5 Echocardiographic Assessment of Ventricular Systolic Function

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CASE PRESENTATION

A 72-yr-old Caucasian male with a history of coronary artery disease and congestive heart failure was relatively well until he began experiencing orthopnea, paroxysmal noctural dyspnea, and acute deterioration 2 d before admission.

Past medical history: he suffered a myocardial infarct 6 mo earlier and underwent cardiac

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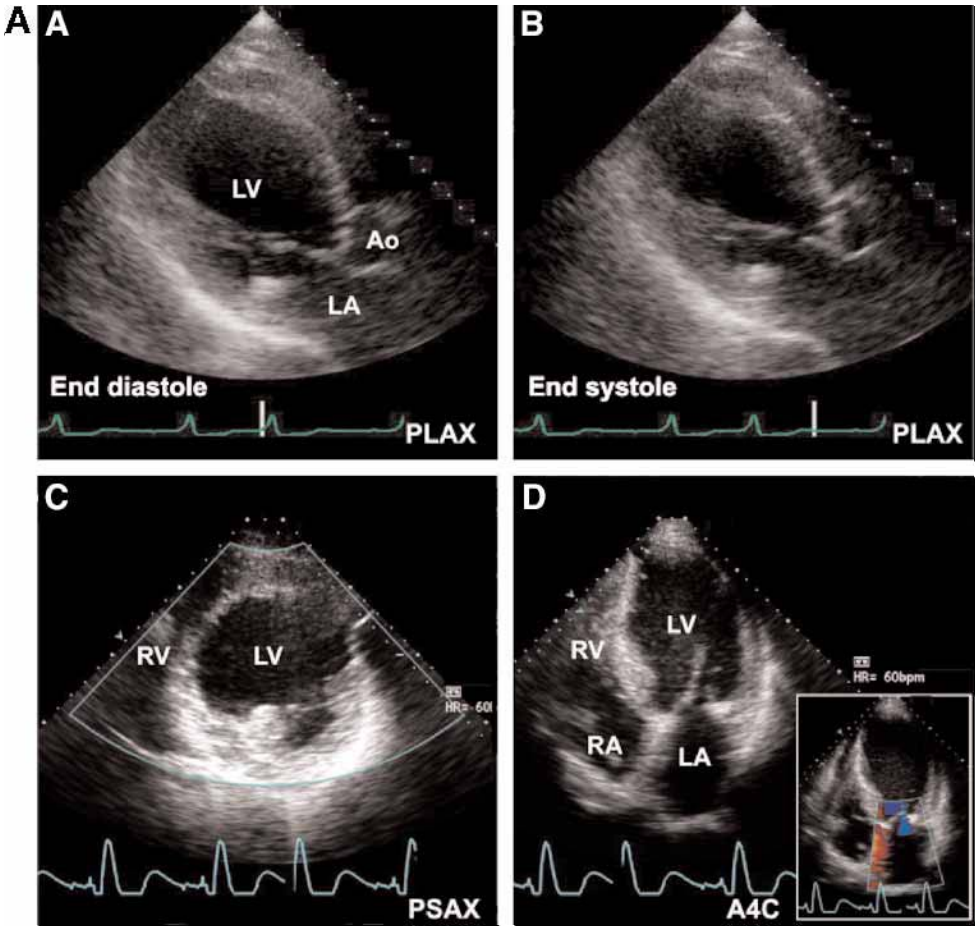

Fig. 1. *(Continued)*

catheterization and coronary angiography, which revealed significant three-vessel disease. Two years previously, he underwent placement of an implantable cardioverter defibrillator with biventricular pacing following episodes of ventricular tachycardia. His additional medical problems included hypertension, dyslipidemia, chronic obstructive pulmonary disease, and chronic renal impairment.

On physical examination, he was tachypneic (respiratory rate >40 breaths/min) with a regular pulse of 62 bpm, and blood pressure measuring 180/90 mmHg. His oxygen saturation on pulse oximetry was 93% on room air. He was afebrile and acyanotic. His jugular venous pressure was elevated and inspiratory crackles were heard halfway up both lung fields posteriorly. His chest X-ray showed signs of pulmonary edema with enlarged cardiac silhouette. Echocardiographic assessment of left ventricular function revealed a moderately dilated left ventricle (LV) with severely reduced global systolic function with regional variation. Select images from his echocardiogram are shown in Fig. 1A–D (please *see* companion DVD for corresponding video).

A major clinical application of echocardiography is the assessment of ventricular systolic function. This is a fundamental part of the standard echocardiographic examination, but is especially important in patients with heart failure and post-myocardial infarction (Fig. 2). Two-dimensional (2D) and Doppler echocardiography plays important roles in the diagnosis, management, and risk stratification of patients with systolic dysfunction.

Common causes of LV dysfunction in industrialized countries are listed in Table 1. Precipitating factors

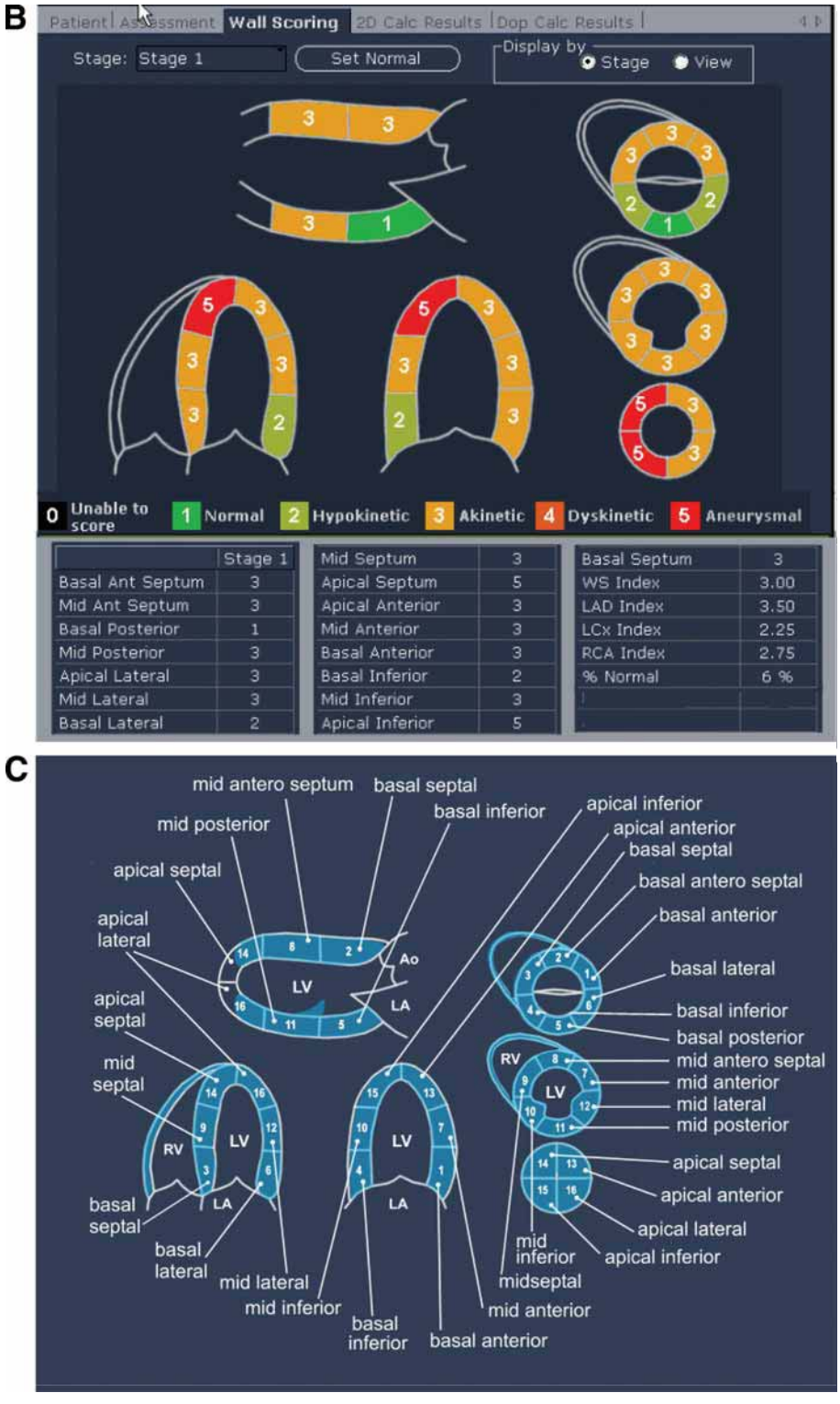

Fig. 1. *(Continued)*

(Table 2) should always be sought and the examination should be interpreted within this wider context (Table 3). This chapter discusses echocardiographic assessment of systolic function.

ECHOCARDIOGRAPHIC ASSESSMENT OF LV SIZE

Assessment of LV size is one of the most important components of quantitation of ventricular function.

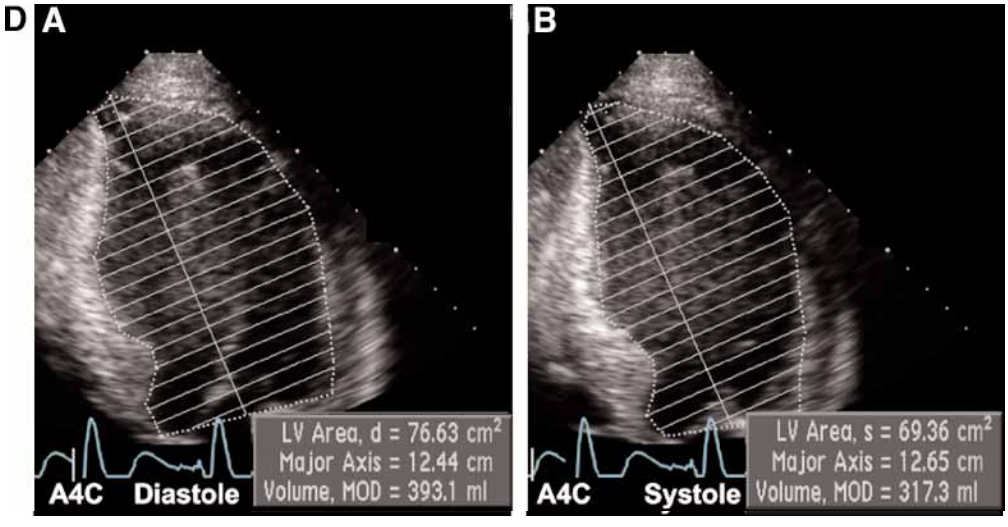

Fig. 1. **(A)** A 72-yr-old man with history of coronary artery disease and heart failure. The left ventricular ejection fraction measured 15%. The apical septal and apical inferior segments were aneurysmal. The entire anterior wall, mid- and distal lateral walls, anterior septal, midposterior segment, midseptal segments, midinferior segment, and basal septal segments were akinetic. The basal lateral and basal inferior segments were hypokinetic. The basal inferior segment contracts and thicken normally. Right ventricular size was not enlarged and right ventricular systolic function was preserved. **(B)** Computerized record of regional wall motion scores. Computerized record of left ventricular wall motion of patient in **A** with wall motion score index (WI) of 3.1 (normal = 1) (please *see* **C**). **(C)** Left ventricular segmental nomenclature. Left ventricular segmental nomenclature according to the American Heart Association/American Society of Echocardiography recommendations (*see* Fig. 10A,B). **(D)** Left ventricular volumes calculated by the biplane method of discs. The recommended method of quantifying left ventricular ejection fraction employs volumetric calculations using two orthogonal biplanes according to the method of discs, (*see* Fig. 14). (Please *see* companion DVD for corresponding video.)

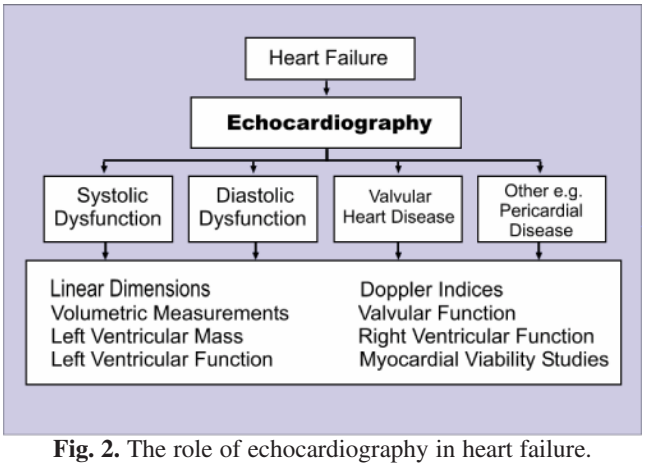

Fig. 2. The role of echocardiography in heart failure.

Qualitative and quantitative data derived from echocardiography, e.g., LV dimensions and wall thickness, can influence patient management and serve as potent predictors of outcomes (Table 4). In patients with chronic stable coronary artery disease, there is a consistent relationship between heart size and outcomes. As heart size increases, so does mortality. The same applies to patients without heart failure. Data from the Framingham Heart Study showed that even in patients without a history of heart failure or myocardial infarction, LV size (by M-mode echocardiography) was an important predictor of subsequent risk of heart failure.

Table 1
Common Underlying Causes of Ventricular Dysfunction

- Ischemic heart disease (~75% in industrialized countries)
- Cardiomyopathies
- Pressure overload states
  - Hypertensive heart disease
  - Valvular heart disease: aortic stenosis
- Volume overload states
  - Valvular heart disease: aortic incompetence, mitral regurgitation
  - Ventricular septal defect
- Rapid ventricular rate states
  - Sustained ventricular tachycardias (e.g., atrial fibrillation with rapid ventricular response)
- Congenital heart disease

LV DIMENSIONS BY M-MODE

The oldest and still widely used method for linear measurements of LV size is M-mode echocardiography. It is simple, reproducible, accurate (when properly applied), and provides excellent endocardial border definition (owing to high frame rate). The American Society of Echocardiography (ASE) recommends measurement of LV dimensions with the M-mode line perpendicular to

Table 2
Common Precipitants of Heart Failure

- Therapeutic noncompliance
- Arrhythmias
- Acute ischemia, including myocardial infarction
- Systemic or cardiac infection, e.g., myocarditis
- Physical, environmental, and emotional stress
- Pulmonary embolism
- High output states, e.g., anemia, thyrotoxicosis, pregnancy
- Drugs and toxins, including nonsteroidal anti-inflammatory drugs, ethanol

Table 3
Pathophysiological Mechanisms in Heart Failure

Structural abnormalities	Cellular/cardiac myocyte abnormalities: necrosis, fibrosis, hypertrophy, excitation–contraction coupling Left ventricular remodeling: dilatation, increased sphericity, aneursymal dilatation Coronary artery abnormalities: stenosis, endothelial inflammation
Functional abnormalities	Mitral regurgitation Myocardial "stunning" or hibernation Arrhythmias
Neuro-hormonal influences	Renin-angiotensin-aldosterone system Sympathetic nervous system Others
Comorbidities	Age, coronary artery disease, diabetes, hypertension, renal dysfunction, metabolic syndrome, anemia

Table modified from Jessup M, Brozena S. Heart failure. N Engl J Med 2003;348:2007–2018.

the long axis of the heart and immediately distal to the tips of the mitral valve leaflets in the parasternal long-axis view (Fig. 3).

Measurements are taken at end-diastole (d)—defined as the beginning of the QRS complex—but preferably using the at the widest LV cavity diameter, and at end-systole (s)—using the narrowest LV cavity diameter. The leading-edge convention of the ASE is the recommended method of measurement. The diastolic measurements obtained are the interventricular septal wall thickness, the LV internal diameter at end diastole (LVIDd) and posterior wall thickness. In systole, the LV systolic diameter (LVIDs) is measured (Fig. 3). Calculations of other indices of LV systolic function, e.g., LV ejection fraction (EF), volumes, and mass can then be performed (Table 4).

Table 4
M-Mode Parameters Used to Assess Left Ventricular Systolic Function

LVID (LVIDs < 3.7 cm; LVIDd < 5.6 cm are normal)
Left ventricular WT Percent change in WT = (WTs – WTd/WTs)
Left ventricular volume Prolate ellipse calculation: volume= $\pi/3$ $(LVIDd)^3$ Teichholz formula: volume = [7/(2.4 + LVIDd)] $(LVIDd)^3$
Ejection fraction (EDV – ESV)/EDV
Fractional shortening (FS) (%) = (LVIDd – LVIDs)/LVIDd
Mitral valve E point—septal separation (normal > 7 mm)
Left ventricular mass $(Mass_{LV}) = 0.8 \times [1.04\ (IVS + PWT + LVIDd)^3 - LVIDd^3] + 0.6$ g

LVIDs, left ventricular internal diameter at end systole; LVIDd, left ventricular internal diameter at end diastole; WT, wall thickness; WTs, wall thickness at end systole; WTd, wall thickness at end diastole; EDV, end diastolic volume; ESV, end systolic volume; IVS, septal wall thickness, PWT, posterior wall thickness.

LIMITATIONS OF M-MODE MEASUREMENTS

A common pitfall of M-mode measurements is the nonperpendicular alignment of the M-mode line in relation to the long axis of the LV. This leads to overestimation of ventricular dimensions. Two-dimensional (2D)-guided M-mode measurements can aid proper alignment thereby minimizing error.

Another challenge is to accurately identify the endocardial and epicardial borders and avoid confusion with contiguous structures, e.g., chordae, trabeculations near the posterior wall, and false-tendons. The endocardial border is distinguished from ventricular trabeculations and chordae by its appearance as a continuous line of reflection throughout the cardiac cycle. The latter structures appear intermittently. The epicardium lies just anterior to the highly echo-reflective parietal pericardium (*see* Chapter 3, Figs. 13 and 14).

A major drawback of M-mode measurements is that these are valid only when LV geometry is normal. When LV geometry is abnormal, as in aneurysmal remodeling or in the presence of regional wall motion abnormality following myocardial infarction, M-mode measurements of heart size may be misleading. An exponential relationship exists between ventricular diameters and ventricular volumes. M-mode parameters, and indeed all other parameters of LV systolic function, are dependent on ventricular loading conditions.

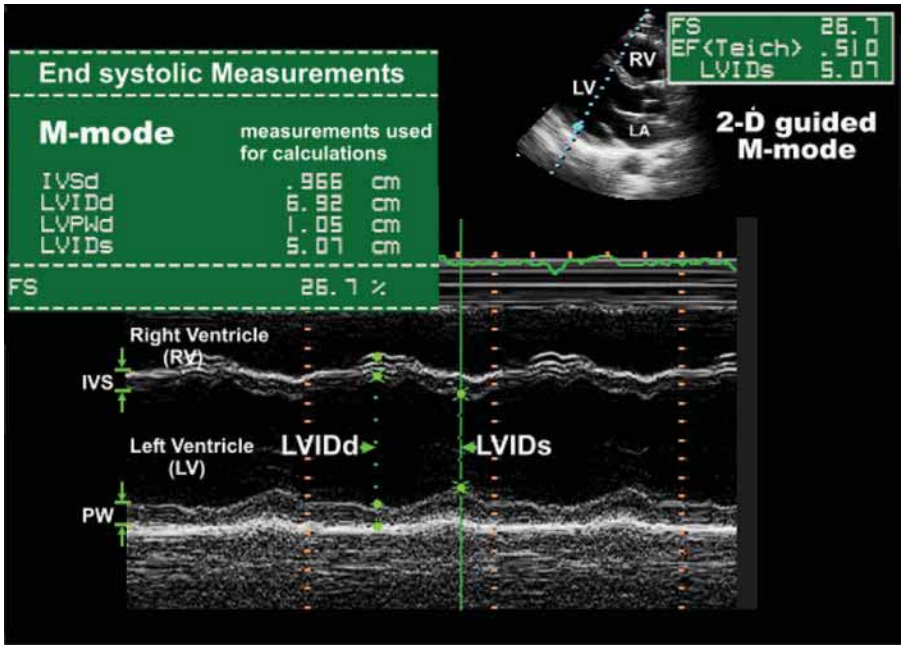

Fig. 3. Two-dimensional-guided M-mode measurements and derived indices. M-mode is simple, reproducible, and accurate when ventricular geometry is normal. It provides good endocardial resolution. The ejection fraction (EF, Teich) is an automated calculation based on the Teichholz method (*see* Table 4).

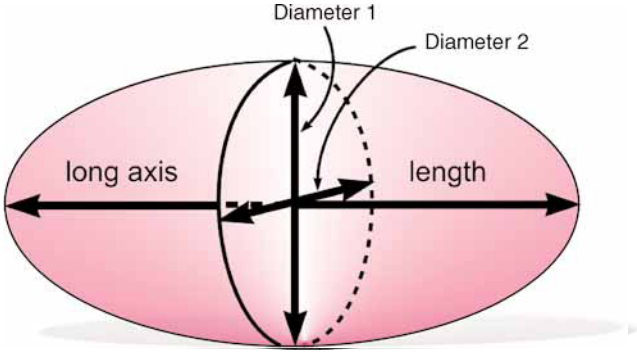

Fig. 4. Prolate ellipsoid. One geometric model used to calculate left ventricular volumes from on M-mode measurements assumes an elliposoid shape for the left ventricle. This model uses diameters (D) and length to calculate areas and volumes. A hemi-ellipsoid model is preferred in left ventricular volumetric and mass quantification using two-dimensional echocardiography (Figs. 13 and 15).

LV VOLUMES AND EF BY M-MODE

Estimates of LV volumes and EF by M-mode rely on geometrical assumptions of LV morphology. The simplest formula cubes the LVIDd. Another calculates volume using the formula for a prolate ellipsoid (Fig. 4). These measures become even more inaccurate when applied to dilated ventricles. The Teichholz method (Table 4) is commonly used to calculate ventricular volumes from M-mode measurements (from which EF can be calculated). However, this method is only recommended when ventricular geometry is relatively normal (*see* Chapter 3, Fig. 14D). Specifically, in patients with myocardial infarction involving the apex, M-mode measurements, which are obtained at the base of the heart, will underestimate ventricular size and overestimate ventricular function.

LV PARAMETERS BY 2D ECHOCARDIOGRAPHY

2D echocardiography is the primary modality used for qualitative and quantitative assessment of ventricular systolic performance (Table 5). In postmyocardial infarction and heart failure patients, 2D echo has great utility in their management and risk stratification. An inverse relationship exists between cardiovascular morbidity, mortality, and LV systolic function—specifically LVEF. EF, however, is not the only predictor of survival in patients with advanced heart failure.

LIMITATIONS OF 2D ASSESSMENT OF LV SYSTOLIC FUNCTION

2D echocardiography is not a true tomographic technique (like cardiac computed tomography or cardiac magnetic resonance imaging). Off-axis measurements,

Table 5
Two-Dimensional Parameters in Left Ventricular Function

Qualitative/semi-quantitative parameters	*Quantitative parameters*
• Global function: ventricular wall motion and thickening	Left ventricular wall dimensions: Wall thickness Internal diameters: LVIDd, LVIDs
• RWM assessment	MWFS: from linear measures of diastolic and systolic cavity sizes and wall thickenesses:
• Visual estimation of ejection fraction	Inner shell = ([LVIDd + SWTd/2 + PWTd/2]^3 – LVIDd^3 + LVIDs^3) $^{1/3}$ – LVIDs $\text{MWFS} = \frac{([\text{LVIDd} + \text{SWTd}/2 + \text{PWTd}/2] - [\text{LVIDs} + \text{inner shell}])}{(\text{LVIDd} + \text{SWTd}/2 + \text{PWTD}/2) \times 100\%}$
• Longitudinal ventricular shortening	Left ventricular quantification Biplane method of discs (modified Simpson's rule) Multiple diameter method Others based on assumptions for left ventricular geometry, e.g., cylinder-hemiellipse, biplane ellipsoid, hemisphere-cylinder, bullet, models
• Mitral annular motion	Left ventricular ejection fraction (%) = [(EDV – ESV)/EDV] × 100% Left ventricular mass (Mass_{LV}) = 0.8 × [1.04 (LVIDd + PWTd + SWTd)^3 – (LVIDd)^3] + 0.6 g Left ventricular wall stress (σ) Meridional wall stress Circumferential

Table modified from Recommendations for Chamber Quantification. American Society of Echocardiography, 2005.
RWM, regional wall motion; LVIDd, left ventricular internal diameter at end diastole; LVIDs, left ventricular internal diameter at end systole; MWFS, mid-wall fractional shortening; SWTd, septal wall thickness; PWTd, posterior wall thickness; EDV, end diastolic volume; ESV, end systolic volume; FS, fractional shortening.

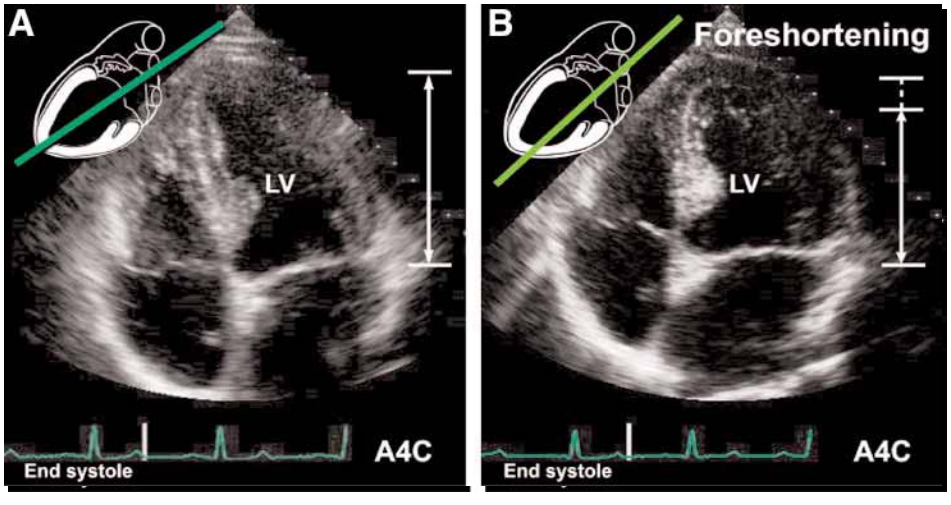

Fig. 5. Left ventricular foreshortening. Foreshortening (shown right) occurs when the imaging plane does not transect the center of left ventricular apex (left). It is a common source of error in left ventricular quantification in two-dimensional echocardiography. (Please *see* companion DVD for corresponding video.)

e.g., foreshortening, easily occur (Fig. 5; please *see* companion DVD for corresponding video). Distortions of LV geometry seen in patients with ischemic heart disease pose challenges to 2D assessment (Figs. 1A,B and 6).

QUALITATIVE AND SEMI-QUANTITATIVE MEASURES OF LV SYSTOLIC FUNCTION

Making linear measurements of cardiac chamber dimensions by 2D echo follows the principles outlined

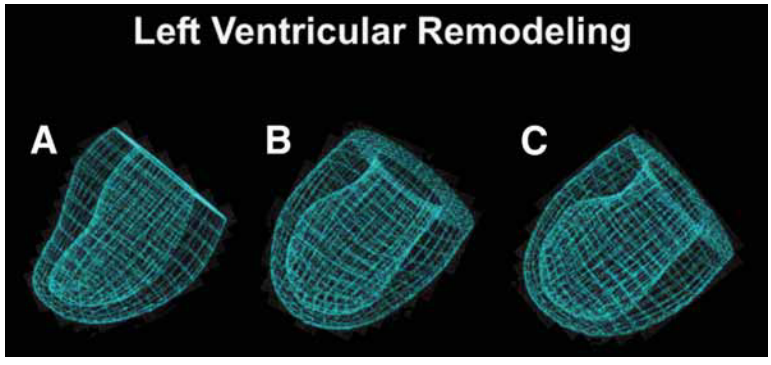

Fig. 6. Three-dimensional (3D) images showing gross distortion of left ventricular geometry post-myocardial infarction. 3D representation of progressive remodeling of the left ventricle in a patient with a large anterior-apical myocardial infarction. Note the progressive distortion of the ventricular geometry with time **(A–C)**.

using M-mode described earlier (*see also* Chapter 3, Fig. 16). "Eyeball" estimates of LVEF are routinely used in clinical practice, but interobserver variability is high, and should be "calibrated" by quantitative measurements.

Accurate assessment of ventricular wall movements during the cardiac cycle is dependent on image quality. Optimal image acquisition is influenced by patient characteristics, operator skill, and instrument settings. Proper patient positioning helps to optimize imaging of parasternal and apical views (Chapter 3, Fig. 9). Images are best acquired at end-expiration or during quiet respiration.

Failure to accurately visualize the endocardial border introduces uncertainty into 2D measurements. To minimize this, techniques to improve endocardial border definition, e.g., harmonic imaging, B-color imaging, LV opacification with contrast agents, and Doppler based techniques are often employed (Figs. 7 A,B and 8; please *see* companion DVD for corresponding video).

QUALITATIVE GRADES OF LV SYSTOLIC FUNCTION

Normal ventricular walls thicken during systole—a manifestation of myocardial fiber shortening—as both ventricles contract. Ventricular systolic contraction is accompanied by a reduction in ventricular cavity size and can be qualitatively assessed as normal, reduced, or hyperdynamic (Fig. 9). Normally, 60–70% of ventricular end-diastolic volume is ejected during each cardiac cycle.

Reduction of LV systolic function can be estimated to the nearest 5 or 10% by an experienced observer. EF of 55% or more is generally considered normal. EF between 40 and 55% is considered mildly reduced; EF between 30 and 40% is considered moderately reduced; EF less than 30% is considered severely reduced. Global reduction of systolic function is frequently accompanied by regional variation.

When the EF exceeds 70%, it is considered to be "hyperdynamic." EFs exceeding 75% manifest as near obliteration of ventricular cavity when viewed from the parasternal or apical windows. This can be seen in hypovolemia or in patients with hypertrophic cardiomyopathy.

Estimations of EF by experienced sonographers correlates well with other quantitative measures of EF. It is, therefore, a practical first step in qualified hands.

GRADING REGIONAL WALL MOTION

Regional LV wall motion assessment generally employs the 16-segment model recommended by the ASE (1989), or the more recent 17-segment model (which adds an additional region for the apex). Ventricular segment scores are assigned based on two qualitative measures of ventricular wall behavior during systole: (1) wall movement (contraction) and (2) wall thickening. Graded scores of contractility of the individual segments range from a normal score of 1 to the worst score of 5 (Figs. 1B and 10A; please *see* companion DVD for corresponding video for Fig. 8). The myocardium of a dysfunctional segment thickens less, or becomes thinner, during systole.

A segment that shows noticeable reduction in contractility is *hypokinetic* and assigned a score of 2. A segment that barely moves or thickens during systole is *akinetic* (score = 3). *Dyskinetic* myocardium moves paradoxically during systole (score = 4). *Aneurysmal* myocardium remains deformed during diastole. The integrated wall motion score is the sum of the scores divided by the number of scored segments. A wall score index of 1 indicates normality. Larger scores reflect more severe degrees of systolic dysfunction (Fig. 1B; please *see* companion DVD for corresponding video).

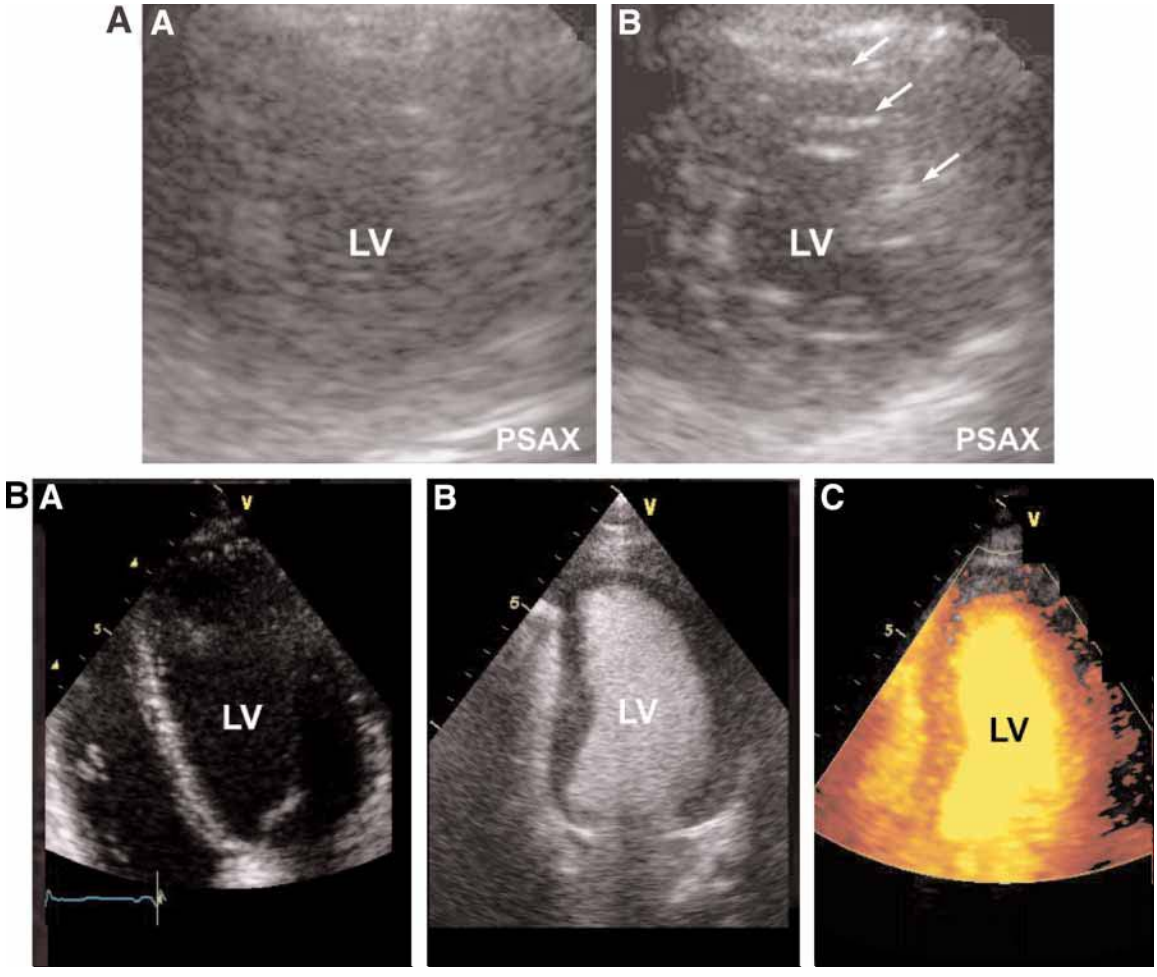

Fig. 7. (**A**) Tissue harmonic imaging. Even with suboptimal imaging (**left panel**, parasternal short axis view of the left ventricle) tissue harmonic imaging markedly improves endocardial definition. Note reverberation artefacts (arrows) arising from ribs. (**B**) Left ventricular opacification/endocardial border definition. Contrasts methods assist in delineating the endocardial border. Left ventricular opacification using microspheres (e.g., Optison® or Definity®) are popular (compare **left** and **middle panels**). Another myocardial contrast imaging technique is shown in **right panel**. (Please *see* companion DVD for corresponding video.)

In an effort to standardize nomenclature of myocardial segments across other cardiac imaging specialties, the American Heart Association (2002) issued a unifying 17-segment model (Fig. 10B). Using this model, LV segments 1–6 are at the base (mitral valve level), segments 7–12 are in the middle (papillary muscle level), segments 13–16 occupy the apical region, and segment 17 represents the very tip of the apex. The latter does not encroach into the ventricular cavity. The segmental numerical model can be matched to the more practical anatomical descriptive terminology as shown in Fig.10B.

The segmental numerical model nomenclature also corresponds well with coronary artery distribution (*see* Chapter 3, Fig. 58; Chapter 7, Figs. 3–6). From this, various indices of coronary artery territory involvement, e.g., left anterior descending artery, may be derived.

LIMITATIONS OF REGIONAL WALL MOTION ASSESSMENT

Regional wall motion assessment is heavily influenced by image quality. Endocardial border definition deteriorates when still frames are acquired from digital video files. The audio/video interleaved (AVI) and the digital imaging and communications in medicine (DICOM) video format are the two most popular. Digital videos consist of multiple still frames in rapid succession, usually in the order of 30–60 frames per second. When the video is stopped, and a single still frame is selected, image quality characteristically degrades, including endocardial border definition.

Angulation of the transducer during acquisition of short-axis views may misrepresent true segmental

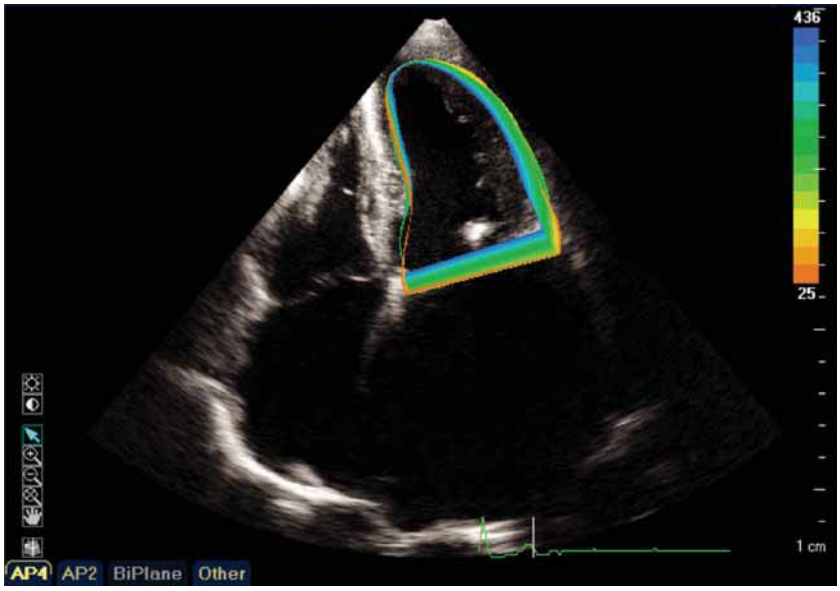

Fig. 8. Dynamic endocardial border analysis. Dynamic left ventricular endocardial border analysis is shown in this patient with mitral stenosis (and a severely dilated left atrium). (Please *see* companion DVD for corresponding video.)

anatomy and is avoided by using the recommended technique (Fig. 11). Translational and rotational movements of the heart during the cardiac cycle cannot be avoided, but can be minimized by acquiring images during end-expiration. Care should be taken to avoid triggering the Valsalva maneuver.

Restricted septal movement can be mistaken for septal hypokinesis or akinesis. Apparent hypokinesis of the septum can be seen following any surgery that breaches the pericardium. Closer observation of the septum will often show normal systolic thickening in the absence of true ischemic injury.

Paradoxical septal motion in the presence of otherwise normal septal myocardium is seen in right ventricle (RV) pressure and volume overload states (Chapters 18 and 21), pericardial effusion and constrictive pericarditis (Chapter 10), or with certain arrhythmias, e.g., left bundle branch block.

QUANTITATIVE MEASURES OF LV SYSTOLIC FUNCTION

Comparisons of LV end-diastolic and end-systolic dimensions form the basis of quantitative estimates of LV function, e.g., fractional shortening and EF (Fig. 12, Table 6). Fractional shortening—the percentage change in the LV minor axis in a symmetrically contracting ventricle—can be derived using the formula:

$$\text{Fractional Shortening (FS)}(\%) = (\text{LVIDd} - \text{LVIDs})/\text{LVIDd} \times 100\%$$

$$\text{FS} = 25\% - 45\% \text{ (normal range)}$$

Volumetric estimates of LV volumes by 2D echocardiography are based on three geometric methods that combine measurements of LV dimensions and area to calculate volume (Table 6; Fig. 13A). These are:

1. Prolate ellipsoid method.
2. Hemi-ellipsoid (bullet) method.
3. Biplane method of discs (modified Simpson's rule).

The prolate ellipsoid method assumes a prolate ellipsoid systolic and diastolic LV geometry). Area-length or length-diameter methods can be used. The single-plane and biplane area-length methods are shown in Fig. 13B,C.

The combined geometric model—of a hemisphere and an ellipsoid (hemi-ellipsoid)—provides a better estimate of LV volume (Fig. 13D), but the biplane method of discs (modified Simpson's rule) is recommended by the ASE and the European Association of Echocardiography. This method does not assume a predetermined geometry of the LV, but instead defines the LV geometry following manual tracing of the acquired LV cavity borders. The LV volume is then quantified by assuming the LV cavity is a stack of elliptical discs whose volumes are quantified and summated (Fig. 14).

Two orthogonal views—apical four-chamber and apical two-chamber—and manual tracing of endocardial borders manually traced at end systole and end-diastole are needed. Automated software divides the LV into a stack of discs oriented perpendicular to the long axis of the ventricle, and summates their individual volumes (Fig. 14).

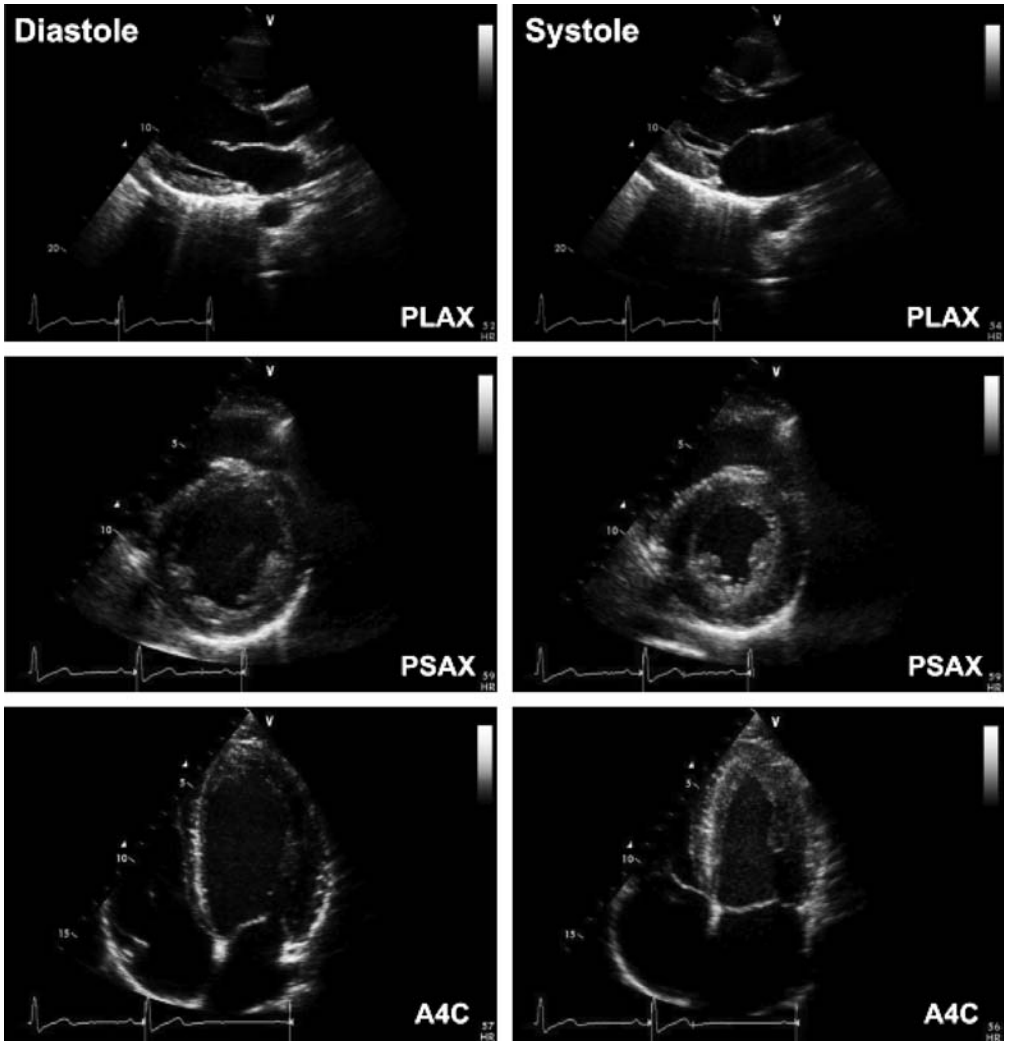

Fig. 9. Normal systolic function. Knowledge of normal left ventricle systolic function is essential for interpreting abnormalities.

From the end-diastolic frame, the end-diastolic volume is calculated. From the end-systolic frame, the end-systolic volume is calculated. The stroke volume (SV) is then:

$$SV = EDV - ESV \text{ (mL)}$$

The EF is therefore:

$$EF = \frac{EDV - ESV}{EDV} \times 100\%$$

Cardiac output (CO) is product of the EF and the heart rate (HR):

$$CO = EF \times HR$$

The advantage of the modified Simpson's method over other volumetric methods listed in Table 5 is that it makes no assumptions about ventricular geometry. Nevertheless, considerable sonographer experience is required as images must be optimized and endocardial borders accurately identified and traced according to convention. Poor endocardial border definition, foreshortened views, and improper technique can compromise this technique.

LVEF shows high correlation, but less striking agreement, with radionuclide ventriculography and contrast cine-angiography. Even quantitative measures of LVEF can be inaccurate owing to limitations inherent in quantification formulae, as well as those of cardiac ultrasonography itself. Interobserver-variability remains a vexing issue in 2D echocardiography.

LIMITATIONS OF VOLUMETRIC MEASURES (EF) OF LV SYSTOLIC FUNCTION

Overwhelming evidence from landmark clinical trials in heart failure and postmyocardial patients have demonstrated the proven value of LVEF assessment on patient management and prognosis. Nevertheless,

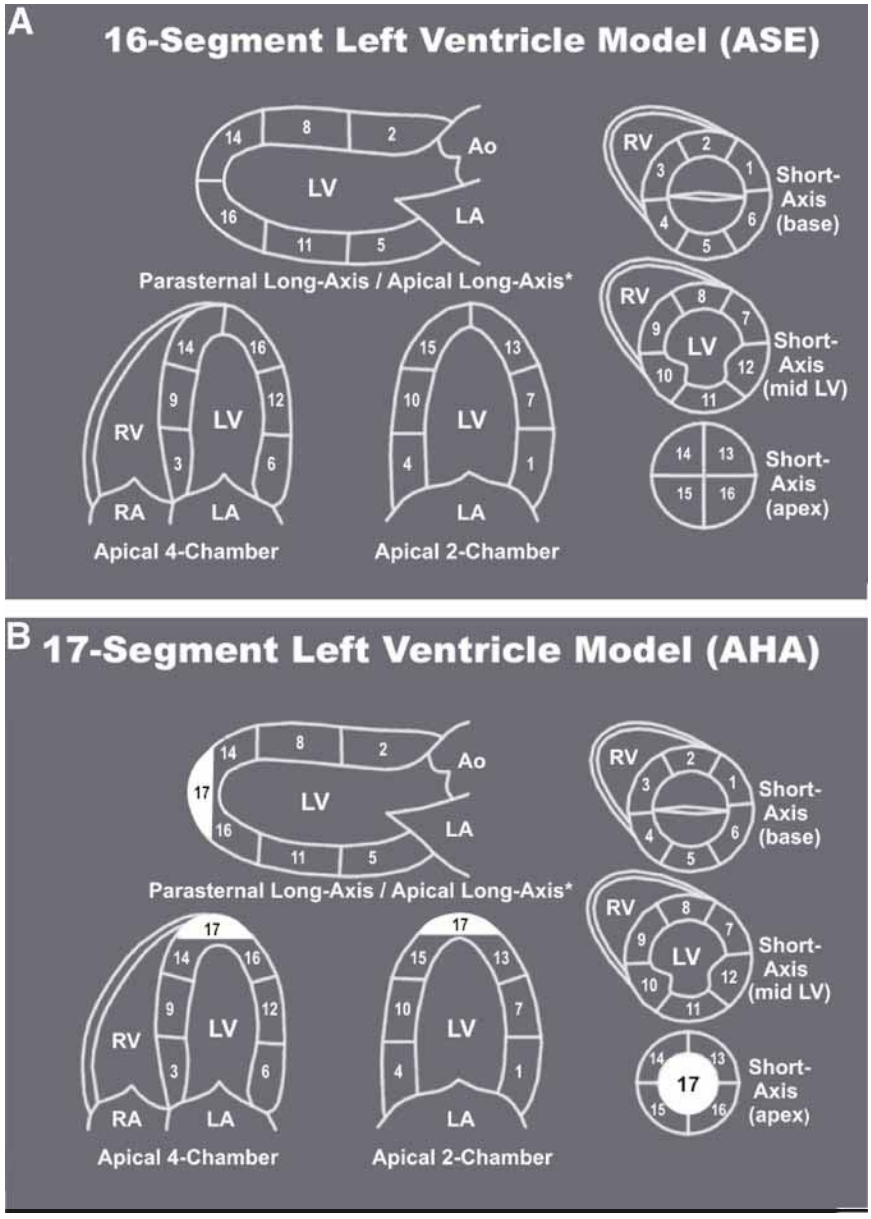

Fig. 10. The American Society of Echocardiography (ASE) issued a 16-segment left ventricle model for wall motion assessment. The American Heart Association's (AHA) 17-segment model has an additional apical segment "cap" added to harmonize left ventricular segment nomenclature with nuclear cardiology and cardiac magnetic resonance imaging. **(A)** A 16-segment model of left ventricular segments (ASE). **(B)** A 17-segment model of left ventricular segments (AHA).

LVEF is not the sole or a complete measure of LV function. Diastolic and other measures of ventricular function are needed because nearly 40% of patients with clinical heart failure have persevered systolic function (normal LVEF). Furthermore, systolic function can be abnormal even in the presence of normal LVEF.

Quantification of LV volumes by 2D echocardiography faces significant technical and clinical limitations. As 2D echocardiography is not a true tomographic technique, LV foreshortening and off-axis views remain a challenge. The LVEF may be normal in patients with acute myocardial infarction, as hypokinesis or akinesis in the affected myocardial territory may be compensated by hyperkinesis in the unaffected segments. The same LVEF in a patient with mitral regurgitation has a different clinical and prognostic implication than in a patient with aortic stenosis.

Therefore, the tendency by many clinicians to request an echocardiographic study for "LVEF only" or

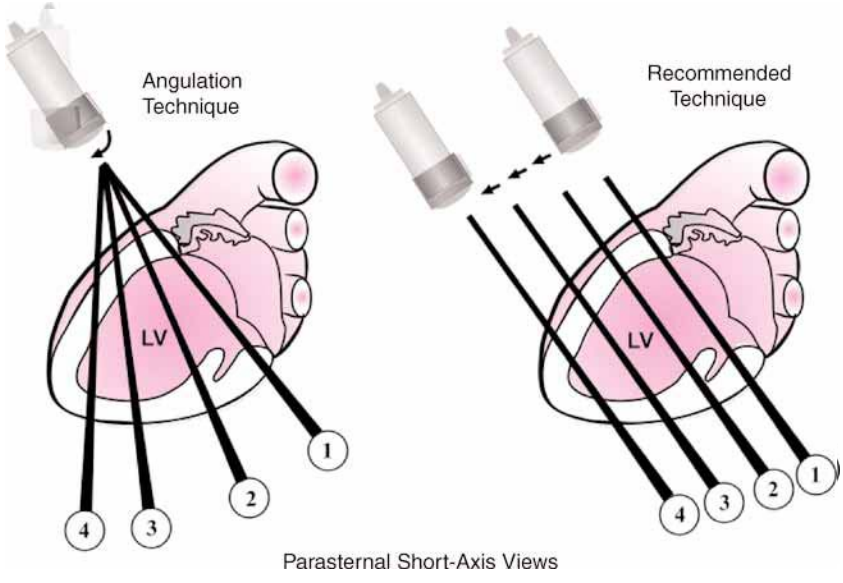

Fig. 11. Angulated vs orthogonal parasternal short-axis imaging of the left ventricle (LV). The angulation technique (left) may acquire short-axis views of the LV segments tangentially, thereby influencing the accuracy of regional wall motion assessment. Images are best acquired at planes orthogonal to the long axis of the left ventricle as shown (right).

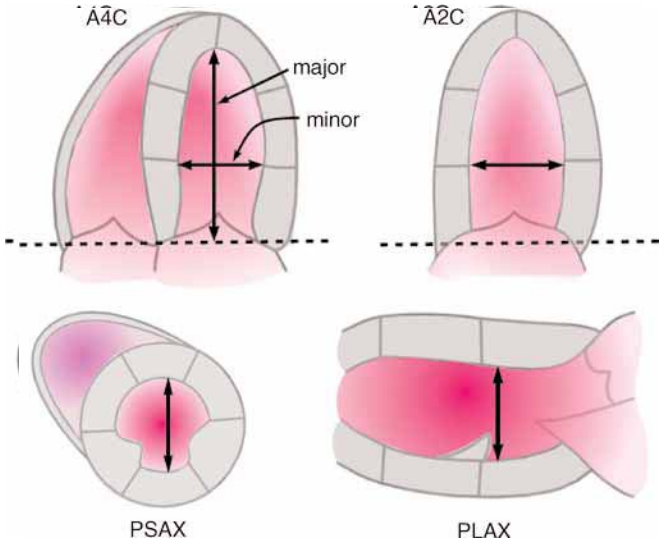

Fig. 12. Left ventricular cavity dimensions.

to equate the LVEF as "the sole measure" of LV function should tempered with other parameters of function.

LV MASS

A clear relationship exists between LV mass and outcomes in cardiovascular disease, especially in hypertension. LV mass is calculated using the formula listed in Table 5 and is based on an area-length method or that for a cylinder hemi-ellipsoid model (Fig. 13). Such measurements of LV mass, whether by M-mode or 2D, essentially subtract ventricular cavity volume from the total ventricular volume to obtain the "shell" or myocardial volume (Fig. 15). This value

Table 6
Cardiac Measurements by Two-Dimensional Echocardiography

Window	*Normal range (cm) (mean – SD)*	*Index adjusted for BSA (cm/m²)*
PLAX	PLAX	PLAX
LVIDd	3.5 – 6.0	2.3 – 3.1
LVIDs	2.1 – 4.0	1.4 – 2.1
Fractional shortening	25 – 46	—
PSAX (papillary muscle level)	PSAX (papillary muscle level)	PSAX (papillary muscle level)
LVIDd	3.5 – 5.8	2.2 – 3.1
LVIDs	2.2 – 4.0	1.4 – 2.2
Fractional shortening	25 – 43	
A4C	A4C	A4C
LVIDd major	6.9 – 10.3	4.1 – 5.7
LVIDd minor	3.3 – 6.1	2.2 – 3.1
LVIDs minor	1.9 – 3.7	1.3 – 2.0
Fractional shortening	27 – 50	

BSA, body suface area; PLAX, parasternal long-axis ; LVIDd, left ventricular internal diameter at end diastole; LVIDs, left ventricular internal diameter at end systole; PSAX, parasternal short-axis; A4C, apical four chamber.

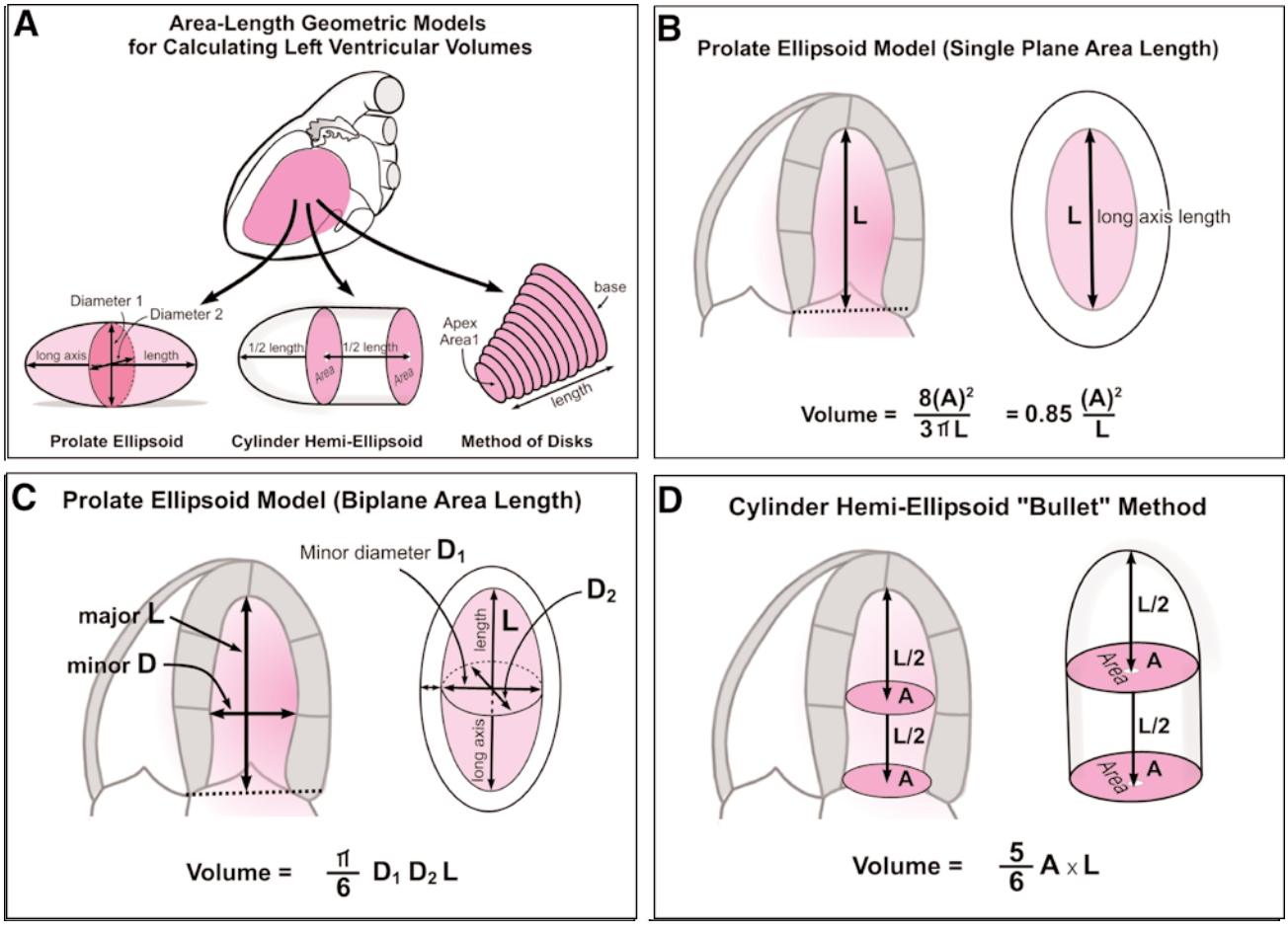

Fig. 13. Geometric models to estimate left ventricle (LV) volumes by two-dimensional echocardiography use short-axis area multiplied by long-axis length. Comparison of volumes at end-systole and end-diastole can be a measure of LV systolic function.

multiplied by the density of the myocardium gives the LV mass.

$$\text{Left ventricular mass } (\text{Mass}_{LV}) = 0.8 \times [1.04\ (\text{IVS} + \text{PWT} + \text{LVIDd})^3 - \text{LVIDd}^3] + 0.6 \text{ g}$$

Accurate measures are crucial, as errors will be cubed.

3D Echocardiography

3D echocardiography will likely replace current echocardiographic methods of calculating ventricular mass and volumes in the near future (Figs. 16A–D; please *see* companion DVD for corresponding video). The limitations and assumptions of 2D and M-mode echocardiography are overcome by both real-time and off-line reconstructive 3D echocardiography. Modern 3D equipment uses planar array transducer technology to obtain a pyramidal "volume" of data. This makes 3D echocardiography less dependent on the sonographer imaging the correct plane.

ASSESSMENT OF MYOCARDIAL VIABILITY

Dobutamine echocardiography can provide additional information on the LV contractile reserve. This has value in predicting recovery of function following coronary revascularization procedures. Assessment of myocardial viability is also important in heart failure patients.

The important clinical question that frequently confronts the cardiology team is whether coronary revascularization procedures will benefit a particular patient with LV dysfunction. To answer this, we need to help predict the probability of improvement following the proposed revascularization procedure. Nuclear techniques assess myocardial perfusion, but not contractile reserve. A biphasic response on dobutamine stress echocardiography, however, can be a good predictor of improvement in patients scheduled to undergo coronary revascularization procedures (Fig. 17; *see* companion DVD for corresponding video; *see also* Chapter 8).

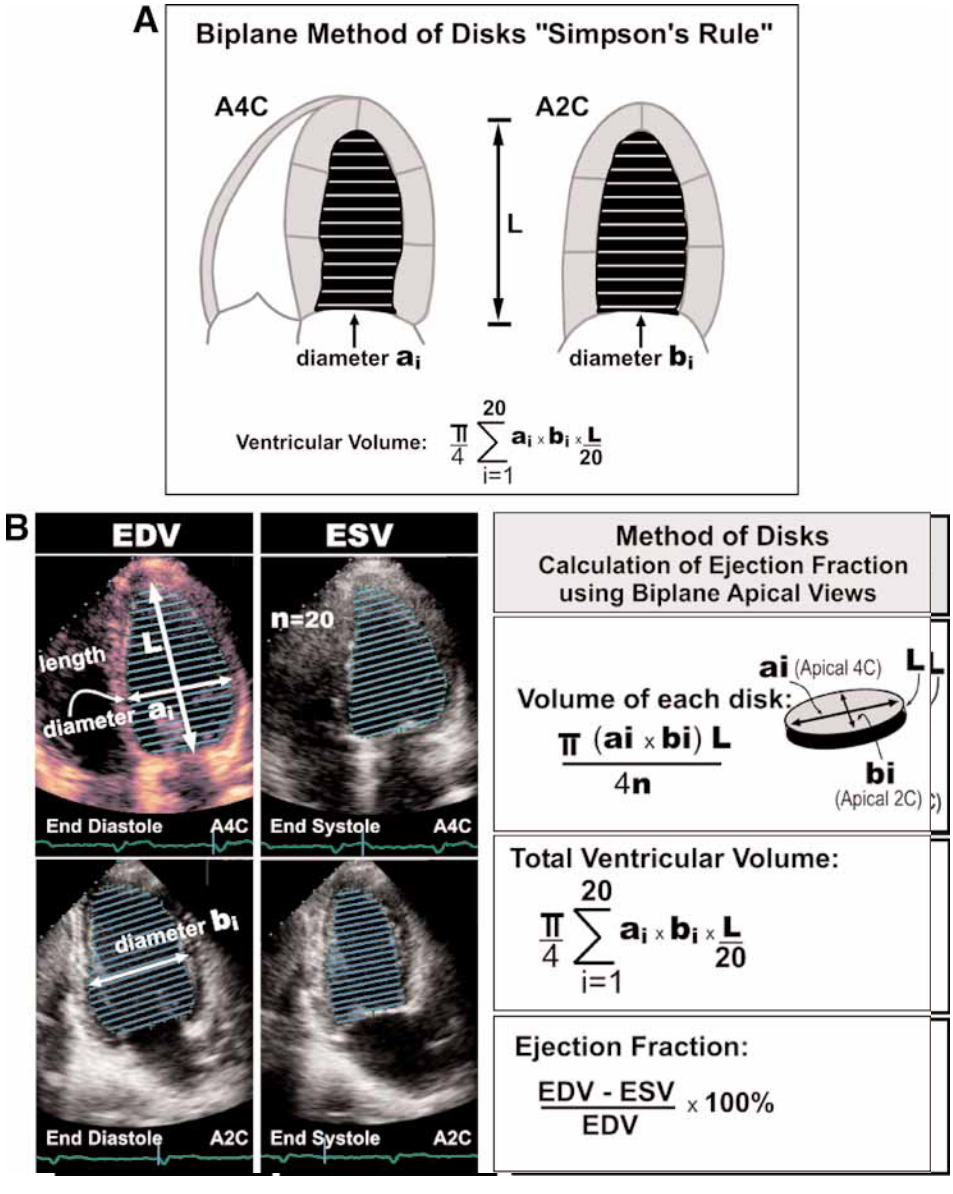

Fig. 14. (A,B) Modified Simpson's method. The American Society of Echocardiography recommends the modified Simpson's method (biplane method of discs) for calculating left ventricular volumes and ejection fraction. Manual tracing of ventricular endocardium at end-systole and end-diastole from two orthogonal planes, and summation of the volumes of discs derived, serve as the basis of this calculation.

DOPPLER ASSESSMENT OF VENTRICULAR SYSTOLIC FUNCTION

Doppler assessment provides complementary and alternative indices of ventricular systolic function (Table 7). Traditional Doppler indices are used to calculate SV and CO. SV is calculated from the equation: *volume = area × velocity time integral*; where *area* is the cross-sectional area of ventricular outflow or inflow tract of interest; *velocity time integral* corresponds to the velocity time integral across the same. CO is then calculated according to the equation:

$$CO = SV \times HR$$

Cardiac index calculated by dividing CO by body surface area.

Doppler indices have the advantage in being independent of geometric assumptions used in M-mode and 2D-based calculation of volumes. The most accurate and reproducible Doppler method for calculating SVs uses the left LV outflow tract (LVOT) diameter and the velocity

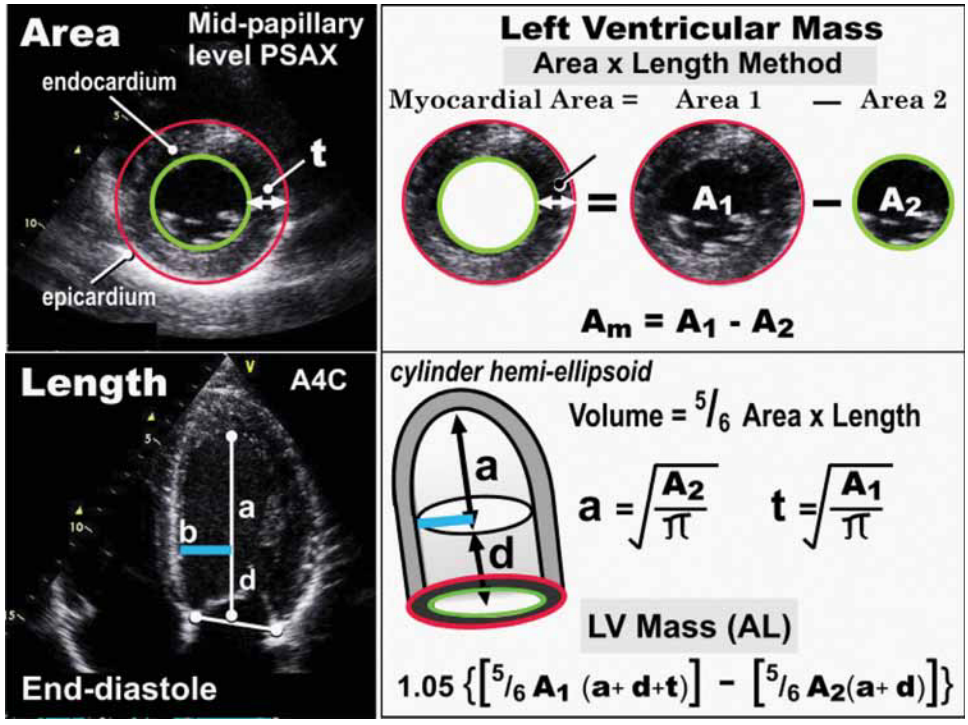

Fig. 15. Left ventricular mass. The area-length method for using a cylinder hemi-ellipsoid of the left ventricle (LV) is the recommended equation for measuring LV mass. It is a simple formula with easily obtainable measurements. End-diastolic measurements using parasternal short-axis (PSAX) and apical four-chamber at the mid- or high-papillary muscle levels are made and inserted into the equation as shown. The sum of a + d is the end-diastolic LV cavity length; b = minor axis radius; t = wall thickness; A_1= total planimetered PSAX area at the mid- or high-papillary muscle level; A_2 = LV cavity planimetered PSAX area.

time integral across the LVOT by pulsed Doppler examination (Fig.18A; *see also* "Continuity Equation" in Chapter 11) LVOT geometry most closely approximates a circle compared with the ellipsoid mitral annulus (Fig. 18B), and is logistically easier to measure than the pulmonary artery diameter (Figs. 18C). Tricuspid annular geometry is complex, and is almost never used to calculate SVs.

Continuous-wave Doppler of the mitral regurgitant jet can reveal clues about LV performance by assessing changes in LV pressure over time (dP/dT). The pressure is measured at two points (at ~1 m/s and 3 m/s after the onset of the mitral regurgitation) and the Bernoulli equation ($dP = 4v^2$) applied. Normal dP/dT is greater than 1200 mmHg/s.

OTHER DOPPLER MEASURES OF VENTRICULAR SYSTOLIC FUNCTION

Tissue Doppler imaging is a useful tool in ventricular diastolic function assessment, but also shows promise in assessing systolic function (Fig. 19). Doppler interrogation of the mitral annulus can provide a measurable index of annular movement and velocity, and the information derived can be extrapolated to assess ventricular function. A good relationship exists between tissue Doppler assessment of myocardial contraction velocity and LVEF.

Tissue velocity imaging employs color codes to reflect ventricular longitudinal shortening using the scheme: red—for movement toward the transducer, and blue—movement away from transducer (Fig. 20; please *see* companion DVD for corresponding video). It is based on the rationale that most of the cardiac muscle fibers are oriented longitudinally. A direct relationship exists between pulsed-wave tissue velocity imaging and ventricular systolic function.

EF is not a load-independent measure of contractility. Load is important when considering contractility and this is not normally accounted for in traditional measures. Newer less load-dependent methods, e.g., Doppler strain imaging, are being investigated. Strain—a dimensionless quantity, measures deformation produced by the application of stress. It represents the percentage change in myocardial fiber length from its original or unstressed dimension (Fig. 21A–C). Comparisons of Doppler velocities at interrogation points along the myocardium are used to measure LV strain.

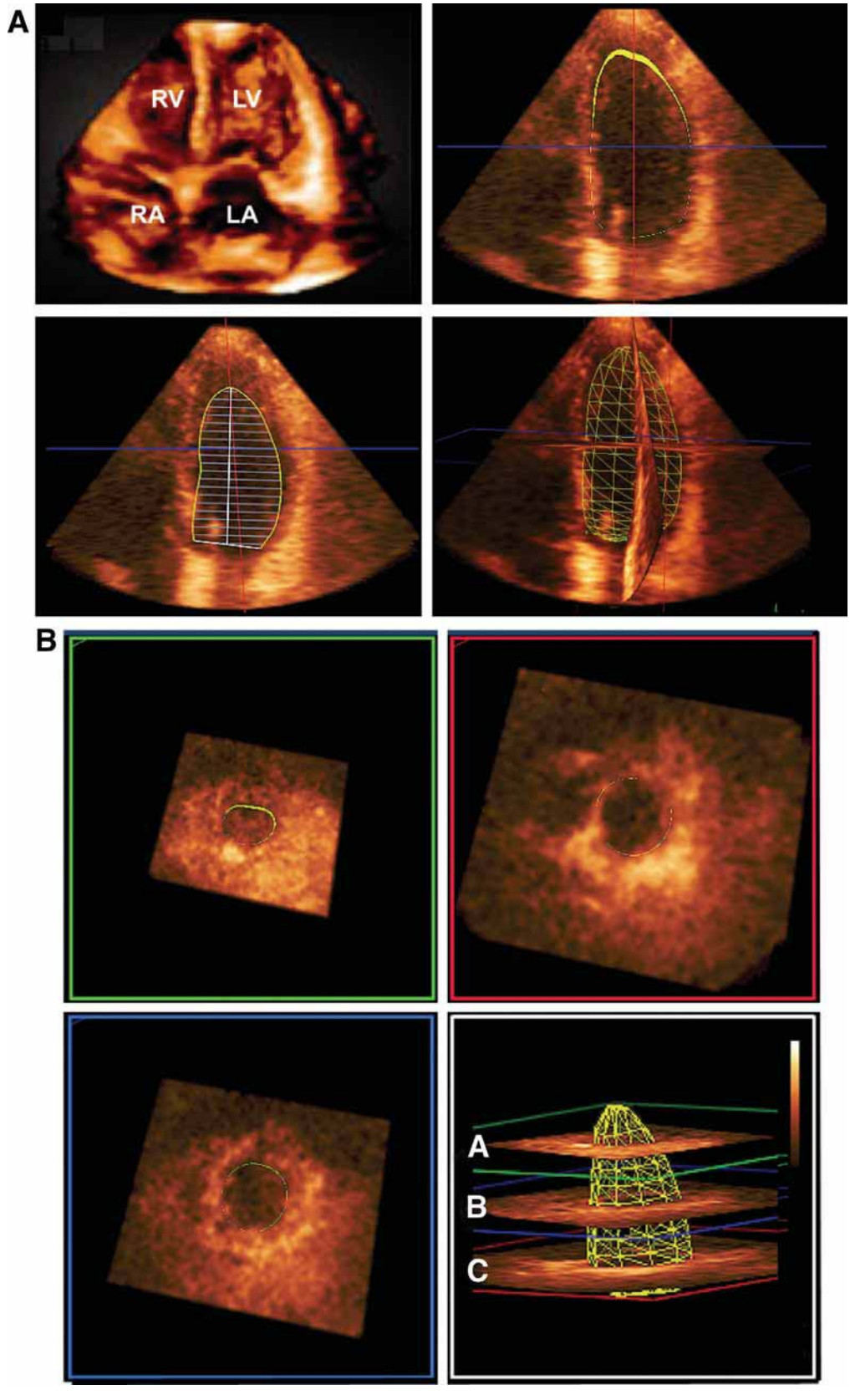

Fig. 16. *(Continued)*

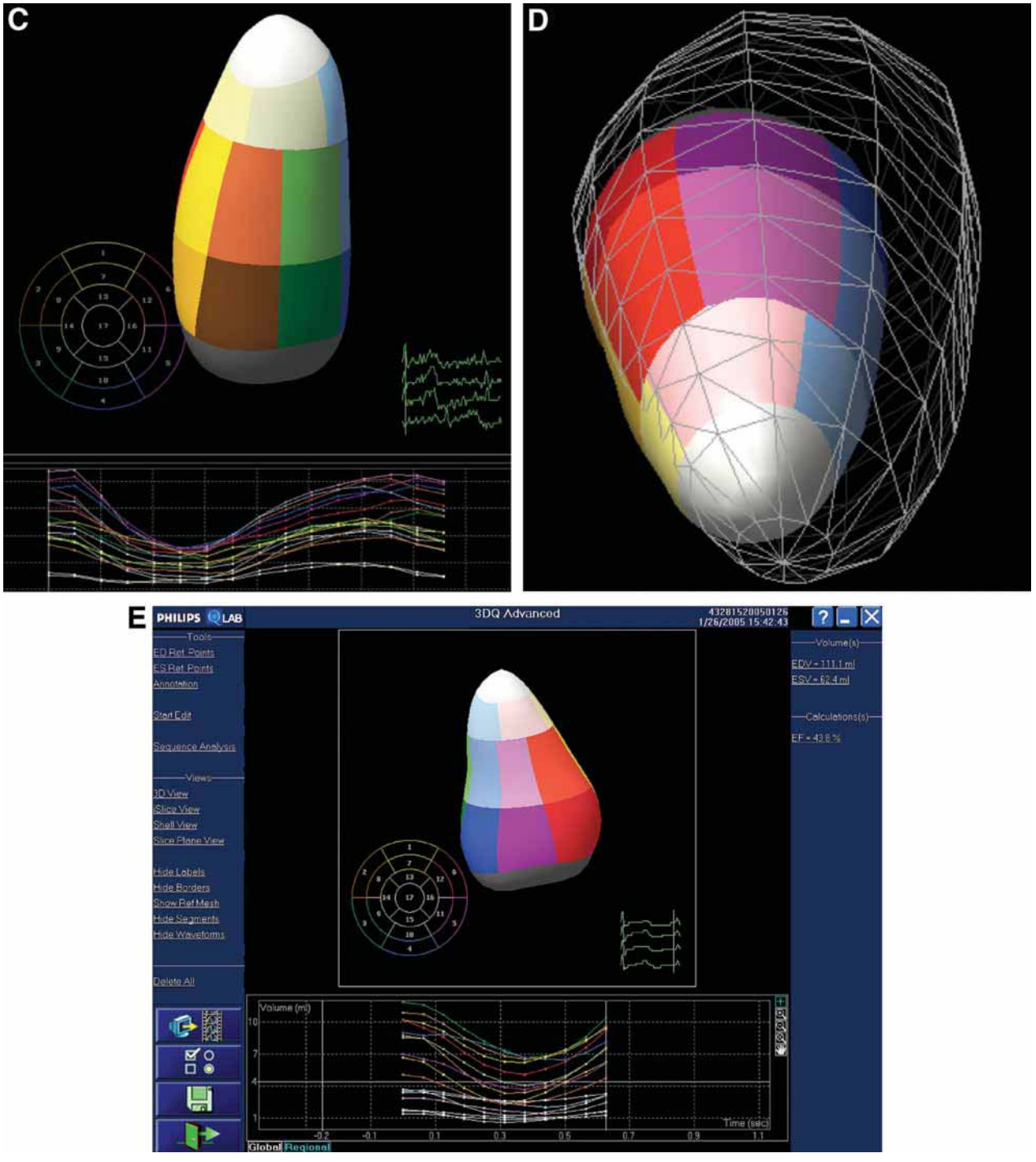

Fig. 16. Left ventricle (LV) quantification by three-dimensional (3D) echo. **(A)** Apical full-volume cropped 3D image. **(B)** Semi-automatic border detection with multiplanar reconstruction (MPR) in 3D echocardiography. **(C)** A 17-segment 3D volumetric data for left ventricular segmental analysis. 3D echocardiography overcomes several limitations of 2D echocardiography in quantification of systolic function including: endocardial border definition, foreshortening, off-axis views, and translational motion. It is slated to supercede 2D echocardiography in the assessment of LV function, mass, and volumetric assessments. 3D echo is especially valuable in right ventricular assessment and quantification, with utility comparable to that of cardiac magnetic resonance imaging. **(D)** LV systolic frame with diastolic reference mesh. **(E)** Systolic frame in patient with cardiomyopathy and asynchrony. (Please *see* companion DVD for corresponding video.)

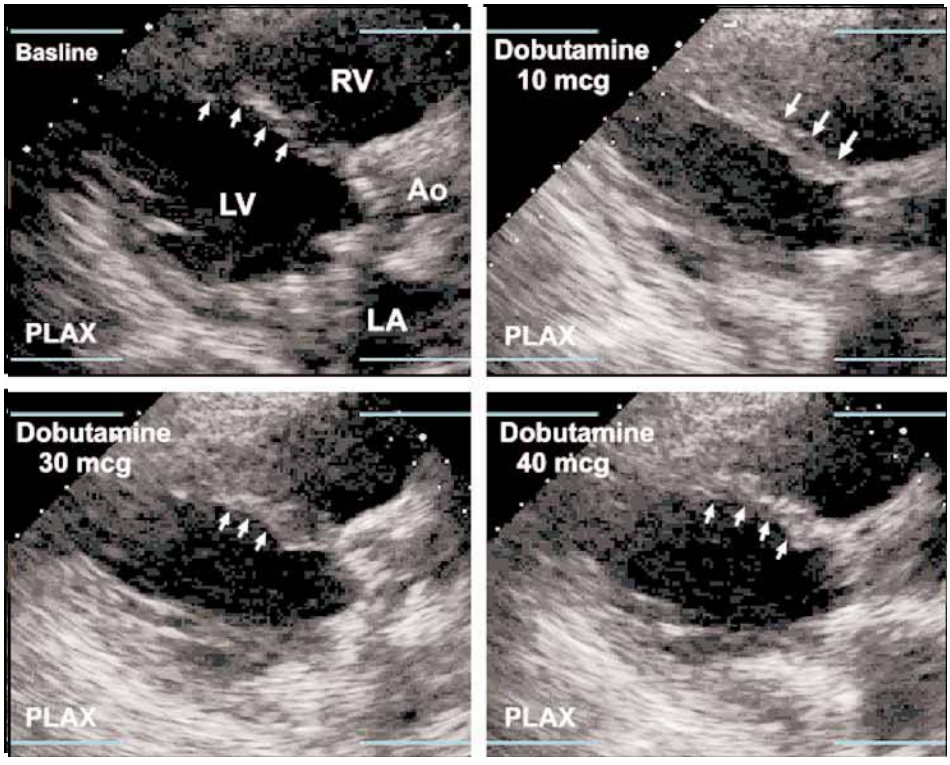

Fig. 17. Dobutamine stress echocardiogram: biphasic response. A biphasic response on dobutamine stress echocardiography may be a candidate for coronary artery revascularization procedures. This patient shows augmentation of a previously poorly functioning region on low dose dobutamine (10 μg), but demonstrates ischemia (decreased contractility) at higher doses. At baseline, this region of the heart (arrows) are hypokinetic—contractility improves at the 5 μg infusion rate, is maintained at 10 μg, worsens at 40 μg. This represents an ischemic region that augmented at low doses that can benefit from revascularization. (Please *see* companion DVD for corresponding video.)

Table 7
Doppler Indices of Left Ventricular Systolic Function

Traditional Doppler indices	*Newer Doppler indices*
$SV = VTI \times CSA = VTI \times \pi r^2$ $= VTI \times \pi D^2/4 = 0.785\ D^2 \times VTI$	TDI/DTI
Measurement sites: LVOT Left ventricular inflow (mitral valve) Pulmonary artery	
$CO = SV \times HR$ CI = CO/body surface area	TVI for left ventricular dyssynchrony
CW Doppler in mitral regurgitation: dP/dt = 32/time (mmHg/s)	Doppler strain imaging: strain and strain rate
Velocity/acceleration times, e.g., aortic flow/velocity acceleration, aortic ejection time	Left ventricular torsion by TDI

SV, stroke volume; VTI, velocity time integral; CSA, cross-sectional area; D, diameter; TDI, tissue Doppler imaging; DTI, Doppler tissue imaging; LVOT, left ventricular outflow; CO, cardiac output; HR, heart rate; CI, cardiac index; TVI, tissue velocity imaging; CW, continuous wave; dP/dT, rate of ventricular pressure rise.

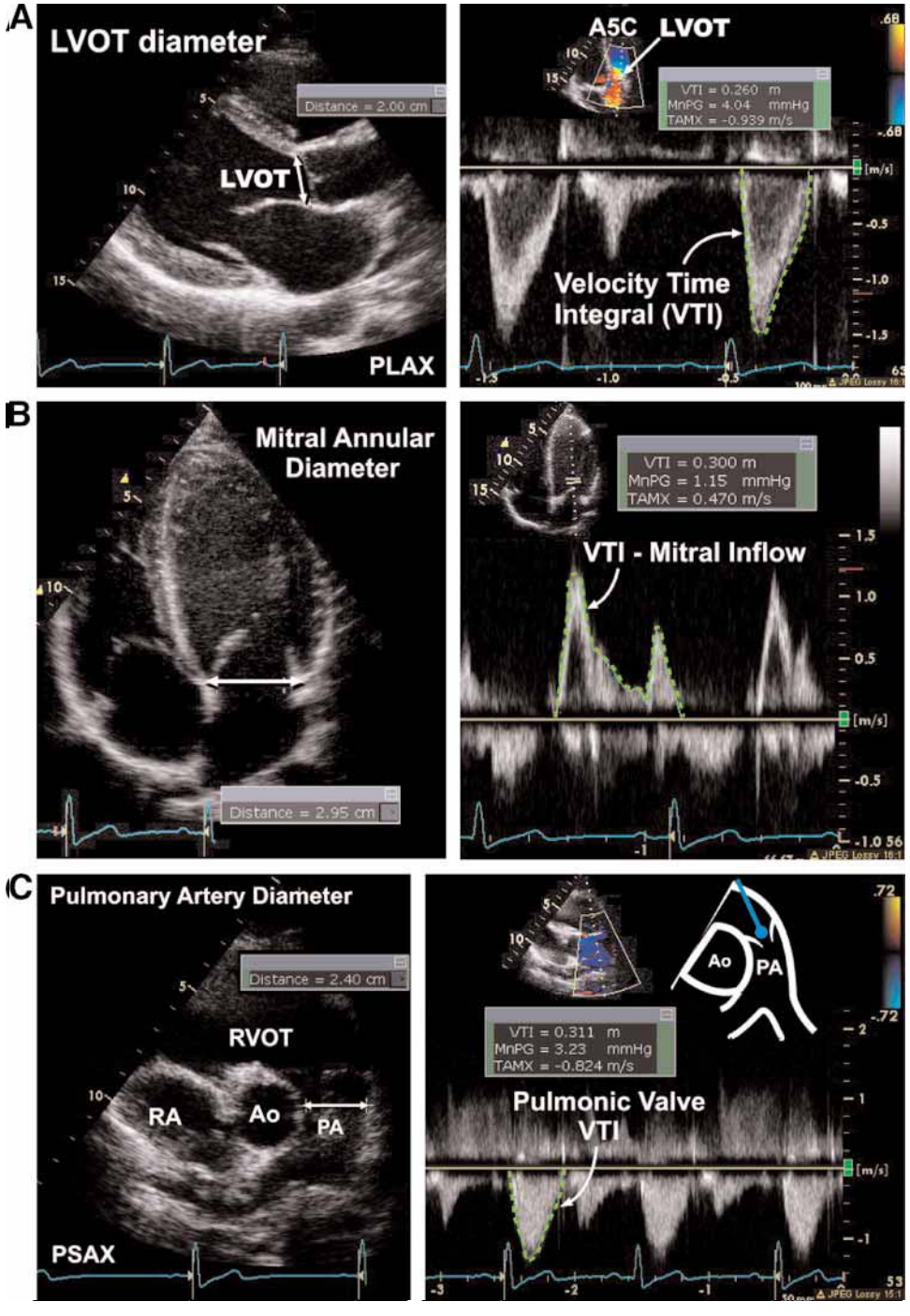

Fig. 18. **(A)** Stroke volume by Doppler (LVOT). **(B)** Stroke volume by Doppler (mitral inflow). **(C)** Stroke volume by Doppler (pulmonary artery).

MYOCARDIAL PERFORMANCE INDEX (TEI INDEX)

The myocardial performance index (MPI) is a Doppler-derived integrated measure of ventricular systolic and diastolic function.

It has been the subject of much interest since its inception in 1995, and has been well received for its ability to assess both LV and RV function in a variety of patients—heart failure, cardiomyopathy, coronary heart disease, heart transplantation, and in prospective clinical trials. It is reproducible, easy to measure and can predict morbidity and mortality in patients with cardiomyopathy and heart failure.

When applied to the LV, it is the sum of the isovolumic contraction and relaxation times (ICT + IRT) divided by

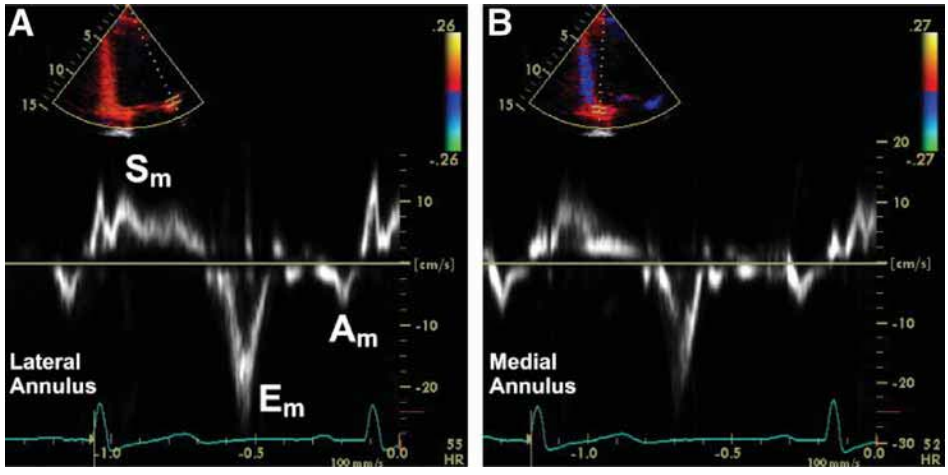

Fig. 19. Tissue Doppler imaging (TDI). Tissue Doppler assesses myocardial velocities during the cardiac cycle. Doppler shift measured at the lateral **(A)** and septal annulus **(B)** are shown. Systolic shifts (S_m) are upward (positive). Shifts away from the transducer (E_m and A_m), reflecting early and late diastolic velocities, are downward (negative).

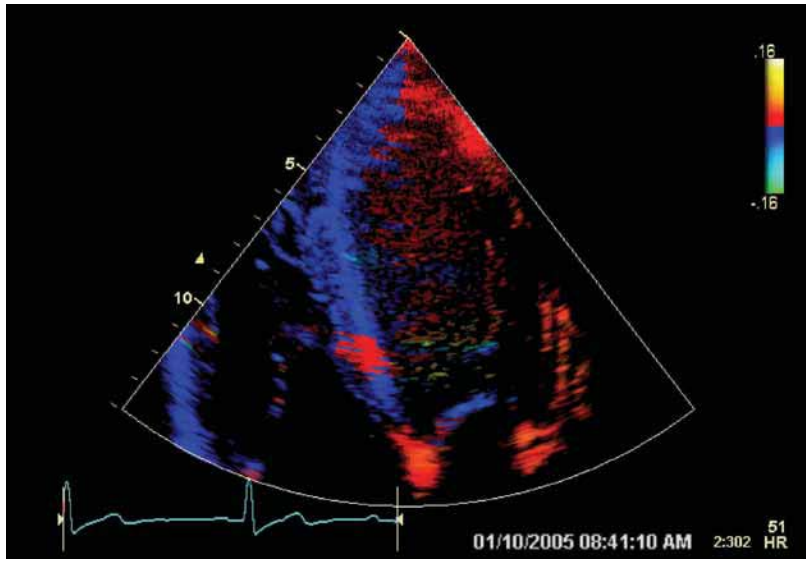

Fig. 20. Tissue velocity imaging (TVI). Differential tissue velocities by color Doppler can detect differential contractility of left ventricle (LV) segments—and color coded as shown. This reflects LV dyssynchrony and impaired LV systolic function. (Please *see* companion DVD for corresponding video.)

the ejection time. These measurements are obtained by Doppler assessment of both LV inflow and outflow and using the formula (Fig. 22):

$$\text{Left Ventricular MPI} = \frac{(\text{ICT} + \text{IRT})}{\text{ET}}$$

The MPI has its limitations. It is not a load-independent measure, and one of its components, the IRT, is less discriminatory in patients with worsening diastolic dysfunction. Therefore, despite its utility, it should complement (not substitute) established measures of LV function, e.g., ventricular volumes and EF.

ASSESSMENT OF RV FUNCTION IN HEART FAILURE AND POSTMYOCARDIAL INFARCTION

In patients with heart failure, RV dysfunction is associated with increased mortality. RV dysfunction is an important predictor of risk and heart failure following myocardial infarction.

MORPHOLOGICAL CONSIDERATIONS

The RV exhibits a far more complex geometry than that of the LV. It is thin walled (<0.5 cm) and assumes

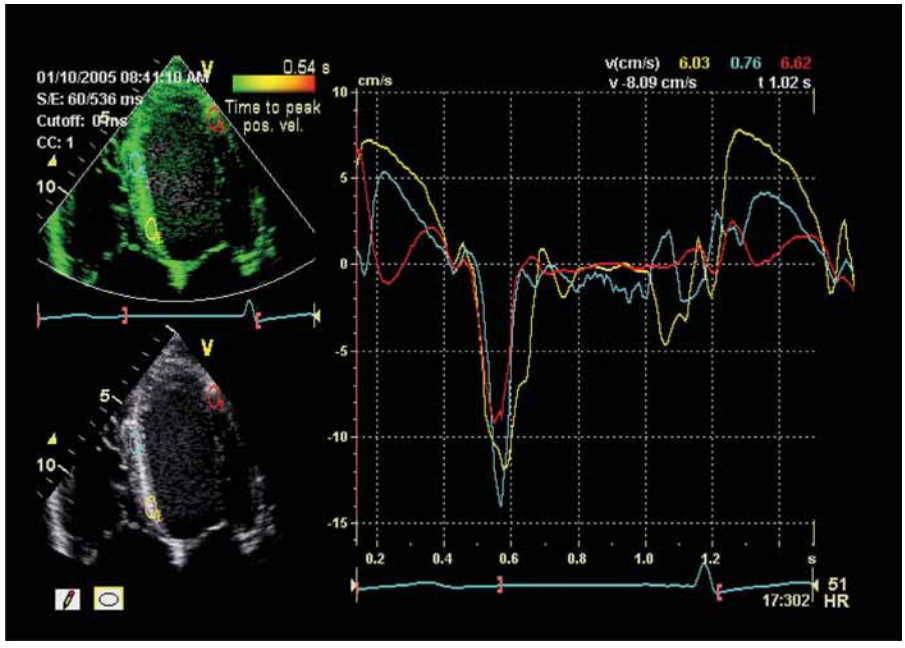

Fig. 21. Doppler strain imaging. Doppler strain imaging showing normal synchronous tissue Doppler tracings of three interrogated myocardial segments are shown.

a flattened pear-shaped appearance folded over the LV (Fig. 23). Such geometry makes it especially difficult to assess by 2D techniques. Most volumetric methods of RV assessment are complex and not well validated, especially in diseased states—when RV geometry is becomes even more complex (Chapter 18).

RV CHAMBER DIMENSIONS

Like the LV, the RV should be assessed using multiple windows. The subcostal window provides the best visualization of the RV free wall. RV size, wall thickness, and systolic function should be recorded, and systolic function described as normal or reduced to varying degrees. RV enlargement can be estimated using the "rule of thirds" in the parasternal long-axis view, or by comparing its size relative to that of the LV in the apical four-chamber view. The RV diameter does not normally exceed one-third the total ventricular width in the apical four-chamber view (Fig. 24; Tables 8 and 9).

Attempts to quantify RV volumes and systolic function by echocardiography include indices like tricuspid annular motion, tricuspid fractional shortening, and RV fractional area change (RVFAC). Tricuspid annular motion refers to the distance the tricuspid annulus moves in the antero-posterior direction. Tricuspid fractional shortening is an assessment

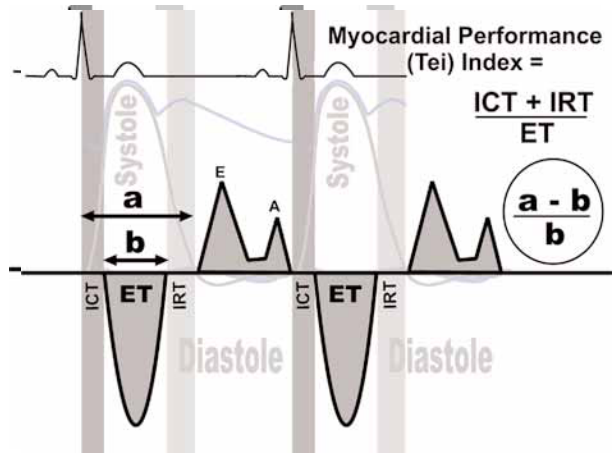

Fig. 22. Myocardial infarction (Tei) index.

of the difference between the maximal and minimal distance between the tricuspid annuli during the cardiac cycle. RVFAC is assessed by measuring RV areas in the apical four-chamber view and comparing the relative change between diastolic and systole. When all three approaches are compared to cardiac magnetic resonance imaging (MRI), the best correlation is seen with RVFAC measurements. RV MPI has also proven a useful measure of RV function. Standards for RV volumetric assessment, however, are yet to be established.

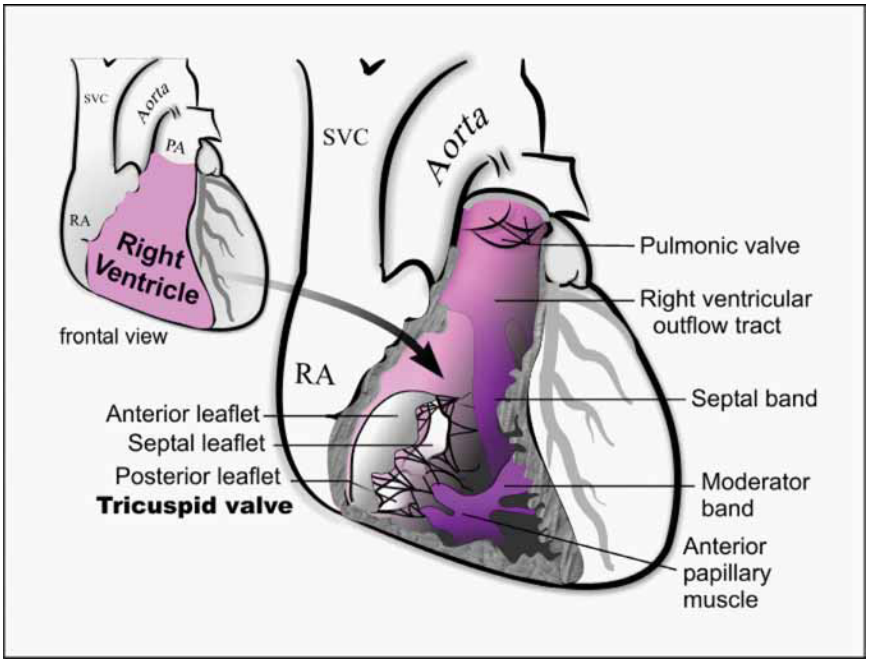

Fig. 23. Right ventricular morphology.

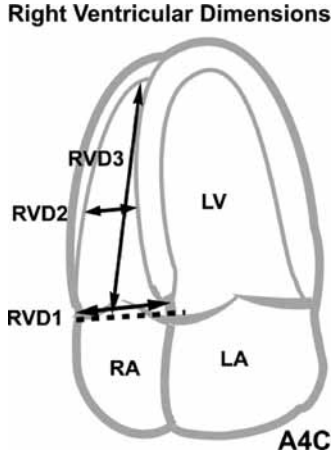

Fig. 24. Right ventricle dimensions (cm). (Please *see* companion DVD for corresponding video.)

ECHOCARDIOGRAPHY TO DETERMINE ETIOLOGY OF SYSTOLIC DYSFUNCTION

Echocardiography can be of use in determining the etiology of systolic dysfunction (Table 1). Patients with ischemic heart disease almost always have discrete regional wall motion abnormalities—most often secondary to prior myocardial infarction. In contrast, global systolic dysfunction, without regional variation, is more suggestive of nonischemic cardiomyopathy. Severe regurgitant valvular heart disease, such as mitral and aortic regurgitation, can lead to a dilated LV with reduced systolic function. Pressure overload conditions, such as aortic stenosis, severe hypertension, or coarctation, usually lead to hypertrophy, although ventricular dilatation and dysfunction can occur late in the course of disease. Various forms of congenital heart disease can lead to systolic dysfunction and can usually be identified on echocardiography. Infiltrative diseases such as amyloidosis can cause severe LV dysfunction and can usually be identified by pathognomonic echocardiographic features. In the case of amyloid, severe LV hypertrophy, "speckled" appearing myocardium, atrial dilatation, nonspecific valve thickening and pericardial effusions are common. Occasionally, hypertrophic cardiomyopathy can ultimately lead to ventricular dilatation and dysfunction (so-called "burnt-out" hypertrophic cardiomyopathy).

USE OF CONTRAST AGENTS IN ECHOCARDIOGRAPHY

As early as the late 1960s, it was noted that intravascular injection of almost any solution resulted in a contrast effect detectable by echocardiography. Contrast microbubbles are used nowadays to complement the imaging process in ultrasound. The advances in imaging modalities in the modern ultrasound systems in tandem with the development of newer agents has made contrast imaging more effective and applicable in daily clinical practice.

Table 8
Reference Limits and Partition Values of Right Ventricular Size

	Reference range	*Mildly abnormal*	*Moderately abnormal*	*Severely abnormal*
RV Dimensions				
Basal right ventricular diameter (RVD1) (cm)	2.0–2.8	2.9–3.3	3.4–3.8	≥3.9
Mid right ventricular diameter (RVD2) (cm)	2.7–3.3	3.4–3.7	3.8–4.1	≥4.2
Base-to-apex length (RVD3) (cm)	7.1–7.9	8.0–8.5	8.6–9.1	≥9.2

Table modified from Recommendations for Chamber Quantification. American Society of Echocardiography (ASE), 2005.

Table 9
Reference Limits and Partition Values of Right Ventricular Size and Function as Measured in the Apical Four-Chamber View

	Reference range	*Mildly abnormal*	*Moderately abnormal*	*Severely abnormal*
Right ventricular diastolic area (cm^2)	11–28	29–32	33–37	≥38
Right ventricular systolic area (cm^2)	7.5–16	17–19	20–22	≥23
Right ventricular fractional area change (%)	32–60	25–31	18–24	≤17

Table modified from Recommendations for Chamber Quantification. American Society of Echocardiography (ASE), 2005.

Contrast Agents

Contrast agents are encapsulated bubbles of gas smaller than the red blood cells and, therefore, capable of circulating freely within the body. The use of contrast dates back to 1968 when Gramiak and Shah first used injected saline to enhance the signals from the blood pool. This was followed years later by encapsulated air bubbles, and more recently by the use of encapsulated low solubility gas bubbles (such as perfluorocarbons). These newer agents are capable of passing through the pulmonary circulation without destruction.

Contrast agents approved for use in the United States to improve LV opacification (LVO) in technically difficult echocardiograms include Optison®, an octafluoropropane within albumin microspheres, Definity™, an octafluoropropane in a phospholipid shell, and Imavist®, a perfluorohexane and other perfluorocarbon gases encapsulated within a surfactant shell. In Europe, Sonovue™, which contains sulfur hexafluoride in a phospholipid shell, is widely used.

The ideal contrast agent should be a nontoxic, easily injectable intravenously (as a bolus or infusion) and should remain stable during cardiac and pulmonary passage for the duration of the ultrasound examination. The agent should have strong echogenic interaction in response to incident ultrasound waves.

Preparation and Administration of the Contrast Agent

The ASE recommends that cardiac sonographers take the appropriate steps to become trained in the preparation and administration of contrast agents. The sonographer is often the first person to recognize the need for contrast, but the physician is ultimately responsible for prescribing its use, which should be done on a case-by-case basis.

Venous access and appropriate instrument settings should be performed prior to preparation of the contrast agent. Each agent has a different method of preparation and administration. Each manufacturer's separate instructions should therefore be followed. The choice between bolus vs continuous infusion depends on the indication for the study and the type of information required. Bolus administration is easier to perform and is sufficient for LVO and Doppler enhancement. However, continuous infusion may be required for myocardial perfusion studies and quantitative analysis. A registered nurse or physician usually administers the injection or infusion of the contrast agent while the sonographer acquires the images.

Ultrasound and Contrast

The mechanical index (MI) represents the normalized energy to which a target (such a bubble) is exposed in an ultrasound field. It gives an estimate of the peak negative pressure to which tissue is exposed. In simple terms, the MI is the intensity of the transmitted ultrasound beam. It varies with the depth in the image. In most of the ultrasound systems, the MI ranges from 0.1 to 2.0. In the absence of attenuation, the MI is maximal at the focus of the ultrasound beam.

Gas bubbles are very effective scatterers of ultrasound waves within the diagnostic frequency range compared to solids. The degree of scattering increases as the MI is increased. Echo signals from microbubbles contain harmonics, which can be detected. Bubble destruction at high MI emits a strong harmonic echo.

There are three main patterns of scattering produced by microbubbles, depending on the peak pressure of the incident sound field. At a peak pressure of less than 100 kPa (MI < 0.1), bubbles produce "linear" oscillations resulting in backscatter enhancement. During this low MI imaging, bubbles act as simple, but powerful, echo enhancers. This is the principle utilized for enhancing spectral Doppler such as in pulmonary venous flow signals. At a peak pressure of 100 kPa to 1 MPa (MI 0.1–1.0), there is "nonlinear" oscillation resulting in harmonic backscatter. This principle is utilized in harmonic B-mode LVO and real-time perfusion imaging. At a peak pressure of more than 1 MPa (MI > 1.0), there is bubble disruption resulting in transient harmonic echoes. This is the principle utilized in power Doppler imaging.

Contrast Imaging Modes

Conventional grayscale imaging results in linear backscatter, and, hence, is useful for enhancement of the LV cavity, providing better endocardial definition (*see* Fig. 7B). The concept of "harmonic imaging" emerged from the observation that "nonlinear" oscillations of the microbubbles results in the generation of "second harmonics." Therefore, imaging can be improved by preferential detection of these "second harmonics" that emanate directly from the microbubbles themselves rather than the tissue. In harmonic B mode imaging, the transmitted frequency typically lies between 1.5 and 3 MHz and the received frequency between 3 and 6 MHz to enable detection of these bubble harmonics.

"Contrast-Specific" Imaging Modalities

Although harmonic imaging improves visualization of bubble harmonics, it imposes some fundamental limitations in bandwidth and hence fails to completely suppress the tissue harmonics. Detection of bubbles in myocardial capillaries (i.e., perfusion), therefore, would require tedious off-line background subtraction to suppress the echoes produced by the tissue. To overcome this, "contrast specific" methods are required for assessment of myocardial perfusion by enhancing contrast harmonics while suppressing tissue harmonics.

Examples of such modalities are:

1. Pulse inversion: high MI technique. By sending two pulses (one inverted) in rapid succession toward the tissue, summation occurs and results in a strong harmonic signal that is exclusively from the microbubbles. However, wall motion artifacts could still attenuate image quality.
2. Harmonic power Doppler: intermittent imaging (high MI technique). The strong, transient echoes produced by bubble destruction provide a highly sensitive method of imaging the microbubbles. The disadvantage is that wall motion (which produces a Doppler shift), is also detected and this potentially interferes with image quality.
3. Low power "real-time" contrast imaging (power pulse inversion/power modulation/coherent imaging): this is a nondestructive, continuous real-time imaging (low MI) technique. In this mode, sequences of more than two pulses are transmitted in alternating phase. Although the sensitivity may be slightly lower than the high power technique, this method allows wall motion information to be available without the need for bubble disruption. This method is much easier to use and avoids many artifacts that occur with high power harmonic imaging. The echoes from the bubbles are well separated from those of tissues, thereby providing better characterization of "real-time" myocardial perfusion.

Clinical Uses of Contrast Echocardiography

LV Opacification

One of the most common clinical indications for echocardiography is in the assessment of regional and global LV function. This should be accurate and reproducible. A pre-requisite for reliable assessment of LV function is accurate visualization of the endocardium. In up to 20% of resting studies, endocardial border definition is suboptimal—defined as the inability to visualize at least two myocardial segments of the LV. The advent of tissue harmonic imaging has significantly improved endocardial definition compared to fundamental imaging.

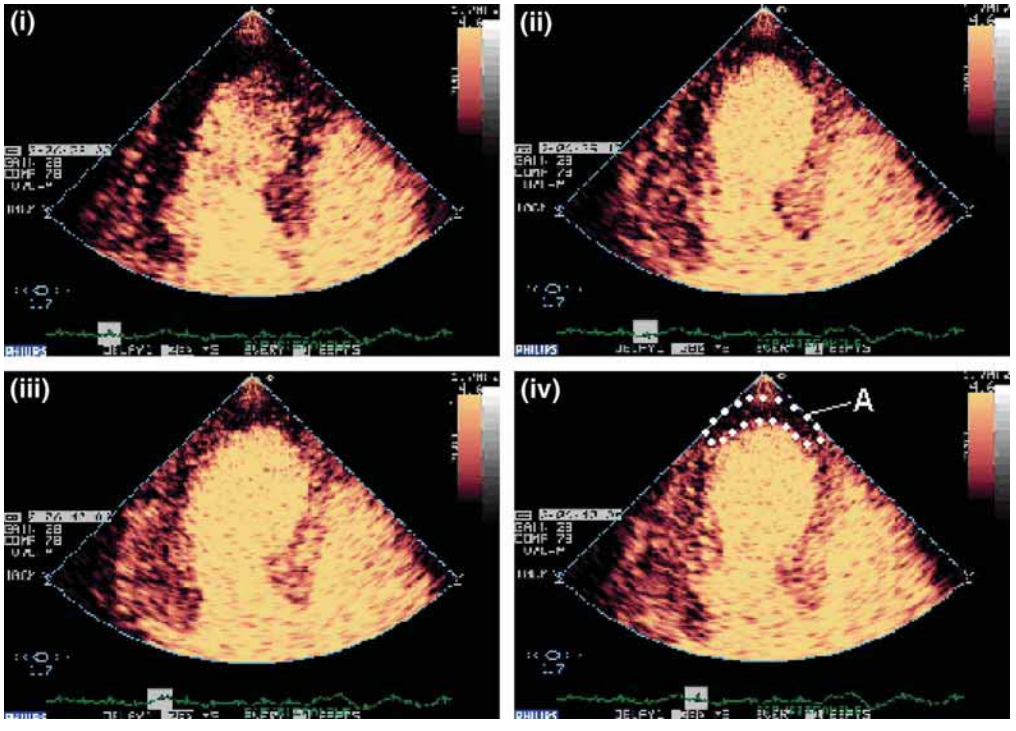

Fig. 25. Frame (i) is immediately following a high power ultrasound flash that destroys the microbubbles within the myocardium. Frames (ii) to (iv) show replenishment of microbubbles in the septum and lateral walls within two heartbeats. A clear apical perfusion defect **(A)** that persists is demonstrated. (Reproduced with permission from R Janardhanan, et al. Myocardial contrast echocardiography: a new tool for assessment of myocardial perfusion. Ind Heart J 2005;57:210–216.)

Nevertheless, 5–10% of studies employing tissue harmonics imaging are still suboptimal.

The primary clinical use of contrast echocardiography is for LVO (Chapter 5, Fig. 7B; Chapter 8, Figs. 8 and 10). By injecting microbubbles that traverse the pulmonary circulation, the LV can be opacified and endocardial definition significantly improved. Studies using contrast-enhanced LVO have shown excellent correlation with MRI in the determination of LV volumes and EF. Currently, this use is the only Food and Drug Administration-approved indication for echocardiographic contrast agents.

Many studies have shown the incremental value of using contrast agents to improve image quality, the percentage of wall segments visualized, and the confidence of interpretation of resting and stress echocardiography images (Chapter 8, Figs. 8 and 10). Contrast agents in stress echocardiography should be used whenever resting image quality is suboptimal.

Contrast agents can assist in the identification of LV thrombi. Approximately 15–45% of echocardiographic studies may fail to identify a LV thrombus. Fundamental imaging from the apical windows may fail to detect apical LV thrombi owing to near-field artifacts. The use of contrast agents permits almost 90% of initially non-diagnostic images to become diagnostic.

Enhancement of Doppler Flow Signals

The accuracy of spectral Doppler velocity measurements depends on obtaining a clear envelope of the Doppler signal. The quality of Doppler recordings, e.g., tricuspid regurgitation velocity, pulmonary venous signals, and so on, can be augmented by using contrast agents. Contrast agents may also be useful in the detection of suspected intracardiac and intrapulmonary shunts.

Echocardiographic Contrast Agents vs Saline Contrast. Echocardiographic contrast agents that traverse the pulmonary circulation differ from agitated saline contrast used for detecting intracardiac shunts. Saline bubbles do not traverse the pulmonary circulation, except when an arterio-venous malformation is present. Because they do not traverse the pulmonary circulation, agitated saline contrast provide no LVO under normal conditions. Echocardiographic contrast agents that traverse the pulmonary circulation should not be used to diagnose intracardiac shunts.

Myocardial Perfusion Imaging

Myocardial contrast echocardiography (MCE) can accurately assess both myocardial blood volume and microbubble velocity (both of which determine myocardial blood flow). Although not yet licensed for this indication, MCE shows great potential as a clinical tool to evaluate myocardial perfusion.

MCE may be superior to techniques like sestamibi SPECT in the detection of myocardial perfusion. This is most likely explained by the superior temporal and spatial resolution of MCE over SPECT. Furthermore, MCE can be performed at the bedside and does not involve ionizing radiations.

MCE detects contrast bubbles at the capillary level within the myocardium and hence, is marker of capillary integrity. This is the principle behind the use of MCE in patients post-MI in the detection of myocardial viability (Fig. 25).

Safety Considerations. Current contrast agents have an excellent safety profile, and complications are rare. Allergic reactions have been occasionally reported. However, in patients with intracardiac or intrapulmonary right-to-left shunts, the potential for adverse events are slightly greater. There are conflicting reports of increased frequency of premature ventricular complexes especially with high MI-triggered imaging. However, this has not been shown in larger studies.

SUGGESTED READING

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