Echocardiogram and Phonocardiogram Related to the Movement of the Pulmonary Valve

Tsuguya Sakamoto, M.D., F.A.C.C., Mokuo Matsuhisa, M.D., Terumi Hayashi, M.D., and Hirofumi Ichiyasu, M.D.

Summary

Pulmonary valve movement and the related acoustic phenomena were investigated using high speed strip-chart echo- and phonocardiographic recording. The opening of the pulmonary valve had no definite relationship to the acoustic phenomena, whereas the pulmonary ejection sound was closely related in time to the early systolic maximal opening of the valve. The concomitant pulmonary ejection systolic murmur faded away by the time of the mid-systolic semi-closure of the valve, where the tiny extrasound occurred in a half of cases. The pulmonary component of the second heart sound occurred after the valve closure, and the time lag maximally reached up to 50 msec. Pulmonary hypertension tended to minimize this delay, giving the so-called single loud second heart sound. Graham Steell murmur started with the pulmonary component of the second heart sound and reached up to the isometric contraction phase beyond the first heart sound.

Additional Indexing Words:
Pulmonary valve movement Pulmonary hypertension Pulmonary ejection sound Pulmonary ejection systolic murmur Mid-systolic extrasound Pulmonary component of the second heart sound Graham Steell murmur Mechanism of production of cardiovascular sound

Although the opening and closure of the cardiac valve have been regarded as having a close relationship to the heart sound production, the direct evidence is a matter of the method available. Up to the present time, some trials to solve the problem by the use of ultrasonic method, either echogram or Doppler signals, have been developed. However, recent ultrasonic technique with high speed strip-chart recording enables us much more accurate comparison with the phonocardiogram.

The present report deals with the phonocardiographic and echocardiographic correlation in regard to the pulmonary valve motion. Since the pulmonary valve echo is easily obtained in cases with pulmonary hypertension, the main sounds and murmurs under investigation are pulmonary ejection...
sound (PEj), pulmonary component of the second heart sound (IIP), pulmonary systolic murmur, and the Graham Steell murmur.

**MATERIALS AND METHODS**

Twelve cases of the pulmonary valve echogram were selected from the previous study\(^{14}\) to examine the phono- and echocardiographic correlation. The selection of the patient was based on the clearly demonstrated valve cusp echo, in which the coaptation of the cusps in both opening and closing points was observed. The strip-chart recording was made using Aloka SSD-90 polygraph, and the echogram, phonocardiograms obtained from 2 different areas simultaneously with an adequate filter, pulse tracings such as carotid pulse, jugular phlebogram, apex cardiogram, or indirect pulmonary artery pulse tracing (IPAPT),\(^{15}\) and electrocardiogram were simultaneously recorded. To identify the heart sounds and murmurs, additional routine phonocardiograms using multi-filter system phonocardiograph\(^{16}\) were utilized in all cases. Echocardiogram was recorded using an unfocused transducer of 2.25 MHz, 1.0 cm in diameter, having a repetition rate of 1,500 impulses per second. Paper speed was 50 mm/sec during observation, and 100 mm/sec was mainly utilized for the measurement.

**RESULTS**

The results are shown in Table I and the illustrative cases are shown in Figs. 1 to 5.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>MSE</th>
<th>Pulm. Ej. SM</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Y. S.</td>
<td>32</td>
<td>F</td>
<td>Eisenmenger syndrome</td>
<td>no</td>
<td>yes</td>
<td>61</td>
</tr>
<tr>
<td>2. T. Z.</td>
<td>46</td>
<td>M</td>
<td>Eisenmenger syndrome</td>
<td>yes</td>
<td>yes</td>
<td>72</td>
</tr>
<tr>
<td>3. T. M.</td>
<td>26</td>
<td>F</td>
<td>ECD</td>
<td>no</td>
<td>yes</td>
<td>61</td>
</tr>
<tr>
<td>4. S. M.</td>
<td>23</td>
<td>M</td>
<td>Eisenmenger complex</td>
<td>yes</td>
<td>yes</td>
<td>77</td>
</tr>
<tr>
<td>5. M. A.</td>
<td>23</td>
<td>F</td>
<td>Eisenmenger complex</td>
<td>no</td>
<td>yes</td>
<td>94</td>
</tr>
<tr>
<td>6. M. N.</td>
<td>32</td>
<td>F</td>
<td>Eisenmenger complex</td>
<td>yes</td>
<td>yes</td>
<td>90</td>
</tr>
<tr>
<td>7. N. N.</td>
<td>37</td>
<td>F</td>
<td>Eisenmenger syndrome</td>
<td>no</td>
<td>yes</td>
<td>72</td>
</tr>
<tr>
<td>8. E. K.</td>
<td>47</td>
<td>F</td>
<td>PSS, PH</td>
<td>yes</td>
<td>yes</td>
<td>86</td>
</tr>
<tr>
<td>9. T. K.</td>
<td>40</td>
<td>M</td>
<td>MS, TI, GS, AF</td>
<td>not clear</td>
<td>69</td>
<td></td>
</tr>
<tr>
<td>10. T. T.</td>
<td>29</td>
<td>M</td>
<td>PMD, CRBBB</td>
<td>no murmur</td>
<td>88</td>
<td></td>
</tr>
<tr>
<td>11. K. S.</td>
<td>9</td>
<td>F</td>
<td>PMD</td>
<td>not clear</td>
<td>91</td>
<td></td>
</tr>
<tr>
<td>12. T. M.</td>
<td>62</td>
<td>M</td>
<td>MS, TI, AF</td>
<td>not clear</td>
<td>73</td>
<td></td>
</tr>
</tbody>
</table>

1. Pulmonary ejection sound (PEj):

PEj appeared 50 to 80 msec after the separation of the valve echo, and it coincided in time with the early systolic maximal opening of the valve in 7 out of 8 cases. An exceptional case showed minimal delay of PEj (0 to 10 msec) (cf. Fig. 3).

2. Pulmonary systolic murmur:

Concomitant short systolic murmur of pulmonary origin was observed in 8 cases. The end of the murmur closely coincided with the mid-systolic semi-closure of the valve (approximation of the cusp echo), and the echo showed re-opening movement thereafter. An exceptional case had the three-fold opening motion, and the murmur extended to the time of the second semi-closure.

At the time of the semi-closure of the valve and the end point of the concomitant systolic murmur, a systolic click of small amplitude was observed in some cases (Figs. 2, 3 and 6).

3. Pulmonary component of the second heart sound (IIP):

IIP occurred 0 to 50 msec after the coaptation of the cusp echo. With increased pulmonary artery pressure, this delay tended to be minimized and finally no apparent delay was observed in cases with severe pulmonary hypertension.

In a case with pulsus alternans, the strong beat showed no apparent

Phonocardiographic Data in 12 Patients

<table>
<thead>
<tr>
<th>Time Intervals (msec)</th>
<th>Q-Op</th>
<th>Q-MOp</th>
<th>Q-PEj</th>
<th>Q-Cp</th>
<th>Q-IIP</th>
<th>(Q-Cp)-(Q-IIP)</th>
</tr>
</thead>
<tbody>
<tr>
<td>70</td>
<td>130</td>
<td>130</td>
<td>430</td>
<td>430</td>
<td>440</td>
<td>-10-0</td>
</tr>
<tr>
<td>80</td>
<td>130</td>
<td>130</td>
<td>400</td>
<td>400</td>
<td>410</td>
<td>-10-0</td>
</tr>
<tr>
<td>80</td>
<td>130</td>
<td>140</td>
<td>410</td>
<td>410</td>
<td>0</td>
<td>-10-0</td>
</tr>
<tr>
<td>60-70</td>
<td>120</td>
<td>120</td>
<td>360</td>
<td>360</td>
<td>0</td>
<td>-10-0</td>
</tr>
<tr>
<td>100</td>
<td>160</td>
<td>160</td>
<td>360</td>
<td>360</td>
<td>370-380</td>
<td>-10---20</td>
</tr>
<tr>
<td>80</td>
<td>130</td>
<td>130</td>
<td>370</td>
<td>370</td>
<td>0</td>
<td>-10-0</td>
</tr>
<tr>
<td>100</td>
<td>160</td>
<td>160</td>
<td>400-410</td>
<td>410</td>
<td>420</td>
<td>-20-0</td>
</tr>
<tr>
<td>70</td>
<td>130</td>
<td>130</td>
<td>350</td>
<td>350</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>90</td>
<td>140</td>
<td>120</td>
<td>320-340</td>
<td>350</td>
<td>380</td>
<td>-30---10</td>
</tr>
<tr>
<td>not appreciable</td>
<td>210</td>
<td>370</td>
<td>410</td>
<td>410</td>
<td>0</td>
<td>-40</td>
</tr>
<tr>
<td>not appreciable</td>
<td>not appreciable</td>
<td>360* &amp; 340**</td>
<td>360* &amp; 350**</td>
<td>0*---10**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100</td>
<td>150</td>
<td>290-300</td>
<td>330-350</td>
<td>330-350</td>
<td>-50---40</td>
<td></td>
</tr>
</tbody>
</table>

atrial fibrillation, PMD: primary myocardial disease, CRBBB: complete right bundle-branch block, AI: aortic insufficiency. All time intervals are expressed by milliseconds. In Case 11, * indicates strong beat and ** indicates weak beat of the alternating pulse. In Case 9 to 12, the pulm. Ej. SM was not identified due to either another murmur or no murmur.
Fig. 1. Echocardiogram of the pulmonary valve in a case of pulmonary hypertension. 46-year-old male. Top; Pulmonary valve echo, phonocardiograms (2L; the second left intercostal space, and Apex), and indirect pulmonary artery pulse tracing (IPAPT) from the maximal point of the pulmonary ejection sound and the second heart sound, which are illustrated in the lower tracing (bottom figure). The pulmonary cusp echo showed typical W-shaped configuration with mid-systolic semi-closure and diastolic
Movemen of Pulmonary Valve

Fig. 2. Pulmonary cusp echo in a case of Eisenmenger syndrome with Graham Steell murmur. 32-year-old female. Phonocardiograms (both medium and high frequency) are recorded from the second left intercostal space (2L). The echo pattern of the pulmonary cusp was typical for pulmonary hypertension (W-shaped systolic movement and diastolic plateau). The pulmonary ejection sound (Ej) occurred at the time of early systolic maximal opening of the valve, but not the time of the valve opening per se. There is only minimal systolic murmur of brief duration, and its end coincided in time with the early systolic semi-closure of the cusp. IIP almost coincided with the time of the valve closure. Graham Steell murmur persists throughout diastole, from the valve closure to the early stage of the valve opening of the next beat.

delay, but weak beat showed 10 msec delay of the IIP (Case 11).

4. Graham Steell murmur:

Graham Steell murmur was observed in 5 cases. In all 5, this murmur

plateau characteristic to severe pulmonary hypertension. The pulmonary ejection sound occurred at the time of the early systolic full opening motion of the cusp, which delayed to the separation of the cusp echo by 50 msec. The pulmonary component of the second heart sound(IIP) has large amplitude just behind to the aortic component(IIA). The closing point of the pulmonary valve is inscribed 5 msec after IIA and 10 msec before IIP. The IIP coincided in time with the incisure of 1PAPT. The end of systolic murmur was expressed by a small click (bottom tracing; H 1, K), and this coincided with the mid-systolic semi-closure of the pulmonary valve echo. In addition, there is a pansystolic murmur of tricuspid regurgitation at the apex, and also is distinct right ventricular heave, which was recorded in the 4th intercostal space laterally to the sternal margin (4L lat. LF). Time lines ; 0.05 sec (top figure) and 0.01 and 0.1 sec (bottom figure).
Fig. 3. Pulmonary cusp echo in a case of moderately elevated pulmonary artery pressure. 26-year-old female. The upstroke of the IPAPT coincided with the time of full opening of the valve, but the pulmonary ejection sound occurs 10 msec later. The W-shaped systolic movement is not clear in this case, because of the high speed and small scaled recording (half scale). However, it is observed that the systolic murmur ends at the time of the peak of "W" arrow) with the small mid-systolic extrasound (arrow). IIP delayed 10 msec to the coaptation of the pulmonary cusp echo.

started with IIP and extended throughout the diastole, and ended after the first heart sound. The end of the murmur was not so definite, but it was disclosed that the murmur persisted beyond the early phase of the pulmonary valve opening up to the time of PEj (cf. Fig. 2).

DISCUSSION

Although the cardiovascular sounds have been studied based on the phonocardiographic and echocardiographic correlation, the pulmonary acoustic events have not been fully elucidated by the same technical means. Previous study of the pulmonary valve echogram has already disclosed frequently the unexpected lack of coincidence of echographic and acoustic events.

First of all, there was big temporal difference between the opening motion of the pulmonary valve (Op) and the pulmonary ejection sound (PEj). This fact favors the classical concept that the semilunar valve opens without noise. In fact, phonocardiogram disclosed no particular heart sound at the time of the separation of the pulmonary cusp echo, or more accurately, the
Fig. 4. Pulmonary cusp echo in a case of congestive heart failure. 62-year-old male. Mitral stenosis, tricuspid insufficiency, and aortic insufficiency. The second heart sound was not split during respiration, but the initial vibration was delayed to the pulmonary cusp coaptation by 40 msec (first 3 beats) or 50 msec (last beat). If the IIP is buried in this second heart sound, the delay should be much more marked. Aortic cusp echo also showed earlier coaptation of the cusp (5-10 msec) prior to the aortic component of the second heart sound (IIA).

latter coincided with the small vibration which was inseparable from the first heart sound.

On the contrary, the close temporal relationship of the PEj and the maximal opening of the pulmonary valve suggests that this acoustic phenomenon is probably due to the sudden check of the pulmonary valve motion, and the mechanism of the sound production seems to be similar to the mitral opening snap, which coincides in time with the full opening of the mitral valve, i.e., E point. In this respect, it seems likely that the abruptness of the valve motion will cause louder PEj, but this was not proved in this small group of the patient.

The pulmonary systolic murmur, frequently observed in cases with pulmonary hypertension, is a flow murmur of various quality. It is interesting enough that this flow murmur also had the definite temporal relationship to the pulmonary valve motion. The mid-systolic approximation of the cusp echo of the pulmonary valve, which gives the "W-shaped" pattern, was quite often observed in cases with pulmonary hypertension and coincided with the end of the systolic murmur. This fact suggests that the effective pulmonary
blood flow ceases with the mid-systolic semi-closure of the valve. In this respect, it is noteworthy that the semi-closure of the valve generates a tiny sound, probably due to the relatively sudden check of the cusp motion. This tiny sound may be observed even in cases with innocent systolic murmur (so-called “relaxation sound” of the pulmonary valve17), though the pitch is less high and never audible. The impression is that the faster the semi-closure, the louder the mid-systolic extrasound. Clinically, this extrasound
Fig. 6. Diagram illustrating the time relationship between phonocardiogram and pulmonary cusp echo (PV) in 8 cases with pulmonary ejection systolic murmur. Pulmonary ejection sound (Ej) coincides with the early to mid-systolic maximal opening of the cusp. The systolic ejection murmur ends at the time of the mid-systolic approximation of the cusp, where the tiny click or extrasound (K) is often inscribed (Cases 2, 4, 6, and 8).

may be the mid-systolic click of non-mitral origin.\textsuperscript{18)}

The second heart sound, in this instance the IIP, is a matter of ample discussion. The time lag of IIP to the pulmonary valve closure (Cp) is quite common, or dare to say, is a rule in both normal and pathological conditions. This is largely due to the time lag of the tension of the valve and artery by the back pressure.\textsuperscript{19)} Since the pulmonary artery has large compliance, the delay of IIP to Cp is normally very long. It takes over 50 msec in normal cases, and the superimposed figure demonstrates that the Cp occurs almost simultaneously with the aortic valve closure (Ca), despite of the presence of the widely split 2 components (IIA and IIP). This is in accord with the statement of Luisada and MacCanon,\textsuperscript{20)} who explained the mechanism of the splitting of the second heart sound. Greater compliance of the pulmonary artery causes a delayed rebound of its pressure pulse over the already closed valve leaflets, so that a more delayed appearance of the IIP as well as the
incisure will take place. Pulmonary hypertension does abolish the interval of the split components of the second heart sound as well as the delay of IIP to the Cp. Thus, it is concluded that the absence of, or minimal, splitting of the second heart sound in pulmonary hypertension is mainly due to the elimination of the time lag due to the back pressure, most probably due to the elevated pulmonary artery resistance or diminished compliance. Although the degree of the delay is much smaller, the same is true to the temporal relationship between Ca and IIA, the delay of the latter is abolished by methoxamine infusion.

The Graham Steell murmur has not been adequately investigated by echo- and phonocardiographic correlation. The present result indicates that this murmur ends after the next first heart sound and reaches up to the PEj. This implies that the reversed blood flow may persist shortly after the opening of the pulmonary valve. This may be unbelievable, however, it should be clarified by another investigation including Doppler method.

Finally, it should be emphasized that the temporal relationship between the acoustic phenomena and the cardiac events studied earlier by either pressure tracings, or flow studies, or cineangiographic observation, or even the echocardiography using analog gate technique, may be criticized and has to be re-studied by the simultaneous echo- and phonocardiographic records using strip-chart at a high speed. This should be of value to clarify the real mode of production of the cardiovascular sounds in health and disease.

REFERENCES


