Abnormal Pulmonary Artery Bending Correlates With Increased Right Ventricular Afterload Following the Arterial Switch Operation

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Abstract

Purpose: In transposition of great arteries, increased right ventricular (RV) afterload is observed following arterial switch operation (ASO), which is not always related to pulmonary artery (PA) stenosis. We hypothesize that abnormal PA bending from the Lecompte maneuver may affect RV afterload in the absence of stenosis. Thus, we sought to identify novel measurements of three-dimensional cardiac magnetic resonance (CMR) images of the pulmonary arteries and compare with conventional measurements in their ability to predict RV afterload. Methods: Conventional measurements and novel measurements of the pulmonary arteries were performed using CMR data from 42 ASO patients and 13 age-matched controls. Novel measurements included bending angle, normalized radius of curvature ($R_c$), and normalized weighted radius of curvature ($R_{cw}$). Right ventricular systolic pressures (as the surrogate for RV afterload) were measured by either recent echocardiogram or cardiac catheterization. Results: Conventional measurements of proximal PA size correlated with differential pulmonary blood flow ($r = 0.49, P = .001$), but not with RV peak systolic pressures ($r = -0.26, P = .18$). In ASO patients, $R_{cw}$ correlated with higher RV systolic pressures ($r = -0.57, P = .002$). Larger neoaortic areas and rightward bending angles correlated with smaller right pulmonary artery $R_c$ ($r = -0.48, P = .001; r = 0.41, P = .01$, respectively). Finally, both pulmonary arteries had significantly smaller $R_c$ compared to normal controls. Conclusions: Pulmonary arteries exhibit abnormal bends following ASO that correlate with increased RV afterload, independent of PA stenosis. Future work should focus on clinical and hemodynamic contributions of these shape parameters.

Keywords

transposition of great arteries, arterial switch, cardiac magnetic resonance imaging (MRI), pulmonary arteries, curvature, PA hemodynamics, PA stenosis

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Introduction

In the era of early primary repair, the arterial switch operation (ASO) is the standard in the surgical management of transposition of great arteries (TGA). In addition to coronary translocation, the repair employs the Lecompte maneuver as first described by Jatene, where the pulmonary artery (PA) bifurcation is placed anterior to the ascending aorta. While long-term survival is excellent, studies have demonstrated long-term impairment in right ventricular (RV) systolic function and diastolic function. The causes of impaired RV performance can be related to perioperative factors (eg, reoxygenation injury) and postoperative complications, with particular highlight on increased RV afterload from PA stenosis. However, the RV can still demonstrate abnormal function.
Abnormal relaxation, and RV hypertrophy even in the presence of normal-sized pulmonary arteries. 

Many previous studies have focused on PA stenosis after the Lecompte maneuver. PA stenosis is the most common indication for reintervention after ASO, with a long-term incidence of approximately 8% over 25 years. Identified mechanisms include decreased postoperative growth and pulsatility of the pulmonary arteries, stretching of the left pulmonary artery (LPA) across the aorta, and surgical technique of coronary excision from the native aortic root, all leading to increased RV afterload. However, abnormal PA flow patterns can still be appreciated in ASO patients with no apparent PA stenosis. These findings suggest alterations in PA hemodynamics, independent of PA narrowing, that are important contributors to RV afterload.

Cardiac magnetic resonance (CMR) imaging and fluid mechanic principles may offer insight into this phenomenon. In a recent study, we used computational fluid dynamics (CFD) in three-dimensional (3D) data sets from CMR of ASO patients and demonstrated the formation of turbulent, vortical flow from abnormal bending in the very proximal branch pulmonary arteries (Figure 1, Supplementary Video 1). Abnormal distortions in pulmonary blood flow profile can also be observed in clinical CMR scans of ASO patients. The flow appearance suggests the classical hydrodynamic phenomenon seen in bent pipes as the curvature increases, expressed in the form of the Dean number/equation. In a bent pipe, flow can separate from the inner wall, transition into turbulence, and result in pressure gradient. Therefore, we hypothesize that in ASO patients, increased RV afterload is influenced by abnormal bending in the pulmonary arteries.

As there are no conventional imaging measurements (CMR or otherwise) that can predict increased RV afterload in ASO patients, we developed a novel set of PA measurements (performed on 3D reconstructions of CMR images) that were inspired by our CFD study to describe the spectrum of unnatural bending in the PA branches. In this study, these novel PA measurements are compared to conventional PA measurements (ie, minimal diameter, cross-sectional area) in their ability to predict RV afterload.

**Patient and Methods**

All ASO patients who underwent a good quality CMR study from January 2014 to December 2017 were included in this institutional review board–approved retrospective study, including TGA variants such as TGA with ventricular septal defect (TGA + VSD) and complex TGA variants such as double-outlet right ventricle (DORV). All included patients had Lecompte maneuver as part of their initial repair. Patients who had conduit placement as part of their initial repair (potentially affecting PA bending) were excluded. Double-outlet right ventricle patients were included as they typically demonstrate a wider range of great vessel orientation which potentially would affect Lecompte position of the pulmonary arteries. Patients with poor imaging quality or significant stent artifact from pulmonary angioplasty (obscuring proximal branch PA anatomy) were excluded. Patients with evidence of elevated pulmonary vascular resistance (confirmed by cardiac catheterization) were also excluded as their primary etiology for increased RV afterload is distal pulmonary vascular disease. Cardiac magnetic resonance data sets from 13 age-matched patients with normal PA anatomy who underwent CMR imaging for a separate clinical indication were also included for comparison.
Cardiac Magnetic Resonance Imaging

All images were performed on a 1.5 T MR scanner (Aera; Siemens Healthcare, Erlangen, Germany) with an 18-channel body matrix array anteriorly. Cardiac magnetic resonance data from ASO patients included contrast-enhanced magnetic resonance angiography, phase-contrast acquisitions, and 3D steady-state free precession sequences. All sequence parameters, including repetition time (TR), echo time (TE), field-of-view (FOV), and voxel size, were performed in accordance with laboratory standards (see Supplementary Materials). Standard clinical measurements of RV volume and function were collated.

Right Ventricular Afterload Measurements (by Echocardiogram/Cardiac Catheterization)

Right ventricular systolic pressure was the surrogate for RV afterload, measured by either standard transthoracic echocardiography (performed within a six-month period of the CMR study) or cardiac catheterization. For transthoracic echocardiography, the RV systolic pressure was estimated by systolic tricuspid pressure gradient as measured by Doppler imaging, added to the right atrial pressure (assumed to be 5 mm Hg). Cardiac catheterization data were collated from a clinically indicated catheterization performed during the same sedation, immediately after CMR. When both echocardiogram and catheterization measurements were available, the cardiac catheterization measurement of RV afterload was used.

Conventional CMR Measurements

Conventional linear and cross-sectional measurements of the great vessels were performed at standardized sites and projections on CMR data (Figure 2A-C), as described by Knobel et al.\textsuperscript{18} and Kaiser et al.\textsuperscript{19} The main pulmonary artery (MPA) axial angle was also measured along the axial plane (Figure 2A) by marking the angle between the neopulmonary root and neoortic root from the median plane (negative = rightward). The pulmonary arteries were measured at their narrowest point to capture pulmonary stenoses, if present (Figure 2B). In order to understand the pulmonary anatomy as it relates to the aortic anatomy, the proximal ascending aorta (prox-AscAo) was defined at the level immediately above the reimplanted coronary arteries; the middle ascending aorta (mid-AscAo) was defined at the level of the pulmonary arteries (Figure 2C). All cross-sectional areas were indexed to body surface area; total PA cross-sectional area was reported as the Nakata index,\textsuperscript{20} and branch PA area as a percentage of the total cross-sectional area.\textsuperscript{21}

Novel Measurements of PA Bending

Novel PA measurements were modeled after the fluid mechanic principles of flow through a bent tube, in the presence of a radial pressure gradient.\textsuperscript{17} To ensure accuracy and reproducibility of novel measurements, 3D digital models of the branch pulmonary arteries were created from the CMR data sets (see Supplementary Materials for methodology) and measured with Rhinoceros 3D (Robert McNeel & Associates, Seattle, Washington) to ensure optimal 3D plane selection and interobserver reproducibility.

i) Normalized radius of curvature ($R_c$) in the proximal right pulmonary artery (RPA) and LPA: This is a measurement of how small the bend is across the proximal portion of the PA. First, the radius of curvature ($R$) was measured along the oblique plane of each PA, by tracing a circle where 25\% of the circumference contacts the curvature (Figure 3). As $R$ decreases, curvature increases. At the optimal plane of measurement (that best reflected the proximal trajectory of the PA), $R$ will be at a minimum; any out-of-plane measurements would likely overestimate $R$ on a single 2D image. Thus, 3D
imaging was used to ensure that the entire PA bend could be visualized, in order to improve both measurement precision and interobserver reproducibility.

To account for PA size and express curvature as a dimensionless unit, the radius of curvature is normalized against the RPA/LPA diameter (D) distal to the bend. The distal PA diameter, rather than the proximal diameter, was used to limit dependency between normalized curvature and proximal PA stenosis:

\[ R_c = \frac{R}{D} \]

ii) Normalized weighted radius of curvature \((R_{c-w})\): In order to account for vessel size changes that may be affected by redistribution of pulmonary blood flow in the presence of unilateral pulmonary stenosis in ASO, differential pulmonary blood flow (differential \%) across each individual PA is used to “weight” the \(R_c/D\) of the RPA and LPA. This is consistent with Harris et al’s concept of incorporating differential \% when calculating unilateral pulmonary vascular resistance.21 Thus, the normalized radius of curvature of the RPA and LPA is weighted accordingly:

\[ R_{c-w} = \frac{R}{D_{RPA}} \times \text{Differential }\%_{RPA} + \frac{R}{D_{LPA}} \times \text{Differential }\%_{LPA}. \]

iii) Bending angle in the RPA/LPA: This is measured from the same oblique plane used for \(R_c\) measurements. The bending angle is the angle between the long axis of the RPA/LPA and MPA (Figure 3). As the bending angle increases, the curvature increases.

### Statistical Analysis

All statistical analyses were performed with MedCalc V12.2 (MedCalc Software, Ostend, Belgium). Comparisons of continuous variables were performed using paired or unpaired t test. Correlations between continuous variables were assessed using Pearson correlation coefficient. Probability values <.05 were considered statistically significant. To assess interobserver variability of novel measurements, the bending angle and radius of curvature were assessed with intraclass correlation coefficient (ICC), by comparing measurements between two independent observers across 15 selected studies (ten ASO patients, five controls). Intraclass correlation coefficient (two-way mixed model for absolute agreement) values were interpreted as follows: 0.00 to 0.10 represent no agreement, 0.11 to 0.40 slight agreement, 0.41 to 0.60 fair agreement, 0.61 to 0.80 moderate agreement, and 0.81 to 1.0 substantial agreement.23

### Results

#### Demographics

Forty-two ASO patients (aged: 13.1 ± 9.4 years, body surface area: 1.3 ± 0.6 m²) were included in the study group (Table 1). Three ASO patients were excluded due to significant stent artifact that obscured proximal PA anatomy on their CMR. One ASO patient was excluded due to the presence of elevated pulmonary vascular resistance. There was a higher prevalence of males (73.8%) in the group, consistent

### Table 1. Patient Demographics.

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<td><strong>Demographics</strong></td>
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<td><strong>Native anatomy</strong></td>
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<td>TGA, VSD</td>
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<td>DORV</td>
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<td><strong>Age at surgery (days)</strong></td>
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<td>TGA</td>
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<td><strong>Subsequent surgical interventions</strong></td>
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<td><strong>Hemodynamic data</strong></td>
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<td>RV ejection fraction</td>
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<td>Maldistribution of pulmonary blood flow</td>
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<td>Differential blood flow to RPA</td>
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<td>RV Systolic Pressure (mm Hg)</td>
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Abbreviations: BSA, body surface area; DORV, double-outlet right ventricle; IVS, intact ventricular septum; LPA, left pulmonary artery; LVOTO, left ventricular outflow tract obstruction; MPA, main pulmonary artery; RPA, right pulmonary artery; RV, right ventricle; RVOT, right ventricular outflow tract; TGA, transposition of great arteries; VSD, ventricular septal defect.
with the known male predominance in TGA. Twenty-two patients (78%) had proximal LPA stenosis, whereas 20 patients (47%) had proximal RPA stenosis. Sixteen (38%) ASO patients had bilateral proximal pulmonary stenosis, 14 (36%) ASO patients had unilateral branch pulmonary stenosis, and 12 (26%) ASO patients had no proximal pulmonary stenosis (see “Subset Analysis” section). Fifty-four percent of patients had evidence of increased RV afterload (RV systolic pressures >25 mm Hg) on echocardiogram and/or cardiac catheterization. Eight (18%) patients had inadequate tricuspid regurgitation or cardiac catheterization data to measure RV afterload. No patients had significant RV dysfunction, with mean RV ejection fraction of 55 ± 7%.

Eight ASO patients also underwent cardiac catheterization at the time of their CMR for the purposes of balloon (7) or stent (1) angioplasty of branch PA stenosis; their hemodynamic results are shown in Supplementary Table 1. The mean RV peak systolic pressure was 50 ± 12 mm Hg. The mean difference between RV peak systolic pressure by echocardiography and cardiac catheterization was 3 ± 13 mm Hg.

### Conventional CMR Measurements

Conventional measurements of the pulmonary arteries in the ASO patients, as shown in Table 2, are consistent with published literature. The mean Nakata index was 154 ± 82 mm²/m². The pulmonary trunk tended to be rightward compared to the aorta, with a mean MPA angle of −13.2 ± 20.4° from the median plane, and flatter along the sagittal plane than the axial plane (13.7 ± 4.0 mm vs 19.1 ± 5.2 mm, P < .0001). The prox-AscAo was larger than the mid-AscAo area (436 ± 164 mm²/m² vs 229 ± 79 mm²/m², P < .0001).

### Novel Measurements

Novel measurements (bending angle, and ) are shown in Table 3. The RPA and LPA in ASO patients had smaller Rc compared to normal controls (0.44 ± 0.06 vs 1.31 ± 0.27, respectively, P < .0001; 0.88 ± 0.64 vs 1.71 ± 0.58, respectively, P = .001). Arterial switch operation patients also had lower Rcw compared to normal controls (0.64 ± 0.35 vs 1.51 ± 0.35, P < .0001). In ASO patients, the RPA had smaller Rc compared to the LPA (P = 0.0001) and larger bending angles compared to the LPA (P = .004). Only the LPA had a smaller bending angle compared to normal controls (72.8 ± 14.9 vs 52.4 ± 10.6, P < .0001).

### Correlations With RV Afterload, Differential Pulmonary Blood Flow, and RV Function

As shown in Table 4, conventional measurements of PA size (Nakata index, RPA/total area ratio) correlated with differential pulmonary blood flow (r = 0.49, P = .0011) and did not correlate with RV afterload (r = −0.26, P = .18). In contrast, as shown in Figure 4, RV afterload and the novel measurement of Rcw are moderately correlated (r = −0.56, P = .002). Of note, Rcw does not correlate with PA size (r = 0.20, P = .2), body surface area (r = 0.16, P = .29), or age.
Subset Analysis of ASO Patients With No Branch PA Stenosis

Out of the 12 ASO patients with no PA stenosis by CMR, 10 had increased RV afterload (mean RV systolic pressures $41 \pm 12$ mm Hg). The RPA and LPA had an $R_c$ of $0.37 \pm 0.23$ and $0.76 \pm 0.60$, respectively, and the $R_{c-w}$ was $0.55 \pm 0.32$. When compared to the rest of the ASO patients, this subset of patients tended to have higher RV afterload and smaller curvatures (Figure 5), although both comparisons were not statistically significant ($P > .05$).

Subset Analysis of ASO Patients With DORV Versus TGA + Intact Ventricular Septum/TGA + VSD

The complex DORV patients had higher incidence of PA reintervention and required subsequent surgical reinterventions, including the pulmonary arterioplasties in Table 1 (out of the four DORV patients, three required pulmonary arterioplasty). In the DORV patients, mean RV pressure was $43 \pm 18$ mm Hg and correlated with $R_{c-w}$ ($R = 0.76$); however, this was not statistically significant ($P = .24$) from low numbers ($n = 4$). Meanwhile, for the ASO patients with TGA + intact ventricular septum or TGA + VSD, the mean RV pressures were not statistically different ($36 \pm 14$ mm Hg) and the correlations with $R_{c-w}$ and RV pressure were preserved ($R = 0.53$, $P = .0025$). The geometrical correlations were also unchanged, MPA versus $R_c$ of RPA: $R = 0.36$, $P = .026$; prox-AscAo versus $R_c$ of RPA: $R = 0.36$, $P = .026$; prox-AscAo versus $R_c$ of LPA: $R = -0.38$, $P = .018$).

Interobserver Variability

The interobserver variability for novel measurements between the two observers demonstrated moderate agreement in bending angle measurements and substantial agreement in $R$ measurements. The RPA and LPA bending angle had an ICC of

Geometrical Correlations

The PA bends were affected by neoaortic size and MPA axial angle. The $R_c$ of the RPA negatively correlated with prox-AscAo indexed area ($r = -0.48$, $P = .0013$), whereas $R_c$ of the LPA positively correlated with the prox-AscAo indexed area ($r = 0.36$, $P = .02$). Greater rightward orientation of the MPA axial angle also correlated with a worse $R_c$ of the RPA ($r = 0.41$, $P = .01$). There were no significant correlations to RPA bending angle or LPA bending angle.

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0.61 (95% confidence interval: 0.18-0.84) and an ICC of 0.73 (95% confidence interval: 0.35-0.89), respectively. The RPA and LPA Rc measurements had an ICC of 0.98 (95% confidence interval 0.94-0.99) and an ICC of 0.95 (95% confidence interval: 0.8656-0.9839), respectively.

Comment

This study is the first to describe novel measurements of abnormal bending after ASO, which are independent from PA stenosis and correlate well with RV afterload. These novel measurements offer insight into the common clinical scenario of repaired TGA with increased RV afterload in the absence of PA stenosis, acting as a surrogate for the complex flow patterns seen across the pulmonary arteries in TGA. These novel findings were found in both simple TGA variants and complex DORV variants. On the other hand, while the conventional measurements in our study were consistent with reported literature, they only take into account PA stenosis and do not correlate with RV afterload. Our novel measurements (Rc, Rcw, and bending angle) quantify a phenomenon previously observed via angiography and described by Chen et al26 and Chiu et al27 as “narrowing of the pulmonary arch window.” The abnormal curvatures and bending angles in the branch pulmonary arteries are likely an effect from multiple geometrical components, particularly enlargement of prox-AscAo and a rightward MPA position. The long-term clinical effect of increased RV afterload in ASO patients warrants life-long monitoring. Arterial switch operation patients generally demonstrate normal RV ejection fraction4; however, their exercise capacity is at least borderline low compared to the normal population, and exercise limitation has been correlated with smaller PA sizes and maldistribution of pulmonary blood flow.31,32 Subtle impairments in RV function also persist after surgical repair, such as decreased RV free wall strain and decreased systolic/diastolic tissue Doppler imaging velocities.5,7 Based on the current understanding of RV pathophysiology in pulmonary hypertension, it is clear that the RV adapts to increased afterload and undergoes remodelling with alterations in regional contractility.34 Indeed, Thattaliyath et al demonstrated that ASO patients have increased RV circumferential strain rate when compared to normal controls.35 However, eventually over time, maladaptive remodeling will occur with the development of eccentric hypertrophy and worse RV systolic/diastolic function.34 As ASO patients get older, longitudinal follow-up is warranted to survey for right heart failure in addition to traditional cardiovascular risk factors.

The relationship of worsening curvatures and increased afterload is consistent with the fundamental fluid mechanic principles. As compared to straight tubes, curved pipes have an additional contribution to flow resistance (in addition to friction losses) where magnitude is directly proportional to the radius of curvature.17,36 A smaller radius of curvature leads to an increase in the local Dean number, one of the dimensionless parameters governing fluid flow regimes in bent pipes.16 The Dean number has also been used to describe hemodynamics in the PA bifurcation, although without any quantitative measurements.15 We performed secondary analysis and found that the RPA/LPA had higher Dean numbers in the ASO patients compared to controls (see Supplementary Table 2). Higher Dean numbers lead to increasingly complex flow structures, possibly involving flow separation along the inner wall and subsequent transition to turbulence (Supplementary Video 1). All these factors contribute to increased RV work to overcome flow resistance in the proximal pulmonary vessels. We conjecture that this circumstance is a physical explanation for the correlation between RV afterload and curvature found in our study.

The effect of abnormal bending on ventricular function has been previously established, particularly relating to aortic arches after coarctation repair. Bruse et al in particular was able to correlate worse left ventricular function and increased left ventricular mass with aortic arches that had greater curvature.37 Increased power losses have been noted in “Gothic” aortic arches relative to the “Romanesque” arch after an aortic arch repair.38 The Dean equation has also been previously used to illustrate arch curvature as a function of flow resistance across an arch.36 Our work applies the same fluid mechanic principles and is equivalent to Bruse et al’s study, except for studying the influence of the ASO pulmonary geometry on the RV.

From a clinical standpoint, Harris et al have demonstrated that differential pulmonary blood flow is predominantly dictated by a combination of pulmonary arterial diameter and differential pulmonary vascular resistance21; however, only the effects of abnormal bending can explain the observations made by Grotenhuis et al, where abnormal PA flow and increased RV mass were present in ASO patients with no significant PA stenosis.6 Our results imply that while discrete, linear PA stenosis can be readdressed with surgery/catheterization (and improve pulmonary blood flow distribution), the remaining substrate of “the pulmonary arch window” would still lead to increased RV afterload. The inherent resistance formed by this abnormal morphologic substrate may explain why reintervention of the pulmonary arteries after the Lecompte maneuver remains challenging and lack satisfactory results.39,40

Limitations

There are several limitations to this study. This is a single-center, retrospective review. The limited size in our study likely affected the subset analysis of ASO patients with no branch pulmonary stenosis. As not all patients underwent cardiac catheterization, both invasive and noninvasive measures of RV afterload were used in this study, potentially limiting the correlations in our study. The negative correlation of age and body surface area with RV systolic pressures indicates a significant positive selection bias that younger/small ASO patients undergoing CMR are more likely to demonstrate PA pathology and increased RV afterload. This selection bias is mitigated by our observation that Rc is independent of age or patient size; thus, age or patient size cannot account for the correlations noted in our study. Additionally, not all patients
had adequate tricuspid regurgitation by echocardiogram or cardiac catheterization data to assess RV afterload. In these patients, the mean $R_{\text{aw}}$ was not statistically different from the rest of the cohort $(0.6 \pm 0.3, P = .7)$; thus, they were not included in correlation analysis to avoid positive/negative selection bias.

Bending angle measurements had significant interobserver variability, likely from inherent limitations of making linear measurements off of 3D geometry (a similar issue is seen in measurements of aortic arch morphometry). The complex PA geometry in DORV patients (with further alterations by PA arterioplasty) was also not elaborated beyond the simple measurements made in this study. Future work will address these limitations with comprehensive, systematic CFD analysis of the entire group of ASO patients, along with the use of 3D statistical shape modeling methods, to clarify the selective contribution of abnormal bends to hemodynamic inefficiency, differential pulmonary blood flow, and changes to PA geometry with time (or subsequent reinterventions).

**Future Directions**

With most ASO patients still in young adulthood, this study provides insight and feedback into the unique hemodynamics that may inform the lifelong congenital care of such patients. With CMR providing optimal surveillance of PA anatomy after the ASO, measurements of bending can be reliably followed as patients age. Alternative surgical treatments, such as the spiral technique, could possibly create a more favorable flow profile from the spiral configuration of pulmonary arteries; however, this technique has little mid-term and long-term outcome data.

**Conclusion**

In our study, novel measurements have been identified that can distinguish pulmonary vessel bending in ASO and is associated with RV afterload. In ASO patients, branch pulmonary arteries demonstrate higher bending angles and decreased radius of curvature when compared to normal controls. Worse curvature is associated with increased RV afterload, independent from PA stenosis. Assessment of PA bends should be considered for potential risk stratification of abnormal hemodynamics after the ASO.

**Declaration of Conflicting Interests**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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**Supplemental Material**

Supplemental material for this article is available online.

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