Brachial arterial blood flow during static handgrip exercise of short duration at varying intensities studied by a Doppler ultrasound method

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ABSTRACT

The purpose of this study was to determine forearm blood flow changes during static handgrip exercise at different intensities in relation to heart rate and blood pressure. Seven active women performed static handgrip exercise at intensities of 10, 30, 50 and 70% maximum voluntary contraction (MVC) in a supine position for 1 min. During exercise at different intensities, the brachial arterial blood flow (Doppler ultrasound method), calculated from vessel diameter, flow velocity and heart rate (measured by ECG), increased to a similar level (137.3 ± 20.2 – 160.9 ± 26.1 mL min\(^{-1}\)) from pre-exercise control value (87.5 ± 14.1 mL min\(^{-1}\)). These increases at the lower intensities were attributable to increased in-flow during one cardiac cycle, whereas at the higher intensities, they were due to increased heart rate. Both systolic and diastolic blood pressure (Finapres) changes increased from 10% MVC (16.1 ± 3.4, 9.0 ± 1.7 mmHg) up to 50% MVC (33.8 ± 6.7, 25.0 ± 4.9 mmHg), but were disproportionately more elevated at 70% MVC (46.1 ± 7.9, 42.9 ± 8.9 mmHg), suggesting neural vasoconstriction had occurred. Immediate post-exercise hyperaemia, used as an indicator of poor blood supply, became greater as the exercise intensity increased. These results suggest that the brachial arterial blood flow was maintained at a similar level during 60-s static handgrip exercise at different intensities by elevating the blood pressure and heart rate, which probably counteracted the increased intramuscular pressure and neural vasoconstriction occurring at the higher exercise intensity. The magnitude of the post-exercise hyperemic response increased as exercise level increased despite increased blood flow to the arm during exercise. This suggests a worsening imbalance in oxygen delivery in forearm muscles at higher levels of exercise.

Keywords blood pressure, brachial arterial blood flow, Doppler ultrasound, exercise intensities, static handgrip.

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During static muscle activity, mechanical compression of the blood vessels increases due to the muscle tension developed by muscular contraction, leading to obstruction of the blood flow to the region (Bonde-Petersen et al. 1975, Barnes 1980), even if metabolic vasodilation occurs. Consequently, the blood flow to the exercising muscle decreases, or even ceases, if the force developed is high enough to overwhelm the increased perfusion pressure. However, there is still controversy concerning blood flow changes during static leg and forearm exercise when the muscle contraction force varies. During static forearm exercise at lower intensities of up to 20% maximum voluntary contraction (MVC) (Barnes 1980) or 30% MVC (Lind & McNicol 1967), forearm blood flow, measured by venous occlusion plethysmography, increased, whereas at higher intensities of 50–80% MVC, it decreased towards (Humphreys & Lind 1963) or below (Barnes 1980) the resting level. In contrast, the leg blood flow during static knee exercise, studied by a thermodilution technique (Gaffney et al. 1990) and a Doppler ultrasound method (Walløe & Wesche 1988), did not increase in proportion to the increased fraction of MVC. It was unclear whether the discrepancy between the results for forearm and leg blood flow changes during static exercise were due to the different regions or the different...
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Methods used to measure blood flow. Furthermore, little information was available concerning heart rate and blood flow during one cardiac cycle with increasing exercise intensity of either forearm or leg. Therefore, the first aim of this study was to determine whether the blood flow to the forearm during static handgrip exercise changed as the contraction force increased and investigate the immediate post-exercise hyperaemia, when mechanical compression ceased, as an indicator of the balance between blood supply and blood flow demand of the forearm (Byström & Kilbom 1990). The second aim of this study was to determine the changes in blood volume sent to the forearm by one heartbeat and heart rate (equal to blood inflow frequency) during static handgrip exercise at various intensities. For this purpose, we used a Doppler ultrasound method, which enabled us to estimate blood flow on beat-by-beat basis.

METHODS

Subjects

Seven normotensive women participated in this study after giving their informed consent. Their mean age (±SD), height and body mass were 22.3 ± 2.9 years, 162.5 ± 5.4 cm and 54.4 ± 7.5 kg, respectively, and they were physically active with no medical problems.

Handgrip exercise

The subjects performed static handgrip exercise with their right (dominant) hands using hand ergometer (Kagaya 1993) in a supine position. Exercise was maintained for 60 s and the loads applied corresponded to 10, 30, 50 and 70% of the predetermined MVC, which averaged 322.3 N (SD 61.6). Prior to the experiment, the subjects were trained so they were thoroughly accustomed to the exercise protocol and could perform static right handgrip exercise without breath-holding and with their other limbs relaxed.

Experimental procedure

All the experiments were conducted in a room with the temperature and relative humidity of which were controlled at 24 °C and 60%, respectively. After a 30-min resting period in a supine position, the parameters were recorded under baseline conditions for 3 min, then during 60-s static handgrip exercise and finally during 3-min recovery period immediately after exercise. There was an interval of at least 10 minutes between experiments to allow the cardiovascular response to recover from the previous static handgrip exercise. The order of performing exercise at different intensities was randomized.

Physiological measurements

Brachial arterial blood flow per min (Qba) was calculated from the integrated blood velocity of one cardiac cycle, vessel diameter and heart rate. Blood velocity was measured by Doppler ultrasound (HP Sonos 1000, USA) with a 5.6 MHz transducer. The gate length was kept at 1.1 mm. Two-dimensional (2D) recording was conducted using a 7.5 MHz transducer with an ultrasound imaging system (HP 77030 A, USA) to estimate the brachial arterial diameter during pre-exercise control period. During each experiment, the transducer was fixed with a holder to the skin over the brachial artery just proximal to the forearm. The position and direction of the transducer were adjusted manually during 2D recording so that the arterial image was recorded at the centre of the artery and the Doppler angle was set at 60° to the direction of blood flow. Heart rate (f c) was determined from the R-R intervals of the electrocardiogram (ECG). Doppler signals were recorded throughout each experiment, except during 2D recording, which was performed during the 2nd min of the pre-exercise control period and at the beginning of exercise. The former recording was used to determine the arterial diameter and the latter for restoring any changes in the position and/or direction of the transducer resulting from muscle action. Ultrasound signals and verbal instructions were recorded on video tape for subsequent analysis. For diameter determination, the brachial arterial diameter was measured 10 times during the second min before exercise for 10 cardiac cycles. Five calculations were made during the systole at the highest blood flow velocity and five during diastole at the lowest velocity were performed and then the mean values of these 10 measurements were used as the diameter for the blood flow calculations. The arterial mean diameters in our subjects ranged from 3.2–3.9 mm. The intra-individual variability within a study, expressed as coefficient variance (SD/mean), was 3.6 ± 1.7 (SD)% for systolic, 3.6 ± 1.4% for diastolic and 3.4 ± 1.3% for mean diameter, respectively. The vessel diameters during resting periods were studied twice for the same subjects, 3–7 days apart, to test for reproducibility. There were no significant differences between two measurements for systolic (3.61 ± 0.24 mm), diastolic (3.27 ± 0.29 mm) and mean diameter (3.42 ± 0.21 mm). The integrated velocities during one cardiac cycle obtained by tracing the envelop of Doppler spectrum, were measured for 3–5 cycles during the third min of the pre-exercise control period and every 10 s during exercise. Immediately after stopping exercise, the integrated velocities were measured for three cardiac cycles. The Qba was calculated using following equations: \[ Q_{ba} \text{ per pulse} = \pi r^2 \] and \[ Q_{ba} = \pi \int v(r) f_c \] where Qba per pulse is Qba during...
one cardiac cycle, VI is the integrated velocity during one cardiac cycle, r is the arterial radius, Qba was brachial arterial blood flow per min and $f_c$ is the heart rate measured at the same time the Doppler signal measurements were recorded. Further details of this method are described elsewhere (Kagaya & Ogita 1992). Blood pressure (BP) was measured, using Finapres (Ohmeda, USA), on a beat-by-beat basis, from a finger of the inactive left hand throughout each experiment and the mean values during the second min of the pre-exercise period and every 10 s during exercise and the recovery period were calculated.

Statistical analysis

One-way analysis of variance (ANOVA) was used to compare the parameters for exercise at different intensities with Scheffe’s post hoc comparisons. Student’s t-test for paired samples was used to compare the values obtained during the last 10 s of exercise and the immediate post-exercise period. Difference at $P < 0.05$ were considered significant. Group values are expressed as mean ± SEM, unless otherwise stated.

RESULTS

Brachial arterial blood flow

Figure 1 shows typical recordings of the Doppler signals from the brachial artery and ECGs during and immediately after termination of handgrip exercise at 10, 30, 50 and 70% MVC. Electromyogram (EMG) recordings from the brachioradial muscles are also shown in this figure. A marked overshoot of the blood flow velocity was observed immediately after exercise cessation. Figure 2 shows peak blood flow velocity ($V_{\text{peak}}$) values during one cardiac cycle during and immediately after exercise at four different intensities and at rest. The $V_{\text{peak}}$ values during exercise differed significantly neither from the resting value nor each other. However, the $V_{\text{peak}}$ increased significantly ($P < 0.01$) immediately after exercise at all intensities, except 10% MVC, in comparison with during exercise, and increased significantly with each exercise intensity increment. The mean blood flow velocity ($V_{\text{mean}}$) during one cardiac cycle changed in a similar manner to $V_{\text{peak}}$. The time courses of $Q_{\text{ba}}$, expressed as the product of $Q_{ba}$ per pulse and $f_c$, are shown in Figure 3. The $Q_{ba}$ at the end of exercise increased to 137.3 ± 20.2 (10% MVC, $P < 0.05$), 160.9 ± 26.1 (50% MVC, $P < 0.05$) and 155.3 ± 20.1 (70% MVC, $P < 0.01$) mL min$^{-1}$, which were significantly higher compared with the pre-exercise control value (87.5 ± 14.1 mL min$^{-1}$). Immediately after discontinuing exercise, at every intensity, except 10% MVC, it increased significantly, and was maintained at a high level approximately for 30 s, and then gradually recovered. Figure 4 shows the changes in $Q_{ba}$ and $Q_{ba}$ per pulse in relation to exercise intensity at the end of and immediately after exercise. The $Q_{ba}$ values during exercise at different intensities did not differ significantly from each other. However, the $Q_{ba}$ immediately after exercise at 10% MVC was significantly lower ($P < 0.01$) than after exercise at every other intensity and that after 70% MVC was higher than after 10% ($P < 0.01$), 30% ($P < 0.05$) and 50% ($P < 0.05$) MVC. The $Q_{ba}$ per pulse at the end of exercise at 30% MVC (2.66 ± 0.45 mL) was significantly higher ($P < 0.05$) than the pre-exercise value of 1.57 ± 0.18 mL. The values during handgrip exercise at 50 and 70% MVC were significantly lower than the 30% MVC value. Immediately after exercise at 10% MVC, the $Q_{ba}$ per pulse increased significantly ($P < 0.01$) to 3.12 ± 0.47 mL and increased further immediately after exercise at the other three intensities (7.22–8.94 mL), which did not differ significantly from each other. The $f_c$ increased when the exercise intensity increased from 30 to 70% MVC (Figure 5) and the $f_c$ values during exercise at 50% ($P < 0.05$) and 70% MVC ($P < 0.01$) were significantly higher than those during 10 and 30% MVC. The highest mean increase observed was 34.7 ± 4.9 beats min$^{-1}$ from the resting value of 54.1 ± 3.1 beats min$^{-1}$.

Blood pressure

The systolic (BP$_s$) and diastolic (BP$_d$) blood pressures increased rapidly upon initiation of static handgrip exercise in a similar manner with the mean blood pressure changes (△BP$_m$) presented in Figure 6 and increased further as it was continued. The magnitudes of the BP$_s$ and BP$_d$ increases from the pre-exercise control value (120.9 ± 3.8, 67.0 ± 1.5 mmHg) increased linearly in relation to the exercise intensity from 10% (16.1 ± 3.43, 9.0 ± 1.7 mmHg, $P < 0.01$) to 50% MVC (33.8 ± 6.7, 25.0 ± 4.9 mmHg), above which they increased more steeply (Figure 7). Both values at 70% MVC (46.1 ± 7.9, 42.9 ± 8.9 mmHg) were significantly higher than those obtained during exercise at 10% ($P < 0.01$) and 30% (24.2 ± 4.1, 17.5 ± 2.8 mmHg, $P < 0.05$) MVC. The calculated forearm vascular conductance decreased gradually from 10% to 50% MVC and a marked decrease from 50% to 70% MVC was observed.

DISCUSSION

The main finding of this study was that the $Q_{ba}$ during 60-s static handgrip exercise at different intensities (10–70% MVC) increased to approximately similar level despite the blood pressure was elevated at higher in-
tensions to promote maintenance of muscle perfusion. These changes were mainly through a change in $Q_b$ per pulse during handgrip exercise at lower intensities (10 and 30% MVC) and through the heart rate increases at the higher intensities (50 and 70% MVC). However, an increasing post-exercise hyperaemia at higher exercise intensity suggested that an imbalance between blood supply and its demand of the forearm during exercise became greater. The forearm blood flow during handgrip exercise was reported to increase with exercise intensity up to 30% MVC (Lind & McNicol 1967, Kilbom & Persson 1982, Jensen et al. 1993) and decrease towards (Humphrey & Lind 1963) or below (Barnes 1980) the resting value at higher intensity. However, the results of Walloe & Wesche (1988) and Gaffney et al. (1990) suggested that the leg blood flow during static leg exercise did not increase with increasing exercise intensity. Although the blood flow we measured in this study included not only muscle blood flow, but also that of other tissues, such as the skin, it is likely that the brachial arterial blood flow changes during handgrip exercise depend predominantly on muscle blood flow changes, because handgrip exercise involving a low level of external work elicited little change in a cutaneous circulation (Taylor et al. 1990). Therefore, the result of this study might suggest that
the muscle blood flow during static handgrip exercise at different intensity was maintained at a similar level, if the vessel diameter during exercise at different intensity did not differ from that during pre-exercise period, because we used pre-exercise control value to calculate blood flow during and post-exercise conditions. However, the diameter changes of human large arterial have been demonstrated for brachial artery (Anderson & Mark 1989) and radial artery (Joannis et al. 1995) through flow-mediated vasodilatation, partly by release of nitric oxide (Dyke et al. 1995). Furthermore the artery diameter would be reduced due to neural vasocostriction (Anderson & Mark 1989, Olesen et al. 1995). Despite the result that the blood velocity in this study did not change significantly and the vessel diameter changes during handgrip exercise might be small (Kagaya & Ogita 1992), we cannot exclude the possibility that we overestimated blood flow during exercise and underestimated it during post-exercise hyperaemia. Therefore, the forearm blood flow changes in relation to exercise intensity should be studied further. During muscle activity, the vasodilator force is enhanced by the metabolites released by the active muscle. However, static exercise increases the intramuscular pressure in direct proportion to the contraction force (Sadamoto et al. 1983, Sejersted et al. 1984, Sjøgaard et al. 1986). These results were obtained in studies on leg muscles and little information on forearm intramuscular pressure during static exercise is available. In our study, the blood pressure increased as the contraction force increased, which probably led to perfusion pressure elevation. However, the peak and mean blood flow velocities during one cardiac cycle did not increase during forearm muscle activity, which suggested the...

Figure 4 Brachial arterial blood flow per min (a) and per pulse (b) changes in relation to exercise intensity during (●, ■) and immediately after (○, □) exercise. ++$P < 0.01$ vs. pre-exercise, **$P < 0.001$ vs. 10% MVC, # $P < 0.05$ vs. 30% MVC, | $P < 0.05$ vs. 50% MVC.

Figure 5 The heart rate changes in relation to load (%MVC). $$$P < 0.01$ vs. 10% MVC, $$P < 0.01$ vs. 30% MVC, $^1P < 0.01$ vs. 50% MVC.
elevated perfusion pressure was counteracted by the increased intramuscular pressure resulting from the increased contraction force. In this situation, increasing the frequency of the systolic phase (=f<sub>C</sub>) will increase the blood in-flow, because the flow velocity is high during systole. Therefore, the blood flow per min was maintained at approximately similar levels during static handgrip exercise at different intensities. At 70% MVC, the blood pressure increased more than that predicted from the rate of increase at the lower intensities, but the blood flow did not increase further. This suggests that other vasoconstrictor forces, such as more intense sympathetic vasoconstriction, in addition to mechanical hindrance may occur at this intensity (Gaffney et al. 1990). In our study, the duration of static exercise was 60 s, which, at a moderate intensities, is unlikely to enhance the muscle sympathetic nerve activity (MSNA) (Saito et al. 1990, Wallin et al. 1992, Seals 1993), although it is possible to augment the MSNA if the exercise intensity is high enough (Seals 1993). The blood flow elevation following exercise was greater and lasted longer as the exercise intensity increased in this study. These findings imply that the blood supply did not meet the demand of the muscles and this supply/demand mismatch was exacerbated at the higher intensities. Central command should be a contributor that determines cardiovascular responses to static exercise (Gandevia & Hobbs 1990, Williamson et al. 1996), but the inadequate blood supply we observed suggests that the muscle chemoreflexes contribute more to these

**Figure 6** Time courses of mean blood pressure changes during static handgrip exercise at various intensities. Symbols, as in Figure 4.

**Figure 7** Systolic (■) and diastolic (□) blood pressure changes related to exercise intensity. **, P < 0.01 vs 10%; #, P < 0.05 vs 30%.}

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responses at higher intensities than low static exercise intensities as suggested by Gandevia & Hobbs (1990). Furthermore, feedback from the contracting muscles changes the cardiovascular response to static exercise (Friedman et al. 1992). Gaffney et al. (1993) demonstrated that the femoral venous oxygen saturation decreased during knee exercise at 15 and 25% MVC, but barely changed at 50% MVC, indicating therefore that exercise at higher intensity is more dependent on anaerobic glycolysis than the oxygen supply. When the circulation was restricted or occluded due to increased mechanical compression, the skeletal muscle oxygen availability was reduced (Hultman & Sjoholm 1983) and the anaerobic ATP turnover rate increased more in type I fibre than in type II fibres (Greenhaff et al. 1993). Taken together, these pieces of evidence suggest that during static handgrip exercise, oxygen availability with respect to the oxygen demand of the muscle decreases as the muscle contraction force increases and anaerobic energy turnover becomes predominant at higher intensities with consequent production of more metabolites that induce vasodilation. However, when the exercise duration was short, the augmented vasodilator force was opposed by the increased compression of the vessel, which resulted in a large difference between the blood flow during and immediately after exercise. In summary, the forearm blood flow measured in the brachial artery by a Doppler ultrasound method, increased during static handgrip exercise and was maintained at similar levels when the contraction force increased from 10 to 70% MVC, although the blood pressure increased disproportionately as the contraction force increased. At the lower exercise intensities, the increased blood flow was attributable to increased blood flow per pulse, whereas at the higher intensities, it was due to increased heart rate. The post-exercise hyperaemia increased as the exercise intensity increased, suggesting the blood supply and demand mismatch will become greater.

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