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**How to write**

**How long does a pediatric echo take?**

**Pediatric echo review.**



How to write an echo report. How long does a pediatric echo take. Pediatric echo review.

**Learning objectives** By reading this article, you should be able to: • Explain the sequential segmental approach to echocardiography used in paediatric cardiology. • Describe methods of evaluating the significance of echocardiographic measurements in paediatrics. • Illustrate the echocardiography findings seen in specific paediatric congenital cardiac conditions. **Key points** • Echocardiography is the primary imaging modality in the assessment of heart disease in children. • The report follows a standard structure covering sequential segmental analysis of cardiovascular structures, measurements and functional assessments. • Cardiovascular measurements in paediatrics are greatly influenced by the patient's size and age; this adds additional challenges to interpretation. • A basic understanding of paediatric cardiac disease and echocardiography enables clinically useful information to be obtained. • Clinicians must interpret the report in the context of the individual patient, considering their diagnosis, current status and the planned procedure. Echocardiography (echo) is the primary imaging modality in the assessment of congenital heart defects (CHD) and acquired heart disease in children, and can provide valuable data to inform perioperative care. Because of the wide spectrum of anomalies occurring in paediatrics, understanding and interpreting the paediatric echo report can be challenging. This article aims to help anaesthetists unfamiliar with paediatric echo to obtain information from the report that is useful in practice. We review the key sequences of the examination and subsequent report, covering cardiovascular structures, haemodynamic measurements and functional assessments, and consider echo findings in specific congenital cardiac conditions. The specific details of various congenital heart lesions and perioperative management are outside the scope of this article. As with all investigations, clinicians must interpret echo results in the context of the clinical history, examination findings and underlying cardiac diagnoses. A discussion of the clinical details of the case with the cardiologist is often better than echo interpretation in isolation, particularly for complex cases. Paediatric echo typically uses ultrasound frequency in the range of 3.5–7.0 MHz. The most common approach is transthoracic with standard acoustic windows, including parasternal views, apical views, subcostal views and the suprasternal view. The relatively thin chest wall in paediatric patients permits images of cardiac anatomy to be achieved readily, particularly of superficial and anterior structures. This article focuses on the more common transthoracic echo (TTE); other more invasive approaches include transoesophageal and epicardial echo. A standard examination involves two-dimensional imaging of cardiac structures, allowing detection of abnormal anatomy or abnormal movement. This is supplemented with M-mode one-dimensional imaging to obtain fine measurements and Doppler imaging (e.g. pulsed wave, continuous wave, colour flow Doppler and tissue Doppler imaging) to assess velocities. Three-dimensional imaging may complement any examination, helping delineate abnormal cardiac anatomy (useful in planning before surgery or catheter interventions) and calculation of ventricular size for more accurate functional assessments. 1 Paediatric echo reports tend to follow a standard format as defined by guidelines published by the American Society of Echocardiography. 2 This includes minimum elements, such as the patient's characteristics, clinical data, echo findings (i.e. defining structural anatomy, measurements of cardiovascular structures, haemodynamic measurements and functional assessments) and a summary. The elements are as follows. Patient data included name, date of birth, unique identifier number. Changes to the patient's condition (e.g. secondary to evolving disease, concomitant illness or in response to treatment) may alter cardiac performance, meaning even recent studies can convey outdated impressions. It is important to consider the current clinical status of the patient and anticipated disease progression to ensure the echo report remains relevant. Attention should be paid to the clinical indication for the study. This could range from a diagnostic investigation for new symptoms to surveillance studies to evaluate disease progression. The indication will direct the examination, with either a complete or a focused study tailored to answer the clinical questions. Diagnostic accuracy depends greatly on image quality, and the report should include a comment relating to this. Suboptimal views may occur in the context of the uncooperative child, unfavourable body habitus or difficult chest wall access. Technically difficult studies may lead to both quantitative and qualitative errors: findings must be interpreted with caution. Be aware that pathology 'not seen' does not necessarily equate to 'not present'. Inclusion of the patient's height and weight allows comparisons based on body surface area. Arterial pressure readings provide context to any intracardiac pressure measurements. Heart rate and rhythm may be relevant clinically. Tachyarrhythmias (e.g. atrial fibrillation or severe sinus tachycardia) can produce mild ventricular systolic dysfunction in an otherwise normal heart. Information on current clinical status may be documented. Artificially altered haemodynamics, such as the presence of haemodynamic support (e.g. inotropes or vasopressor drugs, or cardiac devices) or examinations performed under anaesthesia may influence the findings on echo. A segmental approach is the preferred method of imaging the heart, evaluating the cardiac segments—the systemic and pulmonary venous return, the atria, ventricles and great vessels, in turn. This approach is reflected in the report with cardiovascular structures described in sequence. The following are the key points in segmental sequential analysis: (i) Abdominal situs, cardiac position and apex orientation. (ii) Venous return, atria and interatrial septum. (iii) Atrioventricular connections and valves. (iv) Ventricles and the interventricular septum. (v) Outflow tracts and ventriculoarterial connections. (vi) Extracardiac great vessels. Situs refers to the position of organs within the body or system; situs solitus is the normal arrangement, situs inversus is the mirror image of normal and situs ambiguus is another arrangement. This is applied primarily to the abdominal structures. The positions of the inferior vena cava (IVC), liver and stomach are identified, and this is referenced in a report, with situs solitus describing right-sided IVC, liver and left-sided stomach (Fig. 1). Atrial situs may also be described. The atria are identified morphologically according to their appendages and the septal anatomy. Their relative positions are described: situs solitus describing the morphological right atrium (RA) to the right of the left atrium (LA), situs inversus the RA to the left of the LA and situs ambiguus other arrangements (e.g. atrial isomerism in heterotaxy syndromes or a common atrium with absent septum). This is the position of the majority of the cardiac mass within the thorax relative to the midline. The heart can be predominantly on the left ('levoposition', central 'mesoposition' or on the right 'dextroposition'). This is the orientation of the base-to-apex axis of the heart and is independent of cardiac position. The axis may point to the left 'levocardia', the right 'dextrocardia' or inferiorly (midline) 'mesocardia'. The venous return to the RA normally arises from the superior vena cava (SVC), IVC and coronary sinus. A 'persistent left SVC' is a common variant, present in around 0.5% of paediatric patients and typically drains into a dilated coronary sinus. The IVC diameter and respiratory variation (or collapsibility) can give an indication of right atrial pressures (RAPs). 3 The IVC should collapse >50% with inspiration. Reduced variability suggests RAPs of >10 mmHg. Inferior vena cava dilatation without variability suggests pressures >20 mmHg. Positive-pressure ventilation can obliterate this normal response. The venous return to the LA normally arises from four pulmonary veins. These veins enter the heart posteriorly, and visualisation of all four can be challenging using TTE. The report may confirm drainage of some pulmonary veins (e.g. 'at least two pulmonary veins seen') in the presence of normal pulmonary drainage. Abnormal numbers of pulmonary veins or anomalous pulmonary venous drainage (partial or total) may occur in CHD. In addition to identifying atrial morphology and arrangement, chamber size is assessed and the septum examined for inter-atrial communications (i.e. atrial septal defects (ASDs)). If present, a description of the size, number and location of any defects, and flow direction and velocity should be recorded. Atrioventricular connections can be either biventricular or univentricular. In biventricular connections, each atrium connects to a ventricle. This can either be 'concordant' referring to normal connections, where the RA connects to the right ventricle (RV) and the LA to the left ventricle (LV), or 'discordant', where the RA connects to the LV and the LA to the RV (e.g. congenitally corrected transposition of the great arteries). Univentricular connections describe situations where the atria connect to a single ventricle (e.g. absent LV or RV, or double-inlet ventricle). The nomenclature of the valves follows the respective ventricle irrespective of position or connections, such that the tricuspid valve is associated with the RV and the mitral valve with the LV. Valve morphology, presence of communications between valves (i.e. atrioventricular septal defect) and valve function (including presence of stenosis or regurgitation) will be assessed. Structural assessments of chamber size and shape are made, including evidence of ventricular remodelling, such as dilatation or hypertrophy. The septum is assessed for inter-ventricular communications (i.e. ventricular septal defects (VSDs)) and the septal position in relation to the RV. Right-sided chamber enlargement or increased right heart pressures may result in septal flattening, bowing or paradoxical systolic motion. Assessments of ventricular systolic and diastolic function are made. Multilevel imaging and flow measurements are made along the outflow tracts to identify and quantify any outflow tract obstruction. As with atrioventricular connections, ventriculoarterial connections can be 'concordant', where the pulmonary artery arises from the RV and the aorta from the LV, or 'discordant', where the aorta arises from the RV and the pulmonary artery from the LV (e.g. transposition of the great arteries). Other abnormal connections include double-outlet ventricle, where both arterial trunks (one arterial trunk plus >50% of the other) are committed to the same ventricle and 'common outlet', where a solitary arterial trunk arises from the ventricle (e.g. truncus arteriosus). The ventriculoarterial valves are typically semilunar valves, the nomenclature of which follows the downstream artery (e.g. the valve connecting to the aorta is named the 'aortic valve'). An assessment of valve morphology and function is performed. The aortic root is inspected for the coronary artery origins and the arch examined to determine arch sidedness (normally left-sided) and branching patterns. A right-sided arch can be associated with a vascular ring with potential to cause tracheal or oesophageal compression. Multilevel aortic dimension measurements are taken with flow assessments. Aortic obstruction (e.g. coarctation of the aorta) may be evidenced by flow acceleration (peak gradient >30 mmHg is significant), presence of a diastolic tail on Doppler examination (continuous flow in diastole through a narrowing), aortic dilatation and reduced abdominal aortic pulsatility. 'Flow reversal' may be seen in the presence of severe aortic regurgitation, patent ductus arteriosus (PDA), or other shunt. The main pulmonary artery and branches are evaluated for position and size. Any stenosis or hypoplasia is further assessed with Doppler measurements. Flow reversal may be seen in the presence of pulmonary regurgitation or distal branch pulmonary artery stenosis. An assessment of ductal patency is performed in neonates and infants. If a PDA is present, Doppler evaluation is performed to determine the direction and characteristics of the shunt across the duct and the pressure gradients. 4 Extracardiac structures are visualised during a standard TTE and may be commented on if abnormalities are present (e.g. pericardial effusion, pleural effusions, mediastinal masses, diaphragm motion anomalies and hepatic lesions). A full report usually includes values of cardiovascular measurements (e.g. chamber dimensions, valve areas and flows) in tabular format. In adults, these can be evaluated against single reference ranges. However, in paediatric patients, the normal range is greatly influenced by patient size and age, adding an additional challenge to interpretation. Comprehensive collections of normative values for cardiovascular structures based on age or size are available to aid meaningful evaluation of measurements (e.g. Boston Children's Hospital data). 5 It is often impractical for clinicians to refer to multiple value references, and Z-scores are frequently applied in paediatric practice, providing an approach to normalising cardiovascular measurements for the effect of size and age. The Z-score describes how many standard deviations above or below a size or age-specific group mean a given measurement lies. 6 This allows interpretation of the magnitude of abnormal measurements and is useful in serial assessments of disease as a child grows. 7.8 A normal Z-score lies within two standard deviations of the relevant group mean (between -2.0 and +2.0). Platforms exist to provide instant Z-scores for paediatric cardiology parameters, such as the Cardio Z application. 9 Echocardiography is used to assess the physiology of blood flow in the heart. Although direct measurements cannot be taken, using Doppler imaging, flow, valve function and ventricular performance can be evaluated. Doppler data are typically presented as velocities, but this can be converted to pressure gradients using the modified Bernoulli's equation: Pressure gradient = 4 × velocity<sup>2</sup> (i.e. ΔP = 4V<sup>2</sup>). Abnormal flow through valves and tracts is usually evaluated using pressures and velocities rather than absolute dimensions, which vary with age and size. When interpreting abnormal findings, such as valve stenosis or regurgitation, it is important to view the heart as a whole considering other cardiac lesions and secondary changes. For example, mitral regurgitation may be the consequence of outflow tract obstruction or ventricular dilatation rather than an isolated mitral valve lesion. The ventricular response to any lesion is the most important factor when considering clinical significance. Acute or chronic changes may occur secondary to volume or pressure loading, resulting in ventricular remodelling (e.g. hypertrophy or dilatation) and altered function. For example, aortic stenosis with the addition of secondary left ventricular hypertrophy represents an increased clinical risk. Echocardiography provides dynamic images of ventricular performance. A subjective qualitative visual inspection may be adequate to assess ventricular function, but is prone to variability between observers and between studies. Quantitative assessments of function are preferable; they aim to reduce operator subjectivity and increase reproducibility. 8 Most standard techniques for ventricular assessment are based on chamber size and are load dependent. The conical shape of the LV lends itself to a variety of calculations to assess function. The RV has a more complex non-geometric shape and is more frequently



chamber anatomy, abnormal connections and abnormal loading conditions. The application of three-dimensional echo assessments of the ventricles can be beneficial in this setting.1 Fractional shortening This is the shortening of left ventricular muscle, comparing the left ventricular end-diastolic dimension (LVEDD) and the left ventricular end-systolic dimension (LVESD), as an assessment of function. Measurements are taken at the ventricular base, thus using a regional parameter to assess global function and may be inaccurate in the presence of regional abnormalities. Fractional shortening (FS) is calculated as  $FS (\%) = \frac{LVEDD - LVESD}{LVEDD} \times 100$ . The normal FS is 30–45%.10 Ejection fraction (EF) This is the percentage of the left ventricular end-diastolic volume (LVEDV) ejected during systole. Views required to more accurately assess left ventricular volumes (e.g. Simpson's biplane) can be difficult to obtain in children and therefore, simplified measurements are often used increasing potential for inaccuracies.8 Ejection fraction (EF) is calculated as  $EF (\%) = \frac{LVEDV - LVESV}{LVEDV} \times 100$ . The normal EF is 50–80%.10 Left ventricular systolic performance (dP/dt) Ventricular contractility can be estimated by measuring the strength and speed of contractions. Doppler velocities are used to calculate the rate of left ventricular pressure change during early systole. This is expressed as a change in pressure over a short period of time (dP/dt). The velocity measurements require presence of mitral regurgitation limiting its applicability. The normal dP/dt is  $>1200 \text{ mmHg s}^{-1}$ . Tricuspid annular plane systolic excursion (TAPSE) is the distance the tricuspid annulus moves during systole as a measure of longitudinal shortening (the main mechanism of right ventricular contraction). Normal TAPSE values vary with cardiac size and Z-scores may be used.11 Fractional area change Because right ventricular volumes are challenging to calculate, fractional area change (FAC) is often used in place of EF to estimate right ventricular output. Fractional area change is calculated by measuring the percentage area change in the RV between diastole (right ventricular end-diastolic area [RVEDA]) and systole (right ventricular end-systolic area [RVESA]).  $FAC (\%) = \frac{RVEDA - RVESA}{RVEDA}$ . The normal FAC value is  $>40\%$ .12 Tissue Doppler imaging applies Doppler to myocardial tissue to provide measurement of myocardial tissue velocities and strain patterns, to evaluate regional systolic and diastolic function. This can be useful in CHD as it can be applied to any chamber morphology. However, velocities are sensitive to loading conditions. Tissue Doppler imaging values vary with age and cardiac growth, and are usually presented as Z-scores. The summary will include pertinent positive and negative findings described in the context of the whole cardiac examination. The echo indication should be addressed and comparisons may be made with previous studies. Recommendations for further investigations or treatment may be included. With increasing expertise and advances in ultrasound machinery, the application and diagnostic capability of echo has increased. However, there are limitations to the use of echo. Study findings are equipment and operator dependent, and may vary accordingly. Errors may arise as a result of technical factors (e.g. poor acoustic images, image artefact and patient behaviour), cognitive factors (e.g. misidentification or misinterpretation) and patient factors (e.g. errors secondary to the increased paediatric patient heart rate and complex baseline anatomy).8,13 Measurement errors can be easily amplified through the use of haemodynamic formulae. Other cardiac imaging modalities (e.g. cardiac MRI or catheterisation) may be required to assess cardiac status further. Atrial septal defects account for ~10% of cases of CHD. Clinical significance is dependent on the degree of shunt, which varies with defect size and compliance of the ventricles. Flow is normally predominantly left to right (a slight flow reversal may occur in early ventricular systole). Echo assessment (i) The size and location of any defects, and the direction and magnitude of shunt through the defect are assessed. (ii) Significant left-to-right shunt leads to volume overload of the right heart and pulmonary over-circulation. Right-sided chamber enlargement and later pulmonary artery dilatation may occur (Fig. 2). Tricuspid and pulmonary regurgitation can develop with annular dilatation. Pulmonary hypertension may develop with time. Apical four-chamber view demonstrating a large fossa ovalis atrial septal defect (ASD) (\*). The colour flow Doppler shows flow through the defect in red (indicating flow towards the transducer), confirming left to right flow through the defect. There is evidence of right heart enlargement with moderate dilatation of the right atrium and right ventricle (iii) If an ASD is suspected but is not seen, a 'bubble study' may be performed. Echogenic bubbles within a shaken saline bolus injected intravenously enter the RA; presence of bubbles in the LA confirms an inter-atrial communication. Ventricular septal defects are the most common CHD, often in association with other cardiac anomalies. Clinical significance relates to the degree of shunt and secondary valve or functional compromise. Echo assessment (i) The size, location and number of defects is assessed. (ii) The relationship of the VSD to valve apparatus and altered function (e.g. aortic or tricuspid regurgitation [TR] secondary to valve prolapse or distortion). (iii) Doppler measurements to evaluate direction, velocity and magnitude of ventricular shunt, including estimation of pulmonary to systemic blood flow ratio (Q<sub>p</sub>:Q<sub>s</sub>). (iv) Shunts are predominantly left to right, resulting in volume overload of the left heart; left heart chamber dilatation may occur. Increased pulmonary vascular resistance may develop secondary to increased flow, which may be associated with right ventricular hypertrophy. (v) A VSD is described as 'unrestrictive' when the defect is so large that there is unimpeded flow from the LV to the RV. Tetralogy of Fallot (TOF) is the most common cyanotic heart lesion, encompassing a VSD, overriding aorta, right ventricular outflow tract (RVOT) obstruction and right ventricular hypertrophy. Most children encountered by anaesthetists will be after TOF repair. Depending on the surgical result and cardiac sequelae, these patients may still have clinically significant pathophysiology. Echo assessment Before repair: (i) The anatomy of the TOF is confirmed, including VSD morphology, anatomy of the coronary arteries (implications for surgical repair), pulmonary valve function and severity of RVOT obstruction (Fig. 3). (ii) The degree of RVOT obstruction will determine the shunt through the VSD. Predominant left-to-right shunting is seen in mild obstruction (acyanotic 'pink' TOF); predominant right-to-left shunt is seen in severe obstruction (cyanotic 'blue' TOF). After repair: (i) Pulmonary regurgitation is common post-repair as a result of surgical technique (e.g. transannular patch). Chronic pulmonary regurgitation may result in a cycle of secondary right ventricular dilatation, pulmonary artery dilatation and worsening regurgitation. Right ventricular dysfunction may ensue. (ii) Residual VSDs may be present (e.g. at the margins of the VSD patch or additional VSDs only evident after surgical closure of a larger VSD). (iii) A degree of RVOT obstruction is often present. If significant, right ventricular hypertrophy may persist. The Fontan procedure is a palliative procedure used in children with functionally univentricular hearts. Systemic venous blood flow is rerouted directly to the lungs, creating a Fontan circulation with passive pulmonary blood supply and a single systemic ventricle driving systemic flow. The echo study and interpretation are centred on the underlying cardiac diagnoses and surgical procedures performed, the specifics of which are beyond the scope of this article. Some key considerations are outlined as follows. Echo assessment •The patency of the Fontan pathway is assessed. An unobstructed pathway between the systemic veins, pulmonary arteries and pulmonary circulation is essential for optimal function. Any degree of obstruction will reduce cardiac output. •The pulmonary venous chamber is assessed to establish atrial mixing of blood is adequate and exclude any pulmonary venous obstruction. •Atrioventricular valve regurgitation is common and its severity should be quantified. Atrioventricular valve regurgitation is an important determinant of atrial pressure and, if significant, may diminish the transpulmonary gradient, pulmonary blood flow and cardiac output. •The single ventricle function is evaluated. The ventricular morphology influences measurements, and functional assessments may be challenging especially in the presence of a systemic RV. Systolic function is often preserved in the paediatric patient with diastolic dysfunction more prevalent. •The outflow tract, aortic (or neo-aortic) valve function and aortic patency are assessed. Pulmonary hypertension may be defined as a resting mean pulmonary artery pressure (PAP) of more than 25 mmHg. Echocardiography can be used to estimate right ventricular pressure and PAP to determine severity. Echo assessment •Measurements of right ventricular and pulmonary artery systolic pressure are made. •This is commonly estimated from a TR jet, if present (Fig. 4), using the simplified Bernoulli's principle ( $P = 4V^2$ ). Doppler measurement of a tricuspid regurgitation (TR) jet, to estimate pulmonary artery pressure (PAP) and quantify severity of pulmonary hypertension. The PAP can be estimated from the sum of the estimated or measured right atrial pressure (RAP) and the pressure gradient between the right atrium (RA) and right ventricle (RV). In this image, the peak TR velocity is 3.8 m s<sup>-1</sup>. Using Bernoulli's equation, the pressure gradient between the RA and RV =  $4 \times (3.8)^2 = 57.75 \text{ mmHg}$ . Assuming an RAP of ~5 mmHg (if not being directly measured), the right ventricular pressure is ~64 mmHg. In the absence of any right ventricular outflow tract obstruction, the PAP will also be ~64 mmHg. •Right ventricular systolic pressure (RVSP) = (4 × TR peak velocity)<sup>2</sup> + RAP. •The severity of pulmonary hypertension is typically classified based on the mean RVSP, with mild pulmonary hypertension in the range 30–40 mmHg; moderate in the range 40–70 mmHg; and severe when  $>70 \text{ mmHg}$ .12 •Pressure assessments can be made based on right ventricular size and the inter-ventricular septum. Pressure overload may result in right ventricular hypertrophy with or without dilatation. Right atrial dilatation may also occur. Septal flattening or reverse septal curvature suggests increased right ventricular pressures ( $>40 \text{ mmHg}$ ). •Causative lesions may be identified (e.g. left-to-right shunts). •Right ventricular systolic and diastolic dysfunction may be present. The presence of pericardial effusion is an indicator of right heart failure and associated with poor outcome.14 Hypertrophic cardiomyopathy is a genetic disease characterised by left ventricular hypertrophy. Most patients are asymptomatic or only mildly symptomatic and require regular surveillance echocardiograms to monitor disease progression. Echo assessment •Left ventricular hypertrophy usually involves the inter-ventricular septum. The degree of hypertrophy is measured as wall thickness and may be evaluated using Z-scores. •The magnitude of any left ventricular outflow tract (LVOT) obstruction secondary to hypertrophy is assessed. Peak velocities  $>2 \text{ m s}^{-1}$  are considered significant. Left ventricular outflow tract obstruction is often dynamic and may only be unmasked with provocation. •Diastolic dysfunction may occur as a result of abnormal left ventricular myocardial relaxation. This may manifest with left atrial enlargement and mitral regurgitation. Systolic function is usually normal or hyperdynamic. •'Systolic anterior motion' of mitral valve may be present, contributing to dynamic LVOT obstruction. The paediatric echo report may provide valuable data on a child's cardiac status to inform management of anaesthesia. A basic understanding of paediatric cardiac disease and echo is required to extract clinically useful information from the report. As with all investigations, the echo report must be considered in the context of the individual patient, considering the indication for the scan, their current clinical status and any planned procedures. The authors declare that they have no conflicts of interest. The associated MCQs (to support CME/CPD activity) are accessible at [www.bjaed.org/cme/home](http://www.bjaed.org/cme/home) by subscribers to BJA Education. Matrix codes: 1A03, 2A03, 3D001. Simpson J., Lopez L., Acar P. Three-dimensional echocardiography in congenital heart disease: an expert consensus document from the European Association of Cardiovascular Imaging and the American Society of Echocardiography. *J Am Soc Echocardiogr.* 2017;30:1–27. [PubMed] [Google Scholar]2. Lai W.W., Geva T., Shirali G.S. Guidelines and standards for performance of a pediatric echocardiogram: a report from the task force of the Pediatric Council of the American Society of Echocardiography. *J Am Soc Echocardiogr.* 2006;19:1413–1430. [PubMed] [Google Scholar]3. Kircher B.J., Himelman R.B., Schiller N.B. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. *Am J Cardiol.* 1990;66:493–496. [PubMed] [Google Scholar]4. Mertens L., Seri I., Marek J. Targeted neonatal echocardiography in the neonatal intensive care unit: practice guidelines and recommendations for training. *Eur J Echocardiogr.* 2011;12:715–736. [PubMed] [Google Scholar]5. Lai W.W., Mertens L.L., Cohen M. 2nd Edn. Wiley-Blackwell; Hoboken, New Jersey, USA: 2016. Echocardiography in pediatric heart disease. Appendix 883–901. [Google Scholar]6. Chubb H., Simpson J.M. The use of Z-scores in paediatric cardiology. *Ann Pediatr Cardiol.* 2012;5:179–184. [PMC free article] [PubMed] [Google Scholar]7. Daubeney P.E., Blackstone E.H., Weintraub R.G., Slavik Z., Scanlon J., Webber S.A. Relationship of the dimension of cardiac structures to body size: an echocardiographic study in normal infants and children. *Cardiol Young.* 1999;9:402–410. [PubMed] [Google Scholar]8. Lopez L., Colan S., Frommelt P. Recommendations for quantification methods during the performance of a pediatric echocardiogram. *J Am Soc Echocardiogr.* 2010;23:465–495. [PubMed] [Google Scholar]9. Chubb H., Simpson J.M. Cardio Z application. •Eidem B.W., O'Leary P.W., Cetta F. 2nd Edn. Wolters Kluwer; Alphen aan den Rijn, Netherlands: 2015. Echocardiography in pediatric and adult congenital heart disease. [Google Scholar]11. Koestenberger M., Ravekos W., Everett A.D. Right ventricular function in infants, children and adolescents: reference values of the tricuspid annular plane systolic excursion (TAPSE) in 640 healthy patients and calculation of z score values. *J Am Soc Echocardiogr.* 2009;22:715–719. [PubMed] [Google Scholar]12. Reynolds T. 3rd Edn. Arizona Heart Institute; USA: 2002. The pediatric echocardiographer's pocket reference. ISBN: 0963576771. [Google Scholar]13. Benavidez O.J., Gauvreau K., Jenkins K.J., Geva T. Diagnostic errors in pediatric echocardiography: development of taxonomy and identification of risk factors. *Circulation.* 2008;117:2995–3001. [PMC free article] [PubMed] [Google Scholar]14. Sahay S., Tonelli A.R. Pericardial effusion in pulmonary arterial hypertension. *Pulm Circ.* 2013;3:467–477. [PMC free article] [PubMed] [Google Scholar]



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