Sound Pressure Correlates of the Second Heart Sound

An Intracardiac Sound Study

By James A. Shaver, M.D., Richard A. Nadolny, M.D., James D. O'Toole, M.D., Mark E. Thompson, M.D., P. S. Reddy, M.D., Donald F. Leon, M.D., and Edward I. Curtiss, M.D.

SUMMARY
The sound pressure correlates of the second heart sound were studied in 22 patients during diagnostic cardiac catheterization. Simultaneous right ventricular and pulmonary artery pressures were recorded with equisensitive catheter-tip micromanometers together with the external phonocardiogram and ECG. In 12 patients having normal pulmonary vascular resistance (group 1), pulmonic closure sound was coincident with the incisura of the pulmonary artery pressure curve which in turn was separated from the right ventricular pressure trace by an interval denoted hangout. The duration of this interval varied (33-89 msec), was independent of pulmonary artery pressure or resistance and was felt to be primarily a reflection of the capacitance of the pulmonary vascular tree. The absolute value of this interval during inspiration was very similar to the splitting interval, and when subtracted from the Q-Pa interval, the remaining interval (QRV) was almost identical to the Q-Aa interval, indicating that the actual duration of right and left ventricular systole is nearly equal. Awareness of the existence of the hangout interval and its hemodynamic determinants offers a reasonable mechanism to explain the audible expiratory splitting of the second heart sound found in patients with idiopathic dilatation of the pulmonary artery following atrial septal defect repair and in one additional patient studied with mild valvular pulmonic stenosis. In nine patients with elevated pulmonary vascular resistance approaching systemic levels (group 2), the absolute value of the hangout interval was markedly reduced (15-28 msec) consistent with the decrease in capacitance of the pulmonary vascular bed and the increased pulmonary vascular resistance known to occur in pulmonary hypertension. In those patients where the duration of right and left ventricular systole was nearly equal, narrow splitting of the second heart sound was present. In those patients where selective prolongation of right ventricular systole occurred, the narrow hangout interval persisted, but the splitting interval was prolonged proportionate to the increased duration of right ventricular systole.

Additional Indexing Words:
Phonocardiogram  Right ventricular mechanics  Hangout interval
Equisensitive catheter-tip micromanometers  Audible expiratory splitting
Pulmonary hypertension

Slightly over 100 years ago, Potain recognized splitting of the second heart sound during normal inspiration.¹ In the past two decades, considerable attention has been paid to the second heart sound in both normal and diseased states: Leatham has labeled it the key to auscultation of the heart.² However, such findings as wide splitting of the second heart sound in patients without demonstrable heart disease,³ audible expiratory splitting following atrial septal defect repair⁴ and inappropriately wide splitting in mild pulmonic stenosis⁵ are still not adequately understood. In addition, no ready explanation is yet available of the narrow splitting frequently associated with pulmonary hypertension.⁶⁻⁹ In fact, recent studies by Sutton, Harris and Leatham have challenged the classic observations of Wood regarding the abnormally close splitting of the second heart sound in

From the Department of Medicine, Division of Cardiology, University of Pittsburgh School of Medicine and the Presbyterian-University Hospital, Pittsburgh, Pennsylvania.

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Address for reprints: James A. Shaver, M. D., Director, Division of Cardiology, University of Pittsburgh School of Medicine, 780 Scaife Hall, Pittsburgh, Pennsylvania 15261.

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pulmonary hypertension. This study was undertaken to further define the sound and pressure correlates of the second heart sound and those factors which determine the splitting interval.

**Materials and Methods**

Twenty-two patients were studied during diagnostic cardiac catheterization and were grouped according to their pulmonary vascular resistance. Included in group 1 with normal pulmonary resistance are five patients with innocent systolic ejection murmurs; four patients with atrial septal defects; one patient with idiopathic dilatation of the pulmonary artery; one patient with a patent ductus arteriosus; and one patient with a pericardial cyst. There was no evidence of right ventricular outflow tract obstruction or right bundle branch block in any patient. Although RSR prime complexes in V1 were present in some patients with atrial septal defects, the interval from the Q wave to the onset of right ventricular pressure rise was normal in these patients.

Group 2 consisted of nine patients with elevated pulmonary vascular resistance approaching systemic levels and included three patients with severe mitral stenosis; three patients with idiopathic pulmonary hypertension; one patient with progressive systemic sclerosis; one patient with Lutembacher's syndrome; and one chronic drug abuser with severe pulmonary hypertension, the etiology of which was not determined at postmortem examination. In addition, one other patient, G. C., with mild valvular pulmonary stenosis and normal pulmonary resistance was studied and not included in either group. The pertinent clinical and hemodynamic data of these patients are summarized in table I.

All patients were studied in the supine position. Intracardiac sound and pressure events were recorded by Dallons-Telco catheter-tip micromanometers.* The micromanometer is a variable inductance transducer from which low frequency vibrations are recorded as pressure and higher frequency vibrations (above 40 Hz) are recorded as sound, thus permitting both intracardiac sound and pressure events to be recorded free of contour distortion and without transmission delay. Two catheter-tip micromanometers were introduced through antecubital venotomies and placed into the right ventricle where their pressures were made equisensitive. One micromanometer was then placed into the pulmonary artery just above the valve from which both sound and pressure were recorded (fig. 1). Equisensitivity was checked frequently throughout the study by pullback of the pulmonary artery micromanometer into the right ventricular outflow tract. Great care was taken during the manipulation of these catheters to avoid the production of artifactual sounds. A phonocardiogram was recorded on the chest wall at the point of maximal intensity of the aortic and pulmonic closure sounds together with the electrocardiogram. Continuous respirations were monitored with a nasal thermistor. In addition, simultaneous aortic root pressure was recorded with a Statham catheter-tip micromanometer in some patients. These data were recorded simultaneously on a multichannel Electronics for Medicine photographic recorder at a paper speed of 100 mm/sec with time markers indicating 20 msec (fig. 1).

The following intervals were measured during each study and were the average of at least five complexes taken during a constant phase of inspiration determined by the nasal thermistor.

**Q-P2**—The indirect measurement of the total duration of right ventricular electromechanical systole. The interval from the onset of the Q wave of the electrocardiogram to the first high frequency component of the pulmonic closure sound recorded from the external phonocardiogram.

**Q-A2**—An indirect measurement of the total duration of the left ventricular electromechanical systole. The interval from the onset of the Q wave of the electrocardiogram to the first high frequency compo-

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*Carolina Medical Electronics, King, North Carolina.

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**Figure 1**

(Far left) Two catheter-tip micromanometers are located in the body of the right ventricle where their pressures are made equisensitive. (Near left) One of the equisensitive micromanometers is placed into the pulmonary artery just above the valve from which both sound and pressure are recorded together with the external phonocardiogram and the electrocardiogram. A1 and P2 are clearly seen on both the external and internal phonocardiogram. The Q-A2 and Q-P2 intervals are measured from the onset of the Q wave of the electrocardiogram to the first high frequency component of the aortic and pulmonic closure sounds, respectively. The hangout interval is the 44 msec interval separating the right ventricular pressure curve from the pulmonary artery incisura measured at the pressure level of the incisura. Paper speed = 100 mm/sec; time lines = 20 msec.
Table 1

Clinical and Hemodynamic Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr.) and sex</th>
<th>Diagnosis</th>
<th>CO* (L/min)</th>
<th>PA (mm Hg)</th>
<th>Wedge (mm Hg)</th>
<th>TPR, dynes-sec-em⁻²</th>
<th>PVR, dynes-sec-em⁻²</th>
<th>Hangout (mssec)</th>
<th>Aₚ₋₁ (mssec)</th>
<th>Q₋₁ (mssec)</th>
<th>Q₋₂ (mssec)</th>
<th>Q₋₃ (mssec)</th>
<th>QRV (mssec)</th>
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<td></td>
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<td>24/6 (11)</td>
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<td>166</td>
<td>75</td>
<td>43</td>
<td>48</td>
<td>403</td>
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<td>Normal</td>
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<td>28/12 (16)</td>
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<td>78</td>
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<td>Normal</td>
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<td>18/6 (10)</td>
<td>4</td>
<td>151</td>
<td>91</td>
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<td>152</td>
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<td>PDA</td>
<td>(1.2/1)</td>
<td>14/6 (10)</td>
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<td>Normal</td>
<td>Normal</td>
<td>59</td>
<td>60</td>
<td>458</td>
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<td>8. D. P.</td>
<td>41 F</td>
<td>IDPA</td>
<td>(2.3/1)</td>
<td>32/8 (16)</td>
<td>9</td>
<td>Low</td>
<td>Low</td>
<td>51</td>
<td>59</td>
<td>424</td>
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<td>50 F</td>
<td>ASD</td>
<td>(2.1/1)</td>
<td>32/16 (20)</td>
<td>8</td>
<td>Low</td>
<td>Low</td>
<td>47</td>
<td>46</td>
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<td>ASD</td>
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<td>24/8 (16)</td>
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<td>144</td>
<td>90</td>
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<td>30/10 (16)</td>
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<tr>
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<td>25 F</td>
<td>IPH</td>
<td>4.7</td>
<td>90/50 (60)</td>
<td>4</td>
<td>1021</td>
<td>953</td>
<td>20</td>
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<td>44 F</td>
<td>IPH</td>
<td>2.1</td>
<td>93/35 (50)</td>
<td>5</td>
<td>1905</td>
<td>1713</td>
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<td>35 M</td>
<td>IPH</td>
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<td>60/30 (40)</td>
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<td>970</td>
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<td>IPH</td>
<td>3.1</td>
<td>80/30 (50)</td>
<td>—</td>
<td>1290</td>
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<tr>
<td>17. M. S.</td>
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<td>FSS</td>
<td>2.8</td>
<td>95/40 (65)</td>
<td>—</td>
<td>1857</td>
<td>—</td>
<td>15</td>
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<tr>
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<td>62 F</td>
<td>Lutembacher’s</td>
<td>6.3 (1.4/1)</td>
<td>100/45 (55)</td>
<td>20</td>
<td>691</td>
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<td>428</td>
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<td>RHD; MS</td>
<td>3.2</td>
<td>55/25 (40)</td>
<td>20</td>
<td>1016</td>
<td>908</td>
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<td>PS</td>
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<td>24/8 (15)</td>
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<td>—</td>
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<td>69</td>
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<td>371</td>
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Abbreviations: CO = cardiac output; PA = pulmonary artery pressure; Wedge = pulmonary artery wedge mean pressure; TPR = total pulmonary resistance; PVR = pulmonary vascular resistance; Hangout = the interval separating the right ventricular pressure curve from the pulmonary artery incisura; A₋₁₋₃ = duration of splitting of the second heart sound; Q₋₁ = duration of right ventricular electromechanical systole; Q₋₂ = duration of left ventricular electromechanical systole; QRV = Q₋₁₋₃ interval minus the hangout interval; Group 1 = patients with normal pulmonary vascular resistance; Group 2 = patients with elevated pulmonary vascular resistance; IDPA = idopathic dilatation of the pulmonary artery; ASD = atrial septal defect; IPH = idiopathic pulmonary hypertension; PSS = progressive systemic sclerosis; RHD = rheumatic heart disease; mitral stenosis; PS = pulmonic stenosis.

*In patients with left to right shunts, CO is expressed as pulmonary flow in L/min or as pulmonary-to-systemic flow ratio.
nent of the aortic closure sound recorded from the external phonocardiogram.

Hangout—The interval separating the right ventricular pressure curve from the pulmonary artery incisura as measured from equisensitive micromanometers at the pressure level of the incisura.

From these measured intervals, the following intervals were derived:

A2-P2—The duration of splitting of the second sound. The difference between the Q-P2 and the Q-A2 interval, i.e., the Q-P2 - Q-A2. This interval was also measured directly as the interval between the first high frequency component of the aortic and pulmonic closure sounds as measured from the external phonocardiogram.

QRV—Since the hangout interval is not primarily a reflection of right ventricular electromechanical events but rather a vascular phenomenon, a truer approximation of the total duration of right ventricular electromechanical systole is derived by subtracting the hangout interval from the Q-P2 interval. Such correction is necessary in the pulmonary circulation because of the large value of the hangout interval which may vary from 33 to 89 msec (table 1).

Results

Systemic Circulation

Figure 2 is representative of the sound and pressure relationships of the normal systemic circulation and will serve as a reference for comparison with subsequent illustrations of right-sided events.

Pulmonary Circulation Group 1

Figures 3 through 7 represent the spectrum of sound pressure correlates found in the 12 patients with normal pulmonary vascular resistance. In each patient, P2 is coincident with the incisura of the pulmonary artery tracing which in turn is separated from the right ventricular pressure trace by hangout intervals of varying duration. Figure 3 demonstrates the sound pressure correlates associated with normal physiologic splitting in a 17-year-old male (T. W.) with only a functional murmur, while figure 4 shows the wide inspiratory and expiratory hangout interval responsible for audible expiratory splitting without demonstrable heart disease. Figure 5 is representative of the sound and pressure relationship seen in all four patients with normotensive atrial septal defects (table 1). The mechanism

Figure 2

Sound pressure correlates of the normal systemic circulation. Equisensitive catheter-tip micromanometers are located in the left ventricle and central aorta and their pressures are recorded simultaneously with central aortic sound, external sound, left ventricular dp/dt and the electrocardiogram. A2 is coincident with the incisura of the central aorta which in turn is separated from the left ventricular pressure curve by an interval of 10 msec, denoted hangout. Note the close tracking of the left ventricular and aortic pressure during the protodiastolic phase of systole.

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Figure 3

(Upper Panel): External phonocardiogram recorded from a 17-year-old male (T.W.) demonstrating normal physiological splitting of the second heart sound. A functional systolic ejection murmur is also recorded. (Lower Panel): Simultaneous right-sided sound and pressure correlates found in this patient, which are representative of the sound pressure correlates of the normal pulmonary circulation. A2 and P2 are clearly defined on both phonocardiograms with a splitting interval of 52 msec. P2 is coincident with the incisura of the pulmonary artery which in turn is clearly separated from the right ventricular pressure curve by an interval of 46 msec. The functional systolic ejection murmur (SEM) is recorded by the internal phonocardiogram, confirming its pulmonary origin.
artery pressure and resistance were present in the 12 patients, all values were within normal limits. There was no relationship between the duration of the hangout interval and these minor variations in pressure and resistance, indicating that the hangout interval was independent of either. In no case was delayed pulmonic closure associated with right bundle branch block or with right ventricular outflow obstruction. As can be seen from Table 1, the absolute value of the A2-P2 interval and the hangout interval is very similar. When the hangout interval is subtracted from the Q-P2 interval, a truer approximation of the total duration of right ventricular electromechanical systole (QRV) is obtained. When this interval is plotted against the Q-A2 interval (fig. 8), the relative duration of the right and left ventricular systole can be compared. As can be seen in figure 8, a high correlation between Q-A2 and QRV interval exists (r = 0.99) approaching identity.

**Pulmonary Circulation Group 2**

Figures 9 through 12 demonstrate the two patterns of sound pressure correlates seen in group 2 patients with elevated pulmonary vascular resistance approaching systemic levels. In all nine patients, the absolute value of the rightsided hangout interval was markedly reduced (15 to 28

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**Figure 4**

External sound is recorded simultaneously with central aortic, pulmonary artery and right ventricular pressures in a 16-year-old male (D. Wil.) with a functional murmur and audible expiratory splitting of the second heart sound. Seventy-two msec splitting is present during inspiration. A2 is coincident with the incisura of the central aortic trace, while P2 is coincident with the incisura of the pulmonary artery curve. The pulmonary artery incisura is separated from the right ventricular pressure curve by a 76 msec hangout, an interval almost identical to the splitting interval.

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**Figure 5**

Sound pressure correlates of the second heart sound found in a 24-year-old female (S.R.) with a normotensive atrial septal defect (shunt = 2.3/1). Fixed splitting of the second heart sound is seen. P2 is coincident with the pulmonary artery incisura and is delayed from the right ventricular trace by 48 msec. The delayed pulmonic closure is due entirely to the hangout interval, indicating that the duration of right and left ventricular systole are nearly equal.

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**Figure 6**

Sound pressure relationships responsible for wide, relatively fixed splitting of the second heart sound found in a 41-year-old female (D.P.) with idiopathic dilatation of the pulmonary artery. Prominent pulmonary closure sounds with aftervibrations are recorded on the external phonocardiogram, being nearly coincident with the pulmonary artery incisuras. Note the wide separation of the incisura from the right ventricular pressure trace in all complexes, while splitting is wide and relatively fixed with less than 20 msec movement. A prominent late pulmonary ejection sound is also present.

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Wide physiologic splitting of the second heart sound is demonstrated in a 34-year-old male (C.G.) with mild valvular pulmonic stenosis and aneurysmal dilatation of the pulmonary artery. The major delay in P₂ is due to a wide hangout interval of 60 msec. Note also the loud valvular ejection sound coincident with the sharp anacrotic notch on the pulmonary artery tracing.

msec; table 1) as compared to group 1 patients. In those patients where the duration of right and left ventricular systole were nearly equal (figs. 9 and 10), narrow splitting of the second heart sound was present. In other patients where selective prolongation of right ventricular systole beyond left ventricular systole occurred (figs. 11 and 12), the splitting interval increased proportionately to the increased duration of right ventricular systole, resulting in normal to wide splitting of the second heart sound.

Discussion

It is well established that the aortic component of the second heart sound is coincident with the incisura of the central aortic pressure tracing.11, 12 Both of these phenomena are the results of an abrupt cessation of backward flow in the central aorta when the elastic limits of the valve leaflets are met.13 This sudden change in momentum of blood causes vibrations within the cardiohemic system, the lower frequency vibrations being recorded as a sharp aortic incisura while its higher frequency components are being recorded as A₂.14-16 The temporal relationship between the central aortic incisura and left ventricular pressure curve has been studied in this laboratory with equisensitive catheter-tip micromanometers in six patients during diagnostic left heart catheterization.17 Figure 2 is representative of this group. The purely descriptive term “hangout” was coined to define the interval between the aortic incisura and left ventricular pressure at the level of the incisura and was usually found to be less than 15 msec in patients having a normal systemic circulation. A similar mean value of this interval (15.8 msec) was also recently reported by Kumar and Luisada.18

In order to interpret the significance of this interval, an understanding of the dynamics of normal left ventricular ejection is necessary. During the course of ventricular ejection, once left ventricular contraction has ended and relaxation has begun, the abrupt deceleration and flow reversal of the column of blood being ejected into the aorta are brought about by those factors responsible for opposition to forward flow.19 In the systemic circulation, these are primarily the low distensibility and high resistance characteristics of the arterial tree. Careful examination of figure 2 shows close tracking of the left ventricular and aortic pressure during the protodiastolic phase* with a 10 msec

*An interval, as defined by Wiggers,20 between the end of left ventricular contraction and the aortic incisura. More recently, this has been termed the “diastolic mechanoacoustic interval” by Luisada and MacCanon.21
Sound pressure correlates responsible for narrow splitting of the second heart sound as demonstrated in a 25-year-old female (C.H.) with severe idiopathic pulmonary hypertension. On the external phonocardiogram, $A_2$ is soft and coincident with the incisura of the central aorta and is overshadowed by a booming $P_2$ with aftervibrations occurring 18 msec later, coincident with the pulmonary artery incisura. The incisura is separated from the right ventricular pressure trace by a hangout of only 20 msec. Note the similarity between the right ventricular and pulmonary artery contour and hangout interval as compared to the normal left-sided events (fig. 2). The pulmonary vascular resistance in this patient was 953 dyne-sec-cm$^{-5}$.

Sound and pressure relationships responsible for wide splitting of the second heart sound found in a 62-year-old female (V.J.) with severe pulmonary hypertension secondary to Lutembacher's syndrome. A 60 msec splitting interval is shown on the external phonocardiogram, while the pulmonary artery incisura is separated from the right ventricular pressure curve by only a 20 msec hangout interval. The duration of right ventricular systole goes well beyond the termination of left ventricular systole, as indicated by the central aortic incisura. The 60 msec splitting interval is the sum of the 40 msec prolongation of right ventricular systole plus the 20 msec hangout interval.

Simultaneous left ventricular, right ventricular and pulmonary artery pressure (equisensitive micromanometers) are recorded from a 35-year-old chronic drug abuser (O.S.) with severe pulmonary hypertension. Wide fixed splitting of the second heart sound of 58 msec is recorded on the external phonocardiogram and is associated with a narrow hangout interval of 20 msec. Significant prolongation of right ventricular systole beyond left ventricular systole is responsible for most of the delay in pulmonic closure.

interval separating the incisura at the end of this phase. It is felt that the absolute value of the hangout interval is a good reflection of the total opposition to flow or impedance of the arterial tree. Consistent with this thesis are the following observations:

*Figure 9*

*Figure 10*

*Figure 11*

*Figure 12*
1. A significant increase in this interval during the infusion of isoproterenol, which causes a marked reduction in the resistance as well as an increase in the capacitance of the systemic arterial tree.17

2. A decrease in this interval to almost zero during neosynephrine infusion which causes an increase in the resistance and a decrease in the capacitance of the arterial tree.17

3. A significant prolongation of this interval in patients with aortic stenosis where it is attributed to post-stenotic dilatation of the aorta, thereby increasing the capacitance of the arterial tree.18

Because of the small absolute value of the hangout interval in the normal systemic circulation, subtraction of it from the Q-A2 interval is not necessary for an accurate reflection of the total duration of electromechanical systole.

It has recently been shown that the dynamic events of right ventricular ejection are similar to those described for left ventricular ejection, although minor differences are present.22, 23 During left ventricular ejection, aortic flow velocity rises rapidly, peaks early in systole and is followed by rapid runoff. In contrast, flow patterns during right ventricular ejection show a gradual rise to a lower peak velocity in mid-systole and a more gradual reduction with the termination of right ventricular ejection following left ventricular ejection. The patterns of right and left ventricular pressure are very similar in wave form, but the absolute right ventricular pressure as well as its rate of pressure rise is much lower. This is consistent with the low hydraulic impedance to flow through the pulmonary circulation and is a reflection of the high capacitance and low resistance characteristic of the pulmonary vascular tree. It is therefore no surprise that the hangout interval between the pulmonary artery incisura and right ventricular pressure curve is much greater than the same left-sided event. This phenomenon, however, has only recently been recognized due to the failure of the catheter recording systems commonly used in right heart catheterization to display high fidelity pulse waves free of contour distortion and without delay in transmission.24

All 12 patients with normal pulmonary vascular resistance had hangout intervals during inspiration between 33 and 89 msec with no correlation between the width of this interval and the pulmonary vascular resistance or absolute pulmonary artery pressure. This suggests that the most important factor determining this interval is probably the capacitance of the pulmonary arterial tree.

In some cases, this increased capacitance was obvious and manifested by marked dilatation of the main pulmonary artery (D. P., G. C.), whereas in others, no such obvious factor was present (E. W., D. Wil.). However, awareness of the existence of this interval now allows us to interpret hitherto unexplained auscultatory findings of the second heart sound.

Audible expiratory splitting without demonstrable heart disease as seen in figure 4 can be explained by a wide hangout interval during both phases of respiration. In this instance, obvious dilatation of the central pulmonary artery was not present and a ready explanation for the postulated increased capacitance of the pulmonary circulation is not available. Differentiation of this benign form of audible expiratory splitting from pathologic conditions may often be made by having the patient assume the upright posture. Although the mechanism is not known, the majority of such patients will then close during expiration.25, 26

In all four patients studied with significant left to right shunting at the atrial level who were normotensive, hangout intervals were almost identical to the A2-P2 interval, indicating that the actual duration of right and left ventricular systole is nearly equal, confirming the recent report of Kumar and Luisada.27 All four patients had marked dilatation of the main pulmonary artery and a typical radiographic finding of large atrial septal defect. This longstanding high pulmonary flow leads to dilatation of the main pulmonary artery and perhaps an increase in the capacitance of the entire pulmonary vascular bed as well. It is suggested that the wide splitting observed in patients with normotensive atrial septal defects is primarily a reflection of changes in the pulmonary vascular tree rather than selective volume overload of the right ventricle prolonging right ventricular systole. Consistent with this thesis is the frequent observation of wide audible expiratory splitting which persists following surgical repair of the septal defect.4

In idiopathic dilatation of the pulmonary artery, a similar mechanism can be used to explain the wide, relatively fixed splitting so often observed. In this condition, a deficiency of the elastic tissue in the main pulmonary artery is postulated to be present and responsible for the dilatation of the proximal pulmonary artery.28 As can be seen in figure 6, an early systolic gradient is present and related to acceleration of the right ventricular stroke volume into the dilated main pulmonary artery and a
prominent delayed pulmonary ejection sound is produced by the sudden distention of this vessel.29 During the remainder of the ejection phase, the momentum of the right ventricular stroke volume maintains forward flow in spite of the rapidly falling right ventricular pressure and a persistent negative pressure gradient. In addition, the loss of normal elastic recoil in the dilated pulmonary artery prolongs deceleration and flow reversal during protodiastole, delaying pulmonic closure. Thus the delayed ejection sound is due to ejection of blood into a dilated loose system while the delayed pulmonic closure and wide hangout interval is a reflection of both dilatation of the pulmonary artery and loss of its elastic recoil. As demonstrated in figure 7, this same mechanism can also be used to explain the disproportionately wide splitting of the second heart sound found in mild to moderate pulmonic stenosis when associated with abnormally large post-stenotic dilatation of the pulmonary artery.5

An appreciation of the existence of a normal right-sided hangout interval is essential for a proper understanding of the changes which occur in the second heart sound when pulmonary hypertension develops. Figures 9 and 10 demonstrate the mechanism of narrow splitting of the second heart sound in severe pulmonary hypertension. In both tracings, there is marked narrowing of the hangout interval with close tracking of the pulmonary artery and right ventricular tracing during the latter third of systole. In addition, there is shortening of the protodiastolic phase with the incisura situated high upon the descending limb of the right ventricular pressure curve, a constant finding described by Jonsson in patients with high pulmonary resistance.30 These observations are consistent with the decrease in the capacitance of the pulmonary vascular bed as well as the marked increase in resistance known to occur in pulmonary hypertension. In both patients, the duration of right ventricular systole is only slightly more than that of left ventricular systole and very narrow splitting of the second heart sound occurs. Because of the increased amplitude and higher frequency components of P2 in pulmonary hypertension, narrow splitting of 20 msec can easily be appreciated on auscultation, whereas under normotensive conditions only a single sound can be appreciated at such a short interval. In an Eisenmenger’s ventricular septal defect, where the duration of both the right and left ventricular systole is equal, the second heart sound is single.31

In patients V. J. and O. S., wide splitting of the second heart sound is present, although the pulmonary vascular resistance is elevated to the same level as in C. H. and E. L. who have narrow splitting of the second heart sound. Examination of figures 11 and 12 shows narrow hangout intervals similar to those demonstrated in figures 9 and 10. However, in both patients V. J. and O. S. there is selective prolongation of true right ventricular systole well beyond the duration of left ventricular systole, thus producing wide splitting of the second heart sound. It is evident from these data that the duration of splitting in pulmonary hypertension may be difficult to predict from the absolute pulmonary pressure or the degree of increased resistance. However, as suggested by Shapiro, Clark and Goodwin, a wide split may indicate a more severely compromised ventricle than a narrow split. Similar observations have also been made by Perloff who states that wide persistent splitting becomes a useful sign of abnormal right ventricular performance in patients with primary pulmonary hypertension.5 It is also likely that the wide splitting of the second heart sound associated with acute massive pulmonary embolism is due to selective prolongation of right ventricular systole. Similar selective prolongation of left ventricular systole, producing paradoxical splitting of the second heart sound, has been demonstrated when acute systemic hypertension has been produced.34

Experimental studies in anesthetized dogs by Boyle and Little have been able to demonstrate similar sound pressure correlates during acute pulmonary hypertension. In this study, a changing spectrum of the A2-P2 interval was produced acutely, depending upon the relative effect of progressive pulmonary embolization on the pulmonary vascular bed, as well as its effect on the duration of right ventricular systole.

It can be concluded that the duration of splitting in pulmonary hypertension will be the net effect of two processes: 1) decreased capacitance and increased resistance of the pulmonary arterial tree tending to narrow splitting; 2) selective prolongation of right ventricular systole tending to increase the duration of splitting. It is also clear that varying degrees of splitting may be seen in the same patient during different stages of the disease process producing pulmonary hypertension. This explanation can thus reconcile many of the conflicting reports concerning the nature of splitting of the second heart sound when pulmonary hypertension is present, regardless of etiology.
INTRACARDIAC STUDY OF SECOND HEART SOUND

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JAMES A. SHAVER, RICHARD A. NADOLNY, JAMES D. O’TOOLE, MARK E. THOMPSON, P. S. REDDY, DONALD F. LEON and EDWARD I. CURTISS

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