Wide Frequency Range Force Ballistocardiogram
Its Correlation with Cardiovascular Dynamics

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The ballistocardiograms of young normal subjects as recorded with a wide frequency range technic are described. These records contain high frequency forces not previously described. An analysis of these components in terms of known cardiovascular events strongly suggests that they have definite dynamic significance. In addition, these correlations offer evidence as to the genesis of some forces in the ballistocardiogram.

FORCE ballistocardiograms from a number of different laboratories, utilizing very low frequency or aperiodic systems, have been described and are strikingly similar one to the other.1-3 The records are basically the same as those made from damped high frequency systems recorded as displacement4 and also from undamped direct body displacement when the oscillations induced by the external tissues of the body are electronically cancelled.5 The similarity of the force ballistocardiograms from such varied systems strongly supports the validity of the fundamental principles involved and heralds the end of an era of technical confusion and controversy.

Force ballistocardiograms recorded from this laboratory using a low frequency system of extended frequency range6 have been observed to differ markedly in high frequency detail from any previously described. These new force components have been found to have significant correlation with other events of the cardiac cycle. The observation that various cardiodynamic aberrations alter these forces in a predictable manner offers the possibility of a more rational interpretation of the ballistocardiogram. The present study was designed to verify the reproducibility of the force pattern in young normal subjects, and to examine the apparent correlation between the ballistic deflections and certain measurable events of the cardiac cycle.

METHODS

The force ballistocardiograms of 20 young normal subjects were recorded from the ultra low frequency system previously described.6 This ballistocardiograph is based on the principle of the horizontal pendulum and has a gross weight of 12 pounds. The motion of the bed is sensed as acceleration by a modification of the mercury-sulfuric acid capillary accelerometer, as described by Elliott and associates.7 The upper limit of the calculated linear frequency response of the entire system varies with the square root of the weight of the subject, and ranges from 30 c.p.s. for the lightest subjects to approximately 40 c.p.s. for the heaviest.8 The increased frequency response is in part dependent on the use of a footboard and shoulder straps, to increase the coupling of the body to the bed.

The electrocardiogram, carotid pulse wave, and heart sounds were simultaneously recorded with the ballistocardiogram by a Sanborn Poly-Viso Recorder. The carotid pulse was sensed with an Infra- ton* arterial pickup, using the Control Model E. The heart sounds were recorded by a Sanborn microphone into a Sanborn D-C amplifier. Frequencies over 60 c.p.s. are attenuated by this heart sound system, but the system is adequate for timing the major components of the heart sounds.

For each subject the intervals between the Q wave of the electrocardiogram and each of the consistent deflections of the ballistocardiogram were measured in 5 cycles, taken with respiration suspended at the end of normal expiration. The intervals between Q and the first major vibration of the first heart sound and the 2 major components of the second sound, the upstroke of the carotid pulse, the anacrotic shoulder of the carotid pulse, and the carotid incisura were similarly measured from the same cycles. To facilitate an understanding of the relationship

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between the carotid pressure pulse and the force ballistocardiogram, the carotid pulse wave was graphically differentiated in a few subjects. The pulse wave itself may be considered as a record of displacement, the first derivative as velocity, and the second derivative as acceleration of pressure change. The anacrotic shoulder of the carotid pulse was taken as the segment of most rapid slope change at the top of the initial upstroke. This was found to correspond to the point of maximal initial deceleration of the second derivation of the carotid pressure pulse.

RESULTS

The ballistocardiograms of normal young subjects are strikingly similar, showing consistent and repetitive wave forms. Figure 1 illustrates a typical complex drawn with all the points labeled. Figure 2 is an illustration of the tracings made on 4 young normal subjects with simultaneous electrocardiogram, heart sound, and carotid pressure pulse.

The terminology used subsequently in the description of the ballistocardiographic pattern is a modification of the conventional system, necessitated by the multiplicity of points that require names for descriptive purposes.*

It is not intended as a proposal for a final nomenclature, but rather is used to illustrate more clearly the concepts as to the genesis of the various deflections. The subscripts R or L are used when the peak force so labeled can be confidently ascribed to a definite event occurring in the right or left side of the circulation. A number of points are not labeled, since evidence as to their origin is not presently available.

The normal ballistocardiogram is characterized by a slow small inconstant footward force (the F-G wave) during early systole, which is interrupted by a headward force terminating at a sharp point (termed $H_R$) at .085 second ($\pm .010$) after Q. This is followed by a sharp footward deflection which is interrupted by a headward deflection that reaches a peak (termed $H_L$) at .116 second ($\pm .013$ after Q). This headward force is usually clearly defined, although in some tracings it is indicated only as a slur on the downstroke. From the $H_L$ point, the force vector resumes its footward direction to a nadir (termed $L_L$) at .142 second ($\pm .015$ second) after Q. Very close to this time in many normal records (36 out of the 100 normal complexes measured), there is a small headward force (I+). The major headward force that follows has 3 distinct components, the peaks of which are labeled $J_R$, $J_L$, and $J_D$. $J_R$ and $J_D$ are variable as to amplitude and timing, depending in $J_R$ on the relationship of the measured complex to the phase of the respiratory cycle. The time of these points from the Q wave are: $J_R$—.177 second $\pm .019$; $J_L$—.217 $\pm .019$ second; and $J_D$—.250 $\pm .027$ second. The $J_D$ point is present in about two-thirds of the complexes from the young normal subjects. Following the last J peak ($J_D$) there is a relatively slow decay in the headward force. This is followed by the K point, which is .305 $\pm .024$ second after the Q wave. The K point is variable as to position and time. Following this variable portion of the ballistocardiographic complex there is a sharp headward peak ($L_L$) occurring .39 $\pm .03$ second after Q. This is usually succeeded by 2 footward forces separated by a small but sharp headward peak ($L_R$) occurring .41 $\pm .03$ second after Q. Sometimes these 2 footward forces are apparently superimposed. The earliest diastolic force is slow and diphasic, with the first element

* We are indebted to Dr. Isaac Starr for helpful criticism relative to the nomenclature of the ballistocardiogram.
headward, while the remainder of the diastolic forces in normal subjects are inconstant and of small amplitude and low frequency. The atrial forces are normally of small magnitude and have not been considered here.

The time relationships of these points in 20 normal subjects are presented in table 1. Five complexes from each subject made during held expiration were measured. The mean, range, and standard deviation for each measured phenomenon are given. In addition, measurements from the onset of the QRS complex to the first major deflection of the first heart sound, the carotid upstroke, the carotid shoulder, the beginning of the second heart sound, and the beginning of the second component of the second heart sound are included. The first major deflection of the first heart sound begins at .056 ± .015 second after Q, and was observed to correlate well with the beginning of the G-H upstroke of the ballistocardiograph. The H$_R$ point was not found to correlate with the other events measured. The H$_L$ point in all cases preceded the carotid upstroke from .01 to .04 second (mean .017 ± .007 second). The I$_L$ point (.142 ± .015 second after Q) was found to correlate with the initial positive acceleration peak of the carotid pressure rise in a few subjects from whom these data were obtained by graphic differentiation of the carotid pressure pulse (figure 3). Furthermore, previous work from this laboratory has already established the time of this acceleration peak obtained from carotid pressure pulses in 10 normal subjects as occurring at .14 second after the Q.$^8$

Figure 3 shows a comparison of the ballistocardiograph, the carotid pulse, and the graph-

![Fig. 2. Representative records taken on 4 young normal subjects. The electrocardiogram, heart sounds, carotid pulse, and ballistocardiogram are shown. Notice the consistent patterns and the sharp points. Paper speed is 50 mm. per second. The heavy time lines are at .10-second intervals.](image)

**Table 1.** Ballistocardiographic Times and Correlation with Other Events

<table>
<thead>
<tr>
<th></th>
<th>Mean ± S.D.</th>
<th>Range</th>
<th>No. of Observations</th>
</tr>
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<tbody>
<tr>
<td>Q-S$_1$</td>
<td>.055 ± .015</td>
<td>.03–.09</td>
<td>100</td>
</tr>
<tr>
<td>Q-H$_R$</td>
<td>.085 ± .010</td>
<td>.06–.10</td>
<td>100</td>
</tr>
<tr>
<td>Q-H$_L$</td>
<td>.116 ± .013</td>
<td>.08–.14</td>
<td>99</td>
</tr>
<tr>
<td>Q-CU</td>
<td>.134 ± .014</td>
<td>.11–.16</td>
<td>100</td>
</tr>
<tr>
<td>Q-I$_L$</td>
<td>.142 ± .013</td>
<td>.12–.17</td>
<td>100</td>
</tr>
<tr>
<td>Q-I$_R$</td>
<td>.142 ± .015</td>
<td>.11–.17</td>
<td>36</td>
</tr>
<tr>
<td>Q-J$_R$</td>
<td>.177 ± .019</td>
<td>.14–.22</td>
<td>95</td>
</tr>
<tr>
<td>Q-J$_L$</td>
<td>.217 ± .019</td>
<td>.18–.26</td>
<td>100</td>
</tr>
<tr>
<td>Q-CS</td>
<td>.221 ± .022</td>
<td>.18–.28</td>
<td>95</td>
</tr>
<tr>
<td>Q-J$_D$</td>
<td>.250 ± .027</td>
<td>.21–.32</td>
<td>58</td>
</tr>
<tr>
<td>Q-K</td>
<td>.305 ± .024</td>
<td>.26–.37</td>
<td>86</td>
</tr>
<tr>
<td>Q-L$_L$</td>
<td>.39 ± .03</td>
<td>.33–.46</td>
<td>100</td>
</tr>
<tr>
<td>Q-L$_R$</td>
<td>.41 ± .03</td>
<td>.35–.48</td>
<td>76</td>
</tr>
<tr>
<td>Q-Cin</td>
<td>.41 ± .03</td>
<td>.34–.48</td>
<td>99</td>
</tr>
<tr>
<td>Q-S$_{2a}$</td>
<td>.37 ± .03</td>
<td>.31–.44</td>
<td>100</td>
</tr>
<tr>
<td>Q-S$_{2b}$</td>
<td>.40 ± .03</td>
<td>.35–.46</td>
<td>74</td>
</tr>
<tr>
<td>H$_L$-CU</td>
<td>.017 ± .007</td>
<td>.10–.04</td>
<td>99</td>
</tr>
<tr>
<td>J$_L$-CS</td>
<td>.003 ± .009</td>
<td>-.03+.03</td>
<td>95</td>
</tr>
</tbody>
</table>

Data were obtained from measurements of 100 complexes from 20 subjects. The mean, standard deviation, and range for each series of measurements are given. The right hand column gives the number of the individual waves present in the total 100 complexes. The points measured are from the onset of the QRS complex (Q) to the first major deflection of the first heart sound (S$_1$); to the initial upstroke of the carotid pulse wave (CU); to the shoulder of the carotid pulse wave (CS); to the carotid incisura (Cin), and the first and second components of the second heart sound (S$_{2a}$ and S$_{2b}$). Also, measurements are made from Q to the ballistocardiographic points, as indicated.
ically determined second derivative (or acceleration tracing) of the carotid pulse. The variable \( I_s \) point, when present, occurs at a time closely related to the onset of the carotid upstroke. The \( J_R \) peak was found to vary with respiration in magnitude and, to some extent, in time. It does not correlate with events in the carotid pulse. The \( J_L \) peak, on the other hand, was found to be almost synchronous with the anacrotic shoulder of the carotid pulse (carotid shoulder). The carotid shoulder was taken as the point of maximal deceleration of the carotid pulse pressure rise, and could be selected with fair accuracy from simple inspection of the carotid pulse curve. Figure 3 clarifies the relationship of the carotid shoulder and the point of maximal deceleration of the carotid pulse. A temporal relationship between the shoulder of the carotid pulse and one of the high frequency J peaks has been noted by Rappaport.9 Similarly, the \( J_D \) point may be related to slope changes of the carotid pulse in late systole. This slope change is visible in figure 8. The subscript \( D \) indicates the delayed J wave. The K point has not been shown to be related to the physiologic events as measured. The 2 L peaks, \( L_L \) and \( L_R \), have a close relationship to the first and second components of the second heart sound. \( L_L \) occurs during the rapid downstroke of the carotid incisura, at the time of the first component, and \( L_R \) occurs slightly later with the second component of the second sound. A striking correlation was found between the times of \( H_R \) and \( H_L \) and the reported times of right and left ventricular ejection, respectively. In 10 subjects studied by cardiac catheterization, Braunwald, Fishman, and Cournand10 found the interval between the onset of the Q wave of the electrocardiogram and right ventricular ejection to be \( 0.080 \pm 0.0079 \) second. This is to be compared to a Q to \( H_R \) time of \( 0.085 \pm 0.010 \) second in the present study. Braunwald’s value for Q to left ventricular ejection time is \( 0.115 \pm 0.0117 \) second in 12 subjects, as compared to a Q to \( H_L \) time of \( 0.116 \pm 0.013 \) second found in the present study.

**Physical Considerations**

Acceleration tracings from an ultra low frequency ballistocardiographic system are representations of cardiovascular force, unless there is distortion imposed by the internal body network as mentioned by von Wittern.1 Such distortion does not appear to alter the time relationship of the transmission of force, since the ballistocardiographic waves correlate in time with physiologic events in an entirely reasonable manner, as the present study demon-

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**Figure 3** A diagram of a carotid pulse compared with the graphically determined second derivative of the carotid pulse and the ballistocardiogram. Notice that the point of maximal acceleration of the carotid pulse (as determined by its second derivative or acceleration curve) corresponds in time to the \( I_s \) point of the ballistocardiogram. Also, the point of maximal deceleration of the carotid pulse corresponds to the \( J_L \) point of the ballistocardiogram. Connecting lines (labeled 1 and 2) on the above graph illustrate this relationship. Notice that the segment of most rapid slope change on the carotid pulse curve corresponds to the point of maximal deceleration on the second derivative curve.
strates. Since the acceleration ballistocardiograph from an ultra low frequency system represents cardiovascular force, one must interpret such tracings according to the physical laws concerning force. Newton's second law of motion relates force, mass, and acceleration according to the equation: force equals the product of mass times acceleration. Newton's third law of motion states that for every force there is a simultaneous equal and opposite force. In the case of a low frequency ballistocardiograph in the head-foot axis, the subject is essentially isolated from the environment so far as head-foot forces are concerned. It is, therefore, necessary that for every headward force registered in the ballistocardiograph, a net footward force of equal magnitude exists within the body, and conversely, every footward force in the ballistocardiograph represents a net headward force within the body. Briefly then, one must search for the opposite forces within the cardiovascular system to elucidate each ballistocardiographic deflection. In considering the genesis of a headward wave, then, the only reasonable thesis must predicate a net footward force (or mass acceleration) in the cardiovascular system. This implies nothing about the actual direction of motion of the cardiovascular mass, since the deceleration of a headward motion represents the same kind of force as the acceleration of a footward motion.

**APPLICATION OF PHYSIOLOGIC AND PHYSICAL CONSIDERATIONS**

**Pre-ejection Systole (Onset of QRS to Left Ventricular Ejection).** In normal subjects there is sometimes a small footward force, having its onset prior to the first heart sound. In normal subjects this wave (the F-G wave) is not often visible, and was, therefore, not measured. This wave, however, has been observed to be exaggerated in certain abnormal states, notably those with enlargement of the right ventricle, such as mitral stenosis and cor pulmonale (fig. 4). A similar early presystolic force has been described by others, using high frequency technics. It has been observed to be present with atrial fibrillation, showing its independence from atrial systole.

Following the F-G wave, there is a headward deflection or force having its onset at approximately .05 second after Q. The time of onset correlates with the first major vibration of the first heart sound, and this upstroke (the G-H wave) has been observed to be exaggerated in patients with mitral stenosis, and other conditions causing pulmonary hypertension (fig. 4).

It is postulated that the initial event at the onset of right ventricular contraction is an acceleration of an impulse or "bolus" of blood toward the base of the heart.* This headward acceleration of blood would result in a footward force on the body (the F-G downstroke). The subsequent deceleration (footward acceleration) resulting from the impact of this "bolus" against the closed A-V and semilunar valves would result in a headward acceleration of the body. When the impact force on the pulmonary valve has become sufficiently great to open the valve, there is a sharp headward acceleration of the now unimpeded pulse wave as it flows into the pulmonary artery. This is reflected by a footward acceleration of the body in reaction, causing the downstroke following the H_R point. Meanwhile, an analogous headward force has begun on the left side of the heart because of impact on the aortic valve, and terminates when the aortic valve opens, normally somewhat later than the opening of the pulmonary valve. This impact produces a visible headward deflection in the force ballistocardiogram terminating at the H_L point. The pulse wave then is accelerated headward in the aorta, producing a footward reaction on the body, the H_L-I_L downstroke.

**Ejection Systole.** The H_L point occurs at the time of left ventricular ejection, as measured by Braunwald and as may be predicted from the time of the upstroke in the carotid pulse. The subsequent footward force on the body would be the anticipated reaction to the headward acceleration of blood in the aorta. The ejection from both ventricles contributes to this force, since the ejection from the right ventricle is still present. The correlation of the

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* This peristaltic-like activity of the myocardium was first described by Harvey, and is visible in high speed cinemographs of the heart, though this kind of heart motion in the intact dog has been challenged by Anzola.
IL point in time with the point of maximal initial acceleration of the carotid pulse has been of great interest. The similarity of the force ballistocardiogram and the inverted second derivative of the carotid pressure pulse, as regards both relative amplitude and time, is strikingly demonstrated by figure 3.

Directly recorded second derivatives of the carotid pulse have been obtained from this laboratory, and demonstrate the similarity. The implications of this similarity are discussed below. The fact that the IL point of the ballistocardiogram coincides so closely with the point of maximal initial acceleration of the carotid pressure pulse constitutes evidence that the IL point is related to the point of maximal acceleration of left ventricular ejection. The H2-IL downstroke, then, may prove to be a most important representation of the acceleratory function of the left ventricle, since, as is shown below, the later waves may be more influenced by extracardiac factors.

The IL wave, although measurable in only one third of the normal complexes, has repeatedly been found to be exaggerated in various abnormal states (fig. 5). Its relationship to the carotid upstroke has suggested the possibility that it is related to the initial propagation of the pulse wave into the arch of the aorta.

To facilitate description, the JL point is discussed before JR. The JL point is clearly related to the shoulder of the carotid pulse. Not only is the temporal correlation excellent in our normal subjects, but also in all of the abnormalities thus far studied. The carotid shoulder, as is shown in figure 3, is the point of maximal negative acceleration or deceleration of the carotid pulse. Its magnitude and duration are affected by alterations in aortic elasticity as well as by the acceleration of ejection. The force of deceleration is increased, and comes earlier in instances of increased aortic rigidity. This is reflected by a steep slope of the carotid pulse with the carotid shoulder occurring earlier in the cardiac cycle than is normally the case.

Figure 6 illustrates the observed type of ballistocardiographic alteration in such an instance. The carotid upstroke is steep, with an early shoulder correlating precisely with an early J peak in the ballistocardiogram. It should be noted that JL, the peak corresponding to the carotid shoulder, is now the earliest of the prominent J points. The desirability of physio-

![Fig. 4. The top tracing was taken on a patient with mitral stenosis. The bottom one was taken on a patient with pulmonary hypertension. Both patients were thought to have right ventricular hypertrophy, clinically. The times of the Q onset, the carotid upstroke, and the carotid incisura are indicated. Note the prominence of the F-G and G-H segments. The first major negative deflection after Q is the F-G segment. Paper speed is 50 mm. per second.](image)

![Fig. 5. These 2 ballistocardiographic tracings illustrate the prominence of the IL point seen occasionally in records from patients with heart disease. The first tracing is from a patient with an old myocardial infarction; the second is from a patient with myocarditis, cause unknown. Notice in the second record that the Q to H2, Q to IL, and Q to carotid upstroke times are all delayed in comparison to normal subjects. Paper speed is 50 mm. per second.](image)
logic, rather than sequential identification of the various deflections is strikingly apparent in such records.

The relationship of the carotid pulse as an index of mass acceleration in the central arterial system and the systolic waves of the force ballistocardiogram demonstrates the importance of the arterial pressure pulse as a factor in the genesis of the ballistocardiogram. This relationship is as anticipated. The carotid pulse may be regarded as reflecting changes in mass displacement due to the surge of blood in the arterial system, so that the headward surge of ejection produces an upstroke, and the predominantly footward surge of run-off produces a downstroke. The second derivative of the carotid pulse, then, represents the accelerations of this pulse wave motion. The reaction on the body, according to Newton’s third law of motion, requires that the curve be inverted to correspond to the ballistocardiographic deflections. One would expect the second derivative of the carotid pulse to correlate with the ballistocardiogram only to the extent that the carotid pulse represents a summation of the movements of blood in the arterial system. Local turbulence in the carotid artery would obscure the close relationship between the in-

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**Fig. 6.** The left figure is from a normal subject with a slow heart rate and a Q to carotid upstroke time of .16 second. Notice that the JL point and the carotid shoulder coincide in time. The second tracing was taken from a patient with hypertension. Here, the Q to carotid upstroke time is unusually short (.10 second). However, the JL point still coincides with the carotid shoulder as it did in the previous instance. Paper speed is 50 mm. per second.

**Fig. 7.** This tracing was taken on a normal subject during held inspiration. Recording was begun as soon as the patient had inspired. Notice that the JR point (arrow) is initially the major J point, and that it gets progressively smaller as the breath is held. It is also of interest that the footward segment following HR varies in the same manner. Paper speed is 50 mm. per second.
verted second derivative of the carotid pressure pulse and the left ventricular waves of the ballistocardiogram.*

The above considerations make it apparent that the amplitude and contour of the J wave may be changed by noncardiac factors, since the contour of the arterial pressure pulse is profoundly affected by such factors as elasticity of the vessel walls and viscosity of blood.

The JL point, which normally precedes the JL point described above, has no apparent relationship to the carotid pulse. It has been observed to vary markedly with respiration, increasing in amplitude with inspiration and decreasing with expiration. Also, on held deep inspiration this point may be initially the major J wave, becoming progressively smaller as inspiration is held. This corresponds to the increase in right ventricular output which is presumed to be present initially during inspiration (fig. 7). Because of these findings, the Jr point is thought to be due to the maximal impact of blood from the right ventricle on the pulmonary artery bifurcation. This is analogous to the JL point, thought to represent the maximal impact of ejection from the left ventricle on the arch of the aorta. (The term "impact" as used in the above statements, is synonymous with a rapid deceleration of blood, the term used in the discussion of the JL wave.)

The Jd point is an inconstant peak seen in about half of the young normal subjects (58 out of 100 complexes). It has been observed to be much more prominent in older normal subjects, and particularly in subjects with arteriosclerotic heart disease (fig. 8). The mechanism of this deflection is not well understood, but because of its prominence in older subjects,
and its correlation with slope changes in the carotid pressure pulse, it is thought to be related to physical factors of the arterial system, and to the relative magnitude of late systolic ejection.

The J-K downstroke is probably related to at least 2 factors: to the termination of headward force after Jp, and to the impact of the pulse wave on the peripheral arteries.

Diastolic Period (Beginning with the Onset of the Second Heart Sound). The L peaks are closely related to the second heart sounds. The headward forces, reaching their maximum at the L peaks, are postulated to be due to the brief reversal of flow above the semilunar valves on reversal of the pressure gradient at the end of systole. The L-M downstrokes are thought to be due to the impact of blood on the semilunar valves as they close. Figure 9 shows a tracing taken from a patient with an interatrial septal defect. Notice that the second sound is widely split. Synchronous with the second component of the second heart sound, and following the carotid incisura, there is a large footward deflection. Because of this time relationship and the exaggeration of the second component, the first segment is thought to be due to aortic valve closure, and the second to pulmonic valve closure. This wide splitting of the L points has been observed in subjects with widely split second sounds. In subjects without splitting of the second sound, on the other hand, the L-M waves are very close together, or even superimposed.

Discussion

The points on the force ballistocardiogram described above are those that are consistently seen in records taken on young normal subjects. However, in individual complexes, there are at times minor deflections that are without obvious correlation with the events of the cardiac cycle. Also, in tracings from patients with altered cardiovascular dynamics, the normally present deflections may be diminished, increased, or shifted in time. In such cases, it is necessary to relate the deflections of the ballistocardiogram with other events, such as the heart sounds, the electrocardiogram, and the carotid pulse, in order to identify the deflection.

In some patients with heart disease, there are ballistocardiographic forces that are not readily identified by the above technics. These may be due to abnormal movements of the heart itself, or to a grossly distorted sequence of contraction.

The mechanisms postulated for the genesis of the ballistocardiographic waves are undoubtedly an oversimplification. The primary mechanisms considered are those due to forces caused by acceleration and deceleration of blood. Since any mass acceleration within the body contributes to the total registered force, acceleration of the myocardium itself must make some contribution to the genesis of the force ballistocardiogram. The fact that a force ballistocardiographic complex is obtained with the blood inflow clamped off has been previously reported from this laboratory, and demonstrates that under some circumstances acceleration of the myocardium may play an appreciable role in the ballistocardiographic genesis. In addition, since the head-foot ballistocardiogram records only the head-foot force vector and may be a measurement of the summation of multiple simultaneous forces, some cancellation of forces and some summation of forces may be expected. These factors would, of course, make it difficult to attribute a particular wave as being entirely due to a specific force. However, because of the consistency of the normal ballistocardiographic pattern and the easily recognized, even anticipated changes seen in the records obtained from many abnormal subjects, it is considered that an analysis of the wide frequency range force ballistocardiogram will give considerable information about the timing and relative magnitude of cardiovascular events.

Summary

The force ballistocardiograms of 20 normal subjects, as recorded from an ultra low frequency system having an extended frequency range, are described. These tracings were found to have high frequency components not previously described. The ballistocardiographic forces are considered in terms of other physiologic events. Distinct footward forces are found at the time of right and left ventricular ejec-

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tions. The IJK segment can consistently be separated into 3 and sometimes 4 components. One of these is related to the anacrotic shoulder (the acceleration transient) of the carotid pulse and is considered to be due to deceleration of the initial ejection in the aortic system. Another J component can be related similarly to the pulmonary circulation. Major footward forces correlate in time to the asynchronous closure of the aortic and pulmonic valves.

The mechanism of the production of such forces is considered in terms of physical laws of force as related to accelerations and decelerations of the cardiovascular system.

Ballistocardiograms from patients with various cardiovascular phenomena are presented in terms of the known alterations of cardiovascular physiology. It is found that the ballistocardiographic changes present in these instances are consistent with the hypothesis offered of the genesis of the ballistocardiographic deflections.

**SUMMARIO IN INTERLINGUA**

Es describite le ballistocardiogramma de fortia registrate ab 20 subjectos normal per medio de un systema a frequentia ultrabasse con extension del gamma de frequentias. In iste registrationes, componentes de alte frequentia esseva trovate que non es describite in ulle previe publication. Le fortias ballistocardiographic es considerate in relation a altere evenimentos physiologic. Distincte fortias pedorse es trovate al tempore del ejectiones dextero- e sinistro-ventricular. Le segmento IJK pote regularmente esser separate in 3 e a vices in 4 componentes. Un de iste componentes es relationate al nivello anacrotic (le transiente de acceleration) del pulso carotic e pote esser interpretate como efecto del deceleration del ejection initial a in le systema aortic. Un altere componente J pote esser relationate similemente al circulation pulmonic. Major fortias de direction pedorse es temporalmente correlata- tionate al clauditura asynchron de del valvulas aortic e pulmonic.

Le mecanismo del production de tal fortias es considerate super le base del leges physic de fortia, applicate al accelerationes e decelerationes del systema cardiovascular.

Es presentate ballistocardiogrammas ab patientes con varie conditiones cardiovascular, con attention prestate al alterationes cognoscite del physiologia cardiovascular. Es constatate que le alterationes ballistocardiographic que occurre in iste casos es de accordo con le hypothesis hic formulate in re le genese del deflexiones ballistocardiographic.

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Pheochromocytoma is a relatively uncommon but surgically curable condition. It is a chromaffin type of tumor that masks itself under various forms, notably hypertensive vascular disease. Several clinical features can suggest its presence including fever, tachycardia, respiratory disturbances, glycosuria, and hypermetabolism. The practical value of various pharmacologic tests in regard to their diagnostic usefulness have been analyzed in connection with this particular entity. This report is based not so much on the medical literature as on our own experience. The author especially recommends the use of provocative tests employing histamine, tetraethylammonium or methacholine for patients who exhibit paroxysmal hypertension and adrenergic blocking agents such as phentolamine or piperoxan for the routine screening of the patients who have a persistent elevation of the blood pressure. The factors responsible for false positive or false negative tests that have been encountered in examining such cases, have been discussed. There is presented a description of the technic of the tests, of the selection of patients, and of the criteria for a positive test. Certain conclusions seem to be justified: (1) no single pharmacologic test is considered confirmatory in the diagnosis of the condition, (2) the tests are to be performed only after intelligent selection of patients is made, (3) the nature of the results, whether positive or negative, indicates the necessity for additional tests by additional drugs to confirm or exclude the diagnosis, (4) all patients below the age of 60, in whom there is continuous elevation of the blood pressure, deserve the routine use of such screening tests to exclude pheochromocytoma.

Wendkos
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