The First Heart Sound in Complete Heart Block

Phono-Echocardiographic Correlations

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SUMMARY

Eight subjects with complete atrioventricular heart block were studied with simultaneous echocardiography and phonocardiography to investigate the role of the mitral and tricuspid valves in the genesis of the first heart sound (S1). Mitral valve motion was studied in all cases and tricuspid patterns in three. At P-R intervals less than 0.20 sec, mitral leaflet closure was completed by ventricular systole. Below P-R values of 0.20 sec there was an inverse relationship between P-R and S1 amplitude. With P-R between 0.20 and 0.50 sec atrial systole closed the mitral valve without recordable sound. Beyond P-R intervals of 0.50 sec there was reopening of the mitral valve and secondary closure initiated by ventricular systole which was coincident with S1. The extent of mitral leaflet separation at the onset of ventricular systole correlated with S1 amplitude. Pre-ejection period was not related to S1 intensity. Over a wide range of P-R intervals in two subjects with a split S1, tricuspid valve closure was simultaneous with the second S1 component. It was concluded that atrioventricular valve closure is a necessary event in the production of S1 and that at least in some individuals tricuspid valve closure is related to its second high frequency component.

Additional Indexing Words:
Mitral valve
Echocardiography
Tricuspid valve
Phonocardiography
Pre-ejection period

Variation in first heart sound amplitude in patients with complete atrioventricular block was first reported in 1912 by Griffith.¹ Using simultaneous auscultation and polygraphic techniques he observed that the first heart sound was loudest when the atria were in systole at the time left ventricular contraction began. Subsequent investigators described a soft first heart sound with P-R intervals in the range of approximately 0.2 to 0.5 sec and noted a secondary accentuation of amplitude at longer P-R intervals.² Thirdly found that an increase in first heart sound intensity with long P-R intervals was an unusual finding in elderly subjects.³ Although these earlier workers speculated that the changes in first heart sound amplitude with P-R interval were related to variable mitral valve leaflet position, they were unable to provide definite evidence for this explanation.

Echocardiography provides a precise technique by which auscultatory phenomena may be correlated with valve motion. Using this method to study eight subjects with complete heart block, Shah and coworkers concluded that once closed in diastole by atrial systole the mitral valve did not reopen.ª Their results were in conflict with an earlier report in which two distinct patterns of mitral valve motion in six subjects with congenital complete heart block were recognized.ª

The purpose of this study was to investigate the relation between components of the first heart sound and atrioventricular valve motion using echocardiography in patients with complete heart block in an attempt to gain further understanding of the genesis of the first heart sound.

Material and Methods

Eight subjects with complete heart block were studied, three of them having permanent pacemakers. Several additional elderly subjects were screened but rejected for study because adequate echocardiograms could not be obtained. Five of the group had congenital complete heart block with no other known associated anomalies. The other three, all with fixed rate pacemakers, were older subjects probably with degenerative conducting system disease. The ages, heart rates, electrocardiographic and clinical diagnoses for the group are summarized in table 1.

Simultaneous phonocardiographic, echocardiographic, and electrocardiographic information was obtained on a strip chart at a speed of 100 mm/sec using a Cambridge multichannel photographic recorder. Leatham suction
microphones were placed at the mitral area and in most cases also at the aortic or pulmonic areas. All phonocardiograms were recorded using a medium frequency band setting. Echograms of the mitral and tricuspid valves were obtained using a Smith-Kline ultrasonoscope (Ekoline-20) interfaced with the Cambridge recorder. A half-inch diameter ultrasonic transducer focused at 7.5 cm with a frequency of 2.25 megaHertz and repetition rate of 1000 impulses/sec was applied to the chest using a water soluble coupling jelly. In two cases an external carotid pulse was recorded with the other parameters using a Hellige pulse transducer (Fritz Hellige and Co., Freiburg, West Germany). Lead II of the electrocardiogram was monitored in all subjects.

The initial set of high frequency vibrations of the first heart sound was labelled M1 and if a second component was present it was designated as T1. An approximation of the loudness of these components was obtained by measuring the maximum excursion of their vibrations. In all subjects both anterior and posterior mitral valve leaflets were recorded with particular emphasis being placed on defining closure. The tricuspid anterior leaflet was successfully visualized in three subjects. Closure of the mitral valve was defined as the point on the echogram at which both leaflets became approximated. Tricuspid closure was defined as the maximum posterior excursion of the anterior leaflet. Systolic time intervals were measured in the following manner: Q-A2 was the time from onset of the QRS of the electrocardiogram to the initial high frequency components of the second sound. Left ventricular ejection time was the time from onset of the carotid upstroke to the incisura and pre-ejection period was the difference between Q-A2 and the ejection time expressed in milliseconds. All parameters were measured over at least 30 consecutive cardiac cycles for each subject.

Results

M1, Variations and P-R Interval

In all cases variation of first heart sound intensity, including total absence of an audible component, was appreciated on auscultation. In figure 1 the relationship between P-R interval and the intensity of M1 is graphically illustrated for two subjects who showed patterns representative of the entire group. First heart sound amplitude is expressed in arbitrary units which were normalized to give a maximum value of 40 for each subject. Mitral valve closure was not associated with a recordable high frequency sound when the P-R interval was in the range of 0.20 to 0.50 sec. As the P-R interval progressively decreased from 0.20 sec M1 continued to increase in amplitude. There are no points with a P-R of less than 0.04 sec. At values of P-R greater than 0.50 sec (0.65 sec for subject WM) M1 is again recorded and after an initial rise in amplitude most subjects did not vary substantially with longer intervals. Subject DM was exceptional in that with P-R values ranging from 0.20 to 0.40 sec M1 varied from normal intensity to absent.

Mitral Valve Patterns and M1

M1 consistently occurred precisely at the time of closure of the mitral valve by ventricular systole. There were three distinct patterns of mitral valve motion which are illustrated in figure 2. With P-R less than 0.20 sec the mitral valve was closed by ventricular systole shortly after it had been opened more widely by atrial systole (see cycle 1). This pattern was

Table 1

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yr)</th>
<th>Diagnosis</th>
<th>QRS with duration (sec)</th>
<th>Heart rate (per min)</th>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
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<td>73</td>
<td>ICSD</td>
<td>RV pacing (.13)</td>
<td>65</td>
</tr>
<tr>
<td>NH</td>
<td>55</td>
<td>ICSD</td>
<td>RV pacing (.14)</td>
<td>88</td>
</tr>
<tr>
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<td>71</td>
<td>ICSD</td>
<td>RV pacing (.12)</td>
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<td>SM</td>
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<td>CCHB</td>
<td>normal (.07)</td>
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<td>CCHB</td>
<td>Idioventricular rhythm (.10)</td>
<td>79</td>
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<tr>
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<td>CCHB</td>
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<td>8</td>
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<td>DW</td>
<td>3</td>
<td>CCHB</td>
<td>normal (.08)</td>
<td>75</td>
</tr>
</tbody>
</table>

Abbreviations: CCHB = congenital complete heart block; ICSD = idiopathic conducting system disease; RV = right ventricle.

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Mitral valve patterns at different P-R intervals. Subject SM. Cycle 1 — short P-R with loud M₁. Cycle 2 — intermediate P-R with absent first heart sound. Cycle 3 — long P-R with reopening and secondary closure of mitral valve coincident with a softer M₁. MA = mitral area, MVE = mitral valve echogram, PCG = phonocardiogram, S₂ = second heart sound, M₁ as in figure 1. Tall vertical arrows indicate time of mitral valve closure. P and QRS of electrocardiogram (ECG) indicated by short arrows.

Figure 2

The time of mitral closure related to P-R interval for two subjects. Each point on the graphs represents one cardiac cycle or the average QCM for that P-R interval. QCM = time from Q wave of electrocardiogram to closure of the mitral valve.

Figure 4

The intensity of M₁ is related to the separation of both mitral leaflets at the peak of the R wave of the electrocardiogram for two subjects. As the gap between the mitral leaflets widens at the time of onset of ventricular contraction the loudness of the associated M₁ tends to increase. All subjects showed this same trend and the linear correlation coefficients varied from .84 to .98 (P < 0.001).

In figure 4 the timing of final mitral valve closure is related to P-R interval for two subjects who were representative of the entire group. QCM represents the time in seconds from the onset of the QRS to final closure of the mitral valve as indicated by the echocardiogram. For the three patients with pacemakers Q was defined as the onset of the pacemaker spike. Negative values of QCM indicate that mitral closure occurred before onset of the QRS. QCM for the eight subjects ranged from 0.07 to 0.13 sec when the P-R interval was 0.10 sec and became negative at P-R values ranging from 0.22 to 0.30 sec. An absent M₁ was associated with a QCM of 0.06 sec or less. With increasing P-R intervals QCM became more negative until at ap-

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proximately 0.50 sec (0.65 sec for WM) it abruptly returned to the initial positive values and remained quite constant. This phase represents the pattern of mitral motion in which the valve reopens after approximation by atrial contraction and is finally closed by ventricular systole. As noted previously, subject DM showed a variable pattern of mitral valve motion in the range of P-R values usually associated with permanent atrial closure of the valve.

Tricuspid Valve Patterns and T₁

The anterior tricuspid leaflet was visualized in three subjects, in two of whom (SM and WM) a split first heart sound was recorded. In figure 2 (subject SM) the close temporal relationship between mitral valve closure and M₁ is illustrated. Figure 5, which displays the same subject’s tricuspid valve motion, demonstrates an analogous relationship between tricuspid valve closure and T₁. In the first three cycles of figure 5 tricuspid closure follows M₁ by 0.03 sec. However in the fourth cycle (similar to cycle 2 in fig. 2), with a P-R interval in the intermediate range, the tricuspid valve is closed before ventricular systole and no first heart sound is recorded. Clearly in this subject the atrioventricular valves behave in a parallel fashion over a wide range of P-R intervals but the mitral valve precedes both tricuspid closure and T₁ by 0.03 sec.

In contrast, subject WM displayed nonparallel motion of the atrioventricular valves at P-R intervals in the range of 0.40 to 0.65 sec. This subject’s mitral valve motion is illustrated in figure 6. In the first and third cycles, which have P-R intervals of 0.08 and 0.89 sec, respectively, synchronous mitral closure and M₁ are seen preceding T₁ by 0.03 sec. However in the second cycle (P-R interval of 0.65 sec) the mitral valve is closed by atrial systole and remains in that position for the remainder of this cycle. In spite of the shut mitral valve a loud single first heart sound occurs at the time of an expected T₁. Figure 7, which illustrates this subject’s tricuspid valve motion, provides further evidence that this single first sound is in fact a T₁. Cycle 3 in this figure, with P-R interval of 0.65 sec, illustrates synchrony between tricuspid closure and a single first sound. The tricuspid echogram also demonstrates absence of T₁ when the valve is closed by atrial systole (cycle 1). In the second cycle T₁ and tricuspid apposition follow M₁ by 0.03 sec. It is apparent from figures 6 and 7 that at some P-R intervals the tricuspid valve may reopen during diastole, after closure by right atrial systole, while the mitral valve remains shut. The first sound recorded simultaneously with tricuspid closure in this situation strongly suggests that these two events are related.

The third subject (DW) in whom both atrioventricular valves were recorded had a single first heart sound. The two valves closed coincidently with the onset of this sound at an interval of 0.07 sec after the Q wave of the electrocardiogram. Figures 8 and 9 represent the mitral and tricuspid echograms for subject DM.

Pre-ejection Period and P-R Interval

In two subjects (SM and WM) the carotid pulse was recorded in addition to the phonocardiogram and

Figure 5

Tricuspid valve echogram and split first sound. Subject SM. TVE = tricuspid valve echogram, T₁ = second high frequency component of first heart sound, Q-M₁ = time from Q wave of electrocardiogram to M₁, Q-T₁ = time from Q wave to T₁, other abbreviations as in figure 2.
mitral valve echogram. This made it possible to compare the pre-ejection period with $M_1$ over a wide range of first heart sound intensities. There was a wide scatter of the data with no discernible correlation between these two parameters ($r = -.08$ and $-.16$).

**Discussion**

The relationship between P-R interval and first heart sound intensity in complete heart block described by previous authors was also demonstrated in our subjects. The younger patients with congenital complete heart block showed a somewhat more variable pattern than the older individuals with permanent pacemakers. Atrial systole is followed by a transient augmentation of the gap between leaflets of the atrioventricular valves and thus it is apparent that as the P-R interval shortens the mitral and tricuspid valves will be more widely separated at the onset of ventricular contraction. We have shown that a decreasing P-R interval and a widening mitral valve gap are associated with an increasing $M_1$ intensity. For a given level of ventricular inotropic state, position of the valve leaflets at the onset of systole becomes a principal factor governing first heart sound amplitude. With the P-R interval in the range of 0.20 to 0.50 sec, and the atrioventricular valves closed by atrial systole, ventricular contraction was not followed by a first heart sound.

**Figure 6**

*Mitral valve echogram related to first heart sound components. Subject WM. Abbreviations as in figures 2 and 5.*

**Figure 7**

*Tricuspid valve echogram related to first heart sound components. Subject WM. Abbreviations as in figures 2 and 3.*
Shah and co-workers studied the relation of mitral valve motion by ultrasound and first heart sound intensity in eight patients with complete block having pacemakers. Contrary to our observations they concluded that once atrial systole closed the mitral valve in diastole it remained closed. We demonstrated re-
opening of the mitral valve during diastole with P-R intervals of approximately 0.50 sec or more and eventual closure by ventricular systole which explains the phenomenon of secondary accentuation of the first heart sound. Many of our subjects had longer P-R intervals than were observed in their study which probably accounts for the lack of reopening of the mitral valve in their records. The echocardiographic study of Zaky and associates reports a relation between mitral valve motion and P-R intervals than valve probably accounts for the lack of reopening of the mitral valve in their records. The echocardiographic study of Zaky and associates reports a relation between mitral valve motion and first sound intensity in six patients with congenital complete heart block which is very similar to our findings. However in three of their subjects atrial systole permanently closed the mitral valve over a much narrower range of P-R intervals than in our group.

A recent report by Parisi and Milton states that mitral valve closure as determined by ultrasound follows the initial high frequency components of the first heart sound by an average interval of 10 msec. As further support for their contention that mitral valve closure and the onset of the first heart sound are not simultaneous they refer to a study by Luisada and associates which reports an interval of 23 msec between crossover of left atrial and left ventricular pressures and the onset of the first sound. Faber and Burton, however, have pointed out that because of blood and valve leaflet inertia one would expect some time to elapse from the point of crossover of these pressures and closure of the mitral valve. Furthermore, studies of mitral valve motion by radiographic techniques have demonstrated coincidence of the first heart sound and closure of the mitral leaflets. In our patients closure of the mitral valve, defined as the point of apposition of both leaflets by echocardiography, repeatedly coincided with the onset of the first sound.

We were able to study the relation of tricuspid valve motion to the first heart sound in three patients with congenital complete heart block. In two subjects with a split first sound the second high frequency component was always simultaneous with tricuspid valve closure. One subject demonstrated parallel activity of both atrioventricular valves but tricuspid closure always occurred later than mitral. Nonparallel motion of the atrioventricular valves was apparent in the other subject with a split first heart sound. It was possible to establish that for some cardiac cycles the mitral valve was closed before the occurrence of a single first heart sound which was shown to be coincident with tricuspid closure. In a third subject whose first heart sound was always single the two valves moved in a parallel and coincident fashion. These observations indicate that the two high frequency components of the first heart sound occur simultaneously with closure of the mitral and tricuspid valves respectively. This is consistent with Leatham’s views on the origin of the two main components of the first heart sound. These findings are also in agreement with the report of Waider and associates in which tricuspid valve closure, determined by ultrasound techniques, was found to be coincident with the second group of high frequency vibrations of the first heart sound in a variety of clinical conditions. Further support is added by two echocardiographic studies of the tricuspid valve in Ebstein’s anomaly in which closure of the valve was coincident with the loud delayed second component of the first heart sound. Forty years ago Wofe and Margolies noted independent variation in the intensity of the two first heart sound components in four patients with heart block. They were unable to study valve motion but postulated on the basis of their individual behavior that these components originated from the right and left ventricles.

We were unable to find a correlation between first heart sound intensity and the pre-ejection period. Bashour and co-workers studied the relation between systolic time intervals and P-R interval in patients with artificial cardiac pacemakers for complete heart block. They noted that the pre-ejection period decreased with increases in P-R interval up to a mean value of 0.20 sec at which point it began to increase. This suggested to them that the P-R interval for optimal left ventricular performance was 0.20 sec. We have found an inverse relationship between first heart sound amplitude and P-R interval as it increases to 0.20 sec which suggests that the variation that does occur in first sound intensity in complete heart block is not due to changes that occur in left ventricular function. Further support for this contention is supplied in a canine study by Stept and associates. Using sequential atrioventricular pacing they did not detect any change in maximal rate of rise of left ventricular pressure over a range of P-R intervals from 0 to 0.13 sec in spite of a 119% change in first sound amplitude.

The consistent association between components of the first heart sound and apposition of the atrioventricular valves implies valve closure is a necessary condition for the production of these sounds. This is not evidence that the sound is produced solely by the valve leaflets — other cardiac structures and probably blood vibrate at the time of valve closure. MacCanon and co-workers concluded on the basis of a study in dogs that the movement made by the mitral leaflets on closure could not account for the energy required to produce the first heart sound. We have demonstrated that right sided events alone may be responsible for generation of the first heart sound at some P-R intervals. That the right ventricle can produce a component of the first heart sound has been
hotly contested by some investigators. The second high frequency component of the first heart sound has been attributed by them to the onset of left ventricular ejection and hence has been referred to as an aortic ejection sound. If this was the origin of the second high frequency component (labelled $T_1$ in our records) then it follows that it should be maximal in intensity at a P-R of approximately 0.20 sec when the pre-ejection period is at its minimum in subjects with a variable P-R interval. But not only have we failed to observe an increase in this sound at P-R values of 0.20 sec; we have in fact demonstrated its frequent disappearance between values of 0.20 and 0.50 sec. The hypothesis that both right and left ventricular vibrations, which occur at the time of closure of their respective atrioven tricular valves, are responsible for genesis of the first heart sound appears to us to be more tenable.

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