Echocardiographic and Hemodynamic Relationships of Ejection Sounds

PETER G. MILLS, B.M., M.R.C.P., BRUCE BRODIE, M.D., LAMBERT MCLAURIN, M.D., STEWART SCHALL, M.D., AND ERNEST CRAIGE, M.D.

SUMMARY The physiologic correlates of ejection sounds have been studied by simultaneous phonocardiograms, echocardiograms and high fidelity pressure tracings. Ejection sounds associated with semilunar valve stenosis or hypertension of the systemic or pulmonary circulation occur at the moment of complete opening of the aortic or pulmonary valve recorded echocardiographically. The start of opening of these valves occurs at the onset of the pressure rise in the corresponding great vessel and completion of valve opening always occurs on the pressure upstroke. The ejection sound in the presence of stenotic valves occurs with checking of the opening motion of the thickened valve cusps. Although the hypertensive ejection sounds also occur at the precise moment of full opening of the valve it remains to be seen whether this relationship is causal or coincidental.

EJECTION SOUNDS are a well recognized clinical sign in patients with noncalcific aortic and pulmonary valve stenosis1-2 and there is good evidence to suggest that these sounds are caused by the checking of the opening movement of the thickened semilunar valve.3-4 Ejection sounds also occur in a variety of situations in which the semilunar valves are not stenotic. In such conditions, for example pulmonary hypertension5 or truncus arteriosus,6 the common feature of a dilated great vessel has led to the use of the term "root sounds" to describe the associated ejection sounds.7 Important studies using intracardiac phonocardiography and high fidelity pressure recordings have indicated that these sounds occur slightly before valve ejection sounds8 suggesting that the two categories of ejection sound may have differing origins. Combined echo and phonocardiographic studies, however, have shown that in aortic and pulmonary valve stenosis, truncus arteriosus and pulmonary hypertension, the ejection sounds all occur at the moment of full opening of the corresponding semilunar valve.9-11 Conflicting with the concept that root sounds occur earlier than valve ejection sounds. In an attempt to reconcile these differences, the present study was designed to examine the relationship between ejection sounds, high fidelity pressure recordings, and echocardiographic semilunar valve motion.

The existence and nature of an ejection sound in systemic hypertension is poorly documented and we have therefore performed standard echophonocardiograms on a group of patients with severe systemic hypertension. Further noninvasive studies were also undertaken to clarify the relationship between ejection sounds, semilunar valve opening and tricuspid valve opening, employing in some instances a technique for simultaneously recording two cardiac valves.10 The results will be presented in two categories: A) studies, during cardiac catheterization, of relationships between echocardiographic, phonocardiographic, and high fidelity pressure events; B) studies in which certain echo and phonocardiographic relationships are investigated without high fidelity pressure recordings.

Patients and Methods

Patients

Section A — Combined Cardiac Catheterization/Echocardiographic Studies

All patients selected for this portion of the investigation had been scheduled for routine diagnostic cardiac catheterization and informed consent was obtained from each. Patients were studied during cardiac catheterization only if prior routine echocardiographic studies had shown technically excellent tracings of semilunar valve motion.

Group 1: Patients with Normal Semilunar Valves and No Ejection Sound. In order to determine the temporal relationship between semilunar valve motion and pressure changes in the aorta and the pulmonary artery, 12 patients without semilunar valve stenosis were studied during cardiac catheterization. In six patients, a high fidelity catheter was positioned in the aortic root, and the aortic valve echocardiogram was recorded simultaneously with the high fidelity pressure. In the other six patients, the high fidelity catheter was placed just distal to the pulmonary valve and the pulmonary valve echocardiogram was recorded together with the pulmonary artery pressure.

Group 2: Patients with Ejection Sounds. Eleven patients in whom an ejection sound was present were studied using simultaneous phonocardiography, echocardiography, and high fidelity pressure recordings. Four of these patients had noncalcific aortic valve stenosis, and three had pulmonary hypertension. Pertinent clinical details are contained in table 1.

Section B — Echophonocardiographic Observation of Ejection Sounds by Noninvasive Techniques

1) Fifteen patients with poorly controlled hypertension were studied by combined echophonocardiography, particular attention being paid to tricuspid valve closure and aortic valve opening. The intent was to investigate the possible presence of ejection sounds in patients with severe hypertension, rather than define the incidence of such sounds in the hypertensive population.

2) Owing to the delay in the onset of right ventricular systole compared with left ventricular systole, the interval between tricuspid valve closure and aortic valve opening may be so short as to lead to difficulty in analyzing relationships between sounds and valve motion. We have therefore
studied ejection sounds using simultaneous dual echophonocardiography. Technically satisfactory tracings have been obtained in three patients.

Methods

Recordings were made on an Irex Multichannel Recorder during routine diagnostic cardiac catheterization. All pressures were obtained from a high fidelity micromanometer tipped catheter (Millar Mikrotip). There is a delay in propagation of the pressure pulse of 5 m/sec (2 msec/cm) within the great vessels.19 Care was therefore taken to position the catheter tip approximately 2 cm distal to the semilunar valve. To obtain maximum definition of the relatively short time intervals being studied, a recording speed of 200 mm/sec (1 mm = 5 msec) was employed. This necessitated amplification of the echocardiographic signal to maintain definition of valve motion. All patients were evaluated by standard echophonocardiographic methods6 on the day prior to catheterization to ensure that: 1) the particular valve under study would subsequently be rapidly and accurately located, and 2) its anatomical relationships were defined by the appropriate echocardiographic scans.

Phonocardiograms were recorded at high frequency filter setting with Leatham microphones appropriately located to record the particular sound under study. Echocardiograms were recorded using a Smith Kline Ekoline ultrasonoscope with a 2.25 MHz transducer interfaced to the pediatric amplifier of an Irex Multichannel recorder. The delay in the registration of the echocardiographic signals is approximately 0.1 msec (1/50 mm at the recording speed employed in this study). The technique of dual echocardiography involves the simultaneous registration of echocardiographic signals from two different cardiac structures. Two ultrasonoscopes are interfaced with a single recorder. The technical details have been described previously.22

Results

Section A

Group 1: Relationship between Semilunar Valve Motion and the Rise of Pressure in the Corresponding Great Vessel

Twelve patients with normal semilunar valves were studied with combined high fidelity pressure recordings and echocardiography.

Aortic Valve and Aortic Root Pressure. The initial separation of the aortic valve cusps, recorded echocardiographically, occurs at the time that the diastolic descent of aortic pressure stops (fig. 1). Following this, there is a gradual rise in the aortic pressure over approximately 10 msec and this is mirrored in the gradual initial opening phase of aortic valve motion. Complete opening of the aortic valve always occurs after the onset and during the upstroke of the pressure curve in the aortic root.

Pulmonary Valve and Pulmonary Artery Pressure. Initial separation of the pulmonary valve cusps is rarely seen echocardiographically, and therefore it is difficult to precisely define this point in relation to pressure changes in the pulmonary artery. A typical record is shown in figure 2A. In patients in sinus rhythm, following the "a" dip in the pulmonary valve echocardiogram a gradual posterior motion is seen. This is followed by a more rapid opening mo-
FIGURE 2. Relationship between pulmonary valve opening and pulmonary artery (PA) pressure in two patients with normal pulmonary valves. Panel A shows that initial pulmonary valve opening (Pvoi) is not generally recorded. Following the 'a' dip there is a gradual motion of the visualized cusp up the pulmonary artery, and this is followed by a more rapid opening motion. Complete pulmonary valve opening (Pvoc) occurs on the pressure upstroke. In Panel B from a different patient, the moment of pulmonary cusp separation (Pvoi) was recorded, and this coincides with the start of the pulmonary artery (PA) pressure upstroke. PVE = pulmonary valve echo. Recording speed at 200 mm/sec. Time lines 10 msec.

posterior cusp was seen to coincide with separation of the two cusps.

Group 2: Relationship between the Ejection Sound, the Pressure Pulse, and Valve Motion Recorded Echocardiographically

In each of the eleven patients with ejection sounds, the moment of complete semilunar valve opening occurred during the pressure upstroke - a relationship between pressure change and valve motion similar to that found in the patients with ejection sounds.

In two patients with systemic hypertension and three patients with pulmonary hypertension the ejection sound occurred at complete opening of the respective semilunar valve (fig. 3), these points falling on the respective upstrokes of the pulmonary artery and aortic pressure curves. The hypertensive ejection sounds therefore occurred after the initial rise of pressure in the corresponding great vessel. In four patients with aortic valve stenosis and two patients with pulmonary valve stenosis, the onset of the ejection sound coincided with complete opening of the aortic and pulmonary valves (figs. 4 and 5) and occurred on the pressure upstroke.

Section B. Noninvasive Studies of Ejection Sounds

1) Echophonocardiographic Relationships of Systemic Hypertensive Ejection Sounds

Using conventional echophonocardiography, we have studied 15 patients with severe hypertension. In eight patients, a high frequency sound whose onset coincided with
complete aortic valve opening was recorded. In four patients this sound was of a similar amplitude to the first heart sound at the aortic area, and in the other four it was softer than $S_1$ and consequently difficult to perceive on auscultation. In contrast to the ejection sound seen in aortic stenosis, the aortic hypertensive sound was invariably loudest at the aortic area and was recorded at the cardiac apex in only one patient (fig. 6).

2) Simultaneous Dual Echophonocardiography

The pressure/valve motion relationships studied in Section A cannot take account of atrioventricular valvular events, in particular tricuspid valve closure (TVC). Since TVC has been shown to be a potential cause for a high frequency sound, simultaneous dual echophonocardiography was used to assess critically the temporal relationship between semilunar and tricuspid valve motion in the genesis of ejection sounds. Three patients with ejection sounds have been studied. In aortic stenosis, systemic hypertension and pulmonary hypertension, closure of the tricuspid valve was shown to precede the ejection sound. As in the patients in Section A, the onset of the ejection sound coincided with complete opening of the corresponding semilunar valve (fig. 7) and was separate from TVC.

Discussion

Early studies of the genesis of heart sounds related the phonocardiogram either to valve motion as determined angiographically or to intracardiac and great vessel pressures obtained through fluid-filled catheters. There are serious limitations in using these techniques for the accurate delineation of the rapid sequence of events occurring during early systole. The introduction of high fidelity pressure recordings in this field was an important technical advance because of the elimination of transmission delays of uncertain magnitude. However this technique can only examine valve motion indirectly, and so to clarify further the relationship between ejection sounds, pressure changes and aortic or pulmonary valve motion, we have combined phonocardiography, echocardiographic registration of semilunar valve motion, and high fidelity pressure recordings from the great vessels.

The present study shows that aortic cusp separation occurs as the aortic pressure ceases falling and begins its relatively slow initial rise. The moment of full cusp opening occurs at a time when the pressure upstroke is well established. A similar relationship exists between pulmonary valve motion and pulmonary artery pressure, although in this situation the exact moment of initial cusp separation is more difficult to document. In patients with ejection sounds, these relationships are maintained, and the ejection sound always occurs at the time of complete valve opening. Furthermore, whether associated with semilunar valve stenosis or systemic or pulmonary hypertension, the ejection sound occurs during the pressure upstroke and not at the initial rise of pressure. These observations confirm previous reports regarding ejection sounds of valvular origin, but are at variance with the concept that ejection sounds in systemic or pulmonary hypertension, "root sounds," occur at the onset of pressure rise in the great vessel.

![Figure 4](http://circ.ahajournals.org/)

**Figure 4.** Simultaneous aortic root pressure and aortic valve echocardiogram in a patient with valvular aortic stenosis. The start of the aortic ejection sound (Ao Ej X) coincides with the abrupt checking of the opening movement of the anterior aortic cusp. Both events occur on the pressure upstroke. The onset of the second heart sound (A2) is simultaneous with the incisura and aortic valve closure. The paper speed is 200 mm/sec and the time lines are omitted. PCG MA = phonocardiogram at mitral area.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Pulmonary artery (PA) pressure and opening of the pulmonary valve in a patient with pulmonary valve stenosis. The start of pulmonary ejection sound (P Ej X) coincides with full opening of the pulmonary valve (Pvoc) and occurs on the pressure upstroke.
A possible explanation for this difference may lie in the potential for tricuspid valve closure to cause a high frequency sound. In patients with normal intraventricular conduction, tricuspid valve closure occurs close to both the initial aortic valve opening (figs. 7 and 8) and the onset of pressure rise in the aortic root. Consequently, it would be difficult to be certain of the origin of a sound occurring at this time, particularly if no reliable indicator of tricuspid valve closure such as echocardiography is available. It seems possible therefore that high frequency sounds thought to be caused by a pressure change in the aortic root may in fact have been due to tricuspid valve closure. In the present study simultaneous dual echocardiography has allowed us to separate the possible roles of tricuspid valve closure and semilunar valve opening in patients with hypertensive ejection sounds, demonstrating that these sounds occur at complete opening of the pulmonary or aortic valve and after tricuspid valve closure.

The phonocardiographic characteristics of the ejection sound associated with systemic hypertension are similar to those of pulmonary hypertensive ejection sounds. Hypertensive sounds tend to be poorly transmitted from the aortic and pulmonary areas, and in this respect they differ from ejection sounds associated with aortic valve stenosis. The noninvasive studies show that in systemic hypertension, the ejection sound occurs at the moment of complete aortic valve opening, and may be regarded as an aortic hypertensive ejection sound. It has been suggested that these sounds are an accentuation of one of the normal components of the first sound. Low frequency vibrations occurring after the two high frequency components of the first heart sound are sometimes encountered in routine echocardiographic studies. A striking example of such a sound is shown in figure 8. The noise clearly occurs during aortic valve opening and continues well after tricuspid valve closure. Although this may represent the aortic component of the first heart sound, it seems doubtful that these coarse vibrations could be the basis of the discrete high frequency aortic hypertensive ejection sound shown in figure 6.

The circumstances surrounding the genesis of pulmonary and aortic ejection sounds present a striking analogy with mitral valve opening snaps. In each instance, a high frequency sound is typically associated with noncalcified but stenotic cusps or leaflets, and occurs at the moment of full opening excursion of the valve. However, a sound coinciding with valve opening also may occur when the valve structure appears to be normal and the relationship of onset of the sound to complete opening of the valve is maintained.

Two conclusions may be drawn from the present study: 1) hypertensive ejection sounds have the same temporal relationship to valve motion as do ejection sounds caused by structurally abnormal valves; 2) hypertensive ejection sounds are not related to either the onset of pressure rise or the achievement of peak pressure in the root of the aorta or

---

**Figure 6.** Standard echophonocardiogram of the aortic valve (AVE) in a patient with an ejection sound (Ao Ht X) associated with severe systemic hypertension. As in aortic valve stenosis, the onset of the ejection sound coincides with complete aortic valve opening. In contrast to the ejection sound seen in aortic valve stenosis, the aortic hypertensive sound is relatively soft at the mitral area compared with the first heart sound.

**Figure 7.** Dual echophonocardiogram of the tricuspid (TVE) (upper portion of tracing) and aortic valve (AVE) (lower portion of tracing) in a patient with a stenotic aortic valve. The aortic valve has been recorded from the cardiac apex and consequently its motion resembles that of the pulmonary valve. The cusps are visible during systole, suggesting the presence of a doming valve. The start of the aortic ejection sound occurs at AVC, approximately 30 msec after tricuspid valve closure (TVC dotted line) excluding this event as a possible origin of the sound.
pulmonary artery, but rather occur during the rise in pressure in the respective great vessel.

The results do not settle the precise mechanism of the hypertensive ejection sound. The sounds occur at the moment of full opening of the semilunar valve and therefore probably originate from the valve cusps themselves. However other possibilities not excluded by the present study include tension of the anulus of the semilunar valve and distension of the vessel wall, and further studies will be required to determine which of these mechanisms is responsible for the genesis of hypertensive ejection sounds.

Acknowledgment

We are grateful to the technical staff of the C. V. Richardson Cardiac Catheterization Laboratory for the versatility they exhibited in their contribution to these studies, and to Ms. Betty Horton in the Department of Medical Illustrations for her consistently excellent work. We should also like to acknowledge the secretarial assistance of Mrs. Emmy Woodall and the technical assistance of the staff of the Cardiac Graphics Laboratory.

References

Limitations of the Echocardiogram in Diagnosing Valvular Vegetations in Patients with Mitral Valve Prolapse

P. A. N. CHANDRARATNA, M.D., M.R.C.P., AND E. LANGEVIN, D.O.

SUMMARY In order to assess the reliability of the echocardiogram in detecting valvular vegetations in patients with mitral valve prolapse (MVP), echocardiograms from 85 consecutive patients with mitral valve prolapse were reviewed. Eleven patients had thick shaggy echoes confined to the anterior mitral leaflet; eighteen patients had shaggy echoes on the posterior leaflet; and five had abnormal echoes on both the anterior and posterior leaflets. Only one patient had clinical evidence of infective endocarditis. Redundant leaflets which present multiple surfaces for the production of echoes may explain the abnormal echoes that were observed. Patients with echographic features suggesting mild prolapse less commonly exhibited shaggy leaflet echoes than those with more severe prolapse. Because a significant proportion (40%) of patients with MVP had shaggy echoes which closely resembled those seen in valvular vegetations, we feel that the echocardiogram is of limited value in diagnosing infective endocarditis in patients with mitral valve prolapse.

RECENT STUDIES have indicated that mitral valve prolapse is a common entity. The role of echocardiography in diagnosing mitral valve prolapse is firmly established.

Infective endocarditis of the mitral valve has been shown to produce shaggy echoes on the valve leaflets. Several reports have indicated the susceptibility of patients with mitral prolapse to infective endocarditis. Allen and co-workers followed 62 cases with late systolic murmurs over a mean period of 13.8 years during which five patients developed bacterial endocarditis. Popp observed that 20 to 30% of patients with isolated mitral valve endocarditis have echographic, angiographic or pathologic evidence of prolapse. Since echocardiography is useful in the evaluation of mitral valve prolapse (MVP) and valvular vegetations, it should be of value in the diagnosis of infective endocarditis in patients with MVP. This paper points out the limitations of diagnostic ultrasound in the detection of valvular vegetations in subjects with MVP.

Materials and Methods

A retrospective analysis of the echocardiograms of 85 consecutive patients with mitral valve prolapse was performed. The group consisted of 46 females and 39 males whose ages ranged from nine to 78 years (mean 37 years). The patients were examined in the supine position with a 2.25 MHz, 0.5 inch, 10 cm focus transducer, an Ekoline 20 ultrasonoscope and a Honeywell 1856 recorder. The echocardiograms were performed from the interspace from which the mitral valve could be visualized by perpendicular or near perpendicular placement of the transducer. Care was taken to avoid false positive diagnosis of prolapse due to inferior angulation of the transducer. Optimal damping was used to obtain sharp echoes from the mitral leaflets. A sweep from the aortic root to the left ventricle was made in each instance. The extent of valve prolapse was determined by measuring the vertical distance from the point of maximal prolapse to the CD segment of the mitral valve. The echocardiograms were examined independently by two observers for the presence of thick shaggy echoes on the mitral valve leaflets in diastole. Multiple echoes noted only during systole were not considered abnormal. Multiple, discrete, linear echoes in diastole or mild thickening of the leaflets also were not construed as abnormal.

Results

All the patients had either the classical mid-systolic or holosystolic variety of mitral valve prolapse. Thirty-four patients were observed to have clearly abnormal diastolic echoes. Eleven patients had thick shaggy echoes limited to the anterior mitral leaflet; eighteen were noted to have shaggy echoes on the posterior mitral leaflet; and five patients had abnormal echoes on both leaflets. There was disagreement between the two observers in six cases; these records therefore were not considered abnormal. Mild mitral valve prolapse (< 5 mm) was observed in 38 patients, seven of whom (18%) had shaggy echoes on the leaflets.

From the Division of Cardiology, The University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma.
Address for reprints: Dr. P. A. N. Chandraratna, Division of Cardiology, The University of Oklahoma Health Sciences Center, P.O. Box 20901, Oklahoma City, Oklahoma 73190.
Received February 10, 1977; revision accepted April 15, 1977.
Echocardiographic and hemodynamic relationships of ejection sounds.
P G Mills, B Brodie, L McLaurin, S Schall and E Craige

Circulation. 1977;56:430-436
doi: 10.1161/01.CIR.56.3.430
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/56/3/430

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/