Sound-Pressure Correlates of the Aortic Ejection Sound
An Intracardiac Sound Study
By ARTHUR V. WHITTAKE, M.D., JAMES A. SHAVER, M.D.,
SAMUEL GRAY, III, M.D., AND JAMES J. LEONARD, M.D.

SUMMARY
The sound-pressure correlates of the aortic ejection sound (AES) were studied in six patients without aortic valve disease (group 1) and in nine patients with valvular aortic stenosis (group 2) during diagnostic left heart catheterization. Aortic root sound and pressure events were recorded by a catheter-tip micromanometer simultaneously with the external phonocardiogram, indirect carotid pulse, and electrocardiogram. The AES in group 1 patients was coincident with the onset of pressure rise in the aortic root. This relationship was not altered by drug administration, although variations in the amplitude of the AES occurred. It was concluded that the AES in patients without aortic valvular disease is an exaggerated ejection component of the normal first heart sound, and that it is a root phenomenon related to the forceful ejection of blood into the aorta. The AES in group 2 patients was delayed 24 to 40 msec after the onset of pressure rise in the aortic root, was coincident with the anacrotic notch, and introduced the aortic ejection murmur. Aortic root sound and pressure were recorded simultaneously with aortic valve motion during angiography in two patients utilizing a cine trace system. The AES occurred when the domed valve reached its maximal excursion, and the interval between the onset of aortic pressure rise and the AES was equal to the total time required for the piston-like ascent of the dome. It was concluded that the AES in valvular aortic stenosis is valvular in origin. The amplitude of the valvular AES correlated best with valve mobility on the angiogram, and no correlation existed between its presence and the severity of the valvular stenosis.

Additional Indexing Words:
Valvular aortic stenosis Left heart catheterization Coronary angiography
Phonocardiogram Methoxamine Catheter-tip micromanometer Amyl nitrite
Norepinephrine Isoproterenol

THE existence of a prominent ejection sound or click in various pathologic conditions of the aortic valve or root has been appreciated by clinicians for years. Much interest has centered around the mechanism of the production of this sound and its hemodynamic correlates. There is general agreement that conditions causing dilatation of the aortic root, such as systemic hypertension or aortic root aneurysms, produced prominent aortic ejection sounds by sudden distention of the dilated great vessel with the onset of systole. Opinion is divided regarding the origin of the aortic ejection sound in valvular aortic stenosis. Independent reports by Leatham and Vogelpoel and Reinhold and associates have stated that the ejection sound of valvular aortic stenosis is a root event, arising from the poststenotic dilatation of the aorta and that it occurs after the onset of pressure rise in the indirect carotid pulse.
Clinical and Catheterization Data

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<th>Patient</th>
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<th>Disease</th>
<th>Range of mobility*</th>
<th>Calcification†</th>
<th>Aortic regurgitation†</th>
<th>Comment</th>
<th>Peak gradient (mm Hg)</th>
<th>Valve area (cm²)</th>
<th>OAPR to AES (msec)</th>
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Group 2

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Abbreviations: OAPR = onset of aortic pressure rise; AES = aortic ejection sound; ASHD = arteriosclerotic heart disease; CAS = congenital aortic stenosis; RHD = rheumatic heart disease; MS = mitral stenosis; AS = aortic stenosis; AI = aortic insufficiency.

* Range of mobility: 4+ = normal; 0 = immobile.
† Graded 0 to 4+ with 4+ being the most severe.

Hancock,4 in more recent reports, stated that the ejection sound in valvular aortic stenosis is valvular in origin, and occurs simultaneously with the onset of pressure rise in the indirect carotid pulse. The difference in sound and pressure correlates found by these investigators may well be due to variable time delays and pressure contour distortions which are inherent in conventional pressure recording devices used for obtaining indirect carotid pulses. With the advent of the catheter-tip micromanometer, it is now possible to record both intracardiac sound and pressure free of contour distortion without delay in transmission. In an attempt to define further the origin of the aortic ejection sound and its hemodynamic correlates, the following study was performed.

Methods

Fifteen patients having a well-defined aortic ejection sound at the bedside which was confirmed by external phonocardiography were studied. Six patients without aortic valve disease were studied during selective coronary angiography (group 1). Nine patients with valvular aortic stenosis were evaluated during diagnostic left heart catheterization (group 2). Table 1 summarizes the pertinent clinical and catheterization data of these patients.

Intracardiac sound and pressure events were recorded by a Dallons-Telco catheter-tip micromanometer.* The micromanometer is a variable inductance transducer from which low frequency vibrations are recorded as pressure and high frequency vibrations (above 40 cps) are recorded as sound. This system has a uniform amplitude response to frequencies up to 200 cps. The catheter-tip micromanometer was introduced through a right brachial arteriotomy into the root of the aorta just above the aortic valve. Great care was exercised in placing the tip of the catheter so that it would not interfere with valve motion or produce artificial sounds.

*Dallons Laboratories Inc., 5066 Santa Monica Boulevard, Los Angeles, California.

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by striking the wall of the aorta. From the catheter tip, both aortic root sound and pressure events are recorded simultaneously without transmission delay. External phonocardiograms were recorded by a contact microphone* placed on the chest wall at the point of maximal intensity of the aortic ejection sound. The indirect carotid pulse was obtained from a standard funnel-shaped pickup connected to a P-23Db transducer.† This was placed over the maximal pulsation of the carotid artery to obtain the sharpest possible evidence of onset of left ventricular ejection and a dicrotic notch. Standard lead II of the electrocardiogram was used. These data were recorded simultaneously on a multichannel photographic recording unit* at a paper speed of 100 mm/sec with time markers indicating 0.02 sec (fig. 1).

In two subjects with valvular aortic stenosis (J. C. and G. H., table 1), sound, pressure and electrocardiogram were recorded simultaneously on 35-mm cine film during left ventricular or aortic root angiography with a cine trace system.* This system makes it possible to correlate the time course of the instantaneous sound and pressure events of the aortic root with the motion of the aortic valve.†

Results

Group 1

Figure 1 is representative of the sound-pressure correlates of the aortic ejection sound in patients without aortic valve disease (F.M., table 1). In all six such patients studied, the timing of the external and internal sound events was identical, although the amplitude and frequency response of the two systems were different. All six patients in group 1 showed the following: (1) The onset of the aortic ejection sound was coincident with the

<table>
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<th>Drug</th>
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<td>norepinephrine</td>
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<td>Increased</td>
</tr>
</tbody>
</table>

*Electronics for Medicine, White Plains, New York.
†Statham Instruments Inc., Hato Rey, Puerto Rico.

Figure 1

Patient 1 (F.M.). Sound-pressure correlates of the AES in group 1 patients without aortic valve disease. From top to bottom: External phonocardiogram recorded at the point of maximal intensity of the AES; internal phonocardiogram and direct aortic root pressure recorded from the catheter-tip micromanometer; indirect carotid pulse; and lead II of the electrocardiogram. Note the onset of the AES coincident with the onset of pressure rise in the aortic root. The onset of the pressure rise in the indirect carotid pulse is delayed 40 msec from the onset of pressure rise in the aortic root. The S1 is coincident with the isometric contraction wave in the aortic root tracing. S1 = first component of the first heart sound; AES = aortic ejection sound; A2 = aortic closure. Paper speed 100 mm/sec; time markers = 0.02 sec.

Table 2

Effect of Drugs on Sound-Pressure Correlates of the Aortic Ejection Sound (AES) in Group 1: Relationship of Onset of Aortic Pressure Rise to AES

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The onset of pressure rise in the aortic root. (2) The onset of pressure rise in the indirect carotid pulse was delayed 30 to 50 msec from the onset of pressure rise in the aortic root. (3) The ejection sound frequently introduced a short systolic ejection murmur. (4) The first heart sound was coincident with the isometric contraction wave as recorded from the aortic root. Table 2 and figures 2 and 3 demonstrate the effect of the administration of amyl nitrite, methoxamine, norepinephrine, and isoproterenol to various patients in this group. None of these drugs altered the timing of the sound-pressure relationships outlined above, although changes in the amplitude of the ejection sound occurred (table 2).

Group 2

Figures 4 and 5 are representative of the sound-pressure correlates of the aortic ejection sound in the nine patients with valvular aortic stenosis (J.M. and D.B., table 1). All subjects in group 2 showed the following: (1) The onset of the ejection sound was delayed 24 to 40 msec from the onset of pressure rise in the aortic root and was coincident with the anacrotic notch on the upstroke of the aortic root pressure. (2) The ejection sound introduced the long diamond-shaped murmur characteristic of aortic stenosis. (3) The first heart sound was coincident with the isometric contraction wave and was separated from the ejection sound by an interval free of sound or murmur.

Figure 6 shows a single complex in patient 8 (J.C., table 1) obtained during a left ventricular injection of contrast utilizing a cine trace system. Aortic root sound and pressure are recorded from the micromanometer. The arrows numbered 1, 2, 3, and 4 represent the timing of 4 cine frames taken at 17-msec intervals during the early upstroke of the aortic pressure (fig. 7).

Discussion

In this study, a catheter-tip micromanometer was placed in the aortic root to study the

![Figure 2](http://circ.ahajournals.org/)

**Figure 2**

Patient 1 (F.M.) The effects of methoxamine and amyl nitrite on the sound-pressure correlates of the AES are compared with the resting state. In each tracing the ejection sound is coincident with the onset of pressure rise in the aortic root, which, in turn, always precedes the onset of the indirect carotid pulse. Methoxamine decreases the amplitude of the AES, while amyl nitrite increases it.
Patient 5 (S.C.) The effects of norepinephrine and isoproterenol on the AES are compared. Both agents increase the amplitude of the AES, which is coincident with the onset of pressure rise in the aortic root. The hypertensive effect of norepinephrine prolongs isometric contraction time, thereby separating $S_1$ from the AES, while the pure inotropic action of isoproterenol shortens this period, causing fusion of $S_2$ with the AES.

Figure 4

Patient 7 (J.M.) The sound-pressure correlates of the AES as recorded from this 23-year-old male with congenital aortic stenosis are representative of group 2 patients with valvular aortic stenosis. The onset of the AES is delayed 38 msec from the onset of pressure rise in the aortic root and is coincident with the anatomic notch. This sound introduces the systolic murmur (SM) characteristic of aortic stenosis. $S_1$ is coincident with the isometric contraction wave and is separated from the AES by an interval free of sound or murmur.
six patients having a prominent ejection sound with no evidence of aortic valvular disease (group 1). The second pattern was seen in nine patients with valvular aortic stenosis (group 2).

The pattern in group 1, patients without aortic valvular disease, showed the onset of the aortic ejection sound to be coincident with the onset of the pressure rise in the aortic root and frequently introduced a short systolic ejection murmur. The sound-pressure correlation of this event is identical with the sound-pressure correlation of the ejection component of the normal first heart sound as described by Piemme and co-workers.6 In this study of normal heart sounds in dogs, where flow was recorded by an electromagnetic flowmeter less than 2 cm above the aortic valve and pressure by a catheter-tip micromanometer identical to the one used in this study, the ejection component of the first heart sound began concomitantly with the rise of aortic pressure and the onset of aortic blood flow. Similar observations of the ejection component of the normal first heart sound have been made by Shah and associates,7 utilizing similar recording devices. Both investigators agree that the origin of the ejection component of the normal first heart sound is a root phenomenon related to the forceful ejection of blood into the aorta.

The administration of various drugs (table 2) did not alter the timing of the sound-pressure correlates of the ejection sound. However, isoproterenol and norepinephrine, both powerful inotropic agents, markedly increased the amplitude of the ejection sound. These

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**Figure 5**

Patient 11 (D.B.) The sound-pressure correlates of the AES as recorded from a 32-year-old man with severe congenital aortic stenosis (group 2). The AES is delayed 30 msec from the onset of pressure rise in the aortic root and is coincident with the anacrotic notch. The amplitude of this sound is decreased as compared with the ejection sound in figure 4. That patient (J.M.) had a mobile noncalcified domed valve demonstrated by angiography, while this patient (D.B.) had 2+ calcification of the valve with decreased mobility.

**Figure 6**

Aortic root sound and pressure recorded in patient 8 (J.C.) from the micromanometer above the aortic valve during left ventricular injection of contrast medium. The arrows numbered 1, 2, 3, and 4 represent the timing of 4 cine frames taken at 17-msec intervals during the early upstroke of the aortic pressure. These are reviewed in sequence in figure 7.
Patient 8 (J.C.). The timing of cine frames 1, 2, 3, and 4 are shown in figure 6. Aortic sound and pressure and the electrocardiogram are recorded in the lower left corner of each frame by the cine trace system. The last image recorded reading from left-to-right corresponds to the instant of exposure of the cine frame, thereby allowing simultaneous sound, pressure, and motion correlation. Cine frame 1 demonstrates the closed aortic valve in late diastole as confirmed by the aortic pressure recorded on the cine trace from the micromanometer above the valve. The patient is in the right anterior oblique position, and contrast material is being injected through the transseptal catheter. A short run of prior ventricular tachycardia induced by the injection of contrast medium is seen on the cine trace. In cine frame 2, the domed valve is beginning its ascent coincident with the onset of pressure rise in the aortic root. Note the absence of sound in the aortic sound tracing. Cine frame 3 shows the maximal excursion of the dome when its elastic limits are reached. A prominent ejection sound is recorded on the sound tracing, while a sharp anacrotic notch is inscribed on the aortic pressure. Cine frame 4 demonstrates no further excursion of the valve and the beginning of the ejection murmur. Subsequent frames demonstrate the systolic jet issuing from the orifice of the dome, while a corresponding shudder and ejection murmur are recorded.

Drugs increased the force of contraction, increased the rate of pressure change (dp/dt), and accelerated blood into the aortic root more rapidly. Methoxamine, lacking inotropic effect, decreased the forceful ejection of blood into the aortic root in face of a high peripheral resistance, and a decrease in the amplitude of the aortic ejection sound was seen. The administration of these agents has identical effects on the amplitude of the ejection component of the normal first heart sound as described by both Piemme and associates and Sakamoto and his group.

From these data we conclude that the aortic ejection sound in group 1 subjects without aortic valve disease is an exaggerated ejection component of the normal first heart sound and a root phenomenon. This mecha-
nism would explain the high incidence of audible aortic ejection sounds in clinical conditions associated with a forceful left ventricular ejection such as thyrotoxicosis, anemia, exercise, and other high output states. Likewise, normal left ventricular ejection into a sclerotic aortic root would account for the frequent occurrence of aortic ejection sounds in elderly patients. The triad of a tortuous sclerotic aortic root, a tight noncompliant arterial tree, and a forceful left ventricular ejection would readily explain the loud aortic ejection sound almost always observed in patients with hypertensive cardiovascular disease.

The recognition of the aortic ejection sound as a separate sound at the bedside will depend not only upon its amplitude but also upon its separation from the mitral component of the first heart sound. Intervention such as isoproterenol infusions or exercise, which markedly shortened isometric contraction time, may cause fusion of the two components of the first heart sound, such that only a single sound is appreciated by the ear. Frequently, the two components of this fusion can be distinguished on the phonocardiogram. Likewise, prolongation of the isometric contraction time, as seen in the failing heart and some cases of hypertensive cardiovascular disease, will separate the two components, thus making the ejection sound easily appreciated at auscultation.

The pattern of the sound and pressure relationship seen in group 2 subjects with valvular aortic stenosis was totally different from that observed in group 1 subjects without valvular disease. The first vibration of the aortic ejection sound was delayed 24 to 40 msec from the onset of pressure rise in the aortic root and was coincident with the anacrotic notch in the upstroke of the central aortic pressure (figs. 4 and 5). This ejection sound introduces the long diamond-shaped murmur characteristic of aortic stenosis. Figure 8 compares the timing of the aortic root sound and pressure tracings as recorded from the catheter-tip micromanometer with the aortic root pressure (sidearm of the Telco catheter directly to a Statham P-23Db transducer), indirect carotid pulse, and brachial artery pressure tracing, the pickups for the latter two were also directly connected to a P-23Db transducer. The onset of pressure rise in the micromanometer is 20 msec earlier than its sidearm, and 14 msec earlier than the indirect carotid pulse. The further the distance the recording site is from the aortic root, the greater is the delay in onset of pressure rise. For this reason correction for such delay in pulse wave transmission from the aortic root to other recording sites must be made when tracings from such sites are used as a reference for timing sounds. Also the sharp, initial rise followed in 24 msec by a sharp anacrotic notch seen in the micromanometer tracing is observed only in the aortic sidearm and is seen as a slurred upstroke in the carotid and brachial artery

![Figure 8](https://example.com/figure8.png)

**Figure 8**

*Patient 8 (J.C.) The sound-pressure correlates of the AES as recorded in a 48-year-old man with rheumatic aortic stenosis. Note again the delay of the ejection sound coincident with the anacrotic notch in the Telco tracing from the aortic root. The onset of the rise in the aortic root sidearm, indirect carotid, and brachial artery tracings are all delayed when compared with the onset of the aortic root pressure rise in the Telco tracing. Note also that the sharp initial rise in the Telco tracing followed by the anacrotic notch is seen only as a slurred upstroke in the indirect carotid and brachial artery tracings (see text).*
tracings. With this in mind, we agree with the statement of Hancock that the ejection sound in valvular aortic stenosis is almost simultaneous with the onset of ejection as indicated by the onset of the upstroke of the indirect carotid pulse. However, due to significant and variable delays in pulse wave transmission and distortion of the contour in the the early upstroke of the indirect carotid pulse, information gained from such tracings regarding the timing of the aortic root pressure events with sound events is not valid. For this reason, all subsequent correlation between pressure and sound events will use the aortic root catheter-tip micro-
manometer pressure as a reference.

The mechanism of production of the aortic ejection sound in valvular aortic stenosis can best be appreciated by a consideration of the aortic root sound and pressure events with simultaneously recorded motion of the aortic valve obtained by the cine trace system. In valvular aortic stenosis of both the congenital and acquired type, there is thickening of the valve substance with fusion at the commissures. When viewed by either left ventricular or aortic root injections of contrast material, the stenotic valve has the appearance of a mobile dome whose piston-like ascent may take up to 50 msec until the elastic limits of the valve are met and it becomes a tense dome. The range of excursion of the dome will be determined by its mobility as modified by the degree of commissural fusion and leaflet calcification. One sequence of this piston-like ascent is reviewed in patient J.C. (fig. 7) who has 1+ calcification and moderate mobility of the domed valve. Cine frame 1 demonstrates the closed dome in late diastole. In cine frame 2, the ascent of the dome begins coincident with the onset of pressure rise as recorded from the micromanometer in the aortic root. Cine frame 3 shows the maximal excursion of the dome when its elastic limits are met. Cine trace recordings of aortic root pressure at this time inscribe a sharp anacrotic notch, while a prominent aortic ejection sound is recorded by intracardiac sound. Cine frame 4 demonstrates no further excursion of the valve and the beginning of the ejection murmur. Subsequent films demonstrate the systolic jet issuing from the orifice of the dome, while a corresponding shudder and ejection murmur are recorded in the aortic root pressure and sound tracings. In a second patient, G.H., an identical sequence of pressure, sound, and motion events was recorded on cine trace system.

From these two representative studies of valvular aortic stenosis, it is evident that the time interval between the onset of aortic pressure rise and the anacrotic notch (or aortic ejection sound as they are coincident) is equal to the total time required for the piston-like ascent of the domed valve. The rise in aortic pressure during this interval is due to the displacement of aortic root blood by the ascending dome without actual flow across the orifice occurring, since ejection begins only after the dome reaches its maximal excursion. These findings are identical to those in the patients with valvular aortic stenosis studied by Epstein and associates. Cineangiographic studies on 10 patients with mobile noncalcified valves demonstrated a mean time of 40 msec for full excursion of the mobile dome, while 11 patients with impaired valve mobility required a mean time of 33 msec for full ascent to occur. In our study, J.M. and S.H. (table 1) are similar to those in Epstein’s group with mobile valves with a mean time of 39 msec from onset of pressure rise in the aortic root to the ejection sound. The remaining seven patients with impaired valve movement had a mean time from the onset of aortic pressure rise to the ejection sound of 29 msec. These almost identical findings in two different studies again indicate that this interval represents the time necessary for the full excursion of the dome, prior to ejection.

Authors in the past who have stated that the aortic ejection sound in valvular aortic stenosis is a root event because the sound occurs after the upstroke of the indirect carotid pulse, have assumed that the pressure crossover of aortic and left ventricular pressure is synonymous to valve opening and
that ejection begins with pressure crossover. Both this study and that of Epstein and associates clearly demonstrate that this statement is not true and that ejection begins only after full ascent of the dome takes place, anywhere from 20 msec to 50 msec following left ventricular and aortic root pressure crossover. Furthermore, as pointed out by Minhaus and Gasul,\textsuperscript{11} if the systolic click originated from the aorta, the systolic murmur would be anticipated to precede the click. This was not observed in any of our cases or in those reviewed in the literature, but rather the aortic ejection sound always introduced the systolic ejection murmur.

The relationships of aortic sound, pressure, and valve motion described strongly point to a valvaral origin of the ejection sound in valvular aortic stenosis. Whether the energy necessary to produce this sound is due to vibrations caused by the sudden tending of the valve leaflets when their elastic limits are met as suggested by Dock,\textsuperscript{12} or to sudden deceleration of blood by the checking action of the tense dome, as suggested by others,\textsuperscript{6, 7, 13, 14} cannot be determined by this study. However, inherent in either of these mechanisms is the ability for the deformed valve to move. In cases in which there is rigid fixation of the aortic valve and excursion of the deformed leaflets is not possible, neither sudden tending of the valve leaflets nor abrupt deceleration of the column of blood could occur, because no pre-ejection ascent of the rigid valve is possible. In this study we have found good correlation of the intensity of the aortic ejection sound with the mobility of the valve. The two patients with the loudest aortic ejection sounds (J. M. and S. H.) had the greatest valve excursion on cineangiography, while patients with less mobility had softer aortic ejection sounds (D. B. and M. D.). No good correlation was found between the aortic ejection sound and the severity of the aortic stenosis, although its presence clearly defines the obstruction at the valvular level as so well pointed out in the past by Hancock.\textsuperscript{4}

The presence of the group 1 type aortic ejection sound is dependent upon the forceful ejection of the column of blood into the aortic root. For all practical purposes, the normal aortic valve imposes no obstruction to the free flow of rapidly accelerating left ventricular blood, and the energy generated is transmitted almost instantaneously to the aortic root. Severe valvular aortic stenosis would be incompatible with this type of normal left ventricular ejection, and we would predict that patients with severe stenosis could not have a group 1 type of aortic ejection sound. Careful examination of the external and root phonocardiograms in all subjects with severe valvular aortic stenosis confirms this prediction and fails to reveal any aortic ejection sounds coincident with the upstroke of the aortic root pressure, but rather, always demonstrates the aortic ejection sound coincident with the anacrotic notch of the aortic pulse. However, one patient (S. H.) with minimal congenital valvular aortic stenosis, as manifested by only a 13-mm peak gradient at rest, exhibited both an aortic ejection sound coincident with the upstroke of the aortic root pressure and an aortic ejection sound occurring 40 msec later, coincident with the anacrotic notch (fig. 9). In this situation there was not enough critical valvular stenosis to prevent free flow of blood across the orifice of the mobile dome during its ascent. This was confirmed by aortic root cineangiography which showed nonopacified left ventricular blood in the aortic root two cine frames (34 msec) before the dome reached its maximal excursion. However, when the minimally deformed mobile dome completed its full ascent, the column of blood was abruptly checked as manifested by the sharp anacrotic notch and the loud valvular aortic ejection sound. The mild degree of obstruction was further confirmed by the lack of significant shudder in the remainder of the upstroke of the aortic root pressure. This patient occupies a unique position in the spectrum of valvular aortic stenosis, in that the flow characteristics across the minimally
deformed and stenotic valve are compatible with aortic ejection sounds of both the valvular and the root type.

In light of recent literature, an excellent analogy of the sound, pressure, and motion correlates of the aortic and mitral valves can be made. The physiologic third heart sound or protodiastolic ventricular gallop, a ventricular event due to rapid ventricular filling, is a counterpart of the aortic ejection sound due to forceful left ventricular ejection into the aortic root. Likewise, the mitral opening snap, which occurs at the moment of maximal descent of the valve as it balloons into the left ventricular cavity during early diastole, is a counterpart of the valvular aortic ejection sound. Recent studies by Wooley and associates\textsuperscript{15} utilizing a catheter-tip micromanometer demonstrated that the opening snap follows left atrial and left ventricular pressure crossover by an average of 36 msec, during which time the valve descent occurs, while cineangiographic studies by Criley and Ross\textsuperscript{16} have shown that flow across the stenotic mitral valve begins only after the maximal descent occurs. In mitral stenosis the intensity of the opening snap is best correlated with the mobility of the valve and may be absent with severe calcification. The presence of severe mitral stenosis is incompatible with the rapid phase of left ventricular filling, and likewise a left ventricular protodiastolic gallop is never heard. Each of these sound, pressure, and

\[\text{Figure 9}\]

\textit{Patient 9 (S.H.) Sound-pressure correlates of the AES as recorded from a 16-year-old boy with minimal congenital aortic stenosis. An aortic root ejection sound (ARES) is present coincident with the onset of pressure rise in the aortic root. An aortic valvular ejection sound (AVES) is also present, occurring 40 msec later and coincident with the anacrotic notch. Note the lack of significant shudder in the remainder of the upstroke of the aortic root pressure (see text).}
motion correlates of the mitral opening snap has its exact counterpart when compared with these correlates of the valvular aortic ejection sound as described in this study. Such obvious analogies have only recently been recognized because of much controversy in the past centering around timing of the pressure, sound, and motion events. With the advent of sophisticated pressure, sound, and motion recording instruments, these measurements can now be made with great accuracy and have led to a unifying concept of the mechanism of cardiovascular sound production.

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Sound-Pressure Correlates of the Aortic Ejection Sound: An Intracardiac Sound Study

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