Magnetic resonance imaging in patients with congenital heart disease

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ABSTRACT  Magnetic resonance imaging (MRI) was conducted with use of the spin-echo technique (0.35 Tesla) in 22 patients with a variety of congenital and cardiovascular anomalies and in 16 normal volunteers. Electrocardiographic (ECG) synchronization of the data acquisition produced transverse, parasagittal, and coronal tomograms that were used to define size and relationship of the great vessels and internal cardiac structures. MRI findings were corroborated by angiography and sector-scan echocardiography. In most patients the diagnosis had been established before the MRI study. MRI detected all of 11 abnormalities at the level of the great vessels, all of six atrial septal abnormalities, and 10 of 11 ventricular septal defects. Images of poor quality resulting from patient motion were obtained in the one instance in which a small ventricular septal defect was not imaged. Of two patients with Ebstein's anomaly, the displacement of the tricuspid leaflets was shown in one patient but was not evident in another. Complex anomalies such as double-outlet right ventricle, uncorrected T-transposition, single atrioventricular valve, single ventricle, and common ventricle were clearly shown by MRI. Initial experience with MRI has indicated the effectiveness of this technique for defining great vessel and internal cardiac anatomy in patients with congenital heart disease. This is accomplished without the use of contrast media and is thus a completely noninvasive technique for cardiovascular diagnosis.


GATED magnetic resonance imaging (MRI) is a completely noninvasive technique for evaluating cardiovascular anatomy.1-3 A recent study has reported its use for defining congenital heart disease.4 In our early experience, we have used gated MRI to examine the central cardiovascular structures from the great vessel level to the liver in patients with congenital heart disease. Segmental anatomy was described by these multiple tomographic images and the results were corroborated by those of cardiac angiography and two-dimensional echocardiography.

Methods

Study population. The study was conducted in 22 patients ranging in age from 2 months to 75 years. There were 12 female patients. The diagnosis had been established by cardiac catheterization and angiography in 21 patients before they underwent MRI (table 1). Twenty-one patients also underwent two-dimensional echocardiographic examination. In one patient in whom the only anomaly was persistent left superior vena cava no other cardiac imaging studies were performed; the diagnosis was suspected from the chest x-ray. The images from 16 normal volunteers (age range 29 to 38 years) were also examined in the study. The studies were performed between March 1983 and March 1984. The youngest patients (ages 3 months to 2½ years) were sedated with 5 mg/kg im pentobarbital sodium at 20 to 30 min before imaging. Normal breathing patterns ensued during the imaging procedure.

Magnetic resonance imager and imaging techniques. The magnetic resonance imager for this study uses a superconducting magnet operating at a field strength of 3.5 kG and a resonance frequency for hydrogen of 15 MHz. This imager has been described in detail previously.2 A multiple-plane selective irradiation technique was used for data acquisition and sectional (plane) images were reconstructed by the two-dimensional Fourier transform technique. The reconstruction matrix was 128 horizontal pixels and was displayed in 256 gray levels, with the brightest area representing the tissues with the greatest magnetic resonance signal intensity. Spatial resolution was 1.6 mm.

The imaging sequence was spin echo at echo delay times (TE) of 28 and 56 msec. The repetition rate (TR), or interval between sets of radiofrequency pulses, was determined by each subject's heart rate and the decision by the operator of whether to initiate a sequence for every heartbeat or for alternating heartbeats. In all subjects images gated to every heartbeat were obtained.

Data for each sectional image were acquired during 512 cycles. Total imaging time varied from 5.1 to 8.5 min; this was determined by the product of the R-R interval of the electrocardiogram, the number of lines along the y (vertical) axis of the reconstruction matrix, and the number of times the signal was averaged (four in the current study). Multisectional imaging was accomplished by sequentially irradiating five adjacent 7.0 mm thick tissue sections (z axis) at 100 msec intervals during each TR. Thus, the time required to apply each pulse sequence
at all five sections was approximately 500 msec. For most patients the heart rate was such that the TR was 600 to 1000 msec, which easily accommodated the time necessary for imaging five separate sections.

Since three multislice series were obtained in each patient, the total imaging time for each patient was approximately 30 to 45 min.

Electrocardiographic (ECG) gating technique. An ECG signal was obtained with use of leads designed for minimal noise generation on the images when used in the presence of high magnetic fields and rapidly changing radiofrequency pulses. This was achieved by transmitting the ECG signal from a preamplifier via fiber optics to the main amplifier and trigger circuits located outside the Faraday cage enclosing the imager. The ECG signal was the input signal to the MRI triggering module, which then activated the initiation of imaging sequences. An operator-controlled time delay between the input signal and activation of the imaging sequence was available. This gating unit has been described in detail previously.

With the multislice imaging technique used for gated MRI in the current study, only one of the five tomographic images conformed to end-diastole. This image corresponded to one initiated with the R wave of the ECG. Each tomographic image was offset in time by 100 msec from the adjacent image.

Image analysis. Ten or 15 adjacent transverse tomographic images were obtained in all subjects; these images encompassed the heart from the level of the great vessels to the apex and usually included the cranial portion of the liver. Five adjacent sagittal images were obtained in eight patients and five coronal images were obtained in three. Images were analyzed to determine the presence and location of structural abnormalities, including the relative wall thicknesses and diameters of the two ventricles. The ratio of the diameter of the pulmonary artery to that of the aorta was measured on the transverse slice 1 cm caudal to the origin of the right pulmonary artery.

Results

Normal anatomy. The transverse images at the level of the base of the heart showed normal position and size of the great vessels (figure 1); the base of aorta was located posterior and rightward from the right ventricular outflow tract. In the normal subjects the internal diameter of the aorta and main pulmonary artery were similar. In normal volunteers from 29 to 38 years old the ratio of the diameter of pulmonary artery to that of the ascending aorta was 0.98 (range 0.9 to 1.1). The diameter of the pulmonary artery was greater than the aortic diameter in only one subject. Sagittal images also showed the origin of the aorta and pulmonary arteries from their respective ventricles (figure 2).

Transverse images through the atria and ventricles demonstrated the atrial and ventricular septa (figure 1). In some normal subjects there was a very faint signal from the central portion of the atrial septum (figure 1); presumably this represents the region of the fossa ovalis. In these instances there was gradual thinning of the atrial septum both anterior and posterior to this region of signal "dropout." The inflow and outflow portions of the ventricular septum were spatially distinct from each other on the transverse images (figure 1). Likewise, the junction of the muscular and membranous portions of the septum was usually identified and the thin septum shared by the right atrium and left ventri-

![FIGURE 1. Gated images at multiple transverse levels from the level of the great vessels (top left) to the level of the liver (bottom right). The pulmonary artery is positioned anterior and leftward from the aorta (curved arrows). Transverse image at the left ventricular outflow region shows the outflow (anterior) portion (open arrows) of the ventricular septum while the image through the region of the mitral valve demonstrates the inflow (posterior) portion (closed arrow). Note the atrioventricular septum separating the left ventricle and the right atrium (small arrow). There is decreased signal intensity in the thinned fossa ovalis region of the atrial septum.](http://circ.ahajournals.org/doi/figure/1)
cle (atrioventricular septum) was also distinct on the transverse images. Coronal and sagittal images also showed various portions of the ventricular septum, but spatial separation and identification of distinctive regions of the septa were clearer on the transverse images.

The series of transverse images extending from the base of the heart to the superior aspect of the liver defined the type of ventricular loop, the relationship of the atria to the ventricles, the relationship of atria to visceral situs, and the atrial connections of the systemic and pulmonary veins (figure 1).

On the other hand, normal cardiac valves were not very well imaged with MRI. The semilunar valves, particularly the pulmonic valve, were not demonstrated with sufficient consistency or precision to exclude the possibility of anomalies of these structures. The pulmonic valve was not identified in any normal subject.

Anatomy of cardiovascular anomalies. Transverse images have in most instances provided precise definition of anomalies of the great vessels and cardiac chambers. In our initial experience, parasagittal or coronal images have not displayed abnormalities that could not be identified on the transaxial image. On the other hand, various components of some congenital anomalies were better defined by visualization in an additional orthogonal plane.

The series of transaxial images of the area from base to apex of the heart permitted the description of segmental cardiac anatomy, including great vessel relationships and abnormalities, the type of ventricular loop and ventricular abnormalities, the visceralatrial relationship, and atrial abnormalities. Tables 1 and 2 list the congenital cardiac abnormalities demonstrated with MRI and those shown by two-dimensional echocardiography and cardiac angiography for each individual patient.

Great vessel relationships and abnormalities. All 10 abnormalities of the great vessels in our patient population were demonstrated by MRI. Transverse images demonstrated several positional abnormalities of the great vessels, including anterior and rightward position of the aorta in a patient with D-transposition, anterior and leftward position of the aorta in a patient with L-transposition of the great vessels (figure 2), and side-by-side position of great vessels in a patient with double-outlet right ventricle (figure 3). A single large vessel arising from the base of the heart was imaged in a patient with truncus arteriosus, type I (figure 4).

The disparity in size of the great vessels, which is a characteristic component of some cardiac anomalies, was evident as an enlarged ascending aorta and small pulmonary artery in a patient with tetralogy of Fallot (figure 5). Likewise, the main and central pulmonary arteries were substantially larger than the aorta in patients with Eisenmenger’s syndrome (figure 2).

The sagittal images clearly demonstrated the origin of the aorta from the right ventricle and that of the pulmonary artery from the left ventricle in patients with transposition of the great vessels. A series of sagittal images also showed truncus arteriosus straddling the two ventricles above a large ventricular septal defect (figure 4) and the origin of the pulmonary artery

FIGURE 2. Transverse images at level of great vessels (top) and ventricles (bottom) in a male patient with uncorrected L-transposition. The aorta (a) is located anterior and leftward from the pulmonary artery (p). Note the large ventricular septal defect. The pulmonary artery is markedly enlarged and there is equivalent thickness of the two ventricles. The image on the bottom shows that the liver and morphologic right ventricle (r) are right-sided and at this level the descending aorta has shifted to the right. Note also the entrance of the inferior vena cava into the right-sided atrium.
from the truncus (figure 4). The sagittal images also revealed the small pulmonary anulus of a patient with tetralogy of Fallot (figure 5) and the diminutive posteriorly positioned pulmonary artery of a patient with transposition and single ventricle.

**Ventricular abnormalities.** Ten of 11 ventricular septal defects in our patient group were demonstrated by MRI, although in one patient results of cardiac catheterization were not definitive with respect to whether the left-to-right shunt was located at the atrial or ventricular level. Defects of both the inflow (posterior) (figure 2) and outflow (anterior) tracts of the septum (figure 5) were apparent on transverse images. Large septal defects located just beneath the aorta (figure 5) and the truncus (figure 4) were identified in patients with tetralogy of Fallot and truncus arteriosus, respectively. There were two patients with small ventricular septal defects, and the defect was identified on the series of transverse images in one patient but not in the other. In the latter patient the images obtained were of poor quality due to patient motion. A rudimentary septum was identified in one patient.

### TABLE 1

**Results of corroborative studies**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age/sex</th>
<th>2D echocardiography</th>
<th>Cardiac catheterization and angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11 yr/F</td>
<td>TGV; single AV valve; common ventricle; IVC connection to LA</td>
<td>d-TGV; valvular PS; common ventricle; common AV valve; common atrium; bilateral SVC; visceral heterotaxy</td>
</tr>
<tr>
<td>2</td>
<td>2 yr/M</td>
<td>d-TGV; VSD; RVH</td>
<td>d-TGV; d-ventricular loop; VSD; ASD; pulmonary arterial hypertension</td>
</tr>
<tr>
<td>3</td>
<td>5 yr/M</td>
<td>VSD; RVH</td>
<td>VSD; ASD; pulmonary arterial hypertension; RAE; RVE</td>
</tr>
<tr>
<td>4</td>
<td>38 yr/M</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>33 yr/F</td>
<td>RVE; RAE; interatrial communication; Ebstein’s anomaly; tricuspid regurgitation</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>51 yr/F</td>
<td>Tetralogy of Fallot with RVH; overriding aorta; VSD; subvalvular PS</td>
<td>Tetralogy of Fallot with RVH; overriding aorta; VSD; subvalvular PS; hypoplastic pulmonary anulus</td>
</tr>
<tr>
<td>7</td>
<td>48 yr/F</td>
<td>Ebstein’s anomaly; marked RAE; small RV; tricuspid regurgitation</td>
<td>Ebstein’s anomaly; possible VSD</td>
</tr>
<tr>
<td>8</td>
<td>30 yr/F</td>
<td>Single ventricle (LV type); TGV with aorta arising from left-sided RV outflow chamber; subvalvular PS</td>
<td>Single ventricle; d-TGV; inverted RV outflow chamber; valvular and subvalvular PS</td>
</tr>
<tr>
<td>9</td>
<td>35 yr/M</td>
<td>RVH (severe); RAE (moderate); ‘probable’ VSD</td>
<td>VSD; pulmonary arterial hypertension (Eisenmenger’s syndrome)</td>
</tr>
<tr>
<td>10</td>
<td>19 yr/F</td>
<td>Truncus arteriosus (type unknown); RVH (moderate)</td>
<td>Truncus arteriosus (type I); VSD; pulmonary arterial hypertension (Eisenmenger’s syndrome)</td>
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<tr>
<td>11</td>
<td>6 yr/F</td>
<td>d-TGV; RVH</td>
<td>d-TGV</td>
</tr>
<tr>
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<td>3½ yr/M</td>
<td>d-TGV; RAE; RVE</td>
<td>d-TGV</td>
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<tr>
<td>13</td>
<td>3 mo/M</td>
<td>d-TGV</td>
<td>d-TGV</td>
</tr>
<tr>
<td>14</td>
<td>33 yr/F</td>
<td>DORV; VSD</td>
<td>DORV; VSD; pulmonary arterial hypertension (Eisenmenger’s syndrome)</td>
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<tr>
<td>15</td>
<td>26 yr/M</td>
<td>VSD (small outflow type)</td>
<td>Small VSD</td>
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<tr>
<td>16</td>
<td>75 yr/F</td>
<td>Atrial septal aneurysm</td>
<td>—</td>
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<tr>
<td>17</td>
<td>36 yr/F</td>
<td>RAE; RVE; RVH; enlarged pulmonary artery; ‘possible’ ASD</td>
<td>ASD; pulmonary arterial hypertension</td>
</tr>
<tr>
<td>18</td>
<td>5 yr/M</td>
<td>LAE; LVE; VSD</td>
<td>Secundum ASD; small VSD</td>
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<tr>
<td>19</td>
<td>43 yr/F</td>
<td>LAE; LVE; ‘probable’ VSD</td>
<td>Small VSD; infundibular PS</td>
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<td>20</td>
<td>40 yr/M</td>
<td>Asymmetric septal hypertrophy</td>
<td>Primum ASD; mitral regurgitation; subaortic stenosis (outflow septal hypertrophy)</td>
</tr>
<tr>
<td>21</td>
<td>39 yr/F</td>
<td>‘Probable’ ASD</td>
<td>Common atrium; Eisenmenger’s syndrome</td>
</tr>
<tr>
<td>22</td>
<td>4 yr/M</td>
<td>Not available</td>
<td>d-TGV; VSD patch; RV to PA conduit</td>
</tr>
</tbody>
</table>

**TVG = transposition of the great vessels; AV = atrioventricular; IVC = inferior vena cava; LA = left atrial (or atrium); VSD = ventricular septal defect; RVH = right ventricular hypertrophy; RVE = right ventricular ectopy; RA = right atrial ectopy; RV = right ventricle (or ventricular); DORV = double-outlet right ventricle; ASD = atrial septal defect; LAE = left atrial ectopy; LVE = left ventricular ectopy; SVC = superior vena cava; PA = pulmonary arterial; PS = pulmonary stenosis.**
with a common ventricle and a hypoplastic inverted right ventricular outflow chamber was apparent on coronal images from another patient with single ventricle.

Dilation and hypertrophy of the ventricles were evident on transverse images and the severity of right ventricular hypertrophy was shown in patients with Eisenmenger’s syndrome, tetralogy of Fallot (figure 5), and transposition of the great vessels (figure 2). Alterations in the contour of the ventricles were also observed in association with some congenital complexes: the normal elliptical contour of the left ventricle was altered by reversal of septal curvature in patients with transposition and intact ventricular septum and in those with Eisenmenger’s complex. The sagittal images were useful in visualizing the right ventricular thickness, infundibular and annular hypoplasia, and hypertrophied trabecular bands crossing the ventricular chamber in a patient with tetralogy of Fallot (figure 5).

Atrialization of the inflow portion of the right ventricle was well defined on transverse and coronal images in one patient with Ebstein’s anomaly (figure 6). Likewise, the displacement of the septal leaflet into the right ventricle was well visualized (figure 6). On the other hand, atrialization of the ventricle and displacement of the tricuspid leaflets were not evident by MRI in another patient with Ebstein’s anomaly. The abnormal findings in this patient were confined to marked right atrial and moderate right ventricular enlargement.

Atrial abnormalities. Transverse images showed defects in the atrial septum in all five patients with this anomaly (figure 7). In each of these patients the cranial portion of the atrial septum was clearly visible and the residual septum adjacent to the defect was visualized distinctly and had substantial magnetic resonance signal intensity (figure 7). This appearance is in contrast to the poor signal intensity of the septum adjacent to the signal dropout of the central portion of the atrial septum in some normal patients. An aneurysm of the atrial septum was identified by MRI in one patient.

Complete absence of the atrial septum was demonstrated on sequential imaging in a patient with common atrium. MRI also showed severe right ventricular hypertrophy consistent with Eisenmenger’s syndrome, which complicated this abnormality. A huge atrial septal defect and common atrioventricular valve were apparent in a patient with the complete form of atrioventricular canal defect. Normal drainage of the pulmonary veins into the left atrium was demonstrated in each of the patients with an atrial septal defect.

Abnormalities of systemic venous drainage were observed in two patients. A persistent left superior vena cava was identified lateral to the pulmonary artery on transverse images. Drainage of the left inferior vena cava into the left-sided (pulmonary venous) atrium was shown on transverse images of the atria and cranial portion of the liver (table 2). A transverse liver was also evident in this patient with visceral heterotaxy.

Discussion

Gated MRI is capable of demonstrating a wide spectrum of congenital cardiovascular abnormalities. Transverse images at the level of the great vessels demonstrated abnormalities in the positions of these
vessels, such as transposition and double-outlet right ventricle. Transverse images at the level of the ventricles defined the dimensions of the ventricular chambers and position of the ventricles relative to each other, as well as abnormalities of the ventricular septum and images at the atrial levels identified visceral-atrial situs, venous connections, atrioventricular valve abnormalities, and atrial septal defects. The data from the transverse images were supplemented by multisectional imaging in the coronal and sagittal planes. However, these latter images infrequently uncovered abnormalities that were not already evident on the multisectional transverse images. Cardiac valves, especially the pulmonic valve, were not completely visualized in most patients. In one instance Ebstein's anomaly was not evident on the magnetic resonance image. Thus, this early experience with MRI in patients with congenital heart disease shows considerable potential for this technique in the noninvasive evaluation of these diseases, but also indicates some limitations of the method at this stage in its development.

It should be emphasized that the current study was not intended to, nor does it, evaluate the clinical efficacy of MRI in relation to two-dimensional echocardiography or other noninvasive cardiovascular imaging techniques. Most of the patients were selected for the current study because of a previous anatomic diagnosis that has been established in all cases by cardiac catheterization and angiography; in most cases a two-dimensional ECG examination had also been performed before the ECG-gated MRI study.

The results of the current study and a recent report by Fletcher et al. indicate that a wide variety of cardiac anomalies can be visualized with this completely noninvasive imaging technique. Gated MRI, like echocardiography, is a completely noninvasive imaging technique, since excellent natural contrast exists between the blood pool and walls of cardiac chambers and walls of blood vessels. For cardiovascular imaging with MRI there is no need for administration of exogenous contrast media.

We used MRI at a magnetic field strength suitable...
for defining the density distribution of protons, i.e., hydrogen, in tissues, and the intensity of the magnetic resonance signal of any focus in the image was modified by magnetic relaxation times of the protons within the tissues. The spin-echo technique used in this study is responsive to both $T_1$ and $T_2$ relaxation times as well as the proton density of the tissues. The MRI technique permits the accentuation of the contribution of these three factors in producing differential contrast among soft tissues by variation in pulse-sequence parameters. For instance, a short interpulse delay (TR value) accentuates $T_1$ differences. Gating to every heartbeat results in a short TR, but this can be increased by gating to every second or every third beat. At any TR value, $T_2$ differences are emphasized on images generated by a long echo delay ($TE = 56$ msec) compared with those generated by the short echo delay ($TE = 28$ msec). However, it should be noted that the gating window is doubled on the images when $TE = 56$ msec is used, and this may result in some degradation in image clarity with the longer $TE$.

The effect of blood flow on magnetic resonance images has been examined in preparations in vitro. Generally, blood flowing in a laminar fashion at normal velocity (10 to 15 cm/sec) produces little or no MRI signal. The loss or reduction in signal intensity depends on the fraction of hydrogen nuclei that are mobile and the velocity of motion. However, with certain imaging parameters, and at the initial slice at which flowing blood encounters the radiofrequency pulse sequence, considerable signal intensity can be observed from flowing blood. Moreover, blood flowing at low velocities can produce considerable MRI signal intensity. An intraluminal signal is present within blood vessels on images gated to the diastolic phase of the cardiac cycle.

Some form of physiologic gating is required to consistently produce sharply defined images of the cardiac structures. The continual motion of the heart results in considerable loss of signal intensity from the myocardial wall. The most clinically useful mode of gating on our initial experience was synchronization of the imaging sequence to the R wave of the ECG. In contradistinction to computed tomographic scans, ECG gating of the pulse sequence does not add to the data acquisition period. The multislice imaging technique is accomplished in a time similar to that required for nongated sequences; a five-slice data acquisition en-

**FIGURE 4.** Transverse and parasagittal images in an adult patient with truncus arteriosus, type I. Transverse images demonstrate a large single vessel (truncus, T) and an outflow type of ventricular septal defect (curved arrow). The sagittal image (bottom) shows the truncus arteriosus (T) straddling the two ventricles and the large ventricular septal defect. The relationship of the right atrium (open arrow) and left atrium (closed arrow) to the truncus are shown on the transverse image. R = right ventricle.
compassing most of the heart requires approximately 5 to 7 min.

There are some potential pitfalls associated with use of MRI in patients with congenital heart disease that became evident during our initial experience, as well as some potential advantages to be exploited in the future. One pitfall is that the midportion of the atrial septum, the site of the fossa ovalis, is thin and consequently signal intensity is very low in this area. As with two-dimensional echocardiography, the loss of signal intensity at this site in the atrial septum can be misinterpreted as an atrial septal defect. However, it is likely that the thin fossa ovalis can be distinguished from a septal defect with MRI in most instances. In the normal subject there is gradual thinning and diminution in signal intensity toward the middle of the septum on transverse images, while in the patient with secundum defects there is an abrupt absence of signal surrounded by a septum that is thickened on both sides of the defect. Another limitation of MRI at the present time is that fixed imaging planes (transverse, coronal, sagittal) are required and these are not optimal for evaluation of cardiac valves in a plane perpendicular to the leaflet cusps of cardiac valves. The limitation with regard to visualization of valve components is being addressed at our institution with the development of isotropic volume imaging, which allows the reconstruction of thin (1.8 mm) slices oriented in any plane through the volume from which data has been acquired. This approach also eliminates gaps between

FIGURE 5. Transverse and parasagittal images in an adult patient with tetralogy of Fallot. The image at the level of the great vessels (top left) shows the enlarged ascending aorta (a) and small main pulmonary artery. Likewise the image at the base of the heart (top right) reveals the severe narrowing in the region of the pulmonic anulus (arrow). The defect in the outflow portion of the septum is evident (bottom left). The parasagittal images display a hypertrophied trabecular band in the body of the right ventricle (small arrow) and the severe narrowing of the infundibulum and anulus (curved arrow).
slices into which the valves may unfortuitously fall.

Apparent advantages of MRI compared with other noninvasive imaging techniques are the large field of view, the clear definition of the endocardial interface of cardiac walls, and the exquisite demonstration of the main and central pulmonary arteries. The latter attribute may prove to be very important in the evaluation of patients with pulmonary atresia and certain types of truncus arteriosus.

The eventual role of MRI relative to other imaging modalities for the evaluation of patients with congenital heart disease is unclear at present. The interest, design, and number of patients in the current study are not such that any conclusion can be drawn regarding clinical efficacy of MRI in these patients. It does seem clear that advantages will have to be shown for this modality so that its clinical efficacy may be established in relation to less expensive techniques, such as echocardiography. However, MRI, with further development, offers three intriguing improvements in the assessment of heart disease and possibly the response to therapeutic interventions: direct tissue characterization, noninvasive blood flow measurements, and assessment of myocardial metabolism in vivo.

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