Changes in the Ballistocardiogram after Exercise in Normal and Abnormal Subjects

By Donald H. Makinson, M. R. C. P.

Ballistocardiograms were taken before and at intervals after mild exercise. The normal response has been ascertained by a statistical study of results obtained on 40 healthy subjects. The response of patients varies widely; in some the change after exercise is much diminished, but in others the response is abnormally increased. In some of the form of the ballistocardiogram, normal at rest, becomes abnormal after exercise, so the exercise test will often bring out abnormalities not detected by observations made at rest. Additional evidence was secured that the ballistocardiogram may become abnormal before the clinical picture of coronary heart disease develops.

Physicians for centuries have been striving to secure objective means of assessing cardiac function by examining the heart during or after exercise. A full account of these is contained in Master's paper on "The Two-Step Test of Myocardial Function." In recent years the ballistocardiograph has made available further evidence of abnormalities in many persons with diseased hearts. Hitherto this test has been applied almost solely to resting subjects and it seemed reasonable to expect that an even more delicate test would be secured by obtaining these records while the heart was subjected to a strain, as a slightly diseased heart might be able to behave normally when the body was at rest, but be unable to cope with the situation in a normal manner when increased work was demanded of it. As subjects must not move while ballistocardiograms are being taken satisfactory records cannot be secured during exercise, but they can be obtained immediately after exercise while the heart is still stimulated. Records made at this time and at intervals until the cardiac function returned to normal after exercise have been secured on 98 subjects, 41 of whom were healthy persons, the rest patients from the wards or outpatient departments of the University Hospital. From the results obtained on the healthy group normal standards have been developed, largely by statistical methods. When the results obtained on the patients are compared with these normal standards it is clear that evidence of cardiac dysfunction can be readily secured by this exercise test, and that abnormalities not identified by records obtained at rest can be demonstrated in many instances.

In 1938 a study of this nature was commenced in this laboratory by M. W. Stroud and E. M. Evans, then undergraduate medical students working under Dr. Starr's direction. Later Dr. Starr made a few observations himself but the work was soon abandoned owing to the war. The records obtained in this early work were made available to me and I have remeasured them and recalculated the results. In addition many more subjects were tested, so that the great majority of the tests were conducted by myself in 1948–49. But the old records were valuable because they not only provided independent confirmation of the later results but also permitted me to secure long after-histories on some of the more interesting cases.

Methods

Subjects

A total of 98 exercise experiments was performed upon 41 subjects who considered themselves to be healthy, and 57 who were clinically abnormal. The control group was drawn from medical students, faculty members and hospital employees. Their ages and sexes are given in table I. The 57 patients were secured from the wards and outpatient departments of the University Hospital; all had been subjected to the routine hospital study and these data were employed in assessing their clinical diagnosis and status. Thirty-eight were males, 25 fe-
males, and they can be subdivided as is shown in table 4.

The 13 patients included in the rheumatic heart disease group consisted of 8 with rheumatic mitral disease alone, none of whom had ever been in congestive failure. Four of these, aged 20, 41, 20 and 30, all in Functional Class I, had mitral stenosis alone and were not receiving digitals. Two, aged 38 and 52, who had auricular fibrillation in addition to mitral stenosis, were receiving digitals and they were placed in Functional Classes II and III, respectively. In 2 further patients, aged 25 and 27, placed in Functional Classes II and III, mitral stenosis was associated with mitral incompetence. Of the remaining 5 patients, one, aged 34, in Functional Class I, had mitral stenosis and aortic stenosis; two, aged 20 and 30, in Functional Classes I and II, had mitral stenosis and aortic incompetence; and two, aged 20 and 21, in Functional Class I, had pure aortic incompetence.

None of the 9 patients with the anginal syndrome, whose ages ranged from 47 to 77, gave a clinical history of myocardial infarction, although one individual showed electrocardiographic changes suggestive of an old posterior myocardial infarct.

The 4 patients with essential hypertension, aged 34, 51, 56 and 67, had blood pressures of 190/125, 150/100, 190/110 and 170/105 when at rest. None of these had evidence of renal involvement nor had they been in congestive failure. One, however, a man aged 51, showed electrocardiographic evidence of left bundle branch block.

The 2 patients with thyrotoxicosis had basal metabolic rates of +58% and +62% and neither showed any disturbance of heart rhythm, nor was there other evidence of thyroid heart disease.

The 11 cases of neurocirculatory asthenia were characterized by complaints related to the circulation, especially dyspnea on exertion and precordial pain not definitely related to exertion, but extensive hospital studies failed to reveal any evidence of organic disease.

The 3 patients with postural hypotension were aged 44, 44 and 38; they experienced symptoms of vertigo and faintness associated with a fall in blood pressure on assumption of the erect posture. One of these cases has been described in detail. Incapacity of the other two was not so great.

The 3 patients with hypothyroidism were aged 43, 35 and 52; they had basal metabolic rates of -10%, -36% and -20%, respectively.

The miscellaneous group consisted of 2 cases of traumatic A-V fistula and single cases of pheochromocytoma, syphilitic aortic regurgitation, Addison's disease, arteriosclerosis, emaciation (cause unknown), postoperative thyroidectomy with bundle branch block, tetrology of Fallot, diabetes mellitus, polycythemia vera and coartation of the aorta.

**Table 1.—Distribution of Normal Subjects with Regard to Age and Sex**

<table>
<thead>
<tr>
<th>Age in Years</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-29</td>
<td>12</td>
</tr>
<tr>
<td>30-39</td>
<td>4</td>
</tr>
<tr>
<td>40-49</td>
<td>5</td>
</tr>
<tr>
<td>50-59</td>
<td>4</td>
</tr>
<tr>
<td>60-69</td>
<td>30</td>
</tr>
</tbody>
</table>

The 3 patients with hypothyroidism were aged 43, 35 and 52; they had basal metabolic rates of -10%, -36% and -20%, respectively.

The miscellaneous group consisted of 2 cases of traumatic A-V fistula and single cases of pheochromocytoma, syphilitic aortic regurgitation, Addison's disease, arteriosclerosis, emaciation (cause unknown), postoperative thyroidectomy with bundle branch block, tetrology of Fallot, diabetes mellitus, polycythemia vera and coartation of the aorta.

**Technics**

All subjects were examined more than two hours after their last meal and ballistocardiograms were taken according to the usual technic, after a fifteen-minute rest period. Mild exercise was performed on the Master steps, 2 steps each of 9 inches, arranged so that the top step has a lower step on each side of it. The number of trips across the steps was graded according to age and weight. Half the number of trips recommended by the Master tables were performed in half the time allotted by him, so my subjects took from 9 to 12 trips across the steps in forty-five seconds.

The first ballistocardiogram was secured after fifteen minutes' rest in the horizontal position. The subject then arose and walked over the steps. At the completion of the exercise he immediately lay down and a ballistocardiogram was taken as soon as possible. This was usually about ten seconds after cessation of exercise but the extremes were five and thirty seconds. Additional records were secured at 50, 90 and 180 seconds after completion of exercise. Figure 1 gives several typical examples of the records secured.

Blood pressure was estimated at rest and in the intervals between the taking of ballistocardiograms after exercise.

**Analysis of the Records**

When the record was normal in form typical large and small complexes were selected and measured. Cardiac output was calculated according to the following formula:

\[
\begin{align*}
\text{Percentage deviation from normal} = & K \sqrt{2 \int I dt + \int J dt} \sqrt{c \times \frac{\text{Pulse rate}}{\text{Body weight}}} - 100 \\
\end{align*}
\]

\(\int I dt\) and \(\int J dt\) being the areas of the I and J waves of the ballistocardiogram in mm. sec. and \(c\) the duration of the cardiac cycle in seconds, and \(K\) being obtained from Starr's table. The ballistocardiogram was adjusted so that 280 Gm. displaced the light spot 1 cm.

The effect of exercise on the circulation was estimated in the following way which is best made clear by an example. Subject EWC, after fifteen minutes' rest, had a circulation which was -21% below the mean value of healthy resting subjects,
age and weight being considered. At 10, 50 and 90 seconds after exercise his circulation deviated by +40%, +1% and −16% from the same mean value of healthy resting subjects. Therefore, the changes due to exercise were recorded as +61%, +22% and −5%. Data so calculated were averaged and the results appear in table 2 which defines the normal standards.

After all my experiments had been completed, a method for the estimation of cardiac strength from the ballistocardiogram was published, so it seemed of interest to calculate standards for the normal

<table>
<thead>
<tr>
<th>Time after Exercise</th>
<th>No. in Group</th>
<th>Changes in cardiac output after exercise*</th>
</tr>
</thead>
<tbody>
<tr>
<td>sec.</td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>5-30</td>
<td>40</td>
<td>+47.4</td>
</tr>
<tr>
<td>50</td>
<td>40</td>
<td>+14.25</td>
</tr>
<tr>
<td>90</td>
<td>40</td>
<td>+4.87</td>
</tr>
<tr>
<td>180</td>
<td>40</td>
<td>+3.97</td>
</tr>
</tbody>
</table>

* Differences between the resting cardiac output of each subject before exercise and the values found after exercise.

When the record was abnormal in form the method of recording used by Starr and Mayock was adopted. First the proportion of abnormal complexes present was estimated, and second the abnormalities of form of the complexes were described in detail. But in order to simplify this presentation, the records have been classified merely as normal or abnormal. The estimation of cardiac output was not attempted if the ballistic form was abnormal. However, if only one complex in each respiratory cycle was abnormal, the cardiac output was calculated from a representative small normal complex and a representative large complex, in the usual manner.

The statistical analysis was performed according to the methods of Fisher. The word significant will be used only in the statistical sense, indicating a probability of 95 in 100 or over that the result was not due to chance alone.

**RESULTS**

**Results Secured on Healthy Persons.** A statistical analysis of the response to exercise of all members of the normal group failed to reveal any significant correlation with age or sex and so the group was analyzed as a whole. Table 2 gives the results of this analysis. Immediately after exercise had ceased the cardiac output was found to average almost 50 per cent above the resting level. This increase subsided rapidly so that the resting value was almost, but not quite, attained at 180 seconds after exercise had ceased. We propose to regard as abnormal any case which deviates from the mean by more than twice the standard deviation.

**Average Results Secured in Various Types of Disease.** Table 4 gives standard deviations for the increase in cardiac output after exercise obtained both in the normal group and in the clinically abnormal cases grouped according to the chief diagnosis. The averages show that an increase in cardiac output after exercise, significantly greater than normal, was found in patients with rheumatic heart disease during the first ninety seconds after exercise had ceased. In those with neurocirculatory asthenia the average increase of circulation immediately after exercise was very close to the normal, but the increase was much more prolonged, becoming significantly greater than the normal at fifty seconds. Both cases with thyrotoxicosis showed increases after exercise which far ex-

<table>
<thead>
<tr>
<th>Time after Exercise (Sec.)</th>
<th>No. in Group</th>
<th>Changes in cardiac strength after exercise*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td>5-30</td>
<td>40</td>
<td>+44.7</td>
</tr>
<tr>
<td>50</td>
<td>39</td>
<td>+20.9</td>
</tr>
<tr>
<td>90</td>
<td>39</td>
<td>+13.7</td>
</tr>
<tr>
<td>180</td>
<td>37</td>
<td>+8.0</td>
</tr>
</tbody>
</table>

* Difference between the spread of the complexes (I, J, L, J) for each subject before exercise and the values found after exercise.
ceed the average normal at each test made during the first ninety seconds after exercise, but because of the small number tested statistical significance was attained at only one of the three estimations.

In angina pectoris the average increase immediately after exercise was very close to the normal but increased cardiac activity persisted much longer. At fifty seconds after exercise the mean was significantly higher than normal and for the rest of the test the difference in the averages is striking even though statistical significance was not quite attained. An analysis of the individual results is given in table 5, and shows that 4 of the 9 cases did not have an excessive increase after exercise; however, no significant clinical differences could be found between those who did and those who did not show an abnormal increase in cardiac output.

In contrast, the cases of hypothyroidism, postural hypotension and essential hypertension, judging by the means of each group, responded to the exercise by a smaller increase than the normal which was significant at the first test in all three groups.

The failure of the 4 cases of hypertension to increase their circulation after exercise is especially striking and indeed the whole pattern of their response is abnormal, the mean diminishing until it reaches a point not only significantly below the normal, but also 27 per cent below the average attained before exercise.

**Results in Individual Patients.** The normal standards permit testing the normality of individuals as well as of groups, so all the 57 members of the abnormal group have been thus tested. This has been done in two ways, by studying the changes of estimated cardiac output after exercise, and by neglecting the values found before exercise and concentrating attention on the levels obtained after it. When the change due to exercise was studied and the findings in each patient compared with the normal range, defined as the mean value ± twice the standard deviation, it was found that 28 of the 57 patients gave an abnormal response to exercise. In 9 of these the circulation

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**Table 4.—Average Change of Cardiac Output after Exercise from Each Subject’s Resting Value**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Subjects</th>
<th>5–30 secs.</th>
<th>30 secs.</th>
<th>90 secs.</th>
<th>180 secs.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Normal</td>
<td></td>
<td>41</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>R.H.D.</td>
<td>47.4</td>
<td>24.2</td>
<td>14.25</td>
<td>14.3</td>
<td>4.89</td>
</tr>
<tr>
<td></td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td>3.97</td>
</tr>
<tr>
<td>Angina</td>
<td>48.8</td>
<td>27.4</td>
<td>31.5*</td>
<td>31.4</td>
<td>13.4</td>
</tr>
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<td></td>
<td>9</td>
<td></td>
<td></td>
<td></td>
<td>6.1</td>
</tr>
<tr>
<td>N.C.A</td>
<td>49.7</td>
<td>71</td>
<td>24.7*</td>
<td>55.5</td>
<td>8.0</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td></td>
<td></td>
<td></td>
<td>1.5</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
<td>58</td>
<td>37</td>
<td>14.5</td>
<td>21.5*</td>
<td>19.5</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td>3.5</td>
</tr>
<tr>
<td>Hypothyroid</td>
<td>15.9*</td>
<td>16.4</td>
<td>19</td>
<td>24</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>Postural Hypotension</td>
<td>34.5</td>
<td>11.3</td>
<td>2.4</td>
<td>1.3</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
<td>2.5*</td>
<td>22.2</td>
<td>2.5</td>
<td>23.4</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td></td>
<td></td>
<td></td>
<td>9.3</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>2</td>
<td>2.5</td>
<td>22.2</td>
<td>2.5</td>
<td>23.4</td>
</tr>
</tbody>
</table>

* Denotes statistically significant difference from corresponding value in normals as determined by Fisher’s "t" test.

increased less, in 19 it increased more than normal after exercise.

The level of the circulation after exercise was of great interest because in this step-climbing test the work performed was automatically adjusted to the size of the subject. Therefore it seemed that useful information might be obtained by neglecting the resting value as irrelevant and concentrating attention on the level of the circulation after exercise. Accordingly the data have been analysed from this viewpoint and table 6 shows the results of the statistical analysis for both the normal and the abnormal groups. The same trends for specific clinical groups are noted in this analysis as in the previous one (Table 4), except for the neurocirculatory asthenia group which shows
TABLE 5.—Changes after Exercise in Patients with Angina Pectoris

<table>
<thead>
<tr>
<th>Age</th>
<th>Functional Class</th>
<th>Cardiac Output (Resting)</th>
<th>Cardiac Output Change from resting values at times from 5 to 180 sec. after exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>5-30 sec. 50 sec. 90 sec. 180 sec.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>64</td>
<td>III</td>
<td>-22</td>
<td>50</td>
</tr>
<tr>
<td>49</td>
<td>II</td>
<td>-13</td>
<td>64</td>
</tr>
<tr>
<td>54</td>
<td>III</td>
<td>36</td>
<td>40</td>
</tr>
<tr>
<td>77</td>
<td>III</td>
<td>-11</td>
<td>71</td>
</tr>
<tr>
<td>47</td>
<td>II</td>
<td>-14</td>
<td>12</td>
</tr>
<tr>
<td>66</td>
<td>III</td>
<td>-5</td>
<td>19</td>
</tr>
<tr>
<td>53</td>
<td>II</td>
<td>-17</td>
<td>48</td>
</tr>
<tr>
<td>47</td>
<td>II</td>
<td>-2</td>
<td>27</td>
</tr>
<tr>
<td>50</td>
<td>II</td>
<td>5</td>
<td>57</td>
</tr>
</tbody>
</table>

TABLE 6.—Average Levels of Cardiac Output after Exercise in Terms of Average Normal Resting Values

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of Subjects</th>
<th>Time After Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>5-30 secs. 50 secs. 90 sec. 180 sec.</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>Normal</td>
<td>41</td>
<td>43.8</td>
</tr>
<tr>
<td>R.H.D.</td>
<td>13</td>
<td>67.5</td>
</tr>
<tr>
<td>Angina</td>
<td>9</td>
<td>44.1</td>
</tr>
<tr>
<td>N.C.A.</td>
<td>11</td>
<td>33.1</td>
</tr>
<tr>
<td>Thyrotoxicosis</td>
<td>2</td>
<td>87</td>
</tr>
<tr>
<td>Hypothyroid</td>
<td>3</td>
<td>8.3</td>
</tr>
<tr>
<td>Postural Hypotension</td>
<td>3</td>
<td>23.6</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
<td>22.7</td>
</tr>
</tbody>
</table>

* Denotes statistically significant difference from corresponding value in normals as determined by Fisher’s “t” test. All results are expressed as per cent deviation from the average resting normal. In this compilation the resting level of the individuals tested has been neglected.

Changes in Ballistocardiogram after Exercise

When the normal range of levels after exercise, defined as the mean value ± twice the standard deviation, is compared with the individual results of the abnormal group, 14 of the 37 persons in this group gave an abnormal response. These 14 subjects are included in the 28 so found when the increase of the cardiac output after exercise is considered (Table 4).

Changes of Form of the Ballistocardiographic Curve after Exercise. In addition to abnormals of the response to exercise detected by abnormal changes in size of the record, and of the cardiac output as calculated from it, the exercise test often gave evidence of abnormality of another kind. In the group of clinically abnormal subjects, 40 had normal tracings at rest and in 12 of these the form became abnormal after exercise. Examples are shown in figure 1 B and C. In 13 of the 17 patients who had abnormal tracings at rest, the abnormality persisted after exercise as one would expect, but it was a surprise to find that in 4 the tracings became normal in form after exercise. Twenty-eight of the clinically abnormal group gave an abnormal cardiac output response to exercise and of these, 18 had normal curves before, 3 of which became abnormal after exercise and 10 gave abnormal curves before, 3 of which became normal after exercise.

The commonest abnormality of form produced by exercise was slurring, widening or splitting of the J wave; this was always found in the first tracing taken after exercise, and the form usually had returned to normal by 180 seconds after exercise had ceased (Fig. 1, B and C). In a few cases the J wave became small soon after exercise, returning to normal later.

Blood Pressure and Pulse Rate Responses. In his experiments, employing the full number of recommended trips, Master found that the blood pressure (systolic and diastolic) and the pulse rate of the majority of his normal subjects had returned to the resting value within two minutes of the cessation of exercise. In the 98 subjects in this series who performed half
the number of trips required by the full Master
test, 14 of the clinically abnormal and 5 of
the clinically normal failed to have a return of
their blood pressure and pulse rate to within

different from the majority. In most subjects,
as one would certainly expect, the maxima
for both cardiac strength and output were
found immediately after exercise was over and
the values declined slowly for the rest of the
period of observation. But some apparently
healthy persons responded in an entirely dif-
derent manner. Thus there were three cases in
which the maximum values were not attained
until 90 or even 180 seconds after the cessation
of exercise. Another subject, apparently in ex-
cellent physical condition but 54 years old, gave
an even more unusual response. He always had
resting values considerably larger than the av-
erage for his age and after the mild exercise
required in this test, he manifested little if any
increase. This subject was tested repeatedly,
always with the same result. Perhaps in view of
his unusually large circulation at rest no in-
crease was needed for such mild exercise, a view-
point in accord with another observation made
on him. When this subject stood upright his
resting circulation was much smaller than when
he lay supine. When given an exercise test with
ballistocardiograms taken in the upright posi-
tion, his circulation increased normally after
exercise. But one of Hickam and Cargill's sub-
jects, judged to have a normal circulation
although suffering from syphilis of the central
nervous system, also had an insignificant in-
crease in cardiac output after mild exercise
although his resting circulation was not un-
usually elevated. So perhaps a certain per-
centage of healthy persons react to mild
exercise mainly by increasing their arterio-
venous oxygen difference. But whatever the
reason for these divergent responses among
healthy persons, the inclusion of their data
has greatly increased the normal range and
rendered the detection of abnormality more
difficult.

Several attempts were made to diminish the
scatter of the normal group and so to improve
the ability to detect abnormality. Normal
standards were made in terms of cardiac
strength\(^1\) and of the level of estimated cardiac
output after exercise, but the results disclosed a
scatter as great as or greater than that of the
changes of cardiac output after exercise. So
no means of reducing the scatter of the normal
standards has been found. However, despite

**Table 7.—Changes after Exercise in Patients with
Rheumatic Heart Disease**

<table>
<thead>
<tr>
<th>Age</th>
<th>Functional Class</th>
<th>Resting Cardiac Output</th>
<th>Percentage increase in Cardiac Output over resting values at times from 5 to 180 seconds after exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>%</td>
</tr>
<tr>
<td>30</td>
<td>I</td>
<td>-14</td>
<td>43</td>
</tr>
<tr>
<td>20</td>
<td>I</td>
<td>-20</td>
<td>77</td>
</tr>
<tr>
<td>41</td>
<td>II</td>
<td>-21</td>
<td>24</td>
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<td>20</td>
<td>II</td>
<td>-29</td>
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</tr>
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<td>38</td>
<td>I</td>
<td>-32</td>
<td>88</td>
</tr>
<tr>
<td>52</td>
<td>III</td>
<td>-19</td>
<td>51</td>
</tr>
</tbody>
</table>

**Mitril Stenosis Alone**

| 25  | I   | -20 | 75 | 8 | 1 | -14 | |
| 37  | III | -35 | 149 | 31 | 39 | 39 | |

**Mitril Stenosis and Mitril Incompetence**

| 34  | I   | -17 | 53 | 31 | 16 | 15 | |

**Mitril Stenosis and Aortic Stenosis**

| 20  | I   | 11  | 15 | 9 | 0 | 4 | |
| 30  | II  | -3 | 62 | 39 | 47 | 38 | |

**Aortic Incompetence**

| 21  | I   | 42  | 41 | 19 | -20 | -34 | |
| 20  | I   | 11  | 121 | 41 | 15 | 5 | |

10 points of the resting value within two min-
utes after exercise.

**Discussion**

It was the purpose of this investigation to
determine whether a study of ballistocardi-
ographic tracings taken after mild exercise would
help further in distinguishing patients with
abnormalities of cardiovascular function. It is
believed that this object has been achieved
but a difficulty has been encountered which
must not be minimized. The results obtained
in healthy subjects scatter so widely that only
marked abnormality can be detected. Much
thought has been given to the reasons for this
diversity of normal response.

The scatter of the response to exercise in
healthy persons is greatly increased by the fact
that some subjects give results of a pattern
CHANGES IN BALLISTOCARDIOGRAM AFTER EXERCISE

Fig. 1
this situation, the abnormalities found in many patients are so great that they may be readily detected by the ordinary statistical criteria and it seems evident that the exercise test provides evidence of an abnormality undetected by other methods in many cases.

It will be recalled that ballistocardiograms taken at rest may detect abnormality in one of three ways. The record may be too large or too small for a subject of that age and weight, abnormalities usually identified by calculating cardiac output from the wave measurements, or the record may be abnormal in form. Ballistocardiograms taken before and after exercise may also be used to detect abnormality in three additional ways; first, after exercise the normal increase in size of the record and in the calculated cardiac output may not take place; second, the cardiac response to exercise may be excessive; or third, the form of the record, normal before exercise, may become abnormal after it.

The results clearly indicate that the exercise test is the more delicate. Thus, among my 98 subjects, tests made at rest indicated that 18 subjects showed abnormality by abnormal change in cardiac output, 17 by abnormal shape of the ballistocardiogram, and 7 in both ways. In the records made after exercise 28 were judged abnormal on the basis of abnormalities of form of the curve not present at rest, and 4 on the basis of both. Thus 28 individuals were picked out as abnormal from the resting ballistocardiographic tracing and 23 additional subjects, whose ballistocardiogram passed as normal at rest, had abnormal ballistocardiographic tracings after exercise. Consideration of the absolute cardiac output after exercise, instead of the increase over resting cardiac output, does not increase the usefulness of the test. Experience with the estimation of cardiac strength is as yet insufficient to form a basis for opinion.

This investigation was designed with the expectation that cardiac abnormality, not to be identified at rest, would be made manifest because a diseased heart would be unable to increase the cardiac output after exercise or, in an effort to do so, would contract in an abnormal manner which would result in distortion of the ballistocardiogram. These expectations have been fully realized in many instances. In my cases of myxedema and essential hypertension the failure to increase the size of the ballistocardiogram after exercise was often conspicuous; Hickam and Cargill found a similar failure to increase cardiac output after exercise in cases of congestive failure. In addition typical examples of the many instances in which abnormality of ballistic form developed after exercise are given in figure 1.

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**Fig. 1.** Ballistocardiograms taken before and after exercise. The reproduction from A to E inclusive is 3/5 actual size; that of F is 4/5 actual size. Time records largest interval = 1 second.

A. Records on D. H. M., a healthy man aged 28, height 6'0'', weight 160 lbs. Left before exercise, right commences 10 sec. after the cessation of exercise. This response is typical of normal subjects.

B. Records of J. C., 49, 5'2'', 107 lb. Taken in 1939 when she was in the hospital ward because of a gastric complaint. The routine study gave no evidence of cardiac disease at that time and none was suspected by the attending physician. Left record before, right record 10 sec. after exercise. Note that the amplitude does not increase after exercise and the form becomes abnormal.

C. Records of J. C. who now has developed angina pectoris, taken in 1949. Left record before, center 10 sec. after, right 240 sec. after exercise. Note that the record taken at rest is still normal, the record taken immediately after exercise is abnormal in form but of increased amplitude. The abnormality disappears by the time of the last record.

D. Records of D. R., aged 47, 5'3'', 163 lb., a case of angina pectoris. Left record before exercise, right at 10 sec. after exercise. Both tracings are abnormal but there is very little increase in amplitude of the complexes after exercise.

E. Records of S. S., aged 48, 5'4'', 131 lb., a case of neurocirculatory asthenia. Left record before exercise, right 15 sec. after exercise. Both tracings are abnormal but there is considerable increase in amplitude of the complexes after exercise.

F. Records of J. B., aged 58, 5'74'', 132 lb., a case of toxic nodular goiter. Left before exercise, right 25 sec. after exercise. The records at rest and after exercise are both abnormal in form but the amplitude of the complexes does increase after exercise.
Also not unexpected was the increased response of the cases diagnosed as neurocirculatory asthenia; this is in keeping with the concept\textsuperscript{13} that inability to regulate the circulation properly is a fundamental factor in this condition. A similar abnormality in the cases of thyrotoxicosis is consistent with the well known instability of the circulation seen not only in patients but also in experimental animals given large doses of thyroid. The differences from the normal in these 2 cases were large; significance was not attained more frequently solely because of the small number.

But it is characteristic of many investigations that they raise as many problems as they solve. The finding that my cases of rheumatic heart disease and of angina pectoris gave, on the average, an excessive response to exercise came as a complete surprise. So the clinical data were carefully studied to see if this most unexpected finding could be understood or explained away. Table 7 gives the findings secured in the cases of rheumatic heart disease in detail.

It will be recalled that the response to exercise has been measured in terms of the findings at rest, so it was possible that the unexpected abnormality mentioned might be due to a diminished circulation at rest rather than to an unusually high level during exercise. Reference to table 7 shows that this is in part true; 7 of 9 cases without aortic incompetence had resting circulations below the average normal. But careful study of these data shows that this is not the full explanation, for in 3 cases the increase after exercise exceeded 120 per cent and in many other cases the abnormal prolongation of the response is conspicuous.

Another possible explanation deserves comment. In cases of aortic regurgitation the ballistocardiogram measures the true stroke volume, not the amount of the circulation. In this condition an increase in the amount of blood regurgitated after exercise in the presence of a normal increase in circulation after exercise, would cause an abnormal increase in ballistic amplitude. But in only 4 of the 13 cases of rheumatic heart disease was this lesion diagnosed so this is obviously not the explanation for most cases.

A similar study was made of the cases of angina pectoris in my series. The mild exercise asked of these patients was well within their capacity and pain was not induced in any instance. Nothing was found which would explain the finding that their response to exercise tended to exceed the normal, rather than the reverse. Apparently therefore, some cases with demonstrated organic heart disease respond to mild exercise by an abnormal increase in cardiac function, an increase of magnitude, of duration, or of both.

Apparently we are dealing with a real phenomenon which at first thought seems strange, as one would certainly expect a diseased organ to respond to a constant stimulus less actively than a normal one. However, evidence is accumulating that this expectation is by no means always realized. Thus digitalis,\textsuperscript{16-18} theophylline ethylene diamine,\textsuperscript{18-19} and hypertonic glucose injected intravenously\textsuperscript{20, 21} all cause a greater increase in cardiac output, or an increase of longer duration, or both, in persons with diseased hearts than in normal persons. So perhaps one should not be surprised that the same is true of mild exercise; the diseased heart being frequently over-responsive to stimulation and so less well adjusted to the demands which may be put upon it. And this finding is in support of the old clinical conception that “functional” heart disease is a common complication of organic heart disease.

Equally puzzling is the finding, in 4 patients, that ballistocardiograms abnormal in form during rest either improved or became completely normal in form after exercise. Probably this finding is comparable to that described by Starr and Mayock\textsuperscript{2} in which certain patients, giving ballistocardiograms abnormal in form while they lay at rest, gave a more normal record after they arose and stood upright. It is hard to think of a satisfactory explanation for this finding. It has been suggested that in an early stage of myocardial disease, cardiac weakness might be most apparent when the heart is under no compulsion and so is “taking it easy”; when an increased circulation is demanded, the cardiac reserves of strength are called out and the deficiency overcome.

Mention has been made of the result obtained in the one healthy subject whose circula-
tion, which at rest was at or above the upper normal limit, increased but little after exercise, making him abnormal when judged by the standards in table 2 because the percentage increase was so small. In this case the absolute amount of the circulation after exercise did not differ from that of most normal subjects, so perhaps no increase was needed. This observation stimulated us to study the relation between the absolute amount of the circulation at rest and the percentage increase after exercise. Using all our data (98 cases), there was significant negative correlation between these two values \((r = 0.32)\) so that persons with low resting cardiac outputs can be expected to have a greater increase after exercise than those with high resting values. However, when (in an attempt to improve our normal standards) we studied the same relationships for the healthy persons only, the correlation was not significant.

Evidence for a relationship between ballistic abnormalities and coronary heart disease continues to accumulate rapidly. Three of my subjects, tested with the expectation that they belonged in the normal group, because they considered themselves to be healthy and because there were no abnormal clinical findings, were found to have ballistocardiograms which were abnormal in form both before and after exercise. One of these, a man of 62, developed angina pectoris three months after completion of the investigation. The others were both over the age of 55, university professors, and accustomed to indulge in ordinary activity without distress. In one other subject the tracing, normal at rest, became abnormal in form after exercise; he was 42, a coal miner by occupation, and gave no other evidence of cardiac disease.

An interesting case which illustrates the value of taking ballistocardiographic records after exercise is given by the following 10 year after-history.

Case Report. J. C., a 49 year old widow, was admitted to the University Hospital in March, 1939, for investigation of her gall bladder function following an attack of jaundice lasting three months. She had no complaints suggestive of cardiovascular disease at this time, the heart being reported as clinically normal and blood pressure 130/80. X-ray films showed a normal cardiac contour. The electrocardiogram, however, showed low voltage T waves in all leads. A ballistocardiogram taken after exercise at this time (Fig. 1 B) showed that although the tracing was normal at rest, after exercise slurring of the J waves became most noticeable. Her resting cardiac output at this time was \(-13\) per cent. When the tracings were reviewed in 1949 it was decided to attempt a follow-up study and for this purpose the patient was readmitted to the University Hospital in March of that year. On this occasion she gave a history of angina pectoris always precipitated by exertion and relieved by rest. These attacks have been present since 1943. Clinical examination revealed no significant abnormality in any system and the blood pressure was 120/65. A further exercise test (Fig. 1 C) was carried out which again showed a normal resting ballistocardiogram which still became abnormal with slurring of the J waves after exercise. The resting cardiac output was \(+5\%\).

The case of J. C. reported above is the first instance of a patient with a ballistocardiogram normal at rest but abnormal after exercise, who has developed coronary heart disease in the years which followed.

**Summary and Conclusions**

1. Results of the analysis of ballistocardiograms taken before and at intervals after mild exercise in 41 normal subjects and 57 patients, many with various types of heart disease, are presented.

2. Normal standards of cardiac output response after exercise are defined and applied to the results obtained in patients with heart disease.

3. Twenty-eight subjects were found to have abnormal ballistocardiograms at rest. An additional 23, whose ballistocardiograms at rest were normal, gave abnormal tracings after exercise; in a few cases the reverse was true.

4. Patients were encountered who failed to respond to the exercise by increasing their cardiac output, and others showed excessive response to the exercise.

5. It is considered that ballistocardiograms after exercise give additional information on myocardial function which is not obtained by other clinical methods or by the ballistocardiogram at rest.
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