STATE-OF-THE-ART REVIEW ARTICLES

Methods for Assessment of Left Ventricular Systolic Function in Technically Difficult Patients with Poor Imaging Quality

Kai Hu, MD, Dan Liu, MD, Markus Niemann, MD, Sebastian Herrmann, MD, Philipp Daniel Gaudron, MD, Georg Ertl, MD, and Frank Weidemann, MD, *Würzburg, Germany*

The assessment of left ventricular (LV) systolic function is often the most important information obtained during clinical echocardiography. Although LV systolic function may be visually estimated in many patients with or without contrast opacification, technically difficult patients may require alternative methods for evaluating LV systolic function. In this review, the authors describe several surrogate echocardiographic methods that might be helpful for the evaluation of LV systolic function in patients with poor image quality, including endocardial border delineation by contrast agents, mitral annular plane systolic excursion, mitral annular velocity derived from tissue Doppler, systolic time intervals, mitral regurgitation–derived LV dP/dt, and estimation of cardiac output by Doppler echocardiography. After a short introduction to the various issues involved, the authors propose a method for suitable measurement. In addition, indications and clinical implications, as well as limitations, of the different methods are discussed. (J Am Soc Echocardiogr 2013;26:105-13.)

Keywords: Poor image quality, Contrast echocardiography, Mitral annular plane systolic excursion, Doppler echocardiography, Tei index

As one of the most widely used diagnostic examinations in cardiology, echocardiography has been routinely used for diagnosing and monitoring patients with suspected or known cardiovascular disease. In daily clinical echocardiography, left ventricular (LV) systolic function can be correctly assessed using various echocardiographic imaging methods in the majority of patients. However, the assessment of LV systolic function remains a challenge in a small proportion of patients with poor image quality, caused mainly by obesity, lung disease, tachycardia, or cardiac translocation. The quantitative assessment of ventricular function in these patients is still difficult despite the use of transducers with variable frequencies and harmonic imaging as well as the application of various advanced echocardiographic techniques, such as strain rate imaging, speckle-tracking imaging, and three-dimensional echocardiography. Besides poor image quality, there are other situations that make the determi-

nation of LV systolic function difficult, such as in patients with atrial fibrillation, LV hypertrophy, or mitral regurgitation. The purpose of this review is to summarize the clinical applications and limitations of several echocardiographic methods that can be used to evaluate LV systolic function in patients with poor image quality.

CONTRAST ECHOCARDIOGRAPHY

Contrast echocardiography using ultrasound contrast agents plays an essential role in clinical diagnosis in patients with technically suboptimal echocardiographic images.[1-3]

Contrast Agents

Contrast echocardiography involves the interaction of microscopic gas bubbles with ultrasonic waves to enhance the recognition of the blood pool and/or the blood-tissue interface. The first agents capable of left-heart contrast after intravenous injection (first-generation agents) were air bubbles stabilized by encapsulation (Albunex; Molecular Biosystems, Inc., San Diego, CA) or by adherence to microparticles (Levovist; Bayer Schering Pharma AG, Berlin, Germany). In second-generation agents, replacing air with a low-solubility fluorocarbon gas stabilized the bubbles (Optison, GE Healthcare, Waukesha, WI; Definity, Lantheus Medical Imaging, North Billerica, MA; SonoVue, Bracco Diagnostics Inc., Princeton, NJ), further increasing the duration of the contrast effect. The aforementioned agents are untargeted microbubbles, and targeted microbubbles are presently in preclinical development.

Implementation of Contrast Agents

Details of the implementation of contrast agent, including joint training of physicians, sonographers, and nurses, have been introduced in recent guidelines.[4] Briefly, contrast enhancement is indicated in difficult-to-image patients at rest when echocardiographic image

http://dx.doi.org/10.1016/j.echo.2012.11.004

quality does not permit the adequate assessment of cardiac structure and function. Specifically, contrast enhancement for stress echocardiography is not recommended for all patients but should be considered on a case-by-case basis, depending on image quality.[5] To ensure quality control and maximize benefit to patients, the American Society of Echocardiography recommends that contrast echocardiography be performed by appropriately trained cardiac sonographers and physicians with level 2 or level 3 training in laboratories that have been successful in establishing contrast agent use.[4]

Optimization and Clinical Applications

The mechanical index reflects the output acoustic power. Standard clinical echocardiography imaging uses a mechanical index of about 1.0, but a lower setting (<0.6) is usually optimal for LV opacification during contrast echocardiography to avoid bubble destruction.[6] Common causes of setting artifacts include inadequate focus position, inadequate ultrasound transmit frequency, and excessive receive gain.[7] Tissue signals in the left ventricle may not be distinguishable from the contrast signals, because of inadequate contrast dose (the so-called anticontrast effect) and can be avoided by injecting a slightly larger contrast dose.[7] Attenuation is particularly problematic in parasternal windows, in which dense opacification of the right ventricle may obviate visualization of the left ventricle, and can be prevented by using apical views, in which attenuation is lowest and usually subsides by waiting for contrast washout. Attenuation can also be caused by rapid infusion or high-concentration contrast agent. Instead of a bolus, continuous slow infusion and slow flush are recommended.[7,8] Swirling artifacts may result from high mechanical index, high frame rate, insufficient contrast agent, or LV dysfunction with low flow at the apex.[7] Moving the focus position toward the base may help avoid attenuation and swirling. Adjusting the transducer position along the rib space or holding respiration during image acquisition can help reduce chest wall artifacts and wall motion artifacts.[7,8]

Contrast agent use is particularly valuable for the evaluation of LV structure and function in difficult-to-image patients with reduced image quality for rest or stress echocardiography. It can improve endocardial visualization and the assessment of LV structure and function and reduce variability and increase accuracy in LV volume and ejection fraction (EF) measurement.[9-11] Contrast agent is recommended when two or more adjacent poorly visualized segments are seen on standard echocardiography.[4] Contrast agent use also allows accurate assessment of LV volumes and EF in the intensive care unit when standard tissue harmonic imaging does not provide adequate cardiac structural definition.[10] Stress echocardiography, in combination with contrast agent use, can obtain diagnostic assessment of segmental motion and thickening at rest and stress.[11,12] Other suggested applications include confirming or excluding LV structural abnormalities (apical hypertrophic cardiomyopathy, LV noncompaction, LV aneurysm [Figure 1 and Video 1; available at www.onlinejase.com], pseudoaneurysm, and myocardial rupture) and intracardiac masses (tumors and/or thrombi).[6,13,14]

Safety and Limitations

A large number of studies have proved that contrast echocardiography is safe in clinical practice.[15-18] A large retrospective analysis of 18,000 patients showed that there was no significant difference in mortality between patients who received contrast and those who did not in the acute setting.[16] However, serious allergic reactions have been observed at a very low incidence (1 in 12,000 to 1 in 15,000).[16,17] As shown in the updated guidelines on the safety of echocardiographic contrast agents of the US Food and Drug Administration in June 2008,[4] contraindications to perflutren-containing ultrasound contrast agents (Definity and Optison) include (1) right-to-left, bidirectional, or transient right-to-left cardiac shunts; (2) hypersensitivity to perflutren; and (3) hypersensitivity to blood, blood products, or albumin (Optison only). Additional contraindications include acute myocardial infarction, worsening or unstable heart failure, serious ventricular arrhythmias or high risk for arrhythmia, respiratory failure, severe emphysema, pulmonary emboli, or other conditions that cause pulmonary hypertension.

MITRAL ANNULAR PLANE SYSTOLIC EXCURSION

LV longitudinal shortening is a sensitive parameter reflecting cardiac pump function[19,20] and can be evaluated by measuring long-axis mitral annular plane systolic excursion (MAPSE).[21] The measurement of M-mode-derived MAPSE does not require high imaging quality, because of the high echogenicity in the atrioventricular annulus.

Measurement

MAPSE can be measured from four sites of the atrioventricular plane corresponding to the septal, lateral, anterior, and posterior walls using the apical four-chamber and two-chamber views on M-mode echocardiography. In healthy hearts, the values of lateral MAPSE are usually somewhat higher than those of septal MAPSE.[22] Mondillo *et al.*[23] also demonstrated that MAPSE was lower at the septum and anterior wall in comparison with the lateral and inferior levels in healthy middle-aged individuals. The M-mode cursor should be aligned parallel to the LV walls. The systolic excursion of the mitral annulus should be measured from the lowest point at end-diastole to aortic valve closure (the end of the T wave on the electrocardiogram; Figure 2).

Clinical Implications

The average normal value of MAPSE derived from previous studies for the four annular regions (septal, anterior, lateral, and posterior) ranges from 12 to 15 mm.[21,23,24] MAPSE < 8 mm was associated with a depressed LV EF (<50%), with specificity of 82% and sensitivity of 98%.[21] Mean MAPSE ≥ 10 mm was linked with preserved EF (≥55%), with sensitivity of 90% to 92% and specificity of 87%.[25,26] In addition, mean MAPSE < 7 mm could detect an EF < 30% with sensitivity of 92% and specificity of 67% in patients with dilated cardiomyopathy with severe congestive heart failure.[24] A recent study by Matos *et al.*[27] showed that MAPSE measurement by an untrained observer was a highly accurate predictor of EF determined by an expert echocardiographer.

Limitations

The association between MAPSE and EF is valid only in normal or dilated left ventricles,[28,29] whereas the correlation is rather poor in patients with LV hypertrophy.[30] Another limitation of this parameter is that small localized abnormalities (i.e., small areas of fibrosis) cannot be detected, because MAPSE can evaluate only the longitudinal function of the entire LV wall and is unable to evaluate segmental function.
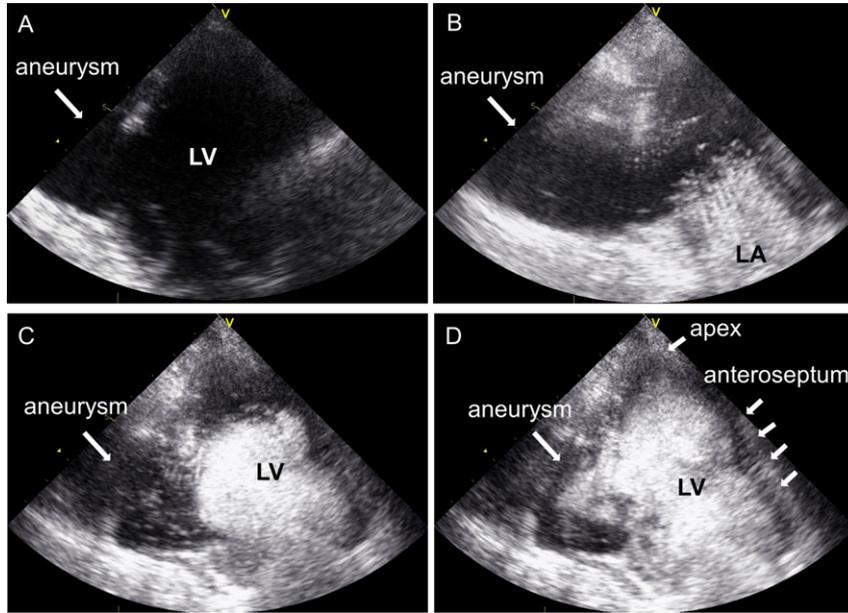
Figure 1 Noncontrast **(A)** and contrast **(B–D)** images of a giant left ventricular aneurysm at the posterior wall as a complication of prior myocardial infarction. The vague left ventricular apex and anteroseptum **(A)** became clear after contrast administration **(D)**. The contrast agent entered the left atrium (LA) **(B)**, left ventricle (LV) **(C)**, and then the left ventricular aneurysm cavity **(D)**.
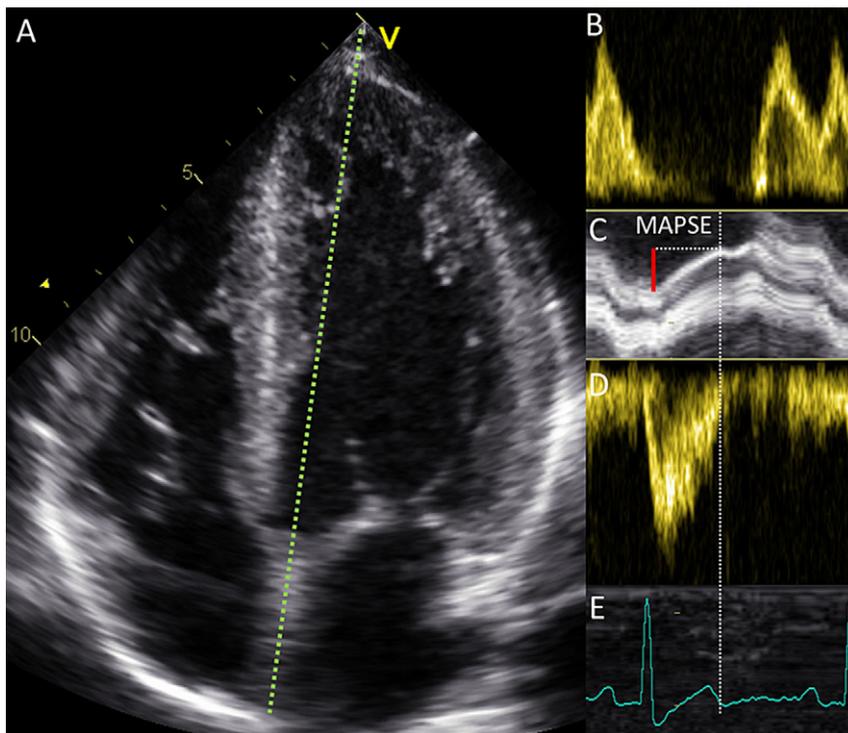

Figure 2 MAPSE measurement. **(A)** MAPSE is measured by M-mode echocardiography in the apical four-chamber view. **(B)** Pulsed Doppler recording of mitral inflow. **(C)** MAPSE. **(D)** Pulsed Doppler recording of aortic outflow. **(E)** Electrocardiogram. MAPSE should be measured from the lowest point to the highest point during systole.

MITRAL ANNULAR VELOCITY DERIVED FROM TISSUE DOPPLER

Doppler tissue imaging (DTI) has become an established component of the diagnostic ultrasound examination. This technique detects low-velocity frequency shifts of ultrasound waves to calculate myocardial velocity. DTI offers the promise of an objective measure to quantify regional and global LV function through the assessment of myocardial velocity data. The velocity traces can be extracted from the basal segments of the left ventricle in most patients, even when the overall image quality is bad. The decrease in systolic velocity on pulsed DTI correlated significantly with both

systolic shortening ($r = 0.90$) and regional myocardial blood flow ($r = 0.96$) in patients with reduced coronary blood flow.[31,32]

Measurement

The measurements of systolic mitral annular velocity (Sm) should be taken at the peak of myocardial systolic velocity, in accordance with recent guidelines.[33] In the apical four-chamber view, the DTI cursor should be placed at the septal side of the mitral annulus in such a way that the mitral annulus at the septum moved along the sample volume line. In normal myocardium, a Doppler velocity range of -20 to 20 cm/sec is recommended to avoid aliasing. As shown in Figure 3, three major velocities can be recorded: the positive systolic velocity when the mitral ring moves toward the apex (Sm) and two negative diastolic velocities when the mitral annulus moves away from the apex (one during the early phase of diastole [Em] and another in the late phase of diastole [Am]). By moving the sample volume to the lateral site of the mitral annulus, systolic and diastolic velocities of the LV lateral wall can be recorded. These velocities can be extracted by pulsed-wave tissue Doppler and in addition also by color tissue Doppler. It is important to note that color tissue Doppler data are mean data, and extracted velocities are approximately 20% lower than the pulsed-wave tissue Doppler velocities. Pulsed tissue Doppler–derived Sm measurement is more often used in daily practice. Pulsed tissue Doppler–derived Sm was significantly lower at the septum (8.3 ± 1.7 cm/sec) than at the inferior (9.5 ± 1.9 cm/sec) and lateral (9.9 ± 2.4 cm/sec) levels in healthy middle-aged individuals.[23]

Clinical Implications

Tissue Doppler data can be rapidly acquired in almost all patients for the estimation of global LV function.[32] Mitral annular velocity is also a quite sensitive indicator for inotropic stimulation–induced alterations in LV contractility.[34] LV function assessment by mitral annular velocity on DTI is valuable especially when endocardial delineation is suboptimal.[35] Ruan and Nagueh[36] showed that Sm had the best correlation with LV EF ($r = 0.65$, $P < .03$), and Sm < 7 cm/sec was the most accurate parameter in identifying patients with LV EFs $< 45\%$ (sensitivity, 93%; specificity, 87%).[36] Sm was also a strong predictor of cardiac mortality or rehospitalization for worsening of chronic heart failure in patients with LV dysfunction.[37] Nikitin et al.[38] found that DTI-derived Sm < 2.8 cm/sec was associated with worse survival in patients with chronic heart failure and LV EFs $< 45\%$.

Limitations

DTI only quantifies myocardial motion. It cannot differentiate whether velocities are caused by active or passive movement, so global cardiac motion and tethering effects of adjacent myocardium may result in "false" velocity increases of dysfunctional segments.

SYSTOLIC TIME INTERVALS

Systolic time intervals can provide useful information concerning the performance of the left ventricle.[39-41] Thus, isovolumetric contraction time, preejection period (PEP), LV ejection time (ET), and the PEP/LV ET index have been studied extensively as measures of cardiac systolic function.[42] More recently, a combined myocardial performance index (termed the Tei index, the sum of isovolumetric contraction time and isovolumetric relaxation time divided by LV ET) was introduced and serves as a clinically useful parameter for analyzing global cardiac function. The longer the isovolumetric phases, the higher the Tei index and the worse the ventricular perfor-
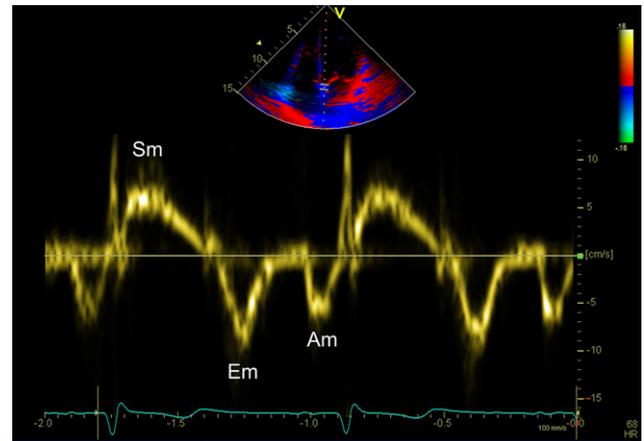

Figure 3 Mitral annular velocity derived from TDI. In the apical four-chamber view, the DTI cursor is placed at the septal side of the mitral annulus in such a way that the mitral annulus at the septum moves along the sample volume line. Three major peak velocities can be recorded: the positive systolic velocity (Sm) when the mitral ring moves toward the apex and two negative diastolic velocities when the mitral annulus moves away from the apex (one during the early phase of diastole [Em] and another in the late phase of diastole [Am]).

mance. The index is easily measured, noninvasive, and reproducible and is rather independent of heart rate and systolic or diastolic blood pressure.[43] It can be used for LV and right ventricular function even when image quality is poor.[44-49]

Measurement

The PEP consist of electromechanical delay and the isovolumetric contraction time, which is the time interval between the start of ventricular depolarization and the moment of aortic valve opening. The LV ET is defined as the time interval of LV ejection, which occurs between the opening of the aortic valve and its closure. A healthy heart exhibits a short PEP and a long ET, while myocardial dysfunction is typically evidenced by prolonged PEP and shortened LV ET. The Tei index can be measured by pulsed Doppler as well as by DTI. The Tei index can be easily derived using conventional pulsed Doppler echocardiography, as described by Tei et al.[50] The mitral inflow velocity is recorded from apical four-chamber views, with the sample volume positioned at the tips of the mitral leaflets during diastole. The LV outflow tract (LVOT) velocity is recorded from apical long-axis views with the sample volume positioned just below the aortic valve. The index is defined by the equation $(a-b)/b$, where a represents the interval between the cessation and onset of mitral inflow, and b represents the ET of the LV outflow (Figure 4A). DTI has also been used to calculate the Tei index, which correlates well with pulsed Doppler measurements.[51,52] All interval measurements by DTI can be performed within one cardiac cycle, as illustrated in Figure 4B. The Tei index is calculated as $(a'-b')/b'$, where a' represents the interval from the end of Am wave to the onset of the Em wave, and b' represents the time from the onset to the end of the Sm wave. The mean normal values of the Tei index for the left ventricle by the pulsed Doppler and DTI methods are 0.39 ± 0.05 and 0.35 ± 0.09, respectively.[50,53] In adults, LV indexes < 0.40 are considered normal.

Clinical Implications

The Tei index appears to have close correlation with the widely accepted systolic and diastolic hemodynamic parameters[54] as well as
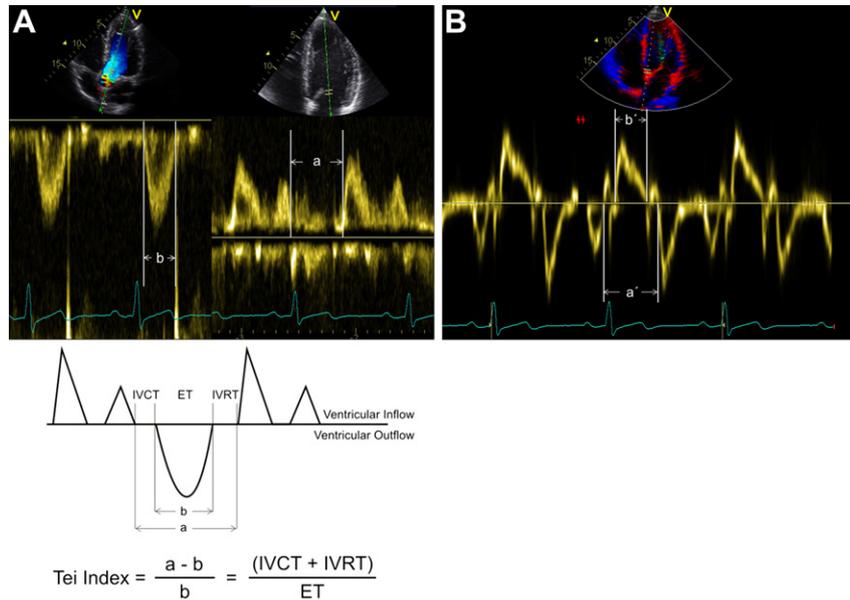
$$\text{Tei Index} = \frac{a - b}{b} = \frac{(\text{IVCT} + \text{IVRT})}{\text{ET}}$$

Figure 4 **(A)** Measurement of Tei index using pulsed-wave Doppler echocardiography. a = interval between cessation and onset of mitral or tricuspid inflow, which is the sum of isovolumetric contraction time (IVCT), ET, and isovolumetric relaxation time (IVRT); b = LV or right ventricular ejection time. **(B)** Measurement of Tei index using tissue Doppler echocardiography. The Tei index is calculated as $(a'-b')/b'$, where a' represents the interval from the end of the Am wave to the onset of the Em wave, and b' represents the time from the onset to the end of the Sm wave.

potential for clinical application in the assessment of overall cardiac performance.[55] The index has been proposed as a useful method for the evaluation of global LV performance in congestive heart failure,[56,57] congenital heart diseases,[58] and valvular heart disease[59,60] and for monitoring interventional therapies.[61] Furthermore, the Tei index has been shown to have strong prognostic value in severe cardiac diseases, such as dilated cardiomyopathy,[62] cardiac amyloidosis,[44] and myocardial infarction.[63] Patients with cardiac amyloidosis with higher Tei indexes (>0.77) have a higher overall and cardiac-related mortality risk than patients with lower values (≤0.77).[44] A prognostic study by Sasao *et al.*[63] suggested that a Tei index > 0.70 was the only significant explanatory factor for cardiac death or developing congestive heart failure in patients with acute myocardial infarction after successful primary angioplasty. Møller *et al.*[64] investigated 799 patients with acute myocardial infarctions and followed them for a median of 34 months, finding that patients with Tei indexes > 0.68 had a fourfold increase in mortality compared with those with Tei indexes < 0.46.

Limitations

Data from large-scale epidemiologic studies regarding the application and the clinical impact of the Tei index are lacking. One major limitation of the pulsed Doppler Tei index is that both relaxation and contraction velocities cannot be measured simultaneously within one cardiac cycle, whereas DTI enables the measurement of both relaxation and contraction velocities simultaneously.[52] Pulsed Doppler Tei index is therefore not feasible in patients with variable beat-to-beat duration, as in atrial fibrillation, frequent supraventricular and ventricular extrasystole, atrioventricular conduction abnormalities, and significant atrial tachycardia.[65] In addition, the Tei index should not be used in patients with ventricular pacing and in those with intraventricular dyssynchrony.[65]

Estimation of Left Ventricular dP/dt

The rate of pressure rise in the ventricles (dP/dt) is a good index of ventricular performance that is sensitive to changes in contractility and insensitive to changes in afterload but can be mildly affected by changes in preload.[66,67] The clinical utility of dP/dt has been limited, however, by the fact that its measurement conventionally requires the insertion of an intraventricular catheter. The estimated mean rate of pressure change (dP/dt) in preejection systole from the mitral regurgitation continuous-wave Doppler tracing has been used as a nongeometric index of LV systolic function.[68] A number of studies have confirmed a good correlation between Doppler-derived noninvasive LV dP/dt and catheter-derived invasive dP/dt.[69-71]

Measurement

Continuous-wave Doppler tracings of mitral regurgitation velocity curves are needed for the measurement of LV dP/dt. The flow direction of the mitral regurgitation must be detected by color Doppler imaging using an apical window in which the sample volume should be positioned correctly with minimal angulation at the level of the annulus. It is important to note that in eccentric significant mitral regurgitation, it may be difficult to record the full envelope of the jet because of its eccentricity. Mean LV dP/dt is commonly calculated by measuring the time required for the mitral regurgitation jet to increase in velocity from 1 to 3 m/sec during the isovolumetric contraction time, which reflects a 32 mm Hg change during this time period between the left ventricle and the left atrium, derived from the modified Bernoulli equation (Figure 5). The average normal value of mean LV dP/dt from the Doppler regurgitant velocity spectrum is >1,200 mm Hg/sec (<27 msec). A value < 1,000 mm Hg/sec (>32 msec) was associated with reduced LV function.[72]

Clinical Implications

LV dP/dt is less influenced by afterload, wall motion abnormalities, or the variations in ventricular anatomy and morphology commonly encountered in patients with congenital heart disease.[63,64] It is more
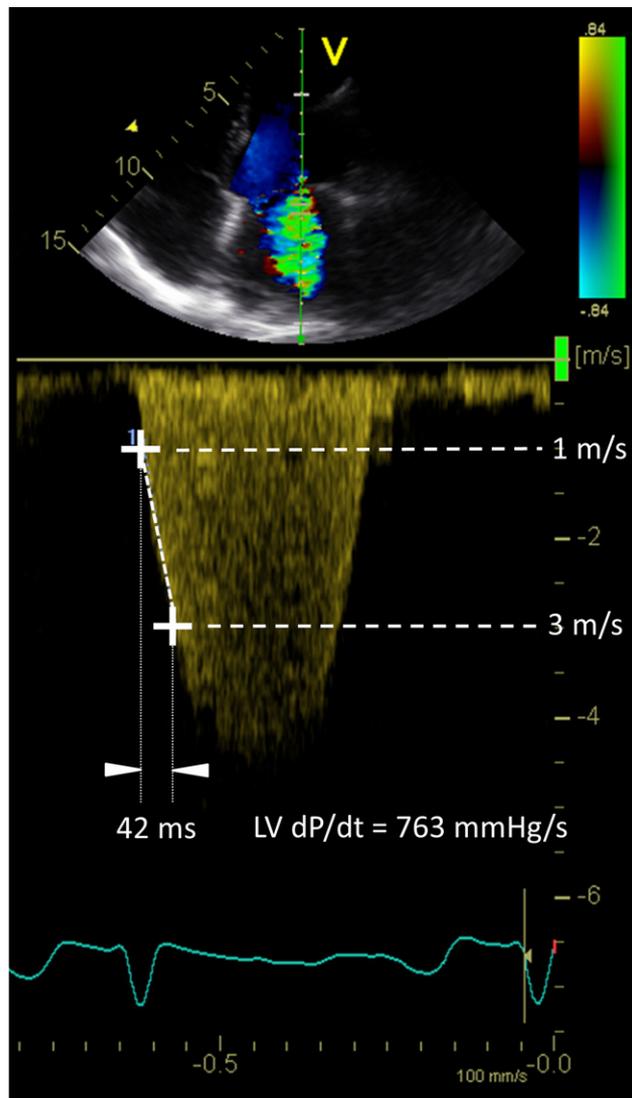
Figure 5 Measurement of the rate of pressure rise (dP/dt) in the left ventricle. Continuous-wave Doppler recordings of the mitral regurgitation (MR) velocity curve is obtained from an apical view. LV dP/dt is calculated by measuring the time required for the MR jet to increase in velocity from 1 to 3 m/sec, which reflects a 32 mm Hg change during this time period between the left ventricle and the left atrium, derived from the modified Bernoulli equation.

related to myocardial contractility than chamber performance. LV dP/dt can be used to determine alterations in myocardial contractility in patients with congestive heart failure, and mitral regurgitation can be detected in the majority of patients with congestive heart failure.[71] The major advantage of this Doppler method is that it provides a noninvasive estimation of LV dP/dt on a beat-by-beat basis, which makes it ideal for serial measurements in patients with congestive heart failure with mitral regurgitation.[70,71] Kolias *et al.*[73] found in patients with chronic congestive heart failure with reduced LV EFs that LV Doppler-derived dP/dt < 600 mm Hg/sec was associated with lower event-free survival compared with dP/dt ≥ 600 mm Hg/sec.

Limitations

A complete, well-delineated velocity spectral envelope from mitral regurgitation is mandatory for accurate measurements of instantaneous

pressure gradients. As with any Doppler velocity measurement, the ultrasound beam should be aligned parallel to the velocity vectors of the mitral regurgitation flow to prevent underestimation of the pressure gradients. Careful scanning is necessary to obtain maximal velocity spectra. Additionally, the full envelope of the mitral regurgitation jet is sometimes difficult to obtain in eccentric significant mitral regurgitation, which may limit the use of measuring LV dP/dt in patients with eccentric mitral regurgitation.

ESTIMATION OF CARDIAC OUTPUT BY DOPPLER ECHOCARDIOGRAPHY

The Doppler technique enables the noninvasive determination of LV stroke volume (SV) and cardiac output (CO), which might be useful in identifying and projecting trends for cardiac function. Doppler-estimated CO correlates well with invasive estimation using a thermodilution pulmonary artery catheter.[74-76]

Measurement

SV and CO are calculated as follows (Figure 6): CO (L/min) = SV (mL) × heart rate (beats/min), where SV (mL) = cross-sectional area (cm^2) × velocity-time integral (cm). The velocity-time integral is obtained by tracing the envelope of the peak velocity detected throughout systole. The cross-sectional area of the region of the heart from which the blood flow velocity was extracted is calculated from its diameter (area = πr^2). The preferred site for determining SV and CO is the LVOT.[33,77] LVOT diameter is measured in the parasternal long-axis view in midsystole from the white-black interface of the septal endocardium to the anterior mitral leaflet, parallel to the aortic valve plane and within 0.5 to 1.0 cm of the valve orifice. LVOT velocity is recorded with pulsed Doppler, because the blood flow velocity in the LVOT needs to be extracted to obtain the velocity-time integral, whereas accurate estimates of the location of the source of a Doppler shift in a patient are difficult to achieve with continuous-wave Doppler. The largest of three to five measurements should be taken, because the inherent error of the tomographic plane is to underestimate the LVOT diameter. The normal range of CO is 4.0 to 8.0 L/min. It is important to note that the correct measurement of LVOT is essential for correct CO determination.[78,79] In some cases, variations in cardiac anatomy and orientation may not permit parallel alignment of the Doppler beam with the LVOT in the proposed plane, which can result in underestimation of the true CO.[80]

Clinical Implications

For patients in the intensive care unit, the ultrasonic determination of CO is likely to be supplementary rather than a replacement for invasive methods. It may be used to screen potential candidates for invasive monitoring. Because the technique is noninvasive, measurements can be repeated as often as necessary, making it suitable for studying serial CO changes in individual subjects.[80,81] Other areas of possible application include early hemodynamic monitoring in patients after myocardial infarction or with decompensated heart failure and intraoperative hemodynamic monitoring in noncardiac operations.[82]

Limitations

In the presence of any LVOT obstruction, high flow velocities caused by flow acceleration through the LVOT or narrowing will lead to
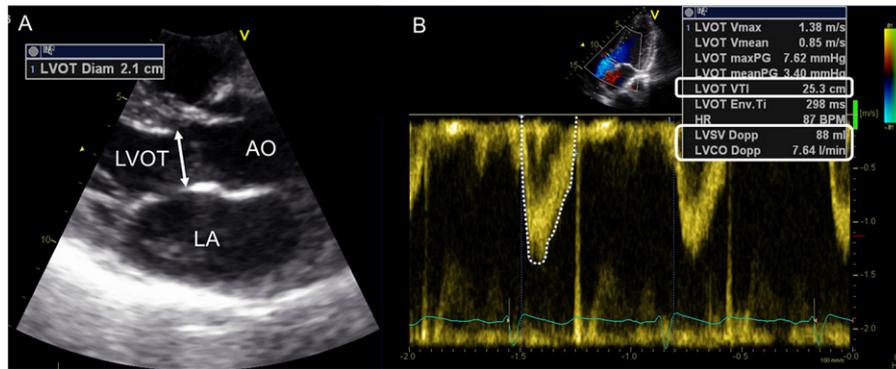
Figure 6 Measurement of LV CO using pulsed-wave Doppler echocardiography. **(A)** LVOT diameter. The cross-sectional area (CSA) is calculated as $\pi \times$ (LVOT diameter/2)^2. **(B)** Pulsed-wave Doppler recording of LVOT velocity obtained from the apical five-chamber view. The velocity-time integral (VTI) is obtained by tracing the envelope of LVOT flow. SV (mL) = CSA (cm^2) $\times$ VTI (cm), and CO (L/min) = SV (mL) $\times$ heart rate (beats/min). *AO*, Aortic root; *LA*, left atrium.

overestimation of CO. Additionally, it should be noted that SV at rest may be normal or even increased in dilated hearts with impaired systolic function. Thus, SV may not accurately reflect overall systolic LV function in patients with dilated hearts.

CONCLUSIONS

In general, most of the methods described here are either dependent on movement of the mitral annulus from the base toward the apex of the left ventricle (MAPSE and Sm) or based on Doppler methods that rely on ultrasound scattering rather than specular reflection from targets that are (optimally) oriented perpendicular to the direction of the ultrasound beam. These methods take advantage of recording motion relative to the apex (along the axis of the ultrasound beam), which is still feasible even when B-mode image quality is not good. Contrast echocardiography is not only based on the scattering of incident ultrasound at a gas-liquid interface to increase the strength of returning signal but also takes advantage of microbubble oscillations that can be detected using harmonic imaging.

The methods described here are useful for evaluating LV systolic function in patients with unsatisfactory image quality. Contrast echocardiography is certainly the best method for defining LV systolic function, but this method requires appropriately trained cardiac sonographers and physicians in laboratories that have been successful in establishing contrast agent use. MAPSE is a simple method and can assess longitudinal function of the complete LV walls, but it cannot detect small localized myocardial abnormalities. Other methods (Tei index, pulsed-wave Doppler–derived CO, dP/dt, DTI) are based on Doppler techniques and can be used to asses LV systolic function even in case of poor endocardial delineation. It is essential for echocardiographers to be aware of the merits and limitations of individual methods when choosing practical techniques for individual patients to achieve the optimal measurement of LV systolic function in daily practice.

REFERENCES

1. Crouse LJ, Cheirif J, Hanly DE, Kisslo JA, Labovitz AJ, Raichlen JS, et al. Opacification and border delineation improvement in patients with suboptimal endocardial border definition in routine echocardiography: results of the phase III Albunex Multicenter Trial. J Am Coll Cardiol 1993;22:1494-500.

2. Cohen JL, Cheirif J, Segar DS, Gillam LD, Gottdiener JS, Hausnerova E, et al. Improved left ventricular endocardial border delineation and opacification with Optison (FS069), a new echocardiographic contrast agent. Results of a phase III multicenter trial. J Am Coll Cardiol 1998;32:746-52.

3. Allen MR, Pellikka PA, Villarraga HR, Klarich KW, Foley DA, Mulvagh SL, et al. Harmonic imaging: echocardiographic enhanced contrast intensity and duration. Int J Card Imaging 1999;15:215-20.

4. Mulvagh SL, Rakowski H, Vannan MA, Abdelmoneim SS, Becher H, Bierig SM, et al. American Society of Echocardiography consensus statement on the clinical applications of ultrasonic contrast agents in echocardiography. J Am Soc Echocardiogr 2008;21:1179-201.

5. Plana JC, Mikati IA, Dokainish H, Lakkis N, Abukhalil J, Davis R, et al. A randomized cross-over study for evaluation of the effect of image optimization with contrast on the diagnostic accuracy of dobutamine echocardiography in coronary artery disease. The OPTIMIZE trial. JACC Cardiovasc Imaging 2008;1:145-52.

6. Stewart MJ. Contrast echocardiography. Heart 2003;89:342-8.

7. Zamorano JL, Fernández MAG. Contrast echocardiography in clinical practice. Berlin, Germany: Springer Verlag; 2004.

8. Burgess P, Moore V, Bednarz J, Carney D, Floer S, Gresser C, et al. Performing an echocardiographic examination with a contrast agent: a series on contrast echocardiography, article 2. J Am Soc Echocardiogr 2000;13:629-34.

9. Reilly JP, Tunick PA, Timmermans RJ, Stein B, Rosenzweig BP, Kronzon I. Contrast echocardiography clarifies uninterpretable wall motion in intensive care unit patients. J Am Coll Cardiol 2000;35:485-90.

10. Yu EH, 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.

11. Hoffmann R, von Bardeleben S, ten Cate F, Borges AC, Kasprzak J, Firschke C, et al. Assessment of systolic left ventricular function: a multicentre comparison of cineventriculography, cardiac magnetic resonance imaging, unenhanced and contrast-enhanced echocardiography. Eur Heart J 2005;26:607-16.

12. Rainbird AJ, Mulvagh SL, Oh JK, McCully RB, Klarich KW, Shub C, et al. Contrast dobutamine stress echocardiography: clinical practice assessment in 300 consecutive patients. J Am Soc Echocardiogr 2001;14:378-85.

13. Thanigaraj S, Schechtman KB, Perez JE. Improved echocardiographic delineation of left ventricular thrombus with the use of intravenous second-generation contrast image enhancement. J Am Soc Echocardiogr 1999;12:1022-6.

14. Kirkpatrick JN, Wong T, Bednarz JE, Spencer KT, Sugeng L, Ward RP, et al. Differential diagnosis of cardiac masses using contrast echocardiographic perfusion imaging. J Am Coll Cardiol 2004;43:1412-9.

15. Anantharam B, Chahal N, Chelliah R, Ramzy I, Gani F, Senior R. Safety of contrast in stress echocardiography in stable patients and in patients with suspected acute coronary syndrome but negative 12-hour troponin. Am J Cardiol 2009;104:14-8.

16. Kusnetzky LL, Khalid A, Khumri TM, Moe TG, Jones PG, Main ML. Acute mortality in hospitalized patients undergoing echocardiography with and without an ultrasound contrast agent: results in 18,671 consecutive studies. J Am Coll Cardiol 2008;51:1704-6.

17. Wei K, Mulvagh SL, Carson L, Davidoff R, Gabriel R, Grimm RA, et al. The safety of Definity and Optison for ultrasound image enhancement: a retrospective analysis of 78,383 administered contrast doses. J Am Soc Echocardiogr 2008;21:1202-6.

18. Dolan MS, Gala SS, Dodla S, Abdelmoneim SS, Xie F, Cloutier D, et al. Safety and efficacy of commercially available ultrasound contrast agents for rest and stress echocardiography: a multicenter experience. J Am Coll Cardiol 2009;53:32-8.

19. Carlsson M, Ugander M, Mosen H, Buhre T, Arheden H. Atrioventricular plane displacement is the major contributor to left ventricular pumping in healthy adults, athletes, and patients with dilated cardiomyopathy. Am J Physiol Heart Circ Physiol 2007;292:H1452-9.

20. Jones CJ, Raposo L, Gibson DG. Functional importance of the long axis dynamics of the human left ventricle. Br Heart J 1990;63:215-20.

21. Simonson JS, Schiller NB. Descent of the base of the left ventricle: an echocardiographic index of left ventricular function. J Am Soc Echocardiogr 1989;2:25-35.

22. Carlhäll C, Wigström L, Heiberg E, Karlsson M, Bolger AF, Nylander E. Contribution of mitral annular excursion and shape dynamics to total left ventricular volume change. Am J Physiol Heart Circ Physiol 2004; 287:H1836-41.

23. Mondillo S, Galderisi M, Ballo P, Marino PN. Left ventricular systolic longitudinal function: comparison among simple M-mode, pulsed, and M-mode color tissue Doppler of mitral annulus in healthy individuals. J Am Soc Echocardiogr 2006;19:1085-91.

24. Alam M, Höglund C, Thorstrand C, Philip A. Atrioventricular plane displacement in severe congestive heart failure following dilated cardiomyopathy or myocardial infarction. J Intern Med 1990;228:569-75.

25. Alam M, Höglund C, Thorstrand C. Longitudinal systolic shortening of the left ventricle: an echocardiographic study in subjects with and without preserved global function. Clin Physiol 1992;12:443-52.

26. Silva JA, Khuri B, Barbee W, Fontenot D, Cheirif J. Systolic excursion of the mitral annulus to assess septal function in paradoxic septal motion. Am Heart J 1996;131:138-45.

27. Matos J, Kronzon I, Panagopoulos G, Perk G. Mitral annular plane systolic excursion as a surrogate for left ventricular ejection fraction. J Am Soc Echocardiogr 2012;25:969-74.

28. Hoffman EA, Ritman EL. Invariant total heart volume in the intact thorax. Am J Physiol 1985;249:H883-90.

29. Carlhäll CJ, Lindstrom L, Wranne B, Nylander E. Atrioventricular plane displacement correlates closely to circulatory dimensions but not to ejection fraction in normal young subjects. Clin Physiol 2001;21:621-8.

30. Wandt B, Bojö L, Tolagen K, Wranne B. Echocardiographic assessment of ejection fraction in left ventricular hypertrophy. Heart 1999;82:192-8.

31. Derumeaux G, Ovize M, Loufoua J, Andre-Fouet X, Minaire Y, Cribier A, et al. Doppler tissue imaging quantitates regional wall motion during myocardial ischemia and reperfusion. Circulation 1998;97:1970-7.

32. Gulati VK, Katz WE, Follansbee WP, Gorcsan J III. Mitral annular descent velocity by tissue Doppler echocardiography as an index of global left ventricular function. Am J Cardiol 1996;77:979-84.

33. Quiñones MA, Otto CM, Stoddard M, Waggoner A, Zoghbi WA. Recommendations for quantification of Doppler echocardiography: a report from the Doppler Quantification Task Force of the Nomenclature and Standards Committee of the American Society of Echocardiography. J Am Soc Echocardiogr 2002;15:167-84.

34. Gorcsan J III, Deswal A, Mankad S, Mandarino WA, Mahler CM, Yamazaki N, et al. Quantification of the myocardial response to low-dose dobutamine using tissue Doppler echocardiographic measures of velocity and velocity gradient. Am J Cardiol 1998;81:615-23.

35. Shimizu Y, Uematsu M, Shimizu H, Nakamura K, Yamagishi M, Miyatake K. Peak negative myocardial velocity gradient in early diastole as a noninvasive indicator of left ventricular diastolic function: comparison with transmitral flow velocity indices. J Am Coll Cardiol 1998;32:1418-25.

36. Ruan Q, Nagueh SF. Usefulness of isovolumic and systolic ejection signals by tissue Doppler for the assessment of left ventricular systolic function in ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol 2006;97: 872-5.

37. Wang M, Yip GW, Wang AY, Zhang Y, Ho PY, Tse MK, 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.

38. Nikitin NP, Loh PH, Silva R, Ghosh J, Khaleva OY, Goode K, et al. Prognostic value of systolic mitral annular velocity measured with Doppler tissue imaging in patients with chronic heart failure caused by left ventricular systolic dysfunction. Heart 2006;92:775-9.

39. Garrard CL Jr., Weissler AM, Dodge HT. The relationship of alterations in systolic time intervals to ejection fraction in patients with cardiac disease. Circulation 1970;42:455-62.

40. Hodges M, Halpern BL, Friesinger GC, Dagenais GR. Left ventricular pre-ejection period and ejection time in patients with acute myocardial infarction. Circulation 1972;45:933-42.

41. Park SC, Steinfeld L, Dimich I. Systolic time intervals in infants with congestive heart failure. Circulation 1973;47:1281-8.

42. Carvalho P, Paiva RP, Couceiro R, Henriques J, Antunes M, Quintal I, et al. Comparison of systolic time interval measurement modalities for portable devices. Conf Proc IEEE Eng Med Biol Soc 2010;2010:606-9.

43. Weissler AM, Harris WS, Schoenfeld CD. Systolic time intervals in heart failure in man. Circulation 1968;37:149-59.

44. Tei C, Dujardin KS, Hodge DO, Kyle RA, Tajik AJ, Seward JB. Doppler index combining systolic and diastolic myocardial performance: clinical value in cardiac amyloidosis. J Am Coll Cardiol 1996;28:658-64.

45. Czernik C, Rhode S, Metze B, Schmalisch G, Buhrer C. Persistently elevated right ventricular index of myocardial performance in preterm infants with incipient bronchopulmonary dysplasia. PLoS One 2012;7:e38352.

46. Said K, Hassan M, Baligh E, Zayed B, Sorour K. Ventricular function in patients with end-stage renal disease starting dialysis therapy: a tissue Doppler imaging study. Echocardiography 2012;29:1054-9.

47. Belham M, Kruger A, Pritchard C. The Tei index identifies a differential effect on left and right ventricular function with low-dose anthracycline chemotherapy. J Am Soc Echocardiogr 2006;19:206-10.

48. Kim WH, Otsuji Y, Seward JB, Tei C. Estimation of left ventricular function in right ventricular volume and pressure overload. Detection of early left ventricular dysfunction by Tei index. Jpn Heart J 1999;40:145-54.

49. Niemann M, Breunig F, Beer M, Hu K, Liu D, Emmert A, et al. Tei index in Fabry disease. J Am Soc Echocardiogr 2011;24:1026-32.

50. Tei C, Ling LH, Hodge DO, Bailey KR, Oh JK, Rodeheffer RJ, 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.

51. Tekten T, Onbasili AO, Ceyhan C, Ünal S, Discigil B. Novel approach to measure myocardial performance index: pulsed-wave tissue Doppler echocardiography. Echocardiography 2003;20:503-10.

52. Schaefer A, Meyer GP, Hilfiker-Kleiner D, Brand B, Drexler H, Klein G. Evaluation of tissue Doppler Tei index for global left ventricular function in mice after myocardial infarction: comparison with pulsed Doppler Tei index. Eur J Echocardiogr 2005;6:367-75.

53. Cui W, Roberson DA, Chen Z, Madronero LF, Cuneo BF. Systolic and diastolic time intervals measured from Doppler tissue imaging: normal values and Z-score tables, and effects of age, heart rate, and body surface area. J Am Soc Echocardiogr 2008;21:361-70.

54. 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.

55. LaCorte JC, Cabreriza SE, Rabkin DG, Printz BF, Coku L, Weinberg A, et al. Correlation of the Tei index with invasive measurements of ventricular function in a porcine model. J Am Soc Echocardiogr 2003;16:442-7.

56. Bruch C, Schmermund A, Marin D, Katz M, Bartel T, Schaar J, et al. Tei-index in patients with mild-to-moderate congestive heart failure. Eur Heart J 2000;21:1888-95.

57. Ikeda R, Yuda S, Kobayashi N, Nakahara N, Nakata T, Tsuchihashi K, et al. Usefulness of right ventricular Doppler index for predicting outcome in patients with dilated cardiomyopathy. J Cardiol 2001;37:157-64.

58. Eidem BW, O'Leary PW, Tei C, Seward JB. Usefulness of the myocardial performance index for assessing right ventricular function in congenital heart disease. Am J Cardiol 2000;86:654-8.

59. Bruch C, Schmermund A, Dagres N, Katz M, Bartel T, Erbel R. Severe aortic valve stenosis with preserved and reduced systolic left ventricular function: diagnostic usefulness of the Tei index. J Am Soc Echocardiogr 2002;15:869-76.

60. Haque A, Otsuji Y, Yoshifuku S, Kumanohoso T, Zhang H, Kisanuki A, et al. Effects of valve dysfunction on Doppler Tei index. J Am Soc Echocardiogr 2002;15:877-83.

61. Nearchou NS, Tsakiris AK, Lolaka MD, Karatzis EN, Tsiafoutis IN, Flessa CD, et al. Influence of angiotensin II receptors blocking on overall left ventricle's performance of patients with acute myocardial infarction of limited extent. Echocardiographic assessment. Int J Cardiovasc Imaging 2006;22:191-8.

62. Dujardin KS, Tei C, Yeo TC, Hodge DO, Rossi A, Seward JB. Prognostic value of a Doppler index combining systolic and diastolic performance in idiopathic-dilated cardiomyopathy. Am J Cardiol 1998;82:1071-6.

63. Sasao H, Noda R, Hasegawa T, Endo A, Oimatsu H, Takada T. Prognostic value of the Tei index combining systolic and diastolic myocardial performance in patients with acute myocardial infarction treated by successful primary angioplasty. Heart Vessels 2004;19:68-74.

64. Møller JE, Egstrup K, Køber L, Poulsen SH, Nyvad O, Torp-Pedersen C. Prognostic importance of systolic and diastolic function after acute myocardial infarction. Am Heart J 2003;145:147-53.

65. Karatzis EN, Giannakopoulou AT, Papadakis JE, Karazachos AV, Nearchou NS. Myocardial performance index (Tei index): evaluating its application to myocardial infarction. Hellenic J Cardiol 2009;50:60-5.

66. Mahler F, Ross J Jr., O'Rourke RA, Covell JW. Effects of changes in preload, afterload and inotropic state on ejection and isovolumic phase measures of contractility in the conscious dog. Am J Cardiol 1975;35:626-34.

67. Quinones MA, Gaasch WH, Alexander JK. Influence of acute changes in preload, afterload, contractile state and heart rate on ejection and isovolumic indices of myocardial contractility in man. Circulation 1976;53:293-302.

68. Rhodes J, Udelson JE, Marx GR, Schmid CH, Konstam MA, Hijazi ZM, et al. A new noninvasive method for the estimation of peak dP/dt. Circulation 1993;88:2693-9.

69. Nishimura RA, Tajik AJ. Determination of left-sided pressure gradients by utilizing Doppler aortic and mitral regurgitant signals: validation by simultaneous dual catheter and Doppler studies. J Am Coll Cardiol 1988;11:317-21.

70. Chen C, Rodriguez L, Guerrero JL, Marshall S, Levine RA, Weyman AE, et al. Noninvasive estimation of the instantaneous first derivative of left ventricular pressure using continuous-wave Doppler echocardiography. Circulation 1991;83:2101-10.

71. Bargiggia GS, Bertucci C, Recusani F, Raisaro A, de Servi S, Valdes-Cruz LM, et al. A new method for estimating left ventricular dP/dt by continuous wave Doppler-echocardiography. Validation studies at cardiac catheterization. Circulation 1989;80:1287-92.

72. Poelaert J, Skarvan K. Transoesophageal echocardiography in anaesthesia and intensive care medicine. London, United Kingdom: BMJ Publishing Group; 2004.

73. Kolias TJ, Aaronson KD, Armstrong WF. Doppler-derived dP/dt and -dP/dt predict survival in congestive heart failure. J Am Coll Cardiol 2000;36:1594-9.

74. Chand R, Mehta Y, Trehan N. Cardiac output estimation with a new Doppler device after off-pump coronary artery bypass surgery. J Cardiothorac Vasc Anesth 2006;20:315-9.

75. Lewis JF, Kuo LC, Nelson JG, Limacher MC, Quinones MA. Pulsed Doppler echocardiographic determination of stroke volume and cardiac output: clinical validation of two new methods using the apical window. Circulation 1984;70:425-31.

76. Tan HL, Pinder M, Parsons R, Roberts B, van Heerden PV. Clinical evaluation of USCOM ultrasonic cardiac output monitor in cardiac surgical patients in intensive care unit. Br J Anaesth 2005;94:287-91.

77. Baumgartner H, Hung J, Bermejo J, Chambers JB, Evangelista A, Griffin BP, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. Eur J Echocardiogr 2009;10:1-25.

78. Ihlen H, Amlie JP, Dale J, Forfang K, Nitter-Hauge S, Otterstad JE, et al. Determination of cardiac output by Doppler echocardiography. Br Heart J 1984;51:54-60.

79. Baumgartner H, Kratzer H, Helmreich G, Kuehn P. Determination of aortic valve area by Doppler echocardiography using the continuity equation: a critical evaluation. Cardiology 1990;77:101-11.

80. Feinberg MS, Hopkins WE, Davila-Roman VG, Barzilai B. Multiplane transesophageal echocardiographic Doppler imaging accurately determines cardiac output measurements in critically ill patients. Chest 1995;107:769-73.

81. Robson SC, Boys RJ, Hunter S. Doppler echocardiographic estimation of cardiac output: analysis of temporal variability. Eur Heart J 1988;9:313-8.

82. Huntsman LL, Stewart DK, Barnes SR, Franklin SB, Colocousis JS, Hessel EA. Noninvasive Doppler determination of cardiac output in man. Clinical validation. Circulation 1983;67:593-602.