Twenty-Year Studies with the Ballistocardiograph

The Relation between the Amplitude of the First Record of "Healthy" Adults and Eventual Mortality and Morbidity from Heart Disease

By Isaac Starr, M.D., and Francis C. Wood, M.D.

The long-term follow-up of persons in whom one is interested, while always difficult to carry out, provides information of a type not duplicated by any other method of clinical investigation. This presentation relates the amplitude of ballistocardiograms to what we have called the physiologic age of the heart, and we have studied the relationship between such estimates of cardiac age, made about 20 years ago, and the after-histories of the subjects up to the present time, making use of the ordinary clinical criteria to judge the presence or absence of heart disease.

The large series of persons studied on our ballistocardiographs since our first instrument was constructed in 1936 can be divided into two main groups: patients attending the hospital because they were ill, and healthy persons who came to be tested at our request, chiefly to provide us with normal standards. This paper is concerned solely with the group who were healthy when first tested. Our long-term studies on the patients will be reported later.

Each healthy subject entered the study when his first ballistocardiogram was taken; this was in 1936 for a few, in 1937, 1938, and 1939 for the majority. A few others, chiefly medical students, entered the series from 1940 to 1943. The study was closed in 1959. The mean duration of the follow-up period is 20.5 years, the median is 21 years, and the range is from 23 to 17 years.

Work on the normal standards had hardly been completed when the onset of war disrupted our plans and research activities related to immediate needs took precedence over all others. That something of interest was happening in the "normal standard" group became apparent after the war was over, so the data were studied and the first report followed. In a second study 80 persons were brought back for retesting with the ballistocardiograph, approximately 10 years after the first test. Preliminary reports of our recent experience have also been made.

The present study is based not only on the further after-histories of those who were studied 10 years ago, but also on the after-histories of many others not included in the previous reports. In this study we aimed at securing data from 200 healthy persons who could be followed for a long period of time after their first ballistocardiograms, and we slightly exceeded our aim; the number finally attained was 211. We would have liked to keep the sexes even, but we found it was much easier to secure men, mostly doctors and medical students who worked in the hospital, than women; and in the completed series, the men outnumber the women by a ratio of about 5 to 1.

During the long studies, our series divided itself into two groups, those who remained in or near Philadelphia and whom we continued to see almost daily, and those who moved away with whom we maintained contact by mail and by seeing them occasionally.

This communication is concerned with findings related to groups of our subjects. Discussion of the many interesting things that happened to individuals during the 20-year period will be reserved for later communications.
Methods and Sources of Information

The Group Studied

All our subjects belonged to the white race. Their ages, when the first ballistocardiogram was taken, ranged from 22 to 85 years. The series consisted of 174 men and 37 women. At the time of the first test, 112 were doctors, of either medicine or philosophy, and 64 were medical students. The others were drawn from established employees of the medical school or hospital and from the friends and family of the first author.

Ballistocardiograms

The original ballistocardiograms were all taken on our first high-frequency table,7 the subjects being in the horizontal position with their heels in firm contact with the footboard. All these records were taken after a rest period of at least 15 minutes lying on the apparatus. Blood pressure was taken during this rest period. No record was taken less than 2 hours after a meal.

The follow-up ballistocardiograms were taken by the same technic on the same original instrument. In addition records were secured on both modern high-frequency and ultra-low frequency instruments.

Other Information

With those that remained in or near Philadelphia, our contact could hardly have been bettered. In this group are the 65 surviving men and 23 surviving women who have recently been given a complete cardiovascular study, the majority in Dr. Wood’s office, a few in the hospital because they were ill. Also in this group can be placed the 30 men and four women who died during the period of observation after a careful study either in the University Hospital or in some other hospital in whose staff we have confidence. Therefore, this group, which totaled 122 persons, will be called the “maximum-contact” group. In these persons our opportunities for observing the development of disease could hardly have been bettered.

Our contact with the rest of our subjects has also been close, although we have not had the opportunity of making a complete cardiovascular study recently, and have had to rely for our recent information on occasional interviews and on a questionnaire sent by mail. All the men of this “lesser-contact group” were doctors well acquainted with the nature of the study and its importance. Our letter inquiring about their present health was answered by all but three persons; the possibility that these three might have failed to respond because they were dead was rendered unlikely by finding that they were still on the active list in the files of our alumni association, and listed as in practice by the medical directory.

Considering both groups together, the results of thorough cardiovascular studies are available to us in all but 15 of the 211 subjects. All but three of these 15 are doctors, now in good health, who have promised to return for reexamination, but who have not yet done so.

In both these groups, we know with certainty whether each subject is alive or dead. Nor do we think that there is much danger of our being seriously mistaken in determining the presence or absence of serious heart disease in any case, but in doing the statistical analysis of this aspect of our results, we have given most attention to the maximum-contact group of 122 persons. To add the data secured from the remainder causes no change in our conclusions.

Our Subjects’ Health at the Time of the First Test

No one with a history of rheumatic fever was admitted to the series. All were actively working at the time of the first test and considered themselves to be in good health. Each, when asked whether he knew of any abnormality that would disqualify him from admission to a series of persons normal from a cardiovascular viewpoint, answered, “No,” with the few reservations to be mentioned below.

Thus we have two cases of arrested tuberculosis in the series, two of mild diabetes, and one of mild pernicious anemia, asymptomatic on liver extract. In six of the older subjects, the blood pressures taken at the time of the original test were above the old normal standards of 150/100, but in only one could hypertension be diagnosed by the more modern standards of Master et al.,8 and in him only the systolic pressure exceeded the normal range.

Two older subjects had suffered from single episodes that might have been mild cardiac infarction; indeed, this was suspected by doctors attending them at the time. In our opinion, however, the evidence was inconclusive, and both survived the 20-year period in excellent health; so we saw no reason to exclude them.

One subject had had an acute episode of substernal pain and electrocardiographic changes suggestive of pericarditis. He recovered, and has had no further cardiac difficulty during the next 25 years. So we saw no reason to exclude him.

Two of our subjects had had a brief attack of atrial fibrillation some years before the first test. Two persons, examined frequently in the University Hospital during the study, died suddenly and unexpectedly at 67 and 75 years of age. One had had a loud systolic murmur for years, the other developed one before death; so aortic stenosis was suspected, and, if true, some abnormality may have been present when the original test was made.
Table 1

Classification of Final State of Health

<table>
<thead>
<tr>
<th>Group</th>
<th>Description</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Undoubted cardiac disease</td>
<td>Cardiac infarction</td>
</tr>
<tr>
<td></td>
<td>Persons thus classified exhibited one or more of these criteria</td>
<td>Congestive failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sudden death without hemiplegia or obvious noncardiac cause</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Angina pectoris</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Permanent atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Permanent atrial flutter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Physical signs typical of valvular heart disease or pericarditis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Permanent complete heart block</td>
</tr>
<tr>
<td>II</td>
<td>Doubtful cardiac status</td>
<td>Bundle-branch block</td>
</tr>
<tr>
<td></td>
<td>Persons thus classified lacked any of the criteria listed under group I and exhibited one of these criteria without other evidence of heart disease</td>
<td>A-V conduction defect</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Abnormal orthodiagram</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Doubtful ECG</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Suspicious but not diagnostic murmurs</td>
</tr>
<tr>
<td>III</td>
<td>Hypertension without evidence of cardiac disease</td>
<td>The standards of Master et al. were used.</td>
</tr>
<tr>
<td></td>
<td>Persons thus classified showed none of the criteria of group I and II</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>No evidence of heart disease or hypertension</td>
<td></td>
</tr>
</tbody>
</table>

In summary, the total information now at our disposal indicated that of our 211 healthy subjects one might have had aortic stenosis at the time of the original (1939) test, one may have had mild essential hypertension, two might have had a small coronary occlusion, and one a mild pericarditis before the first test. Of the remaining 206 it seems most unlikely that anyone had any cardiac abnormality, detectable by the clinical tests used routinely today, at the time the first ballistocardiogram was taken.

If these five persons, possibly not completely healthy when the first test was made, are omitted from the series, the conclusions drawn from the statistical analysis of the results are unchanged.

Inspection and Measurement of the Original Ballistocardiograms

Almost all the old records were available for inspection. For those that had disappeared we used the measurements and description in the old record book. Measurement of the vertical distance between the tips of the recorded I and J waves gave the maximum amplitude of each complex. The sum of the amplitudes of typical large and small complexes of the respiratory cycle, which we call \( I + J + I_2 + J_2 \) defines the amplitude of any record, and we have used this value in the statistical analysis. This value divided by two gives the average amplitude of the ballistocardiogram that appears in the figures. These values are recorded in the tables in millimeters, but they can also be expressed in units of force. Our instrument was kept adjusted so that a deflection of 10 mm. corresponded to 250 Gm. of force, and the relation between force and deflection was linear.

Precautions to Avoid Bias in Analyzing the Results

We doubt if bias can be altogether avoided in any study, but one should take what steps he can to minimize it. To this end the cardiovascular status of the subjects who became ill was decided by Dr. Wood, at a time when he had no knowledge of the ballistocardiograms.

Data concerning the original ballistocardiograms were assembled and analyzed by Dr. Starr. It was impossible to do this without knowledge of the present health of the persons concerned. But the great majority of the records, all except some of those taken on certain medical students during class demonstrations, had been measured many years ago within 24 hours of taking them. So in this study we decided to use the measurements made many years ago rather than to make new measurements that might be biased by knowledge of the outcome.

Accordingly, using the criteria given in table 1, Dr. Wood divided the subjects into the four subgroups shown in that table, in accordance with the changes in their health, if any, that took place during the long study. This classification of the subjects from their after-histories, the age at which each entered the study, the amplitude of the original ballistocardiograms, and the duration.
TWENTY-YEAR STUDIES WITH THE BALLISTOCARDIOGRAPH

Mortality

In figure 1, the mortality of the whole group is compared with the expected mortality of a group of similar age and race residing in Pennsylvania. For the calculation of the "life table," which permits this comparison, we are indebted to Dr. Stanley Schor, of the Department of Public Health and Preventive Medicine, who made use of the age-sex-race adjusted Pennsylvania State Life Table 19. This figure shows clearly that the mortality in our group was always less than that to be expected; indeed, the difference is impressive.

The males of this group were next divided into three subgroups approximately equal in size, comprising those with large, medium, and small ballistocardiograms. For each of these three subgroups, the expected mortality was calculated, this time by Dr. Schild, again using the Pennsylvania State Life Tables. In figure 2, this expected mortality can be compared with the actual mortality for each subgroup of our series. As in the series as a whole, there was less mortality than the expected in each of the subgroups studied.

Inspection of the data given in tables 2 and 4, shows that those dying of all causes tended to have small ballistocardiograms when first tested about 20 years ago. Seventy-three per cent of men with original records smaller than 8.5 mm. have died during the period of observation; only 4 per cent of men with records larger than this died during the study. If the

of the follow-up are to be found in tables 2, 3, 4, and 5. Table 4, containing the data on those who died without developing heart disease, and the very long table 5, the data of those who remained healthy, have been filed with the Library of Congress from which they can be secured by anyone interested.*

Results

*Mortality

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*Table 4, containing data on those dying of causes other than heart disease, and table 5, data on those remaining in good health, have been deposited as document no. 6564 with the ADF Auxiliary Publications Project, Photoduplication Service, Library of Congress, Washington 25, D.C. A copy may be received by remitting with the order $1.25 for photoprints or $1.25 for 35 mm. microfilm. Make checks or money orders payable to: Chief, Photoduplication Service, Library of Congress.

Circulation, Volume XXIII, May 1961
### Table 2

**Summary of Those Who Developed Undoubted Heart Disease**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age at 1st ballooning (yr.)</th>
<th>Average ballooning amplitude (mm.)</th>
<th>Time of onset after 1st ballooning (yr.)</th>
<th>Time of death after 1st ballooning (yr.)</th>
<th>Pertinent clinical findings and diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MEN</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D. G.</td>
<td>63</td>
<td>3.5</td>
<td>6</td>
<td>11</td>
<td>Recovered from first attack coronary occlusion; died 5 years later, of another</td>
</tr>
<tr>
<td>X. L.</td>
<td>46</td>
<td>4</td>
<td>7</td>
<td>7</td>
<td>Dropped dead at wheel of his auto</td>
</tr>
<tr>
<td>X. E.</td>
<td>63</td>
<td>4</td>
<td>3</td>
<td>18</td>
<td>Angina pectoris 15 years; died of coronary occlusion, necropsy</td>
</tr>
<tr>
<td>H. B.</td>
<td>59</td>
<td>4.5</td>
<td>2</td>
<td>5</td>
<td>Typical coronary occlusion followed by angina pectoris</td>
</tr>
<tr>
<td>G. E.</td>
<td>74</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>Angina pectoris for 1 year; died of coronary occlusion</td>
</tr>
<tr>
<td>G. Y.</td>
<td>58</td>
<td>5.5</td>
<td>17</td>
<td>17</td>
<td>Coronary occlusion, died in first attack, necropsy</td>
</tr>
<tr>
<td>X. E. V.</td>
<td>51</td>
<td>6</td>
<td>12</td>
<td>22</td>
<td>Coronary occlusion, angina for years, hypertension, atrial fibrillation; dropped dead sitting in chair</td>
</tr>
<tr>
<td>I. G.</td>
<td>57</td>
<td>6</td>
<td>5</td>
<td>5</td>
<td>Developed angina pectoris, later pneumonia with empyema, died suddenly during convalescence; necropsy showed advanced coronary arteriosclerosis and recent infarct of lateral wall of left ventricle</td>
</tr>
<tr>
<td>C. O.</td>
<td>55</td>
<td>6</td>
<td>4</td>
<td>4</td>
<td>Developed diabetes and hypertension; died of arteriosclerotic heart disease</td>
</tr>
<tr>
<td>D. N. S.</td>
<td>55</td>
<td>6</td>
<td>13</td>
<td>21</td>
<td>Developed murmur suggesting aortic stenosis, later dyspnea on exertion, relieved by digitalis; dropped dead in R. R. station</td>
</tr>
<tr>
<td>D. H. B.</td>
<td>85</td>
<td>6.5</td>
<td>2</td>
<td>7</td>
<td>Basal systolic murmur, enlarged heart, abnormal electrocardiogram; diagnosed arteriosclerotic heart disease</td>
</tr>
<tr>
<td>S. S.</td>
<td>63</td>
<td>6.5</td>
<td>8</td>
<td>13</td>
<td>Developed hypertension followed by repeated bouts of congestive failure</td>
</tr>
<tr>
<td>K. J. D.</td>
<td>53</td>
<td>6.5</td>
<td>7</td>
<td>15</td>
<td>Developed loud systolic murmur at apex and base; dropped dead on street</td>
</tr>
<tr>
<td>F. G.</td>
<td>59</td>
<td>6.5</td>
<td>2</td>
<td>12</td>
<td>Typical severe coronary occlusion followed by 2 minor attacks</td>
</tr>
<tr>
<td>K. K. T.</td>
<td>44</td>
<td>6.5</td>
<td>7</td>
<td></td>
<td>Developed angina pectoris, exact onset difficult to date, working actively</td>
</tr>
<tr>
<td>F. E.</td>
<td>69</td>
<td>7</td>
<td>3</td>
<td>4</td>
<td>Developed angina pectoris; attacks of dyspnea; killed by a fall</td>
</tr>
<tr>
<td>T. I.</td>
<td>51</td>
<td>7</td>
<td>11</td>
<td>14</td>
<td>Severe attack of coronary occlusion, later congestive failure; necropsy</td>
</tr>
<tr>
<td>D. H. A.</td>
<td>63</td>
<td>7.5</td>
<td>3</td>
<td>13</td>
<td>Had 4 mild attacks of coronary occlusion</td>
</tr>
<tr>
<td>I. E. E.</td>
<td>62</td>
<td>8</td>
<td>4</td>
<td>7</td>
<td>Repeated attacks of pulmonary edema; dropped dead</td>
</tr>
<tr>
<td>N. E.</td>
<td>65</td>
<td>8</td>
<td>7</td>
<td>7</td>
<td>Acute coronary occlusion, heart ruptured; necropsy</td>
</tr>
<tr>
<td>C. N.</td>
<td>48</td>
<td>8</td>
<td>3</td>
<td>17</td>
<td>Acute coronary occlusion followed by angina; necropsy</td>
</tr>
<tr>
<td>D. N.</td>
<td>63</td>
<td>8.5</td>
<td>7</td>
<td>10</td>
<td>Developed hypertension and congestive failure</td>
</tr>
<tr>
<td>H. P.</td>
<td>44</td>
<td>8.5</td>
<td>13</td>
<td></td>
<td>Typical acute coronary occlusion 13 years after 1st ballisto, good recovery</td>
</tr>
<tr>
<td>K. V.</td>
<td>53</td>
<td>10</td>
<td>6</td>
<td></td>
<td>Developed angina pectoris, rare attacks atrial fibrillation, Parkinson's disease</td>
</tr>
<tr>
<td>M. L.</td>
<td>50</td>
<td>11</td>
<td>16</td>
<td>21</td>
<td>Developed loud systolic murmur, later atrial fibrillation and repeated attacks of congestive failure; necropsy</td>
</tr>
</tbody>
</table>
tension without apparent impairment in their hearts; these are marked in Table 5.

Figures 3 and 4 show the relation between the size of the initial ballistocardiogram and the development of undoubted heart disease, a doubtful cardiac status and hypertension in the men and women of our group. These figures show the status at the time the period of observation was terminated, in March 1959, without regard to the differences in duration of the periods of observation. From this arrangement of our data, those dying without developing heart disease (Table 4) have been omitted. Obviously heart disease developed with far greater frequency among those with small than those with large ballistocardiograms during the long period of observation, and this difference is highly significant.

Figure 5 illustrates the development of undoubted heart disease in the maximum-contact group. In this figure the data were arranged in order of the size of their initial ballistocardiograms in column A, the dots representing those that developed heart disease; the circles, those that remained healthy in the next 5 years. In column B, we have recorded similarly the onset of undoubted heart disease during the period of the sixth to eleventh years of observation. At the onset of that period, the subject, being 5 years older, would have been expected to have smaller ballistocardiograms for that reason. The expected diminution in amplitude during this period has been calculated from regression equation "a" of Table 7, and the symbols for each subject have been moved downward, a distance corresponding to 1.5 mm. on the scale on the left. Column C shows the incidence of onset of undoubted heart disease from the twelfth to the seventeenth years, similarly adjusted for the expected diminution of ballistocardiogram amplitude with age.
Life expectancy for the 174 men in the series in comparison with the number who actually died during the 17 years of observation. The men have been divided into three groups of approximately equal size. A, those with large ballistocardiograms at the initial test; B, those with middle-sized records; C, those with small records.

What happened in the first 5 years of exposure is very striking. Of those with original ballistocardiograms below 8.5 mm. in average amplitude, 38 per cent developed undoubted heart disease during this period. In contrast, no one with a ballistocardiogram larger than 8.5 mm. developed any cardiac abnormality during the first 5 years; a highly significant difference. Later, presumably accompanied by a diminution of their ballistocardiograms as their age advanced, other cases of heart disease developed in those whose records were expected to fall below this level.

The results secured in the women shown in figure 6 are similar. Again there is one exception to the usual finding.

**Discussion**

Our subjects, all followed for 17 years and many for longer, had a mortality in every year of the study smaller than that to be expected from the actuarial tables of life expectancy. Indeed, at the end of the 17-year period of exposure common to all, the deaths numbered less than two thirds of those expected. It was indeed an unusually healthy group that provided the subjects for this study.

As is so generally the case with healthy persons, the form of the initial ballistocardiograms of our group was normal with few exceptions. So no information concerning the significance of abnormalities of ballistocardiographic form, the abnormality so commonly found in patients, can be derived from this study. The significance of abnormalities of form, touched on in an earlier paper, has been studied again and will be reported in a paper to follow this, which will be concerned with the after-histories of the patients we have been able to follow for long periods.

In this laboratory, our records have always been calibrated, so that we can determine not only abnormalities for form, but also abnormalities of amplitude. The first reflects chiefly the coordination of the cardiac contraction; the second, chiefly its strength. So we have arranged our subjects according to the strength of the heart's beating, in order to compare each subject's position in the arrangement with his after-history.

During the 23 years of the study, however, there has been a marked improvement in ballistocardiographic instrumentation. Our original instrument, the best of its kind for almost 20 years, is now outdated. Yet it seems evident that this has no effect on the conclusions we are prepared to draw from its records. To permit a comparison between the old and new records, ballistocardiograms taken within 15 minutes of one another have been secured on a large number of persons by (a) our original instrument, (b) our modern high-frequency instrument, and (c) our ultra-low frequency instrument. All these records were calibrated in terms of force. Statistical analysis of 50 consecutive tests of this kind demonstrates that the over-all amplitude of records secured on our old instrument is significantly larger than that of either of our more modern instruments. But, while the absolute values are significantly different, the correlation between amplitudes of ballistocardiograms secured on the same subjects by old and more modern methods is very strong indeed; thus, when one compares the old and the modern high-frequency records, \( r = 0.91 \); comparing...
the old high-frequency and the modern ultra-low frequency records, \( r = 0.86 \), the two coefficients not being significantly different. The regression equations are given in table 6. These highly significant correlations are almost identical with the test-retest correlation, \( r = 0.905 \), secured by Tanner\(^6\) for our old high-frequency instrument. Therefore, while the numerical values for amplitude given in the figures will not apply exactly to results secured by a modern method, there is every reason to believe that, if our original records had been secured by any good modern method, the persons of our series, if arranged in order of their record’s amplitude, would be in essentially the same order as is shown in our figures. So, our conclusions, based on this order and not on absolute values, should be found valid for records obtained by modern instruments.

In any study of the significance of such an arrangement, the women and men should be treated separately, for women have ballistocardiograms smaller than men, not because they are smaller in size, but because they are women.\(^{10-12}\)

As soon as the men and women of our series are arranged in order of the average amplitude of their records, one important fact becomes obvious immediately: there is a very strong relationship between the ages of the subjects and the amplitudes of their ballistocardiograms. This has long been known,\(^4, 10, 11\) but we now have additional information. The facts about this important relationship and their significance must now be discussed in detail.

**Age and the Amplitude of Ballistocardiograms**

It is a well-known axiom of mathematical logic that two quantities, each equal to a third, are equal to one another. One can also say with confidence that two series of measurements, each positively correlated with a third series, are probably correlated with one another. For this mathematical reason alone, since the incidence of coronary heart disease increases with age, it is to be expected that any series of measurements found to be strongly correlated with the later development of coronary heart disease, will also be found to be correlated with age. Indeed, if any plan proposed for the prediction of heart

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**Table 3**

**Summary of Those Whose Cardiac Status Could Not Be Passed As Altogether Normal. The Group Called “Cardiac Status Doubtful”**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age at 1st ballisto (yr.)</th>
<th>Average amplitude at 1st ballisto (10 mm.=280 Gm.) (mm.)</th>
<th>Pertinent clinical findings and diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>N. O.</td>
<td>49</td>
<td>5.5</td>
<td>Developed hypertension, occasional paroxysms of tachycardia; exercise tolerance good</td>
</tr>
<tr>
<td>P. J. S.</td>
<td>53</td>
<td>8.5</td>
<td>Attacks of atrial fibrillation for 20 years, has developed gout and slight hypertension; good exercise tolerance</td>
</tr>
<tr>
<td>X. D.</td>
<td>30</td>
<td>10</td>
<td>Right bundle-branch block developed between 1951 and 1957; no history of onset, no other evidence of heart disease; extremely obese</td>
</tr>
<tr>
<td>F. F. E.</td>
<td>60</td>
<td>10.5</td>
<td>Left bundle-branch block, brief attack of atrial fibrillation. Exercise tolerance good for his years; heart normal size</td>
</tr>
<tr>
<td>F. D. N.</td>
<td>54</td>
<td>11</td>
<td>Developed systolic apical murmur and slight cardiac enlargement; exercise tolerance good</td>
</tr>
<tr>
<td>F. R. S.</td>
<td>43</td>
<td>11</td>
<td>Right bundle-branch block, developed between 1951 and 1958; no symptoms nor other evidence of cardiac disease; exercise tolerance excellent</td>
</tr>
<tr>
<td>N. G.</td>
<td>39</td>
<td>11</td>
<td>Labile hypertension, enlarged cardiac silhouette, normal exercise tolerance</td>
</tr>
</tbody>
</table>

*All had a complete cardiovascular examination including electrocardiogram and fluoroscopy that was negative except for the findings mentioned.*
The disease did not provide data that correlated with age, the plan might properly be regarded with suspicion.

The amplitudes of ballistocardiograms, strongly correlated with the later development of heart disease in our data, are also strongly correlated with the ages of our subjects. The regression equations relating these amplitudes to the ages of the 174 men in our series were calculated for us by Dr. Schild, and the results are given in table 7. The correlation coefficient is 0.74, values of 0.25 being significant for $p = 0.01$. Therefore, the possibility that chance might account for this association is altogether negligible. Young people tend to have ballistocardiograms conspicuously larger than those of old people.

Both previous studies and inspection of the dot diagram itself, however, suggested that the regression might be slightly curved. Accordingly, we tested the relation between age and the square root of the amplitude of the ballistocardiograms. In our 174 male subjects $r = 0.77$, a value a little higher, but, tested by Fisher's $z$ transformation, not significantly different from the previous correlation.

Some of the 174 males in our series, however, developed heart disease during the study, and in these our data indicate that some abnormality was present when the initial ballistocardiogram was made.

So Dr. Schild was also asked to calculate regressions for those of the maximum contact group who lived for 17 years after their first ballistocardiograms without developing any form of heart disease. These results are given as equations a and b in table 8 and the corresponding dot diagrams are in figures 7 and 8. Any possibility of incipient heart disease having been eliminated by the long follow-up, we regard these regressions as providing the best definitions of relations between age and ballistocardiogram amplitude in healthy persons, and we find them of great interest.

Our findings can also be compared with information about the decline of other physiologic functions as age advances. Figure 9 is based in part on a figure given by Strehler and Mildvan, which illustrates the decline with age of basal metabolic rate, nerve conduction velocity, maximal breathing capacity, standard glomerular filtration rate, and cardiac output per minute per kilo of body.

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**AV. BALLISTOCARDIOGRAM AMPLITUDE**

![Figure 3](image)

*Relation of the size of the original ballistocardiogram to the development of undoubted heart disease, a doubtful cardiac status or hypertension diagnosed according to the criteria given in table 1. The exact diagnosis of each case who developed heart disease can be found in table 2 or 3. This figure shows the status of the group of men as of March 1959, when the series was closed. The eight persons dying from causes other than heart disease are omitted from this figure. Circles, no heart disease developed; dots, those who developed undoubted heart disease, crosses superimposed indicate that they died from it; horizontal bars, those who developed a doubtful cardiac status; vertical bars, those who developed hypertension without evidence of heart disease.*
TWENTY-YEAR STUDIES WITH THE BALLISTOCARDIOGRAPH

AV. BALLISTO AMPLITUDE

![Graph showing AV. BALLISTO AMPLITUDE](image)

**Figure 4**
Relation of the size of the original ballistocardiogram to the development of heart disease in women. Status of the group in March 1959. Those dying from other causes than heart disease have been omitted. The period of observation ranged from 17 to 23 years. Symbols as in figure 3.

weight. These authors express the decline of these functions with age by the per cent of the reserve capacity remaining at different ages. The relation between age and ballistocardiogram amplitude, given by equation a, table 7, can be expressed the same way, and this has been shown as the line AB in figure 9, where it lies in the midst of other measurements. So our findings for the heart are in close agreement with what is known about the aging of other bodily functions. According to our data, the reserve capacity of the heart of healthy persons diminishes at a rate of 1.4 per cent per year. Strehler and Mildvan's estimates of the rate of loss of the other human physiologic reserve capacities with age, range from 0.5 to 1.3 per cent per year. So the heart seems to age a little faster than the other organs about which we have this inform-

**Figure 5**
Relation between age and the development of undoubted heart disease in men of the maximum contact group. The adjustment for advancing age is explained in the text. Column A. Dots = those developing undoubted heart disease within 5 years after entering the study. Circles = those living during the first 5 years of the study without developing any type of heart disease. Column B. Dots = those developing undoubted heart disease within the period between 6 and 11 years after they entered the study. Circles = those living 11 years without developing any type of heart disease. Column C. Similar data for the period 12 to 17 years. As one passes from A to B and from B to C, the total number diminishes as the data of persons dying without developing heart disease are withdrawn.
in older persons, death is attributed to heart disease more frequently than to disease of any other organ.

On the Physiologic Age of the Heart

It is a truism that some grow old before their time, as if the aging process progressed unduly rapidly. The conception can also be applied to the organs and the idea of the unduly aged heart is a familiar one to the medical profession. But we know of no previous attempt to give exact expression to the heart’s physiologic age.

When the amplitude of the ballistocardiogram is known, physiologic cardiac age can be calculated for each of our male subjects from equations g or h of table 8, which were derived by solving regressions a and b of that table, for age. The two equations give very similar, but not identical estimates of physiologic cardiac age, and we do not know which is the more useful. We have not calculated similar regressions for the women of our series because of their small numbers, but similar equations calculated from other data might be used. For records secured by any modern instrument, an equation to give the physiologic age of the heart of any subject could be derived by combining equation g or h of table 8 with the equation of table 6, which pertains to the type of modern instrument being used.

As far as the statistical analysis of our data is concerned, it makes no difference to the conclusions whether or not we use the amplitude of our record directly or after converting it into physiologic age. However, knowledge of the physiologic age of the heart of each subject permits us to explore the possible advantage of using physiologic age, instead of chronologic age, to enter standard life expectancy tables.

Studies on the Relation between Chronologic Age and Physiologic Cardiac Age as Judged by Ballistocardiogram Amplitude

In the upper half of figure 7 is the dot diagram showing the relation between age and average ballistocardiogram amplitude for the men of the maximum contact group who remained healthy, and its equation is given, in slightly different form, in table 8. This same regression is placed in the lower half of figure 7 together with the dot diagram and regression of those who developed undoubted heart disease. Obviously, the dots representing those who developed heart disease do not conform to the regression of those who remained in health. In figure 8, similar to figure 7 except that age is related to the square root of amplitude, the distinction between the “normals” and the “cardiacs” is even clearer.

Inspection of figures 7 and 8 shows that the “cardiacs” and “normals” do not cover...
the same age range, and a certain advantage is gained, though something is also lost, by restricting the comparison to the age range both have in common, 43 to 65 years. The regression equations of the normal subjects within this range are also given in table 8 for this reason.

That the cardiac group is significantly different from the normal group can now be demonstrated in two ways. First, the slope of the regressions of the cardiac group, equation e of table 8, is found to be significantly different from those of the regressions of the whole normal group, equation a, (t = 3.6); and also from that of the restricted normal group, equation c, (t = 5.3).

Second, from the age of each subject and from equation c of table 8, an expected ballistocardiogram amplitude can be calculated for each "cardiac" subject in the common age range from the data secured on those who remained healthy. This expected amplitude was paired with the amplitude found for that subject, and the difference determined. The ballistocardiogram amplitude found for the 24 "cardiacs" in the common age range averaged 4.8 mm. smaller than the expected, for this difference t = 4.24, so the difference is highly significant.

We can now go further and classify the cardiac subjects according to the time required to develop heart disease. The eight who developed cardiac disease within 5 years gave initial ballistocardiograms that should have averaged 32 per cent larger than the value found, a significant difference, t = 3.05.

For eight who developed cardiac disease between 6 and 10 years, their ballistocardiograms should have averaged 38 per cent larger than was found, but because of greater scatter conventional significance was missed by a narrow margin, t = 1.77. Among the eight who developed cardiac disease between 11 and 17 years after the first test, their ballistocardiograms should have averaged 26 per cent larger than the values found, another significant difference, t = 2.09. It should be noted that a higher significance was found for those who developed heart disease soon than for those who developed it later, as one would expect.

Paired Experiments

We next sought to neutralize the effect of chronologic age by pairing the subjects with one another.

A cardiac group, all men and women, who developed undoubted heart disease within 17 years after the first test, and a control group, those of comparable age who lived 17 years after their first ballistocardiogram without developing either undoubted heart disease or a doubtful cardiac status, were arranged according to their age at their entrance into the study; as in table 9.

Inspection of table 9 shows that in pairing these data we had to make several choices and the problem was to make them without bias. Since inspection of table 9 seemed to indicate that the controls—those who did not develop heart disease—had larger ballistocardiograms than those of similar age who did, this hypothesis was set up and the
Table 8

Regression Equations Relating the Age of the Subjects of the Maximum-Contact Group and the Amplitude of Their Original Ballistocardiograms*

<table>
<thead>
<tr>
<th>Group studied</th>
<th>Equation number</th>
<th>Regression equations</th>
<th>Standard deviations of the data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>about its mean</td>
</tr>
<tr>
<td>Normal Group</td>
<td>a</td>
<td>[ B = 48.8 - 0.54 A ]</td>
<td>9.4</td>
</tr>
<tr>
<td>Those not developing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( n = 59 )</td>
<td>b</td>
<td>[ \sqrt{B} = 7.3 - 0.05 A ]</td>
<td>0.90</td>
</tr>
<tr>
<td>Those not developing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>aged 43-65 inc. ( n = 20 )</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac Group</td>
<td>e</td>
<td>[ B = 23.6 - 0.16 A ]</td>
<td>5.0</td>
</tr>
<tr>
<td>Those developing</td>
<td>f</td>
<td>[ \sqrt{B} = 4.9 - 0.02 A ]</td>
<td>0.62</td>
</tr>
<tr>
<td>undoubtedly heart</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>disease in 17 years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( n = 20 )</td>
<td>g</td>
<td>[ PA = 90.4 - 1.85 B ]</td>
<td></td>
</tr>
<tr>
<td>Those not developing</td>
<td>h</td>
<td>[ PA = 146 - 20 \sqrt{B} ]</td>
<td></td>
</tr>
<tr>
<td>developing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>heart disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>( n = 27 )</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*A = Chronologic age (years); P = Physiologic age (years); B = Ballistocardiogram amplitude (mm. when 10 mm. = 280 Gm.). As our measure of amplitude the sum of the amplitudes of I and J waves of typical small and large complexes of the respiratory cycle has been used.

choices made so that they would tend to defeat it.

Thus, in our first calculation, pairs were made according to the following rules. 1. For every cardiac case a control was sought of the same calendar age; if none was available, a control 1 year older was used; or, if none was available, a control 2 years older. 2. When there were two pairs of the same or comparable ages, which individual was paired with which was decided by tossing a coin. 3. When, within one age group, more than one cardiac had to be compared with only one control, or vice versa, an average value was used to pair with the single value. 4. No datum was used twice.

By means of these rules, 16 pairs of men were secured and one cardiac, I.E.E., had to be left without a mate. Similarly, two pairs of women were secured; and for one cardiac woman, F. E., no control could be found.

In each pair we now subtract the average ballistocardiogram amplitude of the cardiac subject from that of the control and so obtain a series of differences. The averages of these differences would approach zero if the cardiac group and the controls tended to have ballistocardiograms of similar amplitude. So we can now ask ourselves the question, is the mean of the differences we find significantly different from zero. For the 16 pairs of men, the mean difference in ballistocardiogram amplitude is 3.0 mm., the controls having the larger values; \( t = 3.07 \), so the likelihood of chance explaining the findings is negligible. If one adds the two pairs of women, the mean of the differences is 2.75 mm. in favor of the controls and \( t = 3.13 \).

Two more pairs can be secured by using two persons, F.E.E. and F.D.N., from the doubtful cardiac status group (table 3) as controls. This seems a reasonable thing to do for both subjects consider themselves to be in good health at present and both have an exercise tolerance excellent for their age of 76 and 80 years. This gives us 20 pairs, and
TWENTY-YEAR STUDIES WITH THE BALLISTOCARDIOGRAPH

the mean difference in ballistocardiogram amplitude is now 2.95 mm. in favor of the controls. This is a striking difference, after thus neutralizing the effect of age, the ballistocardiograms of the "cardiacs" still average 25 per cent smaller than those of controls. For these 20 pairs \( t = 3.64 \).

To assure ourselves that unconscious bias did not enter into the high significance of the results given above, we now, with conscious bias, selected our pairs in every way possible to defeat the hypothesis that, with age neutralized, the ballistocardiograms of the controls were larger than those of persons who would later develop heart disease. Wherever, in the previous study, we had paired the data of one control with the average of that of several cardiac subjects, we now paired the control with the cardiac subject with the largest ballistocardiogram, and did not use the data of the others at all. Conversely, when one cardiac person had been paired with several controls, the control with the smallest ballistocardiogram was now used, and the others were discarded. When thus weighted against the hypothesis with all the bias we could muster, the mean difference for the 16 pairs of men first studied was still 2.03 mm. in favor of the controls and \( t = 2.23 \), a value still significant.

So we must conclude that, with age neutralized, persons who will live 17 years without developing heart disease are still likely to have larger ballistocardiograms at the beginning of that period than those who will develop heart disease.

Pairs of another sort can also be made. Nine of those who developed undoubted heart disease during the period of observation have a ballistocardiogram amplitude that can be matched exactly with that of one or more subjects of the same sex who remained healthy. Three more pairs are secured by using members of the doubtful group as controls, an addition justified by the fact that their general health and well-being are altogether normal. However, as more than one control is usually available, a choice must be made and the difficulties, discussed before, apply.

Relation of amplitude of the initial ballistocardiogram to the development of undoubted heart disease in women of the maximum contact group. The adjustment of amplitude for columns B and C is explained in the text. Columns and dots as in figure 5.

By use of the average of all where more than one value is available, when ballistocardiogram amplitude and sex are thus paired out, those who developed undoubted heart disease average 15 years older than those who remained healthy, and \( t = 7.04 \), a striking finding. When one chooses one mate for each cardiac subject with maximum conscious bias against the hypothesis that the ages are different the cardiac group averages 7 years older, and \( t = 2.56 \), and this is still significant. So, there is no reasonable doubt that, in our series, when the amplitude of ballistocardiograms are equal, the older person has a greater likelihood of developing undoubted heart disease. This is further evidence that chronologic age and ballistocardiogram amplitudes, though strongly correlated, do not represent the same physiologic aspect of our subjects.

**Interquartile Ranges**

In column A of figure 5 the data from men developing heart disease within 5 years, and
from those who remained well for that period, are arranged according to the amplitude of their ballistocardiograms at the initial test. The interquartile range of the "cardiac" in this group is 30.3 per cent of that of the whole group. If the same data are arranged according to the chronologic ages of the subjects, the corresponding value is 43.5 per cent.

Corresponding values for those who developed cardiac disease from 6 to 11 years after the initial test, are, when arranged by ballistocardiogram amplitude, as in column B of figure 5, 28 per cent; when arranged by age, 67 per cent. For those who developed heart disease from 12 to 17 years after the initial test, when arranged by amplitude, as in column C of figure 5, this value is 44 per cent; when arranged by age, 61 per cent. The arrangement by amplitude of ballistocardiogram always results in a more compact group of those who are to develop heart disease than does the arrangement by age; so physiologic age, derived from this amplitude, should serve better than chronologic age to identify those who will become abnormal.

Other Statistical Studies

Extensive studies by biserial correlation, and by the chi-square technic supported, but did not add to the conclusions to be drawn from the studies already given; so these results will not be given in detail. The relation between ballistocardiogram amplitude and the later development of heart disease was always at least as strong as that with age, but there was often no simple way of testing for the significance of the difference.

The Ballistocardiogram Amplitude as an Indicator of Mortality

Diminishing conspicuously with age, ballistocardiogram amplitude is a successful predictor of mortality; the question is, does it do so better than age itself. Our attempt to answer this question has been limited, for the moment, to comparisons between the mortality found in our series and that expected from the life table, as is shown in figures 1 and 2. It is difficult to interpret these findings, for it must always be remembered that the life table indicates the mortality to be expected, not in healthy persons as in our series, but in the whole population, both sick and well. In our data, only for the period between 5 and 9 years does the mortality of those with small ballistocardiograms go below the value expected, and even here the difference is small; so it would appear that we were better at predicting morbidity than mortality. In another series, the excess mortality of cases with angina, that is the mortality in excess of that to be expected from their age, was only 7 per cent for men and 5 per cent for women, values, we believe, much less than most doctors would expect; and this is true in our cases also. The after-histories of our hospital patients, in a study now nearing completion, contain many more deaths than
TWENTY-YEAR STUDIES WITH THE BALLISTOCARDIOGRAPH

729

Figure 8

Relation of the square root of ballistocardiographic amplitude and chronologic age. The symbols and lines are similar to those of figure 7.

occurred in this series of healthy persons and give promise of throwing much more light on the relation of the ballistocardiograms to mortality. Further discussion will be deferred until these data can be analyzed.

Final Remarks Concerning the Physiologic and the Chronologic Age of the Heart and Its Relation to the Development of Coronary Heart Disease

So many interesting studies can be performed on a body of data as unique as this, that it is difficult to know where to stop either the statistical analysis or the discussion. For this reason the complete data have been filed in the Library of Congress so that anyone interested could secure them for further study. With several general comments we will bring this report of the present study to a close.

Inspection of figures 7 and 8 shows that, in sharp contrast to the results secured on those who remained in health, in the group who later developed cardiac disease the regression of ballistocardiographic amplitude with age is almost flat; indeed, the slope is not significantly different from zero. So in the cardiac group we have failed to demonstrate a significant relation between the cardiac forces, as measured by ballistocardiographic amplitude, and chronologic age. One wonders if there is not a level below which the cardiac forces cannot go, if the patient is to survive in health. Those that are to develop heart disease, whatever their chronologic age, are all so close to this lower limit that the normal relation between age and these forces has been lost.

Finally we must discuss the meaning of our results in terms of present knowledge of aging, and of the heart disease that so often develops as age advances. Our finding that ballistocardiograms of persons later to develop heart disease, are significantly smaller
than those of the controls, is consistent with the view that the disease process, which later advanced until it produced characteristic symptoms, was present at the time of the first test to a degree sufficient to weaken the cardiac forces, but insufficient to cause the typical picture of disease. We believe this is the interpretation that would occur first to most doctors, especially since the hypothesis is consistent with the recent evidence that coronary heart disease occurs more frequently in younger persons than was formerly supposed.

Nevertheless, while we cannot deny the possibility that incipient coronary arterial disease accounted for our findings in the "cardiac" cases, we are more inclined to interpret our results from another viewpoint which is in better accord with our findings in the healthy persons of our series. Here we must pause to emphasize the strength of our position. Our belief that these persons were indeed healthy at the time of our tests is based, not only on the usual clinical studies which, alas, are so often inadequate for the detection of incipient disease, but also on the long after-histories. In these cases we know that no manifest heart disease developed for 17 years or longer. We know of no other clinical study providing such strong evidence that those we classify as free of heart disease at the time of our test, were so in truth.

Despite their excellent cardiac health, the decline in the cardiac forces as age advances...
is very obvious in these subjects; evidence for it is seen both in the regressions reported here, and in the tests made repeatedly on individuals during the long course of the study. This decline with age is so nearly a universal phenomenon that it cannot be attributed to coronary heart disease, which, though found with increasing frequency as age advances, is by no means always present. So we prefer to think of this weakening of the "normal" heart with age as analogous to the well-known weakening of the peripheral muscles as age advances, a universal phenomenon certainly not to be attributed to scleroses of the supplying vessels. Perhaps chemical or physical in nature, the aging process is doubtless of infinite complexity, and it is not to be identified with any structural abnormality of which we are aware.

With such thoughts in mind we are not ready to attribute the unduly small ballistocardiograms of those who later developed coronary heart disease to the existence of coronary arteriosclerones at the time of the initial test. We are content with the thought that the hearts of some of our subjects aged more rapidly than those of others, and that, in the former, the heart diseases characteristic of advancing age developed with much greater frequency, as one would expect.

It was the school of James Hope, of MacKenzie, and of Lewis, that for so long emphasized the importance of cardiac muscle function in any consideration of heart disease and its disabilities. The data presented in this paper support the validity of that viewpoint.

Summary

A group of 211 healthy persons, gathered together from 23 to 17 years ago to provide normal standards for the ballistocardiograms, has been followed to the present time. This study is concerned with the group as a whole, or with subgroups. The study of individuals will appear in later communications.

With a few minor exceptions, the group, at the time of the initial test, had ballistocardiograms normal in form. The death rate of the group during the period of the study was much lower than was to be anticipated from standard life expectancy tables for the state of Pennsylvania.

Although the ballistocardiograms of this group were normal in form, there was great variation in the amplitude of the records. Since these records were calibrated in terms of force, this can be interpreted as due to differences in the force of the heart's contraction. Those whose hearts contracted with little force at the initial test later suffered from death and cardiac disability, chiefly coronary heart disease, in far greater numbers than those whose hearts contracted strongly.

The interpretation of this striking finding is bound up with that of another; in our data, as in that of others, there is strong correlation between the ages of the subjects and the amplitudes of their ballistocardiograms. The heart tends to weaken and beat with less coordination as it grows older.

Exact expressions can be given to the "normal" rate of decline of cardiac function with the years by the slope of the regression between age and ballistocardiogram amplitude found in the group of those who remained healthy for 17 years after the test. The normal rate of cardiac decline, as age advances, agrees closely with that calculated for several bodily functions. The cardiac decline seems a little more rapid than that of these other bodily functions, but the difference is small and its significance is doubtful.

In our data, among those who later developed undoubted heart disease, there was no significant tendency for the older people to have smaller ballistocardiograms than the younger.

From the chronologic age of each subject who later developed undoubted heart disease, and the age-amplitude regression of those who remained healthy, one can calculate for each "cardiac" subject, the amplitude expected if he had been in perfect health. On the average this expected value far exceeds the values found. So most of these hearts were performing abnormally for their age although manifest clinical evidence of heart disease had not yet appeared.
When the effect of age is eliminated by pairing the results directly, the relation between the amplitudes of the initial ballistocardiograms and the later development of heart disease remains significant for \( p = 0.05 \). Indeed, in such pairs with similar ages, those who later developed heart disease had initial ballistocardiograms that averaged 25 per cent smaller than those of their mates who remained healthy for the next 17 years. This is additional evidence that the hearts of those who were to develop myocardial disease were, at the initial test, like the hearts of much older normal people; that is, the physiologic age of their hearts far exceeded the chronologic age.

Acknowledgment
The great bulk of the statistical analysis was performed by Dr. Albert Schild. Dr. S. I. Askowitz computed the more elaborate chi squares. The first author did the remainder, and also provided an independent check on the more important computations.

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ISAAC STARR and FRANCIS C. WOOD

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