Pulse Pressure Response to a Standard Exercise Stress

By G. H. Heidorn, M.D., M.S.

The response of pulse pressure to a standard exercise stress was utilized as the basis for an exercise tolerance test measuring left ventricular efficiency. Clinically normal individuals and asymptomatic individuals who had had a myocardial infarction were studied. Significant differences were noted between these groups. A phenomenon described as a "lag in response" was detected in many subjects with coronary artery disease. This may be a reflection of the delay in oxygen use and in other metabolic activities demonstrated by diseased myocardial tissue after an exercise stress.

THERE is a clinical need for an objective, reliable, and easily performed functional test that measures left ventricular efficiency. The purpose of this report is to present initial observations on the response of pulse pressure to a standard exercise stress in apparently normal individuals and in those who had a myocardial infarction. Pulse pressure response shows promise of being a sufficiently sensitive index to warrant its use in the clinical evaluation of left ventricular efficiency.

METHODS AND MATERIALS

One hundred and two men older than 40 years were studied. They included 50 apparently normal individuals (average age 50.2 years), 22 who had an anterior myocardial infarction 6 months to 9 years before this study was undertaken (average age 56.8 years), and 30 individuals who had a posterior myocardial infarction 3 months to 10 years preceding this study (average age 54.9 years). The "normal" individuals were asymptomatic, had no major illnesses or surgery in the past, were receiving no medication, had a normal physical examination, and a normal 12-lead electrocardiogram. There were no cardiac murmurs of any type upon auscultation; no more than minimal retinal arteriolosclerosis was detected; and a random blood pressure was below 150/90. Subjects who had a prior myocardial infarction were likewise asymptomatic at the time of the study, had the characteristic electrocardiographic evidences of a myocardial infarction either primarily anterior or primarily posterior in location, and were in full-time employment.

The procedure of the exercise tolerance test was as follows: A complete history and physical examination were performed, and a standard 12-lead electrocardiogram was obtained. A 15-minute rest period in the supine position followed. If apprehension was apparent, the rest period was prolonged until apprehension disappeared. If it persisted, the subject was eliminated from the study. Brachial artery blood pressure was recorded at rest in the supine position by a mercury sphygmomanometer. The disappearance of Korotkoff sounds was used as the diastolic end-point. The subject then exercised over the Master 2-step apparatus. The number of trips was based on his age and weight. A single Master exercise rather than a double test was preferred. Blood pressure, again in the supine position, was recorded immediately after exercise, between ½ to 1 minute, and 2 and 4 minutes after exercise. Periods up to 10 minutes after exercise were initially used, but did not appear to offer additional information. Radial artery pulse rates were also recorded at these intervals.

The relative safety of the single Master exercise in patients who had had a myocardial infarction is demonstrated by the fact that no adverse reactions (including angina pectoris) were observed during 89 exercise periods in 52 patients with coronary artery disease. Larger numbers of patients have been exercised in other reports without adverse reactions.

Results were recorded as absolute rise in pulse pressure and as per cent rise in pulse pressure (as compared to resting values) immediately following exercise. True work per beat of the left ventricle in moving blood within the aorta was calculated according to the formula published by Starr.

True work (Gm.M.) = 0.54 + 68.4 pulse pressure (mm. Hg) age (years).

RESULTS

Pulse pressure invariably rose immediately after exercise in apparently normal individ-
FIG. 1 Top. The individual response of pulse pressure in mm. Hg immediately after the completion of a standard, single Master exercise. A, normal controls; B, the anterior myocardial infarction group; C, the posterior myocardial infarction group. Results below the solid continuous line are arbitrarily selected as abnormal. Dotted line, average value for each group.

Fig. 2 Bottom. The individual per cent rise of pulse pressure immediately after exercise as compared to the resting value. A, normal controls; B, the anterior myocardial infarction group; C, the posterior myocardial infarction group. Values below the solid continuous line are arbitrarily selected as abnormal. Dotted line, average value for each group.

FIG. 3. Two representative individuals illustrating the "lag in response" phenomenon encountered in the myocardial infarction groups. Vertical dotted line at 0, completion of exercise. Values preceding this line are resting values.

pressure immediately after exercise, or a 63 per cent (range 18 to 128 per cent) rise in pulse pressure when compared to resting values. Individuals with a prior posterior myocardial infarction averaged a 26 mm. Hg (range —2 mm. to 68 mm. Hg), or, a 51 per cent (range —4 to 142 per cent) rise in pulse pressure. Those who had a prior anterior myocardial infarction averaged only an 18 mm. Hg (range —4 mm. to 38 mm. Hg), or, a 34 per cent (range —7 to 95 per cent) rise in pulse pressure immediately following exercise. The group differences are portrayed in a more apparent fashion in figures 1 and 2.

If rise in pulse pressure of less than 12 mm. Hg is arbitrarily selected as an abnormal response, 8 (36 per cent) of the group with anterior myocardial disease, 5 (17 per cent) of the group with posterior myocardial disease, and none of the normal group demonstrated an abnormal response. Also, if a rise in pulse pressure of less than 25 per cent immediately after exercise as compared to the resting value is arbitrarily selected as an abnormal response, 10 (45 per cent) of the anterior myocardial infarction group, 5 (17 per cent) of the posterior myocardial infarction group, and 1 of the 50 normal individuals fell below this dividing per centile. It is to be emphasized that these are arbitrarily selected standards that are subject to change as experience widens.

A "lag in response" (fig. 3) is defined as
Pulse pressure response to exercise

A greater pulse pressure at 1 minute after exercise than immediately after exercise. Pulse pressure is normally greatest immediately following exercise. A "lag in response" in this study was a phenomenon characteristic of a diseased myocardium. It was observed in 8 (36 per cent) individuals with a prior anterior myocardial infarction (2 of whom had an otherwise normal result), and 11 (37 per cent) times in those with a prior posterior myocardial infarction (6 of whom had an otherwise normal result). It was not noted in apparently normal individuals. The "lag in response" phenomenon was most dramatically illustrated by 2 patients with coronary artery disease in whom the pulse pressure actually fell 4 and 7 per cent, respectively, immediately after exercise (figs. 1 and 2).

Pulsus alternans after exercise was detected in some patients by the mercury sphygmomanometer. This was recorded as an abnormal response. Pulses alternans was observed in 2 individuals in both the anterior and posterior myocardial infarction group (3 of these 4 subjects had an otherwise normal response to the exercise stress). It was not noted in the normal group.

In summary, the following responses were arbitrarily selected as abnormal ones: a rise in pulse pressure of less than 12 mm. Hg or less than 25 per cent immediately after exercise, a "lag in response," and pulsus alternans after exercise. Usually, more than one of the above responses were observed in an individual who had an abnormal result. At least one of these abnormalities was noted in 13 (59 per cent) of those with anterior myocardial disease, 13 (43 per cent) of those with posterior myocardial disease, and in 1 apparently normal subject who had an 18 per cent rise in pulse pressure, but, had an otherwise normal response.

It is of interest in the light of this study to evaluate some of the indices that have been used as a guide to cardiac function in the past. The older criteria considered a normal response to be a return of systolic and diastolic blood pressure and of pulse rate to within 10 "points" of the resting value 2 minutes after a standard single Master exercise. These criteria have been reported to be of insufficient sensitivity. This impression is confirmed by this study. Pulse rate did not return to within 10 beats per minute of the resting rate in 5 patients with an anterior myocardial infarction, 3 patients with a posterior myocardial infarction, but also in 6 normal subjects. Likewise, the return of pulse pressure to within 10 mm. Hg of the resting value within 2 minutes was an insensitive index. An "abnormal" result based on this criterion was present in 17 normal individuals, as well as in 16 patients with posterior myocardial disease and in 12 patients with anterior myocardial disease.

Normally, a fall in diastolic blood pressure is expected after exercise. However, this is not an invariable finding. Diastolic blood pressure fell after exercise in only 30 of the 50 normal subjects, 12 of 30 individuals who had posterior myocardial infarctions, and 10 of 22 individuals with anterior myocardial disease. Although diastolic blood pressure usually fell following exercise in normal subjects and usually rose in subjects with coronary artery disease, it was not a sufficiently sensitive index for use as an additional measure of response to exercise. There was no apparent correlation in the coronary artery disease group between rise in diastolic pressure and the occurrence of an otherwise abnormal or normal result.

Among other indices determined in this study were postexercise changes in true work per beat of the left ventricle in moving blood within the aorta. These data were calculated according to Starr's formula. The values are not herein tabulated because changes in true work were comparable to pulse pressure changes. Interestingly, true work per beat at rest was generally alike in all 3 groups studied. Normal individuals averaged 74.1 Gm. M. per beat; the anterior infarction group averaged 65.1 Gm. M.; and the posterior infarction group averaged 67.3 Gm. M. Immediately after exercise, the normal group averaged 115.5 Gm. M. per beat; the anterior myocardial infarction group averaged 82.7 Gm. M.; and the posterior myocardial infarc-
TABLE 1.—Comparison of Pulse Pressure Responses at Varying Time Intervals in Patients with a Prior Myocardial Infarction

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Time Since Infarction (months)</th>
<th>Pulse pressure response &lt;mm. Hg (%)&gt;</th>
<th>Interval clinical changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3</td>
<td>2 (lag)</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>34 (65)</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>54</td>
<td>- 4 (lag)</td>
<td>steady electrocardiographic improvement</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>8 (17)</td>
<td>25 lb. weight loss and electrocardiographic improvement</td>
</tr>
<tr>
<td>5</td>
<td>36</td>
<td>6 (12)</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>38 (95)</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>16 (40)</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>3</td>
<td>2 (lag)</td>
<td>electrocardiogram returned to normal</td>
</tr>
<tr>
<td>9</td>
<td>10</td>
<td>28 (48)</td>
<td>15 lb. weight reduction, improved electrocardiogram</td>
</tr>
<tr>
<td>10</td>
<td>48</td>
<td>8 (14)</td>
<td>none</td>
</tr>
<tr>
<td>11</td>
<td>20</td>
<td>24 (35)</td>
<td>none</td>
</tr>
<tr>
<td>12</td>
<td>4</td>
<td>30 (75)</td>
<td>electrocardiogram returned to normal</td>
</tr>
<tr>
<td>13</td>
<td>29</td>
<td>26 (42)</td>
<td>none</td>
</tr>
<tr>
<td>14</td>
<td>3</td>
<td>42 (78) no pulsus alternans</td>
<td>none</td>
</tr>
<tr>
<td>15</td>
<td>4</td>
<td>24 (39) no lag</td>
<td>none</td>
</tr>
</tbody>
</table>

In order to lend reliability to an exercise tolerance test, the results must be reproducible in successive testings. Reproducibility of results was determined in many individuals included in the myocardial infarction groups (table 1). Generally, the response of pulse pressure to exercise is reproducible in the same individual at varying time intervals. When results are at variance, the differences can usually be explained by changes in the clinical status of the patient. However, at times no clinical explanation for differences in response was at hand (table 1, patients 5, 11, 14, and 15).

DISCUSSION

The clinical determination of left ventricular efficiency is based chiefly upon an evaluation of symptoms. Dyspnea is of prime importance in this regard. Dyspnea, however, is a subjective symptom. Its appearance in a given individual is governed by many psychologic and physiologic factors. It is not necessarily an objective measurement of the functional status of the left ventricle. Exercise tolerance tests have been utilized in an attempt to measure the status of the left ventricle in a somewhat more objective fash-
Pulse pressure offers an advantage as an index of ventricular function that pulse rate and systolic and diastolic blood pressures do not possess. Pulse pressure has been more closely correlated with the actual work per beat of the left ventricle in moving blood within the aorta than have the other measurements. The response of pulse pressure to a standardized exercise stress was, therefore, utilized as the basis for an exercise tolerance test to measure the functional efficiency of the left ventricle.

The magnitude of pulse pressure rise as measured immediately after the completion of a single Master exercise was used as the index of response. Generally, the average rise of pulse pressure was greater in apparently normal individuals than in those asymptomatic individuals who had had a myocardial infarction (figs. 1 and 2). Also, the response was poorest in those subjects who had electrocardiographic evidence of a primarily anterior myocardial infarction. Although this series is too small to derive conclusions, this difference is perhaps related to the observation that the greatest proportion of the work of systolic ejection is performed by the interventricular septum and the anterior wall of the left ventricle.

The "lag in response" phenomenon as previously described is of considerable interest. It is apparently a characteristic of diseased myocardial tissue. It was not noted in normal subjects, but was present in more than a third of those with a prior myocardial infarction. This phenomenon is very likely related to the lag in oxygen use and to the delay in the mobilization of other metabolic activities that is exhibited by diseased myocardial muscle after an exercise stress. This delay in metabolic mobilization in the acute period following stress probably results in a lessened work ability of the muscle, a deficient cardiac output, and a lessened pulse pressure response. It is important to emphasize that the "lag in response" phenomenon would not be detected if physiologic measurements are first made 2 minutes after exercise is completed as is done in previously reported tests. Measurements should be made in the immediate period following exercise.

The occurrence of pulsus alternans after exercise was interpreted as an abnormal response and was added to the other criteria. Pulsus alternans after exercise was detected by the sphygmomanometer in 4 patients with coronary artery disease, but was not noted in normal subjects. Usually more than one of these criteria that indicate an abnormal response were met in any given patient who demonstrated an abnormal result. At least one of them was filled in 59 per cent of individuals who had a prior anterior myocardial infarction and in 43 per cent of those who had a prior posterior myocardial infarction. On only one occasion did an individual in the apparently normal group demonstrate an abnormal result. This was an isolated occurrence of a less than 25 per cent rise in pulse pressure. It is to be remembered that all of the subjects included in the group with coronary artery disease were asymptomatic. The only clinical residua of a myocardial infarction were contained in the electrocardiogram. Only an expected degree of dyspnea was experienced during exercise. It was therefore not possible to correlate pulse pressure responses with symptomatology. One might speculate that in many instances the response of pulse pressure to
exercise reveals abnormal myocardial function sooner than does the appearance of dyspnea.

Pulse pressure responses were generally reproducible upon repeat testings at varying time intervals (table 1). When the results were at variance, the differences could usually be explained by changes in clinical status such as significant weight reduction or changes in the electrocardiogram. At times, no explanation for the variations were at hand.

This exercise tolerance test has revealed a significantly greater incidence of abnormal responses in asymptomatic subjects with coronary artery disease than in a total of 70 apparently normal individuals. This fact encourages further study of this procedure. It is important in the performance of this test that pulse pressure be measured immediately and 1 minute after exercise is completed in order to detect the "lag in response" phenomenon, which seems to be a phenomenon characteristic of diseased myocardial tissue. The applications of this test would seem to be greatest as a screening procedure, as an evaluation of individuals with bundle-branch block, or in those with borderline electrocardiographic changes. Investigations in this regard are in progress.

SUMMARY

The response of pulse pressure to an exercise stress, standardized as to the age and weight of the individual and performed on the Master 2-step apparatus, was utilized as the basis for an exercise tolerance test. Pulse pressure was selected in preference to other physiologic indices such as pulse rate, systolic and diastolic blood pressure, etc., as it is more closely correlated with the true work per beat of the left ventricle in moving blood within the aorta.

The magnitude of pulse pressure rise as recorded immediately after the completion of the standard exercise showed considerable variation between 50 apparently normal individuals and an asymptomatic group of comparable ages composed of 22 individuals who had had an anterior myocardial infarction and 30 individuals who had had a posterior myocardial infarction.

An abnormal pulse pressure response to exercise was arbitrarily selected as follows: (1) a rise in pulse pressure immediately following exercise of less than 12 mm. Hg or less than 25 per cent, (2) a "lag in response," (3) or pulsus alternans after exercise. At least one of the above criteria of abnormality was noted in 59 per cent of individuals who have anterior myocardial disease and in 43 per cent of individuals who have posterior myocardial disease. There was only an isolated occurrence of an abnormal result in the normal group. In general, subjects with posterior myocardial disease performed better in this study than did those with anterior disease.

A "lag in response" is defined as a greater pulse pressure at 1 minute after exercise than immediately after the exercise was completed. This phenomenon was specific in this study for individuals with a prior myocardial infarction and occurred in more than a third of this group.

The use of pulse pressure response to exercise as a measure of left ventricular function shows promise of being sufficiently sensitive for practical clinical application and merits further study.

ACKNOWLEDGMENT

The technical assistance of Mrs. Helen Jordan, R.N., Mr. John W. House, and Miss Ruth Ann Knouse is gratefully acknowledged.

SUMARIO IN INTERLINGUA

Le responsa del pression del pulso a stress per exercitio—standardisate con respecto al etate e al peso del subjecto e execute con le apparato bigradate de Masters—esseva usate como indice in un test del tolerantia de exercitios. Le pression del pulso esseva seligite plus tosto que altere indices physiologie—per exemplo le frequentia del pulso, le pression sanguinee systolic e diastolic, etc.—proque illa es plus directemente correlationate con le ver labor que le ventriculo sinistre executa per pulso individual in mover le sanguine intra le aorta.
Le magnitudes del augmento in pression del pulso mesurate immediatamente post le completion de un exercitio standard variava considerabilemente inter (1) 50 apparentemente normal individuos e (2) un grupo de individuos asymptomatic de etates comparabile, includente 22 individuos che habeva habite un infarcimento antero-myocardial e 30 qui habeva habite un infarcimento postero-myocardial.

Anormalitade del responsa del pression del pulso al stress de exercitio eseva definite arbitrarimente como respondente al sequente criterios: (1) Un augmento in le pression del pulso immediatamente post le exercitio amon-tante a minus que 12 mm de Hg o a minus que 25 pro cento, (2) "retardo del responsa," o (3) pulso alternante post exercitio. Al minus un de iste tres criterios de anormalitate eseva notate in 59 pro cento del individuos con morbo antero-myocardial e in 43 pro cento del individuos con morbo postero-myocardial. In le grupo normal il habeva solmente un occurrientia isolate de un resultato anormal. In general, subjectos con morbo postero-myocardial exiva melio in iste studio que subjectos con morbo antero-myocardial.

"Retardo del responsa" es definite como le occurrientia de un pression del pulso plus grande 1 minuta post le exercitio che imme-diamente post su completion. Iste phenome-no eseva, in le presente studio, specific pro individuos con previe infarcimento myocardial e eseva notate in plus que un terto de illes.

Le uso del responsa del pression del pulso a standards de exercitio como mesura del function sinistro-ventricular promitte mostrar se sufficientemente sensibile pro practic objectivos clinic. Illo merita investigaciones additional.

REFERENCES
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