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Reactive Hyperaemia in the Forearm Skin of Highly Trained Windsurfers

Abstract

We undertook this study to determine whether long-term high intensity exercise would modify cutaneous endothelial-dependent vasodilation. We compared a group of 9 highly trained windsurfers (mean age: 24.5 ± 1.6 years) to a control group of 8 sedentary individuals (22.9 ± 0.4 years, NS). Laser Doppler was used to measure cutaneous blood flow in the resting state (baseline), during post-occlusive hyperaemia (endothelium-dependent vasodilation), and local heating to 42°C. Lipid profile was similar in both groups. Resting heart rate was significantly lower in windsurfers. Baseline cutaneous vascular conductance (CVC) values were similar in both groups (0.059 ± 0.016 and 0.051 ± 0.009).

During reactive hyperaemia, normalized peak CVC value was significantly higher in the windsurfers group (1775.4 ± 286.9 and 826.4 ± 121.7% baseline CVC; p = 0.01). Normalized peak CVC value in response to local heating (42°C) was not significantly different between both groups (2359.4 ± 346.1 and 1467.7 ± 282.6% baseline CVC). Endothelium-dependent vasodilation in cutaneous microcirculation is significantly enhanced in the forearm skin of highly trained windsurfers when compared to sedentary controls.

Key words

Exercise - laser Doppler - endothelium - vasodilation

Introduction

Exercise training induces adaptation of the cardiovascular system, resulting in improvement of oxygen intake by both cardiac and skeletal muscles [10,35]. Adaptation mechanisms include improvement of endothelial function via modifications in synthesis and release of endothelium-derived factors. These beneficial effects have been shown in animal models [4,7] and in human [13,15,21]. On the contrary, studies after bed rest or simulated microgravity exposure showed that vasodilator responses were reduced [5,25]. Altered response is suggested to be related to several factors in this case: reduction in plasma volume, decreased ability to translocate blood from the splanchnic region to the skin [25], and reduction in local heat-induced nitric oxide (NO) production and/or responsiveness to NO [5].

Increase in forearm blood flow after exercise training suggests that elevated shear stress contributes to endothelial adaptation and cardiovascular protective effects of exercise [15,20,32]. Reactive hyperaemia (RH) to an ischemic block is thought to reflect endothelial function and to be mediated mainly by the release of NO and prostacyclin from endothelium [1,6,8,22,26]. However, mechanisms that are involved in this response remain controversial as some authors suggest that NO does not play a role in RH in skin [36,37]. Previous clinical studies investigated RH by ultrasonography of brachial artery or venous occlusion plethysmogra-
phy in the forearm [8, 17, 34]. Adaptive changes may be also present in cutaneous vasculature that has the advantage of being easily accessible for monitoring using Laser-Doppler (LD) technique [18, 23].

However, effect of exercise has been poorly evaluated with this technique [24]. The purpose of the present study was to determine whether physical conditioning is associated with enhanced endothelium-dependent vasodilation in cutaneous vasculature of healthy men when compared to sedentary controls. LD and RH were used to evaluate endothelial function in cutaneous microcirculation [2, 3]. Local heating to 42°C was used as an alternate method to induce vasodilation in our subjects.

Methods

This study included 9 male windsurfers and a control group of 8 sedentary healthy males. Athletes were training at least 3–4 times per week, each lasting at least 3 h, and were participating in international competitions. They had sailed for 11.7 ± 1.6 years and undertook on an average 10.4 ± 0.7 h of sailing per week. During the month preceding LD measurements, training encompassed 13.7 ± 2.2 h per week.

After written informed consent all subjects undertook a screening program that comprised a medical and training history, baseline blood pressure, anthropometric characteristics (height, weight, maximal forearm girth, and wrist girth), and lipid profile. All subjects were non-smokers, normotensive, and were not on any medication at the time of the study.

All procedures were performed in a quiet temperature-controlled-room at 24.0 ± 0.3°C. Subjects were asked to empty their bladder before measurements and to remain in the supine position. LD measurements started at least after 20 min of rest. LD probe (Periflux PF 4001 – 2, Perimed, Uppsala, Sweden) was always placed on the ventral site of dominant forearm, 5 cm below the elbow bend in order to avoid site to site variations [18, 19, 29]. Cutaneous blood flow was measured from a small volume of skin (−1 mm³) using laser beam at 650 nm wavelengths. To obtain rapid-inflation and deflation during RH, an arm cuff was connected to an automatic device (Hokanson, Model E20, Bellevue, WA, USA) and placed around the upper arm. Blood pressure was measured before and after the protocol. Heart rate was monitored (Polar S610 Finland). Baseline measurements were performed for at least 3–4 min duration.

After baseline measurements, the arm cuff was inflated to 200 mmHg for 5 min and RH was recorded after cuff deflation (endothelium-dependent response).

The recovery period was defined until blood flow returned to baseline values. Thereafter, the measure site was warmed to 42°C for 5 min in order to reach a stable plateau of blood flow value using a thermostatically controlled heater of 32 mm diameter (PF 450 Perimed, Uppsala, Sweden), which could be fixed on the same cutaneous site. The LD probe was placed in the middle of this area.

All traces (baseline, RH, and local heating) were recorded using Perisoft V5.10 (Perimed Software). The signal intensity depends on velocity and concentration of moving blood cells in the examined site and gives a semi quantitative assessment of microvascular blood concentration. This latter parameter was chosen according to a previous report suggesting that during the initial phase of RH, the high value of LDF signal is produced by an increasing number of erythrocytes in the vasodilated cross-section than an increase in moving blood cells velocities [16].

Baseline blood flow is the mean value of all readings over a stable 2-min period. Area under the hyperaemic curve is the area from release of ischemia till recovery. Peak flow was defined as the highest recorded blood flow following cuff deflation (RH peak) or following local heating (T peak). Time to peak (s) is defined as the time to reach RH peak after cuff deflation. TR (s) is the time between RH peak and the fall to 50% of this value. T (s) is the time between deflation and return to baseline value.

For data analysis, CVC was indexed as cutaneous blood flow (in arbitrary concentration units) divided by mean arterial blood pressure (in mmHg) and normalized to baseline values. The arbitrary units correspond to the voltage of the analogue signal of the LD flowmeter with the zero value corresponding to the blood flow value during arterial occlusion. Data were digitised and stored on a computer for analysis. Results are expressed as mean ± standard error (SE). Values obtained in the two groups were compared using t-test. The difference was considered statistically significant if p < 0.05.

Results

Subjects characteristics are summarized in Table 1. Maximal forearm girth was significantly higher in the windsurfers, which was not surprising as it reflects windsurfing effect on forearm musculature and demonstrates the intensity of their physical training. No statistical differences were found in wrist girth, height, and weight. Heart rate was significantly lower in windsurfers. Diastolic blood pressure was similar in the two groups, while systolic and mean blood pressures were significantly higher in the control group. Lipid profile was similar in both groups.

Table 1: Subjects characteristics

<table>
<thead>
<tr>
<th></th>
<th>Windsurfer n = 9</th>
<th>Control n = 8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>24.5 ± 1.6</td>
<td>22.9 ± 0.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179.9 ± 2.2</td>
<td>181.3 ± 2.0</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.7 ± 2.7</td>
<td>72.9 ± 1.3</td>
</tr>
<tr>
<td>Maximal forearm girth (cm)</td>
<td>28.8 ± 0.3</td>
<td>26.8 ± 0.5***</td>
</tr>
<tr>
<td>Wrist girth (cm)</td>
<td>16.9 ± 0.3</td>
<td>16.6 ± 0.3</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>120.6 ± 2.7</td>
<td>130.0 ± 2.8*</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>67.5 ± 2.5</td>
<td>75.0 ± 3.3</td>
</tr>
<tr>
<td>Mean</td>
<td>85.0 ± 2.2</td>
<td>93.2 ± 2.6*</td>
</tr>
<tr>
<td>Resting heart rate (beats/min)</td>
<td>53.8 ± 2.3</td>
<td>67.0 ± 3.6**</td>
</tr>
</tbody>
</table>

Values are means ± SE. *p < 0.05, **p < 0.01
ализed RH CVC peak in windsurfers was significantly higher than in controls (1775.4 ± 286.9 and 826.4 ± 121.7% baseline CVC; p = 0.01). Normalized peak CVC value in response to local heating (42°C) was not significantly different between both groups (2359.4 ± 346.1 and 1467.7 ± 282.6% baseline CVC, NS) (Fig. 1).

Furthermore when we analysed RH traces, we found a significantly higher area under the hyperaemic curve in windsurfers than in controls (3675.1 ± 307.4 and 1418.1 ± 256.0 Concentration Units × s respectively; p = 0.00005). T_{recovery} was significantly higher in windsurfers (144.0 ± 14.2 s) than in controls (96.7 ± 15.6 s; p = 0.04). T_{1/2} was not different in windsurfers (20.6 ± 3.3 s) when compared to controls (29.5 ± 10.5 s; NS). Time to peak response was not statistically different in both groups (23.5 ± 3.9 s in windsurfers and 19.8 ± 2.3 s in controls; NS) (Fig. 2).

**Discussion**

The main result of this study is the beneficial effect of training on endothelial function of cutaneous microcirculation as RH peak measured by LD in windsurfers forearm skin is significantly higher when compared to sedentary controls. The observed difference in RH at rest would indicate a true difference in the capacity of endothelial cells to induce vasodilation, probably as a...
result of repetitive increases in blood flow during training ses-
sessions. The other result of the study is the lack of difference in T
peak between both groups. Response to local heating has been
demonstrated to be mediated by at least two independent
mechanisms: a fast-responding vasodilator system mediated by
axon reflexes and a more slowly responding vasodilator system
that relies on local production of NO [27]. This may explain the
lack of significant effect of training on the response to this pro-
Vative test.

These results are in agreement with other studies that showed
beneficial effect of exercise on endothelium-dependent vasodila-
tion [11,14,15,28,31]. Most of these studies used plethysmogra-
phy or ultrasound techniques concomitantly to RH. Only one
study evaluated this effect on human skin vasculature using ace-
tyline iontophoresis and showed enhanced endothelium-de-
pendent vasodilation in athletes (long distance runners) [24].
The main interest of our study is that our technique is simple
and does not require any drug application. Although this proto-
col studied endothelium-dependent vasodilation differently (RH
instead of acetylcholine iontophoresis), and a different trained
population (windsurfers), our results are similar. Therefore ben-
eficial effect on forearm skin is probably not only a regional effect
related to the fact that forearms are mainly solicited in windsurf-
ners. Exercise is known to exert a generalized effect on the vascu-
lature by increasing NO activity in other vessels than those that
perfuse the actively working muscle [12,33]. In a study evaluat-
ing fitness-induced improvement of endothelial function by ple-
thysemography and RH, O’Sullivan showed that the magnitude of
differences between fit and sedentary individuals was not relat-
ed to the amount of arm activity [31].

Postischemic RH results mainly from the release of endothelium-
derived relaxing factors and from the stimulation of adenin-tri-
phosphate-sensitive potassium channels [1,6,8,22,26]. NO, the
most important endothelium-derived relaxing factor, contribu-
tes to all phases of RH including peak flow in normal human
vasculature [6,17,26,34]. In contrast, some authors found that
dermal vasodilation in response to acetylcholine is mediated
predominantly by prostanooids rather than NO [3,30]. Further-
more, it was suggested that NO does not directly mediate RH in
the skin by other authors [36,37]. Prostaglandins appeared also
to be important factors during RH in studies using plethysmogra-
phy while NO contribution seemed to be modest [8]. Although
the exact mechanisms involved in RH are not completely under-
stood, this provocative test associated to LD is considered a suit-
able method to assess endothelial function in the skin microcir-
culation. The aim of our study was not to evaluate each factor
contribution in this response.

We also evaluated the effect of exercise on other parameters.
Most of them are related to response duration. Time to peak dur-
ring RH was not significantly different between windsurfers and
controls. This result is similar to that found in rock climbers us-
ing plethysmography [9]. The second parameter, TR_{25}, was not
different between both groups. Our values are similar to those
reported by O’Sullivan [31]. Therefore these two parameters do
not seem to be interesting to follow exercise effect on endothe-
lium-dependent vasodilation.

However, two other parameters were found to be significantly
different between our groups: T_{recovery}, and the area under the hy-
peraemic curve. These parameters are significantly higher in
windsurfers particularly the area under the hyperaemic curve.
This parameter was previously showed to be reduced by infusion
of L-NMMA (a specific NO synthase inhibitor) [17,26,34]. How-
ever, unlike our technique which evaluates skin microcircula-
tion, these studies used plethysmography which evaluates fore-
arm blood flow including skin and muscle. As NO synthase was
suggested not to play a major role in RH in skin, comparisons
should be cautious. Are these two parameters (T_{recovery} and
the area under the hyperaemic curve) more specific of NO produc-
during RH? Further studies are necessary to prove this as-
sumption.

Study limitations

We included a small number of subjects. However, our 2 groups
were homogenous with regard to their arterial risk factors and
the relevant difference between them was exercise.

Resting heart rate and systolic blood pressure were significantly
lower in windsurfers than in controls which may have influenced
the results. However, although these two parameters were lower
in the windsurfers, RH peak was significantly higher in this
group which emphasizes our results.

Although not the optimal standardization method, peak (RH and
local heating) CVC values were normalized to baseline values.
Standardization would have been best accomplished by infusion
of sodium nitroprusside or hyperthermia to cause maximal vas-
dilation. As we preferred the use of a simple protocol, maximal
dilation was not available in this study.

In conclusion, physical conditioning results in enhanced endo-
thelium-dependent vasodilation in the cutaneous microcircu-
lation, as demonstrated by the higher RH peak in windsurfers than
in controls. On the contrary, it does not induce any significant ef-
efct on response to local heating (42°C). LD and RH seem to be an
interesting tool to follow training effect on endothelial function in
skin microcirculation.

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