Echocardiographic Observations on the Mechanism of Production of the Second Heart Sound

By Premindra A. N. Chandraratna, M.D., M.R.C.P., Jose M. Lopez, M.D., and Lawrence S. Cohen, M.D.

with the technical assistance of Dale Gindlesperger

Summary

This investigation was designed to determine the echocardiographic correlates of the aortic and pulmonary components of the second heart sound. Simultaneous recordings of the ECG, phonocardiogram, carotid pulse tracing and the echocardiogram of the aortic valve were made in 54 patients. The tracings were recorded at a paper speed of 100 mm/sec, with 20 msec time lines. Aortic valve closure (AVC) on the echocardiogram occurred before the onset of the aortic component of the second heart sound (A2) in all but two patients. The mean AVC-to-A2 interval was 12 msec with a range of 5 to 25 msec. Simultaneous recordings of the pulmonary valve echocardiogram, ECG, phonocardiogram and carotid pulse tracing were made in 14 patients. Pulmonary valve closure (PVC) on the echocardiogram preceded the pulmonary component of the second sound (P2) in all patients. The mean PVC-P2 interval was 60 msec with a range of 30 to 75 msec. We conclude that aortic valve closure and pulmonary valve closure are not directly responsible for the genesis of the second heart sound (S2). These observations are consonant with the theory that S2 is caused by deceleration of columns of blood resulting from semilunar valve closure, throwing the cardiohemic system into vibration.

Additional Indexing Words:
Ultrasound  Pulmonary valve closure  Aortic valve closure

The Origin of the second heart sound has been thought to be due to semilunar valve closure. More recently, Criley and his associates used rapid cineangiographic films and demonstrated that the second sound occurred several milliseconds after valve closure. They also confirmed the association of the second sound with the incisura of the aortic pressure pulse. MacCanon and co-workers observed that aortic valve closure in the dog occurred several milliseconds before the aortic incisura. They concluded that the second heart sound was not caused by coaption of the aortic valve leaflets, but by the same mechanisms which are responsible for the incisura.

Echocardiography has been extensively used to study intracardiac structures. Gramiak and Shah described the echocardiographic features of the aortic root, and of the normal and diseased aortic valve. Echocardiographic detection of the pulmonary valve was also described by Gramiak and his co-workers.

We used echocardiography to investigate the temporal relationship of aortic and pulmonary valve closure to the two components of the second sound. The results of our study constitute this report.

Materials and Methods

Sixty patients were studied. The group consisted of 40 males and 20 females. Their ages ranged from 10 to 83 years. Twenty-eight patients had coronary artery disease; five had congenital heart disease; nine had mitral valve disease; 14 had no demonstrable heart disease; two had cardiomyopathy; one patient had aortic incompetence; and one had idiopathic pulmonary hypertension.

The electrocardiogram, phonocardiogram, echocardiogram and external carotid pulse tracing were recorded simultaneously at a paper speed of 100 mm/sec with 20 msec time lines. The phonocardiograms were recorded from the second or third interspace at the left sternal border, using the logarithmic setting (12 dB octave slope) on an Electronics for Medicine recorder. The echocardiogram was recorded using a 1 cm focus, 0.5 inch, 2.25 MHz transducer, a commercially available ultrasonoscope and an Electronics for Medicine DR-8 strip chart recorder. Aortic root and aortic valve echoes were obtained by placing the transducer at the third or fourth interspace at the left sternal edge and by angulating it superiorly and medially. Long strips were recorded for analysis. The time interval between aortic valve closure and the aortic component of the second sound was obtained by averaging the measurements obtained in 5 to 10 beats.

From the Division of Cardiology, Mount Sinai Medical Center, Miami Beach, and the University of Miami, Coral Gables, Florida.

Address for reprints: P.A.N. Chandraratna, M.D., Division of Cardiology, Mount Sinai Medical Center, 6300 Alton Road, Miami Beach, Florida 33140.

Received August 14, 1974; revision accepted for publication October 14, 1974.
Simultaneous recordings of the pulmonary valve echocardiogram, electrocardiogram, phonocardiogram and external carotid pulse tracing was made in 14 patients. The paper speed used was 100 mm/sec, with 20 msec time lines. The time interval between pulmonary valve closure and the pulmonary component of the second heart sound was calculated from these data by averaging the measurements from 5 to 10 beats.

Of the 60 patients, 46 had echocardiograms of the aortic valve alone; eight had both pulmonary and aortic valve recordings; and six had only the pulmonary valve recorded.

### Results

The aortic valve closure on the echocardiogram (AVC) was seen to precede the aortic component of the second heart sound (A2) in all but two patients. The AVC-to-A2 interval ranged from 5 to 25 msec with a mean of 12 msec (SD ± 5). The individual data are given in table 1. Figure 1 illustrates a record of the electrocardiogram, external carotid pulse tracing, phonocardiogram and the echocardiogram of the aortic valve of patient R. T., a 50-year-old man with a mitral Ball prosthet. Aortic valve closure is seen to occur before the first high frequency transient of the aortic component of the second sound. A recording on the same patient at a paper speed of 200 mm/sec and 20 msec time lines is illustrated in figure 2. The separation between AVC and A2 is clearly discernible.

Pulmonary valve closure (PVC) preceded the pulmonary component of the second sound (P2) in all patients. The PVC-to-P2 interval ranged from 30 to 75 msec with a mean of 60 msec (SD ± 13). Table 2 shows the individual data. A simultaneous record of the electrocardiogram, carotid pulse tracing, phonocardiogram and echocardiogram of the pulmonic valve of patient T. W., a 26-year-old man with a cardiomyopathy, is illustrated in figure 3. Pulmonary valve closure is seen to precede the pulmonary component of the second sound. The patient with idiopathic pulmonary hypertension, and the subject with pulmonary hypertension secondary to an atrial septal defect had short PVC-to-P2 intervals.

Eight patients had both aortic and pulmonary valve echocardiograms. The PVC-to-P2 interval was greater than the AVC-to-A2 interval in all the subjects.

### Discussion

On the basis of phonocardiographic studies, Leatham concluded that the second heart sound was caused by aortic and pulmonary valve closure.1 On the

---

*Patients who had both aortic and pulmonic valve echocardiograms.

Abbreviations: AVC-A4 interval = time interval from aortic valve closure on the echocardiogram to the aortic component of the second sound; CAD = coronary artery disease; N = normal; MVD = mitral valve disease; ASD = atrial septal defect; VSD = ventricular septal defect; CM = cardiomyopathy; PHT = pulmonary hypertension.
other hand, Rushmer hypothesized that heart sounds were due to vibrations of the "cardiohemic system" caused by accelerations and decelerations. The cineangiographic studies of Criley and others, and the animal studies of MacCanon and associates have supported Rushmer’s postulates on the mechanism of production of the second heart sound. Mori and co-workers used intracardiac phonocardiograms and pressure recordings, and external phonocardiograms in a series of dog experiments, to delineate the hemodynamic correlates of the different components of the second sound. They observed that the first component of the second sound was closely related to the incisura of the aortic pressure trace and the second component was related to the pulmonary incisura. Two other vibrations were occasionally observed; one preceded the first component and the other followed the second component. Their results were also in agreement with Rushmer’s hypothesis.

Our study shows that in all but two patients, aortic valve closure precedes the aortic component of the second heart sound by 5 to 25 msec. Similarly, pulmonary valve closure occurs 30 to 75 msec before the pulmonary component of the second sound. These findings are consistent with Rushmer’s and Luisada’s concepts on the mechanism of production of the second heart sound.

The possibility that a delay in the recording system may have influenced our results must be considered. Several observations preclude such an error. Martin and associates found no delay in the external sound when it was compared with intracardiac recordings from a catheter tip micromanometer. Phonocardiographic and echocardiographic observations on Beall mitral valves in our laboratory are also relevant (unpublished observations). We have observed that the opening click of the Beall valve recorded on the external phonocardiogram corresponds exactly with the point of contact of the disc with the cage, as seen on the echocardiogram. Thus, it is unlikely that the recording system introduced a significant error into our observations.
Our study shows that the PVC-P₂ interval is greater than the AVC-A₂ interval. This could be explained on the basis of a greater distensibility and compliance of the pulmonary artery and pulmonary vascular bed as compared with the aorta and the systemic vascular bed. Thus, the rebound of the pressure pulse in the pulmonary artery occurs later. The two patients with pulmonary hypertension had reduced PVC-P₂ intervals, which suggest a reduced compliance of the pulmonary vascular bed in these cases. Shaver and associates used intracardiac catheter tip micromanometers to simultaneously measure aortic and left ventricular pressure pulses. They coined the term "hangout" to define the interval between the aortic incisura and the left ventricular pressure at the level of the incisura. They observed that in patients with normal pulmonary vascular resistance, the "hangout" on the right side of the heart was considerably longer than that on the left side. This was attributable to the higher capacitance of the pulmonary vascular tree.

In summary, the phonocardiographic and echocardiographic correlates of the second heart sound are presented. Our observations suggest that semilunar valve closure is not directly responsible for the genesis of the two components of the second heart sound. Our observations are compatible with the theory that the second heart sound is caused by decelerations of columns of blood resulting from semilunar valve closure, throwing the cardiohemic system into vibra-

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Diagnosis</th>
<th>PVC-P₂ Interval (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. P.M.</td>
<td>58</td>
<td>CAD</td>
<td>60</td>
</tr>
<tr>
<td>2. D.J.*</td>
<td>48</td>
<td>MVD</td>
<td>60</td>
</tr>
<tr>
<td>3. S.H.*</td>
<td>62</td>
<td>CAD</td>
<td>63</td>
</tr>
<tr>
<td>4. C.H.*</td>
<td>29</td>
<td>MVD</td>
<td>75</td>
</tr>
<tr>
<td>5. S.J.*</td>
<td>15</td>
<td>N</td>
<td>70</td>
</tr>
<tr>
<td>6. H.R.*</td>
<td>23</td>
<td>VSD</td>
<td>55</td>
</tr>
<tr>
<td>7. B.K.</td>
<td>31</td>
<td>Idiopathic PHT</td>
<td>35</td>
</tr>
<tr>
<td>8. W.M.*</td>
<td>33</td>
<td>ASD &amp; PHT</td>
<td>30</td>
</tr>
<tr>
<td>9. F.H.*</td>
<td>63</td>
<td>CAD</td>
<td>60</td>
</tr>
<tr>
<td>10. G.J.</td>
<td>24</td>
<td>ASD</td>
<td>70</td>
</tr>
<tr>
<td>11. W.A.</td>
<td>83</td>
<td>CAD</td>
<td>70</td>
</tr>
<tr>
<td>12. R.J.</td>
<td>28</td>
<td>MVD</td>
<td>75</td>
</tr>
<tr>
<td>13. T.W.*</td>
<td>26</td>
<td>CM</td>
<td>60</td>
</tr>
<tr>
<td>14. J.H.</td>
<td>18</td>
<td>N</td>
<td>50</td>
</tr>
</tbody>
</table>

*Patients who had both aortic and pulmonic valve echocardiograms.

Abbreviations: PVC-P₂ interval = time interval from pulmonic valve closure on the echocardiogram to the pulmonary component of the second sound; CAD = coronary artery disease; N = normal; ASD = atrial septal defect; PHT = pulmonary hypertension; MVD = mitral valve disease; VSD = ventricular septal defect; CM = cardiomyopathy.

Figure 3

Echocardiogram of the pulmonary valve (PV) recorded simultaneously with the electrocardiogram, external carotid pulse (CA), and phonocardiogram. (Paper speed 100 mm/sec with 20 msec times lines.) Pulmonary valve closure is seen to occur before the pulmonary component of the second sound (P₂). A₂ = aortic component of the second sound.

References


Circulation, Volume 51, February 1975
Echocardiographic observations on the mechanism of production of the second heart sound.
P A Chandraratna, J M Lopez and L S Cohen

Circulation. 1975;51:292-296
doi: 10.1161/01.CIR.51.2.292

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/51/2/292

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/