Cineradiographic Studies of the Early Systolic Click in Aortic Valve Stenosis

By Ellis J. Epstein, M.D., J. Michael Criley, M.D., Edward B. Raftery, M.D., J. O'Neal Humphries, M.D., and Richard S. Ross, M.D.

Midystolic clicks were first described in 1887 by Cuffer and Barbil- lon\(^1\) and also by Potain\(^2\) and Gallavardin.\(^3\) In 1902 Petit\(^4\) described an early systolic click in pulmonary stenosis and Lian and Welti\(^5\) later demonstrated this finding by means of phonocardiograms. They also noted a similar early systolic click in aortic stenosis. Later publications have confirmed the presence of an early systolic sound in many patients with aortic valve stenosis although opinion is still divided regarding its origin. Leatham and Vogelpoel\(^6\) and Reinhold et al.\(^7\) believe that it arises in the aorta, whereas Woltferth and Margolies,\(^8\) McKusick,\(^9\) and Minhas and Gasul\(^10\) consider that it originates in the aortic valve. Further recent studies have favored the valvular hypothesis.\(^11, 12\)

We have recorded heart sounds simultaneously with hemodynamic and cineradiographic studies in a series of patients with aortic valvular stenosis in order to relate the early systolic sound to pressure changes and to movements of the aortic valve. The purpose of this paper is to present further evidence for the valvular origin of the early systolic click in aortic stenosis.

Patients with valvular aortic stenosis can be classified by cineradiography on the basis of the mobility of the valve.\(^13, 14\) There appears to be a continuous gradation from the thin and freely mobile valve to that which is thickened, rigid, and often calcified. The study of a series of cases suggests that these are progressive stages in the natural history of the disease. Commisural fusion represents the primary pathologic process, which may be either rheumatic or congenital. Fusion of the leaflets results in a mobile perforated dome that obstructs left ventricular ejection. With the passage of time and probably as a consequence of the hemodynamic disturbance, the valve tissue progressively thickens and the dome becomes less mobile. Eventually, calcium is deposited, and a rigid diaphragm separates the left ventricle and aorta.

Characteristics of Systolic Clicks

The early systolic sound in aortic stenosis has a high-pitched clicking quality because it is composed of relatively high frequency vibrations usually of very brief duration (less than 10 msec.), although they may occasionally be prolonged to more than 20 msec. It is best demonstrated on high-frequency or logarithmic oscillographic records (fig. 1) or by the spectral phonocardiogram.\(^9\) The click is well heard over the entire precordium, although usually of maximal intensity at the apex, and it is relatively unaffected by respiration. In contrast, pulmonary systolic clicks are usually of maximal intensity at the second and third left interspaces, are best heard in expiration, and may disappear on inspiration.\(^6, 10\) On auscultation, the click gives the impression of splitting of the first heart sound, but when the second component is a systolic click, it is usually louder, sharper, and more widely separated from the first component.

Methods

In a typical study, a radiopaque polyethylene catheter was introduced percutaneously into the
Figure 1

Patient 3 (B.K.) from group 1. Left ventricular (E) and central aortic (D) pressure tracings with simultaneous phonocardiograms. Paper speed 75 mm. per sec. with 40-msec. time lines. The upper sound tracing (B) was recorded from the aortic catheter and the lower one (C) from the chest wall at the aortic area by an external microphone. There are an early systolic click (X) and midsystolic and a prominent aortic second sound. The systolic click is of greater relative intensity on the intraaortic record. The first heart sound is indicated by (1).

femoral artery and advanced into the ascending aorta. A second catheter is passed through the femoral vein to the right heart and over a transseptal needle into the left atrium and left ventricle. The catheterization is monitored by means of an 8-inch image amplifier and closed-circuit television. With catheters in position above and below the aortic valve, phonocardiograms are recorded externally from the chest wall and also from the catheters by means of an external electromanometer.* In general, the external phonocardiograms obtained just before the cineangiograms have been correlated with the motion pictures by means of the electrocardiogram. The R wave of the electrocardiogram initiates a signal which marks the recording paper and motion-picture film simultaneously, so that each frame of the motion picture can be related to a specific point on the pressure and sound record (fig. 2). To demonstrate the aortic valve, the patient is placed in the left anterior oblique position, contrast material is injected through the aortic catheter, and motion pictures are taken at precisely 60 frames per second on 35-mm. film by a camera synchronized with the x-ray pulses. In some cases, a second injection of contrast material is made into the left ventricle through the transseptal catheter, and occasionally supravalvular injection is made in the right anterior oblique projection.

The sound and pressure tracings are all recorded in partial expiration at a paper speed of 75 mm. per second by means of multi-channel recorder.† There is a transmission delay in the catheter-manometer system of 10 msec., and this has been allowed for in timing the pressure events. A correction has also been made for the delay between the R wave of the electrocardiogram.

*Electronics for Medicine, Model DR-8.

Figure 2

Patient 1. (S.R.) from group 1. Simultaneous left ventricular and central aortic pressure pulses with an intraaortic phonocardiogram. Paper speed 200 mm. per second. There is a systolic click (X) followed by a mid-systolic (S.M.) and a prominent aortic second (A2). Below the tracing there is a 60 cycles per second time signal, each cycle corresponding to one frame of the motion picture. The R-wave of the E.C.G. initiates a square wave signal which depresses the base line and also activates the flag marking the cine film. The latter occurs at the point marked by the arrow with an electro-mechanical delay of 25 milliseconds.

*Dallons Telco, 5066 Santa Monica Boulevard, Los Angeles, California.

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Normal aortic valve: Retrograde aortogram left anterior oblique projection (L.A.O.) These four cine frames demonstrate normal aortic valve opening. In A (upper left) and B (upper right) three sinuses of Valsalva are shown in diastole. (1. right coronary sinus, 2. left coronary sinus, 3. noncoronary sinus.) As systole begins, the valve leaflets open rapidly (C, lower left; D, lower right). The left coronary leaflet is seen in true profile and appears to open wide in this projection. The marker flag can be seen in the upper right hand corner of each photograph. The flag is extended in frame B, thus establishing the temporal relationship between this frame of the motion picture and the other physiologic events.

All the measurements were made independently by two observers and are based on the mean of at least six cardiac cycles. If the findings disagreed, measurements were repeated and a joint decision was reached.

Material

From over 100 patients with aortic valve disease investigated in this laboratory, three groups of patients were chosen for this study.

The first group consists of 10 patients with aortic valvular stenosis who had mobile dome-shaped valves. The second contains 11 patients with dominant aortic valvular stenosis who showed impaired valve mobility and varying degrees of calcification. The third group consists of 11 patients in whom the aortic valve had been replaced by a Bahnson Teflon prosthesis consisting of three individual valve leaflets. All patients in the third group had phonocardiograms after operation and four also had a detailed postoperative cineradiographic study.
Table 1

**Group 1: Aortic Stenosis—Mobile Valves**

<table>
<thead>
<tr>
<th>Case</th>
<th>R wave to LV/aortic cross-over pointa</th>
<th>R wave to onset of valve opening</th>
<th>R wave to completion of valve opening</th>
<th>R wave to early systolic click</th>
<th>Time for aortic valve to open</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. S.R.</td>
<td>40</td>
<td>40</td>
<td>78</td>
<td>82</td>
<td>39</td>
</tr>
<tr>
<td>2. A.R.</td>
<td>33</td>
<td>37</td>
<td>87</td>
<td>85</td>
<td>50</td>
</tr>
<tr>
<td>3. B.K.</td>
<td>58</td>
<td>51</td>
<td>73</td>
<td>70</td>
<td>22</td>
</tr>
<tr>
<td>4. J.L.</td>
<td>35</td>
<td>35</td>
<td>80</td>
<td>80</td>
<td>45</td>
</tr>
<tr>
<td>5. W.S.</td>
<td>30</td>
<td>20</td>
<td>76</td>
<td>73</td>
<td>56</td>
</tr>
<tr>
<td>6. C.H.</td>
<td>60</td>
<td>53</td>
<td>87</td>
<td>70</td>
<td>34</td>
</tr>
<tr>
<td>7. R.G.</td>
<td>30</td>
<td>24</td>
<td>67</td>
<td>70</td>
<td>43</td>
</tr>
<tr>
<td>8. D.K.</td>
<td>67</td>
<td>77</td>
<td>110</td>
<td>109</td>
<td>33</td>
</tr>
<tr>
<td>9. G.W.</td>
<td>43</td>
<td>41</td>
<td>69</td>
<td>67</td>
<td>28</td>
</tr>
<tr>
<td>10. M.P.</td>
<td>30</td>
<td>45</td>
<td>90</td>
<td>65</td>
<td>45</td>
</tr>
</tbody>
</table>

Mean values: 42 42 82 77 40

*a A recording system delay of 10 msec. has been subtracted. All times given in milliseconds.

The Normal Aortic Valve

The normal aortic valve consists of three pliable leaflets which are demonstrated in closure in the left anterior oblique projection in figure 3A. In this projection, the line of attachment of the left coronary cusp lies parallel to the x-ray beam, and hence only this cusp is seen in profile. When the valve opens, the left coronary leaflet (fig. 3C) moves at right angles to the x-ray beam, and hence it appears to move more than the other two. The line of attachment of the noncoronary cusp is perpendicular to the central beam, and thus its motion is parallel to the beam and cannot be appreciated at all in this projection, while the right coronary cusp (fig. 3D) moves on a line 60° away from the central beam, and can be seen to move, but its excursion in this projection is less than that of the left. Opening of the normal aortic valve is probably inaudible, and its closure produces the first component of the second heart sound.

In five patients with normal aortic valves the average time from onset to completion of valve opening was 30 msec. (two frames of the cine-film).

Results

**Group 1. Aortic Stenosis—Mobile Valves**

The main findings in this group of 10 patients are summarized in tables 1 and 2. These patients all had mobile dome-shaped valves without calcification, and history indicates that most, if not all, had congenital aortic stenosis. A systolic jet was present in all, and six patients had hemodynamically important obstruction to left ventricular ejection as manifested by a peak systolic pressure gradient across the aortic valve of at least 70 mm. of mercury. On cineangiography these valves are seen to move upwards like a piston to form a dome-shaped structure. Ejection begins only after the dome has been formed (fig. 4).

Phonocardiography showed that all 10 patients had a loud early systolic click, a mid-systolic murmur and an aortic second sound of at least normal intensity (table 2). The average time from the peak of the R wave to the cross-over point of the left ventricular and aortic pressure pulses was 42 msec. (range 30 to 67 msec). The time from the R wave to the onset of opening of the aortic valve was 42 msec. (range 20 to 77 msec.) and to the completion of the valve opening was 82 msec. (range 67 to 110 msec.). The R peak to systolic click interval was 77 msec. (range 65 to 109 msec.).

Thus the onset of the aortic upstroke, as shown by the pressure pulses, coincided with the start of the valve opening. The systolic click followed 35 msec. later and preceded completion of valve opening by an average of 5 msec. This difference of 5 msec. is within...
the error of the cineradiographic method and is not significant.

**Group 2. Aortic Stenosis—Impaired Valve Mobility**

The findings in this group of 11 patients are shown in tables 3 and 4. These patients had valves characterized by impaired mobility, thickening, and calcification of variable degree. The thickness of the aortic valve could often be appreciated when contrast material was present in both the aorta and the left ventricle, as the non-radiopaque interface between them is due to the valve (fig. 5). Some degree of aortic regurgitation was present in all patients in this group. A systolic jet was seen in eight of the 11 patients, and peak systolic gradients ranged from 75 to 130 mm Hg.

Phonocardiograms showed the typical mid-systolic murmur of aortic stenosis. Very small, early systolic clicks were seen in four patients, and in seven patients there was no systolic click. The aortic second sound was normal in only one patient who had moderate calcification and only moderate restriction of movement. In the 10 remaining patients, the aortic second sound was small in six patients, barely visible in two patients, and absent in two patients (table 4). None of the six patients with rigid valves had a systolic click, and four of these six had very small or absent aortic second sounds.

The average time from the peak of the R wave to the cross-over point of the left ventricular and aortic pressure pulses was 37 msec. (range 12 to 55 msec.). The time from the R wave to the onset of aortic valve opening was 35 msec. (range 7 to 56 msec.) and to the completion of valve opening was 68 msec. (range 45 to 93 msec.). In the four patients with systolic clicks, the time from the R peak to completion of valve opening was 73 msec., and the R peak to systolic click interval was 70 msec. (range 54 to 85 msec.). These findings are similar to those in patients with mobile valves. The onset of the aortic upstroke coincided with the beginning of the valve opening and the systolic click, when present, followed 33 msec. later and preceded

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**Table 2**

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; sex</th>
<th>Aortic Stenosis—Mobile Valves</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. S.R.</td>
<td>23 F</td>
<td>Age &amp; sex: 23 F</td>
</tr>
<tr>
<td>2. A.R.</td>
<td>10 F</td>
<td>Age &amp; sex: 10 F</td>
</tr>
<tr>
<td>3. B.K.</td>
<td>17 F</td>
<td>Age &amp; sex: 17 F</td>
</tr>
<tr>
<td>4. J.L.</td>
<td>28 M</td>
<td>Age &amp; sex: 28 M</td>
</tr>
<tr>
<td>5. W.S.</td>
<td>16 F</td>
<td>Age &amp; sex: 16 F</td>
</tr>
<tr>
<td>6. C.H.</td>
<td>12 F</td>
<td>Age &amp; sex: 12 F</td>
</tr>
<tr>
<td>7. B.G.</td>
<td>7 M</td>
<td>Age &amp; sex: 7 M</td>
</tr>
<tr>
<td>8. D.K.</td>
<td>13 F</td>
<td>Age &amp; sex: 13 F</td>
</tr>
<tr>
<td>9. G.W.</td>
<td>8 M</td>
<td>Age &amp; sex: 8 M</td>
</tr>
<tr>
<td>10. M.P.</td>
<td>22 M</td>
<td>Age &amp; sex: 22 M</td>
</tr>
</tbody>
</table>

+ Present; - Absent.
completion of valve opening by 3 msec. This difference of 3 msec. is within the error of the cineradiographic method.

**Group 3. Prosthetic Aortic Valves**

The phonocardiographic findings in the 11 patients in this group are shown in Table 5, together with the cineradiographic data on the four patients studied by cardiac catheterization. Patients 1 and 2, studied about 12 weeks after operation, are representative of those with successful valve replacements and were found to have a small systolic gradient and only minimal aortic regurgitation. The other two postoperative cineradiographic studies were performed because signs of aortic regurgitation had developed (cases 3 and 4). These four patients all showed mobile leaflets without any visible systolic jet. An early systolic click and a loud aortic second
### Table 4

#### Group II: Aortic Stenosis—Impaired Valve Mobility

<table>
<thead>
<tr>
<th>Case</th>
<th>Age &amp; Sex</th>
<th>Peak Systolic Gradient (mm Hg)</th>
<th>Phonocardiography</th>
<th>Cineraadiography</th>
<th>Degree of Aortic Valve Mobility</th>
<th>Calcification (0-3)</th>
<th>Aortic Regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. D.B.</td>
<td>43 M</td>
<td>75</td>
<td>+ (Soft)</td>
<td>Normal</td>
<td>Moderate</td>
<td>+</td>
<td>1</td>
</tr>
<tr>
<td>2. L.K.</td>
<td>42 F</td>
<td>95</td>
<td>+ (Soft)</td>
<td>Soft</td>
<td>Moderate</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td>3. R.C.</td>
<td>50 M</td>
<td>130</td>
<td>+ (Soft)</td>
<td>Soft</td>
<td>Moderate</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td>4. J.K.</td>
<td>30 M</td>
<td>70</td>
<td>—</td>
<td>Soft</td>
<td>Poor</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td>5. I.H.</td>
<td>48 M</td>
<td>108</td>
<td>—</td>
<td>Very faint</td>
<td>Rigid (T)</td>
<td>+</td>
<td>2</td>
</tr>
<tr>
<td>6. J.R.</td>
<td>45 M</td>
<td>112</td>
<td>—</td>
<td>Very faint</td>
<td>Rigid (T)</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>7. S.H.</td>
<td>50 M</td>
<td>85</td>
<td>—</td>
<td>Soft</td>
<td>Rigid (T)</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>8. M.L.</td>
<td>45 M</td>
<td>80</td>
<td>—</td>
<td>Soft</td>
<td>Rigid (T)</td>
<td>—</td>
<td>3</td>
</tr>
<tr>
<td>9. F.R.</td>
<td>50 M</td>
<td>105</td>
<td>—</td>
<td>Absent</td>
<td>Rigid (T)</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td>10. R.W.</td>
<td>46 M</td>
<td>115</td>
<td>—</td>
<td>Absent</td>
<td>Rigid (T)</td>
<td>+</td>
<td>3</td>
</tr>
<tr>
<td>11. R.F.</td>
<td>40 M</td>
<td>100</td>
<td>—</td>
<td>Absent</td>
<td>Rigid (T)</td>
<td>+</td>
<td>3</td>
</tr>
</tbody>
</table>

+, Present; —, absent; (T), trace of movement, only.

The early systolic click has been recognized as a characteristic feature of congenital aortic stenosis by many authors, but there is disagreement as to its causation. The opening of the aortic valve coincides with the systolic click. Delay in the opening of the aortic valve is also noted, which supports the cause of the systolic click.

### Discussion

In group II, aortic valve opening followed the completion of valve opening by an average of 6 msec. This difference of 6 msec is within the range of 5 to 15 msec. The R wave to systolic click interval was 105 msec. The R wave to the onset of aortic valve opening was 77 msec (range 70 to 90 msec) and to the crossover point of the R wave was 86 msec. The systolic pressure in the aorta was 115 mm Hg.

In the four patients with complete obstruction, the average time from the onset of the R wave to the completion of aortic valve opening was 104 msec. The systolic pressure in the aorta was 110 mm Hg. The systolic pressure in the aorta was 115 mm Hg.
EARLY SYSTOLIC CLICK

Patient 6 (J.R.) from group II: Transseptal left ventriculogram, left anterior oblique projection. A thickened and immobile valve is shown in systole (left) and diastole (right). The dome-shaped appearance is present during all phases of the cardiac cycle. The catheter can be seen entering the left ventricle through the opened mitral valve (dark filling defect) during diastole (right).

Table 5
Group III: Prosthetic Aortic Valves

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Peak systolic gradient (mm. Hg)</th>
<th>R wave to LV/aortic cross-over point*</th>
<th>R wave to onset of valve opening</th>
<th>R wave to completion of valve opening</th>
<th>R wave to systolic click</th>
<th>Time for aortic valve to open</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. F.R.</td>
<td>50</td>
<td>20</td>
<td>70</td>
<td>73</td>
<td>115</td>
<td>105</td>
<td>42</td>
</tr>
<tr>
<td>2. R.C.</td>
<td>50</td>
<td>20</td>
<td>65</td>
<td>90</td>
<td>115</td>
<td>100</td>
<td>25</td>
</tr>
<tr>
<td>3. M.T.</td>
<td>53</td>
<td>5</td>
<td>55</td>
<td>70</td>
<td>87</td>
<td>85</td>
<td>17</td>
</tr>
<tr>
<td>4. J.H.†</td>
<td>21</td>
<td>0</td>
<td>73</td>
<td>75</td>
<td>100</td>
<td>80-100</td>
<td>25</td>
</tr>
<tr>
<td>5. J.B.</td>
<td>55</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. T.A.</td>
<td>28</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. J.R.</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. W.P.</td>
<td>53</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. A.B.</td>
<td>22</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. M.L.</td>
<td>45</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. C.P.</td>
<td>48</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Mean values 66 77 104 98 27

* A recording system delay of 10 msec. has been subtracted. All times given in milliseconds.
† (This patient prolapsed one of the prosthetic cusps and developed severe aortic regurgitation. Two systolic clicks were seen on the phonocardiogram at the times shown in column 7.)

All in this group are males.

Aorta. At the cross-over point of these pressures the aortic valve starts to open. The early systolic click occurs 30 to 40 msec. later and coincides with the completion of valve opening and the beginning of the systolic jet. The visible jet, which may follow the initial impact wave, can be seen to strike the aortic wall about 16 msec. later (1 frame of the motion picture) and systolic expansion of the aorta takes place after a further 16 msec. Valve closure begins at the end of systole at the cross-over point of the left ventricular and aortic pressures. It is completed within 33 msec. (2 frames of the motion picture) and this coincides with the aortic second sound and the dicrotic notch of the central aortic pressure pulse.

Patients with Teflon prosthetic valves...
Table 6

Summary of Data on Three Groups of Patients

<table>
<thead>
<tr>
<th>Group</th>
<th>R wave to LV/aortic crossover</th>
<th>R wave to onset of aortic valve opening</th>
<th>R wave to completion of aortic valve opening</th>
<th>R wave to systolic click</th>
<th>Time for aortic valve to open</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>42</td>
<td>42</td>
<td>82</td>
<td>77</td>
<td>40</td>
</tr>
<tr>
<td>Group II</td>
<td>37</td>
<td>35</td>
<td>68</td>
<td>70</td>
<td>33</td>
</tr>
<tr>
<td>Group III</td>
<td>66</td>
<td>77</td>
<td>104</td>
<td>98</td>
<td>27</td>
</tr>
</tbody>
</table>

All times are given in milliseconds. The values are the average for each group.

Patient 3 (M.T.) from group III: Postoperative study, prosthetic valve. Left ventricular (D) and central aortic (C) pressure pulses with simultaneous phonocardiograms recorded from the aortic area (B). Paper speed 75 mm. per sec. and 40 msec. time lines. There are an early systolic click (X) and a clear aortic second sound. An early systolic gradient can be seen between the left ventricle and the aorta and a correspondingly early systolic murmur.

(group III) have prominent systolic clicks, insignificant pressure gradients, and no visible jet issuing from the valve to strike the aortic wall. Prosthetic valves are thicker and less mobile than normal valves, they appear to have a certain resistance to opening, and a critical pressure gradient must be developed before the valve leaflets snap up and allow ejection to take place (fig. 6). There is a delay of about 11 msec. between the cross-over point of the left ventricular and aortic pressures and the onset of prosthetic valve opening. The systolic click coincides with the completion of valve opening, both events being delayed (table 6). The necessity for an opening pressure gradient may explain the later occurrence of the systolic click in these patients.

Mobility of the aortic valve on cineradiography appeared to be of importance in determining whether a systolic click was present. A prominent early systolic click was present in all patients with a fully mobile valve (table 2), whereas only four of the 11 patients with impaired valve mobility had recordable systolic clicks which were of low intensity (table 4). In six of the seven patients without a systolic click there was only a trace of valve movement or the valve was completely rigid. These valves were the most heavily calcified. Thus as valve mobility becomes impaired, usually in association with calcification, the systolic click becomes less intense, and it is not present when the valve is completely rigid. This may explain previous observations which have shown an inverse relationship between valve calcification and the systolic click.\textsuperscript{11, 12} There was no relationship between the presence and intensity of the early systolic click and the severity of the aortic stenosis as measured by pressure gradient.

A normal or accentuated aortic second sound was present in all the group-I patients with fully mobile aortic valves (table 2), but when valve mobility was impaired the aortic second sound was of very low intensity or absent (table 4). A normal aortic second sound was recorded in only one patient from group II, who still had moderate valve mobility (table 4, case 1). The intensity of the
aortic second sound was not related to the severity of the aortic stenosis. In general, a prominent systolic click was associated with a normal or accentuated aortic sound, and when the systolic click was small or absent the aortic second sound was usually diminished or absent. A loud aortic second sound was usually associated with the prominent systolic click in group-I patients with prosthetic valves.

A jet was present on aortography in all 10 patients with a dome-shaped mobile valve and in eight of the 11 patients with impaired valve mobility (tables 2 and 4). The onset of the systolic jet coincided with the completion of the upward valve excursion and the jet struck the aortic wall 16 msec. later (1 motion-picture frame). No jet was seen in four patients with prosthetic valves, studied by cineradiography, although all had prominent systolic clicks. There was no consistent association between a visible systolic jet and the presence of a systolic click. These observations and the delay before the jet reaches the wall of the aorta are against the theory that impact of the jet is responsible for the systolic click. However, these findings must be accepted with reservation, since the visibility of the jet depends on several factors, including the x-ray exposure and the dose of contrast medium. Moreover, the radiologic jet impact may not represent the moment of energy transfer between the flowing blood and the aortic wall.

The delay between the upstroke of the carotid pulse and the systolic click has been used as evidence for the production of the click by the ejection of blood into the aorta. However, it may be incorrect to equate the upstroke of the carotid pulse with the onset of ejection in patients with dome-shaped mobile aortic valves. When ventricular pressure exceeds aortic pressure the valve moves upwards like a piston and displaces aortic blood before any blood can be seen to issue from the valve orifice. This piston-like ascent may begin as long as 50 msec. before the valve becomes a tense dome (table 1), and therefore it is probable that the upstroke of the aortic or carotid pulse precedes flow through the valve. In group-I patients with mobile valves, the gradient necessary to produce flow through the stenosed valve exceeds that needed to produce systolic doming of the valve, since ejection begins only after the dome has formed.

Maximal expansion of the aorta was timed in several patients and on average found to occur 33 msec. (2 motion-picture frames) after the systolic click and the completion of valve opening. Therefore, completion of rapid expansion of the aorta could not be responsible for the click.

Correlation of sound and aortic valve movements shows that a systolic click is only found if the valve is mobile and that the click occurs as valve opening is completed. Although the movement of the valve and the sound are associated, the mechanism producing the sound has not been determined. It is possible to explain the click as due to tensing of the dome as it snaps upwards in early systole. Since the systolic click occurs at the end of the opening movement of the valve, the sudden arrest of its upward excursion may be responsible for producing sound vibrations. Similarly, the aortic second sound is produced as the valve snaps back toward the ventricle at the end of systole. Dock has shown experimentally that it is possible to produce sounds in valve leaflets by making them tense. Stiff valves gave off louder sounds for a given degree of tension. Despite its mobility, the normal aortic valve does not produce an audible systolic click. In contrast the Teflon prosthetic valve is associated with a loud systolic click. However, these valves are stiffer and more resistant to opening than normal valves, despite the absence of stenosis, and this qualitative structural difference may be related to the production of this sound.

McKusick has suggested that transient sounds may be produced by the sudden interruption of the momentum of local flow. This has also been the view of Rushmer, of Shah et al., and of Piemme and Dexter, who suggested that the vibrations due to abrupt acceleration and deceleration of flow could produce transient sounds. The terminal vibra-
tions of the first heart sound, which are inaudible, have been attributed to ejection of blood into the aorta since they occur after the onset of the aortic pressure pulse. Hancock has suggested that increase of their intensity in abnormal conditions can make them audible and produce a systolic click.

With the onset of ventricular systole, the blood in the ventricle and aorta is accelerated upwards, and a mobile aortic valve moves forward freely with the accelerating column. If the valve is normal, it opens, and ejection proceeds smoothly. If the valve is stenosed, ejection is interrupted by sudden tensing of the valve which cannot swing back to the aortic wall because of the commissural fusion. The column of blood is suddenly checked as the valve becomes tense, and this interruption in ejection may be responsible for the systolic click of aortic stenosis. Tensing of the leaflets and change in velocity of the blood occur simultaneously, and therefore it is impossible to attribute the click to one rather than to the other. The systolic clicks of prosthetic valves might have a similar explanation in that these valves because of their unnatural stiffness do not open completely. The motion of the valves toward the aortic wall stops suddenly due to the stiffness of the material and also to the sutures placed at the commissures. Thus in this situation there is also sudden interruption of the ejection process. When the valve is immobile these blood velocity changes in the ventricle and aortic root, preceding ejection, do not take place and there is no systolic click.

An analogy can be drawn between the early systolic click in aortic stenosis and the “opening snap” of the mitral valve in mitral stenosis. In both disorders, impairment of valve mobility is associated with diminution or absence of the appropriate sound. Thus the early systolic click may be regarded as the “opening snap” of the aortic valve.

Systolic clicks of similar sound frequency are found in many other disorders, such as systemic hypertension, pulmonary arterial hypertension, and pulmonary valve stenosis. It is possible that these clicks are produced by a different mechanism to that proposed for aortic valvular stenosis.

In a patient with aortic valve stenosis, a systolic click implies that the valve is still mobile. If there is no systolic click then valve mobility is poor or absent. The presence of a systolic click is of no help in assessing the severity of the obstruction.

Summary

An early systolic click and a normal or accentuated aortic second sound have been found in cases of aortic valve stenosis characterized cineradiographically by a mobile, dome-shaped valve. Impaired valve mobility was usually associated with a systolic click of low intensity, and in the presence of an immobile aortic valve there was no systolic click. The aortic second sound was correspondingly diminished or absent.

Early systolic clicks and loud aortic closure sounds were also present in patients in whom a diseased aortic valve had been replaced by three Teflon prosthetic leaflets. In most of these patients there was no systolic click before insertion of the prosthesis.

In the patients studied, the systolic click occurred at the end of the opening movement of the aortic valve at the onset of left ventricular ejection. It followed the crossover point of the left ventricular and aortic pressure pulses by a time interval ranging from 15 to 45 msec. (average 33 msec.)

The opening movement of the valve was thought to produce the systolic click by causing sudden tension of the valve membrane or sudden alteration in the velocity of blood flow. By analogy with the opening snap in mitral stenosis, the early systolic click may be regarded as the “opening snap” of the aortic valve. A systolic click is clinical evidence of a mobile aortic valve in patients with aortic valve disease.

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References


Scientific Accuracy

Accuracy is the twin brother of honesty.—TRYON EDWARDS.

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