Journal of Cardiovascular Magnetic Resonance 26 (2024) 101062

Contents lists available at ScienceDirect

Journal of Cardiovascular Magnetic Resonance

journal homepage: www.sciencedirect.com/journal/jocmr

Guidelines/Consensus statement

Society for Cardiovascular Magnetic Resonance guidelines for reporting cardiovascular magnetic resonance examinations in patients with congenital heart disease

Lars Grosse-Wortmann[a,*], Rachel M. Wald[b], Israel Valverde[c], Emanuela Valsangiacomo-Buechel[d], Karen Ordovas[e], Francesca Raimondi[f], Lorna Browne[g], Sonya V. Babu-Narayan[h], Rajesh Krishnamurthy[i], Deane Yim[j], Rahul H. Rathod[k]

[a] Doernbecher Children's Hospital, Department of Pediatrics, Oregon Health and Science University, Portland, Oregon, USA
[b] Toronto Congenital Cardiac Centre for Adults, Peter Munk Cardiac Centre, Toronto, Ontario, Canada
[c] Department of Paediatrics, The Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada
[d] Department of Cardiology, University Children's Hospital, University of Zurich, Zurich, Switzerland
[e] Department of Radiology, University of Washington, Seattle, Washington, USA
[f] Department of Pediatric and Adult Congenital Heart Diseases, Ospedale Papa Giovanni XXIII, Bergamo, Italy
[g] Department of Radiology, Children's Hospital Colorado, Aurora, CO, USA
[h] Department of Adult Congenital Heart Disease, Royal Brompton Hospital, London, United Kingdom
[i] Department of Radiology, Nationwide Children's Hospital, Columbus, Ohio, USA
[j] Department of Paediatric Cardiology, Perth Children's Hospital, Perth, Australia
[k] Department of Cardiology, Boston Children's Hospital, Harvard Medical School, Boston, Massachussetts, USA

ARTICLE INFO

Keywords:
Magnetic resonance imaging
Structured reporting
Reporting
Congenital heart disease
Pediatric cardiology

1. Introduction

Cardiovascular magnetic resonance (CMR) imaging is an integral component of contemporary care for individuals with acquired pediatric and congenital heart disease (CHD), offering a wealth of clinical information. To maximize impact, reports of CMR examinations must convey findings in a manner that is clear, concise, structured, and clinically relevant. The recommendations in this document provide guidance regarding the elements which should be considered in a CMR report, stratified by scan technique and common pediatric and congenital CMR indications. These guidelines, composed by an international panel of experts with in-depth experience in CMR, including cardiologists and radiologists, both pediatric and adult, are officially endorsed by the Society for Cardiovascular Magnetic Resonance

Abbreviations: 2D, two-dimensional; 3D, three-dimensional; 4DF, four-dimensional flow; AA, aortic arch; AAo, ascending aorta; AAOCA, anomalous aortic origin of the coronary arteries; APC, aortopulmonary collateral; aRV, atrialized right ventricle; ASD, atrial septal defect; BSA, body surface area; bSSFP, balanced steady state free precession; BP, blood pressure; CE, contrast-enhanced; CHD, congenital heart disease; CMR, cardiovascular magnetic resonance; CT, computed tomography; DA, descending aorta; DORV, double outlet right ventricle; DST, distal transverse arch; ECG, electrocardiogram; ECV, extracellular volume fraction; EMR, electronic medical record; EF, ejection fraction; fRV, functional right ventricle; IST, isthmus; IVC, inferior vena cava; LA, left atrial; LGE, late gadolinium enhancement; LLPV, left lower pulmonary vein; LPA, left pulmonary artery; LUPV, left upper pulmonary vein; LV, left ventricular; MRI, magnetic resonance imaging; PAPVC, partial anomalous pulmonary venous connection; PC, phase contrast; PDA, patent ductus arteriosus; RA, right atrial; RLPV, right lower pulmonary vein; RPA, right pulmonary artery; RUPV, right upper pulmonary vein; RV, right ventricular; SCMR, Society for Cardiovascular Magnetic Resonance; SVC, superior vena cava; STJ, sinotubular junction; TAPVC, total anomalous pulmonary venous connections; TGA, transposition of the great arteries; VSD, ventricular septal defect

* Corresponding author. Department of Pediatrics, Oregon Health and Science University, 707 SW Gaines Street, Portland, Oregon 97239, USA.
E-mail address: grossewo@ohsu.edu (L. Grosse-Wortmann).

https://doi.org/10.1016/j.jocmr.2024.101062
Received 11 July 2024; Accepted 18 July 2024

(SCMR). It is recognized that the final judgment regarding reporting methodology and report content rests with the specific individual(s) performing and interpreting the CMR examination. Consensus recommendations regarding pediatric and congenital CMR scan protocols have already been published and are outside the scope of this document [1]. With few exceptions, this document focuses on reporting CMR examinations in individuals with CHD. Examinations in subjects with acquired heart disease, including cardiomyopathies, are covered in previously published SCMR guidelines [2]. Selected non-congenital conditions that affect mostly children, such as coronary artery manifestations of Kawasaki disease, are included in this statement.

2. Structured reporting

Clear and accurate reporting of imaging findings is essential for high-quality care for individuals with CHD. Structured reporting uses standardized formats and phrases with an accepted lexicon of terms, as opposed to narrative reports which may be variable in content and consequently more prone to the omission of specific data. Quantitative and qualitative results are captured in pre-defined and searchable data fields. The SCMR recommends applying the principles of structured reporting in order to a) create a consistent approach to reporting within an institution and, ideally, globally among congenital CMR experts worldwide, b) facilitate a more seamless integration of information into the electronic medical record (EMR), enabling the EMR to serve as a central repository of imaging biomarkers, and c) promote high caliber quality improvement and research initiatives. Structured reporting has several advantages over the narrative style, including better reproducibility, ease of comparing serial measurements, and enhanced adherence to clinical guidelines [3]. Structured reporting also reduces the cognitive burden on the reader by reducing the time required to read and extract the important aspects of the report.

3. Post-processing and measurements

It is beyond the scope of this document to delineate various practices for post-processing, as this has been reviewed elsewhere [4]. Vascular measurements, in particular, can vary according to imaging modality (e.g., CMR, computed tomography [CT], echocardiography) [5]. Further variability is introduced by differences in location of acquisition, angulation of slice prescription, timing during the cardiac cycle (e.g., systole or diastole), and acquisition during breath- holding or free-breathing. The effect of heterogeneous practices on quantitative results, especially ventricular volumes, is likely greater in patients with structurally abnormal hearts. The SCMR recommends measuring diameters on reconstructed images that show the vessel in cross-section, most commonly derived from three-dimensional (3D) isotropic contrast- or non-contrast-enhanced (CE) angiography datasets [6]. For arteries with strong pulsatility, the use of gated images is recommended, as discussed below in the aorta section. Clinically reported measurements should ideally use similar measurement techniques as used in the generation of reference or normative values.

4. Normalization of measurements and reference values

The reporting of volumetric and dimensional data should incorporate a comparison with normal reference standards that adjust for the range of pediatric ages and sizes whenever these are available. The best way to account for the effects of somatic growth on cardiovascular dimensions remains controversial and the authors recognize that there is no perfect approach to adjust for body size. The indexing of cardiac and vascular dimensions to body surface area (BSA) is the most widely used clinical method of normalizing chamber volumes and dimensions in pediatric and adult congenital medicine, incorporated in many guidelines and scientific studies, and endorsed by SCMR [6–10]. Cardiac volumetric (e.g., ventricular end-diastolic volume), valve areas, and vascular areas are more linearly related to BSA alone, while vascular and valve diameters are more linearly related to the square root of BSA [11]. There is increasing variance and error at both small and large BSA extremes. For example, while not commonly used in clinical practice, indexing left ventricular (LV) end-diastolic volume to $BSA^{1.3}$ or $BSA^{1.4}$ will improve the regressions and reduce the error variance [11].

The available formulas for estimating BSA (the most commonly used ones are shown in Table 1) can yield significant variability in results, particularly in smaller patients. The Du Bois and the Mosteller formulas are the most widely used in clinical practice. It is important to be consistent in the BSA calculation method between reports and to employ the same BSA equation used to establish reference ranges and z-scores. In children weighing < 10 kg, weight (in kg) and height (in m) should be entered with at least two decimals to reduce inaccuracies that may be incurred by rounding.

Another method of comparing measurements between sexes and among patients of varying sizes is “z-scores” (standard deviations above or below the mean) and percentiles [7,9]. Z-scores between -2.0 and $+2.0$ encompass approximately 95% of a reference population and are generally considered within the normal range Numerous reference standards for chamber and vascular sizes are available and have been derived from contemporary CMR techniques. A number of these are summarized for the pediatric population in Table 2. A synopsis of normal values from various studies in adults and children was provided and later updated by Kawel-Boehm et al. [19,20]. Critical evaluation and recommendation of specific reference ranges are beyond the scope of these guidelines. The authors emphasize the importance of ensuring that the age of the patient being studied is within range of the selected reference standard, that the techniques underlying published reference ranges are comparable to those used in the reported CMR examination, and that sufficient scientific rigor was applied during the creation of the reference values.

5. Sequential segmental description of the anatomy

The segmental approach to CHD by Van Praagh [24,25] and the sequential segmental system by Anderson [26,27] are widely adopted strategies for the description of congenitally malformed hearts. According to this approach, cardiac anatomy is described in three segments—the atria ventricles and great arteries. Each segment is characterized according to its respective anatomic and morphologic features as well as its relationship to and connection with the adjacent segment(s). This method provides clinicians with a uniform language to identify and describe cardiac morphology in a systematic way, with some variability in terminology and conceptual framework propagated by Drs. van Praagh and Anderson. A comprehensive understanding of the standardized terminology used to describe associated conditions and a mastery of the particular language used at their institution is recommended for all authors of congenital CMR reports. A discussion of the fundamentals of cardiac chamber identification and a comparison between the two approaches is beyond the scope of these guidelines. The SCMR suggests following a segmental approach when systematically describing a patient’s anatomy.

6. Report outline

For the remainder of this document, reporting elements in “normal font” are recommended by the SCMR for routine inclusion in CMR reports. Elements that are marked in “*italics*” are considered optional, although these may be routinely incorporated per institutional policies or government regulations.

1. Administrative data regarding performance site
   - Site of service: Physical address and contact information (e.g., phone, fax, email)
   - Scanner: Magnet field strength, vendor, and model
   - *Accreditation entity of the service site and status*; [e.g., American College of Radiology, European Association of Cardiovascular Imaging]

Table 1
Most commonly used formulas for calculation of body surface area.

First author	Year	Formula
Boyd	1935	$0.0003207 \times Ht\ (cm)^{0.3} \times Wt\ (g)^{(0.7285 - (0.0188 \times LOG(Wt\ (g)))}$ [12]
Du Bois	1916	$Wt\ (kg)^{0.425} \times Ht\ (cm)^{0.725} \times 0.007184$ [13]
Gehan	1970	$Wt\ (kg)^{0.51456} \times Ht\ (cm)^{0.42246} \times 0.0235$ [14]
Haycock	1978	$Wt\ (kg)^{0.5378} \times Ht\ (cm)^{0.3964} \times 0.024265$ [15]
Mosteller	1987	Square root ((Ht (cm) × Wt (kg))/3600) [16]
Livingston	2001	$0.1173 \times Wt\ (kg)^{0.6466}$ [17]
Yu	2010	$71.3989 \times Ht\ (cm)^{0.7437} \times Wt\ (kg)^{0.4040}$ [18]

Ht height, *Wt* weight.

2. Patient demographic and clinical data
   - Patient identifier: Medical record number or other unique patient identifier used by the health care delivery system where the CMR examination was performed
   - Date of birth *and age*
   - Sex
   - Height and weight, BSA *and specific formula selected for BSA calculation*
   - Heart rate
   - *Oxygen saturation (recommended in patients with shunts and/or single ventricle physiology)*
   - *Systemic arterial blood pressures (BPs, recommended for studies involving exercise or pharmacological stress, including extremity from which BP was obtained)*
3. Referral data
   - Diagnosis and clinical indication for CMR study, including the specific clinical questions to be answered
   - Referring physician
   - Summarized relevant past medical and/or surgical history
4. Scheduling and performance data
   - Date and time of study
   - *Personnel involved in the performance of study (CMR physicians, CMR imaging fellows, MRI technologists,* etc.*)*
   - Interpreting provider contact details
5. Patient cooperation
   - Requirement for anesthesia or sedation
   - *Requirement for specialized intravenous placement or inability to place intravenous line*
   - *Patient cooperation with breath-holding sequences/use of suspended ventilation if anesthesia was used*

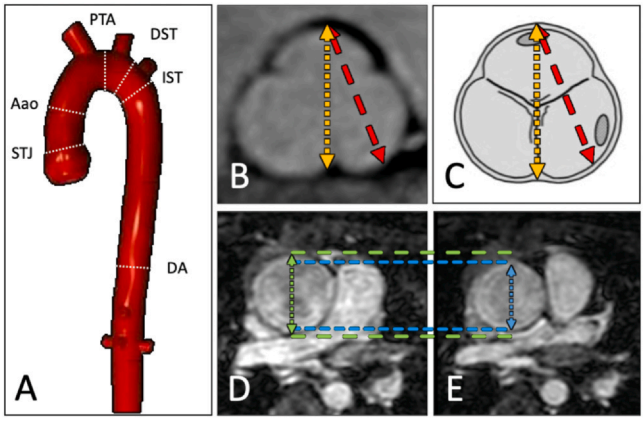

Fig. 1. Aortic measurements. (A) Locations of measurements in the thoracic aorta. (B and C) measurements in the sinuses of Valsalva, cusp to commissure (yellow dotted line) and cusp to cusp (red dashed line). (D and E) Measurements in the ascending aorta using a reformatted ECG-gated 3D balanced steady state free precession acquisition during peak systole (D) and at end-diastole (E). Note the differences in diameter. Aao: ascending aorta, DA: descending aorta at diaphragm, DST: distal transverse arch, IST: isthmus, PTA: proximal transverse arch, STJ: sinotubular junction.

6. Sequences and technique
   - Study quality (good, diagnostic, or poor)
   - Reasons for suboptimal study quality (e.g., arrhythmia, patient motion, inconsistent breath-holding, artifacts, etc.)
   - Specific sequences *and plane of acquisition*
   - For studies requiring vasoactive or positive inotrope pharmacological cardioactive agents (stress), the agent, quantity, duration, and route of administration of the agent(s) and associated medications, presence/absence of any side effects
7. Complications, early study termination, and exam-limiting artifacts
   - Allergic reactions
   - Claustrophobia, limited patient cooperation/tolerance for the study, etc.
   - Artifacts from ferromagnetic implants, arrhythmia, breathing, and other motion artifacts, etc. Given the high prevalence of implanted ferromagnetic materials (e.g., coils, devices, stents, etc.) in patients with CHD, it is important to detail the extent of the artifact as well as mention structures not reliably assessed or evaluated.

Table 2
Commonly applied pediatric normal values.

First author, year	Measurements	Field strength, pulse sequence	Number (% male)	Age range (years)
Ventricular volumes				
Buechel, 2009 [7]	Short-axis ventricular volumes z-scores	1.5T, bSSFP	50 (46%)	7 mo–18 y
Robbers-Visser, 2009 [8]	Short-axis volumes ventricular volumes, indexed to BSA and stratified by age groups	1.5T, bSSFP	60 (50%)	8–17
Sarikouch, 2010 [9]	Axial ventricular volumes, Lambda-Mu-Sigma reference curves	1.5T, bSSFP	114 (48%)	4–20
Van der Ven, 2019 [10]	Short-axis ventricular volumes, Lambda-Mu-Sigma reference curves	1.5T, bSSFP	141(48%)	< 18
Aorta				
Kaiser, 2008 [6]	Aortic arch diameter z-scores	1.5T, CEMRA	53 (57%)	2–20
Voges, 2012 [21]	Aortic arch area, distensibility, pulse wave velocity, Lambda-Mu-Sigma reference curves, z-scores	3T, gradient echo	71 (42%)	2–28
Kutty, 2012 [22]	Ascending aorta diameter, indexed to BSA	1.5T, PC	105 (52%)	4–20
Pulmonary arteries				
Knobel, 2011 [23]	Main and branch pulmonary artery diameters, indexed to BSA	1.5T CEMRA MIP	69 (59%)	2–20
Kutty, 2012 [22]	main pulmonary artery diameter, indexed to BSA	1.5T, PC	105 (52%)	4–20

bSSFP balanced steady state free precession, *BSA* body surface area, *CEMRA* contrast-enhanced magnetic resonance angiography, *MIP* maximum intensity projection, *PC* phase contrast.

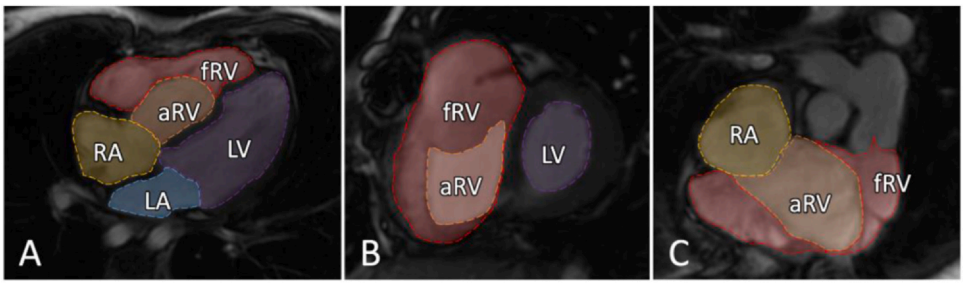

Fig. 2. Ebstein anomaly. The axial (A), short axis (B), and RV outflow tract views (C) demonstrate the aRV and fRV between the apically displaced tricuspid valve and the RV apex. aRV: atrialized right ventricle, fRV: functional right ventricle, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle.

Table 3
Shunt quantification.

	Pulmonary blood flow (Qp)	Systemic blood flow (Qs)
Pre-tricuspid shunts (atrial septal defect, PAPVC)	• Total pulmonary venous flow • Sum of RPA and LPA flows • Main pulmonary artery flow • Stroke volume right ventricle	• AAo flow • Sum of SVC and IVC flows • Sum of SVC and DA flows • Stroke volume left ventricle
Ventricular septal defect	• Total pulmonary venous flow • Sum of RPA and LPA flows • Stroke volume left ventricle	• AAo flow • Sum of SVC and IVC flows • Sum of SVC vena cava and DA flows • Stroke volume right ventricle
Patent ductus arteriosus	• Total pulmonary venous flow • Sum of RPA and LPA flows (distal to ductal insertion) • AAo flow • Stroke volume left ventricle	• Sum of SVC and IVC flows • Sum of SVC and DA flows • Sum of RPA and LPA flows (proximal to ductal insertion) • Stroke volume right ventricle
Combined pre- & post-tricuspid shunts (atrioventricular septal defect, atrial & VSDs, fenestrated Fontan with APCs, unrepaired tetralogy of Fallot with arterial duct or major APCs)	• Total pulmonary venous flow	• Sum of SVC and IVC flows • Sum of SVC and DA flows

AAo ascending aorta, *APCs* aortopulmonary collaterals, *IVC* inferior vena cava, *PAPVC* partial anomalous pulmonary venous connection, *SVC* superior vena cavacava, *RPA* right pulmonary artery, *LPA* left pulmonary artery, *DA* descending aorta, *VSD* ventricular septal defect.

Table 4
Calculation of pulmonary blood flow (Qp), systemic blood flow (Qs), and collateral flow in patients with single ventricle physiology (see Fig. 3 for illustration on flow quantification

Parameter	Methods
Pulmonary flow (Qp)	1) RUPV + RLPV + LUPV + LLPV 2) RPA + LPA + APCs
Systemic flow (Qs)[a]	1) SVC + IVC 2) SVC + DA
Aortopulmonary collateral flow (APC)	1) APCs = (RUPV + RLPV + LUPV + LLPV) - (RPA + LPA) 2) APCs[b] = AAo-Qs

AAo ascending aorta, *APCs* aortopulmonary collaterals, *DA* descending aorta, *IVC* inferior vena cava, *LLPV* left lower pulmonary vein, *LPA* left pulmonary artery, *LUPV* left upper pulmonary vein, *RLPV* right lower pulmonary vein, *RPA* right pulmonary artery, *RUPV* right upper pulmonary vein, *SVC* superior vena cava.

[a] Veno-venous collaterals such as flow reversal in the azygos/hemiazygos system and systemic venous channels draining into the pulmonary veins / atrium should be taken into account if flow can be quantified.

[b] Misses a small amount of systemic-to-pulmonary venous collateral flow if this originates upstream from the SVC (or IVC) measurements.

8. Contrast
   - Use, type, name, volume, dose (in mmol/kg) of intravenous contrast agent
   - Any adverse reactions, if present
9. General anesthesia and sedation
   - For studies utilizing general anesthesia, the reason for the anesthesia (*and details regarding the administration of agents, fractional inspired oxygen, and support*) should be documented in the EMR and, as relevant, in the CMR report.
10. Stress CMR examinations
    Document stress agent used (adenosine, regadenoson or dobutamine or rarely exercise, if exercise whether MRI conditional treadmill or stepper, maximal or submaximal exercise)
    For CMRs incorporating stress testing, systolic and diastolic BP, ST segment/rhythm changes, and the heart rate response for age (and whether targeted values were reached) should be recorded during the following points in time:
    - Before study
    - *At each level of stress*
    - At peak stress
    - *In recovery*

    Other medications, such as beta blockers for coronary artery imaging, are rarely used in pediatric CMR studies, but should be noted when administered during the examination.
11. Findings
    See the sections below on how to report the results of imaging techniques and findings according to specific lesions.
12. Post-processing
    The method of analysis should be reported based on SCMR post-processing recommendations [28]
13. Comparison with previous relevant studies: Dates, types (e.g., CT, echocardiography, catheterization, nuclear medicine) and how key results of current exam compare with prior studies. It should be stated if parameters from the prior study were manually re-measured.
14. Report summary: The SCMR recommends that every CMR report have a clearly demarcated section summarizing the major findings and impressions of the CMR. While there is practice variation with regards to whether this summary is at the beginning or end of the report, it should be consistent between reports at a given

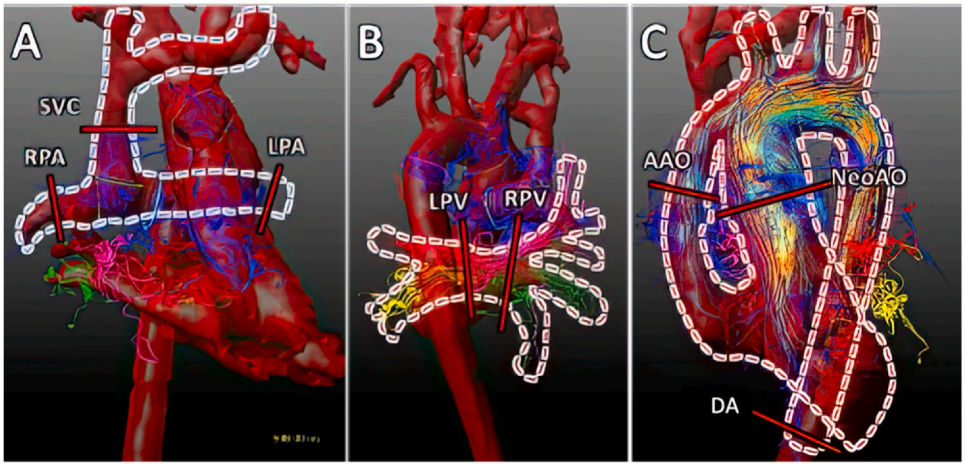

Fig. 3. Blood flow evaluation in a patient after stage two/bidirectional cavopulmonary anastomosis/Glenn operation. Volume-rendered images from a 4DF acquisition. The red lines represent the planes for flow quantification. (A) Systemic venous flow phase (anterior view), (B) pulmonary vein flow phase (posterior view), and (C) systemic arterial flow phase (anterior view). AAO: ascending aorta, LPA: left pulmonary artery, LPV: left pulmonary veins, NeoAO: neo-aorta (native pulmonary artery), RPA: right pulmonary artery, RPV: right pulmonary veins, SVC: superior vena cava, DA: descending aorta.

institution, so that the reader can identify the report conclusion quickly. This section should list the important take-home points for the reader explicitly but concisely.

15. Physician signature: The final report must be reviewed, signed, and dated, either manually or electronically, by the interpreting physician, including medical registration number. Amendments to the report should also be date- and time-stamped.

7. Disease-specific reporting

As outlined above, recommended reporting elements for important lesions that are investigated by CMR are shown below in "normal font" with optional items shown in "*italics*." Space constraints prohibit an exhaustive depiction of all possible findings for each lesion or for the provision of a detailed rationale for the suggested reporting elements. In some instances, recommendations are conditional upon the availability of the necessary images.

1. LV outflow tract and aorta
   The following should be reported for all LV outflow tract and aortic arch (AA) evaluations:
   - Right ventricular (RV) and LV volumes (indexed to BSA) and ejection fractions (EFs)
   - LV mass (indexed to BSA)
   - Right (RA) and left atrial (LA) size (qualitative) *and area ± volume (quantitative)*
   - *Myocardial native T1, extracellular volume (ECV), late gadolinium enhancement (LGE) (optional, if images obtained)*
   - General morphology:
     - AA sidedness
       - Branching of the neck vessels (normal, aberrant artery, variants including bicarotid trunk or separate vertebral artery)
     - Dimensions: Aortic root (ARoot), ascending aorta (AAo), AA
       - Measurements on ECG-gated acquisitions, such as 3D whole heart and cine steady-state free precession (SSFP) sequences are recommended. For the ARoot, in particular, non-gated angiographic have the potential to introduce blurriness due to the vessel distensibility during the cardiac cycle. For the AAo and the AA, measurements on non-gated angiograms are acceptable, although not preferable. Heterogenous approaches exist with regards to the timing of the measurements within the cardiac cycle: Measuring in end-diastole reduces dephasing artifacts in the presence of stenoses; additionally, there is less motion, resulting in better reproducibility for measurements. [29] However, diastolic measurements may result in an under-estimation of the vessel dimension as arteries are largest in systole. If compatibility with echocardiographic guidelines (American College of Cardiology [30] and American Heart Association [31]) is a priority, all aortic measurements except for the annulus should be performed during end-diastole as recommended by the SCMR [4,26]. Importantly, the timing of when measurements are obtained in the cardiac cycle (systole/diastole) should be specified to facilitate clinical interpretation and comparison of measurements in serial studies.

Table 5
Segmental approach to diagnoses in heterotaxy by segmental level.

Visceral or cardiac segment	Heterotaxy with asplenia	Heterotaxy with polysplenia
Systemic venous connections	IVC on same side as (and anterior to) the aorta, hepatic veins drain into IVC or directly into atria Single or bilateral SVC/s, left SVC drainage to roof of common atrium or unroofed coronary sinus	Interrupted IVC (80%), azygos/hemiazygos drainage into right or left SVC, hepatic veins drain directly into atria Single or bilateral SVC/s
Pulmonary venous connections	TAPVC to systemic vein or atria, pulmonary vein stenosis	Normal, PAPVC or TAPVC to atria
Intracardiac anatomy and shunts	ASD or common atrium, AVSD, VSD, DORV, TGA, pulmonary stenosis or atresia, duct-/collateral-dependent circulation, unbalanced ventricles	Normal, ASD, VSD, AVSD, DORV, subaortic obstruction, balanced or unbalanced ventricles
Bronchopulmonary and lung pattern	Bilateral eparterial bronchi, bilateral trilobed lungs, tracheal bronchi, short bronchial lengths	Bilateral hyparterial bronchi, bilateral bilobed lungs, long bronchial lengths
Spleen	Asplenia	Polysplenia—left or right-sided poly-lobulated spleen, rudimentary splenic fragments
Abdominal organs (other than spleen)	Transverse liver, left- or right-sided stomach, midline gallbladder, midgut malrotation	Transverse liver, left or right-sided stomach, normal gallbladder (or small/absent), extrahepatic biliary atresia

ASD atrial septal defect, *AVSD* atrioventricular septal defect, *DORV* double outlet right ventricle, *IVC* inferior vena cava, *PAPVC* partial anomalous pulmonary venous connections, *SVC* superior vena cava, *TAPVC* total anomalous pulmonary venous connections, *TGA* transposition of the great arteries, *VSD* ventricular septal defect.

- Measurements:
  - Three cusp-cusp (maximal inter-sinus dimension, Fig. 1) and maximum cusp-commissure measurements
  - *Three cusp-commissure measurements*
  - Sinotubular junction
  - AAo at the level of the pulmonary artery
  - For the remainder of the aorta, 3D whole-heart acquisitions are preferred. Alternatively, non-gated CE angiography can provide high-resolution images of the aortic lumen. Measurements on cine SSFP sequences are discouraged.
  - Maximum and minimum diameters in cross-section
  - *Smallest cross-sectional area*
  - Length of stenosis to differentiate between focal stenosis and tubular hypoplasia
  - *Proximal transverse AA (proximal to left common carotid artery)*
  - *Distal transverse AA (proximal to left subclavian artery)*
  - Aortic isthmus
  - Descending aorta (DA) at the diaphragm level

1.1. Aortic stenosis
- Anatomical location of the obstruction
  - o Subvalvular:
    - Mechanism, e.g., membrane/ridge, mitral/left atrioventricular valve chordae, systolic anterior motion of the mitral valve, baffle narrowing
    - Minimal LV outflow tract systolic diameter
  - o Valvular:
    - Aortic valve morphology: Number of cusps (unicuspid, bicuspid, tricuspid), presence and location of raphe/cusp fusion, dysplasia, motion restriction, vegetation, abscess
    - Presence of calcification
    - Function:
      - Aortic valve planimetry of the valve opening area (cm^2) on cine
      - Flow velocity measurements by two-dimensional (2D) and even four-dimensional phase contrast (PC) CMR (4DF) can be unreliable, routine reporting of peak velocities is not generally recommended
  - o Supravalvular
    - Location and diameter of the obstruction
    - Coronary artery ostium location relative to the obstruction and presence of ostial stenosis
    - Associated aortopathy (e.g., aneurysm, arch obstruction)
- Pulmonary valve (suitability for Ross procedure, see section 3)

1.2. Aortic valve regurgitation
- Valve morphology (similar to above under “aortic stenosis”)
- Mechanism of regurgitation (aortic dilation, valve degeneration, valve dysplasia, prolapse)
- Regurgitant fraction and volume (including method used)
- Regurgitant jet orientation, *jet width*
- Semiquantitative degree of regurgitation, especially when quantitative metrics are not available
- Aortic dilatation

1.3. Connective tissue aortopathies
- Presence of arterial tortuosity
- *Vertebral artery tortuosity index*
- Presence of dissection
- Prolapse/regurgitation of the atrioventricular and semilunar valves
- presence or absence of Mitral annular disjunction

1.4. Coarctation of the aorta
- Location and severity of obstruction
- Presence, location, and size of a patent ductus arteriosus (PDA)
- Presence and extent of arterial collateral arteries
- *Amount of collateral flow*
- Aortic dimensions as described above
- Post-stenotic dimensions of the DA
- Appearance of other large and medium-sized arterial branches
- Morphology of the aortic valve
- Flow reversal in DA
- Aortic valve morphology
- If post repair, type of repair, and presence or absence of aneurysm

1.5. Inflammatory aortic disease
- Narrowest cross-sectional diameter of each segment, including the abdominal aorta and major branches
- Qualitative (and quantitative, if possible) aortic wall thickness
- Early and late gadolinium enhancement of the aortic wall
- Myocardial inflammation by parametric mapping, T2 weighted imaging, LGE

2. Vascular-mediated airway and esophageal compression

The presence or absence of the following vascular structures should be reported in all cases of vascular-mediated airway and esophageal compression, along with a description of proximity to the tracheo-bronchial tree:
- AA (sidedness of AA and location of DA relative to the spine)
- Brachiocephalic arteries (origin and proximal course)
- PDA (proximal and distal connections) or markers of a residual ductal ligament
- Pulmonary arteries (origin and proximal course)
- Airways: Tracheal/bronchial compression or branching abnormalities
- Esophagus: Course in relation to the vascular structures and presence of any dilatation

2.1. Vascular ring [30–34]

Common causes of a complete vascular ring include (but are not limited to):
- Persistent double AA
- Right AA with anomalous origin of the left subclavian artery and left-sided PDA/ligamentum arteriosum

2.2. Other causes of airway or esophageal compression by head and neck vessels
- Innominate artery compression of the trachea
- Aberrant right subclavian artery compression of the esophagus

2.3. Pulmonary artery sling: anomalous origin of the left pulmonary artery (LPA) from the right pulmonary artery (RPA)
- Right and left pulmonary blood flows
- Presence of stenosis and diameter of the LPA
- Presence of tracheal narrowing and bronchial branching abnormalities

3. RV outflow tract and pulmonary artery

The following should be reported in all RV outflow tract and pulmonary artery evaluations:
- RV and LV volumes (indexed to BSA), mass (indexed to BSA), and EF [35–37]

- RA and LA size (qualitative) *and area ± volume (quantitative)*
- *Myocardial native T1, ECV, LGE (optional, if images obtained)* [38,39], main and branch pulmonary artery size, flow

3.1. Unrepaired tetralogy of Fallot, "tetralogy type" double right ventricle with subaortic ventricular septal defect (VSD), or pulmonary atresia with VSD and major aortopulmonary collateral arteries (APCs)
- RV outflow tract morphology
- Pulmonary valve morphology
- Pulmonary arterial anatomy and lung supply (central pulmonary arteries, APCs, PDA) including descriptions of stenoses and supply to lung fields (sequestered/dual supply)
- AA anatomy (sidedness and branching pattern)
- Pulmonary (Qp): systemic (Qs) blood flows (see section 7 on "shunts" for details)
- Description of VSD (number, location, size)
- Coronary artery proximal course
- Associated lesions

3.2. Post-operative Tetralogy of Fallot, "tetralogy-type" double outlet right ventricle (DORV) with subaortic VSD, or pulmonary atresia with VSD or common arterial trunk
- RV outflow tract morphology and regional function (including patches/conduits and location relative to the sternum)
- Pulmonary valve/right ventricle-to-pulmonary artery conduit
  - o Morphology
  - o Regurgitant fraction and volume[40]
- Tricuspid valve (qualitative assessment, regurgitant fraction, regurgitant volume, see section 5 on "atrioventricular valve anomalies" for details)
- Pulmonary arteries, (see below)
- LV outflow tract morphology and function
- AA anatomy (sidedness and branching pattern)
- ARoot and thoracic dimensions (qualitative); measurements if appear to appear dilated on quantitative assessment.
- Residual lesions (atrial or VSDs, including Qp:Qs)
- *Coronary artery proximal course (optional, unless planning for transcatheter intervention or pulmonary valve implantation which would require more complete delineation of coronary artery course)*

3.3. Branch pulmonary artery stenosis
- Branch pulmonary artery anatomy
- Right to left flow distribution (report ipsilateral pulmonary venous flow if quantification of arterial flow is not feasible)

3.4. Pulmonary arterial hypertension
- Pulmonary arterial anatomy (dilation/aneurysm, thrombus)
- Flow pattern (short, blunted systolic peak; deep nadir between systole and diastole, secondary diastolic peaks)
- *Pulmonary artery distensibility*
- Estimate of RV pressure by systolic septal position (e.g., curvature of the ventricular septum as an estimate of RV pressure as compared with LV pressure)
- Underlying CHD causes, if present

4. Coronary disorders

The following should be reported for coronary disease in children:
- RV and LV volumes (indexed to BSA) and EF
- Wall motion, presence or absence of ventricular thrombus
- LV mass (indexed to BSA)
- *Myocardial native T1, ECV, LGE (if images obtained)*
- LGE and wall motion by coronary distribution territory
- *Stress myocardial perfusion (if images obtained)*
- Coronary anatomy

4.1. Anomalous Aortic Origin of the Coronary Artery (AAOCA) [41]
- Type of anomalous coronary (right, left, circumflex, left anterior descending, multiple)
- Ostial location of each of the coronary arteries: sinus of origin, radial location (central or eccentric within sinus), vertical location (below or above sinotubular junction)
- Spatial relationship of ostia (either remote/separate coronary ostia, adjacent coronary ostia, single coronary ostium branching within the wall, single coronary ostium branching outside the wall)
- Ostial morphology of the AAOCA (round, oval, slit-like)
- Proximal course of the anomalous coronary artery (interarterial, intramural (estimated length of intramural course if applicable), intraseptal, prepulmonic, interarterial, retroaortic)

4.2. Coronary artery fistula
- Sinus or vessel of origin
- Distal connection (to cardiac chamber, to arterial structure, to venous structure)
- Maximum diameter at origin and at connection
- Presence of associated thrombosis or stenosis
- Presence or absence of aneurysm or thrombus

4.3. Kawasaki disease (coronary and other arteries, e.g., subclavian, axillary, iliac)
- Extent of coronary artery disease: vessels involved
- Findings: ectasia, aneurysm, thrombus, stenosis
- Location of lesion
- Length of lesion
- Cross-sectional caliber of coronary artery (when able to be measured)
- Presence of associated thrombosis or stenosis
- *Wall thickening, inflammation if visible (edema on T2 or enhancement on T1 post-contrast)*

5. Atrioventricular valve anomalies

The following should be reported for all atrioventricular valve pathologies(Nishimura et al. [40])
- Valve anatomy including annulus, leaflets and chordae, papillary muscles
- LV volumes (indexed to BSA) and EF
- LV mass (indexed to BSA)
- RA and LA size (qualitative) *and area ± volume (quantitative)*

5.1. Atrioventricular valve regurgitation [42–45]

(Consider using valve cusp views as described by Garg et al. [42])
- Description of the jet (central/eccentric, single/multiple, early/mid/late/pansystolic) and its relation to the Carpentier's classification of the mitral valve cusps
- Regurgitant fraction and volume
- Indirect flow calculation (describe method used in calculation):
  - o Ventricular stroke volume minus ipsilateral semilunar valve forward flow
  - o Difference between RV and LV stroke volumes, if no other valve regurgitation or intracardiac shunts
  - o Difference between right/left atrioventricular inflows, if no other valve regurgitation or intracardiac shunts
  - o Difference between atrioventricular inflow and ipsilateral semilunar valve net outflow

- Direct flow quantification:
  - o *4DF with valve tracking if available*[46]*; 2D PC-based quantification not recommended*

5.2. Ebstein anomaly
- Anatomy
  - o Degree of atrialization of the right ventricle
    - – Atrialized volume and functional RV volumes (Fig. 2)
  - o Distance of displacement of the septal/posterior tricuspid leaflet toward the apex and morphology of the anterior leaflet
  - o Severity of rotation of the tricuspid valve opening toward the RV outflow tract
  - o *Myocardial native T1, ECV, LGE (optional, if images obtained)*
- Function, volumes, and flows
  - o Functional RV EF (excluding the atrialized portion)
  - o *Total right heart volume index = (RA volume plus total RV volume/(LA volume plus LV volume)*
  - o *Findings of cardiomyopathy (non-compaction)*
  - o Tricuspid regurgitation: as detailed above
  - o Quantification of atrial level shunt (see section 7 on "shunts")
  - o RV outflow: Presence and severity of obstruction and regurgitation (see section 3 for details)
  - o Bidirectional cavopulmonary connection (Glenn) physiology, if present (see section 9 for details)

6. Transposition of the great arteries (TGA)

The following should be reported for all CMR evaluations of transposition of the great arteries (TGA):
- RV and LV volumes (indexed to BSA) and EFs
- LV mass (indexed to BSA)
- RA and LA size (qualitative) *and area ± volume (quantitative)*
- *Myocardial native T1, ECV, LGE* (*if images obtained)*

6.1. Post arterial switch repair [47,48]
- Description of RV and LV outflow tract morphology
- Neo-ARoot morphology and dimensions [49]
- Neo-aortic regurgitation fraction and volume
- Neo-pulmonary valve morphology and pulmonary arterial anatomy, including descriptions of external compression, stretching, hypoplasia, or stenosis
- Branch pulmonary artery differential flow volumes
- Atrioventricular valve function
- Description of origin of coronary arteries and their proximal segments
- *Myocardial perfusion at rest and during stress (if performed)*

6.2. Post atrial switch repair [50]
- Anatomic description of systemic and pulmonary venous baffles, including description of external compression, stretching, stenosis, upstream dilatation, or baffle leaks [51]
- Description of RV and LV outflow tract morphology
- PC flow mapping of the systemic veins, including the azygos/hemiazygos veins to detect decompressing flow from baffle obstruction
- Pulmonary artery morphology
- Coronary artery anatomy
- Atrioventricular valve function

7. Shunt lesions

The following should be reported for all shunt evaluations [52]:
- RV and LV volumes (indexed to BSA) and EFs
- LV mass (indexed to BSA)
- RA and LA size (qualitative) *and area ± volume (quantitative)*
- Qp, Qs, and Qp:Qs ratio (see Table 3 for ways to quantify Qs depending on lesion) [53–55]

7.1. Atrial septal defect
- Number and location of communication(s)
- Defect size (longest and shortest diameters from *en-face* views)
- Rim sizes
- Associated lesions (pulmonary venous connections, coronary sinus, atrioventricular valves, ventricular septum)

7.2. Ventricular septal defect
- Number and location of communication(s) and relationship with semilunar valves.
- Defect size (longest and shortest diameters)
- Associated lesions (atrial septum, atrioventricular valves, semilunar valves, outlet septum, arterial relationship, AA)

7.3. Patent ductus arteriosus
- Location including proximal and distal connections
- Morphology, tortuosity, diameters at aortic (ampulla) and pulmonary ends
- *Cross-sectional diameters at aortic and pulmonary end*
- *Length*
- Flow amount and net flow direction across duct
- Flow in DA
- Associated lesions (intracardiac anatomy, great arteries, arch)

8. Pulmonary vein pathologies

The following should be reported for all pulmonary vein abnormalities:
- RV and LV volumes (indexed to BSA) and EFs
- LV mass (indexed to BSA)
- RA and LA size (qualitative) *and area ± volume (quantitative)*
  - o Qp and flow in individual pulmonary veins. If pulmonary venous flow cannot be quantified by direct measurement, it may be estimated by subtracting the flow in the measurable veins from pulmonary arterial flow

8.1. Pulmonary vein stenosis [56]
- Location, severity, and extent of stenosis (long vs short segment, distance from ostium [57,58]
- Narrowest diameter of stenosed pulmonary vein(s)
- Upstream diameter of stenosed pulmonary vein(s)
- *Cross-sectional diameter of* stenosed *pulmonary vein(s)*
- Pulmonary venous flow pattern: Phasic vs non-phasic, peak velocity
- Pulmonary arterial flow pattern (see section 3 on "pulmonary arterial hypertension")
- Associated lesions (e.g., atrial septal defect, VSD)

8.2. Partial anomalous pulmonary venous connections (PAPVCs) [55]
- See section 7 for additional reporting elements
- Connection of individual pulmonary veins (detailed description of anomalous connections, such as distance to cavo-atrial junction, above or below main bronchi and azygos vein)
- Associated lesions (e.g., sinus venosus or secundum atrial septal defect, VSD)

8.3. Scimitar syndrome
- Similar to PAPVCs (see above)
- Description of Scimitar vein including distance to cavo-atrial junction, relationship to diaphragm

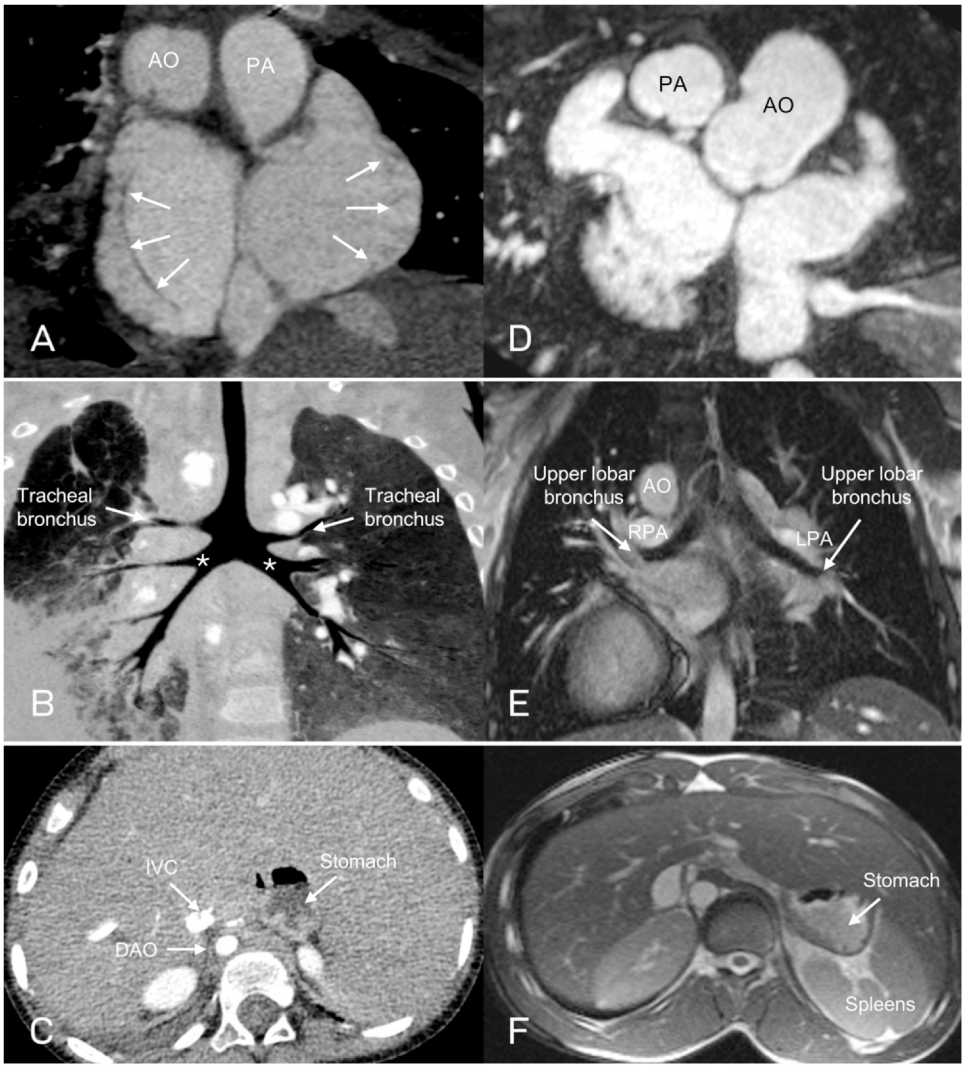

Fig. 4. A. Bilateral atrial appendages are of right atrial appendage morphology with pectinate muscles (arrows) seen along the atrial walls. B. Symmetric airway branching with bilateral tracheal bronchi and short lengths of the main bronchi (asterisks). C. The liver is midline and the abdominal aorta (DAO) and inferior vena cava (IVC) are on the same side of the spine. D. Bilateral atrial appendages are of tubular morphology. E. There are bilateral epbronchial pulmonary arteries and hyparterial upper lobar bronchi. F. The liver is transverse with two spleens seen along the posterolateral wall of the stomach. The IVC was interrupted in this patient. Abbreviations: AO: aorta; LPA: left pulmonary artery; PA: pulmonary artery; RPA: right pulmonary artery (Figure from Yim D, Nagata H, Lam CZ, Grosse-Wortmann L, Seed M, Jaeggi E, et al. Disharmonious patterns of heterotaxy and isomerism: how often are the classic patterns breached? Circ Cardiovasc Imaging 11(2):e006917. American Heart Association. https://www.ahajournals.org/journal/circimaging [64] reprinted with permission).

- Location and extent of stenosis (if present)
- Associated cardiac and pulmonary lesions (systemic arterial collateral from abdominal aorta, lung sequestration, ipsilateral RPA and lung hypoplasia)

9. Single ventricle physiology

The following should be reported for all patients with single ventricle physiology or borderline left ventricles, as applicable according to their stage of palliation (before bidirectional cavopulmonary connection (Glenn), before total cavopulmonary connection (Fontan), and after total cavopulmonary connection (Fontan)):

- RV and LV volumes (indexed to BSA), mass (indexed to BSA), and EFs
  - o In patients with two sizeable ventricles and in whom both contribute to the systemic circulation (e.g., when connected via a VSD), the volumes and EFs of both ventricles may be reported separately and/or combined (e.g., DORV, "remote" VSD, and single ventricle palliation[59]).
  - o In patients with a hypoplastic ventricle and no VSD and semilunar atresia, that ventricle should not be included in volumetric quantification as it does not contribute to the systemic circulation (e.g., hypoplastic left heart with mitral stenosis and aortic atresia).
- RA and LA size (qualitative) *and area ± volume (quantitative)*
- *Myocardial native T1, ECV, LGE* (*optional, if images obtained)*
- Inferior vena cava (IVC), superior vena cava (SVC, if bilateral SVCs, note presence/absence of innominate vein)
- Hepatic veins (describe connections)
- Pulmonary veins
- Cavopulmonary anastomoses
- Fontan pathway, including presence of a fenestration and any baffle leaks
- Presence, location, and size of thrombus
- Branch pulmonary arteries (see section 3). Describe mechanism of obstruction if possible (e.g., compression by dilated AAo) and its relation to airways.
- Outflow tract anatomy (if patent report orthogonal measurements of the native aorta and neo-aorta)
- Damus-Kaye-Stansel anastomosis
- AA anatomy (describe arch sidedness and branching pattern and report orthogonal measurements of the AAo, transverse arch, and aortic isthmus)
- Any vascular shunts (Blalock-Taussig-Thomas, right ventricle-to-pulmonary artery, or central)
- Presence of visible APCs or veno-venous collaterals (including origin and connections)
- Blood flows:
  - o PDA, any vascular shunt (Blalock-Taussig-Thomas, right-ventricle-to-pulmonary artery, or central)
  - o SVC and IVC (below fenestration)

Table 6
Common extracardiac incidental CMR findings.

Location	Common pathology	Imaging findings	Recommendation
Neck	Goiter or focal nodules	Thyroid nodular lesions with signal different than the thyroid parenchyma	Correlation with clinical and laboratory findings with ultrasound and tissue sampling as necessary
Mediastinum/ airway	Lymphadenopathy, tumors, fibrosing mediastinitis, airway compromise	Enlarged lymph nodes, soft tissue masses, distortion of vascular structures, airway narrowing	Chest CT for further assessment of mediastinal masses/lymphadenopathy, fixed vs dynamic airway compromise
Lung/pleura	Lung nodules, Infection, effusion	Focal lung nodules due to infection or neoplasm, lung consolidation vs atelectasis, free or loculated pleural fluid	Correlation with underlying systemic disease and previous imaging to determine need for and type of further workup
Breast	Breast masses	Breast nodular lesions with signal different from breast tissue	Clinical exam and ultrasound as needed
Liver	Benign or malignant masses, diffuse liver disease, regenerating nodules	Nodules with signal different from liver parenchyma, atrophy, diffuse nodularity	Correlation with clinical/laboratory findings and prior imaging to determine further workup for focal or diffuse liver disease
Kidneys	Benign or malignant masses, cysts	Nodules or cysts with signal different from renal parenchyma, congenital renal anomalies like horseshoe kidney or renal agenesis	Correlation with prior imaging to determine stability and need for further characterization by ultrasound
Spleen	Mostly benign masses	Nodules with signal different from splenic parenchyma	Correlation with presence of systemic disease and prior imaging to determine stability and need for further characterization
Bones	Metastatic disease and primary bone tumors	Bone deformity and areas of bone with signal different than normal medullary or cortical bone	Correlation with conventional radiography is usually the next step in the workup of incidental bony abnormalities

- o Azygos/Hemiazygos veins (if sufficiently large to measure, including flow direction)
- o Fontan baffle (above fenestration)
- o RPA and LPA, differential pulmonary arterial blood flow percentages[60]
- o Neo-aorta, native aorta, AAo (above the Damus-Kaye-Stansel anastomosis), DA (at the level of the diaphragm).
- o Pulmonary veins
- o Atrioventricular valve inflow (see section 5 on "atrioventricular valve anomalies" for details)
- o Qp, Qs, APC flow: absolute, as a percentage of systemic flow and as a percentage of pulmonary venous return (see Table 4 and Fig. 3 for quantification options to quantify Qs depending on lesion [61])
- o Semilunar regurgitation

- Qualitative description of lymphatic congestion (including degree and location) and status of the thoracic duct (patent vs occluded), if visible [62]

10. Situs anomalies—heterotaxy

To navigate potential discordant patterns of heterotaxy between segmental levels (e.g., right-sided stomach and midline liver with thoracic situs solitus), we recommend describing the anatomy in detail at individual levels and to avoid categorization for cases that do not display visceroatrial concordance.[63–65] Importantly, these associations are "trends" and are not found in all patients [66]. Table 5 and Fig. 4 illustrate the typical findings associated with asplenia (Fig. 4 A-C) and with polysplenia (Fig. 4 D-F).

The following elements should be included in all patients with heterotaxy:

- Cardiac orientation and position
  - o Levocardia, dextrocardia, or mesocardia
  - o Levoposition, dextroposition, or mesoposition
- Systemic venous connections
  - o Presence and location of renal-hepatic and suprahepatic segments of IVC and connection to atria
  - o Hepatic venous drainage
  - o Presence and morphology of the coronary sinus
  - o Azygos and hemiazygos connections
  - o Unilateral or bilateral SVCs and connections
- Pulmonary venous connections
  - o Presence of total or PAPVCs
  - o Description of pulmonary vein stenosis (if present)
- Intracardiac anatomy and shunts
  - o Atrial appendage morphology
  - o Atrioventricular valves, semilunar valves and great arterial relationship
  - o Ventricular size and morphology of dominant or hypoplastic ventricles
  - o Presence and significance of other intracardiac and/or extracardiac shunts
  - o Morphology of main and branch pulmonary arteries
- Extracardiac associations
  - o Bronchial branching pattern (bilateral eparterial or hyparterial bronchi)
  - o *Lung lobation*
  - o Presence, position, size, and morphology of spleen(s)
  - o Abdominal organ morphology (other than spleen): liver, stomach, *gall bladder, pancreas, adrenals, kidneys, bowel (mal)rotation*
  - o *Abdominal vasculature: abdominal aortic branching, IVC, portal system anatomy, hepatic veins*

11. Extracardiac findings

Common extracardiac findings on CMR imaging are summarized in Table 6.

8. Conclusions

The use of a structured reporting approach to CMR examinations in patients with CHD facilitates the interpretation of the imaging data and sharing of relevant information with the clinical team as well as its use for quality improvement and scientific discovery. Quantitative measurements that are consistently assessed with each CMR examination can identify clinically relevant changes and help with medical decision making and/or for the timing of interventions. A step wise approach, individually tailored for each CHD lesion as outlined above, serves to simplify the report writing, ensure generalizability, promote reproducibility and enhances the clinical relevance of the report.

Funding

None.

Author contributions

Lars Grosse-Wortmann: Conceptualization, Methodology, Project administration, Supervision, Writing – original draft, Writing – review and editing. **Rahul H. Rathod:** Project administration, Writing – original draft, Writing – review and editing. **Sonya V. Babu-Narayan:** Writing – original draft, Writing – review and editing. **Lorna Browne:** Writing – original draft, Writing – review and editing. **Deane Yim:** Writing – original draft, Writing – review and editing. **Rajesh Krishnamurthy:** Writing – original draft, Writing – review and editing. **Francesca Raimondi:** Writing – original draft, Writing – review and editing. **Karen Ordovas:** Writing – original draft, Writing – review and editing. **Emanuela Valsangiacomo-Buechel:** Writing – original draft, Writing – review and editing. **Israel Valverde:** Writing – original draft, Writing – review and editing. **Rachel M. Wald:** Writing – original draft, Writing – review and editing.

Declaration of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. Dr. Rathod receives research grant support from Mezzion Pharmaceuticals.

References

[1] Fratz S, Chung T, Greil GF, Samyn MM, Taylor AM, Valsangiacomo Buechel ER, et al. Guidelines and protocols for cardiovascular magnetic resonance in children and adults with congenital heart disease: SCMR expert consensus group on congenital heart disease. J Cardiovasc Magn Reson [Internet] 2013;15. https://pubmed.ncbi.nlm.nih.gov/23763839/ (accessed April 14, 2022).

[2] Hundley WG, Bluemke DA, Bogaert J, Flamm SD, Fontana M, Friedrich MG, et al. Society for Cardiovascular Magnetic Resonance (SCMR) guidelines for reporting cardiovascular magnetic resonance examinations. J Cardiovasc Magn Reson [Internet] 2022;24:29. http://www.ncbi.nlm.nih.gov/pubmed/35484555 (accessed May 19, 2022).

[3] Bunck AC, Baeβler B, Ritter C, Kröger JR, Persigehl T, Pinto Santos D, et al. Structured reporting in cross-sectional imaging of the heart: reporting templates for CMR imaging of cardiomyopathies (myocarditis, dilated cardiomyopathy, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and siderosis). RoFo Fortschr Geb Rontgenstrahlen Bildgeb Verfahr 2020;192:27–37.

[4] Schulz-Menger J, Bluemke DA, Bremerich J, Flamm SD, Fogel MA, Friedrich MG, et al. Standardized image interpretation and post-processing in cardiovascular magnetic resonance - 2020 update: Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing. J Cardiovasc Magn Reson 2020;22.

[5] Frazao C, Tavoosi A, Wintersperger BJ, Nguyen ET, Wald RM, Ouzounian M, et al. Multimodality assessment of thoracic aortic dimensions: comparison of computed tomography angiography, magnetic resonance imaging, and echocardiography measurements. J Thorac Imaging [Internet] 2020;35:399–406. https://pubmed.ncbi.nlm.nih.gov/32251236/ (accessed May 17, 2022).

[6] Kaiser T, Kellenberger CJ, Albisetti M, Bergsträsser E, Buechel ERV. Normal values for aortic diameters in children and adolescents–assessment in vivo by contrast-enhanced CMR-angiography. J Cardiovasc Magn Reson [Internet] 2008;10. https://pubmed.ncbi.nlm.nih.gov/19061495/ (accessed May 17, 2022).

[7] Buechel EV, Kaiser T, Jackson C, Schmitz A, Kellenberger CJ. Normal right- and left ventricular volumes and myocardial mass in children measured by steady state free precession cardiovascular magnetic resonance. J Cardiovasc Magn Reson [Internet] 2009;11. https://pubmed.ncbi.nlm.nih.gov/19545393/ (accessed May 17, 2022).

[8] Robbers-Visser D, Boersma E, Helbing WA. Normal biventricular function, volumes, and mass in children aged 8 to 17 years. J Magn Reson Imaging [Internet] 2009;29:552–9. https://pubmed.ncbi.nlm.nih.gov/19243036/> (accessed May 17, 2022).

[9] Sarikouch S, Peters B, Gutberlet M, Leismann B, Kelter-Kloepping A, Koerperich H, et al. Sex-specific pediatric percentiles for ventricular size and mass as reference values for cardiac MRI: assessment by steady-state free-precession and phase-contrast MRI flow. Circ Cardiovasc Imaging [Internet] 2010;3:65–76. https://pubmed.ncbi.nlm.nih.gov/19820203/ (accessed May 17, 2022).

[10] Van Der Ven JPG, Sadighy Z, Valsangiacomo Buechel ER, Sarikouch S, Robbers-Visser D, Kellenberger CJ, et al. Multicentre reference values for cardiac magnetic resonance imaging derived ventricular size and function for children aged 0–18 years. Eur Heart J Cardiovasc Imaging [Internet] 2020;21:102–13. https://pubmed.ncbi.nlm.nih.gov/31280290/ (accessed May 17, 2022).

[11] Sluysmans T, Colan SD. Theoretical and empirical derivation of cardiovascular allometric relationships in children. J Appl Physiol (1985) [Internet] 2005;99:445–57. https://pubmed.ncbi.nlm.nih.gov/15557009/ (accessed May 17, 2022).

[12] Boyd E. The Growth of the Surface Area of the Human Body. Minnesota: University of Minnesota Press; 1935.

[13] Du Bois D, Du Bois E. A formula to estimate the approximate surface area if height and weight be known. Arch Intern Med 1916;17:863–71.

[14] Gehan EA, George SL. Estimation of human body surface area from height and weight. Cancer Chemother Rep Part 1 1970;54:225–35.

[15] Haycock GB, Schwartz GJ, Wisotsky DH. Geometric method for measuring body surface area: a height-weight formula validated in infants, children, and adults. J Pediatr [Internet] 1978;93:62–6. https://pubmed.ncbi.nlm.nih.gov/650346/ (accessed April 13, 2022).

[16] Mosteller RD. Simplified calculation of body-surface area. N Engl J Med 1987;317:1098.

[17] Livingston EH, Lee S. Body surface area prediction in normal-weight and obese patients. Am J Physiol Endocrinol Metab 2001;281:E586–91.

[18] Yu CY, Lin CH, Yang YH. Human body surface area database and estimation formula. Burns 2010;36:616–29.

[19] Kawel-Boehm N, Maceira A, Valsangiacomo-Buechel ER, Vogel-Claussen J, Turkbey EB, Williams R, et al. Normal values for cardiovascular magnetic resonance in adults and children. J Cardiovasc Magn Reson 2015;17.

[20] Kawel-Boehm N, Hetzel SJ, Ambale-Venkatesh B, Captur G, Francois CJ, Jerosch-Herold M, et al. Reference ranges ("normal values") for cardiovascular magnetic resonance (CMR) in adults and children: 2020 update. J Cardiovasc Magn Reson 2020;22.

[21] Voges I, Jerosch-Herold M, Hedderich J, Pardun E, Hart C, Gabbert DD, et al. Normal values of aortic dimensions, distensibility, and pulse wave velocity in children and young adults: a cross-sectional study. J Cardiovasc Magn Reson [Internet] 2012;14. https://pubmed.ncbi.nlm.nih.gov/23151055/ (accessed May 18, 2022).

[22] Kutty S, Kuehne T, Gribben P, Reed E, Li L, Danford DA, et al. Ascending aortic and main pulmonary artery areas derived from cardiovascular magnetic resonance as reference values for normal subjects and repaired tetralogy of Fallot. Circ Cardiovasc Imaging [Internet] 2012;5:644–51. https://pubmed.ncbi.nlm.nih.gov/22730421/ (accessed May 18, 2022).

[23] Knobel Z, Kellenberger CJ, Kaiser T, Albisetti M, Bergsträsser E, Valsangiacomo Buechel ER. Geometry and dimensions of the pulmonary artery bifurcation in children and adolescents: assessment in vivo by contrast-enhanced MR-angiography. Int J Cardiovasc Imaging [Internet] 2011;27:385–96. https://pubmed.ncbi.nlm.nih.gov/20652636/ (accessed May 18, 2022).

[24] Van Praagh R. Letter to the editor. Cardiovasc Interv Radiol 1984;7:320–5.

[25] Van Praagh R. Terminology of congenital heart disease. Glossary and commentary. Circulation [Internet] 1977;56:139–43. https://pubmed.ncbi.nlm.nih.gov/872303/ (accessed May 18, 2022).

[26] Anderson RH, Shirali G. Sequential segmental analysis. Ann Pediatr Cardiol [Internet] 2009;2:24–35. http://www.ncbi.nlm.nih.gov/pubmed/20300266 (accessed May 18, 2022).

[27] Anderson RH, Becker AE, Freedom RM, Macartney FJ, Quero-Jimenez M, Shinebourne EA, et al. Sequential segmental analysis of congenital heart disease. Pediatr Cardiol 1984;5:281–7.

[28] Schulz-Menger J, Bluemke DA, Bremerich J, Flamm SD, Fogel MA, Friedrich MG, et al. Standardized image interpretation and post-processing in cardiovascular magnetic resonance - 2020 update: Society for Cardiovascular Magnetic Resonance (SCMR): Board of Trustees Task Force on Standardized Post-Processing. J Cardiovasc Magn Reson 2020;22.

[29] van Hout MJ, Scholte AJ, Juffermans JF, Westenberg JJ, Zhong L, Zhou X, et al. How to measure the aorta using MRI: a practical guide. J Magn Reson Imaging [Internet] 2020;52:971–7. https://pubmed.ncbi.nlm.nih.gov/32374422/ (accessed 2022 May 17, 2022).

[30] Lang RM, Badano LP, Victor MA, Afilalo J, Armstrong A, Ernande L, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015;28:1–39.

[31] Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, et al. ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: executive summary: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association. Circulation 2010;2010:121.

[32] Kellenberger CJ. Aortic arch malformations. Pediatr Radiol 2010;40:876–84.

[33] Rogers DJ, Cunnane MB, Hartnick CJ. Vascular compression of the airway: establishing a functional diagnostic algorithm. JAMA Otolaryngol Head Neck Surg [Internet] 2013;139:586–91. https://pubmed.ncbi.nlm.nih.gov/23787416/ (accessed May 17, 2022).

[34] Hanneman K, Newman B, Chan F. Congenital variants and anomalies of the aortic arch. Radiographics 2017;37:32–51.

[35] Ghonim S, Gatzoulis MA, Ernst S, Li W, Moon JC, Smith GC, et al. Predicting survival in repaired tetralogy of Fallot: a lesion-specific and personalized approach. JACC Cardiovasc Imaging [Internet] 2022;15:257–68. https://pubmed.ncbi.nlm.nih.gov/34656466/ (accessed May 17, 2022).

[36] Geva T. Repaired tetralogy of Fallot: the roles of cardiovascular magnetic resonance in evaluating pathophysiology and for pulmonary valve replacement decision support. J Cardiovasc Magn Reson [Internet]. 2011;13. https://pubmed.ncbi.nlm.nih.gov/21251297/ (accessed May 17, 2022).

[37] Valente AM, Gauvreau K, Assenza GE, Babu-Narayan SV, Schreier J, Gatzoulis MA, et al. Contemporary predictors of death and sustained ventricular tachycardia in patients with repaired tetralogy of Fallot enrolled in the INDICATOR cohort. Heart

[Internet] 2014;100:247–53. https://pubmed.ncbi.nlm.nih.gov/24179163/ (accessed May 17, 2022).
[38] Yim D, Riesenkampff E, Caro-Dominguez P, Yoo SJ, Seed M, Grosse-Wortmann L. Assessment of diffuse ventricular myocardial fibrosis using native T1 in children with repaired tetralogy of Fallot. Circ Cardiovasc Imaging 2017;10.
[39] Hanneman K, Crean AM, Wintersperger BJ, Thavendiranathan P, Nguyen ET, Kayedpour C, et al. The relationship between cardiovascular magnetic resonance imaging measurement of extracellular volume fraction and clinical outcomes in adults with repaired tetralogy of Fallot. Eur Heart J Cardiovasc Imaging 2018;19:777–84.
[40] Wald RM, Redington AN, Pereira A, Provost YL, Paul NS, Oechslin EN, et al. Refining the assessment of pulmonary regurgitation in adults after tetralogy of Fallot repair: should we be measuring regurgitant fraction or regurgitant volume? Eur Heart J 2009;30:356–61.
[41] Krishnamurthy R, Masand PM, Jadhav SP, Molossi S, Zhang W, Agrawal HM, et al. Accuracy of computed tomography angiography and structured reporting of high-risk morphology in anomalous aortic origin of coronary artery: comparison with surgery. Pediatr Radiol [Internet] 2021;51:1299–310. https://pubmed.ncbi.nlm.nih.gov/33755749/ (accessed April 14, 2022).
[42] Garg P, Swift AJ, Zhong L, Carlhäll CJ, Ebbers T, Westenberg J, et al. Assessment of mitral valve regurgitation by cardiovascular magnetic resonance imaging. Nat Rev Cardiol 2020;17:298–312.
[43] Krieger EV, Lee J, Branch KR, Hamilton-Craig C. Quantitation of mitral regurgitation with cardiac magnetic resonance imaging: a systematic review. Heart [Internet] 2016;102:1864–70. https://pubmed.ncbi.nlm.nih.gov/27733535/ (accessed May 18, 2022).
[44] Zoghbi WA, Adams D, Bonow RO, Enriquez-Sarano M, Foster E, Grayburn PA, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr [Internet] 2017;30:303–71. https://pubmed.ncbi.nlm.nih.gov/28314623/ (accessed May 18, 2022).
[45] Uretsky S. Quantification of mitral regurgitation using MRI - American College of Cardiology [Internet]. American College of Cardiology. 2016. https://www.acc.org/latest-in-cardiology/articles/2016/06/28/07/41/quantification-of-mitral-regurgitation-using-mri (accessed February 11, 2024).
[46] Feneis JF, Kyubwa E, Atianzar K, Cheng JY, Alley MT, Vasanawala SS, et al. 4D flow MRI quantification of mitral and tricuspid regurgitation: reproducibility and consistency relative to conventional MRI. J Magn Reson Imaging [Internet] 2018;48:1147–58. https://pubmed.ncbi.nlm.nih.gov/29638024/ (accessed May 18, 2022).
[47] Raimondi F, Aquaro GD, De Marchi D, Sandrini C, Khraiche D, Festa P, et al. Cardiac magnetic resonance myocardial perfusion after arterial switch for transposition of great arteries. JACC Cardiovasc Imaging 2018;11:778–9.
[48] Shepard CW, Germanakis I, White MT, Powell AJ, Co-Vu J, Geva T. Cardiovascular magnetic resonance findings late after the arterial switch operation. Circ Cardiovasc Imaging 2016;9.
[49] Martins D, Khraiche D, Legendre A, Boddaert N, Raisky O, Bonnet D., et al. Aortic angle is associated with neo-aortic root dilatation and regurgitation following arterial switch operation. Int J Cardiol [Internet] 2019;280:53–6. https://pubmed.ncbi.nlm.nih.gov/30660585/ (accessed May 23, 2022).
[50] Samyn MM, Yan K, Masterson C, Goot BH, Saudek D, Lavoie J, et al. Echocardiography vs cardiac magnetic resonance imaging assessment of the systemic right ventricle for patients with d-transposition of the great arteries status post atrial switch. Congenit Heart Dis [Internet]. 2019;14:1138–48. https://pubmed.ncbi.nlm.nih.gov/31816182/ (accessed May 23, 2022).
[51] Arthur L, Schoeneberg L, Angtuaco M, Greenberg SB, Renno MS, Das S. Advanced imaging improves detection of baffle leaks and stenoses after atrial switch compared with transthoracic echocardiography. Int J Cardiovasc Imaging [Internet] 2021;37:2767–72. https://pubmed.ncbi.nlm.nih.gov/33864562/ (accessed May 23, 2022).
[52] Higgins CB, Holt W, Pflugfelder P, Sechtem U. Functional evaluation of the heart with magnetic resonance imaging. Magn Reson Med [Internet] 1988;6:121–39. https://pubmed.ncbi.nlm.nih.gov/3367771/ (accessed May 24, 2022).
[53] Hanneman K, Sivagnanam M, Nguyen ET, Wald R, Greiser A, Crean AM, et al. Magnetic resonance assessment of pulmonary (QP) to systemic (QS) flows using 4D phase-contrast imaging: pilot study comparison with standard through-plane 2D phase-contrast imaging. Acad Radiol [Internet] 2014;21:1002–8. https://pubmed.ncbi.nlm.nih.gov/25018072/ (accessed May 24, 2022).
[54] Beerbaum P, Körperich H, Barth P, Esdorn H, Gieseke J, Meyer H. Noninvasive quantification of left-to-right shunt in pediatric patients: phase-contrast cine magnetic resonance imaging compared with invasive oximetry. Circulation 2001;103:2476–82.
[55] Seller N, Yoo SJ, Grant B, Grosse-Wortmann L. How many versus how much: comprehensive haemodynamic evaluation of partial anomalous pulmonary venous connection by cardiac MRI. Eur Radiol 2018;28:4598–606.
[56] Grosse-Wortmann L, Al-Otay A, Woo Goo H, Macgowan CK, Coles JG, Benson LN, et al. Anatomical and functional evaluation of pulmonary veins in children by magnetic resonance imaging. J Am Coll Cardiol 2007;49:993–1002.
[57] Kotani Y, Zhu J, Grosse-Wortmann L, Honjo O, Coles JG, Van Arsdell GS, et al. Anatomical risk factors, surgical treatment, and clinical outcomes of left-sided pulmonary vein obstruction in single-ventricle patients. J Thorac Cardiovasc Surg 2015;149:1332–8.
[58] Tremblay C, Yoo SJ, Mertens L, Seed M, Jacques F, Slorach C, et al. Sutureless versus conventional pulmonary vein repair: a magnetic resonance pilot study. Ann Thorac Surg 2018;105:1248–54.
[59] Meyer SL, St. Clair N, Powell AJ, Geva T, Rathod RH. Integrated clinical and magnetic resonance imaging assessments late after Fontan operation. J Am Coll Cardiol [Internet] 2021;77:2480–9. https://pubmed.ncbi.nlm.nih.gov/34016261/ (accessed February 27, 2022).
[60] Alsaied T, Sleeper LA, Masci M, Ghelani SJ, Azcue N, Geva T, et al. Maldistribution of pulmonary blood flow in patients after the Fontan operation is associated with worse exercise capacity. https://doi.org/10.1186/s12968-018-0505-4 (accessed February 27, 2022).
[61] Pisesky A, Reichert MJE, De Lange C, Seed M, Yoo S-J, Lam CZ, et al. Prevalence and clinical significance of cardiovascular magnetic resonance adenosine stress-induced myocardial perfusion defect in hypertrophic cardiomyopathy. J Cardiovasc Magn Reson [Internet] 2020;23:134. https://doi.org/10.1186/s12968-021-00782-9 (accessed February 27, 2022).
[62] Biko DM, DeWitt AG, Pinto EM, Morrison RE, Johnstone JA, Griffis H, et al. MRI evaluation of lymphatic abnormalities in the neck and thorax after Fontan surgery: relationship with outcome. Radiology [Internet] 2019;291:774–80. https://pubmed.ncbi.nlm.nih.gov/30938628/ (accessed February 27, 2022).
[63] Loomba RS, Pelech AN, Shah PH, Anderson RH. Determining bronchial morphology for the purposes of segregating so-called heterotaxy. Cardiol Young [Internet] 2016;26:725–37. https://pubmed.ncbi.nlm.nih.gov/26159655/ (accessed September 30, 2022).
[64] Yim D, Nagata H, Lam CZ, Grosse-Wortmann L, Seed M, Jaeggi E, et al. Disharmonious patterns of heterotaxy and isomerism: how often are the classic patterns breached? Circ Cardiovasc Imaging 2018;11(2):e006917.
[65] Jacobs JP, Anderson RH, Weinberg PM, Walters HL, Tchervenkov CI, Del Duca D, et al. The nomenclature, definition and classification of cardiac structures in the setting of heterotaxy. Cardiol Young [Internet] 2007;17(Suppl 2):1–28. https://pubmed.ncbi.nlm.nih.gov/18039396/ (accessed September 30, 2022).
[66] Krishnamurthy R, Chung T. Other Complex Congenital Heart Disease – Heterotaxy, complex spatial relationships, conjoined twins, and ectopia cordis. 1st ed Fogel M, editor. Principles and Practice of Cardiac Magnetic Resonance in Congenital Heart Disease Oxford, UK: Wiley-Blackwell Publishing; 2010. p. 265–86.