Exercise-Related Time Course of Pulsatility Index in Brachial Artery Following Forearm Exercise Assessed by Doppler Ultrasound

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Osada, T. Exercise-Related Time Course of Pulsatility Index in Brachial Artery Following Forearm Exercise Assessed by Doppler Ultrasound. Tohoku J. Exp. Med., 2004, 203 (4), 241-252 — At rest, vascular reactivity assessed by the changes in pulsatility index (PI) is one indicator of vessel stenosis in some clinical/basic science research. However, all types of vessel stenosis do not show an alteration in the PI, because flow perfusion may be maintained by the development of collateral vessels such as in severe arterial stenosis or non-severe arterial stenosis. Therefore at rest, changes in the PI may not always be a precise indicator of vessel stenosis. However, a few studies have used the PI following exercise, which may provide additional information on hemodynamics. The purpose of the present study was to examine the exercise-related time course of the PI in the brachial artery after ischemic or non-ischemic isometric handgrip exercise (IHE) using Doppler ultrasound, and to determine the potential use of this parameter as an indicator of vascular disease. Ten healthy young male subjects performed IHE at 10% and 30% of maximum voluntary contraction (MVC) for 2-minutes (min) with or without arterial occlusion (AO), or 2-min of AO alone. Following each 2-min session, PI was determined during the 5-min recovery period. A significant difference in the recovery PI was observed between IHE, ischemic IHE, as well as AO alone. Exercise with AO significantly increased the reduction in the PI compared to exercise alone, or AO alone, at both 10% and 30%MVC. These results suggest, exercise-induced changes in the time course of the PI during recovery may potentially be a useful diagnostic tool. Exercise-induced ischemic state may potentially be a useful indicator for detecting arteriovascular disease, even if it is not detected by AO alone. —— pulsatility index; brachial artery; isometric handgrip exercise; arterial occlusion

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It is important for cardiovascular disease based arteriosclerosis to be effectively diagnosed for early intervention. This is especially important due to the increasing incidence of hypertensive diseases such as diabetes mellitus (Clarkson et al. 1996) and hypercholesterolaemia (Celermajer et al. 1992; Vogel et al. 1996). Doppler ultrasound provides clinical information on several blood flow parameters including; changes in vessel diameter, blood velocity and pulsatility index (PI) determined by flow profiles (Taylor et al. 1985; Lewis et al. 1986; Clarkson et al. 1996; Kato 1998). Vascular reactivity assessed by the change in the PI has been used as a diagnostic tool for determining peripheral arterial stenosis in some previous clinical/basic science research studies of the cardiovascular system (Nomura et al. 1996; Perko et al. 1996; Kato 1998). In addition, the blood flow dynamics following the release of temporal arterial occlusion (AO) has been used as a model to reflect a limited oxygen supply to healthy active muscles (Osada et al. 2003).

In a previous study, Kato (1998) reported that an alteration in the blood velocity profile may be one indicator of severe obliterative stenotic arterial lesions. This velocity profile tended to be flatter and without a clear peak systolic component, which is quite different from a non-stenotic lesion. On the other hand, Flanigan et al. (1981, 1982) reported difficulty detecting obliterative arterial lesions from the PI or blood velocity profile despite observing a different shape in the blood velocity profiles. It has been suggested the reduced blood flow is offset by the development of collateral vessels, which maintains the oxygen supply to the claudicated limbs even in the presence of a severe stenotic lesion. Therefore, the PI or blood velocity profile under resting conditions may not always be a precise indicator of obliterative arterial lesions.

Intermittent claudication has been previously used during walking exercise to diagnose the initial stages of arteriosclerosis. Prolongation of muscle oxygenation recovery time to resting levels has been found in the calf muscle following walking exercise in patients with peripheral vascular disease (Komiyama et al. 1994; Kooijiman et al. 1997). This finding suggests a combination of exercise and arterial stenosis or occlusion could be a useful approach for determining obliterative arterial lesions. However, in a clinical setting, the PI or blood velocity parameters are normally estimated under resting conditions, not in relation to exercise. Consequently, the time course of blood velocity and the PI in relation to exercise (exercise-induced ischemia due to a limited blood flow supply) may provide valuable information on peripheral vascular disease, which may not be observed under resting conditions.

The purpose of the present study was to examine the exercise-related time course changes in PI from blood velocity parameters in the conduit brachial artery following handgrip exercise with AO (ischemic exercise = exercise state of obliterative stenotic arterial model) and without AO in healthy young males. Furthermore, the possible benefits of exercise as a potential diagnostic tool of obliterative arterial lesions are discussed.

**SUBJECTS AND METHODS**

**Participants**

Ten healthy, untrained male individuals without a history of cardiovascular disease participated in the study. The mean±S.E. (range) age, height and weight were 29.8±2.0 years (21-39 years), 168.8±1.8 cm (158.0-178.0 cm) and 68.8±1.9 kg (62.0-79.0 kg), respectively. The mean±S.E. (range) maximum (100%) voluntary contraction (MVC) measured for the right handgrip was 46.7±2.0 kg (36.0-56.9 kg). The study was conducted according to the principles of the Declaration of Helsinki (1976) with the approval of the institutional Ethics Committee. All of the participants were informed of the nature, purpose and risks involved in the study before giving their written consent to participate.
Experimental design

A practice session helped familiarize the participants with the isometric handgrip exercise (IHE). Participants performed a maximal IHE with the right hand in a supine position using a custom-designed Meiko Handgrip Ergometer (Meiko, Tokyo) connected to a strain gauge to automatically record torque. The right forearm was immobilized to avoid extraneous muscle movement. Maximal torque (N \times m) was defined as MVC, and 10% and 30% of MVC during IHE were defined as 10%MVC-IHE and 30%MVC-IHE, respectively. AO was established in the brachial artery of the forearm by placing a cuff tourniquet inflated to 280-300 mmHg on the right upper arm. In the present study, the IHE combined with artificial ischemia induced by AO was defined as “ischemic exercise” and is analogous to a model of an “exercise state with peripheral vascular disease (measurement site reflects a limited oxygen supply from the upper stream)”.

In other words, exercise enhances the ischemic effect in peripheral vascular disease. Forearm vessel resistance (pulsatility index, see PI section) was measured after IHE with or without AO.

The participants completed five different sessions, 10%MVC-IHE and 30%MVC-IHE with or without AO for 2-min and 2-min AO alone. The five 2-min protocols were as follows: 1) 10%MVC-IHE, 2) 10%MVC-IHE with AO, 3) 30%MVC-IHE, 4) 30%MVC-IHE with AO, and 5) AO alone. Following a 10-min period of supine relaxation, the participants performed the exercise protocols and AO alone. AO alone was assessed first before 10% or 30% MVC-IHE with/without AO. The protocols of 10% and 30%MVC (with/without AO) were performed on separate days. The amount of time between exercise bouts was verified as sufficient to allow blood velocity to return to resting levels. An adequate recovery period of 3 hours was given between the exercise protocols with and without AO. A 5-min recovery period followed the 2-min sessions of IHE or AO alone, during which blood velocity was measured at 1 second, every 5 seconds for the first minute, and then every 30 seconds between 1 and 5 min of recovery. In total, values were taken from twenty-one time points during recovery as shown in Fig. 1.

Blood velocity measurements

Blood velocity was continuously measured and was determined using a Doppler instrument, (SONOS 1500, ultrasound imaging system, HP 77035A, Hewlett Packard, Tokyo) consisting of a real-time, two-dimensional, ultrasonic imager with a pulsed Doppler flowmeter, and a video tape recorder (videocassette recorder AG-7350-P, Panasonic, Tokyo). Blood velocity was measured in the brachial artery of the forearm. Two-

![Five experimental protocols](image)

Fig. 1. Experimental protocol.

Forearm blood velocity and pulsatility index in the brachial artery were continuously measured at rest and during 5-min of recovery. Measurements began immediately upon the completion of the 2-min session (1 second), every 5 seconds for the first minute, and every 30 seconds between 1-5 minutes of recovery.
Fig. 2. Blood velocity profile in one subject.

Blood velocity in the brachial artery shown at rest (A), as well as immediately (within 5 seconds) after the 2-min session for each protocol (B-F). A: at rest, B: AO alone, C:10%MVC-IHE, D: 10%MVC-IHE with AO, E: 30%MVC-IHE and F: 30%MVC-IHE with AO. The peak-systolic maximum blood velocity ($V_{\text{max}}$) and end-diastolic minimum blood velocity ($V_{\text{min}}$) and mean blood velocity ($V_{\text{mean}}$) by integration of the outer (maximum) envelope in the flow profile were automatically determined in this manner. PI was automatically calculated according to the following formulas: $PI = (V_{\text{max}} - V_{\text{min}})/V_{\text{mean}}$ in Fig. 2B. Ischemic exercise shows the higher blood velocity profile (D and F) compared to non-ischemic exercise (C and E).
dimensional echographic images were acquired using a duplex scanner fitted with a 7.5 MHz linear probe at the measuring sites. A probe with a high frequency (7.5 MHz) Doppler signal was used to obtain data from the brachial artery because of its high axial resolution for superficial vessels with a diameter up to 10 mm. A real time imaging system visualized the vessel of the brachial artery 1 cm proximal from the bifurcation in the brachial artery. This allowed the Doppler sample volume to be placed within the lumen of the vessel to obtain Doppler shift signals. The sample volume was maintained at the center of the lumen, and adjusted to cover the width of the vessel and blood velocity distribution. Pulsed Doppler flowmetry analyzed velocity using a 7.5 MHz linear probe at the measuring site. Shallow blood vessels characteristically have low flow velocity; therefore high frequency (7.5 MHz) probes were used. The blood velocity was measured five times at rest, and 21 times during recovery (Fig. 1). An ultrasound beam angle of insonation of less than 60˚ (the angle between the ultrasound beam and the long axis of the vessel) was applied, because wider angles affect the accuracy of the velocity calculation (Gill 1985). Blood velocity was calculated by integration of the outer envelope of the maximum velocity values in the flow profile (Leyk et al. 1994; Isnard et al. 1996; Osada et al. 1999). This was determined by averaging two or three successive cardiac cycles of the vessel. The peak-systolic maximum blood velocity ($V_{\text{max}}$), end-diastolic minimum blood velocity ($V_{\text{min}}$) and time-averaged mean blood velocity ($V_{\text{mean}}$) were automatically determined in this manner. The values of $V_{\text{max}}$ and $V_{\text{min}}$ basically correspond to peak systole and the end-diastolic point, respectively (Fig. 2). Prior to the experiments, intraobserver variability for this operator was determined for the 10 subjects. At rest, the coefficients of variation determined for blood velocity for repeated measurements was 3.5±0.3%. This value is within the acceptable range to provide valid and reproducible data (Osada et al. 2002, 2003; Osada and Rådegran 2002).

**Pulsatility index**

The pulsatility index (PI) quantifies the shape of the blood velocity waveform and was defined as the peak-to-peak amplitude of a waveform, divided by the mean amplitude over the cardiac cycle, as previously described by Gosling and King (1974). Previous studies have reported the physiological significance of PI, which reflects downstream resistance to flow and the correlation between vessel resistance and PI has been closely demonstrated (Gosling and King 1974; Evans et al. 1980; Skidmore et al. 1980; Legarth and Nolsoe 1990). Thus, PI may be used as an effective indicator of changes in peripheral vascular resistance following exercise. Measuring the PI of the blood velocity waveform in the conduit artery supplying the muscle can easily assess vascular impedance after exercise. The index of vessel resistance obtained by using the PI was automatically calculated according to the following formula: $\text{PI} = (V_{\text{max}} - V_{\text{min}})/V_{\text{mean}}$, as shown in Fig. 2B. The average value of the PI obtained from the cardiac cycle at rest was defined as 100%. The PI obtained by the conduit arterial velocity amplitude was only determined at rest or during recovery following exercise, but not during exercise. Since there is a large fluctuation of flow velocity (disturbed velocity profile) during rhythmic muscle contraction (Walløe and Wesche 1988), PI cannot be determined in a conduit artery feeding a working muscle during exercise (it is impossible to detect a clear pulse wave of a blood velocity profile).

Vessel resistance following 1) 10%MVC-IHE, 2) 10%MVC-IHE with AO, 3) 30%MVC-IHE, 4) 30%MVC-IHE with AO, and 5) AO alone, was determined using the PI. The time course (kinetics) of the %PI after 2-min AO alone was measured using an upper-arm cuff tourniquet. The duration of the reperfusion phase for the %PI was defined as the time required from cuff release, to when PI returned to its resting level (100%). In the present study, the reperfusion phase was defined as the time period showing no significant
difference in the %PI compared to resting value. The inflated arm cuff with a pressure of >250 mmHg for 2 minutes initiates the reactive hyperemia after both IHE and AO alone. In each experiment, the pressure achieved by the inflated cuff tourniquet was released within a few seconds by tearing off the cuff belt by the Doppler operator. The operator tried to maintain the probe (sampling volume) at the same position on the measured artery for measuring blood velocity starting from rest, during the 2-min protocol session and 5-min recovery. Furthermore, the sampling volume was fixed at the same position immediately after the 2-min session, even if the measured artery was shifted by the contracted muscle or by pressuring the inflated cuff tourniquet.

**Statistical analysis**

Values are presented as means±s.e. Statistical comparisons within each measured group parameter were performed by one-way analysis of variance (ANOVA) for factor measurements, and the difference for each measurement was determined by Bonferonni’s post hoc comparisons. The level of significance was set at p<0.05.

**RESULTS**

The PI and HR at rest were 3.12±0.05 (mean ±s.e., range 3.01-3.35) and 61±1 (58-64) beats/min, respectively. A previous report found the brachial artery PI values at rest, and immediately after 3-min and 5-min of AO, ranged from 3.0-4.0, 2.0-3.0 and 1.0-2.0, respectively (Legarth and Nolsoe 1990). The PI values obtained in the present study were in a similar range as the previously reported data. The %PI, maximum, mean and minimum blood velocity values in the brachial artery after IHE with or without AO at 10% and 30% MVC, and after AO alone are shown in Fig. 3. The %PI was significantly lower for IHE with, than without AO for up to 60 seconds immediately after IHE at 10%MVC and until 210 seconds at 30%MVC. Furthermore, 1 second immediately after 30%MVC IHE with and without AO, the %PI was reduced to 26% and 38%, respectively, and to 31% and 54% respectively, at 10%MVC. Additionally, %PI was reduced to 37% for AO alone. Moreover, the %PI at IHE with AO was significantly lower than AO alone during the first 150 seconds at 10%MVC, and up to 300 seconds following 30%MVC. Although %PI for AO alone was lower than at 10%MVC over 1-5 seconds, the %PI at 10%MVC was significantly lower than for AO alone between 15-120 seconds. The %PI at 30%MVC was significantly lower than for AO alone between 10-240 seconds.

During the recovery period of AO alone, the %PI returned to resting level within 30 seconds (significantly lower at 1-25 seconds during recovery compared to resting, see ↑ in Fig. 3), which was faster than for both IHE intensities with or without AO. The %PI for IHE with AO tended to be lower than for IHE without AO during the entire recovery phase at both 10% and 30%MVC. Specifically, the %PI for IHE with AO was significantly (p<0.05) lower than without AO from 1-60 seconds at 10%MVC and 1-210 seconds at 30%MVC. The %PI for 30%MVC was lower than 10%MVC, both, with and without AO. Fig. 4 shows the statistical significance in the reduced %PI compared to resting levels. The reduction of the PI was prolonged for higher intensity exercise and under ischemic conditions (AO).

**DISCUSSION**

Stenosis in the artery is characterized by elevation of the blood velocity over the stenotic area, reduction of the velocity caudally to the stenosis and reduction of the peripheral artery resistivity (Lilly et al. 1989; Moneta et al. 1991). Generally, a decrease in the PI following exercise is related to vasodilation in the feeding arterial bed and decreased downstream resistance. Therefore, a discrepancy in the PI dynamics in patients compared to healthy people will occur even if a stenotic lesion is located below or above the measurement site. In other words, more limited blood
Exercise-Related Changes in PI

Fig. 3. Changes in pulsatility index and maximum, mean and minimum blood velocity in the brachial artery after ischemic and non-ischemic isometric handgrip exercise.

The %PI is reduced after ischemic exercise compared with non-ischemic exercise and AO alone. The %PI after AO alone, rapidly returned to resting level within 30 seconds (↑). During recovery, the %PI returned to resting level within 30 seconds (significantly lower at 1-25 seconds during recovery compared to resting), which was more rapid for AO alone than for IHE with or without AO. Statistical significance (p<0.05). Ischemic IHE caused prolonged post exercise hyperemia compared with non-ischemic exercise and AO alone. ●, AO alone; ○, IHE; □, IHE with AO. Values are expressed as means±S.E.
Fig. 4. Comparisons of %PI at various times during recovery.

Percent PI was dramatically reduced immediately after the 2-min session at each protocol. The reduction of PI was prolonged for high intensity exercise (30%MVC-IHE) and under ischemic conditions (with AO). Furthermore, at 30 seconds during recovery, %PI at exercise state was still lower despite the %PI for AO alone had returned to resting level. Statistical significance (p<0.05) compares resting level of 100%, as well as (p<0.05) between ischemic and non-ischemic IHE. Values are expressed as means±e.

A: 300 seconds, B: 240 seconds, C: 180 seconds, D: 120 seconds, E: 60 seconds, F: 30 seconds, G: immediately after.
flow perfusion shows lower blood velocity from the upper stream, while a limited oxygen supply results in an increase in the production of vasodilatory metabolites downstream in the feeding artery. However, it is not easy to detect obliterative arterial stenosis at rest due to various anatomical variations in developed collateral vessels, or if stenosis is located deep below the surface. It was speculated that since exercise enhances the ischemic effect in arteriosclerosis this model might be a useful method for detecting the disease.

The main purpose of the present study was to examine the features of the PI following a model of handgrip exercise with AO (exercise state of obliterative stenotic arterial model ≈ ischemic exercise), handgrip exercise alone (exercise state on healthy arterial model ≈ non-ischemic exercise) and AO alone. An additional goal of this study was to use the changes in the PI as a means of identifying arterial vascular disease. An explanation of the possible merit and the methodological implications of these findings are covered in the following discussion.

Characteristics of ischemic exercise model

The present study clearly demonstrated the prolongation of a reduced PI (≈ reduced vessel resistance) after IHE with AO (ischemic exercise) compared to IHE (non-ischemic exercise), as shown in Figs. 3 and 4. Furthermore, the reduced PI for IHE was larger at 30%MVC than at 10%MVC both with and without AO (Fig. 4). This phenomenon indicates that isometric exercising muscle following AO (limited oxygen supply) has greater recovery blood flow as previously described by Osada et al. (2003), resulting from a reduction in vascular tone and vessel resistance. This could be due to an accumulation of vasodilator metabolites causing exercise-induced hyperemia. The significant differences in the time course of the PI between ischemic and non-ischemic exercise provides important information for assessing muscle hemodynamics in patients with peripheral vascular disease. Moreover, the exercise intensity or duration could be key determinants for the diagnosis of peripheral vascular disease.

In regards to AO alone, the large difference in the time course of PI between AO alone and ischemic/non-ischemic IHE during recovery was clearly demonstrated in Fig. 3. A vascular event initiated by restoration of the circulation after temporal AO was clearly demonstrated. The initial vasodilation (reactive hyperemia) following blood flow perfusion after releasing AO may be caused by a loss of myogenic tone (Bayliss 1902), in other words, a greater reduction in PI immediately after AO is dependent upon transient lower peripheral vascular resistance. In the present study, a 63% reduction in the PI value immediately after 2-min of AO alone rapidly returned to the resting level within 30 seconds, with a lower post occlusive hyperemia (Fig. 3). This value is similar to previous data that showed a 75% reduction in PI immediately after 5-min of AO in the brachial artery (Legarth and Nolsoe 1990). In the present study, the breaking point in %PI, as well as blood velocity at 30%MVC-IHE with AO was seen at 30 seconds (see reperfusion phase, ↑ in Fig. 3). This may be influenced by the initial phase of vasodilation following the release of the tourniquet, which for AO alone represents the period within 30 seconds. As mentioned above, it has been suggested that the reperfusion phase (within 30 seconds after releasing cuff AO due to temporal circulatory arrest) obtained in the present study may influenced by myogenic tone and the degree of vessel distension due to post-occlusive hyperemia. If this is indeed the case, the %PI kinetics after the initial 30 seconds may be more valuable for interpreting circulatory arrest. On the other hand, this reperfusion is observed not only in AO alone, but also in exercise with/without AO. So the significance of this initial 30-second recovery phase remains unclear.

Possible effects of exercise

In the present study, an ischemic exercise model combined with AO (exercise state with
severe arterial stenosis) would indicate a greater production of vasodilator metabolites. A schematic illustration of the possible effect of exercise is shown in Fig. 5. Clinically, the patients with severe limb arterial stenosis have a shorter exercise duration with intermittent claudication due to ischemia in the working limb skeletal muscle. Exercise performance is closely related to exercise capacity, which in turn is related to peripheral factors such as muscle oxygen supply, as well as oxygen consumption in working skeletal muscle.

In Fig. 3, the present data indicated; 1) a statistically significant difference in the %PI between ischemic and non-ischemic exercise was shown at 10%MVC (until 60 seconds) and 30%MVC (until 210 seconds). This may indicate a higher exercise intensity potentially provides better diagnosis for disease. 2) The time duration of the %PI between “ischemic/non-ischemic exercise” and “AO” alone was significantly longer at 30%MVC (until 300 seconds at ischemic and until 240 seconds at non-ischemic exercise) compared to 10%MVC (until 150 seconds at ischemic and until 120 seconds at non-ischemic exercise). This suggests exercise influences hemodynamics more during recovery than circulatory arrest. 3) A significantly longer period with a reduced PI state was seen in ischemic exercise compared to AO alone (see ⋆ in Fig. 3), despite similar %PI values between non-ischemic exercise and AO alone (see ⋆ in Fig. 3). This could be one piece of evidence supporting ischemic exercise corresponds to an exercise state with obliterative arterial disease. This data suggests an exercise-induced ischemic state could be more beneficial for assessing obliterative arterial stenosis by using the differences in the PI recovery time course following exercise at different intensity levels.

Limitations in the present study

It has been frequently argued that hemodynamic limitations are poor predictors of exercise performance in peripheral vascular disease and intermittent claudication (Green 2002). The main argument for this is based upon blood flow data following ischemic exercise. In the present study, it is obvious that the ischemic exercise model used is not identical to physiological conditions or to peripheral vascular disease, but still provides insight to the condition. It could closely resemble the ischemic state model describing the limited blood flow supply during recovery, if moderate cuff pressure in the artery was added during the recovery phase after the 2-min session. However, this would influence the blood velocity profile by pooling the venous blood volume. Thus, applying...
moderate cuff tourniquet pressure was not performed during recovery. Additionally, this study did not determine which exercise intensity and/or duration are appropriate for testing (2-min exercise and 10% or 30%MVC-IHE were only used in the present study).

The difference between forearm (relatively small vessel, the brachial artery, with a small forearm finger flexor muscle mass) and lower limb exercise (relatively large vessel of femoral-iliac artery and large thigh muscle mass) may also influence the PI or blood velocity profile during recovery. Furthermore, it is speculated that the changes in the PI in the measured artery would vary between a proximal and distal lesion of arteriovascular disease, due to the degree of the developed collateral vessel flow. For instance, the velocity response in the femoral artery could be seen as low perfusion flow (due to limited blood flow supply) despite a reduced PI (due to relatively higher value of minimum end-diastolic velocity compared to maximum systolic velocity) in lesions located in the iliac arterial area (proximal). In contrast, lesions below the bifurcation of the femoral artery would show a higher systolic velocity and lower end-diastolic velocity. This results in relatively higher PI values because of higher vascular resistance in distal lesion stenosis.

**CONCLUSION**

It was demonstrated that the reduced PI following IHE was more prolonged with AO, than without, which could be partly due to enhanced forearm vasodilator activity. Ischemic exercise may induce larger changes in recovery PI dynamics. This could potentially be an indice of arteriosclerosis not only at rest, but also following exercise. In future studies, exercise related changes in PI could be examined in patients with peripheral vascular disease.

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