ventricular mass estimated by standard measurements from the echocardiogram.

Only 48% of the patients with hypertrophy had a thickened left ventricular posterior wall. Others have shown that 75% of patients with left ventricular hypertrophy have thickening of the posterior left ventricular wall. Their conclusion was that the primary echocardiographic correlate of left ventricular hypertrophy was an increase in both septal and left ventricular posterior wall thickness. The significance of our study is that a greater percentage of patients with left ventricular hypertrophy can be identified by echocardiography if an estimation of left ventricular mass is used. The primary echocardiographic correlate of left ventricular hypertrophy is not an increase of wall thickness, but rather an increase of estimated left ventricular mass.

In summary, the data from this study show that left ventricular mass, estimated from the echocardiogram, is a better indicator of left ventricular hypertrophy than echocardiographic measurements of left ventricular posterior wall thickness and interventricular septal thickness.

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Echocardiographic Differentiation of Partial and Complete Atrioventricular Canal

JOHN L. BASS, M.D., F. BLANTON BESSINGER, JR., M.D., AND CHERYL LAWRENCE

SUMMARY Retrospective examination of echocardiograms was performed in 34 patients with persistent atrioventricular (A-V) canal who had undergone cardiac catheterization. Characteristic findings in 16 patients with partial A-V canal were lack of continuity of mitral and tricuspid valves, paradoxical interventricular septal motion, definite E and A waves of the mitral valve anterior leaflet (MVAL) echoes and late systolic anterior motion of the mitral valve. Eighteen patients with persistent A-V canal had an interventricular communication and were classified as having the complete form. They characteristically had continuous mitral and tricuspid valves, normal interventricular septal motion, disorganized MVAL echoes, and normal systolic mitral valve motion.

Combining these findings allowed differentiation of 31 of the 34 patients (91%) as having partial or complete A-V canal. Determining the presence of a VSD from loss of interventricular septal echoes was unsatisfactory. The severity of mitral insufficiency could not be estimated from echocardiograms of our patients with partial A-V canal.

PERSISTENT ATROVENTRICULAR (A-V) CANAL represents a broad anatomic spectrum including primum atrial septal defect (ASD), deficiency of septal leaflet of the tricuspid valve and anterior leaflet of the mitral valve, and deficiency of the interventricular septum. Before echocardiography, noninvasive differentiation of the structures involved depended on findings on physical examination, electrocardiogram and chest X-ray. Estimation of mitral insufficiency, degree of deficiency of interventricular septum, and ventricular level shunts has required cardiac catheterization and angiography. Complete and accurate noninvasive evaluation of patients with persistent A-V canal defects may decrease patient morbidity and expense by improving the accuracy of preatherosclerosis diagnosis, the

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Timing of cardiac catheterization, and the estimation of prognosis.

Echocardiographic examination of patients with partial A-V canal was first described by Diamond et al. who found paradoxical interventricular septal motion and increased diastolic right ventricular internal dimension (RVID). This has been attributed to right ventricular volume overload. Others also found narrowing of the left ventricular outflow tract (LVOT) and prolonged diastolic anterograde diastolic echocardiographic motion of the mitral valve anterior leaflet (MVAL) echoes with echoes of the interventricular septum (IVS). This was attributed to a deficiency of the IVS and abnormal position of the mitral valve rather than LVOT obstruction. Multiple systolic echoes and fragmented diastolic echoes of the mitral valve have been reported. Late systolic anterior motion of the mitral valve has been seen. The tricuspid valve echoes in some patients appeared to originate from within the echoes of the IVS.

Differences have been reported in the echocardiograms of patients with complete atrioventricular canal. The descriptions include echoes from the MVAL “crossing” the IVS and being continuous with those of the tricuspid valve. Loss of IVS echoes on scanning from the left ventricle to the aorta has been interpreted as indicating a ventricular septal defect in these patients. IVS motion was found to be normal instead of paradoxical. However, echocardiographic findings have not been consistent enough to allow differentiation between partial and complete atrioventricular canal. In a recent large series reviewed by Hagler, there was paradoxical septal motion in only 42% of his patients with partial A-V canal, and the mitral valve crossed the IVS in 54%. His patients with complete A-V canal were identified primarily on the basis of an absence of large segments of ventricular septal echoes.

To determine if partial A-V canal could be distinguished from complete A-V canal by echocardiogram alone, we retrospectively reviewed all of our patients with persistent A-V canal who had undergone cardiac catheterization. The left ventricular volume overload in patients with partial A-V canal and mitral insufficiency has been felt to abolish paradoxical IVS motion. We evaluated our patients with partial A-V canal to determine if mitral insufficiency affected interventricular septal motion.

Materials and Methods

The records of thirty-four patients at the University of Minnesota Hospitals with persistent A-V canal who had undergone echocardiography immediately before or after cardiac catheterization were reviewed. Patients with associated transposition of the great arteries, double outlet right ventricle or single ventricle were excluded. Ages ranged from six days to sixty years. Diagnoses were assessed by standard hemodynamic and angiographic techniques. If no VSD was present on angiogram, the cardiac defect was classified as a partial A-V canal. If a VSD was present, the cardiac defect was classified as a complete A-V canal.

Standard echocardiographic techniques were employed and transducer selection and gain settings were appropriate for patient size. In most patients, movement of the transducer laterally from the left sternal border resulted in more complete echoes of the mitral valve.

Echocardiograms were examined to assess continuity of mitral and tricuspid valves, IVS motion, motion and configuration of the mitral valve, presence of a VSD, diastolic thickness of IVS and left ventricular posterior wall (LVPW), LVOT diameter, diastolic RVID, aortic root (Ao) and left atrial (LA) diameters. Continuity of mitral and tricuspid valves was assessed on a scan from mitral valve to tricuspid valve across the IVS at the level of the LVOT (fig. 1). When the valves were not continuous, separate openings could be seen when both were recorded simultaneously (fig. 2). If both valves were not recorded simultaneously, timing of opening or closure of the two valves was compared on

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Scan from tricuspid valve to mitral valve in a patient with complete atrioventricular canal. Continuity of mitral and tricuspid valves is demonstrated. TV = tricuspid valve; MV = mitral valve; IVS = interventricular septum.
similar R-R intervals. Motion of the IVS was determined only when recording the left ventricle below mitral valve level. Diastolic configuration of the MVAL echoes was “normal” when definite E and A waves were seen (fig. 3) and abnormal when echoes were disorganized (fig. 4). The mitral valve posterior leaflet (MVPL) echoes were said to have abnormal diastolic configuration when the A wave was more posterior than the E wave (figs. 3 and 4). Late systolic anterior motion of the mitral valve was assessed at low mitral valve level (figs. 3 and 4). This differs from systolic anterior motion of the mitral valve seen in hypertrophic obstructive cardiomyopathy since the D point does not return to the baseline. Two areas were examined for the presence of VSD. One was on a scan from the LV at mitral valve level to the Ao. The other was at the point at which the mitral and tricuspid valves could be recorded simultaneously with the IVS between. A VSD has been said to be present when there is a loss of IVS echoes. The LVOT was measured from C point of the MVAL echoes to the posterior IVS echoes at a level where the left atrial wall was recorded posteriorly (fig. 5). Diastolic RVID could be measured when RV endocardium was recorded. In some patients in whom RV endocardium was not recorded, a minimum diameter was determined from the RV echo-free space. Diastolic thickness of the IVS and LVPW, and aortic root and left atrial diameters were obtained at standard points.

Angiograms were reviewed to assess mitral insufficiency and the presence of VSD. Comparison of left and right ventricular pressures was made and the presence of right-to-left shunting noted.

Results

Sixteen patients were classified by angiography as having a partial A-V canal, and eighteen as having a complete A-V canal.
Partial Atrioventricular Canal

The echocardiographic findings of the 16 patients with partial A-V canal are summarized in figure 6 and listed in table 1. Cardiac catheterization findings are listed in table 2. In 13 of the 16 patients, recordings were adequate to assess continuity of mitral and tricuspid valve openings. All 13 had separate openings or timing differences in openings, and the A-V valves were felt not to be continuous. Closure times were different in the five patients in whom they could be compared. Paradoxical IVS motion was consistently recorded in 12 of the 15 patients with adequate echocardiograms. Two of the other three patients had normal IVS motion on some recordings and paradoxical motion on others. The other patient was the only one with a partial A-V canal and systemic pressure in the right ventricle. The MVAL echoes were recorded in 14 patients and were "normal" in 13. The posterior leaflet echoes of the mitral valve were abnormal in 13 of the 14 patients in whom they were recorded. There was late systolic anterior motion of the mitral valve in 12 of the 16 patients.

Six of the left ventricle to aortic root scans in patients with partial A-V canal were adequate for evaluation and none showed loss of IVS echoes. In eight of the partial A-V canal patients, mitral and tricuspid valves were recorded simultaneously with IVS between, and six appeared to have small areas of loss of IVS echoes. One patient had no loss of IVS echoes on the left ventricle to aortic root scan, but did have loss of IVS echoes on simultaneous recording of mitral and tricuspid valves.

The ratio of IVS to LVPW diastolic thickness in the 13 patients in whom both could be measured was 1.2 ± 0.27 (mean ± SEM). The LVOT could be measured in nine patients. There are no normal values for LVOT in children. In an attempt to standardize this measurement, a ratio of LVOT to Ao was compared. In 26 additional patients without a persistent A-V canal, six patients with an isolated secundum ASD and 20 patients with an isolated VSD, the ratio was 1.0 ± 0.02 (range 0.90 to 1.2). None of the patients had evidence of LVOT obstruction. The nine

![Figure 6](http://circ.ahajournals.org/)

**Figure 6. Summary of echocardiographic findings in patients with partial and complete atrioventricular canal defects.**
partial A-V canal patients had a ratio of 0.78 ± 0.06 (range 0.50 to 1.0). In 10 of the 16 patients, diastolic RVID could be assessed, and was increased in all. All 16 patients had diastolic apposition of the MVAL to the IVS.

Complete Atrioventricular Canal

The echocardiographic findings of the 18 patients with complete A-V canal are summarized in figure 6 and listed in table 3. Cardiac catheterization findings are listed in table 4. The mitral and tricuspid valves were continuous with no timing differences in 14 patients. IVS motion could be evaluated in 10 of these 14 and was normal in eight. One of the patients without normal septal motion had marked tricuspid insufficiency. The other patient had indeterminate IVS motion and there were no hemodynamic or angiographic findings that separated him from the other patients. The MVAL echoes were abnormal in 10 of the 13 patients in whom they were adequately recorded. Systolic motion of the mitral valve could be evaluated in 13 and was normal in all. Thirteen of the 14 patients with mitral-tricuspid continuity had a ratio of right ventricular peak systolic pressure to left ventricular peak systolic pressure of 0.70 or greater.

Four of the 18 patients with complete A-V canal did not have continuity between mitral and tricuspid valves. Three of the four had normal IVS motion and the fourth was indeterminate. The MVAL echoes were normal in two and abnormal in two. All had normal systolic motion of the mitral valve. The ratio of RV peak systolic pressure to systemic peak systolic pressure was less than 0.70 in one patient.

The mitral valve posterior leaflet echoes were recorded in 16 of the 18 patients with complete A-V canal, and were abnormal in 14. One of the six adequate left ventricle to aortic root scans showed a loss of IVS echoes. Sixteen of the patients with complete A-V canal had adequate simultaneous recordings of mitral and tricuspid valves and 15 appeared to have loss of IVS echoes. Two of these areas of loss were small. Four patients did not have loss of IVS echoes on the left ventricle to aortic root scan but did when mitral and tricuspid valves were recorded simultaneously. The ratio of diastolic thickness of IVS to LVWP could be measured in 12 patients and was 2.2 ± 0.47. The LVOT could be measured in 12 of the 18 patients. The LVOT/Ao ratio was 0.69 ± 0.06 (range 0.45 to 1.1). Diastolic RVID was recorded in seven patients and was increased in six.

Discussion

Certain features were common to most of our patients with persistent A-V canal defects. These were abnormalities of the mitral valve including diastolic apposition of the MVAL echoes to the IVS (34 of 34), multiple systolic echoes (34 of 34), abnormal MVPL echoes (27 of 34), and narrowing of the LVOT (14 of 20). Classifying patients as having partial or complete A-V canal by echocardiogram could be accomplished in almost all patients by using other factors. Findings characteristic of partial A-V canal were lack of continuity between mitral and tricuspid valves, paradoxical IVS motion, “normal” MVAL echoes and late systolic anterior motion of the mitral valve. Findings suggesting complete A-V canal were continuity of mitral and tricuspid...
### Table 3. Echocardiographic Findings in Eighteen Patients with Complete Atrioventricular Canal

<table>
<thead>
<tr>
<th>Patient</th>
<th>LVPW</th>
<th>LVS</th>
<th>RVID</th>
<th>LVOT</th>
<th>Ao</th>
<th>MV continuity</th>
<th>Abnormal MV echo</th>
<th>Late systolic anterior motion of MV</th>
<th>IVS motion</th>
<th>VSD (L/V ratio)</th>
<th>VSD (M/V ratio)</th>
<th>BSA (m²)</th>
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*Estimated.

The LVPW/LVS ratio was much higher than that of the pressure ratio of the patient's LV to RV pressure. The restrictive VSD was more common in patients with complete AV canal than with partial AV canal. The IVS/LVPW ratio was higher in patients with partial AV canal than in the patients with complete AV canal. The IVS/LVPW ratio in patients with complete AV canal was higher than in patients with partial AV canal. The IVS/LVPW ratio in patients with complete AV canal was higher than in patients with partial AV canal.
The range of LVOT/Ao ratios was the same for patients with partial and complete A-V canal. While 14 of these ratios were less than those for patients with secundum ASD or VSD, the recording position may have been further from the left sternal border in patients with persistent A-V canal and the values may not have been comparable.

Volume overload of the left ventricle from mitral insufficiency has been used to explain the absence of paradoxical IVS motion in some series of patients with partial A-V canal. Hagler found normal IVS motion in 58% of 24 patients with a persistent A-V canal defect without a VSD. However, all of our patients with partial A-V canal, with the exception of one patient with systemic right-sided pressures, had paradoxical IVS motion. Many of our patients had trace to 2+ mitral insufficiency, but this is usually underestimated on angiography because of dilution by increased pulmonary venous return. Two of our patients were documented to have severe mitral insufficiency. The lower incidence of paradoxical IVS motion in Hagler’s series may be partly explained by the high right-sided pressures (RV/LV peak systolic pressure ratio greater than 0.70 in five of his patients) and the inclusion of one patient with d-transposition of the great arteries.

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