Recommendations for Quantification Methods During the Performance of a Pediatric Echocardiogram: A Report From the Pediatric Measurements Writing Group of the American Society of Echocardiography Pediatric and Congenital Heart Disease Council

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Upon completing the reading of this article, participants will better be able to:

Objectives:

1. Review the techniques for optimizing imaging and quantifying cardiac structures in the pediatric population.
2. Explain the importance of adjusting measurements of cardiovascular structures for the effects of body size.
3. Identify the pediatric quantification protocols for the pulmonary veins, systemic veins, atria and atrioventricular valves.
4. Recognize and apply the recommended echocardiographic methods for the evaluation of left and right ventricular size and systolic function.
5. Define the optimal views and the appropriate anatomic sites for correct measurement of the proximal ascending, proximal and distal arch, and descending aorta.
6. Employ appropriate transducer position and Doppler technique for the anatomic and hemodynamic interrogation of the aorta and aortic valve and the pulmonary artery and the pulmonary valve.

Keywords: Pediatric quantification, Measurements, Z scores, Normative database

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INTRODUCTION

Echocardiographic quantification is crucial in the diagnosis and management of patients with acquired and congenital heart disease (CHD). The American Society of Echocardiography (ASE) and the European Association of Echocardiography have published recommendations on how to measure the size and function of cardiovascular structures in adults, providing reference limits to distinguish normal from abnormal values. Identifying an abnormal measurement helps assess the effect of a disease on the size of a cardiovascular structure, determine when intervention may be necessary, and monitor the effect of the intervention. Examples in which these standards are useful include aortic root dilation in Marfan syndrome and left ventricular (LV) dilation with a ventricular septal defect. However, the size of cardiovascular structures is influenced not only by the hemodynamics of disease states and their treatments but also by confounding factors such as growth, age, genes, gender, race, body composition, basal metabolic rate, hematocrit, exercise, and altitude.

Aside from abnormal hemodynamics, body size is the most powerful determinant of the size of cardiovascular structures; all cardiovascular structures increase in size relative to somatic growth, a phenomenon known as cardiovascular allometry. Expressing measurements relative to body size allows a meaningful distinction between normal and abnormal values in children. It does require the collection of quantitative data from a normal pediatric population to function as the standard against which all measurements are compared. Because there must be agreement on how to measure the size of each cardiovascular structure, this document describes the recommended protocols for the morphometric evaluation of the heart in children with or without CHD, and recommendations at the end of each section refer to measurements that may be useful for the creation of a pediatric normative database. However, the Pediatric Measurements Writing Group emphasizes that the recommended measurements are those that can be performed in a pediatric examination and not necessarily those that must be part of the study.

OPTIMIZATION TECHNIQUES IN IMAGING AND DOPPLER EVALUATION

Standard views are often categorized as "long axis" or "short axis," and these are described in Table 1. General optimization techniques in two-dimensional (2D) imaging have been outlined previously for children. Several technical factors can influence the accuracy of spatial measurements: (1) axial resolution parallel to the ultrasound beam is superior to lateral resolution perpendicular to the beam, so views allowing for linear axial measurements are better than those for which only lateral measurements are available (parasternal views are better than apical views for the aortic annulus); (2) lateral resolution degrades with increasing distance secondary to beam spread, so the transducer should be positioned as close as possible to a structure when only a lateral measurement is available; and (3) for large image depths, the ultrasound resolution often exceeds the pixel resolution of the image display, so decreasing the image depth or magnifying the region of interest can often alleviate the limitations of the monitor resolution.

Quantitative assessment of each structure should be performed in multiple views, and orthogonal planes should be used for noncircular structures such as the atrioventricular (AV) valves. Early reports based on M-mode echocardiography recommended measurements from the leading edge of the near-field reflector to the leading edge of the far-field reflector, and normative data for the proximal aorta in adults have involved leading edge-to-leading edge measurements. However, current guidelines for chamber, annular, and vessel quantification involve measurements of intraluminal dimensions from one inner edge to the opposite inner edge. In addition, published pediatric normative databases based on 2D echocardiography have used inner edge-to-inner edge measurements of vessel diameters. There are two important caveats with these measurements: vascular diameters should be perpendicular to the long axis of the vessel, and valvar and vascular diameters should be measured at the moment of maximum expansion. In other words, the inferior vena cava (IVC) diameter should be measured during exhalation, the mitral valve (MV) and tricuspid valve (TV) annular diameters in diastole, and the aortic valve (AoV) and pulmonary valve (PV) annular diameters as well as arterial diameters in systole. These recommendations are based on hemodynamic considerations, correspond to the methodologies used in published pediatric normative databases, and often differ from the quantification approach used in adults.

General optimization techniques in Doppler echocardiography have been outlined previously for adults, and their utility must address the abnormal valve and vessel positions and unusual flow jets in patients with CHD. Color mapping should precede spectral Doppler interrogation to identify the direction of flow. The audio feature can help optimize alignment, especially given the unpredictable orientation of flow jets in the third dimension. Doppler waveforms should be displayed at a sweep speed of 100 to 150 mm/s to discriminate temporal changes in the velocity flow profile, particularly in children with high heart rates. Simultaneous electrocardiographic display helps correlate the timing of flow with electrical events. Doppler gain and power settings should be optimized to depict the outer edge of the brightest spectral Doppler envelope; only well-defined envelopes should be measured, and “fuzz” or “feathering” beyond modal velocities should be excluded. Mean gradients calculated from the velocity-time integral (VTI) or area under the
velocity curve should be measured from valve opening to closure at the AV and semilunar valves and throughout the cardiac cycle within a blood vessel or at an interatrial communication, incorporating the zero velocity during periods of absent flow. All Doppler measurements should be averaged over 3 consecutive cardiac cycles to account for respiratory variation.

PRINCIPLES AND METHODS FOR ADJUSTING MEASUREMENTS OF CARDIOVASCULAR STRUCTURES FOR THE EFFECTS OF BODY SIZE

The first step in adjusting for body size involves developing a mathematical description of the mean behavior of the measurement within a normal pediatric population. Ideally, this is based on physiologic principles. Body surface area (BSA) appears to be a better parameter of somatic growth in normal children than height or weight alone.15,19 Published equations to calculate BSA often produce variable results, particularly at lower height and weight values,20,23 and some are derived from data that do not include children. The Haycock formula21 (BSA \[m^2\] = 0.024265 × weight [kg]0.5378 × height [cm]0.7644) appears to provide the best correlation between BSA and the size of cardiovascular structures (compared with the formulas of DuBois and DuBois,20 Dreyer and Ray,22 and Boyd23) and is recommended for calculating BSA.15 Because of the linear relationship between cardiac output and BSA24 and the mostly linear relationship between cardiac output and the size of cardiovascular structures,15 “indexing” the size of structures to BSA has become a fairly common practice.25-28 However, assuming that BSA is linearly related to length, area, and volume measurements is mathematically impossible. In addition, BSA-adjusted measurements often manifest a persistent dependence on BSA: the mean value of the BSA-adjusted measurement and the distribution of values around the mean change with increasing BSA (a phenomenon of changing or nonconstant variance known as heteroscedasticity).10,29

Once the mathematical relationship between a measurement and BSA has been determined, the next step involves the confidence intervals and the problem of heteroscedasticity. One approach to find a mathematical descriptor that maintains a stable and constant variance across the range of body sizes might involve transformation of the measurements and/or BSA within a linear or nonlinear regression equation.15,17,19 For example, physiologic principles suggest that distances can be adjusted or normalized to the square root of BSA, areas to BSA, and volumes to BSA 1.5,30 This approach results in a mostly linear relationship between the measurement and the transformed BSA, but it does not eliminate the problem of heteroscedasticity: variance continues to be affected by changes in body size. Another example involves performing a logarithmic transformation of the measurements to minimize the problem of heteroscedasticity.17 However, this method does not effectively describe the population at minimum and maximum BSA values, nor does it fully eliminate heteroscedasticity. There is also no fundamental physiologic explanation for this approach.

An increasingly popular approach in pediatric cardiology to account for the effects of body size and age has been the use of Z scores.15,17,31-36 Calculation of Z scores involves assessment of the distribution of measurement values (by determining the confidence intervals) across a range of body sizes in the normal population. The Z score of a measurement is the number of standard deviations of that value from the mean value at a particular BSA. In other words, a Z score of zero corresponds to a measurement equal to the population mean for that particular BSA. A Z score of +2 or −2 corresponds to
a measurement that is 2 standard deviations above or below the mean for that particular BSA, thresholds that usually represent the upper or lower limits of normal. Z scores can be converted to percentiles, though the magnitude of an abnormality is much easier to appreciate with Z scores than with percentiles (for example, a Z score of +4 corresponds to the 99.8th percentile, and Z score of +10 corresponds to the 99.9th percentile). The major advantage of using Z scores has been the absence of any reliance on a predetermined relationship between the size of a structure and BSA. In addition, there is no assumption that a constant variance exists across the range of body dimensions are significantly different between normal adults and children, and LA volumes indexed to BSA appear to correlate with diastolic function and mitral regurgitation grade.55 Last, LA volumes have been calculated using real-time three-dimensional (3D) echocardiography and a rotation/polyhedral surface algorithm, and these have correlated well with LA volumes measured by magnetic resonance imaging (MRI).46

RA size is usually assessed in an apical 4-chamber view at end-systole just before the TV opens (Figure 2).14,47-50 Major-axis and minor-axis dimensions are significantly different between normal adults and
patients with right ventricular (RV) volume overload (from an atrial septal defect or tricuspid regurgitation). Raw values and indexed values on the basis of BSA in normal adults are available. Planimetered RA areas or RA volumes calculated from the product of RA area and major-axis length may be better at assessing RA size, though the sample sizes in most studies have been small. As with LA volumes, 3D echocardiography may provide a useful means by which to measure RA volumes using the polyhedral surface algorithm.

Recommendations (Table 2): The recommended methods to assess LA size include the measurement of

![Image](image_url)

**Figure 1** Apical 4-chamber and 2-chamber views at ventricular end-systole showing (A) left atrial major-axis length in a 4-chamber view, (B) left atrial planimetered area in a 4-chamber view, and (C) left atrial planimetered area in a 2-chamber view. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
major-axis lengths in apical 4-chamber views and planimetered areas in orthogonal apical views and calculation of volumes using the biplane area-length or the biplane Simpson method. The recommended methods to assess RA size include the measurement of major-axis and minor-axis lengths and planimetered areas in apical 4-chamber views. When the IVC diameter is measured, measurement should be performed above its junction with the hepatic veins just below the diaphragm in subxiphoid short-axis views.

Doppler Evaluation. Pulsed-wave Doppler interrogation of pulmonary and systemic venous flow requires precise placement of the sample volume in the lumen of the vessel >5 mm from its ostium. Because venous flow velocities are low, the Doppler high-pass filter should be minimized. Pulmonary venous flow is usually evaluated in apical or parasternal views, whereas superior vena cava flow can be evaluated in subxiphoid or suprasternal views. IVC flow is best evaluated in subxiphoid views; hepatic vein flow is usually used as a surrogate because the hepatic veins are more parallel to the line of interrogation than the IVC. Characterization of pulmonary and systemic venous Doppler patterns can help with the assessment of atrial and ventricular diastolic function as well as AV valve function (Figure 3). Antegrade flow during ventricular systole (S wave) occurs because of both atrial relaxation and apically directed movement of the AV annulus. Occasionally, it is biphasic secondary to temporal dissociation of the two components. Abnormal retrograde flow during ventricular systole can occur in the setting of significant AV valve regurgitation as well as AV electrical dissociation with atrial contraction against a closed AV valve during ventricular systole. Antegrade flow during ventricular diastole (D wave) is influenced by atrial and ventricular filling properties as well as AV valve patency. With fast heart rates, the S and D peaks may fuse. Retrograde flow during atrial contraction (Ar wave) is frequently augmented when ventricular compliance is decreased.

In fact, a pulmonary vein Ar wave duration that exceeds the MV inflow duration during atrial systole predicts increased LA and LV end-diastolic pressures in the setting of reduced ventricular compliance. Both the systemic venous D and Ar waves can be affected by respiration, with increased D-wave and decreased Ar-wave velocities during inspiration secondary to negative intrathoracic pressure, so these measurements should be averaged over 3 consecutive cycles.

Recommendations (Table 2): Pulmonary venous S-wave, D-wave, and Ar-wave velocities and Ar-wave duration are best measured in apical or parasternal short-axis views.

AV Valves

Morphometric Evaluation. Measurement of MV and TV size helps characterize valvar pathology and diagnose ventricular hypoplasia. Annular diameter and area can be measured by 2D and 3D imaging. Annular area may also be estimated from a single-plane diameter using the area formula for a circle, but the MV annulus is in fact elliptical and saddle-like in shape. A more accurate estimate can be obtained with orthogonal diameters using the area formula for an ellipse. Echocardiographic measurements of MV size generally overestimate postmortem measurements but this may be an artifact of tissue fixation. Published pediatric normative databases have used annular diameters measured in apical 4-chamber (lateral diameter) and parasternal long-axis (anteroposterior diameter) views to calculate MV and TV elliptical annular areas (Figure 4). However, recent studies in adult patients suggest that apical 2-chamber and 3-chamber views are superior to apical 4-chamber views.
views of the MV provide better anatomic alignment and more accurate MV annular dimensions compared with measurements obtained by computed tomography. The difficulty of obtaining adequate 2-chamber views in children often precludes the use of this technique, and most pediatric studies involving various CHDs are based on MV and TV annular measurements obtained in apical 4-chamber and parasternal long-axis views.

The largest diameters during peak filling in early diastole should be measured at the frame after maximum excursion of the leaflets from inner edge to inner edge at the hinge points of the leaflet attachments. Nomograms for MV and TV diameters are available. Although 2D planimetry has been demonstrated to be reasonably accurate in adults with acquired MV stenosis, it is unreliable in the setting of congenital MV stenosis, which is characterized by complex multilevel obstruction with abnormally shaped and often multiple flow orifices precluding a true single-plane “en face” view of the maximum orifice area. Compared with 3D planimetric assessment in patients with mitral stenosis, 2D planimetry overestimates MV area by as much as 88%, depending on valve geometry (domed vs funnel shape) and the position of the transducer relative to the valve orifice.

Recommendations (Table 3): The recommended methods to assess MV and TV annular size include measurement of lateral diameters in apical 4-chamber views and anteroposterior diameters in parasternal long-axis views and calculation of areas using the area formula for an ellipse.

Table 2. Measurements of the Pulmonary Veins, Systemic Veins, and Atria

<table>
<thead>
<tr>
<th>Measurement</th>
<th>View</th>
<th>Timing/Location</th>
<th>Applications</th>
<th>Strengths</th>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA major-axis length</td>
<td>Apical 4-chamber</td>
<td>Ventricular end-systole</td>
<td>LA size</td>
<td>Better than M-mode or 2D antero-posterior length</td>
<td>Foreshortening, Little normal pediatric data</td>
</tr>
<tr>
<td>LA minor-axis length</td>
<td>Apical 4-chamber</td>
<td>Ventricular end-systole</td>
<td>LA size</td>
<td>Same as above</td>
<td>Same as above</td>
</tr>
<tr>
<td>LA planimetered area&lt;sub&gt;4C&lt;/sub&gt;</td>
<td>Apical 2-chamber</td>
<td>Ventricular end-systole</td>
<td>LA size</td>
<td>Same as above</td>
<td>Same as above</td>
</tr>
<tr>
<td>RA major-axis length</td>
<td>Apical 4-chamber</td>
<td>Ventricular end-systole</td>
<td>RA size</td>
<td>Normal adult data</td>
<td>Same as above</td>
</tr>
<tr>
<td>RA minor-axis length</td>
<td>Apical 4-chamber</td>
<td>Ventricular end-systole</td>
<td>RA size</td>
<td>Normal adult data</td>
<td>Same as above</td>
</tr>
<tr>
<td>RA planimetered area</td>
<td>Apical 4-chamber</td>
<td>Ventricular end-systole</td>
<td>RA size</td>
<td>Better than 2D length</td>
<td>Same as above</td>
</tr>
<tr>
<td>IVC diameter</td>
<td>Subxiphoid short-axis</td>
<td>Just below diaphragm</td>
<td>Hydration status</td>
<td>Respiratory variation</td>
<td>No normal pediatric data</td>
</tr>
<tr>
<td>Pulmonary vein S velocity</td>
<td>Apical or parasternal short-axis</td>
<td>Systole</td>
<td>LV diastolic function</td>
<td>LA function, MV function</td>
<td>Depends on alignment</td>
</tr>
<tr>
<td>Pulmonary vein D velocity</td>
<td>Apical or parasternal short</td>
<td>Diastole</td>
<td>Same as above</td>
<td>Depends on alignment</td>
<td></td>
</tr>
<tr>
<td>Pulmonary vein Ar velocity</td>
<td>Apical or parasternal short</td>
<td>Diastole</td>
<td>Same as above</td>
<td>Depends on alignment</td>
<td></td>
</tr>
<tr>
<td>Pulmonary vein Ar duration</td>
<td>Apical or parasternal short</td>
<td>Diastole</td>
<td>Same as above</td>
<td>Depends on alignment</td>
<td></td>
</tr>
</tbody>
</table>

4C, 4-chamber; 2C, 2-chamber; ai, minor-axis slice radius in apical 4-chamber view; A<sub>4C</sub>, area in the apical 4-chamber view; A<sub>2C</sub>, area in the apical 2-chamber view; bi, minor axis slice radius in apical 2-chamber view; h, shortest LA major-axis length in either apical 4-chamber view or 2-chamber view; IVC, inferior vena cava; LA, left atrial; MV, mitral valve; N, number of slices; RA, right atrial.

*One frame before MV or TV opening.

Figure 3. Pulmonary vein Doppler pattern. Ar, Peak retrograde flow velocity during atrial contraction; D, peak antegrade flow velocity during ventricular diastole; S, peak antegrade flow velocity during ventricular systole.
Doppler Evaluation. Doppler interrogation of ventricular inflow is best performed with the help of color mapping in apical views where transducer position and angulation changes are frequently needed to optimize alignment. When MV or TV stenosis is suspected, the VTI of the inflow tracing from continuous-wave Doppler interrogation is used to calculate the mean gradient and assess the severity of obstruction. It is important to remember, however, that the transvalvar gradient is dependent on the diastolic filling period and can be augmented by the faster heart rates in children. Stenosis can also be evaluated by measuring the pressure half-time (the time needed for the peak early diastolic pressure to decline by 50%) or calculating the effective orifice area from the continuity equation (the stroke volume or the product of cross-sectional area and the blood flow VTI at that location is preserved at each position along a closed system). However, these methods are also limited by the faster heart rates in children, correlate poorly with data derived from catheterization in the setting of congenital AV valve stenosis, and are not recommended for routine use in children. Quantitative assessment of MV and TV regurgitation has been discussed previously for adults. Some of the recommended Doppler methods have included measurement of the vena contracta diameter and the regurgitant jet area as well as calculation of the regurgitant volume, regurgitant fraction, and effective regurgitant orifice area from the continuity equation and from the proximal isovelocity surface area phenomenon. However, the utility of these indices in children is limited, and they have not been validated.

Pulsed-wave Doppler analysis of MV inflow velocities is used frequently to assess LV diastolic function. The sample volume is best positioned in the left ventricle at the tips of the valve leaflets (distal to the annulus) because both the peak early diastolic velocity (E wave) and the peak velocity during atrial contraction (A wave) decrease significantly in value as the sample volume is moved.
toward the atrium (Figure 5A). The isovolumic relaxation time (IVRT), representing the time from AoV closure to MV opening, can be measured from the aortic component of the second heart sound using a phonocardiogram to the onset of diastolic flow in the MV Doppler tracing or by using simultaneous continuous-wave Doppler interrogation of LV inflow and outflow in an apical 3-chamber view (Figure 5B). The deceleration time from the peak E-wave velocity to its return to baseline in mid-diastole is another parameter of diastolic function that is sensitive to ventricular relaxation and compliance as well as atrial pressure (Figure 5C). However, deceleration time and other diastolic indices based on the E and A waves are limited by their dependence on loading conditions, and their utility in children is often precluded by fusion of the E and A waves resulting from rapid heart rates.

### Table 3 Measurements of the Atrioventricular Valves

<table>
<thead>
<tr>
<th>Measurement</th>
<th>View</th>
<th>Timing</th>
<th>Applications</th>
<th>Strengths</th>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lateral MV diameter (MVDₐ)</td>
<td>Apical 4-chamber</td>
<td>Diastole*</td>
<td>MV size</td>
<td>Reproducible Normal adult data&lt;sup&gt;72&lt;/sup&gt;</td>
<td>May not be as good as apical 2-chamber and apical 3-chamber measurements&lt;sup&gt;74&lt;/sup&gt;</td>
</tr>
<tr>
<td>Antero-posterior MV diameter (MVDₐₚ)</td>
<td>Parasternal long-axis</td>
<td>Diastole*</td>
<td>MV size</td>
<td>Same as above</td>
<td>Same as above</td>
</tr>
<tr>
<td>Lateral TV diameter (TVDₐ)</td>
<td>Apical 4-chamber</td>
<td>Diastole*</td>
<td>TV size</td>
<td>Same as above</td>
<td></td>
</tr>
<tr>
<td>Antero-posterior TV diameter (TVDₐₚ)</td>
<td>Parasternal long-axis</td>
<td>Diastole*</td>
<td>TV size</td>
<td>Same as above</td>
<td></td>
</tr>
<tr>
<td>MV E wave peak velocity</td>
<td>Apical 4-chamber</td>
<td>Diastole</td>
<td>LV diastolic function</td>
<td>Depends on alignment &amp; sample volume location</td>
<td>Depends on loading conditions</td>
</tr>
<tr>
<td>MV A wave peak velocity</td>
<td>Apical 4</td>
<td>Diastole</td>
<td>LV diastolic function</td>
<td>Same as above</td>
<td></td>
</tr>
<tr>
<td>MV A wave duration</td>
<td>Apical 4</td>
<td>Time from to end of A wave</td>
<td>LV diastolic function</td>
<td>Fast heart rates in children → fusion of E and A waves</td>
<td></td>
</tr>
<tr>
<td>MV deceleration time</td>
<td>Apical 4</td>
<td>Time from E wave peak velocity to return to baseline</td>
<td>LV diastolic function</td>
<td>Fast heart rates in children → fusion of E and A waves</td>
<td></td>
</tr>
<tr>
<td>Isovolumic relaxation time (IVRT)&lt;sup&gt;†&lt;/sup&gt;</td>
<td>Apical 3-chamber</td>
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*CW, Continuous wave; MV, mitral valve; MVDₐ, lateral MV diameter; TV, tricuspid valve; TVDₐ, lateral TV diameter.
†Maximum diameter.

<sup>5</sup> Measured by blood flow Doppler evaluation rather than by tissue Doppler evaluation, as described in Table 4.
LV diastolic filling can also be characterized by using several calculations:\textsuperscript{86} the ratio between the E-wave and A-wave velocities; the E-area and A-area fractions, comparing the VTI during early diastole (E area) and during atrial contraction (A area) with the total area under the diastolic curve; the area or filling fraction in the first 33\% (one third filling fraction) or the first 50\% (one half filling fraction) of diastole; the ratio between pulmonary vein Ar-wave duration and MV A-wave duration as discussed previously\textsuperscript{57} (Figure 5D); and peak ventricular filling rates from the product of the E velocity and MV annular cross-sectional area. Because filling rates may vary with cardiac output, it may be more useful to calculate peak filling rates normalized to stroke volume (PFR\textsubscript{SV}) using the following equation:\textsuperscript{87}:

$$\text{PFR}_{SV} = \frac{\text{peak E velocity}}{\text{MV VTI}}$$

However, an important limitation with this approach involves the fact that calculations of LV inflow (flow across the MV annulus) and peak filling rates using pulsed-wave Doppler interrogation do not account for MV annular displacement away from the transducer during atrial contraction.

Figure 5 (A) Pulsed-wave Doppler pattern of mitral inflow showing peak velocities during early diastole and atrial contraction, (B) continuous-wave Doppler pattern of mitral inflow and LV outflow showing IVRT, (C) pulsed-wave Doppler patterns of mitral inflow showing deceleration time (DT), and (D) pulsed-wave Doppler patterns of pulmonary venous flow and mitral inflow showing the duration of flow during atrial contraction. A, Peak mitral flow velocity during atrial contraction; A Dur, time duration of mitral flow during atrial contraction; Ar Dur, time duration of retrograde pulmonary venous flow during atrial contraction; D, peak antegrade pulmonary venous flow velocity during ventricular diastole; DT, deceleration time; E, peak mitral flow velocity during early ventricular diastole; IVRT, isovolumic relaxation time; MV, mitral valve; PV, pulmonary vein; S, peak antegrade pulmonary venous flow velocity during ventricular systole.
study variability, and relies on the skill of the interpreter. Given the importance of accurate, reproducible LV measurements cannot be overstated. Although published ASE recommendations for chamber quantification in adults have been used extensively in children, data on the accuracy and reproducibility of these measurements in pediatrics are scant. In addition, there are limitations to the published methods adjusting for body size in adults as discussed previously. Several linear and volumetric methods to assess LV size have been described and integrated into routine clinical practice, each with distinct advantages and weaknesses, and these are discussed in this section.

In general, LV size should be measured during both diastole and systole, defining end-diastole as the frame with the maximum chamber intraluminal area and end-systole as the frame with the minimum area. However, these definitions are problematic because they rely on visual estimates of areas rather than a quantitative frame-by-frame analysis. In addition, the minimum area occurs at different times in short-axis and long-axis views. During isovolumic contraction, the long axis first shortens and then elongates (the reverse process occurs during isovolumic relaxation). In contrast, the short-axis area first increases and then decreases progressively during isovolumic contraction. Given these limitations, end-diastole can be defined as the frame at which the MV closes and end-systole as the frame preceding MV opening.

Short-axis or minor-axis measurements of LV internal diameter and septal and posterior wall thickness can be obtained in parasternal views (Figure 6), though occasionally these measurements are available only in subxiphoid views. The maximum short-axis dimension is often located at the level of the MV leaflet tips or chords in young patients and more apically at the level of the papillary muscles in older patients and some adults. It is important to note that linear measurements characterize LV size only in one dimension and may misrepresent an abnormally shaped chamber. Short-axis diameters should be considered a surrogate for LV size only when the LV short-axis geometry is circular, a condition often not met in patients with CHD or other abnormal hemodynamic states. Linear measurements can be obtained from long-axis or short-axis views and from M-mode tracings or 2D images. The ASE guidelines for adults recommend linear minor-axis measurements of the left ventricle in parasternal long-axis views because this ensures a perpendicular orientation between the measurement and the LV long axis. In addition, limited parasternal windows can overestimate minor-axis diameters with oblique measurements in apically located short-axis views, a problem not seen with long-axis views in nonstandard parasternal locations. However, a long-axis view does not account for the lateral motion of the left ventricle seen in many children and it does not guarantee circular LV short-axis geometry throughout the cardiac cycle. It also forces the use of a single diameter, in contrast to the multiple diameters available from 2D short-axis images. Consequently, the short-axis view is the recommended approach because it allows one to choose the diameter with the best blood-endocardium interface, a definite advantage when dealing with LV trabeculations. In addition, normal pediatric data from M-mode short-axis views are available.

M-mode echocardiography has provided better temporal and spatial resolution than 2D imaging in the past. However, M-mode measurements in long-axis views can overestimate LV minor-axis diameters compared with 2D measurements. In addition, measurements along a line crossing the midpoints of the ventricular septum and posterior wall (on the basis of the location of the papillary muscle groups) can be difficult by M-mode. Refinements in transducer technology and image processing have recently provided 2D imaging with improved resolution and clear delineation of the blood-endocardium interface. Therefore, 2D short-axis imaging is the recommended approach to obtain LV short-axis measurements averaged over 3 consecutive cardiac cycles (Figure 6). Ideally, a combination of long-axis and short-axis views should be used to ascertain that the short-axis or minor-axis measurement is perpendicular to and crosses the midpoints of the ventricular septum and posterior wall and that the LV short-axis geometry is circular throughout the cardiac cycle.

Two-dimensional volumetric methods require quality LV images from parasternal short-axis, apical, and/or subxiphoid views in which the LV major-axis length and area of the LV endocardial border can be measured. The basal border is defined as the line connecting the MV annular hinge points. The LV length is measured from the basal border midpoint to the apical endocardium, requiring clear images of the apical endocardium without foreshortening the left ventricle. The endocardial border is traced manually, requiring clear delineation of the blood-endocardium interface (convention excludes the papillary muscles when tracing the endocardial border, leaving them included in the blood pool). The biplane Simpson method to calculate LV volumes by summation of equidistant disks is used frequently in adults, with few data validating its accuracy and reproducibility in children. It involves tracing the LV endocardial border in apical 4-chamber and 2-chamber views and using the formula

![Figure 6](image)
\[ V = \frac{\pi}{4} \sum_{i=1}^{N} a_i \times b_i \times \frac{L}{N} \]

where \( V \) is volume, \( a_i \) is the minor-axis slice radius in the apical 4-chamber view, \( b_i \) is the minor-axis slice radius in the apical 2-chamber view, \( L \) is the LV major-axis length, and \( N \) is the number of slices (Figure 7). Some have suggested that the apical 3-chamber view can be substituted for the apical 2-chamber view. In children with abnormally shaped left ventricles, the modified Simpson algorithm using a combination of short-axis and long-axis views may be better than the biapical algorithm described above. LV volume can also be measured with the area-length or bullet method using the formula \( V = \frac{5}{6} \times \text{short-axis basal area} \times \frac{L}{N} \) (Figure 8). Here, the short-axis basal area is measured from parasternal or subxiphoid short-axis views, and LV length is measured from apical 4-chamber or subxiphoid long-axis views. The truncated ellipse method is similar to the area-length method, with a somewhat different formula requiring the additional measurement of the LV minor-axis diameter from an apical 4-chamber view.

LV mass can be calculated from M-mode or 2D linear measurements and this approach has been used extensively in adult clinical trials and epidemiologic studies. It has been used in children, though accuracy and reproducibility data, especially in infants, are scant. The most common method is to measure volumes using one of the approaches discussed previously. LV mass is then calculated by subtracting the endocardial volume from the epicardial volume and multiplying this difference by 1.05 g/mL, the myocardial-specific density. LV volume and mass can also be measured using 3D echocardiography, and growing experience suggests that better accuracy can be achieved compared with 2D methods when MRI is used as the gold standard. Initial reports in pediatrics are encouraging, particularly considering that 3D echocardiography does not rely on geometric assumptions, an important advantage in patients with CHD and abnormally shaped ventricles. However, the feasibility, applicability, and reproducibility of this approach in clinical practice warrant further investigation.

LV systolic function can be evaluated as pump function (global chamber performance) or myocardial function (performance of cardiac myofibers). Global systolic pump function is dependent on myocardial force generation characteristics (contractility) as well as preload, afterload, and heart rate, whereas myocardial function represents myocardial contractility independent of loading conditions and heart rate. Numerous echocardiographic methods have been used to evaluate both properties of LV systolic function, and these can be divided into geometric and nongeometric parameters. Geometric parameters require LV dimension or volume measurements and are influenced by LV shape. Nongeometric parameters do not require these measurements, are not affected by LV shape, and rely on Doppler echocardiography and other techniques.

The most commonly used geometric parameters of global LV systolic function are the linear shortening fraction (SF), fractional area change, and the volumetric ejection fraction (EF). These methods are affected by loading conditions, but fractional area change and EF are less sensitive to abnormal chamber geometry and regional
abnormalities. SF can be calculated using LV minor-axis internal diameters obtained from a standard M-mode tracing or from 2D images using the equation $SF = (end-diastolic dimension − end-systolic dimension)/end-diastolic dimension$. As discussed previously, the 2D approach provides better display of the midpoints of the ventricular septum and posterior wall. EF is calculated from the equation $EF = (end-diastolic volume − end-systolic volume)/end-diastolic volume$; both end-diastolic and end-systolic volumes are measured using any of the 2D or 3D methods described previously. Extrapolation of EF from linear LV minor-axis diameters is discouraged, because of inaccuracies resulting from geometric assumptions. A relatively load-independent index of myocardial function is useful when following patients at risk for abnormal afterload, such as patients undergoing chemotherapy and those infected with human immunodeficiency virus. One such method involves the relationship between velocity of circumferential fiber shortening adjusted for heart rate and end-systolic wall stress, and this index of contractility is relatively independent of preload and incorporates afterload and heart rate in its assessment (Figure 9). However, this index applies only when the LV is normally shaped.

Recommendations (Table 4): The recommended methods to assess LV size and function include a linear approach and a volumetric approach. The linear method involves measurement of short-axis diameters and wall thickness and calculation of SF and the velocity of circumferential fiber shortening adjusted for heart rate and end-systolic wall stress from 2D short-axis images obtained in parasternal or subxiphoid short-axis views. The volumetric method involves (1) measurement of areas from the same 2D or 3D short-axis images; (2) measurement of long-axis lengths from 2D or 3D long-axis images obtained in apical 4-chamber or subxiphoid long-axis views; and (3) calculation of volumes, EF, and mass using 2D or 3D measurements.

Doppler Evaluation. Tissue Doppler evaluation involves pulsed-wave Doppler interrogation of myocardial motion rather than blood flow, and this modality has provided new nongeometric parameters to assess ventricular function. Both AV valves have circumferential annular attachments to the ventricular myocardium, and each annulus is displaced along the longitudinal axis away from the apex in diastole and toward the apex in systole. MV annular motion is assessed at its lateral and septal junctions, whereas TV annular motion is assessed only at its lateral junction (Figure 10). The apical 4-chamber view provides an ideal window for the ventricular longitudinal axis, with little lateral motion (“rocking”) of the annulus during the cardiac cycle. Transducer position and angulation should be optimized to maintain Doppler alignment parallel to the direction of maximum annular motion. In children, annular velocities are best measured with a sample volume gate length of <5 mm. Because of the low-velocity Doppler signal of the myocardium, the Nyquist limit should be decreased to maximize the deflection size on the display (generally 15-30 cm/s) while using the lowest filter settings. In addition, decreasing the overall gain and maintaining the dynamic range at 30 to 35 dB can minimize the “noise” resulting from the low-amplitude and relatively high-frequency signal of blood flow. Pulsed tissue Doppler velocities are generally higher than velocities measured from color tissue Doppler imaging by 10% to 20%, and the two techniques cannot be used interchangeably to assess myocardial velocities.

The velocities of several diastolic and systolic peaks can be measured on tissue Doppler tracings of annular motion (Figure 11). Two negative diastolic peaks occur when the annulus moves away from the apex and can usually be identified separately as early diastolic annular motion (e' wave), which reflects ventricular recoil from a contracted state, and annular motion during atrial contraction (a' wave), which is affected by both ventricular diastolic and atrial systolic function. A positive systolic peak represents annular motion toward the apex during systole (s' wave). IVRT can be measured from the end of the e' wave to the onset of the e' wave, and isovolumic contraction time (IVCT) can be measured from the end of the a' wave to the onset of the s' wave. It is important to recognize that IVRT assessed by AV valve annular motion may not correlate with IVRT assessed by blood flow Doppler interrogation, particularly when diastolic dysfunction is present, because IVRT appears to be less influenced by filling pressures and to correlate well with $\tau$ (the LV relaxation time constant). A velocity peak is frequently seen during isovolumic contraction, and the isovolumic acceleration calculated as this velocity divided by the time to peak velocity is an index of systolic function. Age-related reference values of annular velocities and time intervals have been published for children and adolescents. Other Doppler indices of LV systolic and diastolic function have been reported. The estimated mean or peak isovolumic rate of pressure change (dP/dt) from the mitral regurgitation continuous-wave Doppler tracing has been used as a nongeometric index of LV systolic function. The velocity ratio of the blood flow Doppler-derived mitral inflow E wave to the tissue Doppler-derived e' wave has been used to assess LV diastolic function. Color M-mode measurements of the early diastolic flow propagation velocity from the MV to the apex correlate well with $\tau$ and provide another means by which to evaluate LV filling; as LV relaxation becomes abnormal, the rate of early diastolic flow propagation into the left ventricle decreases. Pediatric experience with these methods is limited, and their accuracy and reproducibility in children remain unknown. The myocardial performance index, calculated as isovolumic relaxation time plus isovolumic contraction time divided by ejection time and measured either by spectral or tissue Doppler analysis, has been used to assess combined LV systolic and diastolic function, and reference values in adults and children are available. More recently, myocardial deformation analyses with measurements of strain, strain rate, and ventricular torsion by tissue Doppler or speckle-tracking echocardiography have received attention as...
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**Calculation**

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<td>LV mass</td>
<td>Biplane Simpson with apical 4- and 2-chamber views</td>
<td>(EDV&lt;sub&gt;epi&lt;/sub&gt; – EDV) x 1.05 g/ml</td>
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<td>End-systolic volume (ESV)</td>
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<td>Velocity of circumferential fiber shortening corrected for heart rate and normalized to end-systolic wall stress</td>
<td>M-mode 2D</td>
<td>VCF = SF/ET&lt;sup&gt;c&lt;/sup&gt; VCF&lt;sub&gt;c&lt;/sub&gt; = SF/ET&lt;sub&gt;c&lt;/sub&gt; ESS = (P&lt;sub&gt;es&lt;/sub&gt; x R&lt;sub&gt;es&lt;/sub&gt;)&lt;sup&gt;c&lt;/sup&gt;/T&lt;sub&gt;es&lt;/sub&gt;</td>
<td>LV systolic function</td>
<td>Independent of preload Accounts for afterload and heart rate Normal pediatric data&lt;sup&gt;113&lt;/sup&gt;</td>
<td>Inappropriate for abnormal LV shape Time-consuming</td>
</tr>
<tr>
<td>Early diastolic velocity ratio</td>
<td>Tissue Doppler</td>
<td>E/e'</td>
<td>LV diastolic function</td>
<td></td>
<td>Depends on alignment (angle-dependent) Depends on loading conditions</td>
</tr>
<tr>
<td>Isovolumic acceleration (IVA)</td>
<td>Tissue Doppler</td>
<td>MV peak isovolumic annular velocity/time to peak velocity</td>
<td>LV systolic function</td>
<td>Same as above</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>, Minor axis slice radius in the apical 4-chamber view; <sup>b</sup>, minor axis slice radius in the apical 2-chamber view; <sup>c</sup>, End-diastolic area; <sup>d</sup>, end-diastolic epicardial area; <sup>e</sup>, end-diastolic diameter; <sup>f</sup>, end-diastolic length; <sup>g</sup>, end-diastolic epicardial length; <sup>h</sup>, end-diastolic posterior wall thickness; <sup>i</sup>, end-diastolic septal wall thickness; <sup>j</sup>, end-systolic area; <sup>k</sup>, end-systolic epicardial area; <sup>l</sup>, end-systolic diameter; <sup>m</sup>, end-systolic length; <sup>n</sup>, end-systolic epicardial length; <sup,o</sup>, end-systolic posterior wall thickness; <sup>p</sup>, end-systolic septal wall thickness; <sup>q</sup>, end-systolic wall stress; <sup>r</sup>, end-systolic septal wall thickness; <sup>s</sup>, ejection time; <sup>t</sup>, ET corrected for heart rate; <sup>u</sup>, left ventricular length; <sup>v</sup>, left ventricular; <sup>w</sup>, mitral valve; <sup>x</sup>, number of slices; <sup>y</sup>, pressure at end-systole; <sup>z</sup>, radius at end-systole; <sup>aa</sup>, wall thickness at end-systole; <sup>ab</sup>, three-dimensional; <sup>ac</sup>, two-dimensional; <sup>ad</sup>, velocity of circumferential fiber shortening; <sup>ae</sup>, VCF corrected for heart rate.

<sup>1</sup>Maximum minor-axis diameter or area.

<sup>†</sup>Minimum minor-axis diameter or area.

<sup>‡</sup>Measured by tissue Doppler evaluation rather than by blood flow Doppler evaluation, as described in Table 3.
potentially useful indices of LV function. The utility of these new techniques in children deserves further study.

Recommendations (Table 4): When tissue Doppler evaluation is performed at the medial and lateral MV annulus, the recommended measurements and calculations include peak e', a', and s' velocities; IVRT; IVCT; isovolumic acceleration; and the E/e' ratio.

Right Ventricle

Morphometric Evaluation. Two-dimensional echocardiography has been used to assess RV size and function, though it generally underestimates RV volumes compared with MRI. In addition, there are inherent limitations to the application of linear and cross-sectional area measurements to a geometrically complex chamber, and the right ventricle is technically difficult to evaluate by echocardiography because of its anterior retrosternal position. Nevertheless, guidelines on the assessment of RV wall thickness, size, and systolic function in adults have been published. The RV free wall thickness is difficult to quantify, though it can be measured in subxiphoid long-axis or parasternal views at end-diastole, making sure to avoid regions with significantly coarse trabeculations. As with the left ventricle in an apical 4-chamber view, the RV basal border is defined as the line connecting the TV annular hinge points. The RV basal and midcavity minor-axis diameters and RV major-axis length can be measured at end-diastole (defined as the frame at which the TV closes), taking care not to foreshorten the right ventricle (Figure 12). Again, 2D measurements of the RV have correlated weakly with MRI measurements, especially in the setting of RV volume overload.

RV long-axis area can be measured by planimetry, and RV fractional area change has been used as an index of RV systolic function. Multiple formulas have been proposed to estimate RV volume by 2D echocardiography. However, all of these methods have significant limitations, with little or no data regarding utility, accuracy, and reproducibility in children, and the best method for routine 2D measurement of RV volume remains controversial. RV EF can be calculated using these volume estimation methods and is regarded as a load-dependent index of RV systolic function. However, this approach has correlated only modestly with measurements from MRI and radionuclide imaging. Tricuspid annular plane systolic excursion (TAPSE) is another measure of RV systolic function that correlates well with EF as measured by radionuclide angiography and 2D echocardiography in adults. It measures longitudinal shortening of the right ventricle in an apical 4-chamber view, usually acquired by placing an M-mode cursor through the tricuspid annulus. Published normal values for TAPSE in children are available, though its clinical significance is still under investigation.

Recommendations (Table 5): The recommended methods to assess RV size include measurement of end-diastolic diameters at the basal and midcavity levels, end-diastolic length, and end-diastolic and end-systolic planimetered areas in apical 4-chamber views. The recommended methods to assess RV systolic function include TAPSE and fractional area change in apical 4-chamber views.

Doppler Evaluation. The peak velocity of the tricuspid regurgitation jet provides a good estimation of RV systolic pressure, particularly when RA pressure is low. Many of the previously described Doppler indices used to characterize LV function have been applied to the right ventricle. For example, tricuspid annular displacement measured by tissue Doppler evaluation can help assess RV systolic and diastolic function. The dP/dt estimated from the tricuspid regurgitation continuous-wave Doppler tracing has also been used to assess RV systolic function. RA pressure and RV diastolic function can be assessed by the IVC collapsibility index, hepatic venous flow indices, TV inflow velocities, the velocity ratio of TV E wave to TV e' wave, the velocity ratio of TV E wave to color M-mode inflow propagation, and RV IVRT. In addition,
Antegrade flow across the PV at end-diastole may suggest restrictive RV physiology. The myocardial performance index measured by conventional or tissue Doppler evaluation has also been used to assess combined RV systolic and diastolic function. However, the normal values obtained from these two approaches, however, can be significantly different, and the appropriate reference values should be used. Newer methods for assessing RV systolic and diastolic function, such as speckle tracking for strain and strain rate analyses and 3D echocardiography, deserve further study.

Figure 12 Apical 4-chamber view at end-diastole showing (A) right ventricular basal and midcavity diameters, (B) right ventricular length, and (C) right ventricular area. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
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<td>2D</td>
<td>End-diastole*</td>
<td>RV size</td>
<td>Normal adult data†</td>
<td>Foreshortening&lt;br&gt;Depends on good blood-endocardium border&lt;br&gt;Depends on distinct apical endocardium&lt;br&gt;Depends on loading conditions&lt;br&gt;Poorly predicts RV volume measured by MRI[^143]&lt;br&gt;No normal pediatric data</td>
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<td>Apical 4-chamber</td>
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<td>End-diastolic mid-cavity diameter</td>
<td>2D</td>
<td>End-diastole*</td>
<td>RV size</td>
<td>Normal adult data†</td>
<td>Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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<tr>
<td>End-diastolic length</td>
<td>2D</td>
<td>End-diastole*</td>
<td>RV size</td>
<td>Normal adult data†</td>
<td>Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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<tr>
<td>End-diastolic area (EDA)</td>
<td>2D</td>
<td>End-diastole*</td>
<td>RV size</td>
<td>Normal adult data†</td>
<td>Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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<tr>
<td>End-systolic area (ESA)</td>
<td>2D</td>
<td>End-systole†</td>
<td>RV size</td>
<td>Normal adult data†</td>
<td>Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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<tr>
<td>Tricuspid annular plane systolic excursion (TAPSE)</td>
<td>M-mode</td>
<td>Tricuspid annulus</td>
<td>RV systolic function</td>
<td>Correlates to EF&lt;br&gt;Quick and simple&lt;br&gt;Reproducible</td>
<td>Normal values not adjusted for body size[^152]&lt;br&gt;Not evaluated in children with CHD&lt;br&gt;Depends on loading conditions</td>
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<tr>
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<td>Apical 4-chamber view</td>
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<td>Tricuspid regurgitation (TR) jet peak velocity</td>
<td>Apical or parasternal</td>
<td>Systole</td>
<td>Estimation of RV systolic pressure</td>
<td></td>
<td>Depends on alignment (angle-dependent)&lt;br&gt;Trivial TR may not provide accurate estimate of RV pressure</td>
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<tr>
<td>Peak TV annular velocity in early diastole (e')</td>
<td>Tissue Doppler</td>
<td>Early diastole at lateral &amp; medial TV annulus</td>
<td>RV diastolic function</td>
<td>Reproducible&lt;br&gt;Good temporal resolution&lt;br&gt;Normal pediatric data[^117, ^118, ^123, ^124, ^126, ^127, ^129]&lt;br&gt;Depends on alignment (angle-dependent)&lt;br&gt;Depends on loading conditions&lt;br&gt;Not useful for regional wall motion abnormalities</td>
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<td>Apical 4-chamber</td>
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<tr>
<td>Peak TV annular velocity at atrial contraction (a')</td>
<td>Tissue Doppler</td>
<td>Atrial contraction at lateral &amp; medial TV annulus</td>
<td>RV diastolic function</td>
<td>Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above</td>
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<td>Apical 4-chamber</td>
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<tr>
<td>Peak TV annular velocity in systole (s')</td>
<td>Tissue Doppler</td>
<td>Systole at lateral &amp; medial TV annulus</td>
<td>RV systolic function</td>
<td>Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above</td>
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<td>Apical 4-chamber</td>
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<tr>
<td>Peak TV annular velocity in isovolumic contraction</td>
<td>Tissue Doppler</td>
<td>Isovolumic contraction at lateral TV annulus</td>
<td>RV systolic function</td>
<td>Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above</td>
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<td>Apical 4-chamber</td>
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<tr>
<td>Isovolumic relaxation time (IVRT')</td>
<td>Tissue Doppler</td>
<td>Time from end of s' wave to beginning of e' wave</td>
<td>RV diastolic function</td>
<td>Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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<tr>
<td>Time to peak velocity in isovolumic contraction</td>
<td>Tissue Doppler</td>
<td>Isovolumic contraction at lateral TV annulus</td>
<td>RV systolic function</td>
<td>Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above&lt;br&gt;Same as above</td>
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<td></td>
<td>Apical 4-chamber</td>
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(Continued)
Recommendations (Table 5): When tissue Doppler evaluation is performed at the TV annulus, the recommended measurements and calculations include peak e', a', and s' velocities; IVRT; and isovolumic acceleration.

Ventricular Outflow Tracts and Semilunar Valves

Morphometric Evaluation. Subvalvar and valvar outflow tract measurements adjusted for the effects of body size help evaluate potential outflow tract hypoplasia and annular dilation. The transducer imaging plane should be parallel to the outflow tract long axis (to allow for an axial measurement of the outflow tract), and magnification of the region of interest should be used. Although the subvalvar cross-section in both outflow tracts is often elliptical in shape, a circular shape is assumed and a single diameter measured. The maximum dimension of the narrowest subvalvar LV outflow
The tract diameter is best measured in parasternal long-axis views during early to mid-systole, and this value has been used to calculate stroke volume and cardiac output in adults. In contrast, the subvalvar RV outflow tract diameter has been variably measured in parasternal long-axis and short-axis views. Reference values for subvalvar diameters along both outflow tracts are available in adults. Data on the utility, accuracy, and reproducibility of these measurements in children are scant.

Table 6 Measurements of the Ventricular Outflow Tracts and Semilunar Valves

<table>
<thead>
<tr>
<th>Measurement</th>
<th>View/Location</th>
<th>Timing</th>
<th>Applications</th>
<th>Strengths</th>
<th>Weaknesses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subvalvar LV outflow tract</td>
<td>Parasternal long-axis</td>
<td>Systole*</td>
<td>LV outflow tract size</td>
<td>Normal adult data</td>
<td>Avoid oblique alignment</td>
</tr>
<tr>
<td>Aortic annulus diameter</td>
<td>Parasternal long-axis</td>
<td>Systole*</td>
<td>Aortic annulus size</td>
<td>Reproducibility</td>
<td>Non-circular geometry</td>
</tr>
<tr>
<td>Subvalvar RV outflow tract</td>
<td>Parasternal long- or short-axis</td>
<td>Systole*</td>
<td>RV outflow tract size</td>
<td>Normal adult data</td>
<td>Avoid oblique alignment</td>
</tr>
<tr>
<td>Pulmonary annulus</td>
<td>Parasternal long- or short-axis</td>
<td>Systole*</td>
<td>Pulmonary annulus size</td>
<td>Reproducibility</td>
<td>Non-circular geometry low antero-lateral resolution</td>
</tr>
<tr>
<td>LV outflow tract peak velocity</td>
<td>Apical 3-chamber, suprasternal long-axis, or right parasternal</td>
<td>Systole</td>
<td>Outflow tract obstruction</td>
<td>Depends on alignment</td>
<td>Affected by ventricular systolic function</td>
</tr>
<tr>
<td>RV outflow tract peak velocity</td>
<td>Subxiphoid short-axis, apical 4-chamber with anterior sweep, parasternal long-axis with leftward anterior sweep, or parasternal short-axis</td>
<td>Systole</td>
<td>Outflow tract obstruction</td>
<td>Depends on alignment</td>
<td>Affected by ventricular systolic function</td>
</tr>
</tbody>
</table>

LV, Left ventricular; RV, right ventricular.
*Maximum diameter.

Figure 14 Aortic root (Ao Root), sinotubular junction (STJ), and ascending aorta (AAo) diameters in a parasternal long-axis view at mid-systole. LV, Left ventricle; RPA, right pulmonary artery; RV, right ventricle.

Figure 15 Proximal transverse arch (PTA), distal transverse arch (DTA), and aortic isthmus (AI) diameters in a suprasternal long-axis view. LCCA, Left common carotid artery; LSA, left subclavian artery; RPA, right pulmonary artery.
AoV and PV annular diameters are best measured with magnification in parasternal long-axis views from the inner edge of the proximal valve insertion hinge point within the arterial root to the inner edge of the opposite hinge point (Figure 13). As with the subvalvar RV outflow tract, PV annular and main pulmonary artery diameters can also be measured in parasternal short-axis views. However, these measurements are often underestimated because they rely on the lateral imaging plane, with its relatively low resolution, and because an oblique orientation is often the only one available in these views. Both annular diameters have been measured at variable times during the cardiac cycle. For example, measurements of the aortic annulus and root in diastole have been recommended for children and adults, and reference values are available. However, systolic values for annular diameters appear to correlate best with intraoperative measurements. In addition, outflow tract imaging during mid-systole provides more consistent display of valve hinge points and intraluminal diameters, which in turn can be used to calculate stroke volume and cardiac output. Therefore, AoV and PV annular measurements during mid-systole are recommended in children.

Recommendations (Table 6): The diameters of the subvalvar LV outflow tract and aortic annulus are best measured in parasternal long-axis views during early to mid-systole. The diameters of the subvalvar RV outflow tract and pulmonary annulus can be measured in parasternal long-axis or short-axis views during mid-systole, using the largest diameters for documentation.

Doppler Evaluation. Doppler interrogation of the LV outflow tract is usually performed in apical 3-chamber, right parasternal, or suprasternal long-axis views, whereas the RV outflow tract can be interrogated in parasternal long-axis, subxiphoid short-axis, or modified diastolic views.

Figure 16 (A) Left main coronary artery (LMCA), proximal left anterior descending coronary artery (Prox LAD), distal left anterior descending coronary artery (Dist LAD), circumflex coronary artery (Circ), and proximal right coronary artery (Prox RCA) diameters in a parasternal short-axis view; (B) distal right coronary artery (Distal RCA) diameter in an apical 4-chamber view with posterior angulation; and (C) posterior descending coronary artery (PDCA) diameter in a parasternal long-axis view with posterior angulation. LA, Left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle.
apical views with anterior angulation. The measured velocity can vary significantly from one view to another, especially because the geometry and orientation of the outflow tracts may vary significantly among different patients, so the view used for the measurement should always be included in the report to maintain consistency in subsequent studies. Recommendations from the ASE and the European Association of Echocardiography on the quantification of AoV and PV stenosis in adults are available. The severity of total subvalvar and valvar outflow tract obstruction is usually determined from measurement of the maximum instantaneous and mean gradients by continuous-wave Doppler interrogation, and the cleanest and highest velocity spectral Doppler envelope should be used. Pulsed-wave Doppler interrogation using a low-frequency transducer can sometimes evaluate the contribution of subvalvar stenosis in the setting of multiple levels of obstruction, though it is important to remember that the dominant resistor of stenoses in series always masks the hemodynamic effects of the more distal levels of obstruction. In the setting of marked ventricular dysfunction and consequently low cardiac output, the Doppler-derived gradient does not always represent the severity of the obstruction. In fact, a modest gradient with significant ventricular systolic dysfunction should be considered as relatively severe stenosis. In addition, the severity of PV stenosis may not be accurately assessed when a large ventricular septal defect or patent ductus arteriosus results in equalization of ventricular and arterial pressures. In the setting of ventricular dysfunction or a large shunting lesion, abnormalities in semilunar valvar morphology (thickening, doming, commissural fusion) and annular size are as useful as the Doppler-derived gradient in assessing severity.

The maximum instantaneous gradient measured by Doppler echocardiography is different from the peak-to-peak gradient measured by catheterization, partially secondary to pressure recovery, a phenomenon that is particularly important in children. More severe degrees of aortic stenosis and a larger ascending aorta relative to annular size result in more turbulence and less pressure recovery. Because the ascending aorta is frequently less dilated in children with aortic stenosis compared with adults, pressure recovery can contribute substantially to the difference between Doppler-derived gradients and those obtained by catheterization, resulting in differences as high as 20% to 40%. The effective orifice area across a diseased valve can also help assess the degree of valvar stenosis, and valvar area can be measured by 2D planimetry or calculated by using the continuity equation, as described for the AV valves. Similar to the problems with the MV and TV, 2D planimetry of AoV and PV area is usually unreliable because of the irregular funnel-like doming of the stenotic valve and the difficulty with obtaining a reliable “en face” view of the true leaflet opening. The continuity equation also permits calculation of the AoV area, though measurements of the subvalvar LV outflow tract diameter can vary by as much as 5% to 8% in adults. This variability is exaggerated in the pediatric population, in which the smaller elliptical subvalvar cross-sectional area may increase the potential for error, thereby precluding routine use of this approach in children with small outflow tracts and semilunar valves. Quantitative assessment of AoV and PV regurgitation has also been discussed previously for adults, and the utility of vena contracta diameter and regurgitant jet area as well as the continuity equation and proximal isovelocity surface area phenomenon in children are limited and have not been validated.

Recommendations (Table 6): The maximum instantaneous and mean gradients along the LV outflow tract are best measured in apical 3-chamber, suprasternal long-axis, or right parasternal views. The gradients along the RV outflow tract are best measured in subxiphoid short-axis, modified apical 4-chamber, parasternal long-axis, or parasternal short-axis views.

Aorta, Coronary Arteries, and Pulmonary Arteries

Morphometric Evaluation. Measurement of arterial vessels helps identify patients with diverse vascular abnormalities, such as Marfan syndrome and Kawasaki disease. The proximal aorta is frequently dilated in the setting of a connective tissue disease (such as Marfan syndrome) or a bicuspid AoV. In contrast, narrowing at the sinotubular junction associated with supravalvar aortic stenosis is frequently seen in the setting of Williams syndrome. The proximal aorta should be measured at the following levels in a parasternal long-axis view at the moment of maximum expansion: the aortic root at the sinuses of Valsalva, the sinotubular junction, and the ascending aorta as it crosses in front of the right pulmonary artery (Figure 14). Optimal imaging of the entire proximal aorta is not always available in the standard parasternal window, and a high left parasternal view located one or two rib interspaces superior to the standard location may be required. Often, a high right parasternal view in a right lateral decubitus position is better at displaying the entire proximal aorta. After the aortic arch sidedness has been established and the branches identified in
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<td>Systole*</td>
<td>Proximal aorta size</td>
<td>Normal adult data(^1) Normal pediatric data(^{15,17})</td>
<td>May not reflect largest diameter in abnormally shaped aortic roots(^{102}) Depends on alignment</td>
</tr>
<tr>
<td>Aortic sino-tubular junction diameter</td>
<td>Parasternal long-axis, high left parasternal, or high right parasternal</td>
<td>Systole*</td>
<td>Proximal aorta size</td>
<td>Same as above</td>
<td>Same as above</td>
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<tr>
<td>Ascending aorta diameter</td>
<td>Parasternal long-axis, high left parasternal, or high right parasternal at level of RPA</td>
<td>Systole*</td>
<td>Proximal aorta size</td>
<td>Same as above</td>
<td>Same as above</td>
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<tr>
<td>Proximal transverse arch diameter</td>
<td>Suprasternal long-axis between RCCA and LCCA</td>
<td>Systole*</td>
<td>Aortic arch size</td>
<td>Same as above</td>
<td>Same as above</td>
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<tr>
<td>Distal transverse arch diameter</td>
<td>Suprasternal long-axis between LCCA and RSA</td>
<td>Systole*</td>
<td>Aortic arch size</td>
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<tr>
<td>Aortic isthmus diameter</td>
<td>Suprasternal long-axis distal to RSA</td>
<td>Systole(^x)</td>
<td>Aortic arch size</td>
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<tr>
<td>Descending aorta diameter</td>
<td>Subxiphoid short-axis at level of diaphragm</td>
<td>Systole*</td>
<td>Descending aorta size</td>
<td>No normal pediatric data</td>
<td></td>
</tr>
<tr>
<td>Left main coronary artery (CA) diameter</td>
<td>Parasternal short-axis</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td>Normal pediatric data(^{36})</td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Proximal left anterior descending CA diameter</td>
<td>Parasternal short-axis</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td>Normal pediatric data(^{36})</td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Distal left anterior descending CA diameter</td>
<td>Parasternal short-axis</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td>Normal pediatric data(^{36})</td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Left circumflex CA diameter</td>
<td>Parasternal short-axis</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td>Normal pediatric data(^{36})</td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Proximal right CA diameter</td>
<td>Parasternal short-axis</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td>Normal pediatric data(^{36})</td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Distal right CA diameter</td>
<td>Apical 4-chamber with posterior sweep</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td></td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Posterior descending CA diameter</td>
<td>Parasternal long-axis with rightward posterior sweep</td>
<td>Maximum</td>
<td>Coronary artery size</td>
<td></td>
<td>Depends on good spatial &amp; contrast resolution</td>
</tr>
<tr>
<td>Main pulmonary artery (MPA) diameter</td>
<td>Parasternal short-axis</td>
<td>Systole*</td>
<td>Pulmonary artery size</td>
<td>Normal adult data(^1) Normal pediatric data(^{15,17})</td>
<td>Incomplete visualization secondary to anterior location</td>
</tr>
<tr>
<td>Right pulmonary artery (RPA) diameter</td>
<td>Parasternal, high left parasternal, or suprasternal short-axis</td>
<td>Systole*</td>
<td>Pulmonary artery size</td>
<td>Normal adult data(^1) Normal pediatric data(^{15,17})</td>
<td>Incomplete visualization secondary to anterior location</td>
</tr>
</tbody>
</table>

(Continued)
and proximal right coronary arteries (Figure 16). Occasionally, the main, proximal and distal left anterior descending, circumflex, parasternal short-axis views should be used to measure the left with the anterior and posterior descending coronary arteries. or artifacts that resemble the coronary arteries, a particular problem used when possible to avoid erroneous measurement of cardiac veins expansion. Low-scale color mapping with dual display should be plane. Measurements should be made at the moment of maximum left subclavian artery) (Figure 15). In addition, the descending aorta arteries), and the aortic isthmus (narrowest aortic segment distal to the transverse arch (between the left common carotid and left subclavian arteries), the distal and ascending aorta are best measured during mid-systole and aortic isthmus diameters are best measured during parameters at the levels of the aortic root, sinotubular junction, and aortic isthmus (narrowest aortic segment distal to the left subclavian artery) (Figure 15). In addition, the descending aorta may be measured in a subxiphoid short-axis view at the level of the diaphragm. The coronary arteries may become dilated with increased flow or inflammation. They are often difficult to image in a single 2D plane. Measurements should be made at the moment of maximum expansion. Low-scale color mapping with dual display should be used when possible to avoid erroneous measurement of cardiac veins or artifacts that resemble the coronary arteries, a particular problem with the anterior and posterior descending coronary arteries. Parasternal short-axis views should be used to measure the left main, proximal and distal left anterior descending, circumflex, and proximal right coronary arteries (Figure 16). Occasionally, the left anterior descending coronary artery is better visualized in a parasternal long-axis or a modified parasternal view (between the long-axis and short-axis views). The distal right coronary artery is best seen along the right posterior AV groove in modified apical views with posterior angulation, and the posterior descending coronary artery is best seen along the posterior interventricular groove in a suprasternal short-axis sweep, the aortic arch is measured in a suprasternal long-axis view, though a modified right parasternal view is occasionally better, particularly in neonates. Measurements should be performed at the following levels: the proximal transverse arch (between the innominate and left common carotid arteries), the distal transverse arch (between the left common carotid and left subclavian arteries) and the aortic isthmus (narrowest aortic segment distal to the left subclavian artery) (Figure 15). In addition, the descending aorta may be measured in a subxiphoid short-axis view at the level of the diaphragm. The coronary arteries may become dilated with increased flow or inflammation. They are often difficult to image in a single 2D plane. Measurements should be made at the moment of maximum expansion. Low-scale color mapping with dual display should be used when possible to avoid erroneous measurement of cardiac veins or artifacts that resemble the coronary arteries, a particular problem with the anterior and posterior descending coronary arteries. Parasternal short-axis views should be used to measure the left main, proximal and distal left anterior descending, circumflex, and proximal right coronary arteries (Figure 16). Occasionally, the left anterior descending coronary artery is better visualized in a parasternal long-axis or a modified parasternal view (between the long-axis and short-axis views). The distal right coronary artery is best seen along the right posterior AV groove in modified apical views with posterior angulation, and the posterior descending coronary artery is best seen along the posterior interventricular groove in modified parasternal long-axis views with rightward posterior angulation.

Assessment of pulmonary artery size is important in children with various forms of CHD. When pulmonary arterial flow is diminished (as in tetralogy of Fallot), the branch pulmonary arteries are typically small. In contrast, isolated PV stenosis, tetralogy of Fallot with a dysplastic PV, Marfan syndrome, and pulmonary hypertension are all associated with pulmonary artery dilation. The pulmonary arteries can be evaluated in parasternal or suprasternal short-axis views, though suprasternal measurements exhibit less variability, presumably because of less translational cardiac motion in this view. The diameters of the main pulmonary artery (between the pulmonary sinotubular junction and bifurcation) and the branch pulmonary arteries can be measured in a parasternal short-axis view (Figure 17). The right pulmonary artery can also be measured as it crosses behind the ascending aorta in a suprasternal short-axis view, whereas the left pulmonary artery can also be measured at its origin from the main pulmonary artery in a left anterior oblique or sagittal plane in a suprasternal or high left parasternal (“ductal”) view.

Recommendations (Table 7): The proximal aortic diameters at the levels of the aortic root, sinotubular junction, and ascending aorta are best measured during mid-systole in parasternal long-axis, high left parasternal, or high right parasternal views; the proximal and distal transverse arch and aortic isthmus diameters are best measured during mid-systole in suprasternal long-axis views; and the

| Table 7 | (Continued) |
|---|---|---|---|---|---|
| Measurement | View/Location | Timing | Applications | Strengths | Weaknesses |
| Left pulmonary artery (LPA) diameter | Parasternal, high left parasternal, or suprasternal short-axis | Systole* | Pulmonary artery size | Normal adult data¹ Normal pediatric data¹⁸⁶,¹⁷ | Incomplete visualization secondary to anterior location |
| Ascending aorta peak velocity | Apical 3-chamber, suprasternal long-axis, or right parasternal | Systole | Aortic outflow tract obstruction | Depends on alignment Difficult with multiple levels of obstruction |
| Aortic isthmus peak velocity | Suprasternal long-axis | Systole | Aortic arch obstruction | Requires correction for proximal velocity Depends on alignment Affected by PDA |
| Abdominal aortic Doppler | Subxiphoid short-axis at level of diaphragm | | Aortic arch obstruction Diastolic reversal from aortic regurgitation or aortic fistulous connection | |
| MPA peak velocity | Parasternal short-axis or apical 4-chamber with anterior sweep | Systole | Pulmonary outflow tract obstruction | Depends on alignment Difficult with multiple levels of obstruction |
| RPA and LPA peak velocity | Parasternal or suprasternal short-axis or high left parasternal | Systole | Branch pulmonary artery obstruction | Depends on alignment Difficult with multiple levels of obstruction |

¹Maximum diameter.

CA, Coronary artery; LCCA, left common carotid artery; LPA, left pulmonary artery; MPA, main pulmonary artery; PDA, patent ductus arteriosus; RCCA, right common carotid artery; RPA, right pulmonary artery; RSA, right subclavian artery.
descending aorta diameter is best measured during mid-systole in subxiphoid short-axis views at the level of the diaphragm. The left main, proximal and distal left anterior descending, circumflex, and proximal right coronary artery diameters are best measured at the moment of maximum expansion in parasternal short-axis views; the distal right coronary artery diameter is best measured at the moment of maximum expansion in apical 4-chamber views with posterior angulation; and the posterior descending coronary artery diameter is best measured at the moment of maximum expansion in parasternal long-axis views with rightward posterior angulation. The main, right, and left pulmonary artery diameters are best measured during mid-systole in parasternal, high left parasternal, or suprasternal short-axis views.

**Doppler Evaluation.** Doppler evaluation of vascular structures helps identify and characterize obstruction. Similar to the AoV, Doppler interrogation of the proximal aorta is best performed in apical 3-chamber, right parasternal, or suprasternal long-axis views. The aortic arch should be evaluated in a suprasternal long-axis view with step-by-step pulsed-wave Doppler interrogation from the proximal transverse arch to the proximal descending aorta. The flow velocity in the distal transverse arch is usually too high to be ignored in the simplified Bernoulli equation, so calculated gradients along the arch should account for the proximal velocity. Pulsed-wave Doppler interrogation of the abdominal aorta in a subxiphoid short-axis view (displaying the abdominal aorta long axis) just below the diaphragm often gives the first clue that aortic arch obstruction exists. The normal pattern reveals a brisk upstroke and return to baseline, and blunting with delayed or no return to baseline is an important indicator of significant obstruction proximal to the sample site. In addition, the normal abdominal aortic Doppler pattern often reveals a brief early diastolic flow reversal secondary to aortic recoil and coronary artery flow, and holodiastolic flow reversal suggests a large aortopulmonary shunt (such as a patent ductus arteriosus) or significant aortic regurgitation.

Doppler interrogation of the main pulmonary artery is best performed in parasternal short-axis views or modified apical views with anterior angulation. In normal neonates, the branch pulmonary arteries are frequently relatively narrow, often originating from the main pulmonary artery at a slightly more acute angle. The associated flow acceleration along the proximal branch pulmonary arteries results in a benign murmur which typically resolves by 3 to 4 months of age. Standard protocols involve pulsed-wave Doppler interrogation at the origin of each branch. The line of interrogation should be parallel to the axis of the branch pulmonary arteries, and this is best performed in parasternal or suprasternal short-axis views or in a high left parasternal transverse view ("pant-leg" view). Occasionally, a modified left subclavian view will provide the best angle of interrogation along the proximal right pulmonary artery. In contrast, Doppler interrogation of the proximal left pulmonary artery is often better performed in a high left parasternal or sagittal ("ductal") view.

**Recommendations (Table 7):** The abdominal aortic Doppler pattern is best evaluated in subxiphoid short-axis views. The maximum instantaneous gradient along the ascending aorta is best measured in apical 3-chamber, suprasternal long-axis, or right parasternal views. The maximum instantaneous gradient along the aortic isthmus is best measured in suprasternal long-axis views and should account for the proximal velocity along the transverse aortic arch. The maximum instantaneous gradient along the main pulmonary artery is best measured in parasternal short-axis or modified apical views with anterior angulation. The maximum instantaneous gradient along the right and left pulmonary arteries is best measured in parasternal or suprasternal short-axis or high left parasternal views.

**CONCLUSIONS AND LIMITATIONS**

Quantification in pediatric echocardiography requires a consensus on what and how measurements should be made in a standard protocol, and this document has been constructed as a "manual of operations" to address this need. Although it presents a comprehensive list of guidelines when performing measurements during a pediatric echocardiogram, there are several limitations to this document. First, it does not explore the relative value of each measurement as it pertains to prognosis and outcome and therefore does not represent a list of measurements which should be performed on the basis of accuracy, reproducibility, and prognostic importance. In addition, it does not provide guidelines on how the measurements should be included in an echocardiographic report, especially in this era of structured reporting. Last, it does not fully address measurements obtained by 3D echocardiography and myocardial deformation analysis, two techniques that are currently undergoing extensive evaluation and will likely play an important role in clinical practice. This document, however, does standardize quantification methods as the first step in the task of generating a normative database that encompasses the range of body sizes and ages encountered in the pediatric population.

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**REFERENCES**

1. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440–63.


