Ballistocardiographic Evaluation of the Cardiovascular Aging Process

By Arthur J. Moss, M.D.

Cardiovascular aging is a complex phenomenon associated with a gradual, yet progressive, deterioration in cardiovascular function.1-3 The etiology of this deterioration in function is poorly understood, but atherosclerosis and, in particular, coronary artery disease seem to be a factor.3 Cardiovascular aging and atherosclerosis appear to be universal phenomena, but their rate of development and the extent of myocardial involvement are variable. When there is accelerated cardiovascular aging, clinical disease develops at an unusually early age. Thus, coronary atherosclerosis is not limited to older age groups, and coronary artery disease has been demonstrated in a large number of young, healthy men aged 20 to 40.4-7

The clinical evaluation of the cardiovascular aging process has been difficult, for usually there are no signs or symptoms detectable until overt disease is present. The ballistocardiogram has been used for many years in the evaluation of cardiovascular function. This technic offers a unique means of studying the change in cardiovascular dynamics with age. Recently, Reeves et al.5,9 described an ultralow frequency acceleration ballistocardiograph that met all theoretic considerations of biophysical design.10-14 This ballistocardiograph was clinically adaptable, had high test-retest reliability, and revealed considerable new information about cardiovascular events. The purpose of the present study is to elucidate with the Reeves technic the changing ballistocardiographic pattern with advancing age in an overtly healthy male population, and thus to evaluate the cardiovascular aging process.

Methods and Material

Subjects

The subjects consisted of 307 overtly healthy men, aged 18 to 54, on active duty in the U.S. Navy; most were U.S. Navy pilots. The subjects were seen randomly as part of a required annual physical, and all had been screened for evidence of hypertension, rheumatic heart disease, and symptomatic cardiovascular disease. All subjects were considered to be in excellent health. The number of subjects in each of four arbitrary age groups (18 to 24, 25 to 34, 35 to 44, 45 to 54), the mean age of each group, and the average height and weight with the ranges observed are presented in Table 1. On each subject a ballistocardiogram and a standard 12-lead electrocardiogram were taken.

Apparatus

The ballistocardiograph (Fig. 1) was a modification15 of the ultra-low frequency model described by Reeves et al.8,9 The bed platform (13 lb.) was made of honeycomb aluminum* and was suspended from a wooden frame by three 36-inch long, 3/64-inch twisted seven-strand stainless-steel cables. A hydraulic lift system raised the bed platform and disengaged it from cable support when not in use. When freely suspended by the cables, the bed was free to move in head-foot and lateral directions. A single-action joint attached between the foot of the bed and the wooden frame allowed unrestricted head-foot motion, but limited the lateral motion to the head end of the table. In the head-foot and lateral directions the suspended bed had a natural frequency of 0.42 cycle per second, with a damping factor of 0.04. The lower end of the undistorted frequency response of the bed was 0.5 cycle per second. For details on this aspect of the apparatus the analysis of Burger should be consulted.16

In the head-foot axis only, the motion of the bed was sensed as acceleration by an electronic accelerometer.1 The accelerometer gave readings

*Honeycomb Corporation of America, Bridgeport, Connecticut.
TABLE 1  

Population Characteristics

<table>
<thead>
<tr>
<th>Age groups</th>
<th>Number of subjects</th>
<th>Mean age (yr.)</th>
<th>Height (in.) Mean</th>
<th>Range</th>
<th>Weight (lb.) Mean</th>
<th>Range</th>
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<td>81</td>
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<td>70.0</td>
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<td>45-54</td>
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<td>69.5</td>
<td>64.75</td>
<td>174.2</td>
<td>140-215</td>
</tr>
</tbody>
</table>

comparable to the electrokinetic device of Elliott et al. It had a flat frequency response up to 40 cycles per second, was adequately stable, and gave a satisfactory output with no phase shift. The accelerometer was attached to the underside of the bed at the approximate theoretic heart position, and the signal was fed into a strain-gage amplifier on a Sanborn Poly-Viso direct recorder. The polarity of the accelerometer was placed so that a headward force produced an upward deflection of the recording stylus and a footward force a downward deflection. The lateral motion of the bed was not recorded, for there were significant rotational and translational resonant body artifacts in this direction.14

The frequency response of the entire system in the head-foot axis was flat from 1 to 25 cycles per second, and usable to 40 cycles per second. To obtain this frequency response tight coupling of the body to the bed was necessary. This coupling was accomplished by use of a footboard, and by shoulder, chest, and waist straps.

Standardization of the ballistocardiograph was carried out daily. After the Sanborn Poly-Viso was allowed to warm up for a period of 30 minutes, a 100-pound loading weight was placed upon the bed platform. The bed was suspended, and the bridge balance of the strain-gage amplifier was balanced. A standardization pendulum (fig. 2) was placed at heart level on the 100-pound loaded bed with the ballistic pendulum free to move in the head-foot axis. The total weight of the pendulum and its housing was 4,400 Gm.; the effective weight of the ballistic part of the pendulum, including the arm weight, was 1,568 Gm. With the paper speed of the Sanborn Poly-Viso running at 5 mm. per second, and the strain-gage attenuation switch set at the "× 4" position, the ballistic pendulum was set free from an 11.5-degree angle, which the arm of the pendulum made with a vertical line passing through the central axis. The pendulum delivered approximately 286,000 dynes at each turning point of its swing. The sensitivity of the strain-gage amplifier was adjusted so that the total amplitude between the trough and peak of the tenth recorded swing was 21.5 mm. (fig. 2). The amplitude of the trough-to-peak deflection represented the force delivered by the pendulum at two consecutive turning points in its swing. Thus, a 21.5-mm. deflection was equivalent to about 572,000 dynes when the bed was loaded with 100 pounds. A calibration curve, which revealed the relationship between loading weight and amplitude of the ballistocardiographic deflection, indicated that there was a 35 per cent curvilinear diminution in the over-all acceleration amplitude as the weight on the bed was increased from 140 to 220 pounds. This 35 per cent diminution in amplitude is accurate to within 1.5 per cent of the theoretic calculated value for this 80-pound weight change.

Procedure

The actual recording of a ballistocardiogram on a subject was as follows. After approximately a 2-minute rest period, the subject was placed on the bed platform and coupled to the bed by the footboard and straps. Electrocardiographic leads were attached to the limbs, and an Infraton pulse-wave recorder was placed over the right carotid artery. The bed was suspended, and the strain-gage attenuation switch set at "× 1." The paper speed was run at 50 mm. per second, and a simultaneous recording of the electrocardiogram (lead II), head-foot ballistocardiogram, and carotid pulse was obtained during a 10-second period with respiration held in the mid phase. The total procedure for each subject lasted less than 5 minutes. A ballistocardiographic recording on a healthy 22-year-old subject is presented in figure 3.

Ballistocardiographic Analysis

The standard nomenclature18 for labeling the ballistocardiographic deflections was used throughout this paper. A labeled schematic diagram of an acceleratory ballistocardiogram and simultaneous electrocardiogram is shown in figure 4.

Measurements

An arbitrary baseline for each ballistocardiographic complex was assumed to lie along the horizontal line that passes through the top of the H wave. On each ballistocardiogram the fol-
Photograph of the ultra-low frequency acceleration ballistocardiograph. The bed platform is suspended from the wooden frame by three wires. The subject is coupled to the bed by the use of a footboard, and by shoulder, chest, and waist straps.

Following measurements were made on three consecutive complexes, and the average value was obtained:

1. HI angle—the acute angle that the slope of the ejection deflection (HI wave) made with a true vertical passing through the H point was measured to the nearest 0.5 degree.

2. HI, HJ, HK, amplitudes—the total vertical amplitudes from the H baseline to the I, J, and K points were measured to the nearest millimeter. These amplitudes were converted to absolute units of force in dynes.*

3. HK/HI ratio—the ratio was obtained by dividing the HK amplitude by the HI amplitude.

4. Q-H time—the time duration from the Q wave of the simultaneous electrocardiogram to the H point of the ballistocardiogram was measured to the nearest 0.01 second.

5. H-Jp, H-L times—the time durations from the H point to the peak of the J wave (Jp) and the L point were measured to the nearest 0.01 second.

Descriptions

There were a number of characteristics of the ballistocardiogram that did not lend themselves to precise measurement. When these characteristics were described, however, pertinent aspects of the ballistocardiogram were elucidated. The following groups of arbitrary criteria were set up to describe more completely the ballistocardiographic complex. In almost every case the three consecutive patterns analyzed were nearly identical.

1. Contour of the I-J-K complex—the over-all sharpness of the ballistocardiographic deflections at the I-J-K points was graded according to the following scale:

   Contour 1—the three components of a given complex were rounded with no sharp points at the changes in deflection.
   Contour 2—two components of a given complex were rounded, and one component had a relatively sharp change in deflection; the over-all pattern was more rounded than sharp.
   Contour 3—one component of a given complex was rounded, and two components had relatively sharp changes in deflection; the over-all pattern was more sharp than rounded.
   Contour 4—the three components of a complex had sharp changes in deflection, but not markedly so.
   Contour 5—all components of a given complex showed very sharp changes in deflection.

2. Ejection pattern—the HI ejection wave was described as being either straight, interrupted, concave, or convex in shape; notation was made when the I wave was flat, or if an I+ wave* was present.

3. J wave—the predominant shape of the J wave was described as monophasic, biphasic, triphasic, or serrated; notation was made when a Jd wave* was present.

4. Approximate amplitude of the LM, diastolic, and GH waves—the LM and GH amplitudes were obtained by measuring to the nearest millimeter the vertical distance occupied by these waves. The diastolic wave amplitude refers to the maximum vertical deflection in the sinusoidal wave pattern that follows the MN upstroke in each ballistocardiographic complex. The average amplitude of the LM, diastolic, and GH waves, obtained from the measurement of three consecutive patterns, was graded as follows: small—the average amplitude 3 mm. or less; medium—the average amplitude 3+ to 6 mm.; and large—the average amplitude greater than 6 mm.

The total analysis of a ballistocardiogram by this method consisted of seven quantitative measurements and six descriptive terms (fig. 4). The heart rate was calculated from the measured time interval between the first two of the three consecutive complexes analyzed in each tracing. The
analysis of an entire ballistocardiographic tracing required about three to five minutes. From these recorded data the ballistocardiographic pattern can be visualized, and a very accurate reproduction of the original ballistocardiogram can be constructed.

Electrocardiographic Analysis

Standard analysis of the electrocardiograms of the 307 subjects was made with the following measurements noted: P-R interval, QRS duration, frontal plane QRS and T axes, frontal plane QRS-T angle, and amplitude of S in V2 plus R in V5. An electrocardiogram was read as abnormal if (1) S-T depression of greater than 1 mm. was present in any lead; (2) generalized low-voltage T waves were present with flat to inverted T waves in leads I, V5, or V6; (3) left axis deviation beyond minus 60° was present; (4) a pathologic Q wave in any lead was seen. Thus, an electrocardiogram read as abnormal by these criteria strongly indicated coronary artery disease. In the series of subjects studied, no patterns of bundle-branch block were present.

Results

Change in the Ballistocardiographic Pattern with Age

The mean measurements, ranges, and standard deviations obtained from the analysis of the ballistocardiograms in 307 male subjects are presented by age groups in table 2. As one would expect, the heart rate increased slightly with age; it was 10 beats per minute faster in the oldest as compared with the youngest age group. The mean HI angle remained rather constant and there was no significant change with age. The mean HI and HJ forces (dynes) and the mean HK/HI ratio showed a progressive, almost linear, age-related change. The HI and HJ values decreased with age, whereas the HK/HI ratio increased (fig. 5). Some of the effects of age on the ballistocardiogram are apparent from these graphs.

The mean HK value showed a slight non-progressive increase with age. The mean value for the Q-H time interval remained the same in all four groups, yet the spread of values was slightly greater in the older ages. The mean H-Jp time duration was 0.01 second longer in the oldest group than in the other three age levels. The H-L time interval was 0.02 second shorter in the oldest three age groups than in the youngest. Also, the mean systolic ballistocardiographic contour (1-rounded, 5-sharp) became progressively more rounded with age.

In table 3 is presented the frequency of the various ballistocardiographic deflections. An interruption in the ejection deflection (HI wave) was observed in one third of the individuals in the 18 to 24 age group, and it was a considerably more frequent occurrence in this group than in any of the other age levels. In almost all cases the interruption in the HI wave occurred 0.03 second or less after the H point, and the interrupted HI wave usually was associated with a relatively wide HI angle (greater than 12 degrees). An interrup-
tion 0.04 second or greater after H rarely appeared. Concave and convex HI waves occurred infrequently in all age groups. An I+ deflection as well as a flat I wave were seen most frequently in the older ages. There were three individuals in the 18 to 24 age group whose records showed an I+ wave. In each case the I+ deflection was 0.03 second after H and the HI angle was relatively acute, being less than 7.2 degrees. In the oldest age group there were four individuals with an I+ wave. In these individuals the I+ wave was 0.04 to 0.06 second after H and this late I+ deflection was associated with a wide HI angle of 12.0 degrees or greater.

The J wave was observed to be completely monophasic in the youngest age group, and it became somewhat polyphasic with age. The J_d wave was a very discrete entity in about two fifths of the cases in the 18 to 24 age group, and always occurred later than 0.22 second after H. When the J_d wave was present in the oldest age group, however, it measured 0.16 to 0.18 second after H and appeared as a change in deflection on the descending limb of the JK wave. When a late discrete J_d wave was present, the J wave itself showed the same slope on the ascending and descending limbs. However, when the J_d wave was not discernible, the J contour was skewed on its descending limb as if another wave (J_d) had been incorporated under it.

Table 3 also reveals that the LM wave was frequently smaller in the oldest age group than in the youngest. However, the presystolic GH wave showed precisely the opposite change with age. The diastolic wave size was generally small, and the frequency of its occurrence did not change with age.

From the composite measurements and descriptions presented in tables 2 and 3, a ballistocardiogram typical of each age group was drawn (fig. 6). The subtle change in the ballistocardiographic pattern with age can best be appreciated by closely examining the progressive alteration in the various deflections and waves in these redrawn complexes. It should be emphasized that these patterns were drawn from the mean values obtained at each age level. The difference in the mean weight

**Figure 3**

Simultaneously recorded electrocardiogram, ballistocardiogram, and carotid pulse taken on a 22-year-old man. The paper speed is 50 mm. per second and the heavy time lines are at 0.10 second intervals. The first pattern is labeled, and the J_d wave is clearly evident on this tracing.
for the four age groups was less than 9 pounds. Thus, the weight discrepancy between age groups is not a factor causing the ballistocardiographic change with age in these redrawn complexes.

Criteria for Ballistocardiographic Abnormality

The mean values, standard deviations, and frequency occurrence of the various ballistocardiographic deflections at four age levels have been presented in the previous section. Many of the ballistocardiographic deflections changed progressively with age (figs. 5 and 6; tables 2 and 3). Since rheumatic heart disease and hypertension were absent in the population under study, then it is highly probable that the changing ballistocardiographic pattern reflected functional cardiovascular aging.

The criteria for ballistocardiographic abnormality were derived from the three ballistocardiographic measurements (HI, HJ, HK/HI) which progressively changed with age. A ballistocardiographic measurement (HI, HJ, or HK/HI) was defined as abnormal if it was two standard deviations or greater from the mean value at age 26, in the direction of the measurement change with age (fig. 7, table 4). An abnormal ballistocardiogram was one containing one or more of the abnormal measurements that indicated advanced or accelerated cardiovascular aging.

There are three possible gradations of ballistocardiographic abnormality depending upon the number of "abnormal" measurements in a given tracing. The degree of ballistocardiographic abnormality was classified arbitrarily as grade I, II, or III. A grade-I abnormality had only one abnormal measurement present in the ballistocardiogram; grades II and III had combinations of two or three abnormal measurements, respectively. When two or more abnormal measurements occurred together in the same tracing, it seemed reasonable to conclude that a more severe degree of accelerated cardiovascular aging existed.

By means of these criteria of abnormality, ballistocardiographic patterns were drawn to represent the kinds of abnormality that could theoretically be obtained (fig. 8).

The Abnormal Ballistocardiogram

There were 61 persons in this overtly
BCG ANALYSIS

HI = 12 mm
HJ = 10 mm
HK = 6 mm
HK/HI = 0.50

HI angle = 10.0°
Q-H = 0.09 sec.
H-Jp = 0.14 sec.
H-L = 0.32 sec.

Contour — 4
Ejection — interrupted
J wave — monophasic
LM wave — medium
Diastolic wave — small
GH wave — medium

Figure 4

Skematic labeled diagram of the simultaneously recorded electrocardiogram and ballistocardiogram. A method of analyzing the ballistocardiogram is presented in the lower portion of the figure.

healthy population with abnormal ballistocardiograms according to our criteria. The percentage of all individuals exhibiting one, two, and three abnormal ballistocardiographic measurements at the various age levels is presented in table 5.

The percentage of individuals with a grade-I abnormality increased progressively with age. The more severe ballistocardiographic abnormalities (grades II and III) began to develop in the fifth decade and gave the oldest age group a relative predominance of abnormal patterns. These results suggested a ballistocardiographic conversion with age from grade I to grade II, and from grade II to grade III.

In the 49 grade I cases (table 5) there were 25 HJ, 21 HK/HI, and 3 HI abnormalities.
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In the 11 grade-II cases, the combined HJ-HK/HI abnormality predominated. The frequent occurrence (14.6 per cent) of more than one ballistocardiographic abnormality in the 45 to 54 age group suggested a common factor etiologically responsible for the combination of abnormalities.

Figure 9 is a smoothly drawn graph plotted from the data presented in table 5. A grade-I ballistocardiographic abnormality was evident in 16 per cent of the population by age 35. A grade-II abnormality was present in about 16.5 per cent of the individuals by age 50. At age 50, about 40 per cent of the population had no evidence of ballistocardiographic abnormality.

On the assumption that an abnormal ballistocardiogram deteriorates from grade I, through grade II, to grade III, then from the graphs in figure 9 the attack rate for a grade-I abnormality and the conversion rate from grade I to grade II can be calculated* (fig. 10). There appears to be a rather constant attack rate before age 40 and a sharp increase thereafter. The rate of conversion to a grade-II abnormality showed a very significant rise in the fifth decade. One cannot say from this graph whether the individuals who converted to grade II in the fifth decade were the ones in whom a grade-I abnormality developed during the early portion of the 20 to 39 age period. That is, there may be a 15- to 20-year lag between the development of a grade-I abnormality and the conversion to grade II, but this is not the only possibility.

The electrocardiograms of all (fig. 10) 307 subjects were analyzed, and the mean values for the various measurements and the frequency occurrence of an abnormal electrocardiogram are presented in table 6. An abnormal electrocardiogram indicative of coronary artery disease was obtained from five individuals (9.3 per cent) in the 35 to 44 age group, and from 8 individuals (19.5 per cent) in the 45 to 54 group. In table 7 are presented the ballistocardiographic analyses of the 13 subjects with abnormal electrocardiograms. Ten of them (77 per cent) had one or more ballistocardiographic measurements that met the criteria of abnormality. Thus, a high correlation existed between an abnormal ballistocardiogram and electrocardiographic evidence of coronary artery disease in this group of individuals.

Discussion

The cardiovascular aging process has been evaluated ballistocardiographically in 307 men. The percentage of individuals with the initial appearance of accelerated cardiovascular aging increased progressively with age. Also, a more severe degree of aging appeared significantly for the first time in the fifth decade. The frequency-age distribution of the grades of ballistocardiographic abnormality seen in the present study were remarkably similar to the incidence-severity distribution of coronary artery disease reported in a number of necropsy studies in the literature. Enos et al.\(^7\) in their study of the incidence of coronary disease in U. S. soldiers killed in action in Korea showed that in the 20 to 30 age

\*See Appendix II.
group range about 77 per cent of the hearts showed some gross evidence of significant coronary atherosclerosis, with 10 per cent showing advanced coronary artery disease. Spain et al. in their consecutive autopsy series on 73 white men killed suddenly by trauma showed that severe coronary artery disease (grades 3 and 4 on a scale of 4) began to increase between ages 36 and 40, and then rose rapidly to involve 28 per cent of the population in the 41 to 55 age range. A graph relating the frequency of severe coronary atherosclerosis to age can be drawn from Spain's data, and this curve is similar in shape to the incidence graph of the grade-II ballistocardiographic abnormality presented in figure 9 of the present study. White et al. as well as Saphir et al. showed that the most striking increase in the incidence of severe coronary atherosclerosis occurred in the fifth decade, a finding similar to the frequency of a grade-II ballistocardiographic abnormality seen in the present study.

There have been a large number of ballistocardiographic studies that demonstrated a relationship between a qualitatively abnormal tracing and overt symptomatology of coronary artery disease. Unfortunately, the majority of these studies have been done with a ballistocardiographic instrument that did not meet basic theoretical considerations of design. Thus, the results of those studies are not strictly comparable with the data presented in this paper. Suffice it to say, there is

Table 4
Criteria for Ballistocardiographic Abnormality

<table>
<thead>
<tr>
<th>HI (dynes X 10^3)</th>
<th>HJ (dynes X 10^3)</th>
<th>HK/HI (ratio)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;69.1</td>
<td>&lt;35.1</td>
<td>&gt;1.02</td>
</tr>
</tbody>
</table>

Figure 5
Progressive change in the HI, HJ, HK/HI values with age. The solid lines are the regressions for the mean. The dotted lines are the lines of best fit through the values two standard deviations from the mean. Top. Progressive diminution in the HI force. Middle. Progressive diminution in the HJ force. Bottom. Progressive increase in the HK/HI ratio.
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Figure 6
Ballistocardiographic patterns constructed from the mean measurements at four age levels. Notice the progressive diminution in the HJ amplitude and the increase in the HK/HI ratio. Also, the over-all contour of the complexes becomes more rounded with age.

general agreement that regardless of the ballistocardiographic technic used, gross alterations in pattern are present with increasing frequency beginning at age 50; also, an abnormal tracing before age 50 seems to have prognostic significance. More recently, Smith et al., using a ballistocardiographic instrument similar to the one described in this study, demonstrated a flat, low-amplitude J wave on the acceleratory tracing of a 45-year-old pilot in whom a myocardial infarction developed 2 years later. The ballistocardiogram on this patient was presented in a photograph and, with the assumption that the individual was of normal weight, it corresponds to at least a grade-I abnormality by the present criteria.

There are many factors associated with advancing chronologic age that must be considered in evaluating the cause for the ballistocardiographic change with age. The possible alteration in the ballistocardiogram with varying heart rates is one of the considerations, since there was slight acceleration in

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the heart rate with advancing years. Frederick and Eddleman\textsuperscript{35} pointed out that the delivery of the stroke volume and its interaction with the functional state of the vascular tree determined the form of the ballistocardiographic complex. Normal variation in the heart rate due to the interplay of autonomic influences on the sinoatrial node would not be expected to alter significantly the pattern of cardiac contraction or the peripheral vascular resistance. Extremes in heart rate, however, probably would be associated with a change in the basic ballistocardiographic pattern due to an underlying alteration in the circulatory hemodynamics. Since the total average increase in heart rate between the youngest and oldest age groups in this study was only 10 beats per minute, it is unlikely that this minor acceleration in heart rate played any role in the ballistocardiographic change with age.

The alteration in the ballistocardiographic pattern with changes in body weight is pertinent. The amplitude of the ballistocardiographic deflection is inversely proportional to the total bed mass. Thus, as the weight on the ballistocardiographic bed was increased from 140 to 220 pounds, there was a 36 per cent diminution in over-all amplitude of the recorded pattern. This weight-amplitude relationship was taken into consideration and each acceleration amplitude was converted into absolute units of force in dynes.

It is conceivable that the effect of the aging process on the turgor or resiliency of the supporting structures of the body played a role in altering the ballistocardiographic complex in the older age groups. Starr\textsuperscript{36-38} in his necropsy perfusion experiments, produced a wide range of ballistocardiographic patterns in the same individual simply by altering the stroke force, the volume of ejected blood, and the rate of application of the delivered force. Frederick and Eddleman\textsuperscript{35} believed that the forces operating strictly within the cardiovascular system were the major determinants of the ballistocardiographic pattern. The vibromechanical properties of the body were studied by Tannenbaum et al.,\textsuperscript{13} who demonstrated that the inherent frequency of the body mass was not correlated significantly with age or anthropometric measurements. There was a high correlation, however, between natural body damping and age, and this factor may be one of the extracardiac phenomena chronologically related to the changing ballistocardiographic pattern.

A consideration of major importance is the changing direction of the cardiac force vector with age. A number of investigators\textsuperscript{39-41} have shown that the ballistic systolic forces are directed longitudinally in young persons and have a more lateral representation in the older age groups. In the present study only longitudinal (head-foot) forces were recorded. All ballistocardiograms at the present time have

\textbf{Figure 7}

The arbitrary levels of abnormality for the HI, HJ, HK/HI measurements. In each case the level of abnormality was set at two standard deviations from the mean at age 26, in the direction of the change with age.
resonant body artifacts in the lateral recording of motion, and thus the conclusions drawn from the lateral ballistocardiogram must be interpreted cautiously. Scarborough et al., working with the high-frequency Starr bed, showed that the LJ vector force changed from a head-foot to a lateral direction with increasing age. March demonstrated similar results with Dock direct-body electromagnetic pickup. Honig and Tenney, using an aperiodic ballistocardiogram with properties similar to the Reeves instrument, reiterated the changing position of the frontal plane vector from longitudinal to lateral orientation with age. The etiology of this directional change in the systolic ballistic forces with age has not been explained. It is unlikely that it represents a simple anatomic shift in the position of the heart. More likely, it probably represents a circulatory aging process in terms of altered cardiac contractility and vessel distensibility.

Starr introduced the concept of "presbycardia" to explain the changing ballistocardiographic pattern with age in the so-called normal person. He thought that the heart muscle became less powerful, i.e., the work output per unit of time diminished, in the older years. The etiology of this diminished power may be due to the effects of subclinical coronary artery disease, as coronary atherosclerosis is a progressive process in the age
## Table 7

**Ballistocardiographic Patterns on 13 Subjects with Electrocardiographic Evidence of Coronary Artery Disease**

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs.)</th>
<th>Ht. (inches)</th>
<th>Wt. (lb.)</th>
<th>Heart Rate (degrees)</th>
<th>Force (x10^4 dynes)</th>
<th>HK/HI</th>
<th>Time (sec.)</th>
<th>Polyphasic J wave</th>
<th>Contour (scale 1-5)</th>
<th>Grade BCG Abn.</th>
<th>ECG</th>
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<td>36</td>
<td>68</td>
<td>152</td>
<td>58</td>
<td>7.5</td>
<td>120.1</td>
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<td>0.10</td>
<td>0.13</td>
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<td>68</td>
<td>186</td>
<td>110</td>
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<td>87.0</td>
<td>62.0</td>
<td>112.1</td>
<td>1.28*</td>
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<td>0.14</td>
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<td>J. B. D.</td>
<td>38</td>
<td>67</td>
<td>153</td>
<td>57</td>
<td>11.8</td>
<td>90.8</td>
<td>24.5*</td>
<td>79.0</td>
<td>0.87</td>
<td>0.11</td>
<td>0.12</td>
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<td>H. E. J.</td>
<td>41</td>
<td>70</td>
<td>175</td>
<td>63</td>
<td>11.8</td>
<td>182.1</td>
<td>120.3</td>
<td>85.4</td>
<td>0.46</td>
<td>0.06</td>
<td>0.15</td>
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<td>T. G. L.</td>
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<td>68</td>
<td>177</td>
<td>86</td>
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<td>90.4</td>
<td>69.6</td>
<td>114.3</td>
<td>1.26*</td>
<td>0.10</td>
<td>0.17</td>
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<td>Q. R. N.</td>
<td>45</td>
<td>69</td>
<td>133</td>
<td>66</td>
<td>24.7</td>
<td>48.0*</td>
<td>101.6</td>
<td>50.4</td>
<td>1.05*</td>
<td>0.07</td>
<td>0.16</td>
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<td>G. H.</td>
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<td>70</td>
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<td>71</td>
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<td>84.4</td>
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<td>70</td>
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<td>64</td>
<td>16.8</td>
<td>131.0</td>
<td>40.4</td>
<td>138.3</td>
<td>1.06*</td>
<td>0.09</td>
<td>0.17</td>
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<td>68</td>
<td>199</td>
<td>97</td>
<td>20.5</td>
<td>147.1</td>
<td>57.9</td>
<td>212.0</td>
<td>1.44*</td>
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<td>0.17</td>
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<td>79</td>
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<td>64.1*</td>
<td>38.3</td>
<td>64.1</td>
<td>1.00</td>
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<tr>
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<td>178</td>
<td>75</td>
<td>13.2</td>
<td>111.8</td>
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<td>0.17</td>
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<td>B. W. W.</td>
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<td>15.1*</td>
<td>201.4</td>
<td>1.30*</td>
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<td>V. N. P.</td>
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<td>70</td>
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<td>48.8</td>
<td>90.6</td>
<td>1.86*</td>
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*Indicates that the values met the criteria of abnormality.
The frequency occurrence by age of the three grades of ballistocardiographic abnormality. The points on this graph were obtained from the data presented in Table 5. The dotted lines represent extension of the curves beyond the last recorded data point at age 47.7.

Figure 9

The group studied. It seems reasonable that the pattern of myocardial contractility would be altered either from relative coronary insufficiency or from secondary myocardial fibrosis. Thus, the ballistocardiographic change with age demonstrated in this overtly healthy population may represent the effects of diminished cardiac power from progressive coronary atherosclerosis. If this is the case, then an abnormal ballistocardiogram may be a sensitive indicator of early, subclinical coronary artery disease.

There were 13 individuals in the population with electrocardiographic evidence of coronary artery disease. Ten of them had an abnormal ballistocardiogram, and this association lends further support to the possible relationship between an abnormal ballistocardiogram and coronary artery disease. Three in this group of 13 had ballistocardiograms that were entirely normal in all respects. It is possible that the ballistocardiogram in these three subjects failed to reveal accelerated cardio-lovascular aging coincident with coronary artery disease. An equally good possibility is that coronary artery disease was not present even though the electrocardiogram was "abnormal." Although these three persons had generalized low-voltage T waves in their resting electrocardiograms, their records showed a normal QRS axis, a narrow QRS-T angle, and no S-T abnormality. One of the three subjects (H.E.J.) had negative exercise tests, which included a double Master's step test, and 3- and 5-minute Harvard step tests. Another (H.H.R.) had a borderline response to a 3-minute Harvard exercise test with an 0.5 mm. S-T depression in leads II, aVF, and III 3 minutes after the completion of the exercise. It is entirely possible that all three subjects with normal ballistocardiograms did not have coronary artery disease, and the generalized low-voltage T waves in their resting electrocardiograms had another etiology. Only long-term follow-up of these persons will solve the question.
If a relationship between an abnormal ballistocardiogram and coronary artery disease exists, then the ballistocardiogram may be useful as a predictor of underlying ischemic heart disease. Recently, a 37-year-old pilot was seen for cardiac evaluation, and 3 weeks after the examination he died of an acute myocardial infarction. The following is a case report of this person with pertinent clinical, ballistocardiographic, and necropsy findings.

R. W. B. (no. 3648, U.S.N.S.A.M.) was a 37-year-old male aviator seen on June 30, 1959, for cardiac evaluation. There were no recent symptoms. However, 11 months previously he had experienced a very transient episode of hemianesthesia and partial aphasia for which no explanation could be found. Electroencephalogram and carotid angiogram were normal, as were all other clinical studies at that time.

Physical examination was entirely normal except for slightly increased arterial light reflex in the ocular fundi. The carotid pulses were full and equal bilaterally. Cardiac examination was normal, with a blood pressure of 144/88 mm. Hg in the supine position. Neurologic examination revealed no abnormality. The patient's weight was 169 pounds, height 71.5 inches.

Laboratory data revealed a normal resting and exercise electrocardiogram (3-minute Harvard step test, one step every 3 seconds). Cardiac x-rays and fluoroscopy were normal. Blood lipid studies revealed a fasting serum cholesterol of 229 mg. per cent, and an atherogenic index of 74. Ballistocardiogram (fig. 11) showed a grade-II abnormality with significantly divergent HI and HK/HI values (HI=64.2×10^6 dynes; HK/HI=1.54).

Since the ballistocardiogram was the only abnormality detected, the patient was returned to full-duty status. Three weeks later the patient died suddenly of an acute myocardial infarction. Autopsy findings* revealed extensive coronary atherosclerosis involving both the right and left coronary arteries. About 2 cm. below the ostium of the left coronary there was an acute occlusion of the vessel, probably secondary to hemorrhage below an arteriosclerotic plaque. Myocardial infarction of about 24 hours was present. The in-

*The autopsy was performed by the Pathology Department, Gunter Air Force Hospital, Montgomery, Alabama.
Ballistocardiographic change with age have been presented. The validity of the relationship between an abnormal ballistocardiogram, accelerated cardiovascular aging, and coronary artery disease requires further substantiation from follow-up studies. If the results presented in this paper stand the test of time, then the possible diagnosis of early subclinical coronary artery disease from an abnormal ballistocardiogram appears promising.

Summary

A clinically adaptable, ultra-low frequency acceleration ballistocardiograph that met all theoretic considerations of biophysical design was used to evaluate the cardiovascular aging process in 307 overtly healthy men aged 18 to 54.

A new practical method for quantitative ballistocardiographic analysis is described, and the changing ballistocardiographic pattern with advancing age is elucidated.

Criteria for ballistocardiographic abnormality are established in terms of accelerated cardiovascular aging, and the degree of abnormality is graded (I-III).

The initial appearance of accelerated cardiovascular aging (grade-I abnormality) was present on the ballistocardiogram in 16 per cent of the population by age 35. A more severe degree of aging (grade II) was evident in 16.5 per cent of the individuals by age 50. There was a rather constant attack rate for the initial development of an abnormal ballistocardiogram (grade I) during the 20 to 39 age period, and an accelerated attack rate after age 40. The rate of conversion to a more severe grade of ballistocardiographic abnormality increased significantly in the fifth decade.

The relationship between an abnormal ballistocardiogram, accelerated cardiovascular aging and coronary artery disease is discussed.

Appendix

I. Conversion of acceleration amplitude measurements to absolute units of force in dynes

It is known that:

\[ F = m \times a. \]  

(1)

F is force in dynes, m is mass in grams, and a is acceleration in cm./sec.².
Let $M$ be the mass of the subject in pounds. The total weight of the ballistocardiographic bed with electrocardiographic electrodes is 14 pounds. Then:

$$m = (M + 14) \text{ lbs.} \times \frac{1 \text{ kilo}}{2.2 \text{ lb.}} \times \frac{1000 \text{ Gm.}}{1 \text{ kilo}}$$

$$m = 453.6 \times (M + 14).$$

(3)

It was found by calibration that 1 mm. of ballistocardiographic amplitude is equal to 0.12 cm./sec.

Thus,

$$a = 0.12 A,$$

(4)

where $A$ is the ballistocardiographic acceleration amplitude in millimeters.

Combining equations (3) and (4) into the original equation (1), we have:

$$F = 453.6 \times (M + 14) \times 0.12 A$$

(5)

$$F = 54.4 \times (M + 14) \times A.$$  

(6)

II. Derivation of the formulae for calculating attack and conversion rates for ballistocardiographic abnormality.

Let $P(I) [Q(j)]$ be the proportion of persons in the total population who show a grade $i$ [grade $j$] ballistocardiographic abnormality in the $n$th $(n+1)$ th year, and $R(i, j)$ the proportion of the total population who had a grade $i$ abnormality in the $n$th year and a grade $j$ abnormality in the $(n+1)$th year.

In drawing figure 10, it was assumed that:

$$P(III) = R(III, III),$$

$$Q(III) = R(III, III) + R(II, III),$$

$$P(II) = R(II, II) + R(II, III),$$

$$Q(II) = R(II, II) + R(I, II),$$

$$P(I) = R(I, I) + R(I, II),$$

$$Q(I) = R(I, I) + R(O, I).$$

The attack rate, $R(O, I)$, and the conversion rate, $R(I, II)$, were obtained by solving the above six equations with data derived from the graph in figure 9 of the text at yearly intervals between ages 18 and 51.

It was assumed that no one "jumped" a ballistocardiographic grade of abnormality in a year's time. Also, it was assumed that no deaths occurred. Even if the death rate were known and taken into consideration, however, the relative position of these curves (fig. 10) would not be affected.

Acknowledgment

I am deeply grateful to Dr. Ashton Graybiel for his encouragement, enthusiasm, and suggestions, which led to this ballistocardiographic study. I wish to thank Dr. Ronald Malt, who constructed the acceleration ballistocardiograph used in this paper. Also, I am indebted to Dr. Marshall Jones for his assistance in the derivation of the formulae for the calculation of attack and conversion rates.

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