Genesis of Displacement of the Human Longitudinal Ballistocardiogram from the Changing Blood Distribution*

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SINCE THE discovery of ballistocardiography about eighty years ago, it has been clear that events related to the heart's mechanical action were responsible for the body's movement.[1] During the development of this field of research it gradually became clear which physical quantities concerning the internal events are measured with the various current types of ballistocardiographs.[2,3] It has generally been recognized nowadays that the ultra-low frequency ballistocardiograph is a suitable instrument to provide data on three related aspects of the over-all circulatory effects, namely, the displacement, velocity (speed) and acceleration of the internal periodical motion of the center of gravity caused by the action of the heart. These quantities are secured from the ultra-low frequency system by recording its displacement, velocity and acceleration, respectively.[2–5]

The quantitative relationship between the events occurring inside the body and the recorded curves is an old problem. The earliest quantitative investigation concerning the relationship between the internal occurrences and the human acceleration (force) tracing was carried out by Starr and Rawson in 1941[6] applying Machella's data. A similar method of prediction of the force trace was later used by Hamilton and co-workers[7] applying their data secured from previous calculations.

This paper will deal with the prediction of all three aspects of the movement of the human center of gravity [displacement, velocity and acceleration (force)] by means of a new and different approach to the problem.

METHOD OF APPROACH AND RESULTS

Events taking place within the circulation change the distribution of the mass within the body with each beat of the heart. In the first place the right and left ventricles eject their stroke volumes into the corresponding arteries during systole, which is only a fraction of one heart cycle, while the run-off from the arterial to the venous beds goes on during the complete heart cycle; thus the quantity of blood in the systemic and pulmonary beds changes continuously with regard to time. Besides this the quantity of blood in the auricles and in the central veins also changes periodically. Moreover, the movement of the heart, of the large vessels and of the soft tissue in which they are embedded may contribute to a changing mass distribution inside the body. The total change in mass distribution may obviously be described as the movement of the center of gravity within the body,[8] and the result of this movement may be recorded in terms of the displacement, velocity or acceleration of the body itself.

In this paper we shall focus our attention on the effect originated by the changing blood content of the ventricles and of the larger arteries in both the arterial trees. When the right and left heart start to eject their stroke volumes into the

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corresponding arteries, the blood flowing into the root of these vessels causes the blood pressure to increase rapidly and the arteries to distend, first at their orifices. The pressure rise and distention thus created run along the arteries in the distal direction; wherever the pressure (pulse) wave appears, the lumen of the vessel increases, while it decreases as soon as the pressure wave passes by. Thus during the cardiac cycle the distribution of mass in the body is altered, first by the changing volume in the ventricles and then by the wave of changing volume progressing down the arteries. The relation between the flow in the arteries, and the familiar pressure wave (the pulse) is, however, not a simple one. In general there is a time (phase) difference between them.[9]

From these considerations it may be concluded that the movement of the center of gravity (the ballistocardiogram) can be estimated from the flow patterns within the circulation. This method of approach, however, requires not only knowledge of the flow patterns at the aortic and pulmonary valves, but also of flow patterns throughout the circulation. Moreover, the relation between flow and pressure (distention) must also be known. A simpler way of approach follows from the same considerations. The degree of distension of the arteries is related to the pressure curves found in the various arteries, and since the latter is well known, the former can be determined if one has knowledge of the elasticity. This short-cut method does not require knowledge concerning the ejection curve of the heart and the flow curves in the arteries.

Method Based on Ventricular Ejection Curves: The first method was, as the original attempt, applied by Starr and Rawson in 1941.[6] These authors used the ejection curve as recorded by Machella[10] (in dogs) and derived acceleration and deceleration of the blood from this curve mathematically. They divided the aorta and the pulmonary artery into small segments. The masses contained by these segments were multiplied by the values of the acceleration and deceleration of the blood. Alignment according to the pulse wave velocity and summation of the results provided an estimate of the contributions of the aorta and pulmonary artery. Addition of the ventricular effect gave an estimate of the acceleration (force) ballistocardiogram. The authors concluded that the ballistic curve is mainly due to ventricular and aortic effects, while the pulmonary contribution turned out to be a very minor one.

Hamilton *et al.*,[7] who objected to Starr's application of Machella's results, used a similar method of prediction of the force ballistocardiogram, but applied ejection curves estimated from previous calculations.[11] Hamilton and co-workers also concluded that the force ballistocardiogram must be mainly attributed to ventricular and aortic occurrences.

Since these investigations more evidence has been produced suggesting strongly that the ballistocardiographic tracings are mainly due to ventricular and arterial events (Starr's cadaver experiments,[12] Burger,[8,13] Elsbach,[4] Dock[14]), though there is not yet complete agreement (Cossio,[15] and Thomas,[16] dog experiments).

Because of this conflict of evidence we have attempted to predict the three aspects of the internal movement of the center of gravity by applying the above-mentioned second method of approach, which has the advantage that the well-known pressure tracings can be used for the various portions of the arterial trees, the elasticity of which can also be estimated. An extensive description of our method of calculation has been given previously[8,17,18] and only the line of thought will be related here.

Method Based on Pressure and Filling of Various Arteries: The larger arteries (down to an internal radius of 1.1 mm) of both arterial trees, summed up in Table I, were subdivided into small segments. The mass of blood above the end-diastolic level—the excess mass—in each of these was calculated as a function of time with intervals of 20 milliseconds. Multiplication of the excess masses with the appropriate distances to a reference plane passed arbitrarily through the center of both ventricles, and summation of the results, gives an estimate of the expected displacement of the center of gravity as far as this is caused by the events inside the ventricles and the larger arteries. The result is shown as the heavy line in Figure 1, marked D-BCG, instant zero being taken as the instant of opening of the

TABLE I

The Arteries in Which the Changing Blood Content is Taken into Consideration. If the Artery Is Paired Both Are Used

I	VI
Aorta ascendens	A. brachialis
Arcus aortae	A. ulnaris
II	A. radialis
Aorta thoracalis	A. interossea volaris
Aorta abdominalis	VII
III	A. coelica
A. iliaca communis	A. gastrica sinistra
A. iliaca externa	A. lienalis
A. profundis femoris	A. hepatica
A. femoralis	A. renalis
A. poplitea	A. mesenterica sup.
IV	A. mesenterica inf.
A. tibialis posterior	VIII
A. tibialis anterior	A. carotis communis sin.
V	A. carotis communis dex.
A. anonyma	A. vertebralis
A. subclavia	IX
A. axillaris	A. pulmonalis
	Ramus sinister (A. pulm.)
	Ramus dexter (A. pulm.)

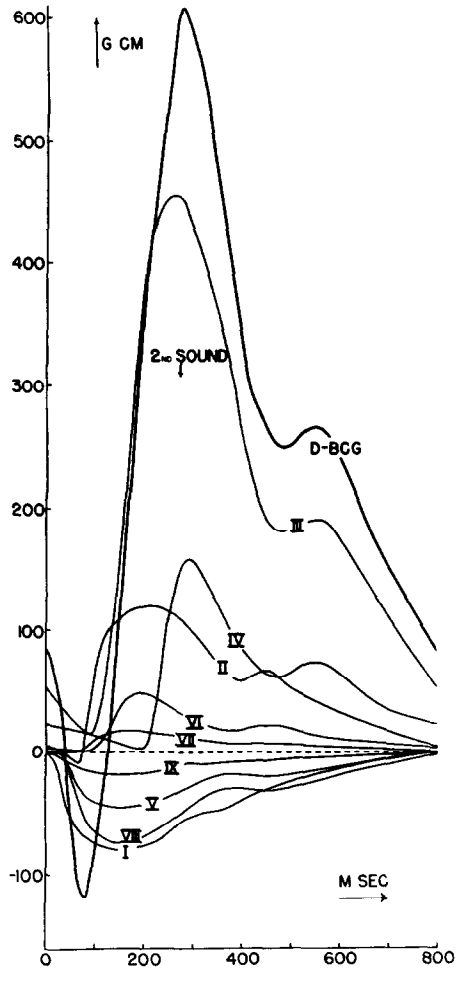

Fig. 1. The predicted longitudinal displacement ballistocardiogram is the heavy line (D-BCG). It is the sum of the contributions of various anatomic portions represented by lighter lines. They are marked by Roman numerals which are identified in Table I.

semilunar valves. The contributions of the various portions of the arterial trees are shown by lighter lines in the same figure. The heavy line is a plot of the sum of the lighter ones.

The other two aspects, velocity and acceleration of the center of gravity, depicted in Figures 2 and 3, have been secured from the data plotted in Figure 1, by differentiating once and twice respectively. The vertical scales of the predicted curves in Figures 1 to 3 should be divided by the total mass of subject and ballistocardiograph to find the amplitude of the predicted displacement, velocity, and acceleration curves. This has been done for displacement, velocity, and acceleration by taking a mass of 75 kg, which is a reasonable average total mass for a healthy person plus an ultra-low frequency ballistocardiograph. The composite plot is given in Figure 4 together with average experimental traces of healthy persons.[19] The amplitudes of the predicted curves are in good agreement with the experimental results.[3,13,20–22]

Not only the peak-to-peak values but also the general shape of the predicted and experimental curves are essentially the same. The analogy holds for the relationships with other phenomena related to the action of the heart as for instance the heart sounds (Fig. 4; see Rappaport[23] for another composite sketch of experimental tracings).

In our method of prediction of the human ballistocardiogram only the contribution of the ventricles and of the larger arteries of both arterial trees were taken into consideration and the great similarity between theoretical and experimental results, which emerged, led us to the conclusion that the normal ballistocardiogram must be attributed mainly to these circulatory events.

By calculating the volume changes in the

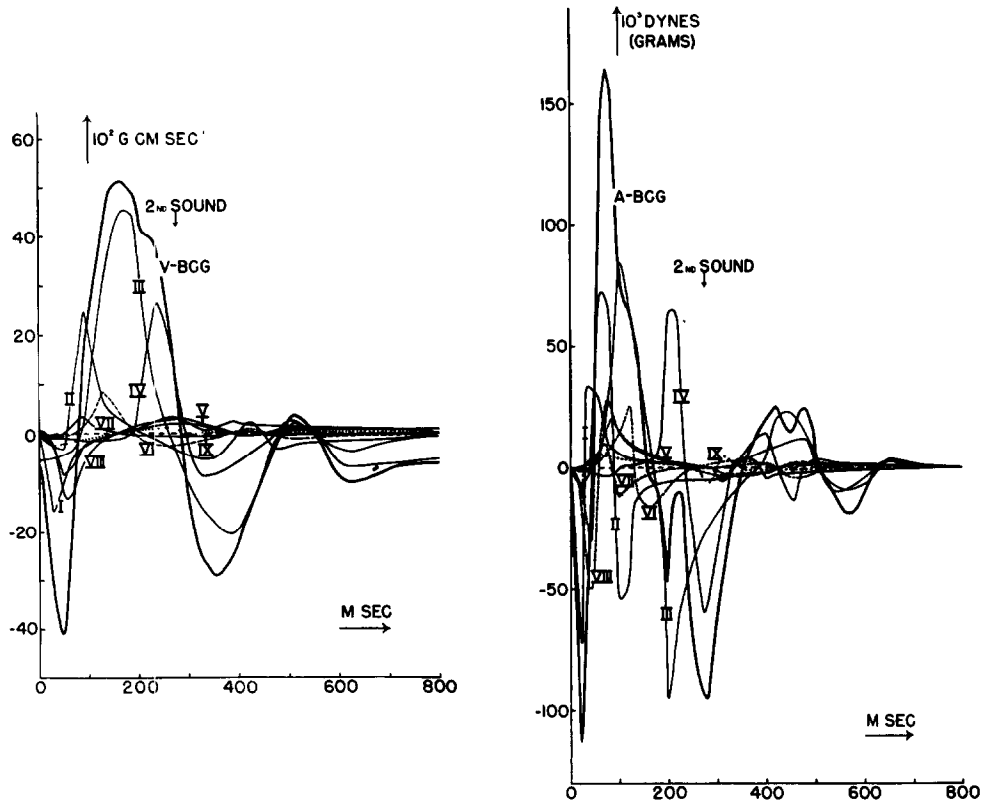

Fig. 2 (left). The predicted longitudinal velocity ballistocardiogram (V-BCG). The lighter lines show the contributions of the various anatomic portions. Fig. 3 (right). The predicted longitudinal acceleration (force) ballistocardiogram (A-BCG). It is the sum of the contributions of the various parts of the arterial trees plotted as light lines.

larger arteries the excess masses present in the limbs at any time can be estimated, and so the plethysmograms of the larger extremities and the trunk can easily be predicted. This calculation was carried out for the forearm and the calf and the results were compared with plethysmograms which have been recorded. This has been discussed in detail elsewhere.[24,25] Our results show a good quantitative agreement between theoretic and experimental curves, and this provides us with an independent check on the validity of our method.

Our conclusion that the normal ballistocardiogram is mainly due to ventricular and arterial effects has also been reached by Klensch and Eger from model experiments in which they proved to be able to produce normally looking arterial pressure and ballistic curves[26,27] by injecting into tubes which simulated the anatomy and elasticity of the blood vessels.

The most striking difference between the experimental and theoretical curves (Fig. 4) is that there is no H wave in the predicted acceleration (force) curve. The H wave is also absent in ballistocardiograms secured in Starr's cadaver experiments in which he replaced the heart by a pump. Indeed, in records secured in living persons, the H wave precedes ventricular ejection and so cannot be due to ventricular systole.

Explanation of Ballistic Movements: It seems worthwhile to discuss the results plotted as the light lines in Figures 1 to 3 a little more closely. It should be noticed first that circulatory effects occurring headward or footward with respect to the reference plane passed through the ventricles by causing a movement of the body's center of gravity, cause the body to move in the opposite direction. Therefore in Figure 5 and in Table II the arteries are distinguished as "headward" and "footward" vessels.

Contribution of a Single Arterial Segment: Let

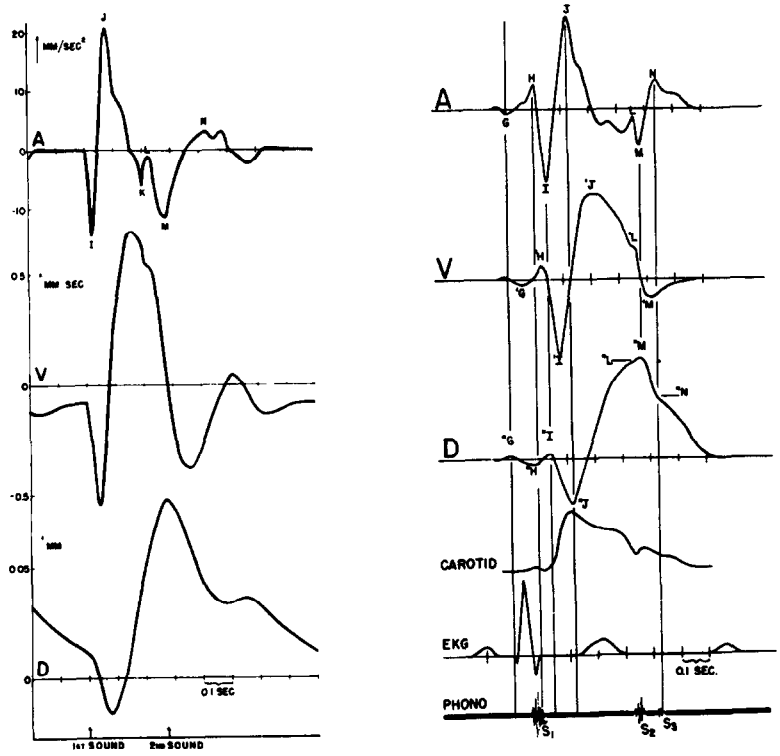

Fig. 4. The right-hand side is a reproduction of average normal acceleration (force, A), velocity (V) and displacement (D) ballistocardiograms secured from an ultra-low frequency ballistocardiograph and published in the Report of the Committee on BCG Terminology by Scarborough and Talbot (*Circulation* 14: 435, 1956; used by permission of the authors and of *Circulation*). The left-hand side pictures the predicted curves taken from Figures 1 to 3 (heavy lines) for reason of comparison.

us now consider the contribution of an arbitrary segment of artery, which is assumed to be so short that the same blood pressure governs throughout its length. A pressure curve, used for an artery with an internal radius of 3.4 mm, is drawn in Figure 5 (top), and is assumed to hold for this segment. This pressure curve gives also the time course of the quantity of blood above the end-diastolic levels. The middle curve depicts the rate (speed) of filling at each instant (first derivative of the top curve). The bottom curve depicts the rate of the change in filling (second derivative of top curve), which indicates acceleration and deceleration in quantity of blood above the end-diastolic level.

Opposite to the curves in Figure 5, the ballistocardiographic effect is indicated. The direction in which the *internal* center of gravity moves is given for "headward" and "footward" segments separately, the *body's* movement being in the opposite direction of the internal effect. When there is a quantity of blood above the diastolic level in a "headward" segment it contributes to a headward displacement of the center of gravity within the body (and the footward displacement of the body to keep its center of gravity unchanged in space). If the amount of blood above the diastolic level is increasing in a "headward" segment of artery (rate of filling positive, middle curve, 2–4; 6–8) then the velocity of the internal center of gravity is in the headward direction. It is in footward direction when the amount of blood above the diastolic level is decreasing (rate of filling negative; 1–2; 4–6; 8–10). The opposite holds for a "footward" segment. When the rate of change in filling is positive (bottom curve, 1–3; 5–7; 9–10) the internal force acts in a headward direction for a "headward" segment. In case

TABLE II

The Arrows Denote the Direction in Which the Mentioned Phenomena Affect the Movement of the Body Regarding Its Displacement, Velocity, and Acceleration. A Downward Pointing Arrow Thus Indicates Footward Effect, and Upward Pointing Arrow Headward Effect

	Displacement tracing	Velocity tracing	Acceleration tracing
"Headward" vessels	Quantity of blood above the diastolic level ↓	Increasing quantity above diastolic level ↓ Decreasing quantity above diastolic level ↑	Rate of change in filling positive ↓ Rate of change in filling negative ↑
"Footward" vessels	Quantity of blood above the diastolic level ↑	Increasing quantity above diastolic level ↑ Decreasing quantity above diastolic level ↓	Rate of change in filling positive ↑ Rate of change in filling negative ↓

the rate of the change in filling is negative (3–5; 7–9), the internal force acts in a footward direction. Again the opposite holds for "footward" segments.

Contribution of Various Portions of Arterial Tree: After this discussion of the contribution of a single segment, the contributions of the various portions of the arterial trees may be looked at a little more closely. The contributions of these portions have been plotted separately in Figures 1 to 3, positive being headward movement of the body. Table II, summarizing Figure 5, may prove helpful in reading these plots. The arrows in Table II denote the direction in which the events in that portion contribute to the *body's* movement. The light lines in Figures 1 and 2 can be easily interpreted making use of Figure 5 and Table II. We shall discuss only the acceleration curves a little further because of the fact that they receive most attention nowadays (Figs. 3 and 4). The downstroke from the base line to the I peak is apparently mostly due to the immediate result of the heart's ejection of blood into the root of the aorta (positive rate of change in filling; Figure 3, I) as was concluded by Starr[22] and Hamilton.[7]

The most important contributions to constitute the I-J upstroke are initially the decrease in the rate of change in filling of the ascending aorta and of the arch (Fig. 3, I) and later the fact that the rate of change in filling of the thoracic and abdominal aorta is positive (Fig. 3, II). The decrease in the rate of change in filling of the thoracic and abdominal aorta and its increase in the iliacs and in the arteries of the upper parts of the legs are the most striking contributions to the first part of the J-K down stroke (Fig. 3, II, III), though the contributions of the other portions are of importance here. The latter part of the J-K downstroke turns out to be mainly caused by the rapidly decreasing rate of change in filling of the iliacs and the arteries of the upper parts of the legs (Fig. 3, III)

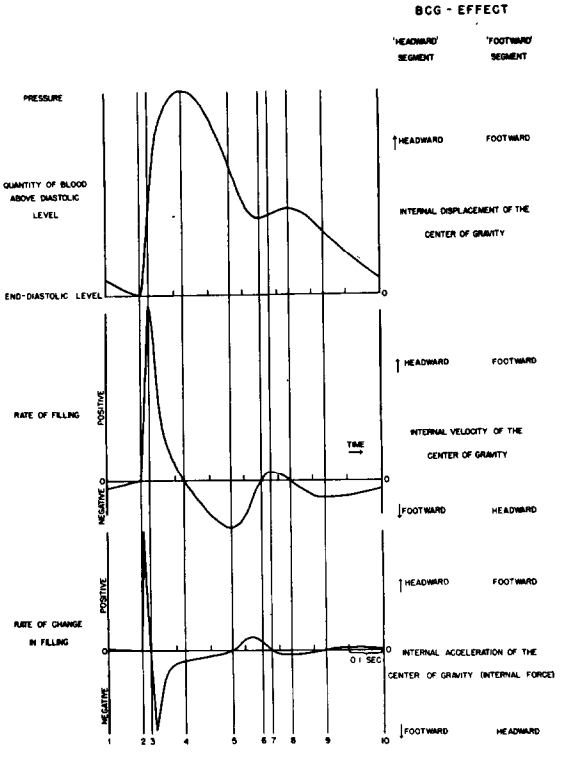

Fig. 5. Evaluation of the ballistocardiographic effects originated in a single segment of artery.

a conclusion also reached by Honig and Tenney.[29] Superposition of the negative rate of change in filling of the last mentioned portion and the positive rate of change in filling of the arteries in the lower parts of the legs (Fig. 3, IV) causes the K-L upstroke. Somewhat later the rate of change in filling of the arteries of the lower legs has turned negative. Superposition of this part of IV and the corresponding part of III causes for the greater part the M peak. For the remaining part of the curve a number of this type of events occurring in different portions are responsible.

This explanation is supported by a recent publication of Reeves and co-workers[28] in which they emphasize the time relationship between the peaks in the second time derivative of the carotid pressure tracing and those in the acceleration record of an ultra-low frequency system. They do not, however, take into account the fact that events in the different parts of the arterial system occur at different instants because of pulse propagation time.

Relation of Amplitude to Rate of Ejection: Starr has shown experimentally[32] that the amplitude of both the initial and terminal complexes depends on the time course of the ejection of blood. If the outflow is abrupt initially, the initial complex shows a large amplitude, whereas the amplitude is small when the initial outflow is gradual. This means that a great acceleration of the blood produces large amplitudes of the force tracing. This is also true for the terminal complexes. An abrupt ending of ejection causes large amplitudes; a gradual stop small ones (Fig. 6). These qualitative observations, that the time course of ejection both at the onset and at the end of ejection are of great importance, are in excellent agreement with our above-mentioned conclusions (compare Fig. 3). Starr's findings have been recently confirmed in model and cadaver experiments carried out by Honig and Tenney.[29] Moreover, the latter authors add the observation that late diastolic vibrations were absent only in those patients without audible evidence of aortic valve closure, which seems to give another confirmation of our theory.

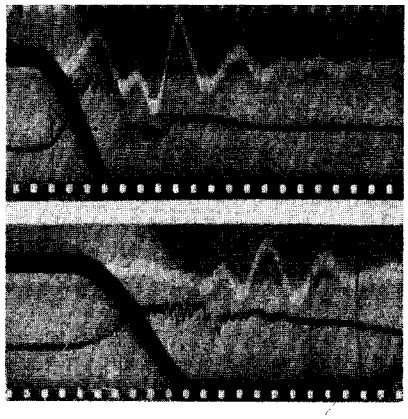

Fig. 6. An example of two simulated systoles in the same cadaver. The injected volume (heavy black line), the central aortic pressure (tiny black line) are recorded simultaneously with the ballistocardiographic force tracing (white line). The latter is recorded from an old type high-frequency ballistocardiograph. Note that the amplitude of the ballistic waves change when the time course of injection changes. (Courtesy of Dr. Isaac Starr).

Discussion

One would expect that if the ballistocardiogram is, for the greater part, based on the circulatory events in the ventricles and in the larger arteries, as is concluded in this paper, the ballistocardiographic phenomena would disappear after occlusion of the vessels immediately around the heart. Cossio[15] and later Thomas[16] and co-workers performed this experiment and observed that, though the pattern changed beyond recognition, the amplitude of the force tracing increased, thus suggesting that other than the above-mentioned circulatory events cause the ballistocardiogram. The most obvious interpretation was then that the heart's movement initiated the recorded tracings. On the other hand, Starr recorded normal traces, except for the lack of an H wave, while he simulated systole at necropsy by injecting into the aorta and pulmonary artery, although the heart was not beating. It was not until very recently that Cossio's experiments have been repeated with improved technics. Honig *et al.*[30] and Scarborough[31] secured the abolition of most of the ballistocardiogram in stopping the circulation while the heart kept beating. These experiments and Starr's critical review of the older dog experiments presented in a previous section of this seminar, seem likely to resolve the old controversy.

The results of the modern dog experiments, of

Klensch's model experiments, and of our quantitative prediction, make it seem very unlikely that the movement of the heart, venous flow, and atrial contraction contribute significantly to the ballistocardiogram, except in the earliest part of systole (H peak in the force trace), a statement which is supported by clinical observations and experiments in dogs (Dock,[14] Honig[30]).

As has been noted above, Starr *et al.* concluded both from their calculations[6] and from results secured in experiments in which they simulated systoles in cadavers,[32] that the contribution to the ballistocardiogram of the movement of blood in the pulmonary circulation is a minor one. To these findings Dock[33] objected that the pulmonary contribution had to be many times bigger than the systemic one in order to explain the respiratory variation in the amplitude and pattern of the ballistocardiogram seen in normal persons. The conception that the movement of blood in the pulmonary circulation makes only a minor contribution to the ballistocardiogram, is strongly supported by our calculation. It may be objected that lack of published information about the physical properties of the pulmonary circulation forced us to assume that these properties were similar to those of the systemic circulation. Because of this assumption, our estimation of events in the pulmonary circulation contains an element of doubt which does not apply to those made on the systemic circulation. Further investigations will be necessary to elucidate this point.

Conclusions

Displacement, velocity and acceleration of the internal center of gravity in the normal human body predicted from physiologic and anatomic data are presented. The predicted curves are essentially equal to the well-known results secured experimentally. It is concluded that the ballistocardiogram must be attributed mainly to the circulatory events occurring in the ventricles and in the larger arteries. The contributions of the various portions of the arterial trees, as estimated from the calculations, are displayed separately. Thus the physiologic events responsible for each part of the ballistocardiogram can be described quantitatively.

The results are critically compared with those obtained from investigations done by others.

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