Analysis of H Wave of Ballistocardiogram

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The observations on which this analysis is based include simultaneous ballistocardiographic and electrocardiographic tracings on subjects with complete heart block, and auricular fibrillation. Deflections of the ballistocardiogram similar to the H wave were seen to occur at a constant time interval after P in the absence of a ventricular systole. However, in fibrillation H waves were also found occasionally. A G wave of the ballistocardiogram is described and the authors discuss the part played by auricular systole, the apex thrust, and venous pressure pulses in producing the G and H waves.

There have been a great number of articles on the origin of the ballistocardiogram since Starr's first report in 1939. It is not the purpose here to review the literature in general but only to discuss a small part of the ballistocardiogram, the H wave. This is the first deflection seen in normal records whether one uses a horizontal or vertical table or a table with a high or low frequency response.

The H wave is an upward deflection whose amplitude varies not only from subject to subject (figs. 1A, 1B), but also in the same subject (fig. 1B). It varies with respiration, usually increasing with expiration (fig. 1A), but it may remain constant or decrease with expiration. The P-H interval, measured from the peak of P (electrocardiogram) to that of H (ballistocardiogram) ranges from 0.2 to 0.3 second in various subjects. It is constant in an individual and averages 0.24 second (fig. 1). The H wave is nearly synchronous with the first heart sound and the apex thrust (figs. 2A and 2B). It begins approximately 0.15 second after mechanical auricular systole (fig. 2C).

Two basic theories have been advanced as to the cause of the H wave. The first was proposed by Hamilton, who related the apex thrust to the H wave, since they occurred at the same time. Accordingly, the vertical component of the apex thrust (isometric contraction of the ventricles) causes a headward movement of the body. Since the heart is fixed at the base, a lateral movement of the ventricles must have a headward component. This theory fits the observed respiratory variation of the H wave very nicely, since the heart is more lateral in expiration, and it would be expected that the vertical component of the apex thrust be more pronounced in expiration. However, the total diastolic filling of the heart is increased in inspiration and decreased in expiration. This would diminish the force of the apex thrust in expiration, thus tending to counteract the increase in the vertical vector. This opposition of forces with respiration helps to explain the number of cases where the H wave showed no respiratory variation, or actually decreased in expiration.

The second theory was offered by Nickerson in 1949. He observed that in complete heart block where P waves occurred separately from the QRS complexes, there were separate ballisitic patterns of small amplitude which were definitely related to the auricular electrical systole (fig. 3A). He postulated that when auricular contraction is in normal temporal relation to ventricular contraction, the H wave is produced by the upward ballistic deflection that results from auricular systole. We have obtained traces of complete heart block in our laboratory which confirm this (fig. 3B). It can be seen that the time relation of electrocardiographic auricular systole (peak of P) to the upward deflection of ballistocardiographic auricular systole (peak of H) in heart block (fig. 3) is within the normal P-H range, since it is
0.25–0.29 second. Therefore, auricular systole probably plays a part in the production of the H wave.

Nickerson felt that the first auricular ballistocardiographic deflection was upward. However, examination of his traces reveals that the first auricular ballistocardiogram deflection is in a downward direction, beginning about 0.15 sec-

graphic auricular complex appear in fig. 3A. The first trace in 3A shows his original labelling of the auricular complexes. Upon examination of other auricular complexes in his records it appears that the true H peak is 0.25 to 0.29 second after the electrocardiographic P wave peak as shown by subsequent labels which are ours; this corresponds to what Nickerson has

Fig. 1. Three traces taken on two normals; records B and C are on the same subject, but at different paper speeds. Note the increased H wave with expiration (compare second and third arrows), in A. Tracings B and C demonstrate G valleys just prior to the H peak. Note the P-G and P-H intervals and compare with figure 3. The larger time lines are 0.2 sec. apart in these figures.

ond after the P wave (figs. 3A and 3B). This could be labelled G. This initial downward deflection is followed by an upward swing of equal or greater amplitude (H), whose peak occurs about 0.27 second after P (fig. 3). The following waves are probably simple oscillations. In a normal with a very slow rate we have observed a G valley (fig. 1C), which could well represent this initial downward auricular deflection. Nickerson's original traces of the ballistocardiograms are greater than 0.2 second.
The time lag of about 0.15 second between the electrocardiographic P wave and the onset of the true time lag is approximately 0.05 second, since it is the mechanical systole rather than the electrical systole with which we are concerned.

Fig. 2. Three records showing the relations of the H peak to the apex thrust (A), heart sounds (B) and (C), and mechanical auricular systole (C). Note that the H wave begins during the first heart sound and just after the apex thrust. The interval from the peak of P to the peak of the first auricular pressure wave is approximately 0.1 second. The interval from the peak of the first auricular pressure wave to the onset of H is approximately 0.15 seconds.

(Record C was obtained from the laboratory of Doctor R. Bruce, Department of Medicine, University of Rochester, New York. Auricular pressure was obtained through a cardiac catheter with a Hathaway transducer.)

of the ballistocardiographic auricular G wave can be explained in this manner: mechanical auricular systole follows electrical auricular systole by about 0.1 second (fig. 2C). Therefore,
probably the deceleration of blood and impulse wave by the ventricles from the auricles that accounts for the initial downward deflection of the auricular ballistocardiographic pattern. The time it takes for the pulse to travel from auricle to ventricle would account for the observed lag.

The origin of the subsequent upward deflection is more obscure; a possible explanation lies in the concept that retrograde flow and impulse of this should occur at approximately the same time as the footward thrust produced by the ventricles. These forces would tend to counteract each other, with the ventricular footward force predominating. However, the innominate

![Image](http://circ.ahajournals.org/)

**Fig. 3.** Records of patients with complete heart block and auricular fibrillation. The tracings in A (sub-labelled 1, 2 and 3) are from Nickerson. The lettering in the first trace in A is Nickerson's. All subsequent lettering is ours. Note that H as labelled by Nickerson does not repeat and is probably an artefact. Nickerson's I valley is the same as our G valley (same interval after P wave), and Nickerson's J peak is the same as our H peak (same interval after P). The first BCG deflection after P is downward (G valleys) followed by H peaks. Note the P-G and P-H intervals, and compare with figure 1.

Tracing C is of a man with auricular fibrillation. Note the H peaks. Since the auricle is fibrillating, these peaks cannot be due to auricular systole. They probably represent apex thrust forces.

travel occur in the great caval veins following auricular contraction. We have seen such retrograde pulsations of dye and blood in the superior vena cava in fluoroscopic movies made during angiocardiography. It is reasonable that this should occur since there are no valves in the caval veins to prevent it. There is little in the inferior vena cava to cause a marked deceleration of this pulse. However, there are several arches in the superior caval system as well as the skull itself which might cause sufficient headward deceleration to account for the headward movement of the ballistocardiograph. The azygos arch occurs 3 to 4 cm. from the right auricle, and a headward thrust produced here would occur at approximately the same time as the footward thrust produced by the ventricles. These forces would tend to counteract each other, with the ventricular footward force predominating. However, the innominate

and subclavian arches are more than 8 to 10 cm. from the right auricle. A headward thrust produced here or in the head would occur after the footward thrust from the ventricles and would account for the lag between auricular mechanical systole and the onset of the auricular H wave of approximately 0.15 second. Therefore, it is possible that the auricular H wave is produced by deceleration of a headward traveling impulse wave in the superior caval venous system or in the head, originating from...
right auricular contraction. It is fair to ignore the pulmonary veins since they approach the left auricle laterally and have tributaries which radiate in all directions. Thus any forces which might occur in this system would tend to balance out as far as the ballistocardiograph is concerned.

**Summary**

1. A theoretic analysis of the H wave of the ballistocardiogram is presented.

2. Since H waves may be seen without sustained auricular contraction, it seems probable that the H wave represents a force produced by the apex thrust as well as auricular contraction. The P-H interval (peak to peak) in heart block averages 0.27 second, which is in the same range as the normal P-H interval. It is postulated that the upward auricular ballistocardiographic stroke may be due to deceleration of an auricular impulse wave by venous arches in the neck, or by the skull, or both.

3. The G valley, a footward deflection occasionally seen in normals with bradycardia, and in auricular ballistocardiographic patterns in patients with heart block, may be due to the deceleration in the ventricles of a footward-traveling auricular impulse. The P-G interval (peak to peak) averages 0.17 second.

**REFERENCES**


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