The Ejection Click of Valvular Pulmonic Stenosis

By Herbert N. Hultgren, M.D., Richard Reeve, M.D.,
Keith Cohn, M.D., and Rima McLeod, B.A.

SUMMARY
Ten patients with valvular pulmonic stenosis were studied by simultaneous external phonocardiograms and intracardiac pressure recordings during successive respiratory cycles to examine the mechanism of the respiratory variation in the ejection click. Selective cineangiograms were performed in four. During inspiration right ventricular end-diastolic pressure (RVEDP) exceeded the pressure in the pulmonary artery (+2.7 mm) and no ejection click was recorded. During expiration RVEDP was lower than the pressure in the pulmonary artery (~3.6 mm) and a click was recorded. The click was present throughout the respiratory cycle when pulmonary artery diastolic pressure consistently exceeded RVEDP. Clicks were associated with sudden “doming” of the valve demonstrated by cineangiography. These data support the valvular origin of the ejection click in pulmonic stenosis and provide the following explanation for the respiratory variation: Inspiratory increase in venous return causes the valve leaflets to move to an open or “domed” position. Ventricular systole at this time produces no sound since there is no slack. With expiration pulmonary artery pressure exceeds RVEDP. Ventricular systole at this time produces an opening motion of the closed, slack leaflets. The click occurs when the opening motion is suddenly checked.

Additional Indexing Words:
Cineangiography Phonocardiogram Hemodynamic studies Intracardiac pressure

Over 60 years ago Petit1 described the occurrence of a loud, early systolic snapping sound at the pulmonic area in patients with valvular pulmonary stenosis. In 1957 Leatham and Weitzman2 described the essential features of the systolic ejection click in pulmonic stenosis and pointed out that it occurs only in valvular stenosis of mild or moderate severity and is absent when infundibular stenosis is present. Subsequently, several workers have described most of the characteristic features of the click.3-7 The pulmonary ejection click is now well recognized as a valuable diagnostic sign. The sound is of brief duration and closely follows the first heart sound. It is best heard at the pulmonic area. In a majority of patients it exhibits a characteristic variation with respiration appearing during expiration and disappearing during inspiration (fig. 1).

Three explanations have been offered for the occurrence of the ejection click. Leatham and Weitzman2 suggested that sudden distention of the dilated wall of the pulmonary artery produced the sound. Absence of the sound in severe pulmonic stenosis was thought to be due to a slower rate of pressure rise in the pulmonary artery. Nadas8 suggested that the sound was produced by the jet of blood hitting the wall of the pulmonary artery. The third explanation presented by McKusick9 suggested that abrupt doming of the stenotic valve diaphragm into the pulmonary artery...
produced the sound, but snapping of the dilated pulmonary artery beyond the stenosis was not excluded as a possible mechanism.

Opinions also differ regarding the cause of the respiratory variation of the sound. Most workers suggest that the decrease in loudness or disappearance of the sound during inspiration is due to attenuation of the sound by inflation of the chest. Crevasse and Logue, however, suggested that slack in the pulmonary artery during expiration favored the occurrence of the click, which they believed was produced by sudden tension of the arterial wall. Inspiration was associated with an increased tension of the arterial wall and therefore attenuation or absence of the click. More recently, Reeve suggested that the occurrence of the click depends upon the position of the valve leaflets at the time of ventricular systole. During inspiration, as the diastolic pressure in the pulmonary artery falls slightly below that in the right ventricle and venous return to the right heart increases, the valve leaflets move quietly to an open or domed position late in diastole. Ventricular systole occurring at this time will not produce an ejection sound because the valve leaflets are already tensed. During expiration, as diastolic pressure in the pulmonary artery pressure rises slightly above that in the right ventricle and venous return is decreased, the valve leaflets remain in a closed or slack position. Ventricular systole will then move the leaflets to an open position, and as this opening movement is suddenly checked by the fusion of the abnormal valve leaflets, the ejection sound occurs. Polis and associates have also suggested that the presence or absence of the ejection click may be determined by the position of the pulmonic valve leaflets at the onset of systole.

It is evident that the mechanism of the ejection click in pulmonic stenosis remains in dispute, and the cause of its respiratory variation will depend upon its mode of production.

It is the purpose of the present study to provide additional evidence in support of the valvular origin of the ejection click and to examine the mechanism of its variation with respiration.

Methods

The elastic properties of the proximal pulmonary artery, pulmonary annulus, and pulmonic valve were examined by laboratory experiments with fresh tissue from adult human hearts obtained at autopsy.

Length-tension relationships were determined in the following manner: Circumferential strips were cut from the pulmonary artery 3 cm distal to the annulus. The pulmonary annulus was removed and separated from the valve leaflets and adjacent pulmonary artery. The pulmonary valve was dissected free, and strips were cut by sharp scissors. Each strip of tissue was trimmed to a width of 3 mm and suspended by a tiny clamp. Weights were attached to the lower end of the strip, and the relationship between length and tension was plotted. The cross-sectional area of each strip of tissue was determined by measurement of the fresh tissue and histologic sections by means of a micrometer. Tension was expressed as grams per square millimeter. A similar technic has been previously described by Harris and associates.

Clinical and hemodynamic studies were performed in 10 patients to determine the pressure relationships across the pulmonic valve during the respiratory cycle. Each patient had been referred to the laboratory for diagnostic evaluation, and the diagnosis of valvular pulmonic stenosis had been confirmed by cardiac catheterization in all and selective angiography in five. Six patients had subsequent corrective surgery.

Hemodynamic data were obtained during diagnostic right heart catheterization in the following manner: Simultaneous pressures were recorded from the right ventricular outflow tract.
and the proximal pulmonary artery by means of a double-lumen catheter. Equisensitive Statham P23Cqb strain gauges were employed and all pressures were referred to the mid-chest. After recording pressures during several respiratory cycles, the strain gauges were interchanged to assure that identical tracings were obtained. A simultaneous phonocardiogram was obtained with the microphone placed at the pulmonic area on the chest. A reference electrocardiogram (lead II) and a pneumogram were recorded. Cardiac output was calculated by the Fick principle, and valve areas were estimated by the use of conventional hydraulic formulas. Clinical data regarding the patients studied are summarized in Table 1.

In five patients, selective cineangiograms were recorded during injection of contrast medium into the outflow tract of the right ventricle. The doming of the pulmonic valve, appearance of the jet in the pulmonary artery, and distention of the main pulmonary artery were observed.

The timing of each event was determined by frame-by-frame analysis of the films with a simultaneous recording of the electrocardiogram.

**Results**

Histologic examination of the normal human pulmonary artery and pulmonary valve reveals that the artery is composed of elastic tissue similar in structure to that of the aorta except for a lesser thickness. The elastic tissue extends to within 2 or 3 mm of the insertion of the valve leaflets where it is replaced by the dense collagenous tissue of the annulus. The valve leaflets are composed of dense collagen. The annulus contains a moderate amount of elastic tissue and the leaflets contain a lesser amount. In valvular pulmonary stenosis the increased thickness of the valve is due almost entirely to an increase in collagenous tissue with few elastic fibers, and in addition there is fusion of the leaflets to produce a domed structure that bulges into the pulmonary artery during ventricular systole. The length-tension relationships of the normal pulmonary artery and valve

![Length-tension relations of proximal human pulmonary artery and pulmonic valve.](chart.png)

**Table 1**

Clinical Data Regarding the Ten Patients Included in the Present Study

<table>
<thead>
<tr>
<th>No.</th>
<th>Initials</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Symptoms</th>
<th>Loudness of murmur</th>
<th>Inspiratory disappearance of click</th>
<th>A1-P2 interval (seconds)</th>
<th>Atrial sound (+ or 0)</th>
<th>ECG R in V1 (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>C.B.</td>
<td>13</td>
<td>F</td>
<td>++</td>
<td>5</td>
<td>+</td>
<td>0.10</td>
<td>+</td>
<td>10</td>
</tr>
<tr>
<td>2</td>
<td>M.M.</td>
<td>33</td>
<td>F</td>
<td>++</td>
<td>4</td>
<td>+</td>
<td>0.10</td>
<td>+</td>
<td>19</td>
</tr>
<tr>
<td>3</td>
<td>A.C.</td>
<td>41</td>
<td>F</td>
<td>++</td>
<td>4</td>
<td>+</td>
<td>0.08</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>R.B.</td>
<td>19</td>
<td>F</td>
<td>0</td>
<td>5</td>
<td>+</td>
<td>0.09</td>
<td>+</td>
<td>17</td>
</tr>
<tr>
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<td>A.S.</td>
<td>58</td>
<td>M</td>
<td>++</td>
<td>3</td>
<td>0†</td>
<td>0.06</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
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<td>30</td>
<td>M</td>
<td>+</td>
<td>4</td>
<td>+</td>
<td>0.10</td>
<td>0</td>
<td>6</td>
</tr>
<tr>
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<td>F</td>
<td>+</td>
<td>3</td>
<td>+</td>
<td>0.06</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>S.M.</td>
<td>16</td>
<td>F</td>
<td>+</td>
<td>4</td>
<td>+</td>
<td>0.085</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>D.W.</td>
<td>17</td>
<td>M</td>
<td>+</td>
<td>4</td>
<td>0†</td>
<td>0.08</td>
<td>0</td>
<td>19</td>
</tr>
<tr>
<td>10</td>
<td>P.H.</td>
<td>17</td>
<td>M</td>
<td>0</td>
<td>3</td>
<td>+</td>
<td>0.05</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>24.4</td>
<td></td>
<td>3.6</td>
<td></td>
<td></td>
<td>0.075</td>
<td></td>
<td>9.1</td>
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</tbody>
</table>

* This patient had polycythemia vera and systemic hypertension.
† Click present throughout respiratory cycle.
leaflets in human hearts are summarized in figure 2. The length-tension curve of the annulus is between the curve for the valve leaflets and the pulmonary artery and is not shown. It is clear that the distensibility of the wall of the pulmonary artery is much greater than the valve leaflets. When tension is applied the valve leaflets will quickly reach their limit of extensibility and then no further elongation occurs. About 10% increase in original length will occur before this limit is reached by the valve leaflets. These data were obtained from normal valves. Stenotic valve leaflets will clearly have a lesser degree of extensibility. The pulmonary artery wall, however, with the same force will elongate to about 45% of its original length before it will reach its limit of extensibility.

Phonocardiographic study revealed the presence of ejection clicks in all patients in the present series. Pertinent data are summarized in tables 1 and 2. Respiratory variation was present in eight patients. Careful study of the tracings revealed that the click disappeared

Table 2

<table>
<thead>
<tr>
<th>No.</th>
<th>Initials</th>
<th>Cardiac index (L/min/m²)</th>
<th>Pulmonic valve area (cm²)</th>
<th>RVs/PAm (mmHg)</th>
<th>Q-click M₁-click (sec)</th>
<th>End diastolic pressure RV/PA (mm Hg)</th>
<th>Gradient RV-PA (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>2.8</td>
<td>.28</td>
<td>130</td>
<td>.105</td>
<td>4.3</td>
<td>6.1</td>
</tr>
<tr>
<td>2</td>
<td>M.M.</td>
<td>2.5</td>
<td>.32</td>
<td>115</td>
<td>.13</td>
<td>5.2</td>
<td>7.3</td>
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<tr>
<td>3</td>
<td>A.C.</td>
<td>3.0</td>
<td>.43</td>
<td>112</td>
<td>.07</td>
<td>3.6</td>
<td>7.4</td>
</tr>
<tr>
<td>4</td>
<td>R.B.</td>
<td>3.5</td>
<td>.50</td>
<td>98</td>
<td>.12</td>
<td>7.5</td>
<td>6.75</td>
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<tr>
<td>5</td>
<td>A.S.</td>
<td>2.5</td>
<td>.60</td>
<td>60</td>
<td>.12</td>
<td>8.1</td>
<td>5.5</td>
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<tr>
<td>6</td>
<td>T.D.</td>
<td>2.8</td>
<td>.37</td>
<td>117</td>
<td>.11</td>
<td>9.5</td>
<td>7.5</td>
</tr>
<tr>
<td>7</td>
<td>M.R.</td>
<td>3.2</td>
<td>.74</td>
<td>41</td>
<td>.135</td>
<td>7.3</td>
<td>5.6</td>
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<tr>
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<td>S.M.</td>
<td>5.2</td>
<td>1.43</td>
<td>40</td>
<td>.11</td>
<td>2.7</td>
<td>2.6</td>
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<tr>
<td>9</td>
<td>D.W.</td>
<td>3.0</td>
<td>.63</td>
<td>63</td>
<td>.11</td>
<td>6.1</td>
<td>6.5</td>
</tr>
<tr>
<td>10</td>
<td>P.H.</td>
<td>3.5</td>
<td>.90</td>
<td>25</td>
<td>.125</td>
<td>6.5</td>
<td>6.0</td>
</tr>
<tr>
<td>Mean</td>
<td></td>
<td>3.2</td>
<td>.62</td>
<td>80.1</td>
<td>.114</td>
<td>6.2</td>
<td>6.1</td>
</tr>
</tbody>
</table>

Abbreviations: RVs = right ventricular systolic pressure; PAm-pulmonary artery mean pressure; Q-click = onset of QRS complex to onset of click; M₁-click = onset of first sound to onset of click; end-diastolic pressure = diastolic pressure prior to onset of systole; gradient RV – PA = gradient across pulmonic valve just prior to the onset of systole. A positive value indicates that the RV pressure is higher than the PA pressure.

Note: A.S. has been omitted from the calculation of mean values since this patient did not exhibit respiratory variation of the click.
The ejection click occurred 0.114 second after the QRS onset and followed the onset of the first sound by 0.060 second (mean values). In two patients (nos. 6 and 9), the click occurred close to the first heart sound and its presence could only be ascertained by an apparent variation in loudness of the first sound. The click occurred later in three patients (mean Q to click interval, 0.13 second).

In addition, a respiratory variation in the timing of the ejection click was observed in two patients (nos. 5 and 10). The Q to
ejection click interval varied from 0.12 second in expiration to 0.08 second in inspiration in one patient (no. 5), and from 0.125 to 0.10 second in the other patient (no. 10). Figure 4 illustrates the earlier occurrence of the click with inspiration (third cycle). In one patient a click occurred during diastole when right ventricular pressure abruptly exceeded pulmonary artery pressure during atrial systole and during early diastolic filling of the right ventricle (figs. 4 and 5). In figure 5 (top) a click occurs during atrial systole in the first two cardiac cycles. In the third cycle, click occurs during the rapid filling phase when the right ventricular pressure rises above the pulmonary artery pressure. Each click occurs at the pressure crossover point. Angiographic studies in the same patient demonstrated that the click occurring under these two hemodynamic circumstances accompanied checking of the doming motion of the valve leaflets toward the pulmonary artery as illustrated in figure 5 (bottom).

A comparison was made of the pressure in the right ventricle just prior to the onset of ventricular systole and the pressure in the proximal pulmonary artery at the same instant throughout several respiratory cycles. In eight of 10 patients the occurrence of the ejection click during expiration was associated with a pressure in the pulmonary artery that was higher than that in the right ventricle. This mean reversed gradient was 1.6 mm Hg. During inspiration the absence of the ejection click was associated with a pressure in the right ventricle that exceeded the pressure in the pulmonary artery. This "forward" gradient was 1.4 mm Hg. These relationships are graphically depicted in two patients in figure 6, and all data are summarized in table 2.

In one patient (no. 5) in whom the ejection click persisted throughout the respiratory cycle, pressure studies revealed that the pulmonary artery pressure prior to the onset of ventricular systole exceeded the simultaneous pressure in the right ventricle during both inspiration and expiration (6.2 mm Hg during inspiration, 4.0 mm Hg during expiration). During inspiration the ejection sound occurred slightly earlier (Q-click = 0.08 sec) than during expiration (Q-click = 0.12 sec). The pressure relationships in this patient throughout several respiratory cycles are summarized in figure 7.

Cineangiographic studies in four patients revealed that doming of the valve preceded the distention of the pulmonary artery by the jet of contrast agent by approximately 0.08 second (mean value).

Discussion

On the basis of the above studies, the following evidence can be presented to support the concept that the pulmonary ejection click in valuable pulmonic stenosis arises by sudden tension of the fused leaflets of the valve and not as the result of distention.
EJECTION CLICK OF VALVULAR PULMONIC STENOSIS

Figure 7
Relation between right ventricular pressure (above) and pulmonary artery pressure (below) prior to systole in a patient whose ejection click did not disappear during inspiration (patient no. 5).

or a jet impact involving the wall of the pulmonary artery.

1. Histologic and length-tension studies of the wall of the pulmonary artery indicate that it is a distensible, elastic structure that would not easily produce a sound when tensed. The valve leaflets, however, are less elastic and quickly reach their elastic limits with little distention. Such structures readily produce a sound when tensed. This has been demonstrated by Dock in an experimental preparation using excised tissue. Pulmonary valve leaflets are capable of producing loud sounds when moving from an open to a tensed position. This has been demonstrated by sounds produced by a cardiac catheter tip being pulled back in diastole across the closed pulmonic valve.

2. The rate of pressure rise beyond the stenotic valve is not rapid enough to produce a sound by distention of the wall of the pulmonary artery. Sounds in systemic arteries occur only if the rate of pressure rise exceeds 800 mm Hg/sec. Other studies have shown that similar sounds occur in veins when the pressure rise exceeds 200 mm Hg/sec. Such rates of pressure rise are not attained in the pulmonary artery even if the stenosis is mild. In four patients in the present study with mild pulmonic stenosis the mean rate of pressure rise was only 20 mm Hg/sec. The remaining patients exhibited even slower rates of pressure rise.

3. Cineradiographic observations in valvular pulmonic stenosis have demonstrated that the checking of the doming motion of the pulmonic valve occurs before the jet of blood strikes the wall of the pulmonary artery. Distention of the wall of the pulmonary artery occurs later. The click occurs at the time the doming motion of the pulmonic valve is checked and not when the jet strikes the wall or the pulmonary artery is being distended. Similar time relationships accompany the ejection click in valvular aortic stenosis.

4. A click may be present when doming of the pulmonic valve occurs with pressure pulses too small to cause any significant distention of the pulmonary artery such as during aortic systole and during the rapid filling phase of the right ventricle.

5. Inspection of the valve structure at surgery reveals a thin, mobile structure that can easily move toward either the ventricle (expiration) or the pulmonary artery (inspiration) with very small pressure differences.

Thus, the ejection sound in valvular pulmonic stenosis has a similar origin to the ejection sound in valvular aortic stenosis. In both situations the sound is faint or absent when the valve leaflets become rigid by marked fibrous thickening or calcification.

Studies of the pressure relationships across the pulmonic valve just prior to ventricular systole support the concepts previously presented by Polis and associates regarding the mechanism of the respiratory variation of the ejection click. During inspiration venous return to the right heart is...
increased and pulmonary artery pressure falls slightly. Right ventricular end-diastolic pressure and the pulmonic valve leaflets move to a domed or open position (fig. 8). If ventricular systole occurs at this time, no sound is produced since the valve leaflets are already tensed. During expiration venous return to the right heart is decreased and pulmonary artery pressure rises slightly. Pulmonary artery end-diastolic pressure exceeds right ventricular pressure, and the pulmonic valve leaflets move to a slack or closed position. If ventricular systole occurs at this time, a sound is produced as the valve opens quickly and is then suddenly checked. This mechanism clearly explains why the sound is usually absent during inspiration and not merely attenuated. Persistence of the ejection sound throughout the respiratory cycle in one patient was shown to be related to a pulmonary artery pressure that exceeded the right ventricular pressure in late diastole throughout the respiratory cycle. The pulmonic valve leaflets were therefore in a domed position at the onset of each systole and could not produce a sound. The presence, absence, or intensity of the ejection click therefore depends on whether the leaflets are slack or already tensed when the pressure pulse occurs. The same general principle applies to variation in loudness of the first sound\(^{24}\) and the absence of the first sound in aortic insufficiency when presystolic closure of the mitral valve may occur.\(^{25}\)

It is apparent from the data presented in this paper that the relation of the ejection click to the first sound (mitral or tricuspid components) may vary from patient to patient and during the respiratory cycle in a given patient, depending upon three factors: (1) right ventricular end-diastolic pressure, (2) pulmonary artery end-diastolic pressure, and (3) rate of initial systolic pressure rise in the right ventricle. Late clicks occur when (1) is considerably lower than (2), and when (3) is slow. Early clicks or clicks summatting with the first sound occur when the pressure

![Diagram](http://circ.ahajournals.org/)

**Figure 9**

Diagram to illustrate the effect of artery end-diastolic pressure and the rate of initial pressure rise in the right ventricle upon the timing of the ejection click. Two levels of pulmonary artery end-diastolic pressure (PA\(_{EDP}\)) are shown for severe pulmonic stenosis (a) and mild pulmonic stenosis (b). In severe pulmonic stenosis PA\(_{EDP}\) is low and the rate of pressure rise in the right ventricle is rapid, hence the click (C\(_1\)) occurs early. In mild pulmonic stenosis (b) PA\(_{EDP}\) is higher and the rate of pressure rise is slower, hence the click (C\(_2\)) occurs later.

![Graph](http://circ.ahajournals.org/)

**Figure 10**

Relation between right ventricular systolic pressure and initial rate of pressure rise in the right ventricle (\(\Delta p/T\)) in 29 patients with varying degrees of valvular pulmonic stenosis and intact ventricular septum.

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gradient between (1) and (2) is small and when (3) is rapid. The effect of these factors upon the timing of the ejection click is graphically illustrated in figure 9. With more severe degrees of pulmonic stenosis, the initial rate of pressure rise in the right ventricle becomes more rapid and thus the ejection click will occur closer to the first sound.\textsuperscript{5, 6} This relationship is illustrated in figure 10. In severe stenosis the click may merge with the first sound and then its presence can only be detected by its respiratory variation in loudness. A useful clue is the presence of a pulmonic component of the second sound. If this is evident by auscultation or phonocardiography, an ejection click is probably present.\textsuperscript{3} A similar relation exists between the ejection click and aortic valve closure sound in aortic stenosis.\textsuperscript{23}

Presystolic sounds in valvular pulmonic stenosis have been observed by several workers,\textsuperscript{3, 26} and an “auricular systolic” murmur of low intensity has also been described in patients who have a prominent a wave in the jugular pulse.\textsuperscript{2} The observations made in one patient (figs. 4 and 5) indicate that in some patients presystolic sounds may arise when the pulmonic valve is abruptly domed into the pulmonary artery by atrial systole. Under these circumstances the forward pressure gradient across the valve may be accompanied by sufficient flow to produce an “auricular systolic” murmur.

Acknowledgment

Mr. John Harding, Stanford Medical School, Class of 1972, performed the length-tension studies.

References

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100 Years Ago

Clear Concepts of Pulmonary Hypertension
Secondary to Disease of the Left
Side of the Heart

Assuming that the second sound of the heart is chiefly due to closure of the semilunar valves, when arresting the backward flow of the blood-column on the arterial recoil, it is manifest that variations in the amount of pressure, increase of arterial elasticity, or want of it, the amount of obstruction to the flow forward, will entail changes in the sound produced. . . . Skoda has shown how mitral lesions, either regurgitant or obstructive, lead to pulmonary congestion and altered blood vessels: the pulmonary artery becomes dilated and thickened, and recoils with increased energy, and, by increasing the pressure on the pulmonary semilunar valves, increases and intensifies the sound. . . . Accentuation of the second sound may be only temporary, which cannot be the case with mitral lesions, whether obstructive or regurgitant. (Temporary mitral regurgitation may arise from irregular action of the papillary muscles; but of this we do not yet know enough to hazard assertions.) . . .

In valvular disease of the left side of the heart, accentuation of the pulmonary second sound is an index that mischief is going on behind the lesion, and that destructive changes highly inimical to life are looming. Finally, accentuation of the second sound is usually found related to the pulmonary semilunar valves, and, when so related, is an index that the balance between the pulmonic and systemic circulations is disturbed.—From J. Milner Fothergill: The diagnostic value of accentuation of the second sound of the heart. Lancet 2: 633, 1869.
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