RESTING AND POSTEXERCISE DIGITAL PLETHYSMOGRAM AND ELECTROCARDIOGRAM IN PATIENTS WITH ISCHEMIC HEART DISEASE

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In the evaluation of the patients with ischemic heart disease, the post-exercise electrocardiogram is one of the most important and widely used procedures and has an established value in the clinical practice. However, it shows only electrical activity of the heart and the informations it gives is therefore, limited. Recently, it has been demonstrated by several investigators that most patients with ischemic heart disease respond to exercise with an abnormal increase in left ventricular end-diastolic pressure, and this has been interpreted as a manifestation of left ventricular failure. It is important, therefore, to have hemodynamic information in addition to electrocardiogram in the assessment of patients with ischemic heart disease. But cardiac catheterization is time-consuming and expensive and incurs risk and discomfort for the patient. Digital plethysmogram is simple and noninvasive to record and has been reported to reflect cardiac output and to be useful in the evaluation of patients with latent heart failure.

The present study was designed to determine (1) what are the digital plethysmographic responses to exercise in patients with ischemic heart disease, (2) whether there are any correlations between the electrocardiographic changes and digital plethysmographic changes after exercise, (3) whether exercise-induced digital plethysmographic changes could be altered by the administration of a cardiac glycoside and finally, (4) whether exercise digital plethysmogram has any additional value in the evaluation of patients with ischemic heart disease.

METHODS

One hundred and nine patients who came to our hospital from May of 1971 to June of 1972, for the evaluation of ischemic heart disease because of chest pain, abnormal electrocardiograms, hyperlipemia, diabetes mellitus and hypertension were included in this study. Those with over heart failure, ST-depression of more than 0.5 mm in the electrocardiogram, congenital heart diseases, acquired valvular heart diseases, cardiomyopathies, neuro-circulatory asthenia, peripheral vascular diseases, and arrhythmias were excluded. No patients had received a cardiac glycoside or a beta-adrenergic blocking agent for at least a week prior to the study. All the patients had received electrocardiographic and digital plethysmographic examinations at least once previously and were familiar with the test procedures and therefore, were thought to be free from anxiety over the procedures. Routine twelve-lead electrocardiograms were taken at rest and immediately after a double Master's two step test with a 6 channel electrocardiograph. Master's two step test was designated as positive or negative, according to whether there was horizontal or downsloping ST depression of

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- Double master's two step test
- Digital plethysmogram
- Ischemic heart disease
- Heart failure
- Ouabain

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more than 0.5 mm below the resting level or not. Digital plethysmograms were obtained from the second finger of the left hand with the patient in the supine position, with simultaneous recording of lead II of the electrocardiogram and taken at rest after lying on bed for 5 minutes, and after double Master’s two step test immediately after 12 leads of the electrocardiogram were recorded, with a photoelectric tube plethysmograph (Model PG 102, Kowa), at the room temperature of 21°C–25°C. Pulse volume was measured from the level of the onset of pulse wave to its highest peak, averaged over 5 consecutive waves and expressed as mV/V. Minute pulse volume was calculated as the product of heart rate and pulse volume and expressed as mV/V/min.

Patients were divided into three groups (Table I): Group I, negative Master’s test (47 patients with a mean age of 55.6±1.7), Group II, positive Master’s test (32 patients with a mean age of 56.1±1.8), and Group III, old myocardial infarction (30 patients including 23 patients with negative Master’s test and 7 patients with positive Master’s test, with a mean age of 59.1±1.4).

Furthermore, of the above patients, fifteen subjects whose pulse volume decreased more than 1.0 mV/V after exercise (Group A) and fifteen subjects whose pulse volume increased more than 1.0 mV/V after exercise (Group B), received 0.075 mg of ouabain intravenously after Master’s test and 40 minutes later, repeated the same test, electrocardiograms and digital ple-
Postexercise Digital Plethysmogram of Ischemic Heart Disease

![ECG and DPG graphs at rest and after exercise for two groups: one with Master's Test negative and one with positive.](image)

Fig. 1. Representative digital plethysmograms (DPG) from patients in Group I (Master's test negative, upper) and from those in Group II (Master's test positive, lower) at rest and after exercise. Pulse volume increased in the patient from Group I (Y.I.) after exercise, while it decreased in the patient from Group II (K.T.) after exercise. Though not shown in the figure, ST-depression of more than 0.5 mm occurred in the left-sided precordial leads, V₅ and V₆ in the electrocardiogram of K.T. after Master's test.

RESULTS

**Digital Plethysmographic Response to Exercise**

Table II shows a summary of digital plethysmographic data before and after exercise for all patients and Fig. 1 illustrates the representative cases from Group I and Group II.

Heart rate (Fig. 2 and 5) increased in all groups after exercise (P<0.05 in Group I and in Group II, P<0.001 in Group III), but rate of increase was greater in Group II and in Group III than in Group I and there was significant difference between Group I and Group III (P<0.05).

Pulse volume (Fig. 3 and 5) increased from 3.9±0.2 mV/V to 4.6±0.2 mV/V in Group I after exercise (P<0.05), but in Group II, it decreased from 4.4±0.3 mV/V to 3.9±0.3 mV/V after exercise, but not significantly (P>0.05). In Group III, there was no significant change (from 4.9±0.4 mV/V to 5.0±0.5 mV/V). When percentage change in pulse volume from rest to exercise was compared, there was significant difference between Group I and Group III (+25±5% versus −11±4%, P<0.005), between Group I and Group III (+25±5% versus +7±6%, P<0.005) and between Group II and Group III (−11±4% versus +7±6%, P<0.01).

Minute pulse volume (Fig. 4 and 5) in-
Fig. 2. Effect of double Master's two step test on heart rate in the three groups. Groups = same as in table I. The vertical lines represent ± SEM.

Fig. 3. Effect of double Master's two step test on minute pulse volume in the three groups. Groups = same as in table I. The vertical lines represent ± SEM.

Increased from 243.6±13.6 mV/V/min. to 329.2±18.8 mV/V/min. in Group I after exercise (P<0.001). In Group II and Group III, it also increased, but not significantly (P>0.05). Percentage increase in minute pulse volume from rest to exercise showed significant difference between Group I and Group II (40±6% versus 4±4%, P<0.005), between Group I and Group III (40±6% versus 25±7%, P<0.05) and between Group II and Group III (4±4% versus 25±7%, P<0.01).

When the ratio of pulse volume increase was plotted against that of heart rate increase, there was a difference of distribution between Group I and Group II and between Group I and Group III, but almost no difference between Group II and Group III. Group I tending to gather in the left upper portion, while Group II and Group III in the right lower portion (Fig. 6).

Pulse volume decreased in 5 of 47 patients.
Fig. 4. Effect of double Master's two step test on minute pulse volume in the three groups. Groups = same as in table I. The vertical lines represent ± SEM.

Fig. 5. Percentage changes from rest to exercise in heart rate, pulse volume and minute pulse volume in the three groups. Groups = same as in I. ± represents mean ± SEM.

Fig. 6. Percentage change in pulse volume after exercise plotted against that in heart rate after exercise in the three groups.
and Group III (P<0.001). There was no significant difference between Group II and Group III. In Group III, 13 of 23 patients with negative Master's test (56.5%) and 5 of 7 patients with positive Master's test (71.4%) showed decreased pulse volume after exercise (Fig. 8). Minute pulse volume decreased in 4 of 47 patients (8.5%) of Group I, in 14 of 32 patients (43.8%) of Group II and in 8 of 30 patients (26.7%) of Group III (Fig. 7) and there was highly significant difference between Group I and Group II (P<0.001) and between Group I and Group III (P<0.005), but there was no significant difference between Group II and Group III. In group III, 5 of 23 patients with negative Master's test (21.7%) and 3 of 7 patients with positive Master's test (42.9%) showed decreased minute pulse volume after exercise (Fig. 8).

**Effects of Ouabain**

Table III shows a summary of data before and after the administration of ouabain.

The administration of ouabain did not cause any significant changes in heart rate, pulse volume and minute pulse volume at rest in both Group A and Group B (Fig. 9). But after exercise...
### TABLE III  EFFECTS OF OUABAIN

<table>
<thead>
<tr>
<th>Group</th>
<th>Heart Rate (beats/min)</th>
<th>Pulse Volume (mV/V)</th>
<th>Minute Pulse Volume (mV/V/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>rest -</td>
<td>exercise</td>
<td>rest -</td>
</tr>
<tr>
<td><strong>Group A (15)</strong> before ouabain</td>
<td>66.4 ± 3.2</td>
<td>82.3 ± 2.9</td>
<td>4.9 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>320.4 ± 39.2</td>
<td>286.0 ± 31.6</td>
<td>28.0 ± 18.4</td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>after ouabain</td>
<td>64.2 ± 3.1</td>
<td>73.8 ± 2.8</td>
<td>4.4 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>280.0 ± 18.4</td>
<td>343.2 ± 30.8</td>
<td>29.2 ± 18.4</td>
</tr>
<tr>
<td><strong>Group B (15)</strong> before ouabain</td>
<td>64.5 ± 1.7</td>
<td>70.6 ± 2.9</td>
<td>4.4 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>278.8 ± 29.2</td>
<td>362.4 ± 28.4</td>
<td>329.8 ± 36.4</td>
</tr>
<tr>
<td>P</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>after ouabain</td>
<td>65.5 ± 2.2</td>
<td>69.6 ± 3.3</td>
<td>4.3 ± 0.7</td>
</tr>
</tbody>
</table>

Abbreviations:  
- **P**=P value;  
- **NS**=not significant at 0.05 level.  
- Values represent means ± standard errors of means.  
- Group A=patients whose pulse volume decreased after exercise; Group B=patients whose pulse volume increased after exercise (see text for further details).

![Graph](image)  
**Fig.9.** Effects of ouabain on heart rate, pulse volume and minute pulse volume both at rest and after exercise in Group A and in Group B. before = before the administration of ouabain; after = after the administration of ouabain. Groups = same as in table 3. The vertical lines represent ± SEM.

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Pulse Volume Increase

%  
100  
93.3%  
P<0.001  

33.3%  

Group A  
Group B  

Minute Pulse Volume Increase

%  
100  
80.0%  
P<0.005  

46.7%  

Group A  
Group B  

Fig.10. Percentage of patients who responded to ouabain with an increased pulse volume (left) and minute pulse volume (right) after exercise.

Groups = same as in table 3.

(93.3%) in Group A and in 5 of 15 patients (33.3%) in Group B (Fig. 10) and there was highly significant difference between the two groups (P < 0.001). Minute pulse volume increased after the administration of ouabain and after exercise in 12 of 15 patients (80%) in Group A and in 7 of 15 patients (46.7%) in Group B (Fig. 10) and there was also a significant difference between the two groups (P < 0.05).

**DISCUSSION**

Digital plethysmogram is a recording of volume changes of the arterioles at the finger tip and its pulse volume is said to reflect cardiac output in the absence of local circulatory disturbances or vasomotor changes. In this study, care was taken to control the emotional state, body position and room temperature which are known to influence the circulation in the fingertips and patients with neuro-circulatory asthenia and peripheral vascular diseases were excluded. Our present study shows that the majority of patients with negative Master's test (89.4%) responded to exercise with an increase in pulse volume and this finding is consistent with that of Zelis and his coworkers who showed that in the normal subjects, cutaneous blood flow declined at the onset of exercise but rose as activity continued, and is interpreted as to show that cutaneous vasodilation occurred in order to eliminate heat produced by muscular exercise. In contrast, the majority of patients with positive Master's test (75.0%) and those with old myocardial infarction (60.0%) responded to exercise with a decreased pulse volume. This finding is very interesting in the light of Zelis and his associates' data which showed that in the patients with heart failure, the cutaneous blood flow fell and remained depressed during the entire period of exercise and no postexercise hyperemia was seen, and is interpreted as to show that cutaneous vasoconstriction occurred despite the need to dissipate heat produced by muscular exercise.

It has been demonstrated that increased vasoconstriction is a characteristic of human heart failure and is an important compensatory mechanism by which arterial pressure is maintained in the face of a low cardiac output and is produced mainly by the increased sympathetic nervous activity. It is then not unreasonable to postulate that the majority of the patients with positive Master's test and those with old myocardial infarction suffered from transient mild heart failure after exercise and that there was an exaggerated response of the sympathetic nervous system to compensate for the inadequate increase in cardiac output and this excessive activity of the sympathetic nervous system overrode the need to eliminate heat generated by the metabolic processes of exercise and caused vasoconstriction.

Our data showing that the patients with positive Master's test and those with old myocardial infarction responded to exercise with more
increase in heart rate than those with negative Master's test are also consistent with this concept.

Very recently, Saltups et al. showed that left ventricular end-diastolic pressure was significantly higher in patients with positive Master's test than in those with negative Master's test on exercise, and that a decrease in stroke volume index occurred in the majority of patients with angina pectoris. The data of several other investigators also show that the majority of the patients with angina pectoris responded to exercise with an abnormal increase in left ventricular end-diastolic pressure and with a decrease or no increase in stroke volume, and this has been interpreted as a manifestation of reversible form of heart failure. Goldschlager et al. reported that the majority of patients with positive treadmill test (76%) were found to have hemodynamic dysfunction at catheterization after exercise.

The administration of ouabain to the patients whose pulse volume decreased after exercise, caused no significant changes in heart rate and pulse volume at rest, but after exercise, it decreased heart rate and increased pulse volume significantly. In contrast, in the patients whose pulse volume increased after exercise, the administration of ouabain decreased pulse volume both before and after exercise, but not significantly. This finding is also very interesting in the light of the data of Mason and his associates who showed that the administration of ouabain caused vasoconstriction in the forearm of normal subjects, while in the patients with congestive heart failure, it contrariwise produced vasodilation. They suggested that this vasodilation was an indirect effect of the drug, resulting from the improvement of the congestive heart failure state and postulated that this improvement diminished reflex vasoconstriction in the patients with heart failure.

From all the above discussions, it is reasonable to speculate that the majority of patients with positive Master's test and those with old myocardial infarction have mild reversible heart failure after exercise.

Our study also shows that the incidence of pulse volume decrease after exercise is greater in positive Master's test group that in old myocardial infarction group and that in old myocardial infarction group itself, the incidence is also greater among patients with positive Master's test than among those with negative Master's test, and this fact might be interpreted as to show that ischemia (ST depression in electrocardiogram) is more important than old myocardial infarction in causing hemodynamic disturbances and heart failure after exercise. The data of Saltups et al., Goldschlager et al., Parker et al., and Fitzibbon et al. are also in agreement with this view.

Another point to be noted is the fact that the patients whose pulse volume decreased after exercise showed the same response to ouabain, even though they included patients with negative Master's test as well as those with positive Master's test and those with old myocardial infarction and that the patients whose pulse volume increased after exercise responded to ouabain in the same way, even though they included patients with old myocardial infarction as well as those with negative Master's test.

These facts demonstrate that digital plethysmogram has a value of its own, different from that of electrocardiogram, and gives an important information concerning the hemodynamics of the heart.

Our data also show that about 10% of the patients with negative Master's test responded to exercise with a decrease in pulse volume, and we postulate that reversible heart failure occurred even in these patients after exercise. The fact that about 30% of patients with widespread coronary atherosclerosis had a negative Master's test and the data showing that hemodynamic abnormality occurred in response to exercise in patients with coronary artery disease who had a negative Master's test, are compatible with this line of thinking.

Digital plethysmogram is simple to record and gives no burden to the patient and can be repeatedly obtained at bedside from any patients, and because it gives an important hemodynamic information as shown here, postexercise digital plethysmogram can be used as a clinical tool in the evaluation of patients with ischemic heart disease.

**SUMMARY**

To assess the cardiocirculatory response to exercise in patients with ischemic heart disease, simultaneous digital plethysmograms and electrocardiograms were taken at rest and after a double Master's two step test. Patients were divided into three groups: Group I, negative Master's test (47 patients), Group II, positive Master's test (32 patients), and Group III, old myocardial infarction (30 patients). Pulse volume decreased in 5 of 47 patients (10.6%) in Group I, in 24 of 32
patients (75.0%) in Group II and in 18 of 30 patients (60.0%) in Group III after exercise, and there was highly significant difference between Group I and Group II (P<0.001) and between Group I and Group III (P<0.001). There was no significant difference between Group II and Group III.

In fifteen patients whose pulse volume decreased after exercise, the administration of 0.075 mg of ouabain did not cause any significant changes in heart rate and pulse volume at rest, but after exercise, it decreased heart rate and increased pulse volume significantly (P<0.05). In contrast, in 15 patients whose pulse volume increased after exercise, the administration of ouabain did not cause any significant changes in heart rate and pulse volume both at rest and after exercise.

It is concluded that exercise digital plethysmogram is useful in the evaluation of patients with ischemic heart disease from the hemodynamic standpoint.

REFERENCES


