Systemic Arterial Blood Pressure during Exercise in Patients with Atherosclerosis Obliterans of the Lower Limbs

By Einar Lorentsen, M.D.

SUMMARY
Systemic intraarterial blood pressure and heart rate have been measured during exercise on a foot ergometer in 13 patients suffering from unilateral intermittent claudication. Each limb was exercised separately for 5 min. Both systolic and diastolic blood pressure increased to significantly higher levels during exercise with the diseased limbs than during exercise with the nondiseased limbs. No significant difference was found in heart rate. After cessation of exercise the blood pressure and heart rate dropped abruptly. For the first 2 min of recovery the systolic blood pressure in the diseased limbs stayed higher than the pressure measured at rest immediately before exercise. Ten patients were also exercised for 2 or 3 min with arrested circulation. During this period no significant differences between the diseased and nondiseased limbs were found in relation to blood pressure or heart rate.

Additional Indexing Words:
Intermittent claudication
Arrested circulation

During rhythmic exercise with complete arrested circulation, and during sustained isometric contractions with or without arrested circulation, an unexpectedly large increase of systemic arterial blood pressure was found.1-5 Exercising muscles receiving an inadequate blood supply seem to be capable of invoking powerful central circulatory reflexes which result in a considerably higher perfusion pressure. These reflexes may also be assumed to operate in circulatory insufficiency during walking in patients with atherosclerosis obliterans of the lower limbs. The objectives of the present study were (1) to compare the changes in systemic arterial blood pressure and pulse rate during exercise with each limb separately in patients with predominantly unilateral intermittent claudication, and (2) to compare the changes in systemic arterial blood pressure and pulse rate during exercise with arrested circulation in the diseased and the nondiseased limbs.

Methods
Observations were made on 12 male and one female patient aged 46-69 years, suffering from predominantly unilateral intermittent claudication due to atherosclerosis obliterans. Their claudication distance varied between 30 and 200 m. Two patients also experienced a vague inconstant distress in the opposite leg during walking. The diagnosis was based on thorough clinical examination including oscillography.

Ten patients had unilateral occlusion of the femoral artery, one patient had unilateral subtotal stenosis of the common femoral artery, and two patients had unilateral occlusion of the common iliac artery. The oscillographic readings at the ankle of the limbs in which claudicatory pains occurred (diseased limbs) had a maximum of 1.5
mm except in one patient with a reading of 3 mm. The oscillographic readings at the ankle of the opposite limbs (nondiseased limbs) was 2 mm in one patient and 3 mm or more in the other patients. In three patients a murmur was heard at the groin or on the thigh of the nondiseased limb.

Arteriography was performed in nine patients, confirming the clinical diagnosis in each patient. Severe stenosis of the iliac or femoral arteries of the nondiseased limbs was not found except in one patient in whom the popliteal pulse was found to be slightly reduced. This patient experienced a vague and inconstant distress in the calf of the nondiseased limb.

No patient had symptoms or signs of heart failure. The ECG at rest was normal in all patients, except in one with a right bundle-branch block. None used drugs.

Systemic arterial blood pressure was measured through a polyethylene catheter, bore 0.8 cm, length 58 cm, introduced percutaneously at the groin. The tip of the catheter was placed in the distal part of the aorta in eight patients and in the iliac artery in five patients. The location of the catheter tip was determined by X-ray transillumination. For pressure measurements a Statham P23Gb pressure transducer and an EME DC preamplifier were used. The pressure curves were recorded on an ultraviolet recorder (Ultralette 5656 ABEM). Zero reference level was 10 cm above the back of the patient. Heart rate was counted from the pressure recordings. Oxygen uptake was determined by the Douglas-bag method.

The exercise was performed on a foot ergometer by plantar flexion of the foot against a graded resistance provided by means of a spring attached to a lever. The height of the heel support was altered so that rotating movements could be performed around an axis which corresponded to the anatomic axis of the ankle joint. The work load during each contraction was calculated to be 0.325 kg. The exercise was timed with a metronome at a rate of 40 contractions/min (i.e., a work load of 13.0 kg/min on each limb).

The examinations were carried out with the patients in supine position. After insertion of the intraarterial catheter, measurements of blood pressure and pulse rate were made during exercise with both limbs for 5 min. This exercise was regarded as a training period for the patient to experience the ergometer work and to become accustomed to the laboratory surroundings. The values obtained during this period are not reported. Thereafter exercise was performed separately with each lower limb for 5 min, starting alternately with diseased and nondiseased limb.

Circulatory arrest was produced by inflating a sphygmomanometer cuff around the distal part of the thigh to a pressure of 100 mm Hg above the systemic blood pressure at rest, but not to exceed 250 mm Hg. In 10 patients the circulation was arrested from the start of the exercise period. Six patients were able to exercise for 3 min with arrested circulation, while four patients could not exercise more than 2 min. At the end of the period the circulatory arrest was suddenly released.

The blood pressure was recorded continuously for 1–2 min immediately preceding each period of exercise, during exercise, and during the first minute after cessation. Thereafter, one recording per minute was made for 6 min during the subsequent recovery period. Blood pressure and heart rate obtained in the resting period immediately before each exercise period were used as reference values in calculating the changes of blood pressure and heart rate during exercise and recovery periods.

Oxygen uptake was measured in 11 patients at rest for 3 min, and during the last 2 min of the exercise period with free circulation.

The significance of the differences in the changes of blood pressure and pulse rate obtained was analysed by use of Student t-test.

The study was performed with the patients' consent as part of a routine catheterization or arteriography.

Results

Blood pressure and heart rate at rest measured immediately before each exercise period did not change significantly during the examination period.

Exercise with Free Circulation

Subjective Responses

All patients were able to carry out 5 min of exercise with each limb separately. In three patients no pain occurred during this exercise, while in the other patients discomfort or pain of varying intensity occurred in the diseased limbs after 1½–3 min. The discomfort increased during the last part of the exercise period. In five patients considerable effort was required to carry out the exercise.

Systolic Blood Pressure

Systolic blood pressure was found to be significantly higher during exercise with the
Table 1

Differences in Arterial Blood Pressure and Heart Rate Differences in Arterial Blood Pressure and Heart Rate

<table>
<thead>
<tr>
<th></th>
<th>Systolic</th>
<th>Diastolic</th>
<th>Heart rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>se</td>
<td>P</td>
</tr>
<tr>
<td>Exercise:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 min</td>
<td>6.77</td>
<td>2.80</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>2 min</td>
<td>8.92</td>
<td>3.81</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>3 min</td>
<td>14.77</td>
<td>4.97</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>4 min</td>
<td>16.08</td>
<td>3.63</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>5 min</td>
<td>18.08</td>
<td>4.27</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Recovery:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1/2 min</td>
<td>8.85</td>
<td>3.03</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>1 min</td>
<td>12.00</td>
<td>3.48</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>2 min</td>
<td>10.38</td>
<td>3.81</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>7 min</td>
<td>3.85</td>
<td>3.65</td>
<td>&gt;0.1</td>
</tr>
</tbody>
</table>

*Tests done on 13 limbs with and 13 limbs without intermittent claudication.

The greatest increase of systolic blood pressure occurred among patients who experienced the greatest discomfort during the exercise. No association was found between the increase of systolic blood pressure and the age of the patients, the duration of symptoms, the systolic blood pressure at rest, or the increase of heart rate during exercise.

Immediately after cessation of exercise the systolic blood pressure dropped abruptly (fig. 1). The values after exercise with the diseased limbs remained significantly higher than the values with the nondiseased limbs during the first minutes of recovery (table 1). After exercise with the nondiseased limbs the systolic blood pressure reached the preexercise level in a few seconds, while the postexercise values of the diseased limbs remained higher than the preexercise values for the first 2 min.

**Diastolic Blood Pressure**

The diastolic blood pressure increased to significantly higher levels during exercise with the diseased limbs than during exercise with the nondiseased limbs (table 1). The average increase of diastolic blood pressure after 5 min of exercise was 19 mm Hg (range 6-35) and 11 mm Hg (range 2-23), respectively (fig. 1).

The diastolic blood pressure rapidly returned to the preexercise level after cessation of exercise, and no significant difference was found after 1/2 min of recovery.

**Heart Rate**

The heart rate increased an average of 16 beats/min (range 6-40) during 5 min of exercise with the diseased limbs, and 12 beats/min (range 4-24) during exercise with the nondiseased limbs. The difference was not found to be statistically significant (table 1). A steady-state level was established after 3 min of exercise. No association was found between the increase of heart rate and the intensity of discomfort experienced during exercise.

The heart rate dropped abruptly when exercise stopped and reached the preexercise level in 5 min (fig. 1).
Systolic Blood Pressure

No significant difference was found between the diseased and the nondiseased limbs in the increase of systolic blood pressure during exercise (table 2). The average increase after 2 min of exercise with the diseased limbs was 42 mm Hg (range 16-66) and 37 mm Hg (range 23-60) after exercise with the nondiseased limbs (fig. 2). The systolic blood pressure for both the diseased and nondiseased limbs after 2 min of exercise with arrested circulation was significantly higher than the pressure after 2 min of exercise with free circulation ($P < 0.02$).

Six patients exercised for 3 min with arrested circulation. The systolic pressure steadily increased. After exercise for 3 min with the diseased limbs the average increase was 49 mm Hg, and 40 mm Hg after exercise with the nondiseased limbs. The highest systolic blood pressure obtained during the study was 302 mm Hg. This patient had a systolic blood pressure of 214 mm Hg at rest.

After cessation of exercise with the nondiseased limbs the systolic blood pressure reached the preexercise level in a few seconds. After exercise with the diseased limbs the values stayed higher for the first minute. No significant difference was found after 2 min of recovery (table 2).

Diastolic Blood Pressure

No significant differences were found during exercise with the diseased and the nondiseased limbs (table 2). The average increase after 2 min of exercise was 23 mm Hg (range 7-39) and 21 mm Hg (range 15-33), respectively (fig. 2). The diastolic blood pressure after 2 min of exercise with arrested circulation was found to be significantly higher than after 2 min of exercise with free circulation ($P < 0.05$).

After cessation of exercise the diastolic blood pressure dropped abruptly and was not found to be significantly different from the preexercise values after ½ min of recovery.

**Exercise with Arrested Circulation**

**Subjective Responses**

Exercise with arrested circulation caused fatigue and/or pain after ½-2 min in diseased and nondiseased limbs. In some of the patients the discomfort lasted for several minutes after cessation of the exercise.
TABLE 2

Differences in Arterial Blood Pressure and Heart Rate Response during and after Ischemic Exercise on a Foot Ergometer*

<table>
<thead>
<tr>
<th>Blood pressure (mm Hg)</th>
<th>Heart rate (beats/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>Exercise:</td>
<td></td>
</tr>
<tr>
<td>1 min</td>
<td>-1.10</td>
</tr>
<tr>
<td>2 min</td>
<td>5.50</td>
</tr>
<tr>
<td>Recovery:</td>
<td></td>
</tr>
<tr>
<td>½ min</td>
<td>10.20</td>
</tr>
<tr>
<td>1 min</td>
<td>9.50</td>
</tr>
<tr>
<td>2 min</td>
<td>8.70</td>
</tr>
<tr>
<td>7 min</td>
<td>5.50</td>
</tr>
</tbody>
</table>

*Tests done on 10 limbs with and 10 limbs without intermittent claudication.

Heart Rate

No significant differences were found in heart rate during exercise with the diseased and the nondiseased limbs (table 2). The average increase after 2 min of exercise with arrested circulation was 16 beats/min (range 4–36) and 18 beats/min (range 10–28), respectively (fig. 2).

After cessation of exercise the heart rate dropped abruptly. No significant difference in heart rate was found between the diseased and the nondiseased limbs (table 2).

Oxygen Consumption

The average oxygen consumption at rest in nine patients was 227 ml/min and during exercise, 384 ml/min.

Comments

Rhythmic muscular work results in a moderate increase in systolic blood pressure and little or no increase in diastolic blood pressure. This pressor response is found to increase with age. The same rhythmic work performed with arrested circulation or sustained contractions gives rise to far higher blood pressure responses.

Both rhythmic ischemic work and sustained isometric contractions involve some discomfort and require a high degree of cooperation from the subjects studied. The circulatory events could be provoked by pain and/or psychic factors. This suggestion is difficult to disprove. However, in the present study discomfort was chiefly experienced during the last part of the exercise period, whereas the pressure rise was greatest during the first half of the exercise period.

Thorough investigations in man have led to the suggestion that active contraction of muscle cells under ischemic conditions causes stimulation of local receptors invoking generalized circulatory pressor reflexes. These reflexes are thought to insure blood flow through the muscles. The magnitude of the circulatory response is found to be directly related to the degree of muscle ischemia during the voluntary contraction. The reflex nature of the pressor response has recently been proved in experiments performed on anesthetized or decerebrated cats.

The fact that a difference in pressure response between the diseased and nondiseased limbs was obtained during exercise with free circulation and not during exercise with arrested circulation supports the suggestion that ischemia plays a fundamental role in the pressor response. The present observations are in agreement with the effect of vascular occlusion observed on the pressor response to sustained isometric muscular contraction.

The blood pressure also increased to a relatively high level during exercise with free circulation of the nondiseased limbs. Exercise on the ergometer used in this study includes an essential part of static muscle contraction performed by a relatively small muscle group. This fact probably explains the high blood pressure increase.
No reports have been published on the comparison of blood pressure increase during walking in patients with and without atherosclerosis obliterans. Intermittent claudication during walking is associated with both discomfort and pain. Thus, much the same conditions prevail in walking as in the present experiments. Therefore, this study may support the suggestion that walking exercise in patients with intermittent claudication causes a higher blood pressure increase than in normal subjects.

In recent years, therapy to increase blood pressure has been advocated in the treatment of severe peripheral arterial insufficiency. The present study may indicate that a sufficient increase in blood pressure is produced by the patient during exercise.

References


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