ARTICLE

Fetal Tricuspid Valve Size and Growth as Predictors of Outcome in Pulmonary Atresia With Intact Ventricular Septum

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ABSTRACT

OBJECTIVE. Pulmonary atresia with intact ventricular septum is a complex congenital cardiovascular anomaly that frequently requires single ventricle palliation. Fetal diagnosis of pulmonary atresia with intact ventricular septum is common, but the natural history of pulmonary atresia with intact ventricular septum diagnosed in midgestation, predictors of neonatal anatomy, and predictors of biventricular repair have not been determined. The objective of this study was to determine whether the size and rate of growth of the fetal tricuspid valve predict neonatal anatomy and biventricular repair.

DESIGN AND RESULTS. Twenty-three fetuses diagnosed with pulmonary atresia with intact ventricular septum between 1990 and 2004 were studied. Of 13 fetuses with a midgestation fetal tricuspid valve z score ≤ -3, 1 achieved biventricular repair, compared with 5 of 5 with a tricuspid valve z score > -3. Of 13 fetuses with a midgestation fetal tricuspid valve z score ≤ -3, 8 were diagnosed postnatally with a right ventricular dependent coronary circulation, compared with none with a tricuspid valve z score > -3. Midgestation and late gestation fetal tricuspid valve z scores correlated with neonatal tricuspid valve z score. The average rate of tricuspid valve growth between mid- and late fetal echocardiograms was significantly lower in patients who did not achieve biventricular repair than in those who did (0.012 ± 0.008 cm per week vs 0.028 ± 0.014 cm per week).

CONCLUSIONS. Fetal tricuspid valve z score and rate of growth predict postnatal outcome in pulmonary atresia with intact ventricular septum. These findings may have important implications for prenatal counseling and selection of patients for fetal pulmonary valve dilation.

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Key Words
echocardiography, congenital heart defects, pulmonary heart disease, pulmonary atresia

Abbreviations
PA/IVS—pulmonary atresia with intact ventricular septum
RV—right ventricle
RVDCC—right ventricule-dependent coronary circulation
TV—tricuspid valve
EGA— estimated gestational age

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Pulmonary atresia with intact ventricular septum (PA/IVS) is a morphologically heterogeneous lesion characterized by variable right ventricle (RV) hypoplasia and abnormal communications between the RV and coronary arteries, with an RV-dependent coronary circulation (RVDCC) in the most severe cases. Single ventricle palliation is required in a substantial proportion of patients.[1–6] Current neonatal management strategies begin with establishment of flow through the hypoplastic RV, with the aim of promoting RV growth and achieving an RV of sufficient size and function to support the pulmonary circulation. Several previous reports suggest that a small tricuspid valve (TV) and the presence of RVDCC in the neonate are predictors of failure to achieve a biventricular circulation.[1,3,4] Given the increasing prenatal detection rate of PA/IVS, the complexity of this defect, and the range of possible outcomes, it becomes important to be able to predict in utero which patients will ultimately achieve a biventricular repair. In addition, with the emerging prospect of in utero valvuloplasty[7,8] for PA/IVS, prediction of postnatal outcome at the time of in utero diagnosis may facilitate appropriate patient selection for fetal intervention. The primary aim of this study was to determine whether TV size and the rate of TV growth in midgestation fetuses with PA/IVS are indicators of the ultimate capacity of the right heart to support a biventricular circulation. The secondary purpose of this study was to determine whether fetal TV size and rate of growth can predict TV size at birth and/or the presence of an RVDCC. We hypothesized that a fetal TV *z* score of ≤ -3, along with slow in utero TV growth, will predict failure to achieve a biventricular repair, small TV at birth, and presence of RVDCC.

METHODS

Patient Selection

The echocardiography database was queried for all fetal studies with a diagnostic code for PA/IVS. Selection was restricted to 1990–2004, because the neonatal management of PA/IVS at our center was consistent during this time period. Live-born fetuses that subsequently had all postnatal interventions (catheter-based and surgical) at Children's Hospital were selected for full analysis. Fetuses that did not go on to live birth and fetuses in which a fetal intervention was attempted were used only for the initial demographic description and were excluded from further analysis. Fetuses with associated Ebstein's anomaly of the TV were excluded, because there was no consistent management strategy for this subset of patients during the study time period. The study was approved by the institutional review committee at Children's Hospital Boston.

Echocardiographic Data

Two-dimensional echocardiograms were reviewed on all of the fetuses and neonates. Archived analog videotapes were digitized into a database using a commercially available product (EchoTrace, Marcus Laboratories, Boston, MA). Studies completed after 2003 were reviewed in their original digital format. The TV was imaged in all of the fetal studies, and annulus size was felt to be a reproducible value without technical limitations. A single fetal echocardiographer (W. T.) blinded to patient outcome measured the TV annulus diameter from the 4-chamber view in all of the fetal echocardiograms. The TV was measured between the hinge points of the TV leaflets with the valve open in diastole. The annulus diameter was expressed as a *z* score to adjust for the estimated gestational age ([EGA] estimated from standard femur length and head circumference measurements) of the fetus, based on unpublished normative data from our institution derived from fetuses without heart disease according to the following formulas: TVp (mean TV diameter for gestational age) = $0.04071 * \text{EGA} - 0.33017$ TVsd (standard deviation of the regression) = $0.00417 * (\text{EGA} - 29.83000) + (0.010976 * \{1.0128 + [(\text{EGA} - 29.83)^2]/2735\})^{0.5}$. The same blinded reviewer measured TV annulus diameter in the 4-chamber view of the first neonatal echocardiogram. Annulus diameters measured on fetal and neonatal echocardiograms were expressed as *z* scores to adjust for body surface area using the formula: TV *z* score = TVz = (TV − TVp)/TVsd.

Figure 1 demonstrates the normative mean TV annulus size in millimeters measured across various times in gestation. *z* scores are derived using the SD around the mean. Other institutions have used similar algorithms for quantitative analysis of fetal cardiac structures.[9] Fetal TV *z* scores were dichotomized into ≤ -3 and > -3 groups based on the findings of Hanley et al[3] and the natural division of patients in our series. To account for variability in EGA at the time of fetal echocardiogram, studies for each fetus were grouped into 1 of 2 windows based on the EGA at the time of the study. The midgestation window was defined as 20 to 29 weeks' EGA. The late gestation window was defined as 30 to 39 weeks' EGA. If 2 echocardiograms were completed within 1 window, measurements from the earlier EGA were used for analysis.

The rate of fetal TV growth was calculated by subtracting the TV annulus diameter (cm) at the first fetal echo from the TV annulus diameter (cm) at the final fetal echo and dividing this value by the number of weeks between these 2 studies. This number was expressed as the rate of fetal TV growth in centimeters per week.

Catheterization, Coronary Angiography, and Management Strategy

The standard practice at our institution is to document coronary anatomy and RV size by angiography in the first days of life. The coronary angiographic technique and criteria for diagnosis of RVDCC for patients with

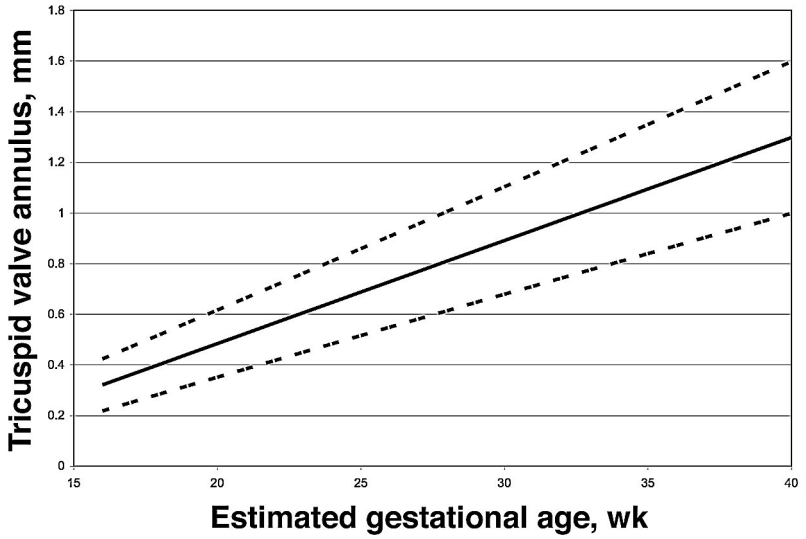

FIGURE 1
Normative fetal TV annulus size by EGA. This graph plots normative TV annulus size (millimeters) against EGA (weeks). —, mean TV annulus across gestation; ----, SD of + 2 and −2.

PA/IVS have been described previously.[10,11] A coronary artery was considered RV dependent if it demonstrated a communicating RV fistula and it was atretic, stenotic, or interrupted proximal to the RV to coronary artery communication, which could lead to ischemia if the RV was to be decompressed. RVDCC was defined as coronary circulation in which there is atresia of 1 or both coronary ostia or obstruction within >1 major coronary artery (right, left main, left anterior descending, circumflex, and/or posterior descending coronary artery). At Children's Hospital Boston, the initial management strategy for newborns with PA/IVS is based on the presence or absence of RVDCC and rarely takes into account RV or TV size. When RVDCC is documented by coronary angiography, a systemic-to-pulmonary artery shunt is placed with the goal of single ventricle palliation. In neonates with non-RVDCC, RV decompression is attempted based on the size of the RV and TV. This is accomplished either by transcatheter balloon valvotomy or surgical RV outflow tract augmentation with or without systemic-to-pulmonary artery shunt. Cardiac catheterization is then performed at 6 months of age to test the adequacy of the TV and RV. The systemic-to-pulmonary artery shunt and interatrial communication may be closed at that time, assuming the RV is able to support the pulmonary circulation. A bidirectional superior cavopulmonary anastomosis is performed in patients in whom the RV is unable to support the pulmonary circulation, and then the cardiac catheterization may be repeated at 1 to 3 years of age to determine suitability for a "one and one-half ventricle" repair or Fontan operation.

Outcomes

Outcomes assessed included achievement of a biventricular repair, TV size at birth, and the presence of RVDCC. A biventricular repair was defined as an RV supporting the pulmonary circulation, a systemic arterial saturation of >90%, and either no interatrial communication or a left-to-right shunt at any remaining atrial communication. Patients with a cavopulmonary shunt (with or without a patent RV outflow tract) were considered not to have a biventricular circulation.

Data Analysis

Comparison of continuous and categorical data between or within groups was performed using the appropriate parametric or nonparametric tests for comparing unpaired or paired samples, respectively. Because definitive biventricular repair is often a staged process in patients with PA/IVS, achievement of biventricular circulation is a time-dependent variable and, therefore, was assessed both as a discrete outcome and by assessment of time to biventricular repair. The time to achievement of biventricular repair was determined by review of catheterization and echocardiographic and pulse oximetry data, using the Kaplan-Meier method, with comparison between groups using the log-rank test. For Kaplan-Meier analysis, patients were censored at the time of death, at the time of definitive single ventricle palliation (ie, Fontan completion, bidirectional Glenn without a patent pulmonary outflow tract), at the time of commitment to a single ventricle management strategy (ie, at the time of diagnosis of RVDCC, an anatomic criteria that is considered at our center to preclude biventricular repair), or at the time of most recent follow-up if a definitive biventricular circulation had not been achieved but remained a possibility (ie, systemic-to-pulmonary shunt or bidirectional Glenn with a patent pulmonary outflow tract). Continuous data are presented as mean ± SD.

RESULTS

Demographics and Diagnostic Data

Between 1990 and 2004, 36 fetuses were diagnosed with PA/IVS, resulting in 25 live births. Of the 11 fetuses not

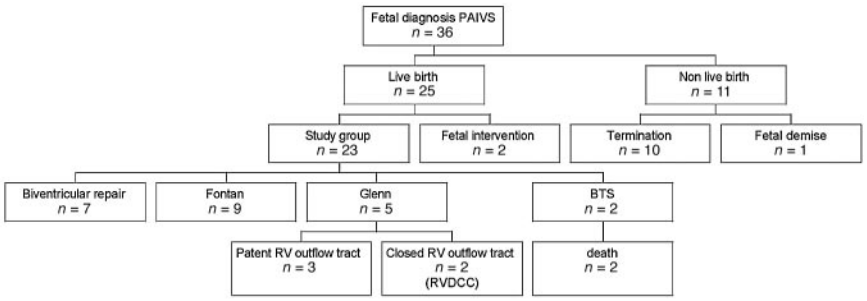

FIGURE 2
Fetal PA/IVS demographics and postnatal outcomes, 1990–2004. The study group contained 23 patients. Of the 5 Glenn shunts, 2 patients have RVDCC and, thus, are expected to continue along a single ventricle pathway. Two deaths occurred, both after BTS, in neonates with severe RV hypoplasia and RVDCC. BTS indicates Blalock-Taussig shunt.

carried to term, there were 10 elective terminations and 1 fetal demise. Of 25 liveborn fetuses, 2 underwent fetal interventional procedures and were not included in this study. Thus, the study group included 23 patients. (Fig 2) The EGA at diagnosis for the original 36 fetuses was 23.4 ± 5.6 weeks. Fetuses not carried to term were diagnosed significantly earlier in gestation than those that were (20.4 ± 3.1 vs 24.5 ± 5.7 weeks; $P = .04$).

Within the study group, the median number of fetal echocardiograms was 2 (range: 1–3). For fetuses with >1 study ($n = 18$), the mean age at the final fetal echocardiogram was 30.6 ± 3.6 weeks' EGA. The mean duration of time from first fetal echo to last fetal echo was 9.8 ± 3.2 weeks. Eighteen patients had ≥1 study in midgestation, and 20 patients had ≥1 study in late gestation. Fifteen patients had an echocardiogram in both the mid- and late gestational age windows.

Outcomes

Among 23 live born patients, there were 7 with a biventricular circulation and 16 with a single ventricle palliation. Twenty one were alive at a median follow-up of 5.4 years (10 months to 13 years), and the 2 deaths occurred in neonates with severe RV hypoplasia and RVDCC who died after a systemic-to-pulmonary arterial shunt. Ten patients were diagnosed postnatally with RVDCC. One patient had coarctation of the aorta requiring surgical repair, and none had significant extracardiac abnormalities. Among the 14 living patients with a single ventricle circulation, 9 had undergone a Fontan procedure, and 5 had a bidirectional superior cavopulmonary anastomosis. Of the 5 patients with a bidirectional cavopulmonary anastomosis, 2 had documented RVDCC and were awaiting a Fontan operation, and the other 3 had a patent RV outflow tract and an open atrial septum at most recent follow-up (Fig 2). Fetuses with TV z score ≤−3 on the midgestation echocardiogram were significantly less likely to undergo biventricular repair ($P = .001$) than patients with a z score >−3 and significantly more likely to be diagnosed with RVDCC ($P = .036$). Fetuses with a TV z score ≤−3 on the late gestation echocardiogram were similarly less likely to undergo biventricular repair ($P < .001$) and more likely to have an RVDCC ($P = .051$) than patients with a z score >−3. In both the mid- and late gestation groups, a TV z score ≤−3 suggested decreased freedom from biventricular repair ($P = .059$ and $P = .014$, respectively).

In utero TV growth was significantly slower in patients who ultimately required single ventricle palliation ($P = .006$) and in patients who developed RVDCC ($P = .033$; Table 1) than in those who did not. Among the 15 patients with measurements in both mid- and late gestation windows, there was a distinct separation between biventricular and single ventricle outcome groups when tracking TV growth from mid- to late gestation (Fig 3). Both mid- ($R^2 = 0.48$) and late ($R^2 = 0.74$) gestation TV z scores correlated with neonatal TV z scores (Fig 4).

DISCUSSION

Among fetuses diagnosed with PA/IVS at our center since 1990, both the fetal TV z score and the in utero rate of TV growth were associated with postnatal outcome. Fetuses with a TV z score ≤−3 on either mid- or late-gestation fetal echocardiograms were significantly less likely to achieve a biventricular repair than those with a

TABLE 1 **Biventricular Versus Single Ventricle Outcome and RVDCC Versus Non-RVDCC in Mid- and Late Gestation by TV *z* Score Group**

Variable	Biventricular Repair	Single Ventricle	*P*	RVDCC	Non-RVDCC	*P*
Mid-TV *z* score						
≤−3 (n = 13)	1	12	.001	8	5	.036
>−3 (n = 5)	5	0		0	5	
Late TV *z* score						
≤−3 (n = 14)	1	13	<.001	7	7	.051
>−3 (n = 6)	6	0		0	6	
Rate TV growth, cm/wk	0.028 ± 0.014	0.012 ± 0.008	.006	0.011 ± 0.008	0.023 ± 0.013	.033

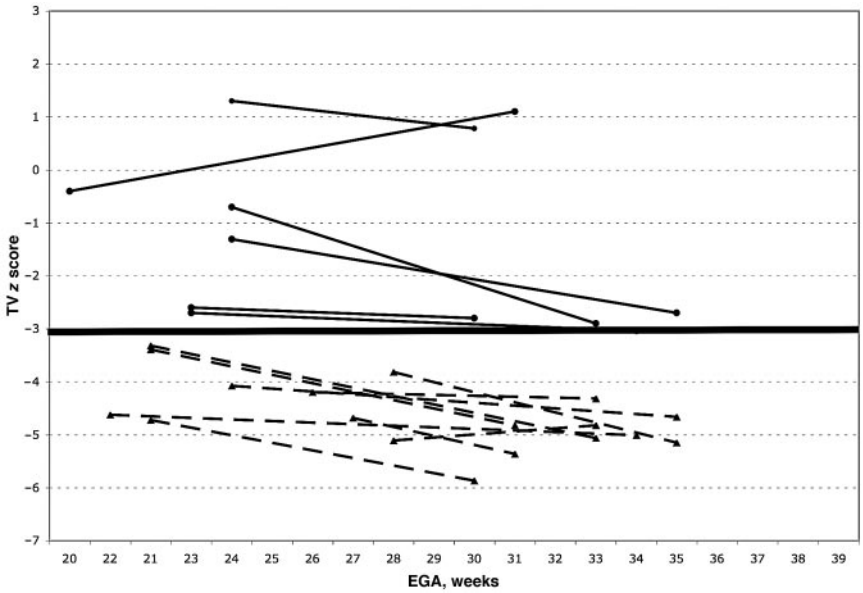

FIGURE 3
TV *z* score at mid- and late fetal echo as an indicator of biventricular repair. ●, TV *z* score for patients who achieved biventricular repair; — connects *z* score measurements between the mid- and late fetal echocardiogram. ▲, TV *z* score for patients who failed to achieve a biventricular repair; ---- connects *z* score measurements between the mid- and late fetal echocardiogram. —, *z* score of −3. Fetuses that did not have a fetal echocardiogram in both the mid- and late gestational age windows are excluded from this graph.

TV *z* score >−3 and were also more likely to have a RVDCC. In contrast, all of the fetuses with a TV *z* score >−3 on either the mid- or late fetal echocardiogram achieved a biventricular repair. A fetal TV *z* score ≤−3 on the midgestational fetal echocardiogram was associated with postnatal diagnosis of RVDCC, and none of the fetuses with a TV *z* score >−3 on mid- or late gestation fetal echocardiograms had RVDCC. In addition, the rate of TV growth in utero was more than twice as slow in the single ventricle patients than in those who achieved a biventricular repair or were born with a non-RVDCC. These findings may have an important impact on the prenatal and postnatal management of the fetus with PA/IVS. The frequency of fetal diagnosis of congenital heart disease is rising steadily, with screening practices affecting worldwide prevalence and types of congenital heart disease, including PA/IVS.[12] At our institution, the frequency of fetal diagnosis of PA/IVS has followed this trend, with prenatal detection rising from 40% of all PA/IVS births between 1986 and 2000 to >65% after 2000. With this increase in prenatal diagnosis, prognostic data may be helpful for prenatal decision-making and perinatal planning. Several studies have correlated neonatal echocardiographic features with angiographic evidence of RVDCC. For example, Garcia et al[13] concluded that abnormal neonatal coronary flow patterns are suggestive of fistulas; Giglia et al[11] suggested that RV volume and TV *z* scores are significantly smaller in neonates with RVDCC; and Satou et al[14] demonstrated that a neonatal TV *z* score of ≤−2.5 has an 80% positive predictive value to predict RVDCC. Despite the use of neonatal echocardiography for predicting the presence of RVDCC, correlating fetal data are lacking. Fetal echocardiographic features of ventriculocoronary connections have been described[15] but may not be predictive of true RVDCC. Fetal indicators of RVDCC are important for 2 primary reasons. First, at our institution, coronary artery anatomy is the primary determinant of postnatal management. In newborns with PA/IVS, decompression of the RV is thought to be essential in promoting growth of the RV and achievement of biventricular repair. Although RV fistulas are common in patients with PA/IVS[3,16] and not considered a contraindica-

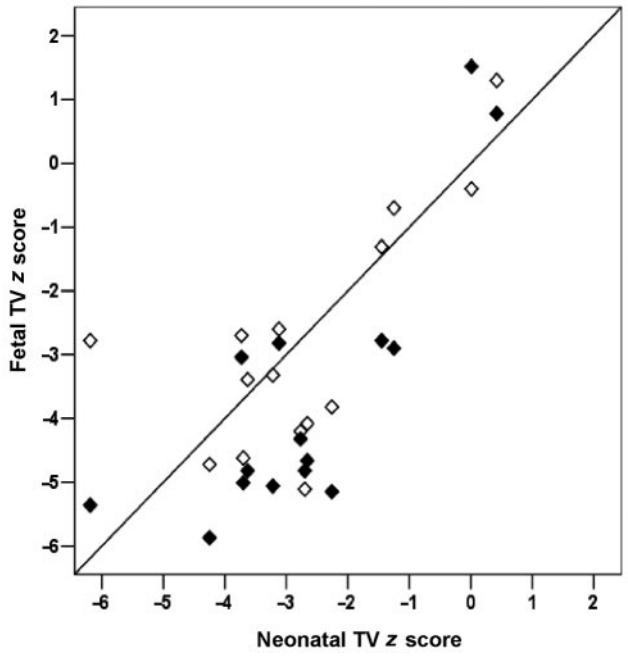

FIGURE 4
Scatterplots demonstrating relationship between early (◇) and late (◆) window fetal TV *z* scores and neonatal TV *z* scores. /, unity. By linear regression, the relationship between early fetal and neonatal TV *z* scores is described by the equation: Neonatal TV *z* score = (0.61 × Early fetal RV *z* score) − 0.97 (R^2 = 0.48). The relationship between late fetal and neonatal TV *z* scores is described by the equation: Neonatal TV *z* score = (0.70 × Late fetal RV *z* score) − 0.28 (R^2 = 0.74).

tion to RV decompression, the presence of RVDCC is a contraindication.[10] At our institution, coronary anatomy is documented in all neonates by angiography. If there is evidence of RVDCC, there is no subsequent attempt to decompress the RV, and the neonate progresses toward staged single ventricle palliation. Second, among patients with PA/IVS, outcomes are worse in those with an RVDCC than in those without. Intermediate follow-up of patients with an RVDCC demonstrated 83% 5-year survival, with the surviving patients at ongoing risk for ischemia.[17] Prediction of an RVDCC in midgestation may aid in prenatal counseling.

PA/IVS is a heterogeneous disease, and not all institutions base their initial management strategy on the presence of RVDCC. Recent reports have demonstrated a tiered grading system for RV hypoplasia, which is predictive of failure to achieve a biventricular repair.[18] Others describe specific RV morphology in an attempt to predict the need for single ventricle palliation.[19] Although our data are derived from the management strategy consistently used at a single institution over the past 15 years, the strong correlations between fetal TV *z* score and both neonatal TV size (a surrogate for RV size) and the presence of RVDCC are likely to be useful regardless of the approach to the management of PA/IVS. In addition, the use of serial fetal echocardiograms to document the rate of in utero growth may aid in parental counseling at centers that rely on RV size as a criterion for palliation. The ability to predict which midgestation fetuses with PA/IVS are likely to have RVDCC and/or to require single ventricle palliation may aid in appropriate patient selection for advanced fetal therapy. There have been several reports of balloon dilation of the pulmonary valve in the fetus, which is based on the theory that decompression of the hypoplastic and hypertensive RV in utero will promote antegrade flow across the TV and PV, thus encouraging RV growth and preventing the development of an RVDCC.[7,8,20] Although much more experience is necessary to substantiate the feasibility and efficacy of fetal balloon pulmonary valvuloplasty, appropriate fetal selection and timing of this procedure may ultimately improve the ability of a fetus with PA/IVS to achieve a biventricular repair postnatally. Additional anatomic factors will almost certainly influence the success of fetal balloon pulmonary valvuloplasty, and careful evaluation of fetal TV size and growth by serial echocardiography may be the first step toward optimal patient selection for fetal intervention.

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