hb) at rest for 5 min, during pre-exercise for 4 min and during moderate and heavy exercises. Open squares show results in moderate exercise, and closed squares show results in heavy exercise.

Fig. 4. Individual relationships between heart rate and total hemoglobin (Total Hb) from 3 min to 20 min in moderate exercise. Relationships in eight subjects are shown in different colors.

Fig. 5. Individual relationships between heart rate and total hemoglobin (Total Hb) from 3 min to 10 min in heavy exercise. Relationships in eight subjects are shown in different colors.
Fig. 1.
Fig. 3.
Fig. 4.
Fig. 5.