Further Studies on a Theory of the Ballistocardiogram

By Abraham Noordergraaf, Ph.D.

In this theory of the normal human longitudinal ballistocardiogram we shall attempt to relate the record, in quantitative terms, to the events in the circulation that we believe to be its genesis.

The variables concerned here fall into three groups: those concerned with the performance of the heart as a pump; those concerned with the position and elastic properties of the vessels that contain the blood and guide its movement; those concerned with the properties of body tissues themselves that influence the transfer of forces arising within the body to its support, the ballistocardiograph. Mainly the first two groups are discussed in this paper. The fourth group, comprising the properties of the instrument, has been discussed extensively.

As the simplest approach to the problem we have taken as our starting point the movement of the body in space, when the body is free to move, with the events of the cardiac cycle. It is of interest to note that the advantages of this approach were recognized by Trotter in 1872, when he commented on Gordon's paper, the first on this subject.

Such an approach is based on a well-known principle. Figure 1 shows a striking example that is within the experience of everyone. If an object is free to move, when the internal position of its center of gravity is altered by forces arising within it, the object alters its position in space so that the position of its center of mass in space remains the same. This is why the fisherman has so much difficulty recovering his hat.

The body, placed in position in which it is free to move in space, will behave similarly. Shortly after the onset of systole, when blood is first driven headward out of the heart to distend the great vessels, the internal center of gravity of blood moves headward in the body. Later, as the pulse wave spreads peripherally, blood accumulates at a great distance from the heart in the more peripheral vessels, so that the center of gravity moves footward within the body. But if the body is free to move in space, the center of gravity of body, blood, and support does not move at all in space; it is the body that moves first footward and then headward during the cardiac cycle (fig. 1).

Such a movement has three interrelated quantities—displacement, velocity, and acceleration—and each of these can be recorded by an appropriately designed ballistocardiograph. It is our aim to calculate what each of these records would look like from data concerned with the physical properties and performance of the heart, circulation, blood vessels, and body tissues. Most of the values needed for this calculation were secured from the literature, some were determined by measurement and experiments made to secure them for this study. In a few instances values consistent with other information on the subject were assumed. From such data we have been able to calculate the theoretical ballistocardiogram from normal values of cardiac performance and tissue properties. And, finally, these theoretical ballistocardiograms have been compared with the records secured on healthy persons by the best modern instruments. Part of these results were reported previously.1, 2

In our previous studies1-5 we computed from physiologic and anatomic data the effects caused by the movement of blood in the ventricles and the larger arteries of both the systemic and pulmonary trees. This accounted for a large part of the ballistocardiogram but

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not for all of it. In the present paper our estimate of the contribution of changing amounts of blood in the pulmonary arteries to the internal movement of the center of gravity has been improved, new data permitting a more accurate estimate. And in this new study we have also calculated the contributions of the changing volume of the small arteries, of the refilling of the heart, of the contractions of the atria and, finally, of the movement of the heart wall itself. The effect of soft tissue motion is also discussed. So the studies reported in this paper present a more complete theory by covering not only the systolic and diastolic but also the presystolic waves of the ballistocardiogram.

In calculating the contribution of each of these items to the movement of the center of gravity within the body, and so to the ballistocardiogram, the same method has always been used. The change in mass at each position at every instance of the cardiac cycle is estimated for every location studied. This is multiplied by the distance of that location to an imaginary plane, perpendicular to the longitudinal axis, and passing through the heart; it is indicated by the line shown passing through the upper body in figure 1. The curves thus obtained for each location are aligned in time and the values for each instant added together to form a composite curve that represents the movement of the body’s center of gravity within the body during the cardiac cycle, assuming that no deformation occurs in the transfer from circulatory system to body frame. This theoretical curve is to be directly compared with the record of the ultra-low frequency ballistocardiograph when displacement is recorded; its first derivative is to be compared with the record of the same instrument when velocity is recorded; its second derivative is to be compared with “force” ballistocardiograms, i.e., the ultra-low frequency record when acceleration is recorded, and the high-frequency ballistocardiogram when displacement is recorded.

The newer contributions to the theory are now taken up in order.

Contribution of the Lesser Circulation

According to the anatomic data on which we previously relied, the relation between the thickness of the wall and the internal radius of the arteries belonging to the lesser circulation is the same as that found in the systemic circulation. Since there was some doubt about the validity of this statement, we made some measurements on the wall thickness of the pulmonary artery and of the ascending aorta in human cadavers. The results suggested that if the thickness of the wall of the pulmonary artery is assumed to equal around 55 per cent of the thickness of the wall in a corresponding location in the aorta, more accurate results would be secured.

Because of the lack of data on the elasticity of the pulmonary arteries, in a previous
In the publication we applied to the pulmonary arteries the same modulus of elasticity $E$ (Young's modulus) that we had used for the systemic tree, namely $E = 4.0 \times 10^6$ Gm. cm.$^{-1}$ sec.$^{-2}$. Recently we have obtained some new data from direct measurements of the elasticity of the pulmonary vessels of cadavers, which made us conclude that the value we had previously used was too high. From Lawton's measurements on excised strips of the human systemic arteries, in which he applied a stress that was a periodic function of time, we calculated the modulus of elasticity. The average value for $E$ turned out to be 3.8, confirming our earlier choice excellently. But from his measurements on the pulmonary artery of a monkey we calculated $E = 2.0$, half the value we used. Moreover, Lawton performed some measurements on the pulmonary and the systemic arteries of one dog, which resulted in values of 4.5 and 6.0, respectively. The latter value is much higher than the values he secured previously from measurements on several dogs, which show an average of 4.8. But we may conclude that the pulmonary artery has a modulus of elasticity considerably smaller than that of the systemic arteries. In this dog the ratio was 1:13.

Besides these recent data we found that static measurements of this modulus had been made by Hochrein several years ago. He measured the elasticity of excised segments of human arteries as well as that of excised strips, but most of his measurements were made with internal pressures larger than those found during life. Hochrein's results show extremely high values for the modulus; the ratio between the moduli for the pulmonary artery and the aorta at normal blood pressures may roughly be approximated as 1:6 from one paper and 1:3 from another.

Table 1

Survey of Anatomic and Physiologic Data Used for Calculating Changing Blood Volume in Pulmonary Arteries during Cardiac Cycle

<table>
<thead>
<tr>
<th>No. of artery</th>
<th>$R$ (cm.)</th>
<th>$r'$ (cm.)</th>
<th>$(E-E')$ (cm.)</th>
<th>$a$ (cm.$^2$)</th>
<th>$S$ (cm.$^2$)</th>
<th>$dS$ (cm.$^2$ sec.$^{-2}$)</th>
<th>Length (cm.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>28</td>
<td>1.40</td>
<td>0.80</td>
<td>1.32</td>
<td>16.5</td>
<td>5.47</td>
<td>74</td>
<td>1.9</td>
</tr>
<tr>
<td>29A</td>
<td>0.95</td>
<td>0.059</td>
<td>0.89</td>
<td>15.1</td>
<td>2.50</td>
<td>31</td>
<td>6.2</td>
</tr>
<tr>
<td>29B</td>
<td>1.05</td>
<td>0.063</td>
<td>0.99</td>
<td>15.7</td>
<td>3.10</td>
<td>40</td>
<td>10</td>
</tr>
</tbody>
</table>

$R$ and $r$ are the external and internal radius, respectively; $S$ stands for the internal cross-sectional area; and $p$ for the pressure. The Poisson ratio of the arterial wall material is assumed to be 0.5. The numbers of the arteries are the same as in reference 4.
The contribution of the right ventricle and the pulmonary arteries to the ultra-low frequency displacement ballistocardiogram as calculated previously (1) and as revised (2). The contribution of left ventricle and systemic arteries as calculated previously (3). The heavy black line gives the total predicted displacement ballistocardiogram due to left and right ventricular and arterial activity (sum of 2 and 3).

previously is marked 1, that with the improved data is marked 2, and is significantly larger, as could be expected from the direction of the change in the numerical values. The left ventricular and systemic contribution is shown separately in figure 2 (marked 3). The heavy black line gives the total predicted movement due to left and right ventricular and arterial activity.2 The effect of movement of blood in systemic vessels is conspicuously larger than that of movement in the lesser circulatory system.

Contribution of Movement of Blood in the Small Systemic Arteries

In the preceding study of blood movement in pulmonary vessels1 and in published papers on that in systemic vessels1,2 the contribution to the ballistocardiogram of pulsatile changes in blood content of the arteries with an internal diameter smaller than 0.22 cm. was not taken into account. It is evident that the rhythmic change in blood content of any small artery with the arrival and departure of the pulse wave must be negligible in comparison to that occurring in a large artery. Since the small arteries so outnumber the large arteries, their total effect is not necessarily close to zero. So an attempt was made to get an idea of the contribution to the motion of the internal center of gravity due to the mass of blood pulsating in the small arteries. Because of lack of reliable data in man the result of the computation must inevitably be a first-order approximation only.

The line of thought that was presented earlier1,4 will also be applied here. Just before the arrival of the pulse there is a certain amount of blood within each segment of artery. With the increase of pressure that marks the pulse the volume of blood contained in each segment increases. The volume of blood above the diastolic volume level, $\Delta V$ at any instant can be estimated from the increase in the internal pressure above the diastolic pressure level at that instant, $\Delta p$ by the following equation:

$$\Delta V = \frac{3V(a + 1)^2}{E(2a + 1)} \Delta p$$  \hspace{0.5cm} (1)

$V$ is the volume of the considered artery, $E$ the modulus of elasticity (Young's modulus) of its wall material, and $a$ the ratio between the internal radius and the thickness of the wall.

Since it is impossible to carry out this calculation for each small artery in the body, some means must be found to deal with them in groups. It should be kept in mind, however, that the pulse wave does not arrive in all parts of the arterial bed at the same time; e.g., it arrives at the coronaries before it reaches the arteries of the calf. In order to take into account the time of arrival the small arteries were divided into groups according to their general location in the human body. The average delay of the pulse in each region of the body had been calculated previously,1 and the same values were used in this study.

The form and amplitude of the pressure wave in the small arteries of each region were assumed to be equal to those in the larger arteries4 supplying that special section. Since
The contribution of the right ventricle and the pulmonary arteries to the ultra-low frequency velocity ballistocardiogram as calculated previously (1) and revised (2). The heavy line shows the total predicted velocity ballistocardiogram due to left and right ventricular and arterial activity.

The effective length of the small arteries has been reported to be very small (Green), we did not correct for the time required for the pulse to traverse these vessels.

The many small arteries comprised in this estimation have of course a whole range of values of internal radii and wall thicknesses, the ratio of which (a) appears in the formula given above. A study of the data compiled from the literature by Horeman indicates that three is a reasonable average value for this ratio in the smaller arteries. The value of $E$ was assumed to be the same as for the large systemic arteries, namely $E = 4.10^6$ Gm. cm.$^{-1}$ sec.$^{-2}$, no other data being available.

The remaining values needed to solve equation (1) are those of the diastolic volumes $V$ for the small arteries of the different sections of the body. Green gives data about the distribution of blood in a 13-Kg. dog arranged according to the order of the branches. He estimates the volume of blood contained by the secondary branches up to the terminal branches to be 4 per cent of the total blood volume. Because of lack of similar data concerning the human arteries we assumed that the smaller arteries of man would contain the same percentage of the total blood volume as they do in the dog, so in our computations $V$ equals about 200 cm.$^3$. But what we actually needed was the value of $V$ for the different sections separately. To approximate these amounts the distribution of the 200 cm.$^3$ of blood over the various regions of the body was
assumed to be proportional to the blood flow through those regions. Data are available concerning these flows. Table 2 lists the values (and thus the proportions) used and the sources from which they were taken. Slightly smoothed-out plots of the results are displayed as light lines in figures 5 to 7. The heavy lines depict the new total estimate, which equals the sum of the predicted total curves given in figures 2 to 4, and of the values plotted as light lines in figures 5 to 7. The contribution of the changing volume of blood in the smaller arteries turns out to be much smaller than that of the large arteries, as was expected. But it amounts to roughly 20 per cent of the total effect on the ballistocardiogram.

The anatomic arrangement of the lobes of the lungs close around the heart suggests that the contribution of the small pulmonary vessels will be so much smaller than that of the corresponding systemic arteries that no estimate was attempted.

### Table 2

<table>
<thead>
<tr>
<th>Regions</th>
<th>Flow (cm³/min.)</th>
<th>Diast. blood vol. in regions (cm³)</th>
<th>Average time delay in arrival of pulse wave (m. sec.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart muscle</td>
<td>250³⁺, ²⁸</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>Head</td>
<td>750³⁺, ²⁸</td>
<td>30</td>
<td>40</td>
</tr>
<tr>
<td>Intercostals</td>
<td>150</td>
<td>6</td>
<td>25</td>
</tr>
<tr>
<td>Hepatic-portal</td>
<td>1500³⁺, ²⁸</td>
<td>60</td>
<td>65</td>
</tr>
<tr>
<td>System</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renals</td>
<td>1250³⁺</td>
<td>50</td>
<td>65</td>
</tr>
<tr>
<td>Legs</td>
<td>850³⁺</td>
<td>34</td>
<td>170</td>
</tr>
<tr>
<td>Arms</td>
<td>250³⁺</td>
<td>10</td>
<td>110</td>
</tr>
<tr>
<td>Total</td>
<td>5000</td>
<td>200</td>
<td></td>
</tr>
</tbody>
</table>

The small arterial vessels are subdivided in regions as listed in the first column. The flow through these regions and their assumed diastolic blood volume are given in columns two and three, and the average time delay before the arrival of the pulse wave after the opening of the aortic valves (reference 1) is summed up in the last column.

**Contribution of Movement of Blood During Cardiac Filling and Atrial Contraction and of Movement of the Heart Itself**

From the time relations it seems obvious that the movement of blood in the arterial trees is not responsible for anything that occurs in the ballistocardiogram just before the first great downstroke, the H-I segment in the force record. Therefore one should look into the events occurring on the venous side, the changes in atrial and ventricular volume during filling, the effect of atrial contraction and the movement of the heart itself, for an explanation of the initial movements in the ballistocardiogram.

A large quantity of information has been gathered about the mechanical activity of the human and mammalian heart from volume and pressure curves, phonocardiograms, electrokymograms, and densigrams,¹⁷, ¹⁸ together with curves displaying the electrical activity. The introduction of new direct methods of measurement by Rushmer et al.¹⁹ has recently provided another source of information concerning cardiac contraction. But not much has been done since Wiggers²⁰ to correlate the various cardiac events, which can be measured...
quantitatively, in order to thus obtain a more general picture of the normal human heart’s behavior.

Nylin\textsuperscript{21} estimated that the residual blood volume in the heart of a recumbent subject averages around 350 cm\textsuperscript{3}. We assumed that 150 cm\textsuperscript{3} is stored in each ventricle and 25 cm\textsuperscript{3} in each atrium. If we assume a stroke volume of 75 cm\textsuperscript{3}, this estimate together with Wig- gers’ curve of the change in ventricular volume gives the value of the ventricular blood volume $V_{ventr}$ at each instant of time. We then assumed that the ventricle has a spherical shape during ejection. The internal radius of the ventricle $r_{ventr}$ during systole with regard to time can then be computed readily from

$$V_{ventr} = \frac{4}{3} \pi r_{ventr}^3.$$}

This gives us the distance between the center of gravity of the ventricle and the annulus fibrosus during ejection. Since it is very likely, however, that the ventricle assumes a less globular form during diastole, this distance cannot be determined from the ventricular volume only. In order to obtain an approximate value we used in addition the mean ventricular electrokymogram, Rushmer’s\textsuperscript{19} direct measurements of the ventricle’s size and Chailllet’s\textsuperscript{22} observations on the motion of the mitral valves. We thus obtained a complete time course of the distance
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between the center of gravity of the ventricle and the annulus fibrosus, which is given as "r_{ventr}" in figure 8.

We were unable to find data on the change in atrial volume with respect to time during the cardiac cycle. But we can get a rough estimate of it in the following manner. The changes in size of both ventricle and atrium together with the motion of the center of gravity of the ventricle inside the thorax determine the movement of the atrial border seen in the atrial electrokymogram, the form of which is well known. We cannot, however, find both quantities from one curve, since we are dealing with a problem of two unknowns obeying one equation. The difficulty lies in distinguishing movement of the atrial border due to contraction and relaxation of the atrium from that due to movement of the heart as a whole. Once the aortic and pulmonic valves have opened the volume of each ventricle diminishes, first rapidly and then slowly. What point of the ventricle may be considered to remain stationary during the process of ejection is a question about which opinions have clashed for centuries. By some the annulus fibrosus, by others the apex has been considered to be fixed in position. Evidence presented by Brecher\textsuperscript{22} both from previous investigations and from his own research supports strongly the conception that the atrioventricular junction moves footward during ejection, thus enlarging the atria. Altmann,\textsuperscript{24} in describing the genesis of the venous pulse, interprets the so-called venous collapse (X-valley) by descent of the atrioventricular junction. Observations of movements in opposite directions of base and apex in cases of calcification in the base are described in the same monograph; Altmann reports excursions up to 3 cm. Also, Wolferth and Margolies\textsuperscript{25} present quantitative proof that the left heart valve area moves footward, while the apex moves headward during contraction with total excursions of 4.5 and 13 mm, respectively; the outer border of the left ventricle was observed to move first outward and then inward, with a total excursion of 4 mm. Sundberg\textsuperscript{26} calculated that 85 per cent of the decrease in ventricular volume can be accounted for by the movement of the base toward the apex. The discordant elements in these observations are doubtless to be attributed to abnormal cardiac movement, as many
of the hearts studied were clearly abnormal.

Searching for what unity there was in these diverse data, we assumed, as an average, that when the normal ventricle contracts the displacement of the base is twice as great as that of the apex. By means of such an assumption one can estimate the motion of the center of gravity quantitatively and so of the heart as a whole. Knowing this we then can determine the change in radius of the atrium from data secured by the electrokymogram. The residual volume being known, the radius and the volume of the atrium can also be calculated (fig. 8).

The change in volume of the atrium plus the uptake of the ventricle determines the venous return, so that the total uptake from the veins, i.e., the inflow into the heart, can be readily calculated. These results are also plotted in figure 8 as "total inflow" and "atrial inflow." This calculated atrial inflow curve can be compared with the few experimental data available. Brecher\textsuperscript{29} has reported measurements made by a bristle flow-meter inserted into the vena cava in closed-chest experiments in dogs. The tracings Brecher obtained experimentally show one small negative and two high positive peaks in about the same phase of the cardiac cycle as in our computed curve. Brecher also found the second positive peak occurring during the ventricle's rapid filling phase to be higher than the first, as in our results. Müller and Shillingford\textsuperscript{27} published a similar tracing taken on a normal subject. This curve shows the same general form as in our results except that the second positive peak is lower than the first.

The aspects of cardiac performance that we have just computed can be compared with well-known changes occurring during the cardiac cycle in figure 8. The vertical lines indicate the transitions of the various phases from one to another during the heart cycle, a method of showing time relationships derived from Wiggers.\textsuperscript{28} The electrocardiogram, phonocardiogram, atrial and ventricular pressures, and change in ventricular volume were drawn in according to Wiggers' data.\textsuperscript{29} An average venous pulse was derived from curves presented by Wiggers,\textsuperscript{29} Molhuyzen,\textsuperscript{29} and Altmann.\textsuperscript{24}

The time relations thus established, we are now in a position to compute the effect on the ballistocardiogram of atrial filling and contraction and of the movement of the heart itself. This was estimated as follows:

The contribution of an atrium equals its mass of blood $\rho V_{atr}$, $\rho$ being the density of the blood and $V_{atr}$ the blood volume contained in that atrium, augmented with $m_{mu}$ the mass of the muscle layer surrounding the blood and multiplied by the distance between the center of gravity of the atrium and reference plane $-(r_{ventr} + \frac{3}{8} r_{atr}) + p$. The factor $\frac{3}{8}$ before $r_{atr}$ is added because the atrium is considered to be a hemisphere. The minus sign is added because of the headward position of the atrium with respect to the reference plane. The
quantity \( p \) is the displacement of the ventricle's center of gravity caused by its special mode of contraction. Thus this contribution equals:

\[
(p_{\text{atrium}} + m_{\text{mus.atrium}}) \left( -r_{\text{ventr}} - \frac{3}{8} r_{\text{atrium}} + p \right)
\]

(2)

Since the center of gravity of each ventricle moves with respect to the reference plane we find another contribution in the same way:

\[
(p_{\text{ventr}} + m_{\text{mus.ventr}}) p
\]

(3)

In the calculation of the total contribution to the ballistocardiogram we have multiplied the sum of products (2) and (3) in the first place by a factor 2 to take care of left and right heart sides, which are thus assumed to behave identically except for their internal pressures, and, in the second place, since the heart's longitudinal axis forms an angle of about 45 degrees with the body's longitudinal axis, by another factor \( \cos 45^\circ = \frac{1}{2} \sqrt{2} \). The total contribution reads therefore

\[
\text{BCG contribution} = \left\{ - (p_{\text{atrium}} + m_{\text{mus.atrium}}) \right\} \left( r_{\text{ventr}} + \frac{3}{8} r_{\text{atrium}} + p (p_{\text{ventr}} + m_{\text{mus.ventr}} + p_{\text{atrium}} + m_{\text{mus.atrium}}) \right) \times 2.5 \sqrt{2}
\]

(4)

The mean masses of the muscle layer of an atrium and of a ventricle are taken as 25 and 140 Gm., respectively, in accordance with Vierordt's data.

Figure 9 shows the calculated contribution to the ballistocardiogram of the filling and emptying of the heart and its motion as a light line. The heavy line represents the calculated total displacement curve, the sum of all the contributions studied. The calculated results for velocity and acceleration curves are given in figures 10 and 11. Figure 12 shows our final results together with average experimental tracings reported by Scarborough et al. and by Rappaport.

Discussion

Figure 12 shows the quite surprising correspondence between the calculated ballistocardiogram and records secured experimentally by the ultra-low frequency ballistocardiograph. Agreement between the direction and the quantitative magnitude of the waves and their relations to one another is as good as one could possibly hope for.

The most conspicuous deviation is in the timing. Thus, in Scarborough's data the second sound is synchronous with the tip of the L wave of the ultra-low frequency 'force' ballistocardiogram; in the theoretical ballistocardiogram the second sound occurs 50 milliseconds later. The reason for these small differences may well be caused by a slightly wrong assumption of the value of the elastic properties of the arterial wall material, i.e., in the value of Young's modulus of elasticity.
Theoretical study throws light on the genesis of the individual waves of the ballistocardiogram. The I and J waves are caused principally by movement of blood in the great arteries of the systemic circulation; the contribution of blood in the pulmonary system is much smaller. Although the presented improvements in the computation of the pulmonary effect have increased the estimate it is still somewhat lower than the values secured by Starr in experiments on cadavers. One should also note that, although the influence assigned to the pulmonary circulation is small, if blood movement in this circulation becomes asynchronous with that in the greater circulation, the forces developed in the pulmonary circulation would be sufficient to cause deep notching in the force ballistocardiogram, as is frequently observed in ultra-low frequency records.

Movement of blood in the small arteries plays a small role in the ballistocardiogram, but is responsible for some 20 per cent of the total effect, which is certainly not negligible.

In discussing the origin of individual waves one must remember that a number of events act simultaneously throughout the cardiac cycle and that a wave tip merely means that the sum of several events is maximal or minimal. Therefore a tip or inflection of the record can, in general, not be attributed to a single physiologic occurrence, which can be done fairly well in the electrocardiogram, although one event may be mainly responsible.
One must recall that a small change of blood distribution in the legs, because of the greater distance from the body's center of gravity, is very effective in altering the position of the center of gravity within the body, and thus altering the ballistocardiogram. The theoretical study supports the generally accepted views of the genesis of the I and J waves of the force record, but it has given new understanding to the waves of the terminal complex L, M, and N. Though these occur in diastole they are not to be attributed solely to cardiac filling but also to the changes in blood distributions as the descending limb of the pulse wave reaches the peripheral vessels.

The physiologic origin of the presystolic waves of the ballistocardiogram has been more uncertain and more debated than any other part of the record. The calculations suggest that, in the ultra-low frequency displacement record the "G" wave is chiefly due to the shift of blood from atria to ventricles accompanying atrial contraction. In the initial part of the "H" and "J" waves, the ejection of blood headward and the displacement of the heart footward compete, bringing about the "I" wave, which is better seen in experimental tracings than in our calculated curves. In the force tracing the GH upstroke precedes ejection and is mainly due to isometric contraction of the ventricles. Ejection begins at the tip of the H wave.

More details can easily be obtained from a comparison of figure 8, depicting the hemodynamic events, and figures 9 to 12, in which the calculated results are plotted.

While the normal ballistocardiogram is thus accounted for theoretically in quantitative terms, we must discuss several other possible effects, which, though probably having only a small effect on normal records, might under pathologic conditions play a considerable role in the ballistocardiogram.

A changing quantity of blood in the central veins would make some contribution to the ballistocardiogram. We could not, however, find sufficient data on which an estimate of the magnitude of this effect could be based. Certainly effects on the body's center of gravity due to changes of blood volume in the superior and inferior venae cavae would tend to cancel each other out, and the central location of the great veins would cause changes of their blood volume to have small influence on the ballistocardiogram. But if the great veins were congested, such effects might be larger.

When an artery changes its volume, surrounding tissue of about the same density as blood must move. It is this movement that is finally transferred to the skin. For reasons of symmetry the effect of this tissue movement on the ballistocardiogram is probably negligible. On the other hand, when the heart changes its volume or its position, or both, lung tissue with a much smaller density is caused to move. This makes the contribution to the ballistocardiogram as calculated in this paper give an estimate that will be somewhat too high.

In this paper it is assumed that there is no deformation in the transmission from the cardiovascular system to the body frame, an assumption that is not likely to be a suitable first order approximation for the higher frequency components of the force ballistocardiogram. This transmission acts as a low-pass filter.

**Conclusion**

The greater part of the normal ultra-low frequency ballistocardiogram, when displacement, velocity, and acceleration are recorded, can be accounted for quantitatively by known hemodynamic events. Although several assumptions have had to be made in the course of the computations, the agreement between the computed curves and those obtained experimentally on healthy subjects appears to be as close as one could possibly expect, except for some difference in timing.

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