Ascending Aortic and Main Pulmonary Artery Areas Derived From Cardiovascular Magnetic Resonance as Reference Values for Normal Subjects and Repaired Tetralogy of Fallot

Shelby Kutty, MD; Titus Kuehne, MD, PhD; Paul Gribben, BS; Eric Reed, MD; Ling Li, MD, PhD; David A. Danford, MD; Philipp B.J. Beerbaum, MD, PhD; Samir Sarikouch, MD, PhD

Background—Cardiac magnetic resonance (CMR) imaging is an important clinical tool for serial follow-up of patients with congenital heart disease, but normative data for great vessel dimensions in pediatric subjects are scarce. We investigated the ascending aortic (AO) and main pulmonary artery dimensions in normal children and young adults in comparison with a cohort of patients with repaired tetralogy of Fallot (TOF).

Methods and Results—Subjects were prospectively enrolled for cardiac magnetic resonance after a standardized protocol in 14 participating centers of the German Competence Network for Congenital Heart Defects. All studies were performed in 1.5-T scanners and used single-slice multiphase acquisitions steady-state free precession and velocity-encoded cine. AO and main pulmonary artery areas were measured. The cohort consisted of 483 subjects: 105 normal controls (55 men; 50 women; and median age, 14 years) and 378 patients with repaired TOF (210 men; 168 women; and median age, 16 years). Among TOF, 35 (9%) had pulmonary atresia, 98 (26%) had a palliative procedure before repair, the mean age at repair was 2.9 years, and 82 (23%) used a transannular patch repair. Great vessel areas correlated well with body surface area and age in controls and reference Z-score values were derived. Z scores for ascending AO areas were larger in TOF compared with controls (mean Z score =1.95, \( P=0.001 \)). In TOF, pulmonary atresia (\( P=0.003 \)), male sex (\( P=0.01 \)) and previous palliations (\( P=0.046 \)) were associated with larger AO areas. Main pulmonary artery area Z scores in surgically modified TOF were smaller on an average than controls (mean Z score =−0.293 \( P=0.001 \)) but not small to the same extent as the AO was large.

Conclusions—This study provides cardiac magnetic resonance reference Z scores for great vessel areas in normal children and adolescents in comparison with a large contemporary cohort of repaired TOF. Male sex, pulmonary atresia, and previous palliations emerged as predictors for larger AO dimensions in TOF.


Key Words: tetralogy of Fallot ■ adult congenital heart disease ■ great arteries ■ pediatric cardiology ■ cardiovascular magnetic resonance imaging

Aortic root and ascending aortic (AO) dilatation is known to occur in tetralogy of Fallot (TOF) and persists despite resolution of right-to-left shunting after surgical repair.1–3 Moreover, late progressive aortic dilatation has been reported in children and adults with repaired TOF.2–4 Progressive dilatation can be associated with increasingly severe aortic regurgitation, which may necessitate aortic valve or root replacement in a small number of patients with repaired TOF.5,6 Other long-term consequences, including ascending AO dissection, rupture, and AO aneurysm, have also been reported after TOF repair.5–8 Follow-up of AO dimensions and AO valve function after repair of TOF is, therefore, important.

Clinical Perspective on p 651

Cardiac magnetic resonance (CMR) imaging has evolved as an important modality for follow-up evaluation of repaired congenital heart disease (CHD). In particular, CMR is commonly applied for serial clinical surveillance in repaired TOF. CMR is especially useful in this setting for quantifying pulmonary regurgitation and right ventricular volumes and
function. CMR has been demonstrated to be an excellent diagnostic modality for the thoracic aorta and superior to echocardiography in the evaluation of pulmonary artery size and morphology after surgical repair.\(^1\)\(^{11}\) Though steady-state free precession cine- and phase-contrast velocity mapping are key components of CMR imaging that are routinely used for assessment of semilunar valves and great vessels, reference values for great vessel dimensions by CMR are limited\(^1\)\(^2\) and data pertaining to patients with repaired TOF are unavailable.

We have created a large CMR database for children and young adults with repaired TOF, as well as normal controls using standardized contemporary steady-state free precession–cine- and phase-contrast CMR imaging techniques within the German Competence Network for Congenital Heart Defects. We have previously used this database to compute age– and sex–specific pediatric reference norms for ventricular volumes, function, and mass and young adults. Our goals were (1) to report CMR–based great vessel measurements in repaired TOF and compare these with normal values, and (2) to identify clinical factors associated with AO dilatation in TOF.

### Study Population

This was a prospective study consisting of patients with repaired TOF and normal children and adolescents. CMR scans were performed exclusively for research purposes and subjects were enrolled between April 2005 and March 2008. In the TOF group, specific exclusion criteria consisted of additional severe heart defects (eg, atrioventricular sepal defect, presence of major aortopulmonary collateral arteries or TOF-absent pulmonary valve syndrome), as well as non-cardiac conditions, such as malignant or systemic inflammatory diseases. The local institutional review committee of the participating institutions approved the study, and written informed consent was obtained from the parents or legal guardians. Details of surgical repair in TOF patients, including any previous palliative procedures, were recorded. Body weight and height were measured, and the procedure was explained using a scaled-down model of the CMR scanner. All subjects were in sinus rhythm. Body surface area (BSA) was calculated using the Dubois formula: body weight (kg) \(0.425 \times \text{height (cm)} \times 0.007184\).

### CMR Technique

All examinations were performed by 1.5-T whole-body scanners using a standardized protocol of the CMR project of the German Competence Network for Congenital Heart Defects published previously\(^1\)\(^3\)\(^,\)\(^4\) and outlined on the website www.kompetenznetz-ahf.de/en/research/clinical-studies/magnetic-resonance-imaging-mri. A 5-element cardiac phased-array coil was used for signal acquisition, and a vector cardio gram–gated steady-state free precession sequence was applied to cover the whole heart. Further to acquisition of steady-state free precession sequences as detailed elsewhere,\(^5\) quantitative phase-contrast flow measurements were performed in all subjects. Flow measurements were obtained for the MPA with the imaging plane exactly midway between the level of the pulmonary valve and the bifurcation of the branch pulmonary arteries and for the ascending AO 1 to 2 cm distal to the sinotubular junction visualizing the whole diameter of the vessel and perpendicular to the vessel (Figure 1). A standard, retrospectively gated gradient-echo sequence with 30 to 40 heart phases was used corresponding to a temporal resolution ranging between 17 to 25 ms; 1 k-space line per phase; repetition time/echo time/time/flip angle, 15 ms/6.5 ms/30°; velocity-encoded value, 150 to 200 cm/s; number of signal averages, 2; slice thickness, 6 mm; acquisition matrix, 144 \(\times\) 128; acquired spatial resolution, 2.1 \(\times\) 2.1 \(\times\) 6\(\text{mm}\); and through-plane, free-breathing, phase-contrast CMR protocol. The phase-contrast sequence was repeated at least once in each study to enhance precision. The sequences obtained had the best possible spatial resolution and thin-slice thickness, without being compromised by the presence of bone or surgical scarring during image acquisition.

### CMR Analysis

All study data were integrated in a central research database after anonymization for data protection. Analysis was performed offline on a computer workstation using dedicated customized software capable of vendor–independent data handling (Chili GmbH, Heidelberg, Germany).\(^1\)\(^3\)\(^,\)\(^4\) Only the studies with unambiguous phase-contrast image(s) of both great vessels suitable for measurements were selected. Any image with an inappropriate vessel shape or phase wrap artifact or inaccurate contour definition because of partial volume effect was excluded from measurement. The phase–contrast cine CMR image allowed semiautomatic vessel border detection and measurement of the cross-sectional area of the great artery from systolic frames with through-plane velocity encoding perpendicular to the axis of blood flow (Figure 1). The zoom tool on the workstation was used to enhance the pixel-by-pixel accuracy of the measurement. The maximal external diameter (\(d_1\)) was measured in the systolic frame of the magnitude image when the great artery size was the largest, as recommended in the current guidelines on thoracic AO disease.\(^11\) In the identical frame, the vessel diameter perpendicular to \(d_1\) was also measured (\(d_2\)). Radii (\(r_1\) and \(r_2\)) were derived and cross-sectional area calculated using the formula \(\pi r_1 r_2\). A repeat measurement of the area was performed for the same frame and the average of 2 measurements was taken. To assess interobserver variability, a second observer repeated AO and MPA area measurements in 50 randomly chosen studies from the normal control group. The primary observer performed repeated measurements (4 weeks after the first set of measurements) in these 50 studies to assess intraobserver variability.

### Statistics

Mean, SD, median, and ranges were determined for continuous variables. Frequencies were determined for nominal and ordinal variables. Two sample \(t\) tests were used to compare demographic and artery dimensions between the TOF and control group. Ascending AO and MPA area measurements were normalized to age and BSA in all subjects using linear regression, and distributions of the measurements in TOF and in controls were generated for illustrative purposes. The regression models derived for AO and MPA maximal cross-sectional diameters, as well as great vessel area measurements, were similar but goodness of fit, as assessed by \(R^2\), was slightly higher for regressions against BSA than for age (AO area versus BSA 0.674, versus age 0.632; MPA area versus BSA 0.696, versus age 0.612). Therefore, normal standards for AO and MPA were established based on BSA, and these were used to generate Z scores representing the number of SDs above or below the population mean. Inter- and intraobserver variability was determined using Bland–Altman analysis to identify possible bias (mean difference) and the limits of agreement (2 SD of the difference). Nonpaired Student \(t\) test was used to compare great arterial area Z scores across groups with differing demographic, clinical, and anatomic features in TOF. A \(P\) value <0.05 was considered significant. Statistical analyses were performed on Microsoft Excel (Microsoft Inc, Redmond, WA) and Minitab 16.1 (Minitab Inc, State College, PA).

### Results

The entire study cohort consisted of 483 subjects. This included 378 patients with repaired TOF and 105 normal controls. At the time of enrolment for CMR, the TOF group...
consisted of 210 men and 168 women, and mean age 17.6±8.0 years (range, 8.0–59.0) and the control group had 55 men, 50 women, and mean age 13.7±4.0 years (range, 4.4–20.4).

Demographics of TOF subjects and normal controls are shown in Table 1. The mean age at complete repair of TOF was 2.9±3.9 years and mean weight was 11.3±6.6 kg. Among TOF, 35 (9%) had pulmonary atresia, representing an extreme variant of the spectrum of TOF. Of the 378 patients in the TOF group, 280 (74%) had a primary repair and 98 (26%) had 1 or more previous palliations before definite repair. Among those who had previous palliations, 80 (21%) received 1 procedure (shunt), and 18 (5%) had 2 or more palliative procedures performed.

Details of intracardiac repair were available in 360 TOF patients. Of the 360 patients, 198 (55%) patients were repaired without a transannular patch, 82 (22.8%) were repaired by use of a transannular patch, and another 80 (22.2%) received pulmonary augmentation procedures in addition to transannular patch. CMR parameters in the cohort of TOF and normal controls have been published elsewhere.13,14 In brief, the volumetric and functional parameters in the TOF cohort were as follows: mean right ventricular end-diastolic volume, 120.4±32.9 mL/m²; right ventricular ejection fraction, 50.2±9.3%; left ventricular end-diastolic volume, 80.9±17.1 mL/m²; left ventricular ejection fraction, 57.0±8.4%; pulmonary regurgitation fraction, 26.8±18.4%; and AO regurgitation fraction, 2.1±4.8%.

The Bland–Altman analysis showed excellent inter- and intraobserver agreement for both AO and MPA area measurements as represented in Figure 2. Individual variable models derived using age and BSA for AO and MPA in healthy controls demonstrated slight superiority of the model using BSA (Figure 3), and therefore, normal standards for AO and MPA areas were established using BSA. Unlike normal controls, the relation between great arterial areas with age or BSA in TOF was not nearly so strong, with wide variance of AO and MPA area measurements about the regression line (Figure 4). The Z scores for AO areas were significantly larger in TOF compared with normal controls (mean Z score=1.95, SEM=0.153, P=0.001, 1 sample t test) demonstrating abnormally large ascending AO in TOF. The MPA area Z scores in

### Table 1. Characteristics of Normal Controls and the Tetralogy of Fallot Group at the Time of Study

<table>
<thead>
<tr>
<th></th>
<th>Normal Controls</th>
<th>Tetralogy of Fallot Group</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=105</td>
<td>n=378</td>
<td></td>
</tr>
<tr>
<td>Men:Women, n</td>
<td>55:50</td>
<td>210:168</td>
<td>0.563</td>
</tr>
<tr>
<td>Men:Women, %</td>
<td>52.4:47.6</td>
<td>55.6:44.4</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>13.7±4.0</td>
<td>17.6±8.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age at repair, y</td>
<td>...</td>
<td>2.9±3.9</td>
<td>...</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>46.5±20.5</td>
<td>56.1±18.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height, cm</td>
<td>152.6±20.6</td>
<td>162.0±15.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BSA, m²</td>
<td>1.4±0.4</td>
<td>1.6±0.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AO diameter, cm</td>
<td>2.2±0.4</td>
<td>2.9±0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AO area, cm²</td>
<td>4.0±1.4</td>
<td>6.5±2.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>MPA diameter, cm</td>
<td>2.3±0.4</td>
<td>2.5±0.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>MPA area, cm²</td>
<td>4.4±1.6</td>
<td>5.2±2.5</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

BSA indicates body surface area; AO, ascending aorta; and MPA, main pulmonary artery.
TOF were smaller compared with normal controls (mean Z score=-0.293, SEM=0.148, P=0.001, 1 sample t test). In TOF, pulmonary atresia (P=0.003), male sex (P=0.01), and previous palliations (P=0.046) were associated with larger ascending AO areas. None of these features were associated with the differences in MPA dimensions. The type of repair (presence of transannular patch versus others) did not demonstrate an association with either of the great artery areas. Comparison of the effects of sex, previous palliation, pulmonary atresia, and transannular patch on AO and MPA area Z scores are shown in Table 2. In controls, the relationships between AO or MPA sizes and BSA, stratified by sex did not show significant differences (Table 3). Reference percentiles were computed from AO and MPA measurements in normal controls and in repaired TOF subjects to display percentiles of the range of values that can be expected in both groups (Figure 5).

**Discussion**

Clinical surveillance of the rapidly growing population of adolescents and adults with repaired or palliated CHD is often achieved with the aid of serial noninvasive cardiac imaging. With increasing use of CMR for assessment of intracardiac and thoracic vasculature after CHD repair, age and BSA specific normative data are important. The present study represents the largest study, to date, of cross-sectional area measurements of the great vessels in repaired TOF and in normal children and young adults. Importantly, these results were derived from a prospective multicenter setting with strict methodological standardization. The data are derived from a large cohort of children and adults with repaired TOF. At the time of clinical presentation, TOF represents a spectrum of disease ranging from cases in which there is severe disproportion of great arterial root dimensions to those in which that disproportion is less dramatic. This diversity of substrate is likely responsible in part for the broad variation in AO and MPA areas in the TOF population observed in this study, which was not seen in controls.

**Aortic Dilatation in TOF**

Echocardiographers have long recognized the phenomenon of AO dilatation in TOF. For example, Niwa et al observed progressive AO dilatation in ≈15% of the adult patients with repaired TOF. The prevalence of AO dilatation varies depending on the criteria used, with some studies reporting as high as 87%. AO dilatation has been reported to occur more frequently in men and in those with pulmonary atresia and right AO arch. We similarly found male sex and pulmonary atresia to be associated with larger ascending AO areas in TOF. No other variable was found to be associated with increased great vessel area in this report. The TOF cohort in the present study was significantly younger at repair than those in published reports on AO enlargement in TOF.

Others have shown a high prevalence of AO dilatation, increased AO stiffness, and reduced AO strain in children after TOF repair compared with normal controls, linking AO stiffening with progressive dilatation. In that series, only the
duration of follow-up after surgery emerged on multivariate analysis as a determinant of echocardiographic AO annular and sinus dilation. AO regurgitation was present in 12% of the children, and they tended to have larger AO at all levels. Data from Dodds et al demonstrate progression of AO valve dysfunction after repair of TOF and pulmonary atresia-ventricular septal defect. Sixteen patients in that series (12 with pulmonary atresia and 4 with TOF) required AO valve replacement at a median interval of 13.5 years after initial repair. Ongoing analysis, including the 1-year follow-up CMR series of our TOF cohort, may help to clarify aspects of increased AO stiffness seen even in the younger patients.

Reduced AO elasticity and distensibility, along with minor degrees of AO regurgitation and reduced left ventricular systolic function, in young adults after repaired TOF is also evident by CMR. Mohiaddin et al in the first report of AO area measurements based on spin echo CMR images in adults have demonstrated age-related dimensional changes in the thoracic aorta. Histological changes of the media similar to changes seen in patients with Marfan syndrome and bicuspid AO valve have been observed in the dilated AO of TOF. These changes of cystic medial necrosis have been described even in the normal aorta related to increasing age and lead to weakening of the wall with dilatation. It is unknown whether these changes result from volume overload before repair or intrinsic AO abnormalities, or because of a combination of the two. Likely some or all of the above factors play a role in the dilatation of AO after TOF repair and necessitate further investigation. Although there is no TOF specific consensus about indications for AO repair or replacement, some have advocated AO root repair or root/ascending aorta replacement when a rate of increase \( \geq 0.5 \) cm/year or diameter of the AO root/ascending aorta \( >5.5 \) cm or progressive AO regurgitation is observed. These are important considerations in repaired TOF with significant AO dilatation, particularly in the context of another surgical indication, such as pulmonary valve implantation.

There is a strong association between sex and the risk of adverse AO outcomes in adults with major CHD. A large national registry of adult CHD showed an overall 33% higher risk of AO aneurysms and dissections and a 30% higher probability of AO surgery in men as compared with women. In patients with TOF, significant sex differences are known to exist for indexed biventricular volumes, mass, and ejection fractions. Larger ascending AO area Z scores shown in the present study have potential implications for sex-specific AO surveillance in repaired TOF, in that men may require more frequent follow-up than women. The higher prevalence of systemic hypertension at age \(<45\) years in men than women, may be a contributor to the sex-related differences in AO dimensions in TOF.

**Pulmonary Artery Size in TOF**

It is interesting that in surgically repaired TOF the MPA, although smaller on an average than normal, is not small to the same extent that the AO is large. We speculate that the surgical augmentation of the hypoplastic MPA at the time...
of TOF repair may preserve a more normal diameter of that vessel after surgery. The small size of the main and branch pulmonary arteries in unrepaired TOF is considered to be reflective of decreased flow, and augmentation of the flow after repair very likely contributes to pulmonary artery growth. In addition, the thin-walled MPA may be particularly well suited to enlargement in proportion with general somatic growth or in response to the nonphysiological stimulus of to-and-fro flow across the pulmonary valve annulus in the common situation of pulmonary regurgitation after TOF repair. It is also likely that MPA may have normal growth in patients operated without transannular patches. Central pulmonary arterial ultrastructure, specifically the volume proportion of elastin, may play a role in pulmonary artery growth after TOF repair. Our results are consistent to the report from Kaulitz et al in a relatively younger cohort of patients that showed sustained pulmonary regurgitation after TOF repair.

Table 2. Comparison of the Effects of Sex, Previous Palliation, Pulmonary Atresia and Transannular Patch on AO and MPA Area Z Scores

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>SE</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>AO area Z score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>2.28</td>
<td>0.22</td>
<td>0.015</td>
</tr>
<tr>
<td>Women</td>
<td>1.53</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>Palliation</td>
<td>2.55</td>
<td>0.13</td>
<td>0.046</td>
</tr>
<tr>
<td>No palliation</td>
<td>1.57</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td>Pulmonary atresia</td>
<td>4.25</td>
<td>0.78</td>
<td>0.003</td>
</tr>
<tr>
<td>Not pulmonary atresia</td>
<td>1.69</td>
<td>0.14</td>
<td></td>
</tr>
<tr>
<td>Transannular patch</td>
<td>1.85</td>
<td>0.20</td>
<td>0.610</td>
</tr>
<tr>
<td>Not transannular patch</td>
<td>2.00</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>MPA area Z score</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>−0.22</td>
<td>0.20</td>
<td>0.699</td>
</tr>
<tr>
<td>Women</td>
<td>−0.34</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Palliation</td>
<td>−0.70</td>
<td>0.32</td>
<td>0.078</td>
</tr>
<tr>
<td>No palliation</td>
<td>−0.06</td>
<td>0.17</td>
<td></td>
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<tr>
<td>Pulmonary atresia</td>
<td>−0.31</td>
<td>0.53</td>
<td>0.965</td>
</tr>
<tr>
<td>Not pulmonary atresia</td>
<td>−0.29</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td>Transannular patch</td>
<td>−0.21</td>
<td>0.22</td>
<td>0.660</td>
</tr>
<tr>
<td>Not transannular patch</td>
<td>−0.34</td>
<td>0.20</td>
<td></td>
</tr>
</tbody>
</table>

AO indicates aortic; MPA, main pulmonary artery.

Table 3. Great Artery Size and BSA Stratified by Sex in the Controls

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>AO diameter/BSA</td>
<td>1.62±0.28</td>
<td>1.70±0.28</td>
<td>0.156</td>
</tr>
<tr>
<td>AO area/BSA</td>
<td>2.91±2.50</td>
<td>2.86±1.61</td>
<td>0.644</td>
</tr>
<tr>
<td>MPA diameter/BSA</td>
<td>1.61±0.40</td>
<td>1.67±0.46</td>
<td>0.386</td>
</tr>
<tr>
<td>MPA area/BSA</td>
<td>3.15±0.58</td>
<td>3.17±0.67</td>
<td>0.893</td>
</tr>
</tbody>
</table>

AO, aortic; BSA, body surface area; MPA, main pulmonary artery.
artery growth after TOF repair resulting in normally developed proximal pulmonary artery system irrespective of age at repair. Our results also demonstrate the range of expected MPA dimensions with respect to BSA in repaired TOF and would, therefore, facilitate decision making in the individual patient.

Study Limitations
The TOF cohort represents both recent and nonrecent surgical repairs, so as to incorporate patients over a wide age range. Therefore, era effects including the variability and advances in surgical techniques must certainly have impacted our observations. Older TOF patients were operated more than 40 years ago and had higher ages at definitive repair. The normal controls consisted of younger subjects with a slightly lower median age. Compared with phase–contrast anatomic imaging used to derive AO and MPA sizes in our study, gadolinium contrast CMR angiography would have offered technical advantages, such as multiple AO measurements at defined positions (AO sinus, sinotubular ridge, and ascending aorta) in the aorta. The magnitude images derived from the phase contrast technique compares well to conventional gradient-echo images in terms of image reconstruction/anatomic accuracy, and it was not feasible to administer contrast in our comparison cohort of normal children who did not have intravenous access and in whom the imaging protocol had to be limited. Moreover, contrast studies would be disadvantageous for serial clinical surveillance and suboptimal in accuracy because of the limited temporal resolution. Finally, longitudinal studies are needed in repaired TOF to determine whether our cross-sectional observations of the TOF population are a true reflection of the modified natural history in individuals.

Conclusions
Ascending AO and MPA cross-sectional areas by CMR are reproducible and have predictable association with BSA in both normal hearts and repaired TOF. The variation in great arterial dimensions at any given BSA is much greater in surgically modified TOF than in healthy controls, and we provide reference data for serial great artery measurements in repaired TOF. AO dilatation in TOF is significantly greater among men, which has implications for a different follow-up surveillance strategy in male TOF patients given the higher risk for severe AO outcomes in men with CHD. Pulmonary atresia and staged surgical treatment with initial palliation also lead to significantly increased AO Z scores in TOF and may also be considered as risk factors for AO complications. MPA size after repair of TOF is on an average smaller than normal, but it is not small to the same extent as the aorta is large.

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Disclosures
None.

References

CLINICAL PERSPECTIVE

Ascending aorta and main pulmonary artery cross-sectional areas can be measured reproducibly in children and young adults using cardiovascular magnetic resonance imaging. Great vessel sizes measured this way increase predictably with body surface area in both normal hearts and repaired tetralogy of Fallot, but great arterial growth characteristics in tetralogy are remarkably different from normal. Aortic growth out of proportion to normal is seen in tetralogy after repair and may be a hazard for some of these patients in the long term. For them, cardiovascular magnetic resonance could play an important role in early identification of disproportionate aortic dilatation. In this series, male sex, pulmonary valve atresia, and previous surgical palliations emerged as predictors for larger ascending aorta areas in tetralogy. These factors may have implications for risk stratification and surveillance strategies in this population. The main pulmonary artery in repaired tetralogy, although smaller, on an average, than normal controls, is not small to the same extent as the aorta is large. Percentile charts for expected great vessel areas as a function of body surface area in normal subjects and in surgically modified tetralogy were generated for clinical reference.
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