Endurance Exercise Training Increases Peripheral Vascular Response in Human Fingers

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Abstract: The purpose of this study was to clarify whether peripheral vascular response to alteration of transmural pressure is changed by endurance exercise training. The healthy male subjects (training group; n=6) performed endurance exercise training that consisted of cycle ergometer exercise 5 d · week⁻¹ and 30 min · d⁻¹ for a period of 8 weeks. Changes in the peripheral vascular response to alteration of transmural pressure in the human finger were measured by a differential digital photoplethysmogram (ΔDPG) and blood pressure during passive movement of the arm to different vertical hand positions relative to heart level. Following 8 weeks of endurance training, percent changes in ΔDPG from heart level in the training group increased significantly (mean±SD, −48.1±7.3 to −58.7±9.3% at the lowered position, 46.1±13.4 to 84.6±8.8% at the elevated position, p<0.05). Similarly, the arterial compliance index, which was calculated from ΔDPG-P wave amplitude and arterial pulse pressure, also significantly changed in the training group over the 8 weeks (5.6±1.3 to 2.7±1.6 mV · V⁻¹ · s⁻¹ · mmHg⁻¹ at the lowered position, 30.0±12.4 to 54.4±18.9 mV · V⁻¹ · s⁻¹ · mmHg⁻¹ at the elevated position). Maximal oxygen uptake (VO₂max) was significantly increased in the training group. On the other hand, the control group (n=6) showed no significant changes in all parameters for 8 weeks. Therefore, these results suggest that endurance exercise training induces an increase in peripheral vascular response to alteration of transmural pressure in the human finger. [Japanese Journal of Physiology, 48, 365–371, 1998]

Key words: photoplethysmogram, peripheral vascular response, endurance training.

Local vascular reaction, which is produced mainly by changes in resistance arteries, was induced by raising or lowering of an extremity relative to heart level [1–3]. The local vascular response has been reported to play an important role in the autoregulation of blood flow in microcirculation [4, 5]. Numerous studies have also documented that local vascular responsiveness to various stimuliants is necessary for the maintenance of blood flow and blood pressure in the tissues and the body as a whole [4–6]. One widely used method to measure blood vessel statement is photoplethysmography. This technique is simple to use and a noninvasive method of measuring microvascular perfusion in a small volume [7–10]. Recently, Takemiya et al. demonstrated that peripheral vascular response to alteration of transmural pressure can be measured by using the differential digital photoplethysmogram (ΔDPG) with movements in arm position [11–15].

The influences of exercise training on the cardiovascular system have been studied extensively and are well documented [16, 17]. The endurance exercise
training induces a change in arteriolar responses to various substances and changes in arterial or arteriolar wall thickness and components [18, 19]. During exercise, peripheral vascular responsiveness not only regulates a maintenance of blood pressure, but also plays a role in the delivery and redistribution of blood flow to the active tissues [19]. To our knowledge, however, no report has been made on whether a change in peripheral vascular response to alteration of transmural pressure occurs as a result of endurance exercise training in humans.

The purpose of this study was to examine the hypothesis that endurance exercise training may contribute to changes in the peripheral vascular response induced by alterations in transmural pressure in humans. For this purpose, ΔDPG with movements in arm position at rest was performed on training and control groups during a period of 8 weeks.

**METHODS**

**Subjects.** Twelve healthy males who were uninvolved in a regular training program and who have no history of cardiorespiratory diseases volunteered to participate in this study. Their physical characteristics and parameters of exercise performance are shown in Table 1. Each subject was assigned randomly to either the training group (n=6) or the control group (n=6). All subjects were informed of the nature of the experiment, and they gave informed consent before testing. No significant differences existed in the physical and physiological parameters between the two groups before training.

**Experimental procedures.** The measurement of peripheral vascular response to the alteration of transmural pressure and maximum oxygen uptake ($V_{O2_{max}}$) was taken in both groups at 2-week intervals commencing at the beginning of the 8 weeks of endurance exercise training. All experiments were performed in a room with a constant ambient temperature that varied from 22 to 24°C over the 8-week experimental period. In the peripheral vascular response measurement, the subjects in both groups spent 30 min of quiet relaxation in a sitting position; then a series of ΔDPG and finger blood pressure (BP) measurements were performed for about 30 min. After these measurements, $V_{O2_{max}}$ was measured.

The subjects of the training group performed exercise training on a bicycle ergometer. Each training session lasted 30 min with a pedaling frequency of 60 rpm. This included a 3 min warm-up at the predicted 50% $V_{O2_{max}}$ intensity, followed by 24 min of training at the predicted 80% $V_{O2_{max}}$ intensity and a 3 min cool-down at the same intensity of the warm-up. The subjects trained 5 d·week$^{-1}$ for 8 weeks.

**Table 1. Physical and physiological characteristics of subjects.**

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>$V_{O2_{max}}$ (l·min$^{-1}$)</th>
<th>$V_{O2_{max}}$ (ml·kg$^{-1}$·min$^{-1}$)</th>
<th>HR$_{max}$ (beats·min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T</td>
<td>23.5±0.8</td>
<td>170.8±6.7</td>
<td>71.4±7.8</td>
<td>3.65±0.45</td>
<td>50.7±5.4</td>
</tr>
<tr>
<td>U</td>
<td>23.8±0.8</td>
<td>173.3±3.9</td>
<td>69.2±7.7</td>
<td>3.39±0.48</td>
<td>49.5±4.6</td>
</tr>
</tbody>
</table>

Values are means±SD. T, training group; U, control group.

**Differential digital photoplethysmogram.** A continuous recording on a differential digital photoplethysmogram (ΔDPG) during changes in arm position was obtained from the forefinger of the left hand by connecting, in series, a digital photoplethysmogram (MLV-2201, Nihon Kohden, Japan) to an AC amplifier with a time constant of 0.03 s. The main P wave of ΔDPG was recorded precisely without fluctuations from the baseline. In a previous study, Takemiya et al. had confirmed that the amplitude of the ΔDPG-P wave was linearly proportional to that of the percussion wave of the digital photoplethysmogram ($r=0.98$) [15].

**Finger blood pressure.** Finger arterial blood pressure (BP) was monitored continuously by using a Finapres blood pressure monitor (Model 2300, Ohmeda, USA). The recording was made from a cuff positioned on the second phalanx of the middle finger on the left hand.

**Arterial compliance index.** The calculated index of arterial compliance was obtained from the amplitude of the differential photoplethysmogram, indicating changes in vascular volume, divided by pulse pressure, indicating changes in vascular transmural pressure [12–14].

**Changing arm position.** To elicit a peripheral vascular response, changes in vascular transmural pressure were produced by changing the arm position, lowering and elevating the upper extremity from the heart level. Arm position changes were performed at a sitting position. The left arm, from elbow to fingers, was placed comfortably on a flat, horizontal board. The passively lowered or elevated arm position was produced by lowering or raising the board until the tip of the finger was 50 cm below or above the heart level.
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Fig. 1. Original recordings of ΔDPG, BP, and PR for different arm position. ΔDPG, differential digital photoplethysmogram; BP, finger blood pressure; PR, pulse rate.

The measurement in the lowered or elevated arm position was performed for about 30–60 s (Fig. 1). This maneuver has been used in the alteration of intravascular pressure of the finger vasculature in previous studies [1, 11–15].

Maximal oxygen uptake. Each subject performed the intermittent incremental load test to determine maximum oxygen uptake (\(\dot{V}O_2\text{max}\)). All subjects underwent several work sessions on a bicycle ergometer (pedaling frequency = 60 rpm) with enough rest periods between sessions. The initial work load was 120 W. Work loads increased by 30–60 W each session until oxygen uptake (\(\dot{V}O_2\)) had leveled off. At submaximal intensity, the subjects exercised for 10 min. They were considered exhausted (usually after five to six rides) when they could no longer pedal for at least 3 min. Subjects breathed through a low-resistance two-way valve, and the expired gas was collected in a Douglas bag during the last minutes of each session. Expired \(O_2\) and \(CO_2\) gas concentrations were measured by mass spectrometry (Perkin-Elmer, 1100), and gas volume was determined by use of a dry gas meter (Shinagawa Dev. N5, Japan). Heart rate (HR) was recorded electrocardiographically (Nihon Kohden, OEC-6501) for the duration of each session. The peak HR value was expressed as HR\(_{\text{max}}\). The criteria for attaining \(\dot{V}O_2\text{max}\) were (1) a leveling off or a decrease in \(\dot{V}O_2\) with an increasing work load, (2) HR±10% of age-predicted maximum (220-age), and (3) respiratory exchange ratio ≥1.0. Oxygen uptake as measured during maximal exhaustive exercise was determined to be maximum oxygen uptake when two of the three criteria were satisfied [20, 21].

Statistical analysis. The analysis of ΔDPG was restricted to the mean amplitude of its main P wave (mV · s\(^{-1}\) · s\(^{-1}\)). Stable P wave amplitudes in each condition were calculated as the average of a 10 s measurement period [11–15]. ΔDPG amplitude values at lowering or raising arm positions were expressed as percent changes from heart level. Also, the arterial compliance index value was expressed as the average (mV · s\(^{-1}\) · mmHg\(^{-1}\)) [11–15]. The differential changes in the parameters during the experimental periods between training and control groups were compared by using a two-way analysis of variance (ANOVA) with repeated measurements. Differences in the parameters during an experimental period within each group were analyzed by the Tukey post hoc test. The values were reported as the mean and standard deviation (SD). The level of significance was established at \(p<0.05\).

RESULTS

Figure 1 shows typical records of the differential digital photoplethysmogram (ΔDPG), finger blood pressure (BP), and pulse rate (PR). The ΔDPG amplitude decreased at the lowered position and increased at the elevated position. PR did not change during movements in the arm position.

The ΔDPG amplitude value at heart level did not change statistically for 8 weeks in both groups. Similarly, finger blood pressure at heart level, at elevated, and at lowered arm positions exhibited no change in both groups throughout the experimental sessions, as shown in Table 2.

A significant difference in the percent change in ΔDPG was evident at the lowered position between training and control groups during the 8 weeks (\(F=2.68, p<0.05\)), as shown in Fig. 2. At the lowered arm position, percent changes in ΔDPG consistently increased in the training group over the 8 weeks of endurance training (−48.1±7.3 to −58.7±9.3%), but not in the control group (−44.0±11.7 to −42.4±7.4%). In the training group the percent changes in ΔDPG at lowered arm position at 6 and 8 weeks were significantly (\(p<0.05\)) higher than before training. Similarly, a significant difference in the percent change in ΔDPG occurred at the elevated position between the training and control groups during the 8 weeks (\(F=13.39, p<0.05\)). The percent changes in ΔDPG at the elevated arm position in the training group consistently increased (46.1±13.4 to 84.6±8.8%), but no changes were detected in the control group, as shown in Fig. 2 (55.0±16.8 to 48.3±20.3%). In the training group, the percent changes in ΔDPG at lowered arm position at 6 and 8 weeks were significantly (\(p<0.05\)) higher than before training.

Figure 3 shows the changes in the arterial compliance index in both groups over the 8 weeks. The arterial compliance index at the lowered arm position consistently decreased in the training group during the 8
Table 2. Values of digital blood pressures in both groups during 8 weeks.

<table>
<thead>
<tr>
<th></th>
<th>0 week</th>
<th>2 weeks</th>
<th>4 weeks</th>
<th>6 weeks</th>
<th>8 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MBP</td>
<td>ΔMBP</td>
<td>MBP</td>
<td>ΔMBP</td>
<td>MBP</td>
</tr>
<tr>
<td>T</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HL</td>
<td>78.8</td>
<td>±7.7</td>
<td>77.9</td>
<td>±7.6</td>
<td>76.1</td>
</tr>
<tr>
<td>Up</td>
<td>38.9</td>
<td>±36.0</td>
<td>34.1</td>
<td>±34.4</td>
<td>35.6</td>
</tr>
<tr>
<td>Down</td>
<td>113.4</td>
<td>±10.4</td>
<td>106.2</td>
<td>±14.4</td>
<td>117.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>±12.1</td>
<td>±9.9</td>
<td>±14.2</td>
<td>±11.0</td>
</tr>
<tr>
<td>U</td>
<td>82.5</td>
<td>±15.0</td>
<td>84.0</td>
<td>±14.6</td>
<td>83.0</td>
</tr>
<tr>
<td>HL</td>
<td>41.3</td>
<td>±35.3</td>
<td>43.2</td>
<td>±33.3</td>
<td>41.8</td>
</tr>
<tr>
<td>Up</td>
<td>±14.9</td>
<td>±7.6</td>
<td>±14.0</td>
<td>±5.1</td>
<td>±16.2</td>
</tr>
<tr>
<td>Down</td>
<td>113.9</td>
<td>±39.6</td>
<td>112.1</td>
<td>±40.7</td>
<td>114.9</td>
</tr>
<tr>
<td></td>
<td>±16.5</td>
<td>±4.3</td>
<td>±24.7</td>
<td>±4.0</td>
<td>±17.0</td>
</tr>
</tbody>
</table>

Values are means±SD (mmHg). T, training group; U, control group. HL, heart level; Up, elevated arm position; Down, lowered arm position; MBP, mean blood pressure; ΔMBP, difference of mean blood pressure between the heart level and elevated or lowered arm positions.

Fig. 2. Changes in ΔDPG at the lowered arm position (A) and at the elevated arm position (B) over 8 weeks of endurance training in the training (n=6) and the control groups (n=6). *Significantly different from before training (p<0.05). Values are means±SD.

Fig. 3. Changes in the arterial compliance index at the lowered arm position (A) and at the elevated arm position (B) over 8 weeks of endurance training in the training (n=6) and the control groups (n=6). *Significantly different from before training (p<0.05). Values are means±SD.

weeks (5.6±1.3 to 2.7±1.6 mV · V⁻¹ · s⁻¹ · mmHg⁻¹), however, no change was observed in the control group (5.2±2.5 to 4.9±2.9 mV · V⁻¹ · s⁻¹ · mmHg⁻¹). A statistically significant difference was noted in the percent change in arterial compliance index between the training and control groups over the 8 weeks (F=2.87, p<0.05). In the training group, the percent changes in compliance index at lowered arm position at 8 weeks were significantly (p<0.05) lower than before training. Similarly, a statistically significant difference in the percent change in the arterial compliance index at the elevated arm position was found between the
Table 3. Values of $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) obtained during 8 weeks of endurance exercise training.

<table>
<thead>
<tr>
<th>Training duration (week)</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>T</td>
<td>50.7±5.4</td>
<td>50.8±4.9</td>
<td>53.2±4.2</td>
<td>53.9±3.3</td>
<td>54.6±3.5*</td>
</tr>
<tr>
<td>U</td>
<td>49.5±4.6</td>
<td>47.7±4.6</td>
<td>48.3±5.1</td>
<td>48.4±4.5</td>
<td>48.7±4.4</td>
</tr>
</tbody>
</table>

Values are mean±SD. T, training group; U, control group. $\dot{V}O_2$ max, maximal oxygen uptake. *Significantly different from before training ($p<0.05$).

training and control groups during the experimental period ($F=3.11$, $p<0.05$). At the elevated position, the arterial compliance index consistently increased in the training group (30.0±12.4 to 54.4±18.9 mV·V$^{-1}$·s$^{-1}$·mmHg$^{-1}$), but not in the control group (31.3±20.8 to 29.4±22.9 mV·V$^{-1}$·s$^{-1}$·mmHg$^{-1}$). In the training group, the percent change in compliance index at elevated arm position at 8 weeks was significantly ($p<0.05$) higher than before training.

Changes in $\dot{V}O_2$ max for the 8 weeks of exercise training are shown in Table 3. A statistically significant difference in $\dot{V}O_2$ max developed between the training and the control groups during the 8 weeks ($F=5.55$, $p<0.05$). In the training group, $\dot{V}O_2$ max at 8 weeks was significantly ($p<0.05$) higher than before training.

**DISCUSSION**

In the present study, it was found that endurance exercise training for the 8-week exercise period induced an increase in the percent change in the ΔDPG amplitude at lowered and elevated arm positions (Fig. 2). In an evaluation for vascular response to transmural pressure, it must be considered that a change in the volume corresponds to a change in the pressure. Thus in this study, the arterial compliance index was calculated by dividing the amplitude of ΔDPG by the pulse pressure [12–14]. No significant changes were found in the finger arterial pulse pressure with movements of arm position during the 8 weeks for both groups, but the compliance index in the training group consistently decreased at the lowered arm position and increased at the elevated position (Fig. 3). Recently, Takemiya et al. suggested that the observation of ΔDPG amplitude with movements of arm position may be the simplest method by which to evaluate the peripheral vascular response in humans [11–15]. Furthermore, local vascular response induced by the raising or lowering of an extremity relative to heart level was mainly due to changes in resistance arteries [1, 3]. Therefore these results suggest that endurance exercise training induced an increase in constrictive and dilative responses of resistance arteries in finger vasculature.

During movements of arm position, several mechanisms may affect the regulation of peripheral vessels. Blood flow regulation or vasoactivity at rest and during dynamic exercise is associated with systemic mechanism, that is, sympathetic nerve activity. Some studies reported a relationship between peripheral vascular response and sympathetic activity [13, 22]. However, there were no statistical differences in ΔDPG amplitude value and blood pressure at heart level in both groups over the 8 weeks. Furthermore, previous studies demonstrated that changes in systemic reactions, for example, HR, arterial blood pressure, and respiration, during the lowering or raising of the arm position relative to heart level were not observed [1, 11, 15]. Similarly, in this study PR did not change during movements of extremities (Fig. 1). Thus the changes of ΔDPG in the finger do represent an effect on the local vascular regulatory mechanism.

It is known that several local vascular regulatory mechanisms exist. One is the myogenic response. The arteriolar myogenic response was first reported by Bayliss [23], and the myogenic response has been well investigated in terms of autoregulation in local blood flow [4, 6, 22, 24–27]. Johnson suggested that the importance of the myogenic mechanism in maintaining homeostasis can be best appreciated by considering the various functions that the peripheral circulation performs [4]. To our knowledge, the effect of exercise training on myogenic response in humans has not been reported. In animal study, however, some investigators observed the influence of the myogenic response on endurance exercise training [25, 28]. They found that exercise training enhances myogenic constrictor responses in resistance arteries, and their results partly correlate with those in this study [25, 28]. Thus it is possible that an enhanced myogenic mechanism may explain the increased peripheral vascular response to the alteration of transmural pressure. The other mechanism that affects local vascular regulation involved endothelial factors. Previous studies have also demonstrated that the endothelium plays an important role in the regulation of arteriolar tone [29, 30] and that acute increases in blood flow stimulate the synthesis of endothelial factors, for example, nitric oxide [29]. Thus in the present study, it is also likely that endurance exercise training for 8 weeks might induce changes in endothelial factors. Because we did not measure those parameters, the influence of exercise training on endothelial factors cannot be ade-
quately discussed. Further investigations are needed to investigate the effects of exercise training on the myogenic response and endothelial-mediated regulatory mechanisms in humans.

Furthermore several studies have shown that exercise training or increased load induces changes in the arterial or arteriolar wall components and thickness [19, 25–28]. Other studies have reported that chronic increased wall stress appears to favor smooth muscle cell hyperplasia with increased wall thickness in the smaller resistance arteries [31–34]. The increase in thickness of the arterial wall in response to elevated pressure has been referred to as “structural autoregulation” [34]. A thickening of the wall would decrease wall tension, and this increase, along with smooth muscle cell hyperplasia, may be related to an enhancement of the vascular response. Since endurance exercise also increases vessel wall stress in arterioles, it may have occurred in the human digital vasculature of the subjects in the present study.

It is thought that the vascular response in resistance arteries plays an important role in blood pressure and/or blood flow regulation during exercise. Higher local vascular response may be an advantageous redistribution of blood flow to the active tissues. Additional research is necessary to test this hypothesis. Furthermore, because this study was conducted on a small number of subjects, further investigations are needed with larger test groups and to compare with other physiological parameters.

In conclusion, we show that endurance exercise training for 8 weeks induced increases in percent changes of ΔDPG amplitude following an alteration of transmural pressure, and a decrease and an increase in the arterial compliance index at the lowered and elevated arm positions in humans. These results suggest that peripheral vascular response to change in transmural pressure in the human finger is increased by endurance exercise training over 8 weeks.

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REFERENCES

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