Imaging of congenital heart disease in adults

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INTRODUCTION

The number of adults with congenital heart disease continues to increase due to significant advances in early diagnosis and management. However, residual or postoperative right and left sided anatomic and haemodynamic abnormalities are common. Cardiovascular imaging is essential in the long-term care of adult congenital heart disease (ACHD). Periodic surveillance imaging is important for detecting haemodynamic change, as symptoms may be late. With age, comorbidity including acquired heart disease also comes into play. The choice and frequency of imaging modality is determined by lesion specific patient characteristics, strengths and weaknesses of imaging modality and institutional resources and expertise. A multimodality imaging approach is often required to obtain all the necessary information.

USE OF DIFFERENT IMAGING MODALITIES IN LIFELONG FOLLOW UP

Echocardiography

Echocardiography remains the workhorse and first line in imaging for assessment of anatomy and physiology. It is widely available, portable, has high temporal resolution and is free from ionising radiation exposure. Small, mobile intracardiac structures such as valves and vegetations and small intracardiac shunts are well shown (Figure 1). Doppler echocardiography is usually the superior method for noninvasive haemodynamic assessment of right ventricular (RV) and pulmonary artery (PA) pressure gradients and valvular pathology and function in ACHD. To assess RV systolic function percentage fractional area change (FAC) and tricuspid annular plane systolic excursion (TAPSE) from a four-chamber view are widely used. TAPSE represents longitudinal contractile function of the RV, is reproducible and easy to measure though it may be influenced by tricuspid regurgitation, abnormal ventricular geometry and recent surgical procedures. Moreover, TAPSE has prognostic value, for example, in Eisenmenger syndrome, (Figure 2). Tissue Doppler imaging of myocardial velocities and speckle tracking for myocardial deformation have both been applied in ACHD for regional and global ventricular myocardial deformation assessment (Figure 3), but their clinical application to the RV in ACHD remains to be elucidated. Transthoracic echocardiography
(TTE) acoustic windows may become suboptimal as patients age or have chest wall deformity from prior surgery. Obtaining 3D echocardiography images that include all of the RV in ACHD is not always possible and RV volumes may be underestimated. Transoesophageal echocardiography (TOE) is useful in selected cases to guide interventional procedures (Figure 4) or further evaluate valvular anatomy.

**CMR and CT**

Cardiovascular magnetic resonance (CMR) allows unlimited access to imaging the heart and thoracic cavity unrestricted by rib spaces, does not involve ionising radiation, and is therefore ideally suited and recommended for long-term ACHD follow-up. In addition to addressing many questions that TTE can answer, CMR is the reference standard for accurate and reproducible quantification of right and left ventricular volumes, mass and function. Other strengths include assessment of degree of valvar dysfunction, especially pulmonary regurgitation, multilevel outflow tract obstruction, shunt quantification (from pulmonary and systemic flows), differential branch pulmonary artery (PA) flow, and non-invasive tissue characterisation. Advances in CMR allow for acquisition and reconstruction of comprehensive data sets in any plane for subsequent analysis of anatomy, function and flow. In patients scheduled for reoperation CMR (or CT) provides information to assess the relationship between vascular structures, the heart and the sternum. Limitations include availability, higher cost, artefacts from stainless steel implants, and relative contraindication in patients with pacemakers or implantable defibrillators (ICD). Magnetic resonance-compatible devices are increasingly used and are desirable for ACHD patients; selected patients with *in situ* conventional devices may safely undergo CMR with appropriate local protocol. However, predicting the impact of artifact noise on image quality is difficult. Siting the device on the opposite side of the chest from the heart may be preferable in ACHD patients.

Multi-detector ECG-gated cardiac computed tomography (CT) has excellent 3D spatial resolution and allows for detailed evaluation of small blood vessels such as coronary arteries (Figure 5), pulmonary veins, (Figure 6), collaterals, arteriovenous malformations, distal PA branches and *in situ* pulmonary thrombosis. Acquisition time is rapid (< 2 minutes) allowing patients who are unable to lie still or flat for
long to be imaged. CT may also have added value in younger patients, for instance to assess aberrant coronary anatomy. Pulmonary parenchyma imaging is also provided which is highly relevant for patients with pulmonary hypertension. Furthermore, CT complements assessment of mechanical heart valve dysfunction, and allows 3D visualization of abcess formation in endocarditis. CT is an alternative to selective coronary angiography in older patients referred for ACHD surgery. However, CT exposes the patient to ionising radiation and iodinated contrast agents, and does not provide information on haemodynamics, flow rate or velocity. CT can be used to acquire ventricular volumes and function but with lower temporal resolution than CMR or echocardiography and at the expense of additional radiation exposure. It tends to overestimate ventricular volumes and is clearly unattractive for serial measurements because of radiation. Multidetector CT enables dose reduction algorithms and is routinely practiced in our centre.

**Chest-x ray, nuclear scintigraphy and cardiac catheterisation**

Chest x-ray is simple, cheap and reproducible and provides diagnostic information; furthermore, cardiothoracic ratio relates to functional class and predicts survival in ACHD (Figure 6). Nuclear scintigraphy has been reserved for selected patients only, for example for myocardial stress perfusion imaging or differential pulmonary blood flow quantification when CMR is not available. Diagnostic cardiac catheterisation and angiography are less frequently performed in ACHD nowadays; they are reserved for specific clinical indications such as calculation of pulmonary vascular resistance or where the diagnosis remains uncertain after noninvasive imaging or for percutaneous interventions.

**IMAGING GOALS IN SPECIFIC CONGENITAL HEART DISEASES**

Selected lesions are discussed in subsections below and the goals of imaging are summarised in Table 1.

**Left ventricular outflow tract obstruction and aortic coarctation**

Echocardiography is used to assess left ventricular outflow tract obstruction for example due to subaortic ridge, aortic valvar and supravalvar stenosis plus aortic coarctation or re-coarctation; Doppler
derived diastolic tail in the descending thoracic aorta and continuous abdominal aortic flow indicate significant coarctation. Additional information, such as aortic valve morphology, presence of ventricular septal defect or left ventricular hypertrophy is sought. Aortopathy involving the aortic root or proximal ascending aorta should be sought on 2D echocardiography. Repaired coarctation patients require screening for the complications of recoarctation or aneurysm formation (Figure 7). Echo Doppler may reveal nonspecific high-flow velocities across the isthmus in patients after coarctation repair given the fact that elasticity of the aortic wall is lost in this trajectory, causing flow velocities to increase even in the absence of stenosis. CMR is the gold standard for assessing LV mass and can be useful to assess multilevel left ventricular outflow tract obstruction, aortic valve morphology, calibre of the entire aorta, and collateral flow. All adults with aortic coarctation should undergo cross-sectional imaging (usually CMR) at least once. CT is more suited for assessing stent lumen and fracture.

Atrial septal defects

The diagnosis of an atrial septal defect (ASD) and/or anomalous pulmonary veins should be considered in adults presenting with right ventricular dilation. TTE is used to assess size and location of the defect (secundum and primum), (Figure 4) TOE is the gold standard for accurately assessing size, number, rims, and relationships to important neighbouring structures to determine suitability for percutaneous device closure. CT and CMR are especially useful for detecting associated anomalous pulmonary veins that insert into the superior vena cava above the level of the azygous vein. Transthoracic echocardiography may be suitable for sinus venosus ASD imaging, however TOE, CMR or CT are often needed to delineate the frequent association of anomalous pulmonary venous return (Figure 8). CMR and CT give information on the distance of the anomalous pulmonary vein from the cardiac mass, which in turn is important for planning the surgical approach to pulmonary venous redirection.

Atrioventricular septal defect

Patients with atrioventricular septal (canal) defects have a common atroioventricular junction with a spectrum of lesions with potential for shunting at atrial level (ostium primum defects), ventricular level or
both atrial and ventricular level. The most common adult complication for repaired patients is left atrioventricular valvar regurgitation (of note this valve has abnormal tri-leaflet morphology). Imaging must also assess for other potential complications including pulmonary hypertension and left ventricular outflow tract obstruction. TTE is usually able to address the majority of concerns and TOE used to assess for suitability for repair versus replacement of the left atrioventricular valve.

**Repaired tetralogy of Fallot and right ventricular outflow tract obstruction**

Patients with repaired tetralogy of Fallot (TOF) constitute one of the largest groups of ACHD patients surviving into adulthood. Residual hemodynamic and electrophysiological abnormalities contribute to increasing morbidity and mortality rates arising in adulthood.\(^{12}\) The surgical strategy for TOF repair, particularly with regards to right ventricular outflow tract (RVOT) reconstruction, has evolved with time.\(^ {13}\) Conduits from the RV to pulmonary artery are sometimes required for TOF repair, for example with associated pulmonary atresia or anomalous coronary arteries. These may be difficult to assess with TTE alone due to their anterior location. A multimodality imaging approach is often utilised.\(^ {14}\) Pulmonary regurgitation is common and is an important factor for the long-term outcome of these patients. Aortic root dilation is also common in patients with repaired TOF in some patients associated with significant aortic regurgitation.\(^ {15,16} \) CMR is recommended for all patients and is especially helpful for multilevel RVOT (including branch pulmonary artery) obstruction, pulmonary regurgitation and RV volumes.\(^ {16}\) RVOT regional wall motion abnormalities and aneurysms are common and contribute to RV systolic dysfunction and adverse ventricular interactions.\(^ {17}\) RVOT akinetic area length predicts the onset of sustained ventricular arrhythmia. Pulmonary regurgitation can be assessed with echocardiography\(^ {18}\) although the gold standard for its quantification is CMR. Free pulmonary regurgitation, that is whereby forward and reverse flow jets are comparable in size, and imaging shows little or no effective remaining valve tissue, typically occurs with regurgitation fraction 30-40%. Quantification of right atrial size is helpful as large right atrial area has been associated with sustained atrial tachyarrhythmias in these patients.\(^ {19}\)
RV volumes quantified by CMR are followed serially for progressive dilatation; it has been suggested that elective pulmonary valve replacement should be considered before RV end-diastolic volume indexed to body surface area reaches 150-160mL/m². RV volumes can also be assessed to define the degree of RV reverse remodelling following valve implantation (Figure 9). Echocardiography can identify the presence of a restrictive right ventricular physiology with the presence of an antegrade “a” wave (end-diastolic forward flow) in the RV outflow tract on pulse wave Doppler throughout the respiratory cycle demonstrating a non-compliant RV which is unable to distend further with atrial systole. This again may be relevant in interpreting pulmonary regurgitant fraction or volume and RV volumes from CMR for timing of pulmonary valve replacement. Left ventricular dysfunction is present in >20% of adults with TOF and is associated with increased mortality.

Focal RV fibrosis on late gadolinium enhancement CMR imaging is associated with adverse clinical prognosticators in adults with repaired TOF; prospective studies are required. The INDICATOR prospective study of 873 repaired TOF patients showed that CMR derived RV and LV ejection fraction predict sustained ventricular tachycardia and mortality.

Cross-sectional imaging prior to catheter-based pulmonary valve reinterventions can be informative regarding the origins and proximal course of the coronary arteries in relation to the RVOT (Figure 10). Furthermore, cardiac CT provides information on the extent of conduit calcification for stent deployment. Prior to redo sternotomy, cross sectional imaging shows the proximity or even adherence of the anterior RV wall and/or ascending aorta to the sternum, thus allowing for surgical planning and avoidance of injury.

**Systemic right ventricle after atrial redirection for TGA or in the setting of congenitally corrected transposition of the great arteries (ccTGA)**

Many surviving adults with TGA would have had atrial switch surgery, (Mustard or Senning operation). Systemic RV dysfunction is a determining factor for late morbidity and mortality. Echocardiographic parameters (RV FAC, TAPSE) can be used for detecting changes in RV function. Total isovolumic time and peak systolic strain measures have prognostic value. Systemic atrioventricular
(tricuspid) valve regurgitation usually reflects progressive RV dilatation and dysfunction and is most sensitively assessed by echocardiography, as is the presence of pulmonary hypertension or baffle leaks (better delineated with the use of contrast). Associated lesions such as pulmonary stenosis and ventricular septal defect can also be assessed. Pulmonary hypertension can be difficult to diagnose when there is no mitral regurgitation but equal LV and RV size on apical four-chamber view is suggestive of pulmonary hypertension or significant baffle leak. Imaging of patients after a Mustard or Senning operation requires assessment of all three baffled atrial flow pathways (superior/inferior caval, and pulmonary venous atrial) for presence and degree of obstruction. Increased flow velocity >1.6 m/s or continuous baffle flow are suggestive of obstruction. CMR in addition to echocardiography is often helpful for assessment of baffle patency.

cCTGA comprises atroventricular and ventriculoarterial discordance (L-loop TGA/double discordance). The spectrum of clinical presentation is wide and depends on associated lesions; presentation in adulthood is possible. The tricuspid valve in cCTGA is often intrinsically abnormal (Ebstein type and may be underdiagnosed) and results in tricuspid regurgitation; the latter may also be secondary to RV dysfunction and annular dilatation. Echocardiography can be used to assess RV dysfunction and tricuspid regurgitation. Diastolic dysfunction can be assessed by Doppler flow of RV filling pattern. Absent Doppler “A” wave in the presence of sinus rhythm and clear p waves often indicate very high filling pressures. CMR allows gold standard quantification of systemic RV ejection fraction. This may be used to enable clinical decision-making with regards to tricuspid valve surgical replacement. CT may be of value in the setting of systemic RV dysfunction with wide QRS when cardiac resynchronisation is contemplated to assess the coronary sinus anatomy. Tissue characterisation by CMR has shown areas of late gadolinium enhancement consistent with focal RV fibrosis.27, 28 (Figure 11). We have recently shown that systemic RV late gadolinium enhancement correlates with histological fibrosis, is associated with clinical disease progression and predicts outcomes.29 justifying its periodic use. In our practice, we consider patients with RV ejection fraction of \( \leq 35\% \) and
extensive RV fibrosis for primary prevention for sudden cardiac death; implantable defibrillator is offered on an individualized basis following multidisciplinary discussion.

**Transposition of the great arteries (TGA) after arterial switch surgery**

Arterial switch procedure, i.e. anatomic repair, involving switching the great vessels and reimplanting the coronary arteries has been the treatment of choice for infants with TGA over the past 3 decades. Supravalvar pulmonary stenosis, neo-aortic root dilatation, aortic valve regurgitation, left ventricular dysfunction, and coronary occlusion are relatively common complications. Echocardiography with Doppler can assess aortic valve and LV function and right ventricular pressures, whereas the main and branch pulmonary arteries are often difficult to image. CMR (or CT) can provide detailed information on RVOT or branch pulmonary artery stenoses. CT is particularly suited to image the proximal coronary arteries, reimplemented during repair. CMR evaluation of coronary origins is also excellent although CT is superior in excluding coronary stenoses. In the case of symptoms, LV dysfunction or LV scar at CMR assessment of viability with CMR stress perfusion and with exercise echocardiography should be performed.

**Single ventricle, Fontan procedure**

Many survivors to adulthood of “single ventricle” physiology have undergone the Fontan operation to re-route systemic venous return to the pulmonary arteries. In the earlier era this was done with an anastomosis from the right atrial appendage to the pulmonary arteries (atriopulmonary Fontan), whereas more recently, total cavopulmonary connection (TCPC), either using an intraatrial/lateral tunnel or extracardiac conduit. The superior vena cava is connected end to side to the top of the right PA, whereas the inferior vena cava is channelled by a patch, flap, or conduit up one side of the right atrium to the pulmonary artery. Thrombus may form in the dilated right atrium in the atrio pulmonary Fontan due to sluggish flow (Figure 12), or in the disconnected pulmonary trunk after TCPC. Failure of the Fontan type circulation ensues with obstruction of Fontan pathways, ventricular and or valvular dysfunction. Restrictive ventricular septal defect in the setting of ventricular arterial discordance is unfavourable, causing subaortic stenosis. Ventricular systolic and diastolic dysfunction determine outcome; echocardiography can assess both. CMR can establish patency of
pathways, exclude thrombus, quantify the volume of the dominant/primary ventricle and its ejection fraction, and has demonstrated areas of ventricular fibrosis more commonly in patients with documented non-sustained ventricular tachycardia.30

SUMMARY

Imaging is fundamental to the lifelong care of ACHD patients. Echocardiography remains the first line imaging for inpatient, outpatient, or perioperative care. Cross-sectional imaging with CMR or CT provides complementary and invaluable information on cardiac and vascular anatomy and other intra-thoracic structures. Furthermore, CMR provides quantification of cardiac function and vascular flow. Cardiac catheterisation is, mostly reserved for assessment of pulmonary vascular resistance, ventricular end-diastolic pressure and percutaneous interventions. There have been further advances in non-invasive imaging for ACHD including the application of advanced echocardiographic techniques, faster automated CMR imaging and radiation dose reduction in CT. As a result ACHD, a heterogeneous population, benefit from appropriate application of multiple imaging modalities matched with tertiary ACHD expertise.

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FIGURE LEGENDS

Figure 1
Acute presentation of endocarditis with vegetation (asterisk) related to previous VSD surgical repair patch leak shown in apical four chamber 2D echocardiography view (A) and magnified from apical five chamber view (B).

Figure 2
Echocardiography indices TAPSE <15mm, right atrial area ≥ 25cm², right atrial to left atrial area ratio ≥1.5 and RV systolic over diastolic ratio ≥1.5 are all predictive of survival in Eisenmenger syndrome. Images show right atrial enlargement (A), prolonged tricuspid regurgitation on Doppler compromising filling (D=diastole, S=systole in B), biventricular hypertrophy and a flattened interventricular septum (C) and impaired TAPSE of 11mm (D), in this patient with longstanding Eisenmenger syndrome.

Figure 3
Speckle tracking of the LV from apical 4-chamber (A), apical 3-chamber (B) and apical 2-chamber (C) views in adult patient with repaired anomalous coronary artery from the pulmonary artery (ALCAPA) syndrome. Despite normal LV ejection fraction, speckle tracking shows LV myocardial dysfunction. The bullseye plot (D) shows that the anterior and inferior segments are akinetic. Total global longitudinal strain is -14%.

Figure 4
Large ASD (asterisk) at 3D TOE (A) with relative lack of a posterior rim (B). Fenestrated ASD from left atrial aspect (C) and from right atrial aspect (D); in these cases 2D echocardiography could potentially underestimate the size of the overall defect/shunt if the plane sampled is perpendicular to the small defect only (dotted arrow).
**Figure 5**

ALCAPA (Ai and Aii) studied with CT preoperatively. Post operative CT (Bi and ii) shows patent left internal mammary graft to mid left anterior descending artery. (Images courtesy of Dr Mike Rubens). Ao; aorta, LAD, left anterior descending, LCA; left coronary artery, PA; pulmonary artery, RCA; right coronary artery.

**Figure 6**

Anomalous right pulmonary venous drainage to the inferior vena cava creating a curvilinear “scimitar” silhouette (arrows) on chest x ray (A) with corresponding images from CT (B and C). (Images courtesy of Dr Mike Rubens).

**Figure 7**

Contrast enhanced cardiovascular magnetic resonance angiography (CE-CMRA) in an adult presenting with systemic hypertension due to severe aortic coarctation (A). CT imaging following endovascular stenting (B). Aneurysm related to endovascular stenting of coarctation studied with cine CMR (C; dotted arrow), and aneurysm related to Dacron patch coarctation repair studied with CE-CMRA (D; arrow).

**Figure 8**

CMR (or CT) may be useful in the diagnosis of sinus venosus defects, which can be at the orifice of the superior (or less commonly inferior caval veins) and to delineate anomalous pulmonary venous drainage. CMR images showing sinus venosus ASD (asterisk) in A and the anomalous drainage of the right upper pulmonary vein to superior vena cava (B). IVC; inferior vena cava, LA; left atrium, RA; right atrium, RUPV; right upper pulmonary vein, PA; pulmonary artery, RCA; right coronary artery, SVC; superior vena cava.

**Figure 9**

Diastolic still image from CMR cine pre (A) and post (B) pulmonary valve replacement for pulmonary regurgitation status post repaired tetralogy of Fallot. Reduction in RV volume and increased LV filling in
Late gadolinium enhancement CMR evidence of ventricular fibrosis/scarring is seen in C; block arrows point to bright areas of scar in the RVOT and dotted arrows to the VSD patch site. Image D is derived from 3D CMR acquisition after segmentation of chambers, outflows and scar using Mimics, Materialise NV (courtesy of collaboration with Drs Veronica Spadotto and Jennifer Keegan). LV; left ventricle, RV; right ventricle

**Figure 10**

Cardiac CT in a patient with RV-PA conduit (A), showing virtually single origin coronary arteries passing between the aorta and narrow segment of conduit (B-D). (Images courtesy of Dr Mike Rubens). Ao; aorta, Cx; circumflex, LAD, left anterior descending, LAD; PA; pulmonary artery, RCA; right coronary artery.

**Figure 11**

CMR images of simple TGA (A,B) and ccTGA (C,D) showing parallel discordant outflows (A,C) and hypertrophic systemic RV (B,D). Late gadolinium enhancement in TGA with atrial redirection surgery (E) and in ccTGA (F); bright areas of enhancement within the RV (arrows) suggestive of fibrosis. Ao; aorta, LA; left atrium, LV; left ventricle, PA; pulmonary artery, PVAC; pulmonary venous atrial compartment, RV; right ventricle, RA; right atrium.

**Figure 12**

Images A and B show patent Fontan pathways status post atriopulmonary Fontan. In patient C thrombus has formed (arrows) due to sluggish flow in the dilated right atrium. In image D, late gadolinium enhancement CMR evidence of rudimentary endocardial RV fibrosis is seen (arrows). Images E and F show patent total cavopulmonary pathways (asterisks). Ao; aorta, IVC; inferior vena cava, LA; left atrium, LPA; left pulmonary artery, LV; left ventricle, RA; right atrium, RPA; right pulmonary artery, SVC; superior vena cava.
### Table 1. Cardiac Imaging Goals in selected Adult Congenital Heart Diseases with strengths and weaknesses of different imaging modalities

<table>
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<th>Echocardiography</th>
<th>Cardiovascular Magnetic Resonance</th>
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| **Strengths** | Widely available  
Portable  
Estimation of pressure gradients  
Valvular pathology (mechanisms and degrees of dysfunction)  
Assessing small fine structures (chordal attachments, vegetations)  
Intracardiac shunts (fenestrations, PFO, VSD) | Quantification of:  
- ventricular size and function  
- regurgitant lesions  
- branch pulmonary artery flow  
- pulmonary: systemic flow  
Myocardial viability | Quantification of:  
- ventricular size and function  
Aortic pathology  
Coronary artery imaging |
| **Weaknesses** | Limited acoustic windows | Artifacts from steel implants  
Relative contraindications in patients with intracardiac leads | Exposure to ionising radiation and iodinated contrast |
| **Lesion** | Echocardiography | Cardiovascular Magnetic Resonance | Computed Tomography |
| Left ventricular outflow tract obstruction, including aortic coarctation | Aortic valve morphology, function and pressure gradient  
Subaortic anatomy (membrane) and pressure gradient  
Aortic root and ascending aortic anatomy  
LV size, function and wall thickness | Ascending, transverse and descending thoracic aortic pathology (dilation, multi-level stenosis and aneurysm)  
Aortic valve morphology  
LV size, function and mass  
Aortic collaterals | Ascending, transverse and descending thoracic aortic pathology (dilation, aneurysm)  
*A Particularly useful in the case of prior stents  
Aortic collaterals |
| Secundum ASD | Atrial septal anatomy  
Pulmonary venous anatomy (possible)  
RV size and function  
Right ventricular pressure  
AV valve morphology | Identification of multiple/ fenestrated ASD  
Identification of rims for device closure  
Pulmonary: systemic flow ratio  
Quantification of RV volumes  
Pulmonary venous anatomy | *rarely used |
| Sinus venous ASD | Atrial septal anatomy  
Pulmonary venous anatomy (possible)  
RV size and function  
Right ventricular pressure | Anomalously draining pulmonary venous connections and drainage  
Atrial septal anatomy  
RV size and function  
Pulmonary: systemic flow ratio | *rarely used |
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<td>LV size and function, Residual intracardiac shunts, Main PA and proximal branch PA stenosis, Neo-aortic root dilation and regurgitation</td>
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<td>Systemic-to-pulmonary collaterals</td>
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*In selected cases*

**Ventricular size and function**

**Residual intracardiac shunts**

**Tricuspid valve morphology & regurgitation**

**Aortic regurgitation**

**LVOT obstruction (pulmonary stenosis)**

**Single Ventricle, Fontan Procedure**

**Ventricular size and function**

**Atrioventricular valve regurgitation**

**Fontan pathway patency**

**Branch PA caliber**

**LVOT obstruction**

**Aortic regurgitation**

**Pulmonary venous compression**

**Aortic-to-pulmonary collaterals**

**Systemic-to-pulmonary collaterals**

Abbreviations: ASD; atrial septal defect, AV; atrioventricular, LV; left ventricle. LVOT; left ventricular outflow tract obstruction, PA; pulmonary artery, PFO; patent foramen ovale, PS; pulmonary stenosis, RV; right ventricle, RVOT; right ventricular outflow tract, VSD; ventricular septal defect
FIGURES

Figure 1
Figure 2
Figure 3
Figure 4
Figure 5

(A i) Ao PA RCA LCA

(A ii) Ao PA RCA LCA

(B i) graft to LAD

(B ii) graft to LAD
Figure 6
Figure 7
**Figure 8**

![Image of congenital heart disease](image-url)
Figure 9
Figure 10
Figure 11
Figure 12