able sign of pulmonary hypertension.\textsuperscript{18} Attenuated motion of the aortic root ordinarily indicates low cardiac output and, in the setting of pericardial effusion, could suggest a misleading diagnosis of tamponade when none exists. Since patients with large pericardial effusions may have coexisting cardiac disease, the final determination of the nature of a motion abnormality of a cardiac valve cannot be made until a repeat echocardiogram is obtained following partial or complete resolution of the fluid accumulation.

References


Pseudoejection Sound in Hypertrophic Subaortic Stenosis

An Echocardiographic Correlative Study

KARL C. SZE, M.D., AND PRAVIN M. SHAH, M.D.

SUMMARY Phonoechocardiographic studies were performed in 23 patients with hypertrophic subaortic stenosis. In ten patients a distinct systolic sound was recorded, usually along the lower left sternal border. In eight of these cases the sound was low or medium frequency. Unlike the ejection click of valvular aortic stenosis, the sound began 40-100 msec after the upstroke of the indirect carotid pulse and occurred close to the initial peak of the carotid pulse.

This sound, termed pseudoejection sound, was associated with systolic anterior movement (SAM) of the anterior mitral leaflet on the echocardiogram. In all six patients studied with simultaneous phonoechocardiograms, the pseudoejection sound coincided with the sudden halting of SAM of the anterior mitral leaflet. Following provocative maneuvers the sound became louder, and its timing, as well as the sharp halting of SAM of the mitral leaflet, occurred earlier in systole.

The pseudoejection sound probably results either from impact of the anterior mitral leaflet against the interventricular septum or more likely from deceleration of blood flow in the left ventricular outflow tract. The echo data support the association of the pseudoejection sound with significant left ventricular outflow obstruction.

THE OCCURRENCE of systolic ejection sounds in idiopathic hypertrophic subaortic stenosis (IHSS) has been debated. A number of investigators have failed to document such sounds in this disease.\textsuperscript{1,4} Others have noted their presence in a small number of individual cases or as an unusual finding in a large group of patients.\textsuperscript{3,7} Braunwald et al.\textsuperscript{3} noted definite early systolic sounds in seven of 64 patients, an incidence of 11%.

Various investigators have commented on atypical features of these sounds. DeJoseph et al.\textsuperscript{8} noted that eight of 42 patients (19%) had delayed systolic ejection sounds. In all cases the systolic sound occurred at the peak of the initial carotid pulse rather than at the onset of the carotid upstroke. They concluded that these sounds were different in origin and nature compared with the ejection sounds in other diseases. In an earlier report Snellen\textsuperscript{9} denied the occurrence of systolic ejection sounds in IHSS, but noted a similar, if not identical, sound. He observed the frequent occurrence of a loud low-pitched systolic sound which developed with the sudden downstroke following the early peak of the carotid pulse, marking the onset or sudden increase of a systolic murmur. Likewise, Hancock\textsuperscript{10} has described a systolic sound of low pitch and delayed onset, simultaneous with the peak carotid pulse. He has termed it a “pseudoejection sound” because of its superficial resemblance to the true ejection sound of valvular aortic stenosis.
The purpose of this study is twofold: to clearly define the characteristics of this systolic sound in IHSS by use of phonocardiography with indirect carotid pulse recordings, and to elucidate its genesis by simultaneous phonoecho-cardiography. The term pseudo-ejection sound will be used to describe this atypical sound occurring in the first half of ventricular ejection.

Methods

Fourteen patients with IHSS were studied prospectively, and the records of nine additional patients were reviewed retrospectively. There were 16 females and seven males. In 21 of 23 cases the diagnosis was established by hemodynamic and angiocardiographic data. In the remaining two patients the diagnosis was made by clinical and echocardiographic criteria. Four patients, all in the prospective group, had undergone ventriculomyotomy, but postoperative cardiac catheterizations were not performed. However, the systolic anterior motion (SAM) of the anterior mitral leaflet persisted in two of these patients on subsequent echocardiograms. Patients with concomitant aortic valve disease were excluded from this study.

Phonocardiograms of the 14 prospective patients were obtained using a multichannel Cambridge recorder with microphones placed over the pulmonic area and lower left sternal border and subsequently over the aortic area and apex. Heart sound recordings were made employing 50, 100, 250 and 350 cps high-pass filters with 24 db per octave rolloff at a paper speed of 50 or 100 mm/sec. Simultaneous indirect carotid pulse and electrocardiographic tracings were obtained. A Unirad echocardiograph with a 2.25 MHz transducer was interfaced to the Cambridge recorder for simultaneous echocardiograms on the strip chart.

All patients studied prospectively were examined in the supine as well as the left lateral positions. Baseline phonocardiograms were initially obtained with carotid pulses and followed by simultaneous echocardiograms. Studies were obtained at rest, during the Valsalva maneuver, and after amyl nitrite inhalation.

The phonocardiograms of the nine patients analyzed retrospectively were recorded in similar fashion using a Sanborn recorder with 50, 100, 200 and 400 cps high-pass filters with 24 db per octave rolloff at a paper speed of 75 mm/sec. Echocardiograms were recorded separately.

The interval between the following events and the pseudo-ejection sound (X) were measured: 1) the initial QRS deflection on the electrocardiogram (Q-X interval), 2) the initial rapid component of the first heart sound (S1-X interval), 3) the onset of the indirect carotid pulse upstroke (C-X interval), and 4) the initial peak carotid pulse (PC-X interval). When the peak carotid pulse occurred after the pseudo-ejection sound, the PC-X interval was recorded as a negative value. All intervals were measured to the nearest 10 msec. Mean values were obtained from at least five beats either at rest or during provocative maneuvers.

Results

Phonocardiograms demonstrated discrete early systolic sounds in ten of 23 patients with IHSS. This group included six of 14 patients examined prospectively and four of nine patients examined retrospectively.

Figure 1 illustrates the pseudo-ejection sound in the ten cases. In contrast to the typical aortic ejection click, the pseudo-ejection sound was low or medium-pitched in all but two cases. It was generally best demonstrated with a 50 or 100 cps high-pass filter. Although clearly recorded, the sound was characteristically difficult to appreciate on auscultation, being obscured by the associated systolic murmur. It was audible in only the two patients in whom it was high-pitched. The pseudo-ejection sound was generally best recorded along the lower left sternal border. In several cases it could only be delineated over the aortic or pulmonic areas due to the prominence of the systolic murmur elsewhere over the precordium.

As noted in table 1, the pseudo-ejection sound followed the onset of the Q wave of the simultaneous electrocardiogram by 150–230 msec. It followed the first heart sound by 70–150 msec. Most strikingly, it followed the upstroke of the indirect carotid pulse by 40–100 msec. The pseudo-ejection sound actually occurred near the peak of the carotid pulse with a range of 30 msec before to 40 msec after its inspersion.

![Figure 1. Phonocardiograms showing pseudo-ejection sounds (x) in ten patients. Examples correspond by number to the patients listed in table 1. The sound was defined as low frequency when it was best recorded with a 50 cps high-pass filter (cases 2, 3, 5, 6, 10), medium frequency when best recorded with a 100 cps high-pass filter (cases 4, 7, 8), and high frequency when it was best recorded with a 250 cps or greater high-pass filter (cases 1, 9). Time lines mark 40 msec intervals.](http://circ.ahajournals.org/Downloadedfrom)
All ten patients with pseudoerection sounds had systolic anterior movement (SAM) of the anterior mitral leaflet on echocardiographic examination. The SAM of the mitral leaflet was readily observable, well-defined, and noted to extend fully to the interventricular septum at rest in seven cases and with provocative measures, i.e., amyl nitrite inhalation or Valsalva maneuver, in nine of the ten cases. In one patient, who was difficult to examine by echocardiography, only fragmentary SAM of the mitral leaflet was recorded on several occasions. In all six patients studied with simultaneous phonoecchocardiography, the onset of the pseudoerection sound was demonstrated consistently to coincide with the sudden halting of anterior movement of the anterior mitral leaflet in the left ventricular (LV) outflow space (fig. 2). Only those beats with abrupt halting of mitral leaflet movement were associated with pseudoerection sounds. Those beats with a rounded pattern of SAM of the mitral leaflet failed to demonstrate pseudoerection sounds.

The pseudoerection sound was frequently absent or inconsistently present during initial supine examination. This occurred despite the presence of SAM of the mitral leaflet. However, this sound could be clearly demonstrated following amyl nitrite inhalation or during the Valsalva maneuver. In one patient the pseudoerection sound was clearly elicited in the post-PVC beat. Figure 3 demonstrates the dynamic nature of the obstruction. Initially only a faint systolic murmur was noted. However, the Valsalva maneuver not only produced the characteristic increase in murmur intensity and change in carotid pulse contour typical of IHSS, but it also evoked a distinct click-like sound. In other cases where the pseudoerection sound was present initially, it moved earlier into systole following the provocative maneuvers. Its temporal relationship to the mitral leaflet movement held despite significant beat-to-beat variations in individual patients of up to 60–70 msec in the S1-X interval. Figure 4 illustrates an example of this behavior. During the peak Valsalva strain, the mitral leaflet reaches the septum earlier in systole and is associated with the pseudoerection sound closer to S1. Figure 5 demonstrates a dynamic response to amyl nitrite inhalation. A change in shape of the SAM of the mitral leaflet is associated with a change in carotid pulse contour and prominence of a low frequency systolic sound.

Four of the 13 patients without demonstrable pseudoerection sound showed persistent and full SAM of the mitral leaflet obliterating the LV outflow space, either at rest or with provocation by amyl nitrite inhalation and Valsalva maneuver. Four patients had only fragmentary and small SAM of the mitral leaflet, and the remaining five patients demonstrated no SAM of the mitral leaflet at rest.

Discussion

The high-pitched ejection sound of valvular aortic stenosis is intimately related to the valve itself, occurring coincident with the maximal doming of the valve cusps. It is essentially an opening snap of the aortic valve. Temporally, it also coincides closely with the onset of the indirect carotid pulse rise.

This study confirms the rarity of true systolic ejection

![Phonoecchocardiogram from patient 4 demonstrating the simultaneous occurrence of the pseudoerection sound with the abrupt halting of systolic anterior movement of the anterior mitral valve leaflet (AMV). The dashed lines are drawn in beats 1 and 3 to illustrate this association. Heart sounds are recorded from the pulmonic area (PA) and cardiac apex with a 100 cps high-pass filter. The time lines occur 40 msec apart in this and subsequent figures. IVS = interventricular septum, EKG = electrocardiogram.](image)

![Phonoecchocardiogram at rest demonstrating only a trivial systolic murmur and absence of pseudoerection sound. The carotid pulse recording is normal. (Right) phonoecchocardiogram during the Valsalva maneuver demonstrating a more prominent systolic murmur (SM) with a distinct pseudoerection sound (X) occurring 40 msec after the carotid upstroke. Note the change in carotid pulse contour. Ao = aortic area, LSE = lower left sternal edge.](image)
TABLE 1. Features of the Pseudoejection Sound

<table>
<thead>
<tr>
<th>Patient</th>
<th>Supine control</th>
<th>Valsalva or amyl nitrite</th>
<th>Frequency</th>
<th>Best location</th>
<th>SAM</th>
<th>Timing*</th>
<th>HR</th>
<th>Q-X</th>
<th>S1-X</th>
<th>C-X</th>
<th>PC-X</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. D.L. +</td>
<td>+</td>
<td>High</td>
<td>LSE</td>
<td>+</td>
<td>+</td>
<td>85</td>
<td>(C) 210</td>
<td>150</td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>2. R.K. +</td>
<td>+</td>
<td>Low</td>
<td>LSE, apex</td>
<td>+</td>
<td>+</td>
<td>75 (PVC) 160</td>
<td>75</td>
<td>N</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. E.D. ±</td>
<td>+</td>
<td>Low</td>
<td>LSE, Ao</td>
<td>+</td>
<td>+</td>
<td>75 (C) 230</td>
<td>150</td>
<td>80</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. E.A. ±</td>
<td>±</td>
<td>Med</td>
<td>LSE, Ao</td>
<td>+</td>
<td>+</td>
<td>55 (C) 230</td>
<td>140</td>
<td>80</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. E.C. −</td>
<td>+</td>
<td>Low</td>
<td>PA</td>
<td>+</td>
<td>+</td>
<td>75 (A) 210</td>
<td>150</td>
<td>N</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. E.S. −</td>
<td>+</td>
<td>Low</td>
<td>LSE, Ao</td>
<td>+</td>
<td>+</td>
<td>75 (A) 230</td>
<td>150</td>
<td>N</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. A.A. +</td>
<td>+</td>
<td>Med</td>
<td>LSE</td>
<td>+</td>
<td>N</td>
<td>100</td>
<td>(C) 170</td>
<td>110</td>
<td>60</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>8. A.J. ±</td>
<td>+</td>
<td>Med</td>
<td>PA</td>
<td>+</td>
<td>N</td>
<td>50 (C) 220</td>
<td>140</td>
<td>N</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. G.R. ±</td>
<td>±</td>
<td>High</td>
<td>Ao</td>
<td>±</td>
<td>N</td>
<td>90 (C) 200</td>
<td>130</td>
<td>100</td>
<td>40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10. J.O. ±</td>
<td>−</td>
<td>Low</td>
<td>LSE, apex</td>
<td>+</td>
<td>+</td>
<td>85 (V) 180</td>
<td>100</td>
<td>70</td>
<td>20</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Simultaneous occurrence of pseudoejection sound with abrupt halting of SAM of the anterior mitral leaflet in the LV outflow space.

Abbreviations: + = consistently present; ± = intermittently present; − = absent; N = not measured; C = control; V = Valsalva; A = amyl nitrite; PVC = premature ventricular contraction; HR = heart rate; SAM = systolic anterior movement; med = medium; Ao = aortic area; PA = pulmonic area; LSE = lower left sternal edge; Q-X, S1-X, C-X, PC-X = intervals from onset of QRS on electrocardiogram, first heart sound, carotid upstroke and initial peak carotid pulse to pseudoejection sound, respectively.

sounds in IHSS, but demonstrates the relatively common presence of a discrete sound occurring somewhat later and termed pseudoejection sound. Characteristics of this sound include: 1) its generally low or medium-pitched quality, 2) frequent beat-to-beat variations in intensity, 3) enhancement by provocative maneuvers known to increase the outflow gradient in IHSS, 4) movement earlier into systole by maneuvers known to intensify outflow obstruction, 5) onset well after the beginning of aortic ejection, actually near the initial peak of the indirect carotid pulse, and 6) its close association with abrupt halting of the systolic anterior motion (SAM) of the anterior mitral leaflet in early systole.

Many of the early systolic sounds described by other investigators are undoubtedly the same as those in the present series of cases. Whether they be labelled pseudoejection sound, 4 low-pitched mid-systolic sound, 9 or systolic "click-like" sound, 9 the descriptions and illustrations published show them to be similar in both timing and pitch. Three of the eight cases studied by DeJoseph et al. also demonstrated enhancement by provocative maneuvers. In two cases the sound was only recorded following amyl nitrite inhalation, and in one case it was brought out by the Valsalva maneuver.

FIGURE 4. (Left) Phonoechocardiogram at rest of patient 1 demonstrating a pseudoejection sound coincident with the abrupt halting of anterior mitral valve (AMV) leaflet movement (dashed lines). The Q-X and S1-X intervals are 210 and 150 msec, respectively. (Right) with the Valsalva maneuver, the halting of SAM of the AMV occurs progressively earlier in systole. The pseudoejection sound moves earlier by the same interval (dashed lines). The Q-X interval shortens to 180 msec in beat 1 and 160 msec in beat 3. The S1-X interval shortens to 120 msec in beat 1 and 90 msec in beat 3. Baseline artifact during the Valsalva maneuver is caused by muscle tremor.
maneuver. Additionally, Kolibash et al. reported a case of IHSS with severe symptoms during pregnancy which lessened after delivery. During pregnancy a prominent pseudoejection sound was recorded 40 msec after the onset of ejection as judged from the E point on the apexcardiogram. Following delivery when symptoms were improved, the sound was no longer recorded. The systolic ejection sounds described by Braunwald et al. may also be pseudoejection sounds. Their illustration clearly shows the "ejection" sound to be of the delayed type, occurring 40 msec after the carotid upstroke and just prior to the peak carotid pulse. Tucker et al. noted definite left-sided ejection clicks in only two of 90 patients with IHSS. However, they also recognized the presence of intermittent nonejection clicks in four patients.

The spatial relationship of the pseudoejection sound with the sudden halting of the SAM of the mitral leaflet clearly suggests the role of the mitral leaflet in the genesis of this sound. The pseudoejection sound may be caused by actual impact of the anterior mitral leaflet as it reaches up to the hypertrophied and bulging interventricular septum. A more likely explanation for the genesis of the pseudoejection sound is the pressure transient arising from the sudden deceleration of blood flow in the left ventricular outflow tract. Endocardial thickening of the septum as well as the anterior mitral leaflet have been observed, possibly representing repeated coaption of the two surfaces.11

Previous studies have suggested the association of abnormal mitral leaflet movement with the localization of the LV outflow obstruction in IHSS. The variable nature of the pseudoejection sound in our patients, its enhancement and movement earlier into systole by provocative maneuvers known to increase the LV outflow obstruction in IHSS, such as amyl nitrite inhalation and Valsalva maneuver, are also consonant with this view of dynamic LV outflow obstruction. Indeed, as the pseudoejection sound moves earlier in systole with these maneuvers, the initial peak of SAM of the mitral leaflet likewise moves earlier. A louder pseudoejection sound, such as that associated with these maneuvers, is presumably secondary to greater deceleration of blood flow in the LV outflow tract or greater impact of the anterior mitral leaflet against the septum. When the LV outflow obstruction becomes less, the pseudoejection sound may become faint or absent. The timing of the pseudoejection sound near the initial peak carotid pulse is also compatible with this view. The rapid reversal of aortic and carotid pulse pressures is thought to be partly due to the obstruction caused by the anterior mitral leaflet in the LV outflow tract.

Correlation of the pseudoejection sound with the severity of outflow obstruction among individual patients remains to be defined since cardiac catheterizations in our patients were not performed concurrently with the present study. All eight patients with systolic "click-like" sounds studied by DeJoseph et al. had LV outflow gradients of 67 mm Hg or greater, either at rest or after provocative measures. However, they found absence of the sound by no means ruled out a large gradient. Similarly, Hancock noted the presence of pseudoejection sounds in patients with more severe forms of IHSS. Echocardiographic studies correlated with hemodynamic data have shown that when SAM of the mitral leaflet consistently obliterates the LV outflow tract, persistent pressure gradients are generally present. When the SAM of the mitral leaflet is inconstant or does not reach the interventricular septum, pressure gradients are latent or labile. Likewise, where no abnormalities at rest are noted,
Echocardiographic Assessment of the Level of Cardiac Compensation in Valvular Heart Disease

ANDREW ROSENBLATT, M.D., RALPH CLARK, M.D.,
JUDITH BURGESS, B.A., AND KEITH COHN, M.D.

SUMMARY The level of cardiac compensation in valvular disease was studied by relating echocardiographic and cardiac catheterization measurements. Three groups — compensated, intermediate, and decompensated — were defined according to the left ventricular angiographic pattern and cardiac output. The echocardiographic ejection indices, percent left ventricular minor diameter shortening, ejection fraction, and fiber shortening rate were significantly higher than normal in compensated mitral regurgitation, lower than normal in compensated aortic stenosis, and within normal limits in compensated aortic insufficiency. In the decompensated state these indices were depressed. Intermediate compensation was best recognized by combining several echocardiographic variables into an echocardiographic score based on multivariate discriminant function analysis. Thus, the compensated volume overload states (aortic and mitral regurgitation) and pressure overload state (aortic stenosis) have separate sets of “normal” echocardiographic values; low ejection indices characterize the decompensated group, while recognition of intermediate compensation requires analysis of multiple echocardiographic variables.

OVER THE LAST SEVERAL YEARS, echocardiography has facilitated the differential diagnosis of various types of valvular and congenital heart disease. Difficulties in assessing the degree of cardiac compensation in valvular disease and these questions relevant to the appropriate timing of cardiac catheterization, angiography, and surgical intervention continue to confront the clinician. Although methods for quantifying ventricular size and contractility have been introduced, none are considered completely reliable, and the establishment of a simple noninvasive means of determining the level of left ventricular function in valvular disease states would permit more “precise” management of patients with these lesions.

This study, then, seeks those echocardiographic features

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