The First Heart Sound in Normal and Ectopic Ventricular Contractions

Mechanism of Closure of the A-V Valves

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Phasic right atrial and ventricular pressures and heart sounds were simultaneously recorded from dogs with normal and ectopic ventricular contractions. Heart sounds and electrocardiograms were recorded from humans. The first heart sound is significantly delayed in the absence of atrial systole. The time between onset of the ventricular pressure rise due to ventricular contraction and the beginning of the first heart sound is minimal at normal A-V intervals. These findings support the view that normally the A-V valves are closed or nearly closed before ventricular systole. In ectopic ventricular beats the A-V valves are closed from an open position by ventricular contraction.

The exact mechanism of closure of the A-V valves has not been determined. Among the factors which may be involved are (a) contraction of the valve ring, (b) eddy currents set up behind the valve leaflets, (c) sudden stoppage of ventricular inflow after atrial systole, and (d) the sudden increase in intraventricular pressure due to onset of ventricular contraction. The relative importance of these factors in valve closure is uncertain, and the validity of some has been questioned.

One important factor in the production of the first sound is the closing and sudden tensing of the A-V valves. Thus, the intensity and timing of the first heart sound has been used to indicate the position and movements of the valve cusps prior to closure. Several studies have suggested that the valve cusps approach closure at the end of diastole.

Recently, we presented an analysis of phasic pressures recorded from the right heart which suggested that the valves are closed or nearly closed before the onset of ventricular contraction by a reversal in the atroventricular pressure gradient following atrial systole. An analysis of the first heart sound recorded simultaneously with intracardiac phasic pressure changes should permit a more detailed study of the mechanisms responsible for closure of the A-V valves. The purpose of this communication is to present such an analysis.

Method

Dogs were anesthetized with sodium pentobarbital (ca 30 mg. per kilogram), and the chest was opened under mild artificial respiration. The atrium was cannulated via the external jugular vein with a large sound. The right ventricle was punctured with a blunt 15 gage hypodermic needle. Blood clotting was prevented by frequent flushing of the cannulas with saline. Right atrial and ventricular pressures were recorded with sensitive calibrated optical Gregg manometers of modified design. The heart sounds were picked up from the exposed heart by a resonator similar to that described by Wiggers and Dean. It was sutured to the A-V groove at the midline of the right ventricle. The sound vibrations reached a crystal microphone by air transmission through a 6 cm. rubber tube having a 1 cm. bore and a small side opening. The electrical impulses were led from the microphone to an amplifier and recorded by a high frequency optical galvanometer. All records were recorded simultaneously and no parallax existed between recording beams. There was no significant time delay in the sound recording. Respiration was interrupted during recording to avoid artefacts incident to lung inflation. A calibration recording of a standard pressure was made at the end of each record, and a complete calibration was made at the end of each experiment. The zero level for all manometers was the level of the animal board.

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* Hathaway Galvanometer Type OA2. 5.0 Mm/ Ma/M 650 cps.
Ventricular contractions without preceding atrial contraction were produced by stimulating both vagi with a strong faradic current. Occasionally, the ventricles were stimulated directly with induction shocks; in the remainder ventricular escape occurred.

Human heart sounds were recorded with a Cambridge phonocardiograph simultaneously with lead II of the electrocardiogram.

**RESULTS AND DISCUSSION**

Optical records from five dogs were suitable for analysis. The pressure curves were of acceptable form. The sound tracings were free of artefacts and of good quality. The onset of the first heart sound was clearly demonstrated and was not obscured by atrial sounds. Representative records of beats with and without atrial systole are presented in figure 1.

Ventricular pressure increases sharply with ventricular systole. The time from onset of this pressure deflection to the first heart sound was measured. This interval is referred to as the heart sound time. In normal beats the heart sound time may be very short, in many records the first sound occurs nearly simultaneously with the increase in ventricular pressure. However, in some beats the heart sound time is clearly measurable. Examples of such records are shown in figure 1. An investigation of this variation in heart sound time was undertaken as a part of this study.

The validity of using the heart sound time to indicate the interval from onset of ventricular contraction to the first heart sound may be questioned if the sudden upward deflection in ventricular pressure does not begin from a stabilized position. This deflection apparently occurs from an unstabilized baseline after atrial systole in some records (fig. 1A). Careful examination of these records, particularly in beats showing long intervals between atrial

![Fig. 1. Representative records of heart sounds (upper line), right ventricular pressure (middle) and right atrial pressure (lower) showing normal (A, B, C) and ectopic ventricular (D) beats. Records B and C slightly enlarged. Time 0.02 second. Pressure in mm. Hg.](attachment:image.png)
and ventricular systole ($A\_V$ intervals) (fig. 1B, C), reveal that ventricular pressure increases slightly during atrial systole and then begins to fall; this decline is abruptly halted, and the pressure stabilizes at a slightly higher level before it suddenly increases with ventricular systole. This momentary stabilization offers some justification for using the heart sound time from beats without atrial systole to indicate the interval between the onset of ventricular contraction and the beginning of the first sound. In any event the heart sound time does represent the interval from the onset of effective contraction to the beginning of the first sound.

The frequency distribution of the heart sound times from five animals measured in 90 beats with atrial systole and 110 beats without atrial systole is presented in figure 2. Some overlap is present; however, the first heart sound occurs significantly later in beats without atrial systole.

Several points are demonstrated by figures 1 and 2 that are worthy of comment. In the first record of figure 1, ventricular pressure increases 2 mm. Hg during atrial systole and reaches 6.5 mm. Hg before the onset of ventricular contraction and 0.035 second after the peak atrial systolic pressure. The mechanism of this delay in transference of pressure from atrium to ventricle remains speculative.

At the onset of the sudden increase in ventricular pressure due to ventricular contraction, marked by the first vertical line in figure 1A, atrial pressure has decreased from its systolic high and is now 5 mm. Hg. This indicates a gradient of 1.5 mm. Hg between ventricle and atrium. As we have pointed out, this small but definite gradient should cause a reversal of the normal flow into the ventricle and thereby move the A-V valves to or towards the position of closure. Subsequent ventricular contraction can then promptly complete closure and put the valve cusps under tension. This mechanism of closure is given added support by the prompt occurrence of the first heart sound in beats with atrial systole. In this case the first sound occurs 0.02 second after onset of the upward ventricular pressure deflection.

When atrial contraction is absent, the pressures in the atrium and ventricle are equal at the onset of ventricular contraction, and a reversal of the A-V gradient does not occur, thereby removing this mechanism for valve closure. As a result, the A-V valves must be closed by ventricular contraction from an open position with considerable regurgitation into the atrium. This manner of closure is illustrated in record D of figure 1. A small pressure wave is present in the atrial pressure pulse during the first 0.045 second of ventricular contraction. This wave indicates a regurgitation of blood into the atrium from the ventricle. The first heart sound begins exactly with the end of this wave, indicating closure of the A-V valves. The late occurrence of the first sound in ectopic ventricular contractions is consistent with these observations.

If A-V valve closure precedes the onset of ventricular contraction, sufficient time must follow atrial systole to permit the valve cusps to swing together. This factor was investigated by plotting the $A\_V$ intervals against the heart sound times. The heart sound times were arranged in groups, the class interval being 0.01 second. The frequency distribution of the $A\_V$ intervals in each group showed a bimodal curve with complete separation into two subgroups, the dividing line being an $A\_V$ interval of 0.160 second. The mean $A\_V$ interval for each subgroup was plotted with one standard deviation to each side (fig. 3). The upper curve shows that increasing the $A\_V$ interval beyond the normal range (0.12 to 0.20 second) causes the heart sound time to increase. The lower curve shows that decreasing the $A\_V$
FIG. 3. Average Aₐ-Vᵣ interval plotted against heart sound time. Vertical lines indicate one standard deviation to each side. Figures are number of observations for each point. See text for discussion.

FIG. 4. Human heart sound and electrocardiograph record showing a normal and a ventricular ectopic beat. Vertical lines indicate heart sound time.

interval from the normal range also increases the heart sound time. Thus, a normal Aₐ-Vᵣ interval will result in the minimal heart sound time, and as the Aₐ-Vᵣ interval deviates from the normal range, the duration of the heart sound time increases. This may explain the range of heart sound times recorded in beats with atrial systole.

These observations support the view that an Aₐ-Vᵣ interval of 0.12 to 0.20 seconds gives optimal time for the A-V valves to close. At Aₐ-Vᵣ intervals greater than this, the valves open again due to return of the normal pressure gradient and flow from the atria into the ventricle. Aₐ-Vᵣ intervals less than this provide insufficient time for the valves to close. In either case, closure must be completed by ventricular contraction.

Variations in the intensity of the first heart sound with changes in the Aₐ-Vᵣ interval have been reported. This observation was not confirmed in our animal experiments, probably because of the method utilized to record the heart sounds.

To extend our investigations to man, simultaneous recordings of the heart sounds and the electrocardiogram were made in subjects with ventricular premature beats. A typical record of a normal and of an ectopic beat is shown in figure 4. The heart sound time was taken as the interval between the beginning of the QRS complex and the onset of the first sound. This interval will be slightly longer than the animal heart sound time previously measured because of the time difference between electrical and mechanical events. The heart sound times in normal and ectopic ventricular beats from six subjects are summarized in table 1. This interval is clearly longer in the premature ventricular beats. These observations agree closely with the heart sound times in 16 subjects with premature ventricular beats reported by Cossio and co-workers.⁵

**SUMMARY AND CONCLUSIONS**

Phasic right atrial and ventricular pressures were simultaneously recorded with the heart sounds from the exposed dog heart. Both normal beats and ventricular beats without preceding atrial contractions were studied.

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**Table 1.**—Heart Sound Times of Normal and Ectopic Ventricular Beats in Humans

<table>
<thead>
<tr>
<th>Subject</th>
<th>Number of beats measured</th>
<th>Average time, seconds</th>
<th>Range, seconds</th>
<th>Number of beats measured</th>
<th>Average time, seconds</th>
<th>Range, seconds</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>.044</td>
<td>.040–.050</td>
<td>26</td>
<td>.136</td>
<td>.115–.150</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>.039</td>
<td>.035–.045</td>
<td>26</td>
<td>.128</td>
<td>.095–.145</td>
</tr>
<tr>
<td>3</td>
<td>12</td>
<td>.065</td>
<td>.055–.060</td>
<td>1</td>
<td>.095</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>23</td>
<td>.056</td>
<td>.046–.063</td>
<td>27</td>
<td>.096</td>
<td>.065–.105</td>
</tr>
<tr>
<td>5</td>
<td>25</td>
<td>.029</td>
<td>.020–.040</td>
<td>25</td>
<td>.073</td>
<td>.060–.100</td>
</tr>
<tr>
<td>6</td>
<td>16</td>
<td>.031</td>
<td>.029–.040</td>
<td>16</td>
<td>.088</td>
<td>.060–.110</td>
</tr>
</tbody>
</table>
The first heart sound occurs significantly later in the absence of atrial systole. The time between the onset of the sudden increase in ventricular pressure due to ventricular contraction and the beginning of the first heart sound reaches a minimum at A-V intervals of 0.12 to 0.20 second. It increases as the A-V interval deviates from this range. The time interval between onset of the QRS complex and onset of the first heart sound was prolonged in humans with ectopic ventricular beats.

These findings support the view that following atrial systole there is a reversal of the atrial-ventricular pressure gradient before the onset of ventricular systole. As a result the A-V valves are closed or nearly closed before ventricular systole. In ectopic ventricular beats the A-V valves are closed from an open position by ventricular contractions.

REFERENCES

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