

Chapter 1

Transthoracic Echocardiography

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Echocardiography has become the most widely used imaging procedure in patients with cardiovascular disease. The basis of echocardiography relies on the transmission and reflection of ultrasound waves from a transducer and interaction with different tissue interfaces to generate a digital image. The images obtained include M-Mode, 2D, 3D and Doppler (CW: continuous wave, PW: pulse wave, color and tissue Doppler) to define both normal and abnormal structures and pathologies. The uses include evaluation of the structures of the cardiac chamber walls, the systolic and diastolic performances of the ventricles, the structure and function of the native or prosthetic cardiac valves, evidence of pericardial effusion and constriction, the appearance of the proximal great vessels and inferior vena cava, and the presence of abnormal intracardiac shunting.

Indications

The American College of Cardiology (ACC) and American Heart Association (AHA) published guidelines for the clinical application of echocardiography in 1997 (with an update in 2003) for various cardiovascular conditions [1]. In 2011, the ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR Appropriate Use Criteria for Echocardiography were published with the level of appropriateness to guide physicians [2]. These of indications are extensive and generally includes usefulness in the acute setting and in patients with cardiac signs and symptoms, patients with valvular heart disease, hypertension, heart failure and cardiomyopathies, cardiac masses and pericardial diseases, suspected cardiovascular sources of emboli, and aortic diseases (Table 1.1).

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Table 1.1 Appropriate indications for transthoracic echocardiography

1.	Symptoms or conditions potentially related to suspected cardiac etiology including but not limited to chest pain, shortness of breath, palpitations, TIA, stroke, or peripheral embolic event
2.	Prior testing that is concerning for heart disease or structural abnormality including but not limited to chest X-ray, baseline scout images for stress echocardiogram, ECG, or cardiac biomarkers
3.	Frequent VPCs or exercise-induced VPCs, Sustained or non-sustained atrial fibrillation, SVT, or VT
4.	Clinical symptoms or signs consistent with a cardiac diagnosis known to cause lightheadedness/presyncope/syncope (including but not limited to aortic stenosis, hypertrophic cardiomyopathy, or HF), Syncope when there are no other symptoms or signs of cardiovascular disease
5.	Evaluation of suspected pulmonary hypertension including evaluation of right ventricular function and estimated pulmonary artery pressure, Routine surveillance (>1 year) of known pulmonary hypertension without change in clinical status or cardiac exam, Re-evaluation of known pulmonary hypertension if change in clinical status or cardiac exam or to guide therapy
6.	Hypotension or hemodynamic instability of uncertain or suspected cardiac etiology
7.	Acute chest pain with suspected MI and non-diagnostic ECG when a resting echocardiogram can be performed during pain, Evaluation of a patient without chest pain but with other features of an ischemic equivalent or laboratory markers indicative of ongoing MI
8.	Suspected complication of myocardial ischemia/infarction, including but not limited to acute mitral regurgitation, ventricular septal defect, free-wall rupture/tamponade, shock, right ventricular involvement, HF, or thrombus
9.	Initial evaluation of ventricular function following ACS, Re-evaluation of ventricular function following ACS during recovery phase when results will guide therapy
10.	Respiratory failure or hypoxemia of uncertain etiology
11.	Known acute pulmonary embolism to guide therapy (e.g., thrombectomy and thrombolytics), Re-evaluation of known pulmonary embolism after thrombolysis or thrombectomy for assessment of change in right ventricular function and/or pulmonary artery pressure
12.	Severe deceleration injury or chest trauma when valve injury, pericardial effusion, or cardiac injuries are possible or suspected
13.	Re-evaluation of known valvular heart disease with a change in clinical status or cardiac exam or to guide therapy
14.	Routine surveillance (>3 years) of mild valvular stenosis without a change in clinical status or cardiac exam, Routine surveillance (>1 year) of moderate or severe valvular stenosis without a change in clinical status or cardiac exam
15.	Routine surveillance (>1 year) of moderate or severe valvular regurgitation without a change in clinical status or cardiac exam
16.	Initial postoperative evaluation of prosthetic valve for establishment of baseline, Routine surveillance (>3 years after valve implantation) of prosthetic valve if no known or suspected valve dysfunction, Evaluation of prosthetic valve with suspected dysfunction or a change in clinical status or cardiac Exam, Re-evaluation of known prosthetic valve dysfunction when it would change management or guide therapy

(continued)

Table 1.1 (continued)

17.	Initial evaluation of suspected infective endocarditis with positive blood cultures or a new murmur, Re-evaluation of infective endocarditis at high risk for progression or complication or with a change in clinical status or cardiac exam
18.	Suspected cardiac mass
19.	Suspected cardiovascular source of embolus
20.	Suspected pericardial conditions, re-evaluation of known pericardial effusion to guide management or therapy
21.	Guidance of percutaneous noncoronary cardiac procedures including but not limited to pericardiocentesis, septal ablation, or right ventricular biopsy
22.	Evaluation of the ascending aorta in the setting of a known or suspected connective tissue disease or genetic condition that predisposes to aortic aneurysm or dissection (e.g., Marfan syndrome), Re-evaluation of known ascending aortic dilation or history of aortic dissection to establish a baseline rate of expansion or when the rate of expansion is excessive, Re-evaluation of known ascending aortic dilation or history of aortic dissection with a change in clinical status or cardiac exam or when findings may alter management or therapy
23.	Initial evaluation of suspected hypertensive heart disease
24.	Initial evaluation of known or suspected HF (systolic or diastolic) based on symptoms, signs, or abnormal test results, Re-evaluation of known HF (systolic or diastolic) with a change in clinical status or cardiac exam without a clear precipitating change in medication or diet, Re-evaluation of known HF (systolic or diastolic) to guide therapy
25.	Initial evaluation or re-evaluation after revascularization and/or optimal medical therapy to determine candidacy for device therapy and/or to determine optimal choice of device
26.	Known implanted pacing device with symptoms possibly due to device complication or suboptimal pacing device settings
27.	To determine candidacy for ventricular assist device, Optimization of ventricular assist device settings, Re-evaluation for signs/symptoms suggestive of ventricular assist device-related complications
28.	Monitoring for rejection in a cardiac transplant recipient
29.	Cardiac structure and function evaluation in a potential heart donor
30.	Re-evaluation of known cardiomyopathy with a change in clinical status or cardiac exam or to guide therapy
31.	Screening evaluation for structure and function in first-degree relatives of a patient with an inherited cardiomyopathy
32.	Baseline and serial re-evaluations in a patient undergoing therapy with cardiotoxic agents
33.	Initial evaluation of known or suspected adult congenital heart disease, Known adult congenital heart disease with a change in clinical status or cardiac exam, Re-evaluation to guide therapy in known adult congenital heart disease

Contraindications

Transthoracic echocardiography has no contraindications, as the use of ultrasound has no adverse effects when used for cardiac imaging. However, ultrasound waves have the potential to cause thermal bioeffects depending on the intensity and length of exposure that are determined by the frequency, focus, power output, depth, perfusion, tissue density; these bioeffects are considered minimal.

Contrast agents, when used, include the following contraindications:

1. Clinical instability with hypotension in such cases as acute myocardial infarction, worsening or clinically unstable heart failure, life threatening ventricular arrhythmias, respiratory failure, severe emphysema, pulmonary embolism;
2. Right-left, bidirectional, or transient right-to-left cardiac shunts;
3. Hypersensitivity to contrast agents.

For additional details about contrast agents, please see Chap. 3 (Contrast echocardiography)

Equipment

The necessary equipment for performing an echocardiographic exam includes the portable echocardiography unit, a suitable ultrasound transducer (typically 2–4 Mhz) and an experienced sonographer or physician. The ultrasound transducer uses a piezoelectric crystal (such as quartz or titanate ceramic) to generate and receive ultrasound waves. The received waves are converted to electrical signals and displayed on the echocardiographic machine. The equipment is portable and allows examination in multiple locations aside from the echocardiography laboratories.

Technique

The echocardiographic examination starts by connecting the ECG electrodes and positioning the patient comfortably in the left lateral decubitus position to obtain optimal images (Obtaining images in supine position are possible if patients are unable to lie on their side). The ultrasound transducer is applied (using a water soluble gel) to the parasternal, apical, subcostal and in some cases suprasternal notch to obtain the usual images of an echocardiographic protocol. Parasternal long axis (PLAX), parasternal short axis (PSAX), apical 4 (A4C), 2 (A2C), and 3-chamber (A3C or long axis), subcostal, and suprasternal notch images are obtained. Doppler echocardiography (color flow, continuous and pulsed wave) is used to determine regurgitant and stenotic flow across valves and measure the velocity, pressure gradients, and volumetric flow. Tissue Doppler (mitral annulus) is used in determining diastolic function.

1. The parasternal long axis view (PLAX) (Fig. 1.1) with the transducer slightly left of the sternum is initiated with the 2D evaluation of a sagittal view of the left ventricle (LV) (long axis view). This view allows evaluation of the structure and systolic function of the LV including LV outflow tract, left atrium (LA), the structure and motion of the aortic valve (AV) right and non-coronary cusps, the proximal aortic size and wall characteristics, and a portion of the right ventricular outflow. The posterior pericardium can also be evaluated for thickening or

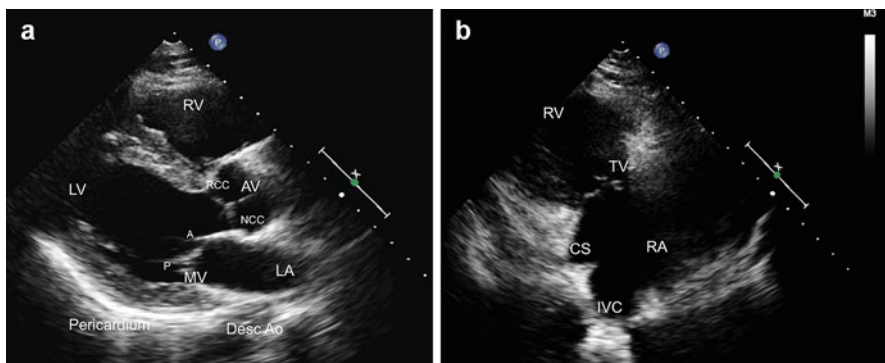


Fig. 1.1 Parasternal long axis (PLAX) views: (a) shows PLAX with the LA, LV, MV-mitral valve with anterior and posterior leaflets, AV-aortic valve with RCC-right coronary cusp and NCC-non-coronary cusp. The RV lies anteriorly and posteriorly the pericardium and descending aorta are seen. In the PLAX, the anteroseptal and inferolateral walls of the LV are evaluated for any wall motion abnormalities. Panel (b) shows an RV inflow view with the RV, RA, the TV-tricuspid valve as well as the ostia of the IVC-inferior vena cava and the CS-coronary sinus

presence of pericardial effusion and more posteriorly pleural effusion. The septum is visualized and information can be obtained on the presence of a ventricular septal defect (VSD).

2. A parasternal right ventricular (RV) inflow (Fig. 1.1) view can be obtained by medial angulation of the transducer from the parasternal long axis position. This allows visualization of the tricuspid valve (TV), right atrium (RA), coronary sinus (CS), and RV inflow tract. Tricuspid regurgitation can be evaluated and using continuous flow (CW) Doppler, right ventricular systolic pressure (RSVP) can be calculated.
3. Parasternal short axis view (PSAX) (Fig. 1.2) is orthogonal to the long axis view. It is obtained by rotating the transducer 90°. Cross sectional evaluation of the LV, mitral valve (MV), AV, and LA are obtained, as well as views of the interatrial septum, RA, TV, RV outflow tract, PV, proximal PA and main PA branches. Doppler studies allow assessment of aortic regurgitation, tricuspid regurgitation, PA velocity, and the presence of pulmonic stenosis or regurgitation. Shunts that can be assessed in this view include membranous and supracristal VSD, patent ductus arteriosus (PDA), atrial septal defects (ASD) and patent foramen ovale (PFO)
4. Apical four chamber (A4C) view (Fig. 1.3): The transducer is placed on the left side of the chest as far inferiorly and laterally as possible to obtain views of the four chambers, including the mitral and tricuspid valves. This view allows assessment of the LV apex, inferoseptal and anterolateral walls and the lateral RV wall. The view is useful for evaluation of global LV systolic performance and the presence of LV apical aneurysm and thrombus. Diastolic performance can be determined by Doppler assessment of mitral inflow, pulmonary vein, and mitral annular (tissue Doppler) motion. This is the best view for assessing the presence and degree of mitral and tricuspid stenosis and regurgitation and is useful for

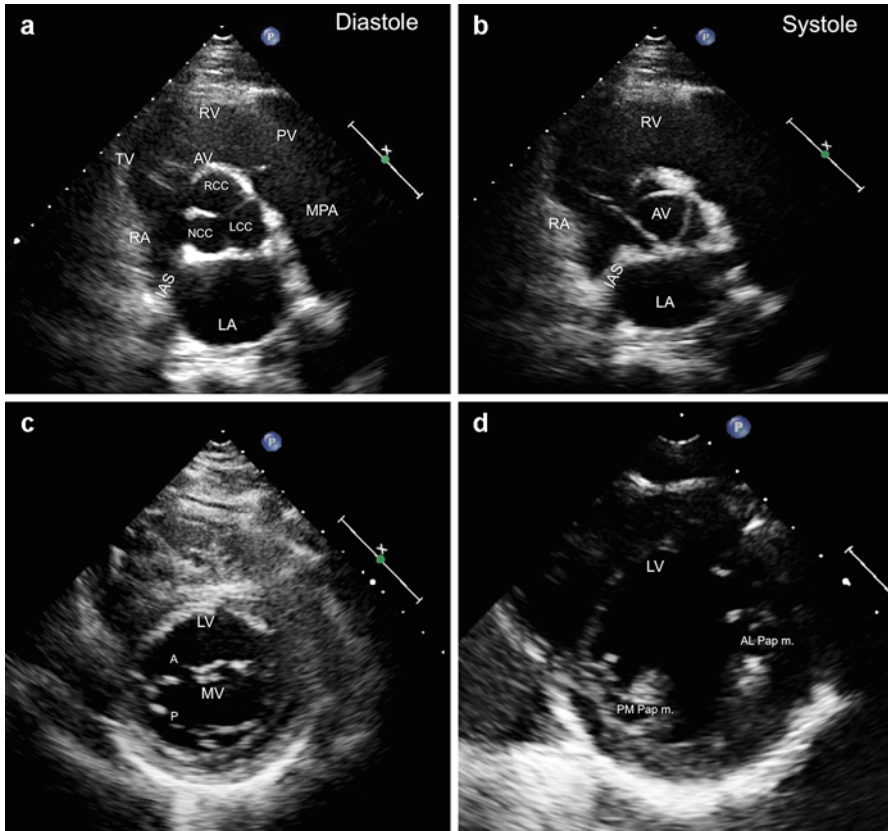


Fig. 1.2 Parasternal short axis (PSAX) views: (a, b) At the aortic level in diastole (AV closed) and systole (AV open). Note the three cusps: RCC, LCC, and NCC (located at the IAS-interatrial septum). Also seen are the LA, RA, IAS with slight dropout, RV outflow, PV-pulmonary vein and MPA-main pulmonary artery. Panel (c) shows short axis of the LV at the mitral valve level showing the anterior and posterior leaflets. Panel (d) shows a short axis of the LV at the papillary muscle level with the AL-anterolateral and PM-posteromedial papillary muscles from [1]

assessing the extent of pericardial effusion, and respirophasic characteristics of mitral and tricuspid characteristics that may include pericardial tamponade. RV and RA invagination or compression also suggesting tamponade can be seen in these views. Doppler interrogation for muscular VSD is best accomplished in this view. Saline intravenous injections for R-L shunts are best determined in this view.

5. Apical 2 chamber (A2C) view (Fig. 1.3): Rotation of the transducer 60° counter-clockwise provides visualization of the LV, MV, and LA. This view allows evaluation of the LV inferior and anterior walls, further evaluation for mitral regurgitation, and may be the best view for demonstration of LV pseudoaneurysm, which usually involves the inferior wall.
6. Apical 3 chamber (A3C) view (Fig. 1.3): Rotating the transducer 60° clockwise and tilting is slightly anteriorly allows visualization of the LV, MV, LA, LV

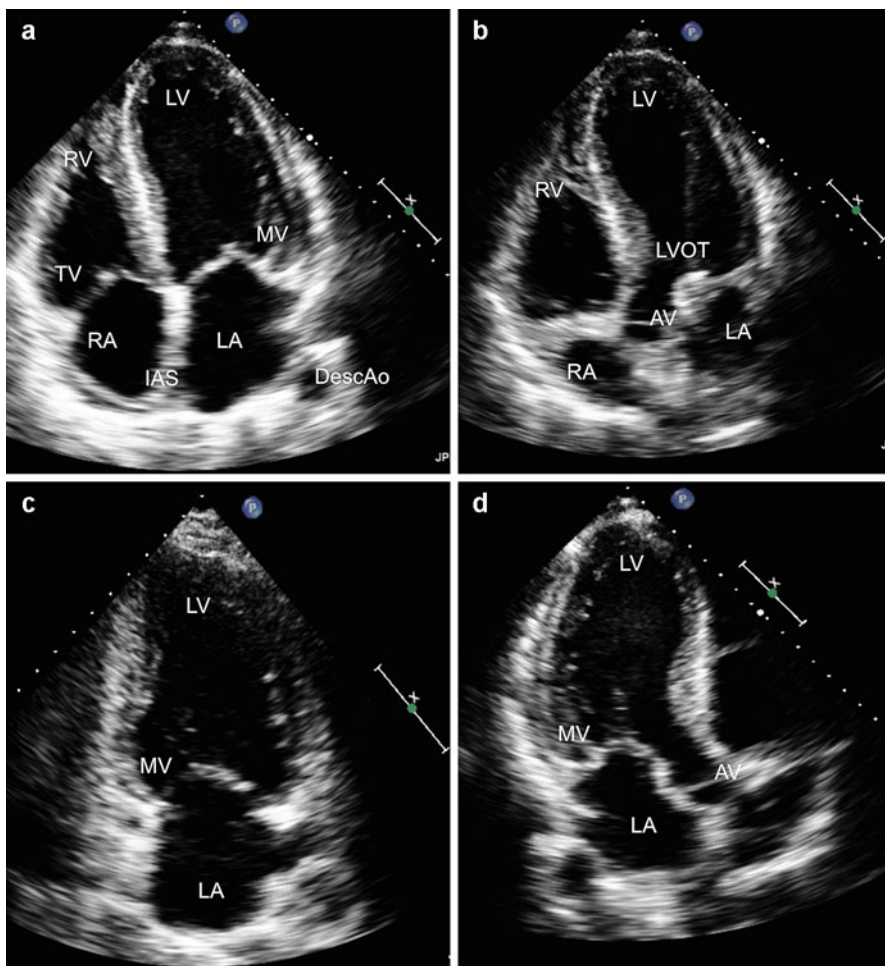


Fig. 1.3 Apical views. (a) Apical 4 chamber (A4C) view showing the LA, RA, RV, LV (anterolateral and inferoseptal walls), and the MV/TV. (b) Apical 5 chamber (A5C) with slight anterior angulation of the transducer, the aorta is opened and the aortic valve can be seen and interrogated with CW to evaluate for AS, and PW of the LVOT to measure the SV-stroke volume. (c) Apical 2 chamber (A2C) showing the LA, MV and LV (Anterior and inferior walls). (d) Long axis (A3C) showing the LA, LV, MV, and AV. Similar to the PLAX, the anteroseptal and inferolateral walls of the LV are seen from [5]

outflow tract, AV and proximal aorta. It is the best view for Doppler interrogation for aortic stenosis, subaortic stenosis and aortic regurgitation.

7. Subcostal view (Fig. 1.4): The transducer is placed in the epigastrium just below the subxiphoid process. It is the best view for evaluating RA and RV free wall motion, and presence of PFO or ASD. The inferior vena cava is best viewed in this location. The diameter of which is measured and used to estimate RA pressure. The abdominal aorta can be seen several centimeters below the diaphragm, which allows the evaluation of aneurysms or dissection at this level.

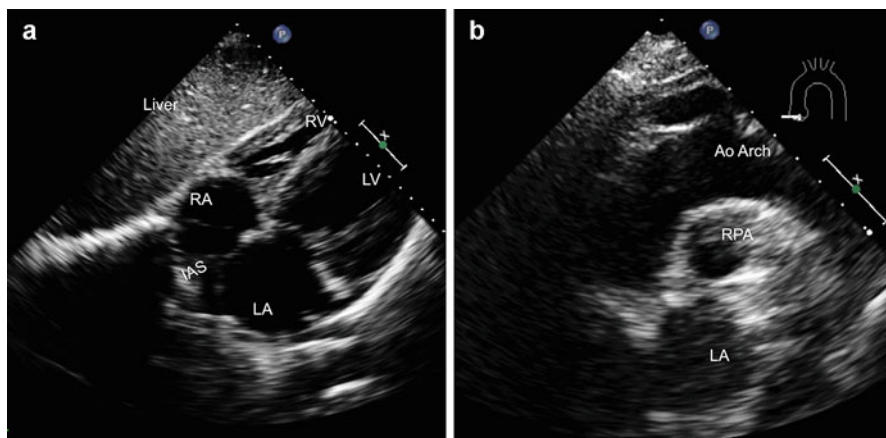


Fig. 1.4 Subcostal and suprasternal TTE views: (a) A subcostal view with the four chambers LA, LV, RA, and RV are seen. This is the best view to evaluate for the presence of IAS-interatrial septal defects such as ASDs or PFOs using color flow Doppler. (b) Suprasternal notch view showing the aortic arch, RPA-right pulmonary artery and LA. This view is used to evaluate for the presence of aortic coarctation and diastolic flow reversal to determine the severity of aortic regurgitation

- Suprasternal view (Fig. 1.4): This view allows Doppler assessment of the ascending aortic velocities in aortic stenosis and can provide 2D imaging of the transverse and proximal descending thoracic aorta. Doppler assessment of the descending aorta in this view may be useful for the diagnosis of PDA and coarctation.

In general, standard images can be obtained in most patients with limitations in those with extremes of weight, with severe lung hyperinflation (i.e. severe COPD), or in patients in which access to the chest wall is precluded in setting of thoracic bone malformations, open-heart surgery or other thoracic surgeries. In difficult to image patients, ultrasound contrast agents are used to improve image quality.

Data Interpretation

Physicians with specialized training should perform data interpretation as established by competency guidelines from the ACC/AHA/ASE to generate a formal report. It is important to use a consistent systemic approach with special attention directed to the clinical question.

A systemic approach includes the following:

Left Ventricle: Chamber dimension, wall thickness, fractional shortening (FS), ejection fraction, regional wall motion abnormalities, stroke volume, diastolic characteristics and presence of VSD [3].

Right Ventricle: Chamber dimension, wall thickness and global function.

Left atrium: size, presence of masses or cor triatriatum.

Right atrium: Size, abnormal motion suggesting pericardial tamponade.

Proximal aorta: size, evidence of dissection and fibrocalcific changes.

Proximal PA: size, presence of PDA.

Valves: Stenosis, regurgitation, vegetations, calcification, prolapse, bicuspid and other AV abnormalities.

Congenital anomalies: PFO, ASD, VSD, cor triatriatum, Ebstein anomaly, transposition of ventricles and other congenital abnormalities.

Pericardium: Pericardial effusion, tamponade, constriction.

Hemodynamic: Hemodynamics play an important role in echocardiography and is helpful in evaluating the degree of right to left or left to right shunting, degree of mitral and aortic stenosis, regurgitant flow, PA systolic pressure.

Flows can be calculated using the formula $\text{Flow Rate} = \text{Area} (\pi r^2) \times V_{\text{max}}$. πr^2 represents the area across a circle assuming the left ventricular outflow tract (LVOT) is circular in shape. The volume is calculated as $\text{Volume} = \text{Area} (\pi r^2) \times \text{VTI}$ (Velocity time integral: measured by tracing the CW or PW signal). The stroke volume (SV) can be calculated using multiple methods, with the use of the LVOT being the most accurate: $\text{SV} = \text{Area (LVOT)} \times \text{VTI (PW LVOT)}$. The SV can also be calculated by $\text{SV} = \text{EDV} - \text{ESV}$ by tracing the LV in end diastole and end systole and using the biplane summation method to calculate volumes.

The pressure gradients across valves can be calculated using the Bernoulli equation $\Delta p = 4 V^2$. Another method to evaluate aortic stenosis is by measuring the mean pressure gradient (PG) by tracing the CW AV signal or approximating it by calculating 2/3 or 0.7 of max PG. The aortic valve area (AVA) is calculated using Newton’s 2nd law of conservation of energy (Flow in = flow out), therefore, $\text{AVA} \times \text{VTI} = \text{CSA (LVOT)} \times \text{VTI (PW at LVOT)}$. $\text{CSA of LVOT} = \pi r^2$. $R = \text{radius of the LVOT}$, $\text{CSA} = \text{Cross sectional area}$, $\text{VTI} = \text{Velocity time integral}$. Lastly, a dimensionless index defined as $\text{DVI} = \text{VTI (LVOT)} \text{ divided by } \text{VTI (AV)}$ is used to estimate severity with a value less than 0.25 signifying severe. This method avoids the error of measuring the LVOT diameter because LVOT squared- results in squaring any error obtained.

Table 1.2 shows the classification of the severity of aortic stenosis.

Mitral stenosis can be evaluated with several methods including the determination of the mean pressure gradient by tracing the CW signal across the mitral valve

Table 1.2 Recommendations for classification of AS severity [4]

	Aortic sclerosis	Mild	Moderate	Severe
Aortic jet velocity (m/s)	<2.5	2.6–2.9	3.0–4.0	>4.0
Mean PG (mm Hg)	–	<20	20–40	>40
AVA (cm ²)	–	>1.5	1.0–1.5	<1.0
Indexed AVA (cm ² /m ²)	–	>0.85	0.60–0.85	<0.6
Velocity ratio	–	>0.50	0.25–0.50	<0.25

PG pressure gradient, AVA aortic valve area

Table 1.3 Recommendations for classification of MS severity [4]

	Mild	Moderate	Severe
Valve area MVA	>1.5	1.0–1.5	<1.0
Mean PG (mm Hg)	<5	5–10	>10
PA pressure (mm Hg)	<30	30–50	>50

The above recommendations are based on the ASE guidelines, however, the AHA/ACC most recent guidelines; define an area >1.5 as progressive, <1.5 as severe, and an area <1.0 as very severe

MVA mitral valve area, *PG* pressure gradient, *PA* pulmonary artery

or by calculating the mitral valve area (MVA) by planimetry using the parasternal short axis at the mitral valve level. Additionally, MVA may also be determined by pressure half time (PHT), which is defined as the time for the LA to LV pressure gradient to fall to half its peak. A deceleration time (DT) can be calculated from the slope and $PHT = 0.29 \times DT$ and $MVA = 220/PHT$ (Table 1.3).

Quantification of valvular regurgitation can be achieved using volumetric methods or PISA. The volumetric methods, uses the principle of “What goes in, must come out”. Therefore, the regurgitant volume (RV) across the MV $RV (MR) = SV (MV) - SV (LVOT)$ and $RV (AI) = SV (LVOT) - SV (MR)$. The ERO (Effective regurgitant orifice) can be calculated by dividing the regurgitant volume (RV) by the VTI of the regurgitant valve (MR or AI). The regurgitant fraction is calculate by dividing the RV by the SV at the valve i.e., if MR then SV of MV and if AI then SV LVOT. Using the PISA method, by adjusting the aliasing velocity and creating a hemisphere of isoveLOCITY, calculations can be made to quantify regurgitation with the ERO measuring lesion severity and RV measuring volume overload from the regurgitation. $ERO = 2\pi r^2 \times Va$ (Aliasing velocity)/ V_{max} of MR, $RV = ERO \times MR$ VTI, and $RF = RV/SV$ of mitral valve. Similar calculations are made for aortic regurgitation. Tables 1.4 and 1.5 demonstrate the ASE criteria for classification of mitral and aortic regurgitation.

The RVSP and PASP can be estimated. The right atrial pressure (RAP) is estimated using the size or diameter of the IVC. If the IVC diameter is <2.1 cm, the RAP is given a value of 3 mmHg. If the IVC diameter is >2.1 cm and collapses with a sniff or <2.1 cm and is non-collapsed, then a value of 8 mmHg is given. If the IVC is >2.1 cm and is non-collapsed, then a value of 15 mmHg is given. The RVSP can be calculated using the TR velocity by applying the Bernoulli equation with $RVSP = 4 V^2 (TR) + RAP$. The pulmonary artery systolic pressure (PASP) is estimated to be equal to the RVSP unless pulmonary stenosis is present.

Finally, shunts such as ASD and VSD can be estimated using the Qp/Qs ratio. The formula to calculate the shunt is $Qp/Qs = RVOT \text{ CSA} \times RVOT \text{ VTI} / LVOT \text{ CSA} \times LVOT \text{ VTI}$.

The assessment of diastolic dysfunction is an integral part of a routine echocardiographic examination especially in heart failure patients [6]. Assessment of diastolic dysfunction usually includes measurement of mitral inflow and peak early filling (E-wave) and late diastolic filling (A-wave) velocities, E/A ratio, deceleration time (DT) of early filling velocity and the IVRT (isovolemic relaxation time). Pulmonary venous flow is performed with a PW Doppler at the right upper pulmonary vein. Measurements include peak systolic (S) velocity, peak antegrade

Table 1.4 Qualitative and quantitative parameters in grading mitral regurgitation severity [5]

	Mild	Moderate	Severe
LA and LV size	Normal	Normal or dilated	Usually dilated
Color flow jet area	Small, central jet	Variable	Large jet (>10 cm ² or >40 % of LA)
Mitral inflow-PW	A wave dominant	Variable	E wave dominant
Jet density-CW	Faint	Dense	Dense
Jet contour-CW	Parabolic	Usually parabolic	Early peaking-triangular
Pulmonary vein flow	Systolic dominance	Systolic blunting	Systolic flow reversal
VC width (cm)	<0.3	0.3–0.69	>0.7
RV (ml)	<30	30–59	>60
RF (%)	<30	30–49	>50
EROA (cm ²)	<0.2	0.2–0.39	>0.4

LA left atrium, LV left ventricle, PW pulse wave, CW continuous wave, VC vena contracta, RV regurgitant volume, RF regurgitant fraction, EROA effective regurgitant orifice area

Table 1.5 Qualitative and quantitative parameters in grading aortic regurgitation severity [5]

	Mild	Moderate	Severe
LA and LV size	Normal	Normal or dilated	Usually dilated
Jet width in LVOT-color flow	Small, central jet	Intermediate	Large jet
Jet density-CW	Faint	Dense	Dense
Jet deceleration rate-CW (PHT, ms)	>500	500–200	<200
Diastolic flow reversal in descending aorta-PW	Brief early diastolic reversal	Intermediate	Prominent holodiastolic reversal
VC width (cm)	<0.3	0.3–0.6	>0.6
Jet width/LVOT width, %	<25	25–64	>65
Jet CSA/LVOT CSA, %	<5	5–59	>60
RV (ml)	<30	30–59	>60
RF (%)	<30	30–49	>50
EROA (cm ²)	<0.1	0.1–0.29	>0.3

LA left atrium, LV left ventricle, LVOT left ventricular outflow tract, PHT pressure half time, PW pulse wave, CW continuous wave, VC vena contracta, RV regurgitant volume, RF regurgitant fraction, EROA effective regurgitant orifice area

diastolic (D) velocity, S/D ratio, and the peak A reversal in late diastole. The time difference between it and the mitral A wave duration (Ar-A) is used in assessing diastolic dysfunction. PW tissue Doppler of the septal and lateral mitral annulus is performed in the A4C. The early diastolic annular velocity e' is integral in the assessment of diastolic function. Normal diastolic function is defined by a septal $e' > 8$ and lateral $e' > 10$. If the values are < 8 and < 10 respectively, multiple parameters are evaluated (E/A, DT, Ar-A, Valsalva $\Delta E/A$) to determine the grade of diastolic dysfunction I, II, or III.

Complications

As mentioned, echocardiography is generally very safe with no complications. The key procedural complications are usually related to contrast administration or inaccuracy of data.

Clinical Vignettes

Case 1

Sixty-five-year-old male with history of hypertension, hyperlipidemia and heavy alcohol and tobacco use was admitted with 2 weeks of worsening dyspnea on exertion, orthopnea and cough with clear sputum production. His vitals include a blood pressure (BP) of 80/44, heart rate (HR) 126 and respiratory rate (RR) of 22. Physical exam is significant for distant heart sounds, few crackles and lower extremity edema. ECG is remarkable for low voltage and electrical alternans. 2D trans-thoracic images are shown in (Fig. 1.5).

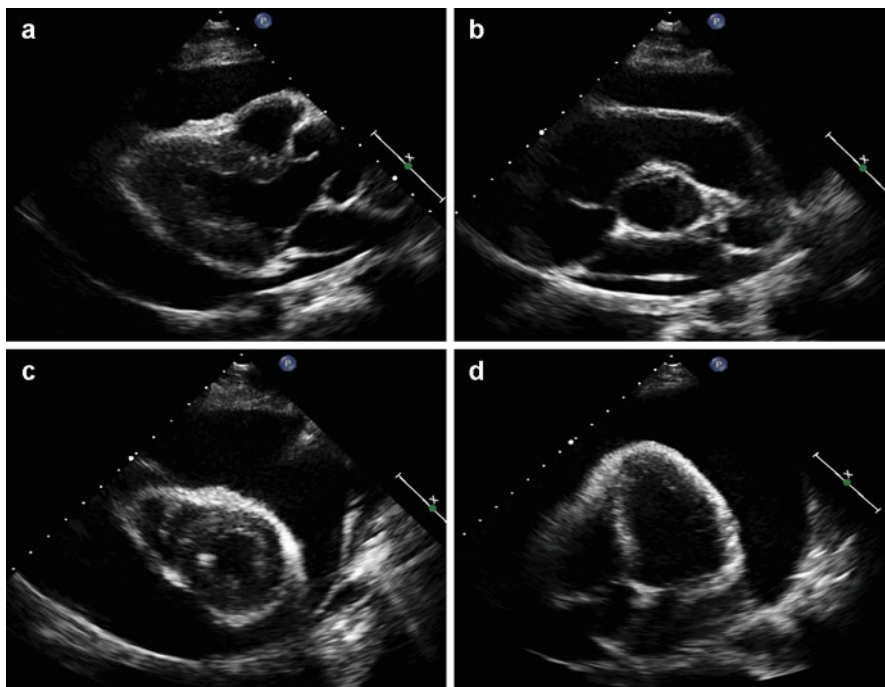


Fig. 1.5 Case #1. (a) Parasternal long axis; (b, c) Short axis, (d). Four chamber view (see text for details)

The TTE images clearly show a large circumferential pericardial effusion that is causing hemodynamic compromise and tamponade. Pericardial effusions are classified as small (less than 1 cm), moderate (1–2 cm), and large (>2 cm). In this case, the effusion is 3–4 cm with diastolic collapse or invagination of the right-sided chambers. The treatment of choice is emergent pericardiocentesis.

Case 2

Eighty-five year old male with hypertension, hyperlipidemia, prostate cancer s/p radiation and surgery presented with worsening clinical status over the last year with episodes of chest pain, dyspnea and an episode of syncope that prompt the admission. His Vitals are stable. Physical examination is notable for a grade 3/6 systolic murmur at the right upper sternal border with radiation to the carotids, S2 is diminished, lungs bibasilar crackles. ECG unremarkable. Transthoracic echocardiographic images are shown in (Fig. 1.6).

The TTE images show severe calcifications of the aortic valve with a slit like opening during systole indicating the presence of severe aortic stenosis (As shown in the upper panels). The lower panels show a continuous wave through the aortic valve, a pulse wave at the LVOT level, and a CW of the aortic regurgitation $\Delta p = 4(4 \text{ m/s})^2$ is 64 mmHg. The mean PG is calculated by tracing the CW across the aortic valve or 2/3 of the Peak PG. AVA is calculated at 0.9 cm^2 indicative of severe AS per guidelines ($\text{AVA} < 1 \text{ cm}^2$) and in symptomatic patients should be referred for aortic valve replacement.

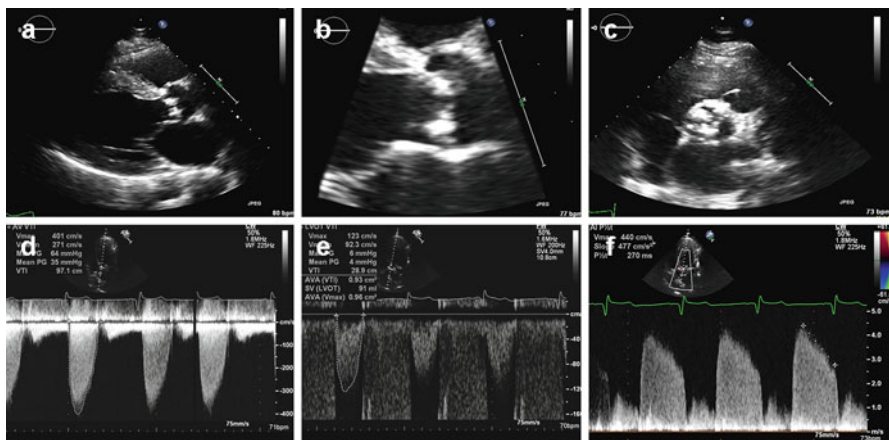


Fig. 1.6 (a) Parasternal long axis (severe calcification of the aortic valve) (b) zoom on aortic valve (c) Parasternal short axis (d) Continuous wave Doppler through the aortic valve to measure the peak and mean aortic stenosis gradients. (e) Pulse wave Doppler at the LVOT to calculate the aortic stroke volume. (f) Continuous wave Doppler through the aortic valve to quantify the pressure half time of aortic regurgitation

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