An Empiric Approach to the Interpretation of the Low Frequency, Critically Damped Ballistocardiogram

By Richard J. Jones, M.D., and Norman E. Goulder, M.D.

For the first time, the range of variation in amplitude and temporal relationships of the first four waves of the critically damped ballistocardiogram are presented for two groups of normal subjects. Characteristic deviations from this range are described in patients with vertical hearts, essential hypertension, coarctation of the aorta and aortic insufficiency. The relationship of the cardiac position to the first two patterns is reported. Explanation is found for these observations in current ballistocardiographic theory.

Since his original description of the ballistocardiogram, Starr\(^1\) has directed most of his efforts toward improving its use as a measure of the cardiac output. While his definition of the range of amplitudes to be expected in normal subjects has been well outlined by Tanner\(^2\), his classification of abnormalities in form has remained qualitative.\(^3\) Indeed, as Hamilton and his associates\(^4\) pointed out, the vibration of the undamped instrument is only impeded or reinforced by the movements of blood in the heart and great vessels, so that more minute temporal analysis would perhaps be unjustified.

Nickerson and his co-workers\(^5\),\(^6\) in describing the low frequency, critically damped apparatus, give only a qualitative description of the form of the resulting record. This instrument has the advantage, however, that all waves are the consequence of forced movements, so that phasic reinforcement and damping need not be considered. It would appear that this type of record would give greater fidelity and reproducibility of form for clinical purposes. Consequently, we have pursued the suggestion of Hamilton\(^7\) to evaluate empirically the ballistocardiograph, but using the low frequency, critically damped instrument to record the cardiodynamic events.

Though Nickerson\(^8\) has referred to some significant points of interest in the development of the H and K waves, and removed the objection of Hamilton and co-workers\(^4\) to the time lag in onset of the I wave, neither he nor Starr has published any tables of the normal limits of relative amplitudes and time sequence of the various waves. In attempting to study deviations from the normal ballistocardiogram in various circulatory disturbances, a group of normals were collected and their records measured carefully with regard to the timing and amplitude of the first four waves. Some rather pertinent deviations from the normal were noted in random conditions and allowed the establishment of certain definite patterns, the understanding of which may further aid in approaching the goal envisaged by Starr\(^8\) of deriving the shape of the cardiac ejection velocity curve from the ballistic impacts.

Material

Over 500 ballistocardiograms have been taken on over 300 subjects, the majority of whom had real or suspected heart disease. This study concerns the comparison of two groups of subjects having normal cardiovascular systems with patients having hypertension and certain other cardiodynamic abnormalities. Normal group 1 is made up of 25 young, active adults—14 men and 11 women—averaging 26.2 years in age (ranging from 17 to 37 years), who were members of the hospital staff and had passed several thorough physical examinations in recent years. They were all of average body build, none being particularly obese or at the extremes of height. While none were trained athletes, all were in good health at the time of recording. One had a slightly
prolonged P-R interval, apparently of congenital origin.

Group 2 normal subjects were drawn from the medical services on the basis of normal cardiovascular systems in the absence of acute illness. The group includes 27 patients, predominantly male, averaging 54.9 years in age (ranging from 38 to 85 years), many of whom doubted had some degree of unrecognized arteriosclerosis or transient hypertension. To exclude cardiac disease, unipolar electrocardiograms were taken in addition to the routine physical examination and admission photofluorograms of the chest. They suffered from no demonstrable cardiac failure, and, though they had been hospitalized, the majority were ambulatory. Though 3 of them gave electrocardiographic evidence of "myocardial abnormality," they were not excluded from the series, because they gave no history of cardiac distress or symptoms of myocardial infarction and their ballistocardiograms had a normal configuration.

Twenty cases of essential hypertension, including 6 who had been previously treated for congestive heart failure, were also studied in similar fashion. This group consisted of 15 women and 7 men, ranging from 26 to 84 years of age (average age 51.9), who had an elevation of the diastolic pressure to over 100 mm. Hg, and who were free of renal failure, congestive heart failure, or clinical coronary artery disease at the time of recording. Four cases of clinically established coarctation have come under observation. Their records are compared with those of the normal and the hypertensive subjects. A group of 19 cases with rheumatic or syphilitic lesions of the aortic valve revealed some points for comparison with the hypertensive pattern.

METHOD

All patients were made to rest 20 minutes on an air mattress atop the ballistocardiograph,* which was adjusted for the weight of each individual to the low frequency of 1.5 cycles per second and critically damped as recommended by Nickerson and Curtis. Usually the study was made in the basal state, or, if serial recordings were not anticipated, the subject was at least three hours postabsorptive. Because of the more evident influence of respiration in patients, recordings were made both during slow respiration and in breath-holding, in duplicate, so that a comparison might be made. For best agreement between the records during slow breathing and those in arrested respiration, it was found advisable to instruct the patient carefully to avoid effort or strain in blowing out a full inspiration, inspiring again and holding the second breath at the mid-point of normal respiration. Only occasionally did the patient have trouble following the commands.

Lead II of the electrocardiogram was taken simul-

* Built in this laboratory by Dr. M. M. Newman.
intervals were present with the slower pulse rates. Its significance in cases of coarctation will be mentioned later. Comparison of the H-I, I-J and J-K intervals shows an increasing dispersion in the normal variation as the cycle progresses.

The depth of the H-I deflection varied from 21 to 50 per cent of the I-J deflection, and might have varied more widely had a greater variety of cardiac positions been sought. The J-K deflection averaged slightly more than the

agreement between "minute volume" values, while breathing and while apneic, within 0.5 liter per minute, and in 75 per cent, within 1 liter per minute.

Older Normal Group. The 27 noncardiac patients composing group 2 showed a greater tendency for a variation of form to be present during quiet breathing, and in 20 per cent, to have such distortion with respiration as to make that record useless for calculating amplitudes. In 2 patients, the H wave was so poorly

developed (or masked by the preceding diastolic wave) as to preclude measuring the H peak, so their H intervals are excluded in the tabular results (table 1).

Four of this group had Q-H intervals of 0.12 second, without evident prolongation of subsequent events in the ballistic pattern, and without unusual shortening of the P-R interval. The other intervals, as can be seen from table 1, tend to be shorter on the whole than in the young, healthy group. One patient barely exceeded 0.20 second for the Q-I interval. The remainder had Q-I durations of 0.14 second or more, except for 1 extremely apprehensive and trembling psychoneurotic patient who had a

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**Table 1.—Time Intervals and Relative Amplitudes of H, I, J and K Peaks of the Ballistocardiogram, as Measured from the Onset of Electrical Systole in Two Groups of Normal Subjects and in Four Cases of Coarctation of the Aorta**

<table>
<thead>
<tr>
<th></th>
<th>Q-H</th>
<th>Q-I</th>
<th>Q-J</th>
<th>Q-K</th>
<th>H-I</th>
<th>I-J</th>
<th>J-K</th>
<th>F</th>
<th>&quot;S.V.&quot;</th>
<th>&quot;M.V.&quot;</th>
<th>H-I Amp as % I-J</th>
<th>J-K Amp as % I-J</th>
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<td>sec.</td>
<td>sec.</td>
<td>sec.</td>
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<td>sec.</td>
<td>sec.</td>
<td>sec.</td>
<td>gm.</td>
<td>cc.</td>
<td>L/min.</td>
<td></td>
<td></td>
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<td>.37</td>
<td>.57</td>
<td>.10</td>
<td>.19</td>
<td>.25</td>
<td>86.9</td>
<td>123.0</td>
<td>9.00</td>
<td>50</td>
<td>158</td>
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<td>.15</td>
<td>.29</td>
<td>.46</td>
<td>.06</td>
<td>.14</td>
<td>.17</td>
<td>29.8</td>
<td>50.6</td>
<td>3.56</td>
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<td>79</td>
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<td>.169</td>
<td>.329</td>
<td>.533</td>
<td>.082</td>
<td>.160</td>
<td>.204</td>
<td>50.4</td>
<td>88.6</td>
<td>6.11</td>
<td>38</td>
<td>105</td>
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<td>± .013</td>
<td>± .013</td>
<td>± .022</td>
<td>± .029</td>
<td>± .011</td>
<td>± .011</td>
<td>± .021</td>
<td>± 13.8</td>
<td>± 20.0</td>
<td>± 1.38</td>
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<td>sec.</td>
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<td>sec.</td>
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<td>.185</td>
<td>.27</td>
<td>59.0</td>
<td>105.0</td>
<td>7.55</td>
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<td>195</td>
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<tr>
<td>Minimum</td>
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<td>.49</td>
<td>.065*</td>
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<td>64.4</td>
<td>4.79</td>
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<td>± .032</td>
<td>± .038</td>
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<td>± .042</td>
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<td>± 37</td>
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<td>6 yr. M</td>
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<td>.37</td>
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<td>35.3</td>
<td>70.9</td>
<td>6.24</td>
<td>48</td>
<td>47</td>
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</table>

* Omitting two patients where no H wave was demonstrable; n = 25.

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I-J in amplitude, but never exceeded 160 and was not less than 88 per cent of I-J.

The "stroke volume" and "minute volume," as estimated by Nickerson's formula, is that calculated during arrested respiration. Much better agreement is obtained in day-to-day comparisons if slow breathing values can be used, though this is not possible in about 10 per cent of normal subjects and 35 per cent of our cardiac patients. Hence, such minute volume determinations are best made during apnea for the purpose of making comparisons with patients. The volume calculations were somewhat higher, on the average, during apnea; but in about 50 per cent of the cases there was
Q-I of 0.11 second and the 2 oldest patients (71 and 85 years of age), who had intervals of 0.12 and 0.09 second. These 2, being well along in years, doubtless suffered from some degree of arteriosclerosis of the major vessels. While the Q-J and I-J maxima in the younger group were never exceeded, the minimum of one or the other was not reached in 12 cases. The Q-K time was shorter than 0.46 second in only 4 instances, and in 6 of the 7 cases in which there was a decrease in J-K amplitude (below 90 per cent of the I-J amplitude), it was shorter than 0.49 second. Conversely, the longer Q-K times (near the upper limit of normal) were always associated with well developed K waves, never with abbreviated or abortive K waves.

The H-I amplitude exceeded 50 per cent of I-J amplitude in one case (59 per cent) but fell below the 20 per cent limit in 5 cases, in 2 of which it was often absent during breath-holding or expiration. The J-K amplitude was frequently smaller than the I-J amplitude, falling below 80 per cent in 6 cases, often not falling below the roughly estimated base line (fig. 1B, C, D). This has been said to be closely associated with coarctation of the aorta,¹⁻⁹⁻¹¹ but none of these patients showed any diminution of the femoral or pedal pulses, or other conditions where circulation to the viscera is impaired. We were impressed, however, by its association with a markedly vertical heart on x-ray study, and routine x-ray films available in 6 such cases indicated a dependent type vertical heart in each case.

Further comment is hardly needed on the question of "stroke volume" and "minute volume" calculations, except to say that they are obviously lower in the older patient group, as previously noted by Starr and Schroeder,¹⁵ and that greater variability is noted between respiration and apnea, and is more often seen from beat to beat. Notching was not met with even in the more unusual tracings of this series.

Coarctation of the Aorta. Four cases of coarctation (table 1) proved by clinical examination, 2 of which were confirmed at surgery (fig. 1E, F), had J-K amplitudes of 59, 70, 74 and 47 per cent, in association with H-I amplitudes of 59, 54, 46 and 48 per cent, of I-J amplitude. The only other abnormality, and the feature distinguishing these cases from the normals with vertical hearts, was a delay in the final development of the K peak, often associated with marked slurring in the J-K segment. The Q-K time in these three cases was 0.57, 0.60, 0.65 and 0.62 second, respectively. The 0.57 second, while not abnormal in itself, is never associated in normal vertical hearts with an abortive K wave, which is ordinarily shortened in time as well as amplitude.

![Fig. 1. Representative ballistocardiograms: A. Normal. B. and C. Normal, with tendency to vertical heart. D. Vertical heart, postlobectomy. E. and F. Coarctation of aorta, confirmed at operation. G. and H. Essential hypertension. I. and J. Clinical aortic insufficiency.](http://circ.ahajournals.org/)

Patients with Uncomplicated Hypertension. A group of 20 patients (table 2) with essential hypertension were studied, of whom 6 had previously been in heart failure. Excluding the latter, there was either a Q-I interval or Q-J interval of shorter duration than any noted in the younger group of healthy normal adults, except for 1 patient, aged 30, whose blood pressure ranged from 180/120 to 114/70 during hospital observation. His was historically a very labile hypertension. Two other hypertensives with associated cardiomegaly had Q-I durations of 0.17 and 0.15 second, but they had an I-J duration less than 0.14 second, the lower limit
noted in group 1. In the 3 men and 3 women who had been in congestive failure due to hypertension, one had a Q-I interval of 0.17 second, the rest falling at 0.12 second or less. There is a statistically significant difference between the mean Q-I intervals in the hypertensive group and those in the normal group 2.

Table 2.—Time Intervals and Amplitudes of the Ballistocardiographic Waves in Twenty Hypertensive Subjects*

<table>
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<tr>
<th></th>
<th>Q-I</th>
<th>Q-J</th>
<th>H-I</th>
<th>I-J</th>
<th>H-I amp. as % I-J</th>
<th>J-K amp. as % I-J</th>
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Maximum...  .175  .33  .08  .22  36  232
Minimum...  .07  .20  .02  .10  0  105
Average...  .125  .29  .05  .142  11  168
Standard deviation ± .028  ± .012  ± .014  ± .029  ± 10  ± 38

* Patients 1 through 6 had previously been treated for heart failure.

It thus appears that essential hypertension is associated consistently with a shorter duration of Q-I or I-J, or both, than occurs in a normal series. That this shortening is not necessarily related to the hypertension is shown by the 2 oldest men in the patient group, who had Q-I durations of 0.09 and 0.12, respectively, without evident hypertension.

Of the 20 hypertensives, the H-I amplitude fell within the normal range in only 5 cases, it being very low or absent in the rest. On the other hand, the J-K amplitude exceeded 160 per cent of the I-J amplitude in 12 cases, and in no case fell below 100 per cent (fig. 1G, H).

A point of interest arises in comparing the hypertensive pattern with the aortic insufficiency pattern. Out of 19 cases with aortic valve lesions and murmurs characteristic of either stenosis and/or insufficiency, 8 were found to have a J-K amplitude exceeding 160 per cent of the I-J amplitude. These cases (fig. 1I, J) more often had an H-I amplitude well within the normal range, and were not accompanied by any shortening of intervals in the early portion of the cycle. In fact, if the H-J interval is used, it reaches the lower limit of normal (0.20 second) in only 3 of the hypertensive patients, and exceeds it in all the aortic valvular lesions associated with deep K waves.

The Effect of Cardiac Position on the Ballistocardiogram. The reciprocal behavior of the I wave and the K wave with vertical hearts (fig. 1B) and with hypertensive hearts (fig. 1G, H) suggested that the amplitudes of these waves might be dependent in part on the position of the left ventricle. X-ray films taken in the upright position in the 20 hypertensive patients and in 31 of the combined normal group, including 5 vertical hearts, permitted the determination of the angle of inclination; the angle between the long diameter of the heart and the dorsal spine. The cosine of this angle (α), which is proportional to the vertical vector of this diameter, was then compared with the H-I amplitude and the J-K amplitude, both expressed as percentage of the I-J amplitude, which has figured prominently in stroke volume estimations of both Starr and Nickerson. The correlation coefficient comparing cosine α with the H-I amplitude in all 51 cases is r = +0.886, and with the J-K amplitude is r = −0.644. Both are significantly different from zero (p = 0.001 for the latter).

It is seen from the scattergrams (figs. 2 and 3) that the line of best fit for these two correlations, including the hypertensive patients, does not evenly split the hypertensive cases in either case. All but 6 cases fall below the best line for the 51 cases in figure 2, and all but 7 lie above it in figure 3. While this series is rather small,
it is at least suggestive that the position of the heart is not the only factor at work to diminish the H-I segment, and increase the J-K amplitude in hypertensives. This could only be definitely established by comparing these hypertensive patients with a normal group having a similar average rotation of the heart to the left. Very few normal hearts reach the degree of rotation seen in hypertensive persons.

**FIG. 2.** The vertical H-I amplitude, expressed as the per cent of the I-J amplitude plotted against the cosine of $\alpha$, the angle between the long diameter of the heart and the dorsal spine, measured from the anteroposterior roentgenograms in 51 cases. The line of best fit is presented. Open circles indicate cases with essential hypertension, diastolic blood pressure over 100 mm. Hg. Dots indicate cases drawn from the normal series.

**FIG. 3.** The vertical J-K amplitude, expressed as per cent of the I-J amplitude, plotted against cosine $\alpha$. (Cases identical with those in fig. 2)

**Discussion**

From analysis of our records, it has become apparent that the critically damped ballistocardiogram must indicate certain characteristics of systolic ejection which are as yet poorly defined. We have used the onset of the QRS of the electrocardiogram as timing the onset of systole. While a carotid pulse wave or a phonocardiographic analysis might be theoretically a better measure of the onset of mechanical systole, we found in a small series of normal subjects as great a variation in the relationship of the I wave of the ballistocardiogram to the onset of the carotid pulse as to the Q of the electrocardiogram. Brief experience with phonocardiograms indicates that they give a more reliable measure of the onset of mechanical systole when the first sound is not obscured by adventitious sounds. We are now routinely employing both electrocardiograms and phonocardiograms. At any rate, the variations we have observed in pathologic cases are apparently much greater than the normal variation in the interval between mechanical and electrical systole.

The I peak has been rather widely accepted, on the basis of experiments with models, as an indication of reversal of aortic blood from the headward to footward direction. Its time of occurrence indicates that the systolic ejection of blood from the heart is not yet concluded, but that the head of the pulse wave has rounded the arch, and, presumably, the predominant flow of blood changes from the headward to the footward direction. Accepting that this is true, a shortening of the "maximum ejection phase" of Wiggers would be expected to hasten the occurrence of the I peak of the ballistocardiogram. That the maximum ejection phase is brief and steep in hypertension would explain the early I peak in this condition. Since the subsequent ballistic waves are dependent also on the initial acceleration of blood, it is not surprising that they may be correspondingly shortened. The particular advantage of the ballistocardiogram over a direct systolic volume curve, were it obtainable in man, lies in the fact that the relatively sharp peak of the I wave is easier to measure than a gradual and
often minute change in the slope of the ejection curve.

While there is a slight tendency for the shorter Q-K times to occur in association with a more rapid pulse, there was no such tendency demonstrable with the Q-I intervals in the narrow range studied. An increase in average pulse rate of 6 beats per minute in the hypertensive group could by no means explain such differences as were observed.

The J peak is determined by the point at which maximal footward acceleration of the blood is succeeded by gradual deceleration, and, according to Hamilton and co-workers,16 this terminates in the K wave peak when reflection of the pulse wave from the lower extremities causes a more significant deceleration in the headward direction. In our older normal subjects with vertical hearts, the amplitude of the J-K segment was quite under-developed, though this was also found consistently with coarctation of the aorta. The distinguishing feature in the latter cases was that the K wave, though under-developed, was also much delayed—i.e., though most of the descending aortic flow was dissipated in collaterals, such a wave as did reach the lower extremities was low in amplitude and delayed, as pulse tracings have shown,14 and so was its reflected wave. Thus, in coarctation of the aorta, there is an abnormally small K peak, delayed beyond any normal limits, associated with a deep I wave. This pattern is quite specific for coarctation and other obstructive lesions of the descending aorta.16

The explanation for a somewhat similar pattern in subjects with an otherwise normal circulation is apparently found in the cardiac position, as indicated by the angle which the long axis of the heart makes with the vertical. Starr and his associates' recognized that the "cardio-aortic axis" could influence the depth of the I wave, but felt that this only needed to be considered in extreme cases. While the spread of our data certainly indicates that other factors are involved, there is an excellent positive correlation and probably a strong dependent relationship between the two. The obvious explanation for this, of course, is that the vertical vector of the movement of the mass of ejected blood and contracting cardiac muscle will be larger in a vertical heart than in the transverse heart, where a larger proportion of the movement is in a horizontal direction, thus dissipating a larger part of the kinetic energy against the wall of the ascending aorta, instead of the arch.

Less to be expected is the good correlation, in the negative direction, between the cosine of the angle of inclination of the heart and the relative vertical amplitude of the J-K segment. The only ready explanation for this occurrence is that the greatest movement of heart and blood in diastole takes place during the same period of early diastole when the K wave is developing, but in a footward direction so that its headward recoil opposes the footward recoil of the body during deceleration of descending aortic blood. Obviously, the more vertical heart will have the greater vertical component for this force and exert a greater nullifying force against the "normal" development of the K wave. Hence, the K wave is deep in the transverse and shallow in the vertical hearts.

The fact that most patients with hypertensive heart disease show relatively deep K waves compared with those few normal persons whose hearts have similar angles of inclination, and that 70 per cent of them lie above the best fitting line for all 51 cases, suggests that some other factors may be operating to enhance this particular effect of cardiac position. Perhaps the greater mass of cardiac muscle or the larger diastolic volume is related. It is also conceivable that a systolic volume ejection curve of the hypertensive type may lead to a greater volume of blood traveling with the head of the pulse wave, thus allowing for deceleration of a greater volume of blood than is usual before this force is quenched by the reflected wave from the periphery.

About half of the cases of aortic insufficiency, with or without stenosis, also have abnormally large J-K segments, relative to I-J. They may be differentiated from cases presenting the hypertensive pattern by their normal or prolonged Q-I and Q-J intervals, and their normal or excessive II-I amplitudes. Again the position of the heart plays a relative role, but such factors as the duration and magnitude of the regurgita-
tion of blood, the slow transmission of reflected waves due to a patulous peripheral vasculature and doubtless other factors, make final evaluation difficult.

Aside from these suggested implications of ballistic theory, our data indicate that the ballistocardiogram may have much greater use clinically than its rather dubious value as a measure of cardiac output. Shortening of the time intervals between the earlier peaks of the ballistocardiogram is present in all well-defined cases of essential hypertension that we have followed to date. That this is not the necessary consequence of an elevated blood pressure is evident from the fact that this shortening is not seen in hypertension due to coarctation, pheochromocytoma, or hyperthyroidism and is not always seen in patients with an early labile essential hypertension. Furthermore, it was noted in 2 elderly men who had no history of hypertension. Arteriosclerosis of the great vessels is such a constant concomitant of old age that it is tempting to suggest that such a ballistic phenomenon derives from the systolic ejection curve induced by an increase in the rigidity of the great vessels by sufficient diastolic pressure elevation or arteriosclerosis. Perhaps further studies will indicate some value in prognosis or better diagnosis in this disorder.

The presence of a prolonged Q-I or Q-J interval has, in our experience, been associated only with serious heart disease: delayed intraventricular conduction, old myocardial infarction, or congestive heart failure. A normal Q-J interval in a patient with marked hypertension must be viewed with the same suspicion as a prolonged interval otherwise.

The assistance these observations may lend to the better determination of the stroke volume by the ballistocardiogram is questionable. Certainly a better definition of various patterns with improved understanding of the related variations in the central pulse wave contour must inevitably lead to closer approximation of stroke volume. On the basis of hypothetic curves postulated by Hamilton and associates, a normal person with a relatively long Q-I interval should have a somewhat higher J peak than one with an interval near the lower limits of normal for the same stroke volume. It is evident that the relative H-I amplitude varies directly and the J-K amplitude inversely with the cosine of the angle of inclination of the heart, so that the I-J amplitude with which they are compared would appear to be the most representative of the stroke volume. Whether the I-J amplitude itself may be influenced similarly by position of the heart has not been determined, though on theoretic grounds there is less reason to suppose so.

If one were to propose a routine system of ballistocardiography, it would be advisable to relate the various peaks to the electro- or phonocardiogram, and to compare the relative amplitudes of H-I, I-J and J-K. Whether such careful temporal measurements would be equally worthwhile with ballistocardiograms made with the high-frequency undamped table of Starr, we are not prepared to state.

**Summary and Conclusions**

1. An approach is made toward describing the pattern of the low frequency, critically damped ballistocardiogram in terms of the amplitude and time relations of its first four waves. The occurrence of H, I, J and K peaks is timed in reference to the onset of systole, and the H-I and J-K amplitudes are expressed as a percentage of the I-J amplitude. These values are found to fall within a rather narrow range in 25 young, healthy adults under the age of 37; exceeded rarely in an older group of 27 patients with presumably normal cardiodynamics; and exceeded in certain definite patterns in essential hypertension, coarctation of the aorta and aortic insufficiency.

2. The “cardiac output,” as compared during slow respiration and apnea, checked within 0.5 liter per minute in half of the normal subjects and within 1.0 liter per minute in only three-fourths of the cases.

3. The H-I amplitude, expressed as a percentage of the I-J segment, was found to have a significant positive coefficient of correlation with the cosine of the “angle of inclination” of the heart. The J-K amplitude was found to have a significant negative coefficient of correlation with the same angle.

4. Four definite patterns are mentioned: (a)
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cocoractation of the aorta—a small K wave (J-K less than 90 per cent of the I-J wave), delayed beyond the upper limits seen in normals (0.57 second) in association with a large I-J segment; (b) the vertical heart—a small K wave, earlier than usually seen, associated with a high-normal I-J amplitude; (c) essential hypertension—a short Q-I or Q-J, below that seen in normal subjects, usually associated with a small or absent H-I segment, and, in half the cases, with an abnormally deep K wave (J-K greater than 160 per cent of I-J); (d) aortic insufficiency or stenosis—a deep J-K (greater than 160 per cent of I-J), associated usually with a normal or deep I-J segment, but always associated with normal or prolonged Q-I or Q-J times.

5. The explanation for these specific ballistocardiogram patterns, based on present ballistocardiographic theory, is discussed.

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