The Effects of Cold Stress Test on Vasomotor Tonus in Normal Controls

H. Bozdemir, Y. Sarica, M. Demirkiran

Summary

Plethysmographic recordings are used to evaluate changes in peripheral vascular tonus. Twenty-six volunteers (15 men, 11 women) with a mean age 31 years were included in this study. Plethysmographic recordings were done both at baseline and +4°C temperatures bilaterally. Significant reductions in systemic blood flow were noted with different maneuvers. Reactive hyperemia occurring after ischemia was local, however local reduction in blood flow after cold test was systemic. As the local temperature increased, the blood flow parameters normalized, too. In normal controls these values normalized in 20 minutes. The changes in vasomotor tonus due to cold test demonstrated a parallel pattern.

Key words : Plethysmography, Cold stress test, Vasomotor tonus. Post-ischemic reactive hyperemia

Introduction

Peripheral vascular circulation can be evaluated with various noninvasive methods including plethysmography. Pulse plethysmography can be used in the diagnosis of reflex sympathetic dystrophy, and cosalgia due to trauma, and peripheral neuropathies.1-6 The aim of this study was to evaluate baseline and cold stress test values with plethysmographic recordings in normal controls and to determine the changes in vasomotor tonus due to cold.

Material and Methods

Twenty-six (15 men, 11 women) volunteers with a mean age of 31 years were included in this study. None of them had a history of a neurologic or a systemic disease, sweating disorder, hypertension, cold intolerance, smoking, or alcohol consumption. They were asked not to take any tranquilizers at least a week before the study.

Plethysmographic recording of pulse volume was performed on index finger, bilateral in some, and the skin temperature was recorded at the 4th finger on the right hand. Vasomotor changes were recorded at baseline, breathholding, valsalva maneuver, hand grip, mental activity, 5-minute ischemia tests and cold test. Baseline recordings and cold stress test were done in all cases while breathholding, valsalva and 5-minute ischemia tests were performed in 22, hand grip in 21, and mental activity in 20.

1. Plethysmographic recordings : A cuff with a pressure system was placed on the 2nd finger of both hands. Each cuff was connected to a different pressure transducer on Hewlett Packard physiography, recording the changes in pulse volume simultaneously. The patient was asked to close his eyes during the recordings. Baseline recordings were performed for at least one minute. At first, the patient was asked to take a deep breath and hold it for 15 seconds (inspiration gasp). Right after this maneuver, pulse volume recordings were done for 1.5-2 minutes of normal breathing. Without any recess, he was asked to perform a valsalva maneuver for 15 seconds then breathe normally for 1.5-2 minutes and recording was carried out. Then he was asked to make a fist on the left hand for 1 minute and recording was performed (hand grip test). Later recordings were performed for 45-60 seconds while the patient concentrated on a task (mental activity).

For 5-minute ischemia test, the index finger of the right hand was tied firmly and the blood flow was prevented for 5 minutes. After loosening, pulse volume recording was done for 5 minutes to evaluate reactive hyperemia due to ischemia. Recordings were bilateral in 7 controls.

For cold stress test, right hand was immersed in +4°C cold water for 4 minutes. After the hand was taken out, pulse volume was recorded at the 1st, 5th, 10th, 20th, 30th, and 60th minutes bilaterally in all.

2. Recording of skin temperature : The changes in the skin temperature were recorded on graphic papers with a thermic censor placed on the 4th finger of right hand. Temperature changes on the finger skin were recorded with a sensitivity of +10°C.

Results

Baseline pulse volume changes : Spontaneous amplitude changes were noted as the pulse volume recordings were carried out at baseline. The
plethysmographic changes were different from case to case, with very variable pulse volume in some but stable pulse volume in others. At breathholding, 55.2 % reduction in pulse volume was observed. There was a rebound after this test with pulse volume reaching up to 112.4 % of the baseline at the 60th second. Valsalva maneuver reduced pulse volume by 40.4 %; there was a rebound value of 105.5 % increase at 60th second after the test. On hand grip test, 54.8 % reduction in the pulse volume was noted at 5th and 10th seconds. However at the 50th second, an increase of 74.7 % was detected. After the test, a decrease to 68.7 % was seen at the 15th second and a rebound value of 113 % increase at the 60th second. Mental activity caused a reduction to 34.8 % in pulse volume, normalizing at 60th second. No significant difference in the pulse volume change was detected between the 2 hands (Table 1).

In conclusion, plethysmographic recordings revealed that i) Breathholding, valsalva, hand grip and mental activity caused a decrease in pulse volume, most prominent with mental activity. There was a rebound effect after the tests, pointing out an increase in blood flow due to vasodilation occurring after vasoconstriction. ii) No reactive volume increase was detected during breathholding, valsalva and mental activity tests. However, at hand grip test, there was a reactive volume increase during the test which did not reach baseline values. iii) The rebound occurring after the tests showed that these tests caused significant vasomotor tonus. iv) These local recordings point out a systemic reaction, because simultaneously similar results were recorded on both index fingers.

Post-ischemic reactive hyperemia : In 22 controls, post-ischemic reactive hyperemia was evaluated, after blocking blood flow for 5 minutes. After the test, blood flow increased by 222 % in the 1st minute and then this response decreased slowly to 144% by the end of 5th minute. This could be because, ischemic tissue metabolites cause a reflex increase in the local blood flow which then possibly decreases as the metabolites decrease. In 7 controls, simultaneous recordings were carried out on bilateral index fingers. Pulse volume increased by 168 % on the right and by 111% on the left after the ischemia. This shows that local ischemia causes local vasodilation.

The effect of cold stress test on pulse volume : Cold stress test caused a prominent decrease in pulse volume in controls. A rebound response exceeding the baseline value was detected at 20th minute. Our results show that cold stress test causes first an increase in vasomotor tonus during the test, and then a decrease after the test with a rebound response exceeding the baseline values. Moreover, pulse volume changes correlated with the local temperature. These results support that local temperature is an important factor in vasomotor tonus. The effect of cold was not local in the study. Vasomotor tonus increased, thus decreasing pulse volume in the left hand as well, while cold test was applied to right hand. But the increase in vasomotor tonus in left hand was not as prominent as it was in right and no rebound effect was observed in left. These results suggest that the effect of locally applied cold is not local; cold causes systemic vasomotor changes.

Discussion

Sympathetic stimuli can cause changes in the peripheral vasomotor tonus. Exercise, deep inspiration and expiration, breathholding, and ischemia are some of such manipulations. Some metabolites occurring during vasoconstriction are thought to cause reactive vasodilation. In this study we have shown that these maneuvers caused vasoconstriction which was followed by a rebound reaching to a maximum in 60 seconds in normal controls. It is already known that ischemia can decrease the peripheral vascular resistance. Our results with 200% rebound detected at post-ischemia test suggested that ischemia was the most prominent vasodilator.

<table>
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<tr>
<th>Table I</th>
<th>Pulse Volume Changes During Vasomotor Tests</th>
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<tr>
<td>Changes in mean values in relation to the baseline</td>
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<tr>
<td><strong>Breathholding</strong></td>
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<tr>
<td>During Test</td>
<td>55.2 % decrease</td>
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<tr>
<td>Rebound 60th sec</td>
<td>112.4 % increase</td>
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<tr>
<td><strong>Valsalva</strong></td>
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<tr>
<td>During Test</td>
<td>40.4 % decrease</td>
</tr>
<tr>
<td>Rebound 60th sec</td>
<td>105.5 % increase</td>
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<td><strong>Hand grip</strong></td>
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<td>During Test 5th sec</td>
<td>54.8 % decrease</td>
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<td>During Test 50th sec</td>
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<td>Rebound 15th sec</td>
<td>68.7 % decrease</td>
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<td><strong>Mental activity</strong></td>
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<tr>
<td>During Test</td>
<td>34.8 % decrease</td>
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<td>Rebound 60th sec</td>
<td>baseline value</td>
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After the cold test vasomotor tonus incerased and local blood flow was blocked. This effect was detected in both the cold-applied hand and the other hand simultaneously.\textsuperscript{14-16} Our observations suggest that the effect of temperature is not local but systemic and this occurs independent of the skin temperature. However, in another experiment it was demonstrated that blood flow was unaffected by nerve block, but mainly by the skin temperature; therefore the authors concluded that even in the absence of nervous signals, local skin temperature affected blood flow.\textsuperscript{7,16-18} Moreover, in several studies, it has been shown that vasomotor tonus was affected by neuromediators like serotonin and epinephrine.\textsuperscript{19} The correlation with these mediators were not included in our study Two questions need an answer in this situation: i) is the effect totally local?, ii) does cold, a powerful sympathetic stimulus, reach peripheral veins through sympathetic nerves and cause vasoconstriction? In the cold-applied area reflex vasoconstriction is detected and this is known to be caused by sympathetic discharges due to cold. We think that cold effect is more than a local change, it causes a diffuse increase in the sympathetic tonus. It can be speculated that the effect of cold is not only due to changes in skin temperature, but also probably to the increase in vasomotor tonus, leading to metabolic changes. Within the limits of our study, we are unable to tell whether this effect is solely by neurogenic or humoral mechanisms.

References