Effect of Blood Volume in Resting Muscle on Heart Rate Upward Drift during Moderately Prolonged Exercise

Takehide Kimura, Ryouta Matsuura, Takuma Arimitsu, Takahiro Yunoki and Tokuo Yano

Laboratory of Exercise Physiology, Graduate School of Education, Hokkaido University

Abstract  The aim of this study was to determine whether the increase in blood volume in resting muscle during moderately prolonged exercise is related to heart rate (HR) upward drift. Eight healthy men completed both arm-cranking moderately prolonged exercise (APE) and leg-pedaling moderately prolonged exercise (LPE) for 30 min. Exercise intensity was 120 bpm of HR that was determined by ramp incremental exercise. During both APE and LPE, HR significantly increased from 3 to 30 min (from 108±9.3 to 119±12 bpm and from 112±8.9 to 122±11 bpm, respectively). However, there was no significant difference between HR in APE and that in LPE. Oxygen uptake was maintained throughout the two exercises. Skin blood flow, deep temperature, and total Hb (blood volume) in resting muscle continuously increased for 30 min of exercise during both APE and LPE. During both APE and LPE, there was a significant positive correlation between total Hb and deep temperature in all subjects. Moreover, there was a significant positive correlation between HR and total Hb (in seven out of eight subjects) during LPE. However, during APE, there was no positive correlation between HR and total Hb (r=0.391). These findings suggest that an increase of blood pooling in resting muscle could be proposed as one of the mechanisms underlying HR upward drift during moderately prolonged exercise.

Introduction  The term “heart rate upward drift” (HR upward drift) refers to the phenomenon of heart rate increasing progressively over time during prolonged exercise with constant work rate and moderate intensity (>50% VO2max). This phenomenon occurs despite the fact that the circulatory system generally shows a steady state and cardiac output remains relatively constant during short and moderate exercise (Ekelund, 1964, 1966, 1967). Therefore, it is thought that HR upward drift is induced by a decline in stroke volume. However, the cause of stroke volume decline has not been fully elucidated.

Rowell (1986) hypothesized that HR upward drift is the consequence of a progressive increase in cutaneous blood flow (CBF) as body temperature rises. The rise in CBF suggests an increase in skin blood volume, leading to a reduction in end-diastolic volume and a decline in stroke volume. However, it has been observed that stroke volume continuously decreased despite a plateau in skin blood volume during the latter half of prolonged exercise for 60 min (Ricardo et al., 1999). Nose et al. (1990, 1994) reported that HR upward drift occurred in spite of attaining a CBF plateau. These studies suggest that the decline in stroke volume might not be related to an increase in CBF. Moreover, Ricardo et al. (1999) reported that HR upward drift after 15 min of prolonged exercise is inhibited by the β-adrenoceptor blocker atenolol. That study showed that stroke volume was not decreased and CBF remained stable after the first 20 min of prolonged exercise for 60 min.

Therefore, we focused on resting muscles as the location where blood is accumulated during prolonged exercise. Wenger et al. (1975) reported that forearm blood flow is increased by leg-pedaling exercise due to increases in central and peripheral temperatures. If blood flow in resting muscles increases during exercise, blood volume in resting muscles should increase. The increase of blood volume would result in a reduction in end-diastolic volume and a decrease in stroke volume.

The present study had two main purposes. The primary purpose was to determine whether HR upward drift was associated with an increase in blood volume in resting muscle during moderately prolonged exercise (30 min). The cause of HR upward drift during prolonged exercise of 45–120 min has been investigated in many previous studies. However, with the advance of time, the cause of HR upward drift is thought to be complex, because HR upward drift is influenced by a variety of elements during the final phase of exercise (i.e., dehydration,
elevation of VO₂, etc.) (Wingo et al., 2005, 2006; Ganio et al., 2006; Hamilton et al., 1991; Gonzalez-Alonso et al., 2000). Therefore, we employed moderately prolonged exercise of 30 min to eliminate the effects of these factors.

Previous studies suggested that HR upward drift was induced by a decline in stroke volume. Consequently, we employed a load of intensity that was a maximum of stroke volume during exercise (HR 120 bpm). In addition, accuracy of stroke volume measurement is not better than accuracy of HR measurement. Therefore, prolonged exercise (60 min~) is necessary to measure stroke volume. However, we employed prolonged exercise of 30 min. Since this duration of exercise is not sufficient for a change of stroke volume to occur, we employed a load of intensity of HR of 120 bpm. We expected that this load of intensity would accurately reflect change of stroke volume.

The secondary purpose of this study was to compare the degrees of HR upward drift when performing upper body exercise and lower body exercise. We hypothesized that these exercises would produce a difference in blood volume due to the difference between muscle volumes in the arms and legs and consequently induce different degrees of HR upward drift.

**Methods**

**Subjects**

Eight healthy and active men provided written informed consent to participate in this study. The mean age, height, and weight of the subjects were 22.1±1.4 years, 171.7±6.2 cm, and 64.9±5.3 kg, respectively. The Ethics Committee of Hokkaido University Graduate School of Education approved the present study.

**Protocol and experimental design**

All exercise tests were conducted in the same room (room temperature: 25°C). Before any experimental testing, each subject completed several practice sessions of both arm-cranking and leg-pedaling exercises. A pedaling rate of 60 rpm was used for both arm-cranking and leg-pedaling exercises in the experimental testing.

Before arm-cranking and leg-pedaling moderately prolonged exercises, peak power output (Wpeak), peak oxygen uptake (VO₂peak), and maximal heart rate (HR max) were determined using a ramp incremental protocol for arm-cranking and leg-pedaling exercises. During arm-cranking exercise, subjects sat on a bench while keeping their lower limbs relaxed. On the other hand, during leg-pedaling exercise, subjects’ upper limbs were placed on a rest.

1. **Ramp incremental exercise**

   Arm-cranking exercise: after 4-minute warm-up at 10 W, the work rate of exercise was gradually increased at a rate of 10 W/min until exhaustion.

   Leg-pedaling exercise: after 4-minute warm-up at 10 W, the work rate of exercise was gradually increased at a rate of 20 W/min until exhaustion.

   The above two exercises were performed randomly with an interval of more than 2 days.

2. **Moderately prolonged exercise**

   Arm-cranking moderately prolonged exercise (APE) and leg-pedaling moderately prolonged exercise (LPE): after resting for 5 minutes, subjects arm-cranked or leg-pedaled for 30 min at a uniform intensity (HR 120 bpm of the measured HRmax). The load of intensity was calculated by a regression equation between values for heart rate and work rate previously obtained by ramp incremental exercise and then established by Watt values. The reason for employing this intensity was a maximal value of stroke volume during exercise of this intensity. We expected that this exercise would induce a marked change of stroke volume and HR upward drift. The above two exercises were performed randomly with an interval of more than 2 days.

   During APE, we instructed subjects to relax the muscles in their lower limbs. Similarly, during LPE, we instructed subjects to relax the muscles in their upper limbs. Relaxation of these muscles was measured by electromyogram. The measurement sites were the vastus lateralis muscle (APE) and flexor carpi radialis muscle (LPE).

**Instruments**

Exercise was performed using an electromagnetic arm-crank ergometer (model 232CXL, Combi) and electromagnetic cycle ergometer (model 232CXL, Combi).

**Measurements**

1. **Expired gas and heart rate**

   Expired gas was measured breath-by-breath using a computerized system (AE-280s, minato Medical Science). Heart rate (HR) was recorded using the same system. Expired gas and HR were continuously measured and recorded during resting and exercise.

2. **Blood volume (total Hb) in resting muscles**

   Total Hb levels were used as an index of blood volume. Total Hb levels were measured using near-infrared spectrometry (NIRS) (HEO200N, OMRON). In the probe used in this study, the distance between the light source and photoreceptor was 30 mm, and the mean depth of light penetration was approximately 15 mm from the skin surface. The NIRS probe was attached to the left vastus lateralis muscle (APE) or to the left flexor carpi radialis muscle (LPE). Changes in total Hb were measured during resting and exercise with a sampling time of 5 seconds. The averages for each 1-min interval were used.

3. **Skin blood flow**

   Skin blood flow was measured using a laser blood flow meter, Omega Flow FLO-N1 (Omega Wave). A probe was attached to the left vastus lateralis muscle (APE) or to the left flexor carpi radialis muscle (LPE). Changes in skin blood flow were measured during resting and exercise. Obtained analog signals were recorded by a data recorder, along with analog-to-digital conversion at a sampling frequency of 10 Hz using...
MacLab/8s (AD Instruments, Castle Hill) for input into a personal computer. The averages for each 30-sec interval were used.

4. Deep temperature in resting muscle

Deep temperature was measured using a Core Temp Monitor (Terumo). The depth of measurement was 10 mm from the skin. The Core Temp monitor probe was attached to the left vastus lateralis muscle (APE) or to the left flexor carpi radialis muscle (LPE). Changes in deep temperature were measured during resting and exercise with a sampling time of 10 seconds. The averages for each 30-sec interval were used.

Statistical analysis

Two-way analysis of variance for repeated measures was used to determine differences between APE and LPE. Total Hb levels were compared using one-way analysis of variance for repeated measures. The Tukey-Kramer test was applied to specify the differences when significant changes were found. The paired Student’s t-test was used to compare changes over time within each group. Correlations between total Hb and heart rate and correlations between total Hb and deep temperature were calculated by Pearson product-moment coefficients. A value of \( p < 0.05 \) was regarded as statistically significant. All data are presented as means ± SD.

Results

Peak values obtained during ramp incremental exercise

During arm-cranking exercise, \( \dot{V}O_2 \text{peak} \) was 2.23 ± 0.26 \( \text{ml} \cdot \text{min}^{-1} \), HRmax was 183 ± 14.6 bpm, and work rate at exhaustion (WR\text{peak}) was 140 ± 21.5 watts. During leg-pedaling exercise, \( \dot{V}O_2 \text{peak} \) was 3.00 ± 0.46 \( \text{ml} \cdot \text{min}^{-1} \), HRmax was 193 ± 11.9 bpm, and work rate at exhaustion (WR\text{peak}) was 251 ± 34.5 watts. The work rate for moderately prolonged exercise was determined on the basis of the results of ramp incremental exercise. The work rates used for APE and LPE were 61 ± 9.7 watts (42 ± 5.6% \( \dot{V}O_2 \text{peak} \)) and 78 ± 16 watts (33 ± 5.1% \( \dot{V}O_2 \text{peak} \)), respectively.

Serial changes in HR during moderately prolonged exercise

There was no significant difference between HR in APE and that in LPE. HR from 3 min to 30 min during both APE and LPE significantly increased from 108 ± 9.3 to 119 ± 12 bpm and from 112 ± 8.9 to 122 ± 11 bpm, respectively.

Serial changes in \( \dot{V}O_2 \) during moderately prolonged exercise

There was no difference between \( \dot{V}O_2 \) in APE and that in LPE. From 10 min to 30 min, \( \dot{V}O_2 \) remained constant at 1.1 ± 0.2 l/min during APE and remained constant at 1.2 ± 0.3 l/min during LPE.

Serial changes in skin blood flow

There was no significant difference between skin blood flow in APE and that in LPE. From 3 min to 30 min, skin blood flow significantly increased from 2.9 ± 0.6 to 9.5 ± 4.9 ml/min/100g during APE and from 4.8 ± 0.9 to 10.2 ± 1.5 ml/min/100g during LPE.

Serial changes in total Hb in resting muscle

Fig. 1 shows a trend graph of total Hb levels in resting muscle. As shown in this figure, total Hb levels in resting muscle continuously increased for the first 30 minutes of exercise during both APE and LPE. Moreover, total Hb levels after 30 minutes of exercise were significantly higher than those after 3 minutes of exercise during both APE and LPE.

Serial changes in deep temperature in resting muscle

There was no significant difference in APE and that in LPE. From 3 min to 30 min, deep temperature significantly increased from 34.7 ± 0.7 to 36.2 ± 0.8°C during APE and from 35.1 ± 0.9 to 36.0 ± 0.8°C during LPE.

Correlations among HR, total Hb and deep temperature in resting muscle

During both APE and LPE, there was a significant positive relationship between changes in total Hb and deep temperature. Moreover, there was a significant positive relationship between changes in HR and total Hb during both APE and LPE. All computed relationships between total Hb and deep temperature and between HR and total Hb are shown in Table 1.

We calculated relative changes (from 3 min to 30 min) between HR and total Hb and between deep temperature and total Hb during APE and LPE, and then we analyzed the correlation factors of these results. The results are shown in Fig. 2. During LPE, there was a significant positive correlation of relative changes between HR and total Hb (\( R = 0.812, p < 0.05 \)). However, there was no significant correlation of relative changes between HR and total Hb during APE (\( R = 0.391 \)). There were no significant correlations of relative changes between deep temperature and total Hb during APE and LPE.

Discussion

The main purpose of the current study was to determine whether the increase in HR that occurs after 3 min of exercise is related to elevated deep temperature and an increase in blood volume in resting muscle. Findings supporting this notion were as follows: 1) during both APE and LPE, there was a significant positive relationship between changes in HR and total Hb, and 2) during both APE and LPE, there was a significant positive relationship between changes in total Hb and deep temperature.

In previous studies, HR upward drift was thought to be induced by a decline in SV during prolonged exercise (Rowell, 1986). Wingo et al. (2006) reported that the increase in HR was suppressed when the decline in SV was inhibited by suppressing the increase in skin blood flow with a cooling fan.
Therefore, an increase in HR in the present study suggests a decline in SV throughout exercise.

In the present study, skin blood flow in resting muscle continuously increased over time. This finding is consistent with data obtained in other studies during prolonged exercise (Hayashi et al., 2002; Ricald et al., 1999). However, skin blood flow is not directly reflected by skin blood volume. Therefore, we measured skin and muscle blood volume in a resting region as total Hb.

Coyle and Gonzalez-Alonso (2001) proposed that HR upward drift is characterized by a progressive decline in stroke volume. In addition, they hypothesized that a decline in stroke volume was induced by hyperthermia and hypovolemia (Fritzsche et al., 1999; Gonzalez-Alonso et al., 1995, 1999). We therefore focused on hypovolemia with blood pooling in resting muscle. We speculated that blood pooling in resting muscle is induced by hyperthermia during exercise.

The positive relationship between changes in total Hb and deep temperature in resting muscle during exercise suggests an increase in blood pooling in resting muscle during exercise. Wenger et al. (1975) reported that forearm blood flow was changed by esophageal temperature and skin temperature during leg-pedaling exercise, indicating that blood volume in resting muscle is changed by deep temperature in resting muscle. In the present study, HR, deep temperature, and total Hb in the resting muscle increased during both APE and LPE. Therefore, moderately prolonged exercise can induce an increase in deep temperature in resting muscle. The gradual increase in deep temperature could induce an increase in total Hb (blood volume) in resting muscle.

The positive relationship between changes in total Hb and deep temperature in resting muscle suggests an increase in blood pooling in resting muscle during exercise. Wenger et al. (1975) reported that forearm blood flow was changed by esophageal temperature and skin temperature during leg-pedaling exercise, indicating that blood volume in resting muscle is changed by deep temperature in resting muscle. In the present study, HR, deep temperature, and total Hb in the resting muscle increased during both APE and LPE. Therefore, moderately prolonged exercise can induce an increase in deep temperature in resting muscle. The gradual increase in deep temperature could induce an increase in total Hb (blood volume) in resting muscle.

The positive relationship between changes in HR and total Hb upward drift is characterized by a progressive decline in stroke volume. It is commonly thought that HR drifts upward with a decline in SV because central blood volume decreases. In addition, central blood flow is influenced by peripheral blood flow. Therefore, it was speculated that
because total Hb in resting muscle is increased, HR is increased.

No relationship was found between changes in HR and total Hb in one subject during APE and in one subject during LPE (Those subjects were different in APE and LPE). In those subjects, total Hb increased, whereas HR plateaued. This is thought to have been caused by a matching between muscle pump and heart rate. In the present study, we established a pedaling rate of 60 rpm during exercise. This pedaling rate is constructed by one arm or leg exercise. Therefore, pedaling rate was 120 rpm during two arms or legs exercise. In addition, because HR at the start of exercise was 120 bpm, there was a possibility that blood circulation of the muscle pump matched blood circulation of the heart. That is, the tempo of ejecting blood in the heart was consistent with the tempo of returning blood by the muscle pump. Consequently, it was thought that this matching caused smooth-flowing blood circulation and inhibited reduction in venous return. In contrast, subjects who showed a positive relationship between changes in HR and total Hb did not exercise at the intensity of HR of 120 bpm at the start of exercise. Therefore, this matching did not occur.

We predicted that HR upward drift in APE would be higher than that in LPE. Since muscle volume in lower limbs is greater than that in upper limbs, we speculated that blood pooling in lower limbs during APE would be greater than that in upper limbs during LPE. However, there was no significant difference between HR upward drift in APE and that in LPE. The reason for this finding may be a static issue or a difference in gravity blood pooling between upper and lower limbs during exercise. HR increased only slightly in both APE and LPE. These increases might not have been sufficient to detect statistical difference. The position of the lower limbs is also lower than that of the upper limbs. This difference in position causes a difference in hydrostatic fluid pressure and a difference in blood volume between the upper and lower limbs.

A load of intensity that was the maximum of stroke volume during exercise (33–42% VO2peak) was used in the present study. However, a load of intensity of 50% VO2peak was used in previous studies. We focused on stroke volume and therefore employed that load of intensity. However, a load of intensity of 33–42% VO2peak may not be sufficient for HR upward drift to occur. Therefore, further study is necessary to reveal the mechanism of HR upward drift.

In summary, HR upward drift was associated with an
increase in blood pooling in resting muscle. The blood pooling would cause a reduction in venous return and a decrease in SV. Therefore, HR upward drift compensates for the decrease in SV to maintain cardiac output. These findings suggest that an increase of blood pooling in resting muscle could be proposed as one of the mechanisms underlying HR upward drift during moderately prolonged exercise. Since a clear conclusion has been not drawn in this study, further research is necessary to verify this.

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References


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Corresponding to: Takehide Kimura, 1–16–402, Nishi 21, Kita 2, Chuou-ku, Sapporo 064–0822, Japan
e-mail: ptswimmer@triton.ocn.ne.jp