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STATE-OF-THE-ART PAPER

Echocardiography in Heart Failure

Applications, Utility, and New Horizons

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Echocardiography is well qualified to meet the growing need for noninvasive imaging in the expanding heart failure (HF) population. The recently-released American College of Cardiology/American Heart Association guidelines for the diagnosis and management of HF labeled echocardiography "the single most useful diagnostic test in the evaluation of patients with HF. . .," because of its ability to accurately and noninvasively provide measures of ventricular function and assess causes of structural heart disease. It can also detect and define the hemodynamic and morphologic changes in HF over time and might be equivalent to invasive measures in guiding therapy. In this article we will discuss: 1) the clinical uses of echocardiography in HF and their prognostic value; 2) the use of echocardiography to guide treatment in HF patients; and 3) promising future techniques for echocardiographic-based imaging in HF. In addition, we will highlight some of the limitations of echocardiography. (J Am Coll Cardiol 2007;50:381–96)

Echocardiography is, according to the recently released American College of Cardiology/American Heart Association (ACC/AHA) guidelines for the diagnosis and management of heart failure (HF), "the single most useful diagnostic test in the evaluation of patients with HF. . .," because of its ability to accurately and noninvasively provide measures of ventricular function and assess causes of structural heart disease (1). An estimated 5 million people have HF, and their ranks are increased by an estimated 550,000 each year (2). Heart failure hospital stays have increased 150% over the last 20 years (2). The lifetime risk of developing HF has been estimated at 20% for the U.S. population. And, although ischemic heart disease is the most common cause of HF, up to 11% of the population without evidence for coronary artery disease will also develop HF (3).

Heart failure is classically described as left ventricular (LV) dysfunction leading to congestion and reduced systemic perfusion, most often manifesting symptomatically as dyspnea and fatigue. After an insult to the myocardium, the LV progressively dilates or hypertrophies, a process followed by spherical remodeling. These morphologic changes cause further stress on the myocardium by increasing wall tension and cause or exacerbate mitral regurgitation, which, in turn, results in further dilatation and contractile dysfunction in a vicious cycle (1). Such remodeling is often the final common pathway for many although not all etiologies of HF.

Because this morphologic process begins before the onset of symptoms, the recent HF guidelines place special emphasis on detecting subclinical LV systolic and diastolic dysfunction (1,4). Several studies have emphasized that standard physical examination maneuvers are suboptimal in detecting either systolic or diastolic LV dysfunction, especially in the preclinical phase. Similarly, physical examination is limited in its ability to accurately characterize the volume and cardiac output status in patients with LV dysfunction (5,6). As a rapid and accurate modality, echocardiography can improve the noninvasive detection and definition of the hemodynamic and morphologic changes in HF. Echocardiography might also be equivalent to catheter-based techniques in guiding therapy and improving outcomes, without the risks and cost of invasive measures (5).

In this review we will discuss: 1) the clinical uses of echocardiography in HF and their prognostic value; 2) the use of echocardiography to guide treatment in HF patients; and 3) promising future echocardiographic techniques for cardiac imaging in HF. We will also highlight some of the limitations of echocardiography (outlined in Tables 1 and 2). We will use the term "echocardiography" in a general sense to refer to all cardiac ultrasound imaging techniques, including M-mode, 2- and 3-dimensional imaging, and spectral

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Abbreviations and Acronyms

CRT = cardiac resynchronization therapy

EF = ejection fraction

HF = heart failure

ICD = implantable cardioverter-defibrillator

LV = left ventricle/ventricular

LVEDP = left ventricular end-diastolic pressure

LVOT = left ventricular outflow tract

TDI = tissue Doppler imaging

SF = systolic fraction of pulmonary venous forward flow

VAD = ventricular assist device

Vp = flow propagation slope of early diastolic left ventricular filling

and color Doppler, which comprise the comprehensive sonographic assessment of cardiac structure and function.

Clinical Measurements and Prognosis

Used for many years to provide structural correlates to the clinical picture of HF, echocardiography can also measure multiple clinically important parameters of cardiac function, including hemodynamic status and LV ejection fraction (EF), volumes, and mass.

Hemodynamics. Intracardiac pressure measurements have traditionally required invasive methods. This limitation, which also precludes serial measurements outside of the intensive care context, can often be circumvented with the use of echocardiographic techniques. In selected patients, echocardiography might be a noninvasive surrogate (Fig. 1). Stroke volume and cardiac output can be estimated from the velocity time integral obtained by pulse wave Doppler recordings in the left ventricular outflow tract (LVOT), multiplied by the LVOT area. Figure 1 illustrates the echocardiographic estimates of right atrial pressure, right ventricular systolic pressure/pulmonary artery systolic pressure, and pulmonary artery mean and diastolic pressures (7–9). All of these measurements require adequate imaging windows and parallel alignment of the Doppler cursor with blood flow to avoid underestimation of Doppler jet velocity and calculated pressure. Stroke volume as measured in the LVOT is overestimated in the presence of significant aortic insufficiency. Small errors in the measurement of LVOT diameter lead to large errors in the calculation of LVOT area. Pulmonary artery pressure estimates require the presence of tricuspid valve regurgitation for systolic pressure and pulmonic valve regurgitation for mean and diastolic pressures as well as an accurate estimate of right atrial pressure (10).

A variety of echocardiographic techniques can determine abnormal diastolic function, increased left atrial pressure and left ventricular end-diastolic pressure (LVEDP). These measurements have demonstrated considerable prognostic value in symptomatic and asymptomatic patients with either preserved or abnormal LV systolic function (11). The adverse prognosis associated with systolic dysfunction is well described, but isolated diastolic HF also carries a poor prognosis, including future development of systolic HF (12).

Diastolic function can be characterized according to severity. Mild diastolic dysfunction—abnormal LV relaxation—can be detected via a decrease in early diastolic flow velocity (E-wave) and a greater reliance on atrial contraction (A-wave) to fill the LV (E/A <1). Moderate diastolic dysfunction—"pseudonormalization"—reflects increasing left atrial pressure at the onset of diastole and an increase in early diastolic flow velocity to a level near that of normal filling (E/A 1 to 1.5). Severe diastolic dysfunction—restrictive filling—occurs when left atrial pressure is further elevated such that early diastolic flow is extremely rapid and left atrial and LV pressures equalize quickly during early diastole (E/A >2, DT <115 to 150 ms). Reduction in preload with the Valsalva maneuver can unmask diastolic dysfunction by changing a pseudonormalized pattern to an abnormal relaxation pattern or a restrictive pattern to a pseudonormalized one (13,14). Persistence of a restrictive filling pattern during the Valsalva maneuver or on follow-up echocardiogram after HF therapy portends a particularly grim prognosis, as shown by Pinamonti et al. (15) and others. These traditional techniques, however, are dependent on heart rate and loading conditions and lack validity in patients with preserved EF.

Table 1 Evaluation of Standard Doppler Echocardiographic Techniques in HF

Technique	Strengths	Limitations
Doppler (hemodynamics)	1. Facile 2. Rapid 3. On-line	1. Requires parallel alignment of Doppler beam 2. Pulmonary and tricuspid valve regurgitation not always present 3. Stroke volume measurement from LVOT overestimated in significant AI
Doppler (diastolic function)	1. Facile 2. Rapid 3. On-line 4. Prognostic	1. Requires parallel alignment of Doppler beam 2. Heart rate dependent 3. Load dependent
2D EF, dimensions and mass	1. Facile 2. Rapid 3. Prognostic 4. On-line	1. Dependent on image quality 2. Foreshortening common 3. High inter- and intra-observer variability 4. Requires geometric assumptions 5. Does not correlate well with clinical status

2D = 2-dimensional; AI = aortic insufficiency; EF = ejection fraction; HF = heart failure; LVOT = left ventricular outflow tract.

Table 2 Evaluation of Novel Doppler Echocardiographic Techniques in HF

Technique	Strengths	Limitations
Real time 3D for EF and volumes	1. Eliminates foreshortening 2. Geometric assumptions not required 3. Simultaneous assessment of all wall segments	1. Highly dependent on image quality 2. Extra expense of software and probe 3. Incremental value over 2D not well established 4. Sonographer expertise required 5. Not widely available
Tissue Doppler, strain, and strain rate	1. Prognostic 2. Most parameters load independent 3. Widely available (tissue velocity) 4. Less dependent on image quality	1. Angle dependent 2. Strain and strain rate require off-line analysis 3. Low signal/noise ratio
Tissue tracking	1. Not angle dependent 2. Able to assess torsional mechanics	1. Extra expense of software 2. Incremental value over TDI not well established 3. Speckles move in and out of plane (requires mathematical assumptions to compensate) 4. Requires off-line analysis 5. Not widely available

3D = 3-dimensional; TDI = tissue Doppler imaging; other abbreviations as in Table 1.

Additional parameters such as: 1) an abnormal ratio of the systolic and diastolic velocities of pulmonary venous inflow (S/D <1); 2) a systolic fraction of pulmonary venous forward flow (SF) of <40%; and 3) an early LV filling flow propagation slope (Vp) of <45 cm/s are less dependent on loading conditions and heart rate and have been shown to be robust predictors of high LV filling pressures and cardiovascular mortality (16,17). These measures are limited by inability to adequately image the pulmonary veins in some patients and by the limited reproducibility of Vp. A ratio of peak early mitral inflow velocity (E) to peak early diastolic myocardial velocity (E') of ≤8 predicts an LVEDP of <15 mm Hg, whereas a ratio of >15 predicts an elevated LVEDP (≥15 mm Hg) (Fig. 1D) (14). The ratio of peak early mitral inflow velocity to slope of the propagation velocity (E/Vp) of ≥1.5 predicts a LVEDP >15 mm Hg and has been shown to have prognostic value in post-myocardial infarction patients (18). Increased left atrial volumes (>32 ml/m^2), which are usually larger in diastolic compared with systolic HF, have been shown to predict morbidity (19). Among multiple diastolic parameters in patients with a broad range of EF and degrees of mitral regurgitation, Rossi et al. (20) have demonstrated that a >30-ms difference between pulmonary vein atrial flow reversal and mitral A-wave durations was the most sensitive predictor of elevated LVEDP >18 mm Hg. Interestingly, E/E' has proven superior to brain natriuretic peptide (BNP) levels in diagnosing volume overload, even in patients with preserved systolic function (21,22). Tables 3 and 4 list patient population, Doppler modality, cutoff values, and outcome measures used in a variety of prognostic studies of diastolic function and filling pressures (12,15–17,19,23–41). Figures 2A and 2B depict strategies for using Doppler techniques to noninvasively estimate filling pressures and characterize the severity of diastolic dysfunction in patients with reduced EF. The strategies reflect the fact that synthesis of multiple parameters is often required to give an assessment of filling dynamics, particularly when poor acoustic windows might limit the ability to make every measurement.

The myocardial performance index. The myocardial performance index (better known as the Tei index) is a simple Doppler parameter that provides global assessment of systolic and diastolic function. The Tei index consists of the ratio of the isovolumic contraction + isovolumic relaxation times/the ejection time—all parameters that can be obtained from Doppler interrogation. The Tei index is independent of heart rate and blood pressure, applies to left and right ventricular systolic and diastolic dysfunction, does not rely on geometric assumptions, and is highly reproducible, although normal values vary with age (42,43). It has been correlated with invasively measured changes in LV dP/dt (44). The prognostic value of the Tei index was initially tested in patients with infiltrative cardiomyopathy and pulmonary hypertension (45). It has subsequently been validated in patients with dilated cardiomyopathy, with a value of >0.77 proving superior to EF in predicting cardiac death and disease severity (46). The Tei index also proved beneficial in predicting the development of HF in a cohort of elderly men without baseline LV dysfunction (47) and in predicting the lack of clinical response to medical treatment in a study including both patients with systolic HF and HF with preserved systolic function (48). Because adequate Doppler images can often be acquired when 2-dimensional (2D) image quality is suboptimal, the Tei index might be particularly useful when other measures of left and right ventricular function are obscured or indeterminate.

EF and dimensions. Traditionally, EF measurements have been visually estimated with important limitations of subjectivity and dependence on highly trained expert interpretation for accuracy. Although symptoms guide the majority of HF management decisions, precise and reproducible EF measurements play an increasingly important role in guiding important interventions. Consequently, quantified, objective measurements of LV systolic function should become standard practice in echocardiography. Al-

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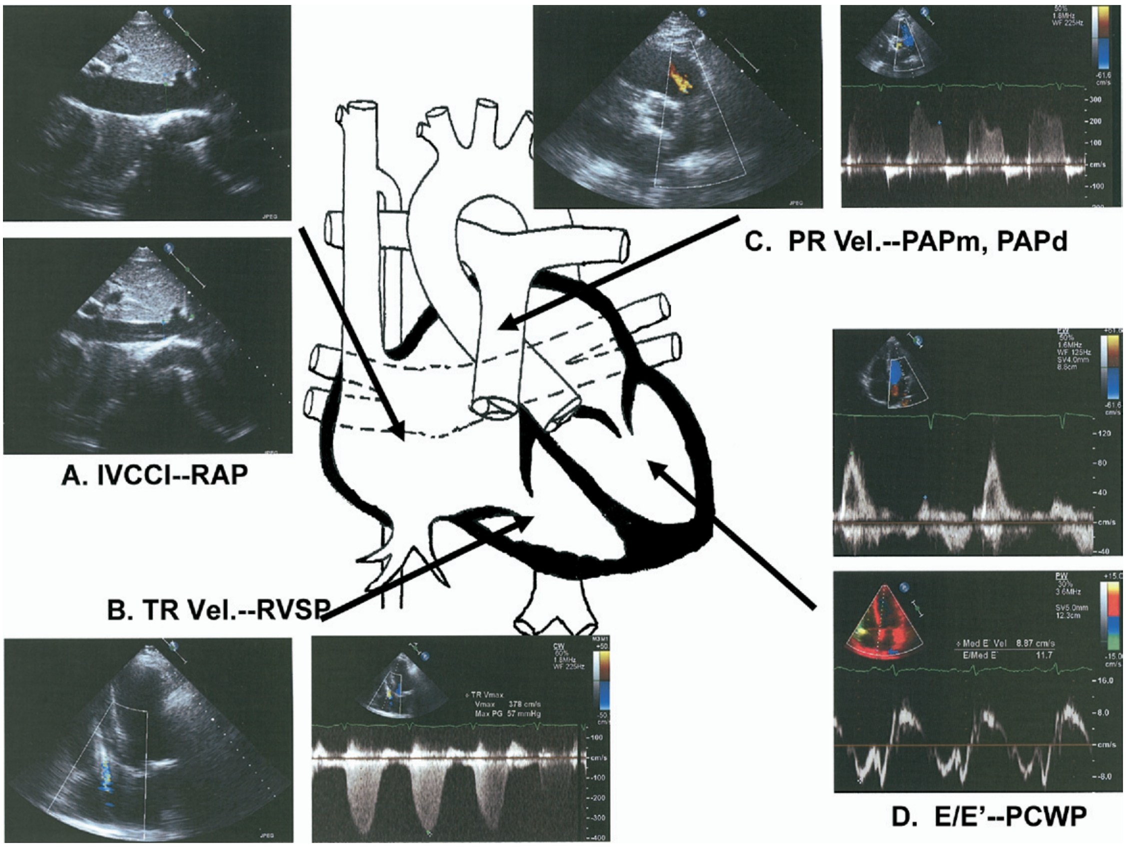

Figure 1 **Illustration of "Echo Right Heart Catheterization"**

(A) Inferior vena caval (IVC) size and degree of collapse yields a range of right atrial pressure (RAP): <1.2 cm with full collapse = RAP 0 mm Hg; 1.2 to 1.7 cm with >50% collapse = RAP 0 to 5 mm Hg; >1.7 cm with >50% collapse = RAP 6 to 10 mm Hg; <50% collapse = RAP 10 to 15 mm Hg; and >1.7 cm with 0% collapse = RAP >15 mm Hg (51). **(B)** The tricuspid regurgitant velocity (TR Vel) is used to estimate the systolic right ventricle—right atrium gradient (and the pulmonary artery systolic pressure, in the absence of pulmonic stenosis). **(C)** The maximal pulmonic valve regurgitant velocity is used to estimate the mean pulmonary artery pressure (PAPm). The end diastolic pulmonic regurgitant velocity is used to estimate diastolic pulmonary artery pressure (PAPd). **(D)** The early mitral inflow (E-wave)/early diastolic mitral valve annular motion (E'-wave) ratio <8 or >15 is calculated to assess PCWP <15 mm Hg or >15 mm Hg, respectively. E/E' = ratio of early diastolic mitral inflow velocity to early diastolic velocity of the mitral valve annulus; IVCCI = inferior vena cava collapsibility index; PCWP = pulmonary capillary wedge pressure; PR Vel. = pulmonic valve regurgitant velocity; RVSP = right ventricular systolic pressure.

though fractional shortening measured from M-mode tracings can quantify LV function, it is valid only in a symmetrically contracting heart without regional variability and is therefore inappropriate for the remodeled ventricles of many HF patients. The new guidelines from the American Society of Echocardiography (ASE) advocate the biplane method of discs for EF quantification and discourage the use of M-mode measurements that rely on geometric assumptions to convert linear measurements to 3-dimensional (3D) volumes (49). An alternative method for volume calculation, useful when the endocardium is not well defined, is the area-length method. This method assumes a bullet-shaped ventricle and involves planimetry of the mid-ventricle short-axis area and the annulus-to-apex length in systole and diastole. With either 2D method, the new ASE guidelines define an abnormal EF as <55%, with the cutoffs for moderately abnormal and severely abnormal at 44% and 30%, respectively. The reference ranges for LV dimensions are best indexed to body surface area, with reference ranges 2.4 to 3.2 cm/m^2 and cutoff values of 3.5 and 3.8 cm/m^2 for moderate and severe dilation, respectively (49).

Image quality in patients with poor acoustic windows has traditionally played a major role in limiting the accuracy of quantification of LV volumes and EF. Tissue harmonic imaging with and without echocardiographic contrast for LV cavity opacification has improved the accuracy and reproducibility of EF measurements (50,51). This method has enabled the accurate assessment of LV function in nearly all patients, irrespective of body habitus, chest wall deformities, or pulmonary diseases (52–54) (Fig. 3). Two-dimensional echocardiography, even when employing these methods, lacks accuracy compared with the gold standards of magnetic resonance imaging (MRI) or radionuclide ventriculography for quantification of EF and volumes (55). The reasons for the consistent underestimation of LV

Table 3 Prognostic Significance of Echocardiographic Diastolic Dysfunction Measures: Single Component Studies

Modality	Patient Population	Cutoff Values	Outcome
Mitral inflow Doppler			
E/A	2,671 elderly patients, no CVD	<0.7 or >1.5	Incident HF
E/A	1,839 hypertensive patients	Age- and heart rate-adjusted ratio below median	Cardiovascular events*
E/A	3,008 Native Americans	<0.6 or >1.5	Death or cardiac death
DT	110 patients, EF <50%, no CAD	<115 ms, persisting after 3 months' HF treatment	Death or transplant at 4 yrs
Peak E	2,671 elderly, no CVD	Continuous	Incident HF
DT	571 patients post-AMI	<130 ms	Death at 4 yrs
DT	79 HF patients, no CAD	<115 ms	Death or transplant
M-mode IVRT	185 elderly HF patients	≤30 ms	Death
Pulmonary vein Doppler			
PV AR dur − MV A dur	145 LV dysfunction patients	≥30 ms	Cardiac death or hospital stay
S/D	115 patients, EF <45%	<1	HF hospital readmission or HF death at 1 yr
Tissue Doppler			
E/E'	250 patients post-AMI	>15	Death
E/E'	45 patients, NYHA functional class III or IV HF	Continuous	Predictor of NYHA functional class, HF hospital stay, cardiac death
E/E'	130 chronic HF patients	>12.5	Composite: cardiac death, HF hospital stay, urgent transplant
E/E'	110 patients hospitalized with HF	≥15	Cardiac death or hospital readmission for HF
E', E/E'	518 patients referred for echocardiography	E' <3 or 3–5 cm/s, E/E' >20	Cardiac death
Systolic mitral annular velocity	185 patients, EF <45%	Continuous	Death or transplant
LA volume	1,375 elderly patients with preserved EF	≥ 32 ml/m^2	Incident HF
Flow propagation			
E/Vp	67 post-MI patients	E/Vp ≥1.5	Death and HF readmission
Vp	125 post-MI patients	Vp <45 cm/s	Cardiac death

*New onset event = myocardial infarction, sudden cardiac death, unstable angina, revascularization, stroke/transient ischemic attack, hospital stay for heart failure (HF), symptomatic aorto-iliac disease, end-stage renal disease.

A = atrial filling velocity; AMI = acute myocardial infarction; CAD = coronary artery disease; CVD = cardiovascular disease; D = diastolic pulmonary vein wave; DT = deceleration time of E-wave; E = early diastolic filling velocity; E' = tissue Doppler early filling velocity; EF = ejection fraction; IVRT = interventricular relaxation time; LA = left atrium; LV = left ventricle; MV A dur = mitral valve atrial wave duration; NYHA = New York Heart Association; PV AR dur = pulmonary vein atrial reversal duration; S = systolic pulmonary vein wave; Vp = flow propagation velocity slope.

volumes and EF involve reliance on geometric assumptions, combined with foreshortening of the LV from transducer positioning errors. This underestimation might be overcome with the use of 3D echocardiography (56).

Although EF and LV dimensions do not correlate with HF symptoms, exercise capacity, or myocardial oxygen consumption (57,58), they do provide crucial prognostic information (59). Morbidity and mortality are closely linked to both EF and LV volumes in HF patients in multicenter trials (60,61). Although influenced by a myriad of demographic and clinical factors, post-myocardial infarction prognosis is most powerfully predicted by EF and LV size. Early studies of acute myocardial infarction survivors using cineangiography and radionuclide ventriculography demonstrated EF <40% and increased LV volumes to be predictors of subsequent cardiovascular mortality and sudden death (62–65). More recent studies using echocardiography have also found EF and LV volumes to be powerful prognosticators for major adverse cardiac events (66,67). The ability of echocardiography to assess global dysfunction and regional wall motion has aided in the assessment of the size of myocardial infarctions. This measurement predicts cardiogenic shock (if >40% of the myocardium is involved), development of chronic HF, and mortality, despite the fact that myocardial stunning and hibernation complicate the prediction of eventual infarct size (68,69).

LV mass. Although LV mass has received less attention in clinical cardiology than EF, it is an important prognostic marker in HF in patients with and without coronary artery disease (70). This observation in smaller studies was confirmed in the echocardiographic substudy of the SOLVD registry and trials, in which investigators examined the effect of LV hypertrophy on clinical outcomes and found that increased LV mass was associated with high mortality and rate of cardiovascular hospital stays, independent of EF (71). Because population studies suggest that the etiology of HF in African-American patients is more likely to be hypertensive than ischemic (72), the routine and accurate measurement of LV mass and its prognostic significance might be even more salient in this population.

Left ventricular mass assessment is subject to the same limitations in reproducibility and accuracy as measurement of LV dimensions (73). The current ASE guidelines recommend mass calculation from linear dimensions with the cubed formula, modeling the LV as a prolated ellipse, because this method has been validated in multiple studies

Table 4 Prognostic Significance of Echocardiographic Diastolic Dysfunction Measures: Composite Studies

Composite Study	Patient Population	Components	Outcome	Degree of Predictive Significance
Mild diastolic dysfunction vs. normal	2,042 patients >45 yrs	E/A <0.75, E/A Valsalva <0.5, E/E' <10, S >D, PV ARdur >MV Adur	Death	HR 8.31
Moderate or severe diastolic dysfunction vs. normal	2,042 patients >45 yrs	E/A 0.75–1.5, DT >140 ms, E/A Valsalva ≥0.5, E/E' ≥10, S <D, PV AR dur − MV A dur >30 ms E/A >1.5, DT <140 ms, E/A Valsalva <0.5, E/E' ≥10, S <D, PV AR dur − MV A dur >30 ms	Death	HR 10.17
Restrictive vs. nonrestrictive	311 HF patients evaluated for transplant	E/A >2 or DT ≤140 ms	Death or transplant	RR 2.4
Restrictive vs. nonrestrictive	98 chronic HF patients	E/A >1 and DT ≤130 ms	Cardiac death or transplant	NA
Restrictive vs. nonrestrictive	100 patients, EF <40%	E/A ≥2 or 1–2 plus DT <140 ms	Cardiac death at 2 yrs	RR 8.6
Restrictive vs. nonrestrictive	193 HF patients, EF <45%	E/A >2, DT <150 ms, E'<8 cm/s	Cardiac death or early transplant	RR 6.62
Restrictive vs. nonrestrictive	63 patients with amyloid cardiomyopathy	E/A >1 and DT <150 ms	Cardiac death at 1 yr	RR 4.87
AR vs. PN vs. RFP	115 patients admitted with HF	E/A <1 or >2, DT <140 or >230 ms, PV AR dur/MV A dur >1.2	Death, HF hospital readmission	PN and RFP more predictive of outcomes than AR

AR = abnormal relaxation; HR = hazard ratio; PN = pseudonormal; RFP = restrictive filling pattern; RR = relative risk; other abbreviations as in Table 3.

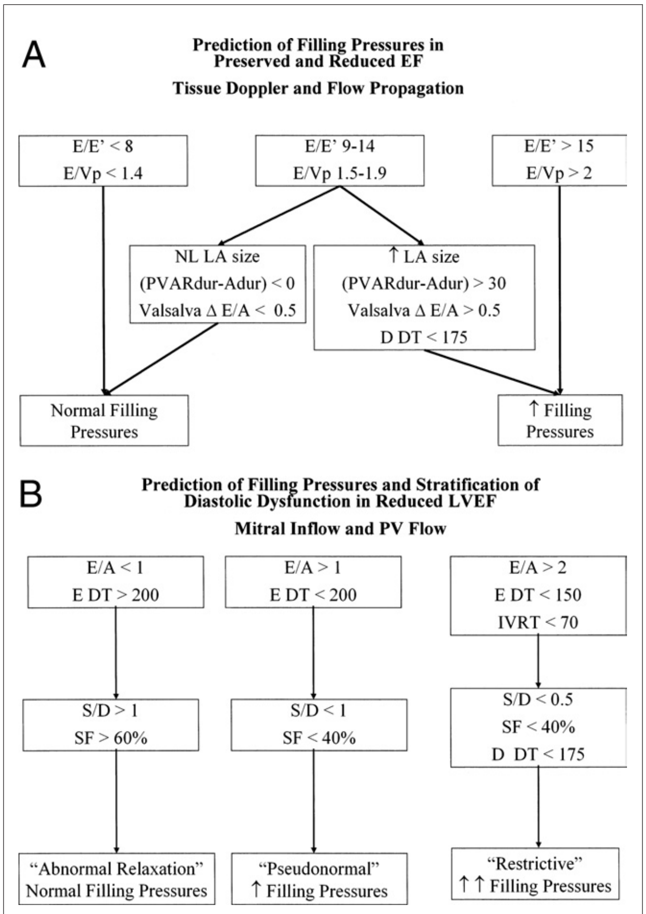

Figure 2 Diastolic Filling Parameters and the Prediction of Normal Versus Elevated Filling Pressure

(A) Prediction of normal versus elevated filling pressure. In patients with preserved and reduced left ventricular ejection fraction (LVEF), normal filling pressures are predicted by normal mitral inflow E-wave to tissue Doppler E'-wave ratio (E/E') and mitral inflow E-wave to flow propagation ratio (E/Vp) values or intermediate values with normal left atrium (LA) size, normal pulmonary vein atrial reversal duration minus mitral inflow A-wave duration (ARdur − Adur), and a minimal change in the E/A wave ratio with Valsalva. Elevated filling pressures are predicted by elevated E/E' and E/Vp values or intermediate values with elevated LA size, prolonged ARdur − Adur, a substantial change in the E to A valve with Valsalva, or a prolonged pulmonary vein D-wave deceleration time (DDT). **(B)** Degree of diastolic dysfunction. In patients with reduced LVEF, mitral inflow E/A, mitral inflow E-wave deceleration time (EDT), and isovolumic relaxation time (IVRT) parameters, confirmed by pulmonary vein S to D ratio (S/D), systolic fraction of pulmonary venous forward flow (SF), and DDT can further define filling dynamics by stratifying diastolic function into "abnormal relaxation" (normal filling pressures), "pseudonormal" (elevated filling pressures), and "restrictive" (very high filling pressures) categories. Valsalva ΔE/A = change in mitral inflow E to A-wave ratio with Valsalva maneuver. (Modified from Nagueh SF, Zoghbi WA. Clinical Assessment of LV Diastolic Filling by Doppler Echocardiography. ACC Current Journal Review. 2001; July/Aug: 49).

with pathologic correlation. The cubed formula lacks precision, however, when applied to many HF patients, because it involves geometric assumptions that are invalid in a nonsymmetrically contracting, remodeled ventricle. Two-dimensional methods, including the truncated ellipsoid and the area-length formula, might be more appropriate for distorted ventricles with regional wall motion abnormalities. These methods, however, rely heavily on geometric assump-

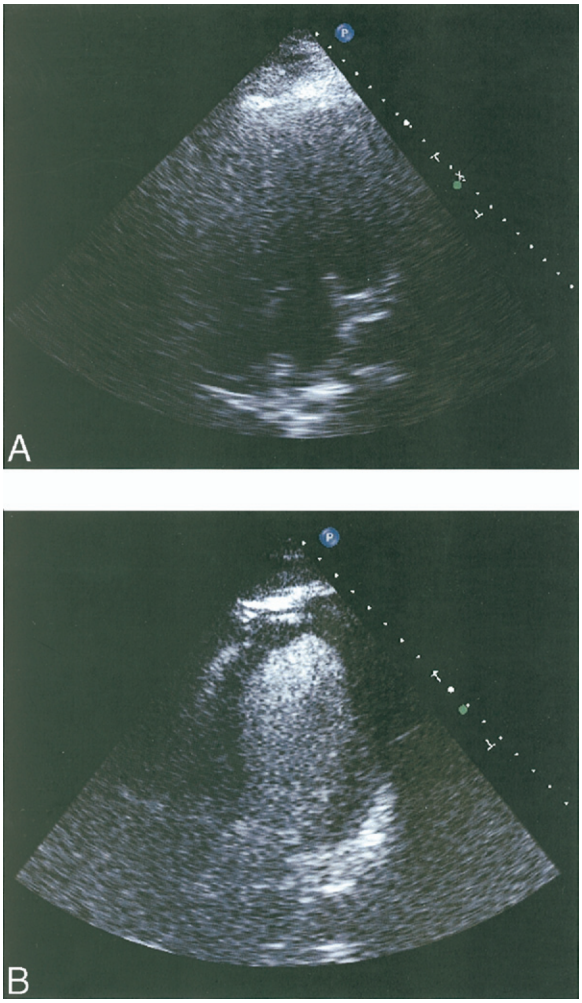

Figure 3 **Contrast Opacification of the Left Ventricular Cavity**

(A) Apical 4-chamber views with poor endocardial definition. **(B)** The same view with improved endocardial definition after echocardiographic contrast administration.

tions. Furthermore, as mentioned, these methods are subject to inaccuracies from foreshortening.

Unlike EF and LV dimensions, LV mass has different cutoff values for men and women and for linear and 2D methods. The reference ranges for women are 67 to 162 g and 66 to 150 g for the linear and 2D methods, respectively. Indexed to body surface area, these ranges are 43 to 95 g/m^2 and 44 to 88 g/m^2. For men the reference ranges are 88 to 224 g and 96 to 200 g, and 49 to 115 g/m^2 and 50 to 102 g/m^2 (49).

The LV mass increases in the remodeled, failing heart, either from increased volumes with myocardial thinning or from wall hypertrophy in patients with hypertensive cardiomyopathy (74). Despite its limitations, the assessment of LV mass provides not only an important research tool to evaluate remodeling but also a precise and prognostically powerful way to characterize clinical status.

Therapeutic Guidance

Echocardiography not only provides clinical measures and prognostic assessments in patients with HF but can also supply information to guide application of HF therapies.

Medications. In addition to the demonstrated benefit of angiotensin-converting enzyme (ACE) inhibitors for patients with both symptomatic and asymptomatic LV dysfunction (75), beta-blocker drugs are beneficial for almost all well-compensated patients with LV systolic dysfunction (76,77), and aldosterone antagonists reduce mortality in New York Heart Association functional class III and IV patients hospitalized for HF with EF ≤35% and in post-myocardial infarction patients with EF <40 % (78,79). Not only does echocardiographic EF measurement commonly establish an indication for these medications but also improvements in EF and LV volumes by echocardiography are used as standard measures of therapeutic effect in many clinical trials of HF medications (80,81). Conversely, echocardiography also supplies an assessment tool for the detrimental effects on LV function of cardiotoxic medications, such as anthracycline chemotherapeutic agents. The EF decrements while taking these medications is often an indication for discontinuation (82,83).

The strategy of combining echocardiographic assessment of filling pressures with BNP measurement has definite prognostic value and might prove one of the most accurate ways to noninvasively guide fluid management. In a study by Dokainish et al. (31), an E/E' value >15, combined with BNP ≥250 pg/ml, measured on the day before discharge, had incremental power in predicting readmission or cardiac death compared with traditional clinical risk factors. If proven to be cost-effective, echocardiography with BNP might become an important strategy for triaging HF patients in acute care settings, for assessing suitability for discharge, or for identifying patients who need more intensive outpatient management.

Implantable cardioverter-defibrillators (ICDs). Recent studies have demonstrated the benefit of prophylactic ICD for the primary prevention of sudden death in patients with reduced EF (84,85). Reimbursement strategies for ICDs therefore rely on EF as a common parameter for placement of these devices in HF patients, and echocardiography is often employed to assess EF (1). Repeat EF assessment at 30 to 40 days after myocardial infarction and after initiation of optimal HF medical therapy is necessary to determine candidacy for ICD. Many patients' EF rise above 30% to 35% cutoff after a month on an appropriate medical regimen, and premature ICD implantation has shown no benefit (86).

Cardiac resynchronization therapy (CRT). Many HF patients lack coordinated contraction of the LV walls (intraventricular dyssynchrony) and between the right and left ventricles (interventricular dyssynchrony). Cardiac resynchronization therapy can restore coordinated contraction with demonstrated improvement in symptoms and survival

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(87). Current recommendations and reimbursement strategies advocate that only patients with EF ≤35%, moderate-to-severe HF symptoms, a widened QRS interval, and sinus rhythm should undergo CRT (1). Nevertheless, it is now clear that not all patients meeting these criteria will respond to CRT; furthermore, it has been recently shown that a subgroup of patients lacking these criteria could benefit from CRT (88). Echocardiographic measurement of dyssynchrony can accurately predict beneficial response in the form of reverse remodeling (reduction in LV volumes, improved EF, and reduced mitral regurgitation) (89), and echocardiographically demonstrated reverse remodeling predicts improved survival (90,91). Currently, different techniques are used to assess dyssynchrony, some of which are discussed in the following text (92). It remains to be seen which measurement or combination of measurements will prove most accurate in predicting beneficial response to CRT. This is an area of active investigation in the PROSPECT (Predictors of response to cardiac resynchronization therapy) trial and other studies (93).

Mitral valve surgery. So-called "functional" mitral regurgitation in HF has traditionally been ascribed to stretching of the mitral annulus and malcoaptation of the mitral valve leaflets. More recent echocardiographic and pathologic investigations have described tethering of the mitral valve leaflets from remodeling-induced displacement of 1 or both papillary muscles and structural changes of the mitral valve itself. These mechanisms rather than annular dilatation might be the main determinants of functional mitral regurgitation (94,95).

The rationale behind repair or replacement of the mitral valve has been the traditional perspective on functional mitral regurgitation; nonetheless, surgery has demonstrated efficacy, even in advanced HF (96). But it is not always clear whether repair or replacement is indicated. Furthermore, the severity of functional mitral regurgitation can be reduced by CRT and by ACE inhibitor therapy without surgery, presumably owing to the effects of reverse remodeling (97).

Traditional echocardiographic evaluation of mitral regurgitation has significant limitations. The mitral valve annulus is saddle-shaped and cannot be fully visualized in 2D imaging planes (98). The mitral valve regurgitant jet, especially when eccentric, is also incompletely visualized in traditional imaging planes, leading to misclassification of mitral regurgitation severity. Similarly, the geometric assumptions involved in calculating mitral regurgitation se-

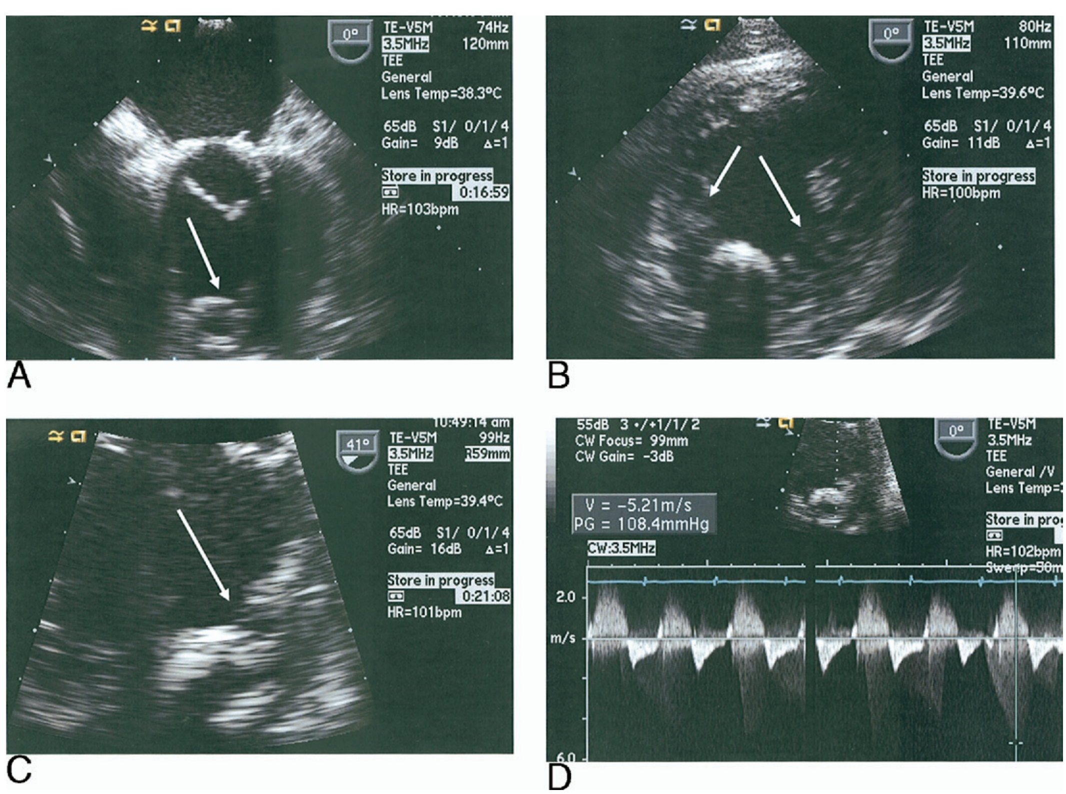

Figure 4 Left Ventricular Assist Device Cannula Obstruction From Trabeculation

(A) Transesophageal echocardiogram in the mid-esophageal 4-chamber view demonstrating inflow cannula of a left ventricular assist device **(arrow)**. **(B)** Transgastric short-axis view demonstrates inflow cannula proximity to an area of hypertrabeculation that is consistent with non-compaction of the left ventricular myocardium **(arrows)**. **(C)** Close-up view in the mid-esophageal 4-chamber view demonstrates an area of hypertrabeculation being pulled into the inflow cannula **(arrow)**. **(D)** Continuous wave Doppler imaging demonstrates high velocities into the inflow cannula, consistent with partial obstruction.

verity with Doppler flow and color Doppler (e.g., calculations of effective regurgitant orifice area) lead to inaccuracies in non-central jets (99,100).

Ventricular reconstruction surgery. A number of ventricular reconstruction surgeries have been proposed for patients with ischemic HF and apical dyskinesis or LV aneurysms (101,102). These surgical mechanical techniques reduce ventricular remodeling and have improved both morbidity and mortality in HF patients in small studies (103,104), but definitive conclusions await the results of ongoing trials (105). Decision-making for these surgical procedures relies heavily on accurate determination of dyskinesis or akinesis, thinning of the apical segment, depressed EF, coexistence of mitral regurgitation, and volumetric measurement (106,107). An elevated LV end systolic volume index >60 ml/m^2, in particular, portends poor postoperative survival (108). Echocardiography provides accurate preoperative modeling to guide the amount of myocardium to be excluded or resected (109). And echocardiography provides an important way to judge the efficacy of these procedures in improving ventricular remodeling, hemodynamic status, and EF.

Ventricular assist devices. Ventricular assist devices (left ventricular assist device = LVAD, bi-ventricular assist device = bi-VAD) are commonly used as bridges to heart transplantation or ventricular recovery and, more recently, have demonstrated both mortality and quality of life benefit as "destination therapy" in patients for whom heart transplantation is not an option (110). Candidates for VAD placement, however, require careful preoperative consideration. Significant intracardiac shunts, such as an atrial septal defect, will be exacerbated by LVAD placement, leading to significant hypoxia (111). Furthermore, significant valvular disease, especially significant aortic stenosis or aortic regurgitation, must be detected to allow valve repair or replacement before VAD implant. Decreased right ventricular function and high pulmonary pressures often necessitate placement of a bi-VAD. Preoperative echocardiography can detect all of these disorders. After implantation, echocardiography can detect thrombus formation within the VAD (112) or other causes of inflow cannula obstruction. (Fig. 4) Doppler and color Doppler imaging can also detect significant inflow and outflow cannulae regurgitation and also assess aortic valve opening and insufficiency (113).

New Horizons for Advanced Echocardiographic Techniques in HF

Echocardiographic techniques applicable to HF patients are advancing rapidly. New techniques have been developed to image myocardial mechanics and provide more precise measurements to guide therapeutic decisions. Although MRI is an established technique for measuring myocardial mechanics and obtaining highly accurate measurements of cardiac size and structures, the evaluation of these parameters by echocardiography is considerably more feasible for

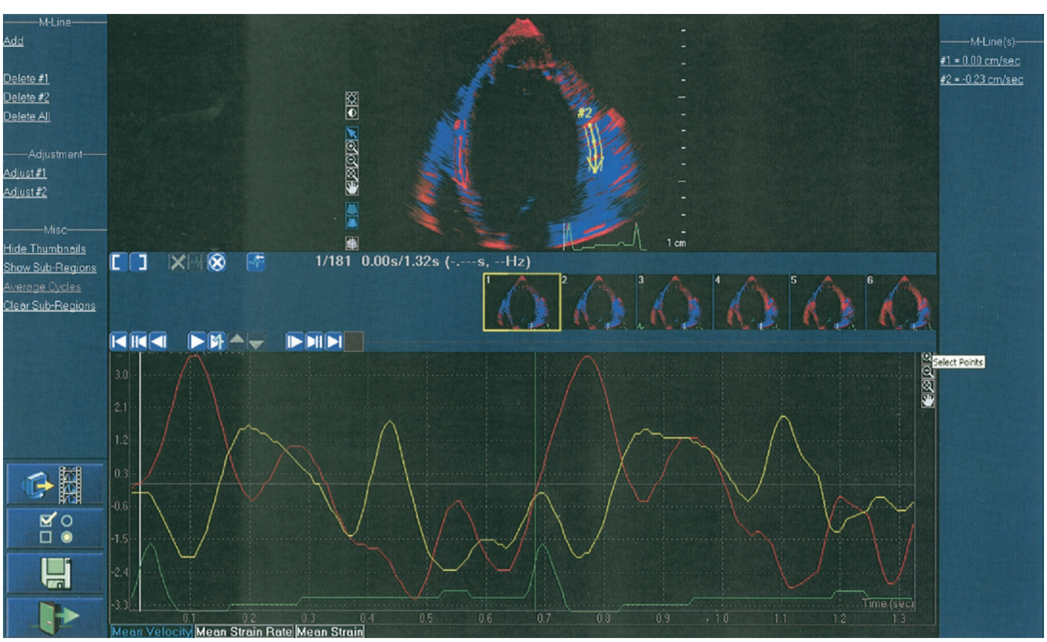

Figure 5 **Tissue Doppler Imaging and Assessment of Regional Function**

Apical 4-chamber view with tissue Doppler imaging showing regional analysis of the contraction patterns of the septal and lateral walls in a patient with dilated cardiomyopathy. Regions of interest (ROI) are drawn at the mid portions of the septal and lateral walls, and the contraction velocities within each ROI in the vertical plane are graphically displayed over time.

clinical use, because it is widely available and repeat assessment can be readily performed.

Myocardial motion, strain, and strain rate. One of the most promising techniques, already used in daily clinical practice, is tissue Doppler imaging (TDI) (Fig. 5). Tissue Doppler imaging has been used with M-mode, 2D, and pulse wave Doppler (114). The peak systolic myocardial velocity, reflective of longitudinal myocardial fiber shortening, has been used to assess systolic function in HF. Yip et al. (115) and Yu et al. (116) noted systolic abnormalities (Sm <4.4 m/s) with TDI in 38% to 52% of HF patients with normal EFs. Their findings suggest that systolic myocardial velocity might provide a more accurate measure of systolic dysfunction than EF. Many HF patients with normal EF might have systolic as well as diastolic HF. In this setting, tissue velocity measures have demonstrated incremental (117,118) and, in a recent study, superior (119) prognostic ability compared with standard echocardiographic measures, including EF.

Tissue velocity measurements are susceptible to artifact from tethering and translational motion (i.e., displacement of the entire heart is recorded as tissue motion of the specific segment being measured). Strain imaging, derived from tissue velocity measurements, overcomes this limitation by measuring actual deformation of the myocardium (expressed as a percentage) in systole and diastole. Strain rate is the inverse of the time to deformation. As measured in the longitudinal direction (base to apex in the apical views), the normal values for strain are 15% to 25% and for strain rate are 1 to 1.5 s^{-1} (120). Like tissue velocity measures, strain and strain rate imaging detect abnormalities of systolic and diastolic function in patients with infiltrative cardiomyopathies (121). In addition, Palka et al. (122) described the use of strain rate to differentiate restrictive cardiomyopathy (reduced early diastolic strain rate, compared with normal hearts) from constrictive physiology (increased early diastolic strain rate).

The identification and measurement of dyssynchrony is one of the most widely published uses of tissue velocity, strain, and strain rate imaging in HF. The TDI techniques predict echocardiographic and clinical response to bi-ventricular pacing with high sensitivity and specificity. Several parameters have been used, such as an intersegmental delay in peak systolic longitudinal motion between segments (abnormal >60 to 65 ms) and the SD of time to peak velocity of 12 segments (abnormal >30 to 31 ms) (123,124). Tissue Doppler imaging can identify the specific regions involved in dyssynchrony as well as the magnitude of dyssynchronous contraction. This information can be available to guide specific placement of bi-ventricular pacing

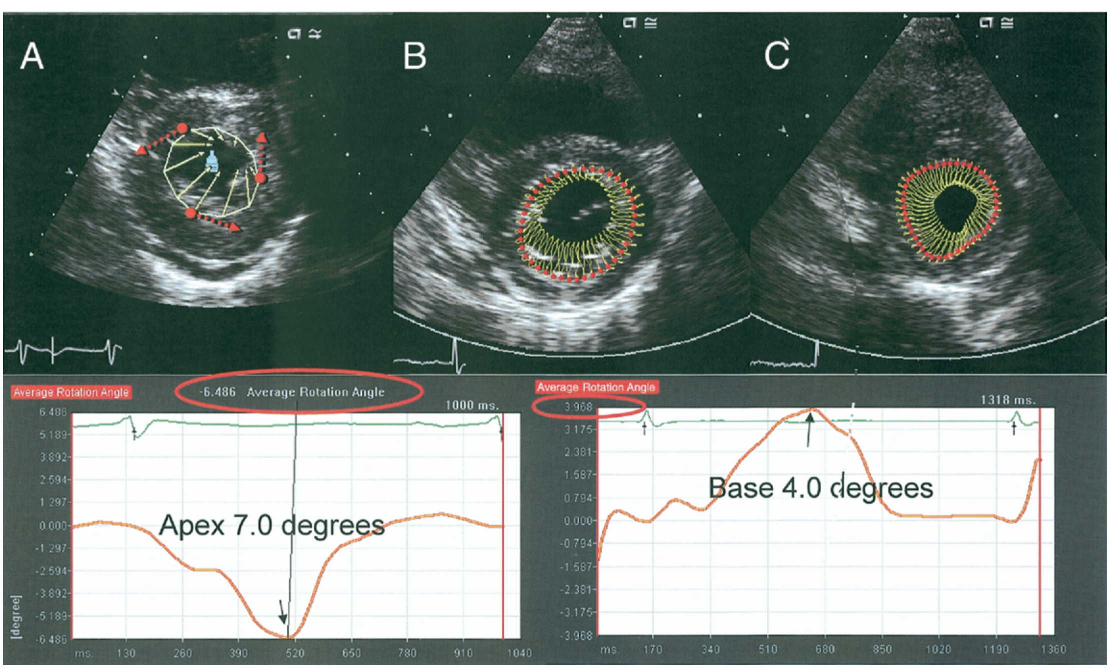

Figure 6 Velocity Vector Imaging

The **upper panels** display radial and rotational mechanics of the left ventricle (LV) by Velocity Vector Imaging (VVI). **(A)** Depiction of radial velocity vectors **(yellow arrows)** in end-systole with the direction of the arrows pointing to a counter-clockwise rotation of the apex. Velocities that are orthogonal to these radial velocities are rotational velocities **(red arrows)**. These velocities are automatically calculated at each location of the arrows around the LV circumference. **(B and C)** Depiction of visual representation of the rotational motion at the base **(B)** of the LV and at the apex **(C)**. The **lower panels** show the magnitude of the rotational motion at the apex and base (degrees of rotation). The apical rotation in this example is −7°, the minus sign depicting counterclockwise rotation. The basal rotation in the clockwise direction is 4°. Thus, the LV twist is 11°, and LV torsion is derived by dividing LV twist by the distance between the base and the apex in centimeters.

leads and assess response during long-term follow-up. Furthermore, as bi-ventricular pacemakers can be set with a delay between right ventricular and LV activation, dyssynchrony measures can been used to optimize these settings, improving intraventricular and interventricular synchrony and hemodynamic status (125).

Tissue tracking. The major limitation to TDI is Doppler angle dependency and problems in assessing regional LV torsional dynamics. In particular, the rotational component of cardiac contraction plays a significant role in LV ejection and relaxation and is poorly imaged by most TDI techniques.

Newer techniques such as "speckle tracking" algorithms involve identification of multiple unique patterns of echocardiographic pixel intensity that are automatically tracked throughout the cardiac cycle. The angular displacement of these pixels can be plotted over time for the apex, mid ventricle, and basal segments. Each pixel's angular displacement is averaged to provide a measurement of both degree and direction of rotational motion for each segment (Fig. 6). This method is not limited by angle dependency and compares favorably with MRI (126,127).

Although the prognostic significance of abnormal ventricular torsion has not been validated in large studies, measurement of rotational motion has shown promise as a sensitive marker for cardiac ischemia (128) and loading conditions (129) and might prove beneficial as a refined measure of LV dysfunction in HF and regional and global dyssynchrony (130). As such, it might become an important marker of the functional significance of remodeling and reverse remodeling in HF patients (131). It might also be able to detect early allograft rejection in transplant patients, potentially circumventing the need for frequent myocardial biopsies. In fact, when combined with 3D imaging techniques, it might prove to be a more sensitive marker for occult LV dysfunction.

3D echocardiography. Previously hampered by the need for cumbersome off-line reconstruction of images, 3D echocardiography has benefited from the development of new matrix array transducers that acquire full volume data sets to allow real-time imaging (132). The ability to visualize all LV walls contemporaneously prevents foreshortening of the LV cavity and allows analysis of regional

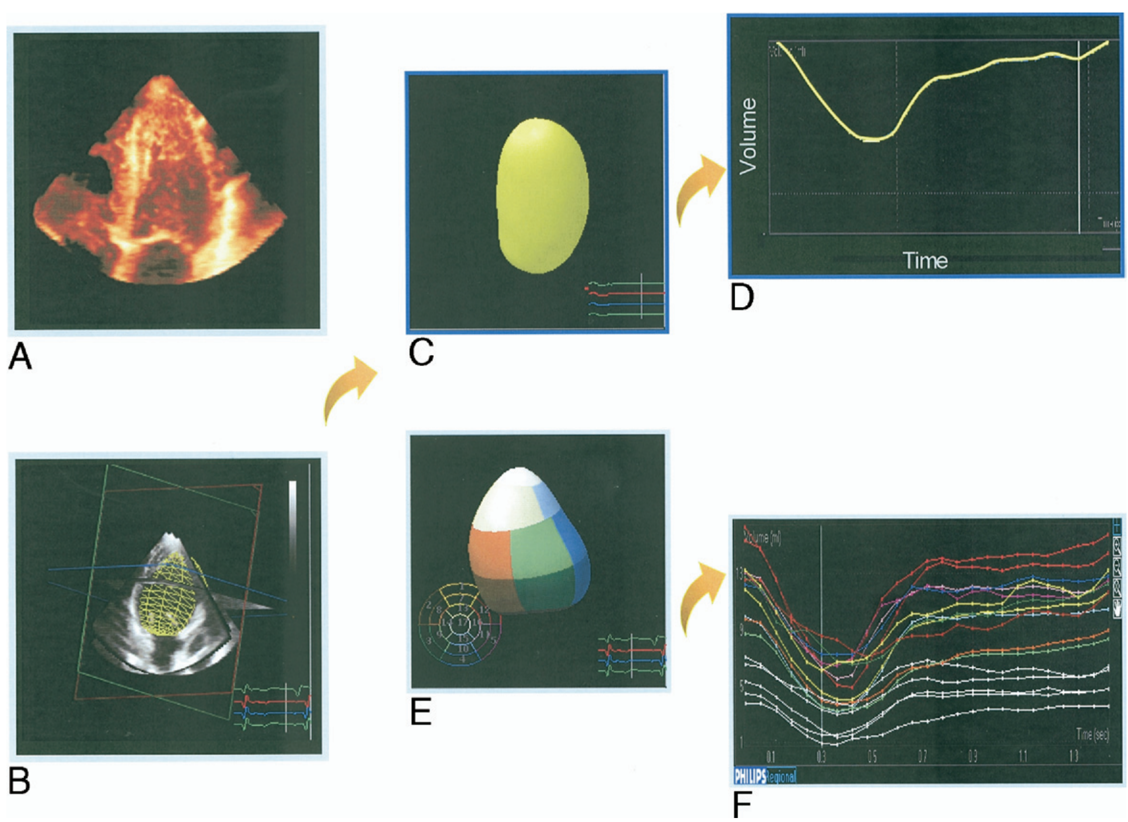

Figure 7 **Real Time 3-Dimensional Echocardiographic Volume Measurements**

(A) A real time 3-dimensional full volume image is acquired in the apical 4-chamber view. **(B)** The image can be cropped and rotated along multiple planes **(red, green, and blue lines)** to isolate the left ventricle (LV) for analysis. **(C)** Application of an automated border detection algorithm yields a cast of the endocardium. **(D)** From the cast, LV cavity volumes can be calculated and tracked over the cardiac cycle. Stroke volume, end systolic and end diastolic volumes, ejection fraction, and filling dynamics can be measured. **(E)** The cast can also be automatically divided into wall segments. **(F)** Regional/segmental volume changes can be tracked over the cardiac cycle, enabling regional wall motion and dyssynchrony analyses.

myocardial function (Fig. 7). No geometric assumptions are required in calculating volumes from a 3D image. In recent studies, 3D echocardiography demonstrated accurate global and regional assessments of LV size and function compared with the gold standard of MRI as well as lower intra- and inter-observer variability than traditional techniques (133). Sugeng et al. (134) showed 3D imaging to be superior to cardiac computed tomography in EF and volumes assessment. Application of new endocardial border detection techniques to 3D images might allow direct calculation of LV volumes and EF, leading to improved reproducibility (135). Mor-Avi et al. (136) demonstrated that, as in volumetric assessments, 3D echocardiography is more accurate and reproducible than methods for LV mass calculation, compared with the gold standard of MRI.

The 3D imaging of global and regional function and LV volumes has not yet translated into clear improvements in or predictions of clinical outcomes. Three-dimensional echocardiography might, in the future, prove beneficial in several areas. The improved precision in measuring EF might guide more appropriate selection of patients for ICD and CRT therapy. Like TDI, 3D echocardiography can assess segmental myocardial motion over time, thereby detecting and characterizing dyssynchrony. Three-dimensional echocardiography has been used in the functional assessment of mitral annular size and tenting volume and improves the echocardiographic measurement of mitral regurgitation (137). A recent review discusses these and other applications of 3D echocardiography in imaging HF patients, such as assessments of atrial size and of right ventricular size and function (138).

Conclusions

Echocardiography is well qualified to meet the growing need for noninvasive imaging in the HF population. Because HF patients often have more than 1 structural and/or functional abnormality contributing to their disease state, echocardiography's versatility in detecting valvular and pericardial pathology along with myocardial disorders yields obvious benefits. Doppler measurements provide important information to direct management of volume status, diagnose and characterize HF with preserved systolic function, and identify patients at high risk for cardiovascular morbidity and mortality. Not surprisingly, the underuse of echocardiography in populations at significant risk for HF is associated with adverse cardiovascular outcomes (139,140). Assessment of LVEF in clinical HF is one of the primary measures in a number of cardiovascular quality improvement initiatives, including the AHA's "Get with the Guidelines" and the ACC's "Guidelines Applied in Practice" (141,142). Echocardiography is well suited to repeated measurements of EF and LV mass in clinical trials and routine patient care. In fact, the new HF guidelines recommend repeat echocardiography for HF patients with changes in symptoms, a clinical event, or a treatment likely to affect cardiac function (1). Echocardiography provides important data for therapeutic decision-making, including defining candidacy for medications, implantable cardiac devices, and surgical procedures. New techniques for the characterization of ventricular mechanics and recent developments in 3D echocardiography hold great promise for improving the quality of care to the growing population of HF patients.

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REFERENCES

1. Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 guideline update for the diagnosis and management of chronic heart failure in the adult: summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). J Am Coll Cardiol 2005;46:e1–82.
2. American Heart Association, American Stroke Association. 2005. Heart disease and stroke statistics: 2005 update. Available at: http://www.americanheart.org/downloadable/heart/1072969766940HSStats2004Update.pdf. Accessed December 3, 2006.
3. Lloyd-Jones DM, Larson MG, Leip EP, et al., Framingham Heart Study. Lifetime risk for developing congestive heart failure: the Framingham Heart Study. Circulation 2002;106:3068–72.
4. Ho KK, Anderson KM, Kannel WB, Grossman W, Levy D. Survival after the onset of congestive heart failure in Framingham Heart Study subjects. Circulation 1993;88:107–15.
5. Capomolla S, Ceresa M, Pinna G, et al. Echo-Doppler and clinical evaluations to define hemodynamic profile in patients with chronic heart failure: accuracy and influence on therapeutic management. Eur J Heart Fail 2005;7:624–30.
6. Badgett RG, Lucey CR, Mulrow CD. Can the clinical examination diagnose left-sided heart failure in adults? JAMA 1997;277:1712–9.
7. Nagueh SF, Kopelen HA, Zoghbi WA. Relation of mean right atrial pressure to echocardiographic and Doppler parameters of right atrial and right ventricular function. Circulation 1996;93:1160–9.
8. Masuyama T, Kodama K, Kitabatake A. Continuous wave Doppler echocardiography of pulmonary regurgitation and its application to noninvasive estimation of pulmonary artery pressures. Circulation 1986;74:484–92.
9. Berger M, Haimowitz A, Van Tosh A, Berdoff RL, Goldberg E. Quantitative assessment of pulmonary hypertension in patients with tricuspid regurgitation using continuous wave Doppler ultrasound. J Am Coll Cardiol 1985;6:359–65.
10. Sorrell VL, Reeves WC. Noninvasive right and left heart catheterization. Echocardiography 2001;18:31–41.
11. Franklin KM, Aurigemma GP. Prognosis in diastolic heart failure. Prog Cardiovasc Dis 2005;47:333–9.
12. Aurigemma GP, Gottdiener JS, Shemanskii L, Gardin J, Kitzman D. Predictive value of systolic and diastolic function for incident congestive heart failure in the elderly: the Cardiovascular Health Study. J Am Coll Cardiol 2001;37:1042–8.
13. Nagueh SF. Noninvasive evaluation of hemodynamics by Doppler echocardiography. Curr Opin Cardiol 1999;14:217–24.
14. Ommen SR, Nishimura RA, Appleton CP, et al. Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: a comparative simultaneous Doppler-catheterization study. Circulation 2000;102:1788–94.

15. Pinamonti B, Zecchin M, Di Lenarda A, Gregori D, Sinagra G, Camerini F. Persistence of restrictive left ventricular filling pattern in dilated cardiomyopathy: an ominous prognostic sign. J Am Coll Cardiol 1997;29:604–12.
16. Dini FL, Dell' Anna R, Micheli A, Michelassi C, Rovai D. Impact of blunted pulmonary venous flow on the outcome of patients with left ventricular systolic dysfunction secondary to either ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 2000;85:1455–60.
17. Garcia MJ, Smedira NG, Greenberg NL, et al. Color M-mode Doppler flow propagation velocity is a preload insensitive index of left ventricular relaxation: animal and human validation. J Am Coll Cardiol 2000;35:201–8.
18. Garcia MJ, Ares MA, Asher C, Rodriguez L, Vandervoort P, Thomas JD. An index of early left ventricular filling that combined with pulsed Doppler peak E velocity may estimate capillary wedge pressure. J Am Coll Cardiol 1997;29:448–54.
19. Takemoto Y, Barnes ME, Seward JB, et al. Usefulness of left atrial volume in predicting first congestive heart failure in patients > or = 65 years of age with well-preserved left ventricular systolic function. Am J Cardiol 2005;96:832–6.
20. Rossi A, Loredana L, Cicoira M, et al. Additional value of pulmonary vein parameters in defining pseudonormalization of mitral inflow pattern. Echocardiography 2001;18:673–9.
21. Mottram PM, Leano R, Marwick TH. Usefulness of B type natriuretic peptide in hypertensive patients with exertional dyspnea and normal left ventricular ejection fraction and correlation with new echocardiographic indexes of systolic and diastolic function. Am J Cardiol 2003;92:1434–8.
22. Dokainish H, Zoghbi WA, Lakkis NM, Quinones MA, Nagueh SF. Comparative accuracy of B-type natriuretic peptide and tissue Doppler echocardiography in the diagnosis of congestive heart failure. Am J Cardiol 2004;93:1130–5.
23. Schillaci G, Pasqualini L, Verdecchia P, et al. Prognostic significance of left ventricular diastolic dysfunction in essential hypertension. J Am Coll Cardiol 2002;39:2005–11.
24. Bella JN, Palmieri V, Roman MJ, et al. Mitral ratio of peak early to late diastolic filling velocity as a predictor of mortality in middle-aged and elderly adults: the Strong Heart Study. Circulation 2002;105: 1928–33.
25. Temporelli PL, Giannuzzi P, Nicolosi GL, et al. GISSI-3 Echo Substudy Investigators. Doppler-derived mitral deceleration time as a strong prognostic marker of left ventricular remodeling and survival after acute myocardial infarction: results of the GISSI-3 echo substudy. J Am Coll Cardiol 2004;43:1646–53.
26. Pinamoti B, Di-Lenarda A, Sinagra G, Camerini F. Restrictive left ventricular filling pattern in dilated cardiomyopathy assessed by Doppler echocardiography: clinical, echocardiographic and hemodynamic correlations and prognostic implications. Heart muscle Disease Study Group. J Am Coll Cardiol 1993;22:808–15.
27. Florea VG, Henein MY, Cicoira M, et al. Echocardiographic determinants of mortality in patients >67 years of age with chronic heart failure. Am J Cardiol 2000;86:158–61.
28. Hillis G, Moller J, Pellikka P, et al. Noninvasive estimation of left ventricular filling pressure by E/e' is a powerful predictor of survival after acute myocardial infarction. J Am Coll Cardiol 2004;43:360–7.
29. Hamdan A, Shapira Y, Bengal T, et al. Tissue Doppler imaging in patients with advanced heart failure: relation to functional class and prognosis J Heart Lung Transplant 2006;25:214–8.
30. Acil T, Wichter T, Stypmann J, et al. Prognostic value of tissue Doppler imaging in patients with chronic congestive heart failure. Int J Cardiol 2005;103:175–81.
31. Dokainish H, Zoghbi WA, Lakkis NM, et al. Incremental predictive power of B-type natriuretic peptide and tissue Doppler echocardiography in the prognosis of patients with congestive heart failure. J Am Coll Cardiol 2005;45:1223–6.
32. Nikitin NP, Loh PH, de Silva R, et al. The prognostic value of systolic mitral annular velocity measured with Doppler tissue imaging in patients with chronic heart failure due to left ventricular systolic dysfunction. Heart 2006;92:775–9.
33. Moller JE, Sondergaard E, Poulsen SH, Seward JB, Appleton CP, Egstrup K. Color M-mode and pulsed wave tissue Doppler echocardiography: powerful predictors of cardiac events after first myocardial infarction. J Am Soc Echocardiogr 2001;14:757–63.
34. Moller JE, Sondergaard E, Poulsen SH, Egstrup K. Pseudonormal and restrictive filling patterns predict left ventricular dilation and cardiac death after a first myocardial infarction: a serial color M-mode Doppler echocardiographic study. J Am Coll Cardiol 2000;36: 1841–6
35. Redfield MM, Rodeheffer RJ, Jacobsen SJ, Mahoney DW, Bailey KR, Burnett JC Jr. Plasma brain natriuretic peptide to detect preclinical ventricular systolic or diastolic dysfunction: a community-based study. Circulation 2004;109:3176–81.
36. Hansen A, Haass M, Zugck C, et al. Prognostic value of Doppler echocardiographic mitral inflow patterns: implications for risk stratification in patients with chronic congestive heart failure. J Am Coll Cardiol 2001;37:1049–55.
37. Traversi E, Pozzoli M, Cioffi G, et al. Mitral flow velocity changes after 6 months of optimized therapy provide important hemodynamic and prognostic information in patients with chronic heart failure. Am Heart J 1996;132:809–19.
38. Xie GY, Berk MR, Smith MD, Gurley JC, DeMaria AN. Prognostic value of Doppler transmitral flow patterns in patients with congestive heart failure. J Am Coll Cardiol 1994;24:132–9.
39. Bruch C, Gotzmann M, Stypmann J, et al. Electrocardiography and Doppler echocardiography for risk stratification in patients with chronic heart failure. J Am Coll Cardiol 2005;45:1073–5.
40. Klein AL, Hatle LK, Taliercio CP, et al. Prognostic significance of Doppler measures of diastolic function in cardiac amyloidosis. A Doppler echocardiography study. Circulation 1991;83:808–16.
41. Whaley GA, Doughty RN, Gamble GD, et al. Pseudonormal mitral filling pattern predicts hospital readmission in patients with congestive heart failure. J Am Coll Cardiol 2002;39:1787–95.
42. Tei C, Ling LH, Hodge DO, et al. New index of combined systolic and diastolic myocardial performance: a simple and reproducible measure of cardiac function—a study in normals and dilated cardiomyopathy. J Cardiol 1995;26:357–66.
43. Spencer KT, Kirkpatrick JN, Mor-Avi V, Decara JM, Lang RM. Age dependency of the Tei index of myocardial performance. J Am Soc Echocardiogr 2004;17:350–2.
44. Tei C, Nishimura RA, Seward JB, Tajik AJ. Noninvasive Doppler-derived myocardial performance index: correlation with simultaneous measurements of cardiac catheterization measurements. J Am Soc Echocardiogr 1997;10:169–78.
45. Yeo TC, Dujardin KS, Tei C, Mahoney DW, McGoon MD, Seward JB. Value of a Doppler-derived index combining systolic and diastolic time intervals in predicting outcome in primary pulmonary hypertension. Am J Cardiol 1998;81:1157–61.
46. Dujardin KS, Tei T, Yeo TC, Rossi AD, Seward JB. Prognostic value of a Doppler index combining systolic and diastolic performance in idiopathic-dilated cardiomyopathy Am J Cardiol 1998;82: 1071–6.
47. Arnlov J, Ingelsson E, Riserus U, Andren B, Lind L. Myocardial performance index, a Doppler-derived index of global left ventricular function, predicts congestive heart failure in elderly men. Eur Heart J 2004;25:2220–5.
48. Mikkelsen KV, Moller JE, Bie P, Ryde H, Videbaek L, Haghfelt T. Tei index and neurohormonal activation in patients with incident heart failure: serial changes and prognostic value. Eur J Heart Fail 2006;8:599–608.
49. Lang RM, Bierig M, Devereaux RB, et al. Recommendations for chamber quantification. J Am Soc Echocardiogr 2005;18:1440–63.
50. Malm S, Frigstad S, Sagberg E, Larsson H, Skjaerpe T. Accurate and reproducible measurement of left ventricular volume and ejection fraction by contrast echocardiography: a comparison with magnetic resonance imaging. J Am Coll Cardiol 2004;44:1030–5.
51. Hundley WG, Kizilbash AM, Afridi I, Franco F, Peshock RM, Grayburn PA. Administration of intravenous perfluorocarbon contrast agent improves echocardiographic determination of left ventricular volumes and ejection fraction: comparison with cine magnetic resonance imaging. J Am Coll Cardiol 1998;32:1426–32.
52. Yu EHC, Sloggett CE, Iwanochko RM, Rakowski H, Siu SC. Feasibility and accuracy of left ventricular volumes and ejection fraction determination by fundamental, tissue harmonic, and intravenous contrast imaging in difficult-to-image patients. J Am Soc Echocardiogr 2000;13:216–24.

53. Spencer KT, Bednarz J, Rafter PG, Korcarz C, Lang RM. Use of harmonic imaging without echocardiographic contrast to improve two-dimensional image quality. Am J Cardiol 1998;82:794–99.
54. Lang RM, Mor-Avi V, Zoghbi WA, Senior R, Klein AL, Pearlman AS. The role of contrast enhancement in the echocardiographic assessment of the left ventricle. Am J Cardiol 2002;90:28–34.
55. Bellenger NG, Burgess MI, Ray SG, et al. Comparison of left ventricular ejection fraction and volumes in heart failure by echocardiography, radionuclide ventriculography and cardiovascular magnetic resonance. Are they interchangeable? Eur Heart J 2000;21: 1387–96.
56. Jacobs LD, Salgo IS, Goonewardena S, et al. Rapid online quantification of left ventricular volume from real-time three-dimensional echocardiographic data. Eur Heart J 2006;27:460–8.
57. Cohen-Solaol A, Tabet JY, Logeart D, Bourgoin P, Tokmakova M, Dahan M. A non-invasively determined surrogate of cardiac power ('circulatory power') at peak exercise is a powerful prognostic factor in chronic heart failure. Eur Heart J 2002;23:806–14.
58. Smart N, Haluska B, Leano R, Case C, Mottram PM, Marwick TH. Determinants of functional capacity in patients with chronic heart failure: role of filling pressure and systolic and diastolic function. Am Heart J 2005;149:152–8.
59. Vasan RS, Larson MG, Benjamin EJ, Evans JC, Reiss CK, Levy D. Congestive heart failure in subjects with normal versus reduced left ventricular ejection fraction: prevalence and mortality in a population-based cohort. J Am Coll Cardiol 1999:33:1948–55.
60. Wong M, Stazewsky L, Latini R, et al. Severity of left ventricular remodeling defines outcomes and response to therapy in heart failure. J Am Coll Cardiol 2004;43:2022–7.
61. Grayburn PA, Appleton CP, DeMaria AN, et al., on behalf of the BEST trial Echocardiographic Substudy Investigators. Echocardiographic predictors of morbidity and mortality in patients with advanced heart failure. J Am Coll Cardiol 2005;45:1064–71.
62. Sanz G, Castaner A, Betriu A, et al. Determinants of prognosis in survivors of myocardial infarction: a prospective clinical angiographic study. N Engl J Med 1982;306:1065–70.
63. Multicenter Postinfarction Research Group. Risk stratification and survival after myocardial infarction. N Engl J Med 1983;309:331–6.
64. Ahnve S, Gilpin E, Henning H, Curtis G, Collins D, Ross J Jr. Limitations and advantages of the ejection fraction for defining high risk after acute myocardial infarction. Am J Cardiol 1986;58:872–8.
65. White HD, Norris RM, Brown MA, Brandt PWT, Whitlock RML, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation 1987;76:44–51.
66. Sutton MSJ, Pfeffer MA, Plappert T, et al., for the SAVE Investigators. Quantitative two-dimensional echocardiographic measurements are major predictors of adverse cardiovascular events after acute myocardial infarction. The protective effects of captopril. Circulation 1994;89:68–75.
67. Cleland JGF, Torabi A, Khan NK. Epidemiology and management of heart failure and left ventricular systolic dysfunction in the aftermath of a myocardial infarction. Heart. 2005;91 Suppl 2:ii7–13.
68. Burns RJ, Gibbons RJ, Yi Q, et al., CORE Study Investigators. The relationship of left ventricular ejection fraction, end-systolic volume index and infarct size to six-month mortality after hospital discharge following myocardial infarction treated by thrombolysis. J Am Coll Cardiol 2002;39:30–6.
69. Marchioli R, Avanzini F, Barzi F, et al., GISSI-Prevenzione Investigators. Assessment of absolute risk of death after myocardial infarction by use of multiple-risk-factor assessment equations: GISSI-Prevenzione mortality risk chart. Eur Heart J 2001;22:2085–103.
70. Cooper RS, Simmons BE, Castaner A, Santhanam V, Ghali J, Mar M. Left ventricular hypertrophy is associated with worse survival independent of ventricular function and number of coronary arteries severely narrowed. Am J Cardiol 1990;65:441–5.
71. Quinones MA, Breenberg BH, Kopelen HA, et al., for the SOLVD Investigators. Echocardiogaphic predictors of clinical outcomes in patients with left ventricular dysfunction enrolled in the SOLVD registry and trials: significance of left ventricular hypertrophy. J Am Coll Cardiol 2005;35:1237–44.
72. Yancy CW. Heart failure in African Americans. Am J Cardiol 2005;96 Suppl 2:3–12.
73. Park SH, Shub C, Nobrega TP, Bailey KR, Seward JB. Two-dimensional echocardiographic calculation of left ventricular mass as recommended by the American Society of Echocardiography: correlation with autopsy and M-mode echocardiography. J Am Soc Echocardiogr 1996;9:119–28.
74. Devereux RB, de Simone G, Ganau A, Roman MJ. Left ventricular hypertrophy and geometric remodeling in hypertension: stimuli, functional consequences and prognostic implications. J Hypertens 1994;12:S117–27.
75. Edner M, Bonarjee VV, Nilsen DW, Berning J, Carstensen S, Caidahl K. Effect of enalapril initiated early after acute myocardial infarction on heart failure parameters, with reference to clinical class and echocardiographic determinants. CONSENSUS II Multi-Echo Study Group. Clin Cardiol 1996;19:543–8.
76. Vantrimpont P, Rouleau JL, Wun CC, et al., for the SAVE Investigators. Additive beneficial effects of beta-blockers to angiotensin-converting enzyme inhibitors in the Survival and Ventricular Enlargement (SAVE) Study. J Am Coll Cardiol 1997;29: 229–36.
77. The MERIT-HF Study Group. Effect of metoprolol CR/XL in chronic heart failure: Metoprolol CR/XL randomized intervention trial in congestive heart failure (MERIT-HF). Lancet 1999;353: 2001–7.
78. Pitt B, Zannad F, Remme WJ, et al. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators. N Engl J Med 1999;341:709–17.
79. Pitt B, Remme W, Zannad F, et al. Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study Investigators. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. N Engl J Med 2003;348:1309–21.
80. Edner M, Bonarjee VV, Nilsen DW, Berning J, Carstensen S, Caidahl K. Effect of enalapril initiated early after acute myocardial infarction on heart failure parameters, with reference to clinical class and echocardiographic determinants. CONSENSUS II Multi-Echo Study Group. Clin Cardiol 1996;19:543–8.
81. The Australia/New Zealand Carvedilol Study Heart Failure Research Collaborative Group. Randomized, placebo-controlled trial of carvedilol in patients with congestive heart failure due to ischaemic heart disease. Lancet 1997;349:375–80.
82. Youssef G, Links M. The prevention and management of cardiovascular complications of chemotherapy in patients with cancer. Am J Cardiovasc Drugs 2005;5:233–43.
83. Tassan-Mangina S, Codorean D, Metivier M, et al. Tissue Doppler imaging and conventional echocardiography after anthracycline treatment in adults: early and late alterations of left ventricular function during a prospective study. Eur J Echocardiogr 2006;7:141–6.
84. Moss AJ, Zareba W, Hall WJ, et al., Multicenter Automatic Defibrillator Implantation Trial II Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med 2002;346:877–83.
85. Bardy GH, Lee KL, Mark DB, et al., Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) Investigators. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. N Engl J Med 2005;352:225–37.
86. Hohnloser S, Kuck K, Dorian P, et al., on behalf of the DINAMIT Investigators. Prophylactic use of an implantable cardioverter-defibrillator after myocardial infarction. N Engl J Med 2004;351: 2481–8.
87. Bristow MR, Saxon LA, Boehmer J, et al. Cardiac-resynchronization therapy with or without an implantable defibrillator in advanced chronic heart failure. N Engl J Med 2004;350:2140–50.
88. Achilli A, Sassara M, Ficili S, et al. Long-term effectiveness of cardiac resynchronization therapy in patients with refractory heart failure and "narrow" QRS. J Am Coll Cardiol 2003;42:2117–24.
89. Pitzalis MV, Iacoviello M, Romito R, et al. Cardiac resynchronization therapy tailored by echocardiographic evaluation of ventricular asynchrony. J Am Coll Cardiol 2002;40:1615–22.
90. Yu CM, Bleeker GB, Fung JW, et al. Left ventricular reverse remodeling but not clinical improvement predicts long-term survival after cardiac resynchronization therapy. Circulation 2005;112: 1580–6.

91. Stellbrink C, Breithardt OA, Franke A, et al. Impact of cardiac resynchronization therapy using hemodynamically optimized pacing on left ventricular remodeling in patients with congestive heart failure and ventricular conduction disturbances. J Am Coll Cardiol 2001;38: 1957–65.
92. Bax JJ, Ansalone G, Breithardt OA, et al. Echocardiographic evaluation of cardiac resynchronization therapy: ready for routine clinical use? A critical appraisal. J Am Coll Cardiol 2004;44:1–9.
93. Yu CM, Abraham WT, Bax J, et al., PROSPECT Investigators. Predictors of response to cardiac resynchronization therapy (PROSPECT)—study design. Am Heart J 2005;149:600–5.
94. Nesta F, Otsuji Y, Handschumacher MD, et al. Leaflet concavity: a rapid visual clue to the presence and mechanism of functional mitral regurgitation. J Am Soc Echocardiogr 2003;16:1301–8.
95. Kwan J, Shiota T, Agler DA, et al. Geometric differences of the mitral apparatus between ischemic and dilated cardiomyopathy with significant mitral regurgitation: real-time three-dimensional echocardiography study. Circulation 2003;107:1135–40.
96. Bolling SF, Pagani FD, Deeb GM, Bach DS. Intermediate-term outcome of mitral reconstruction in cardiomyopathy. J Thorac Cardiovasc Surg 1998;115:381–6.
97. Breithardt OA, Sinha AM, Schwammenthal E, et al. Acute effects of cardiac resynchronization therapy on functional mitral regurgitation in advanced systolic heart failure. J Am Coll Cardiol 2003;41:765–70.
98. Jimenez JH, Soerensen DD, He Z, He S, Yoganathan AP. Effects of a saddle shaped annulus on mitral valve function and chordal force distribution: an in vitro study. Ann Biomed Eng 2003;31:1171–81.
99. Khanna D, Miller AP, Nanda NC, Ahmed S, Lloyd SG. Transthoracic and transesophageal echocardiographic assessment of mitral regurgitation severity: usefulness of qualitative and semiquantitative techniques. Echocardiography 2005;22:748–69.
100. Quere JP, Tribouilloy C, Enriquez-Sarano M. Vena contracta width measurement: theoretic basis and usefulness in the assessment of valvular regurgitation severity. Curr Cardiol Rep 2003;5:110–5.
101. Athanasuleas CL, Stanley AW Jr., Buckberg GD, Dor V, Di Donato M, Silver W, RESTORE Group. Surgical anterior ventricular endocardial restoration (SAVER) for dilated ischemic cardiomyopathy. Semin Thorac Cardiovasc Surg 2001;13:448–58.
102. Suma H, Isomura T, Horii T, et al. Nontransplant cardiac surgery for end-stage cardiomyopathy. J Thorac Cardiovasc Surg 2000;119: 1233–44.
103. Wilhelm MJ, Hammel D, Schmid C, et al. Partial left ventriculectomy and mitral valve repair: favorable short-term results in carefully selected patients with advanced heart failure due to dilated cardiomyopathy. J Heart Lung Transplant 2005;24:1957–64.
104. McGee EC, Grady KL, McCarthy PM. Nontransplant surgical alternatives for heart failure. Curr Treat Options Cardiovasc Med 2005;7:491–501.
105. The STICH trial. Available at: http://www.stichtrial.org. Accessed December 3, 2006.
106. Athanasuleas CL, Buckberg GD, Stanley AWH, et al. Surgical ventricular restoration in the treatment of congestive heart failure due to post-infarction ventricular dilation. J Am Coll Cardiol 2004;44: 1439–45.
107. Yotsumoto G, Sakata R, Ueno T, et al. Late development of mitral regurgitation after left ventricular reconstruction surgery. Ann of Thorac Cardiovasc Surg 2005;11:159–63.
108. Dor V. The endoventricular circular patch plasty ("Dor procedure") in ischemic akinetic dilated ventricles. In: Heart Failure Reviews. Dordrecht: Kluwer Academic Publishers, 2001:87:187–93.
109. Cherniavsky AM, Karaskov AM, Marchenko AV, Mikova NV. Preoperative modeling of an optimal left ventricle volume for surgical treatment of ventricular aneurysms. Eur J Card Thorac Surg 2001; 20:777–82.
110. Rose EA, Gelijns AC, Moskowitz AJ, et al. Long-term mechanical left ventricular assistance for end-stage heart failure. N Engl J Med 2001;345:1435–43.
111. Shapiro GC, Leibowitz DW, Oz MC, Weslow RG, Di Tullio MR, Homma S. Diagnosis of patent foramen ovale with transesophageal echocardiography in a patient supported with a left ventricular assist device. J Heart Lung Transplant 1995;14:594–7.
112. Reilly MP, Wiegers SE, Cucchiara AJ, et al. Frequency, risk factors, and clinical outcomes of left ventricular assist device-associated ventricular thrombus. Am J Cardiol 2000;86:1156–9.
113. Horton SC, Khodaverdian R, Chatelain P, et al. Left ventricular assist device malfunction: an approach to diagnosis by echocardiography. J Am Coll Cardiol 2005;45:1435–40.
114. Gulati VK, Katz WE, Follansbee WP, et al. Mitral annular descent velocity by tissue Doppler echocardiography as an index of global left ventricular function. Am J Cardiol 1996;77:979–84.
115. Yip G, Wang M, Zhang Y, Fung JW, Ho PY, Sanderson JE. Left ventricular long axis function in diastolic heart failure is reduced in both diastole and systole: time for a redefinition? Heart 2002;87: 121–5.
116. Yu CM, Lin H, Yang H, Kong SL, Zhang Q, Lee SW. Progression of systolic abnormalities in patients with "isolated" diastolic heart failure and diastolic dysfunction. Circulation 2002;105:1195–201.
117. Wang M, Yip GW, Wang AY, et al. Peak early diastolic mitral annulus velocity by tissue Doppler imaging adds independent and incremental prognostic value. J Am Coll Cardiol 2003;41:820–6.
118. Troughton RW, Prior DL, Frampton CM, et al. Usefulness of tissue Doppler and color M-mode indexes of left ventricular diastolic function in predicting outcomes in systolic left ventricular heart failure (from the ADEPT study). Am J Cardiol 2005;96:257–62.
119. Okura H, Takada Y, Kubo T, et al. Tissue Doppler-derived index of left ventricular filling pressure, E/E', predicts survival of patients with non-valvular atrial fibrillation. Heart 2006;92:1248–52.
120. Sun JP, Popovic ZB, Greenberg NL, et al. Noninvasive quantification of regional myocardial function using Doppler-derived velocity, displacement, strain rate, and strain in healthy volunteers: effects of aging. J Am Soc Echocardiogr 2004;17:132–8.
121. Koyama J, Ray-Sequin PA, Falk RH. Longitudinal myocardial function assessed by tissue velocity, strain, and strain rate tissue Doppler echocardiography in patients with AL (primary) cardiac amyloidosis. Circulation 2003;107:2446–52.
122. Palka P, Lange A, Donnelly JE, Nihoyannopoulos P. Differentiation between restrictive cardiomyopathy and constrictive pericarditis by early diastolic Doppler myocardial velocity gradient at the posterior wall. Circulation 2000;102:655–62.
123. Bax JJ, Bleeker GB, Marwick TH, et al. Left ventricular dyssynchrony predicts response and prognosis after cardiac resynchronization therapy. J Am Coll Cardiol 2004;44:1834–40.
124. Bax JJ, Abraham T, Barold SS, et al. Cardiac resynchronization therapy: part 1—issues before device implantation. J Am Coll Cardiol 2005;46:2153–67.
125. Sogaard P, Egeblad H, Pedersen AK, et al. Sequential versus simultaneous biventricular resynchronization for severe heart failure evaluation by tissue Doppler imaging, Circulation 2002;106: 2078–84.
126. Helle-Valle T, Crosby J, Edvardsen T, et al. New noninvasive method for assessment of left ventricular rotation: speckle tracking echocardiography. Circulation 2005;112:3149–56.
127. Notomi Y, Lysyansky P, Setser RM, et al. Measurement of ventricular torsion by two-dimensional ultrasound speckle tracking imaging. J Am Coll Cardiol 2005;45:2034–41.
128. Knudtson ML, Galbraith PD, Hildebrand KL, Tyberg JV, Beyar R. Dynamics of left ventricular apex rotation during angioplasty: a sensitive index of ischemic dysfunction. Circulation 1997;96:801–8.
129. Stuber M, Scheidegger MB, Fischer SE, et al. Alterations in the local myocardial motion pattern in patients suffering from pressure overload due to aortic stenosis. Circulation 1999;100:361–8.
130. Vannan MA, Pedrizzetti G, Li P, et al. Effect of cardiac resynchronization therapy on longitudinal and circumferential left ventricular mechanics by velocity vector imaging: description and initial clinical application of a novel method using high-frame rate B-mode echocardiographic images. Echocardiography 2005;22:826–30.
131. Fuchs E, Muller MF, Oswald H, Thony H, Mohacsi P, Hess OM. Cardiac rotation and relaxation in patients with chronic heart failure. Eur J Heart Failure 2004;6:715–22.
132. Sugeng L, Weinert L, Lang RM. Left ventricular assessment using real time three dimensional echocardiography. Heart 2003;89 Suppl 3:iii29–36.
133. Jenkins C, Bricknell K, Hanekom L, Marwick TH. Reproducibility and accuracy of echocardiographic measurements of left ventricular

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parameters using real-time three-dimensional echocardiography. J Am Coll Cardiol 2004;44:878–86.
134. Sugeng L, Mor-Avi V, Weinert L, et al. Quantitative assessment of left ventricular size and function: side-by-side comparison of real-time three-dimensional echocardiography and computed tomography with magnetic resonance reference. Circulation 2006;114:654–61.
135. Corsi C, Lang RM, Veronesi F, et al. Volumetric quantification of global and regional left ventricular function from real-time three-dimensional echocardiographic images. Circulation 2005;112:1161–70.
136. Mor-Avi V, Sugeng L, Weinert L, et al. Fast measurement of left ventricular mass with real-time three-dimensional echocardiography: comparison with magnetic resonance imaging. Circulation 2004;110:1814–8.
137. Sugeng L, Spencer KT, Mor-Avi V, et al. Three-dimensional color flow Doppler: an improved technique for the assessment of mitral regurgitation.Echocardiography 2003;20:265–73.
138. Lang RM, Mor-Avi V, Sugeng L, Nieman PS, Sahn DJ. Three-dimensional echocardiography: the benefits of the additional dimension. J Am Coll Cardiol 2006;48:2053–69.
139. Senni M, Rodeheffer RJ, Tribouilloy CM, et al. Use of echocardiography in the management of congestive heart failure in the community. J Am Coll Cardiol 1999;33:164–70.
140. Kermani M, Dua A, Gradman AH. Underutilization and clinical benefits of angiotensin-converting enzyme inhibitors in patients with asymptomatic left ventricular dysfunction. Am J Cardiol 2000;86:644–8.
141. Get with the Guidelines, American Heart Association. Available at: http://www.americanheart.org/presenter.jhtml?identifier=3027533. Accessed December 3, 2006.
142. Guidelines Applied in Practice, American College of Cardiology. Available at: http://www.acc.org/qualityandscience/gap/or/oregon_gap.htm. Accessed August 23, 2006.