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Determination of Left Ventricular Filling Pressure by Doppler Echocardiography in Patients With Coronary Artery Disease: Critical Role of Left Ventricular Systolic Function

KAZUHIRO YAMAMOTO, MD, RICK A. NISHIMURA, MD, FACC, HARI P. CHALIKI, MD, CHRISTOPHER P. APPLETON, MD, FACC,* DAVID R. HOLMES, JR., MD, FACC, MARGARET M. REDFIELD, MD, FACC

Rochester, Minnesota and Scottsdale, Arizona

Objectives. **This study was designed to determine the usefulness of transthoracic Doppler measurements in detecting increased left ventricular (LV) end-diastolic pressure in patients with coronary artery disease, specifically examining the influence of systolic function on the accuracy of these methods.**

Background. **Studies that have correlated Doppler indexes with LV filling pressures primarily involved patients with LV systolic dysfunction. The reliability of Doppler indexes in estimating filling pressures in patients with coronary artery disease and preserved systolic function is unclear.**

Methods. **Pulsed wave Doppler transmitral and pulmonary venous flow velocity curves and LV pressure were recorded in 83 patients with coronary artery disease.**

Results. **Conventional Doppler indexes (deceleration time of mitral E wave velocity, ratio of peak mitral E to A wave velocities and pulmonary venous systolic fraction) correlated with LV filling pressure in patients with an ejection fraction (EF) ≤50% but not in those with an EF >50%. Previously published regression analysis for prediction of LV filling pressure was accurate in patients with an EF ≤50% but not in those with an EF >50%. The difference between flow duration with atrial contraction in the pulmonary veins and transmitral flow duration with atrial contraction correlated with LV filling pressure in both groups.**

Conclusions. **Analysis of the early diastolic portion of the transmitral or pulmonary venous flow velocity curves can be used to predict LV filling pressures in patients with systolic dysfunction, but are inaccurate in patients with preserved systolic function. The combined analysis of both flow velocity curves at atrial contraction is a reliable, feasible predictor of increased LV filling pressure, irrespective of systolic function.**

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Left ventricular (LV) systolic function is frequently preserved in patients with congestive heart failure (1–4). The diagnosis of isolated diastolic heart failure depends on the demonstration of increased LV diastolic pressure, which requires invasive cardiac catheterization. Certain indexes derived from pulsed wave Doppler transmitral flow velocity curves, specifically those in early diastole (peak mitral E wave velocity, ratio of peak mitral E wave velocity to peak mitral A wave velocity [E/A ratio] and deceleration time of mitral E wave velocity), correlate with LV filling pressures in subgroups of patients with systolic dysfunction (5,6). However, our laboratory has shown a poor correlation between such indexes and LV filling pressures in patients with hypertrophic cardiomyopathy and normal to hyperdynamic systolic function (7). Other studies have reported that the combined analysis of transmitral and pulmonary venous flow velocity curves—the difference between the duration of flow with atrial contraction in the pulmonary veins (pulmonary venous A duration) and that across the mitral valve (mitral A duration)—correlates with LV end-diastolic pressure in a large series of patients (8,9). The overall reliability of these noninvasive Doppler indexes in patients with more common cardiac diseases and isolated diastolic dysfunction is unclear.

The present study was designed to determine the reliability of analysis of transmitral or pulmonary venous flow velocity curves, or both, in detecting increased LV end-diastolic pressure in patients with coronary artery disease, focusing particularly on the influence of systolic function.

Methods

Eighty-three patients with coronary artery disease referred for cardiac catheterization were studied. All patients had sinus rhythm without mitral or aortic stenosis or more than mild mitral or aortic regurgitation. All patients were undergoing elective coronary angiography, and no patient had unstable

From the Division of Cardiovascular Diseases and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota; and *Division of Cardiovascular Diseases, Mayo Clinic Scottsdale, Scottsdale, Arizona. This study was supported in part by grants from the Joseph P. and Jeanne M. Sullivan Foundation, Chicago, Illinois; the Miami Heart Research Institute, Miami, Florida; and the Mayo Foundation, Rochester, Minnesota. Dr. Yamamoto was supported by a Fellowship of the Uehara Memorial Foundation, Tokyo, Japan.

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Address for correspondence: Dr. Rick A. Nishimura, Mayo Clinic, 200 First Street SW, Rochester, Minnesota 55905.

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Abbreviations and Acronyms

E/A = ratio of peak mitral E wave velocity to peak mitral A wave velocity
EF = ejection fraction
LV = left ventricular
ROC = receiver operating characteristic

angina before the procedure or angina during the procedure. For ethical reasons, medications were not withheld before the study. This protocol was approved by the Institutional Review Board of the Mayo Foundation, and written informed consent was obtained from all the patients. The Doppler data from a subgroup of these patients are included in a previous report that focused on a different analysis of the diastolic filling variables (10).

Echocardiographic assessment. Transthoracic two-dimensional and Doppler echocardiographic studies were conducted with the subjects in the fasting state, in the left lateral position and during quiet respiration within 3 h before cardiac catheterization. Blood pressure and heart rate were measured at the time of echocardiography. The transmitral flow velocity curves were recorded with the sample volume at the mitral tips, and the pulmonary venous flow velocity curves were recorded with the sample volume 0 to 1 cm into the right superior pulmonary vein (8,11–13) Continuous wave Doppler echocardiography was used to simultaneously obtain the transmitral flow velocity curve and aortic valve closure click (14). During the pulsed wave Doppler study, the filter settings were minimized. Doppler velocity curves were recorded at a horizontal sweep speed of 100 mm/s.

Left ventricular pressure measurements. Left ventricular pressure was recorded with a 7F, high fidelity, manometer-tipped catheter (Millar Instruments), as described previously (11), or a 6F pigtail catheter connected with a fluid-filled transducer before coronary angiography and left ventriculography. Left ventricular pressure was digitized at 5-ms intervals onto an off-line computer.

Data analysis. Echocardiographic recordings were analyzed with the commercial analysis software supplied with the system. The ejection fraction (EF) was calculated by a modification of the method of Quinones et al. (15), as described previously (16,17): EF (%) = (LV end-diastolic dimension^2 − LV end-systolic dimension^2/LV end-diastolic dimension^2 × 100. Left ventricular mass was calculated from the formula and M-mode conventions recommended by the American Society of Echocardiography (18), and the LV mass index was determined as the ratio of LV mass to body surface area, as described previously (19). The transmitral flow velocity curve was analyzed for measurement of the peak mitral E and A wave velocities, the deceleration time of the mitral E wave velocity (deceleration time) and the mitral A duration (Fig. 1). The pulmonary venous flow velocity curve was digitized for measurement of the time–velocity integral of the systolic and diastolic forward flow velocity curves. The duration (pulmonary venous A duration) and peak velocity of the pulmonary venous velocity curve with atrial contraction were also determined (8–10,20). When the entire pulmonary or mitral A wave velocity curve was not recorded adequately, the difference in pulmonary venous and mitral A durations was assessed by reference to the relative positions of the cessation of mitral A wave flow and the cessation of pulmonary venous A wave flow or the start of pulmonary venous systolic forward flow on the QRS complex (8). Isovolumetric relaxation time was calculated from the aortic valve closure click to the onset of the mitral E wave velocity curve on the continuous wave Doppler echocardiogram (14). Doppler-derived LV end-diastolic pressure (LVEDP) was calculated from a multiple regression analysis proposed by Mulvagh et al. (21):

$$\text{LVEDP} = 46 - 0.22\ \text{IVRT} - 0.11\ \text{AFF} - 0.03\text{DT} - \left(\frac{2}{\text{E/A}}\right) + 0.05\ \text{MAR}$$

where IVRT = isovolumetric relaxation time; AFF = atrial filling fraction; DT = deceleration time; and MAR = time from termination of mitral flow to electrocardiographic R wave.

Left ventricular end-diastolic pressure was measured from the LV pressure tracing at the point just before the onset of an increase in LV systolic pressure. Left ventricular pre–A wave pressure was measured at the point just before the onset of an increase in LV pressure due to atrial contraction. In the patients in whom high fidelity LV pressure recordings were made, the time constant of LV relaxation was measured to assess the rate of LV relaxation. The time constant was

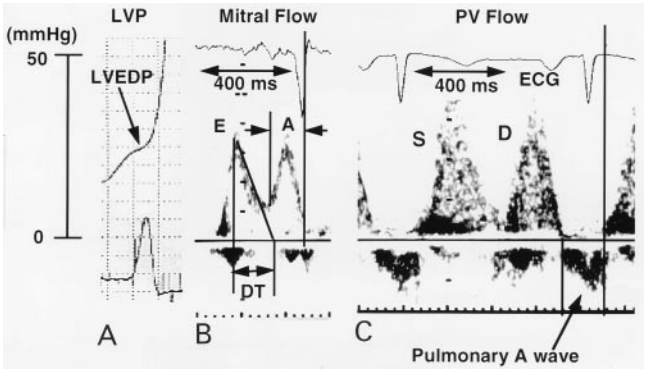

Figure 1. Recordings of **(A)** left ventricular pressure (LVP), **(B)** transmitral flow velocity curve (mitral flow) and **(C)** pulmonary venous (PV) flow velocity curve in a 74-year old patient with an EF of 62%. In this patient, left ventricular end-diastolic pressure (LVEDP) is increased to 25 mm Hg. The pulmonary venous A duration (171 ms) is longer than the mitral A duration (143 ms), and the end of the mitral A wave velocity curve occurs earlier in the QRS complex than does the end of pulmonary venous A wave velocity curve. However, the deceleration time (DT) is not shortened (186 ms); the ratio of peak mitral E wave velocity to peak mitral A wave velocity is not increased (1.1); and the pulmonary venous systolic fraction is not decreased (0.60). A = mitral A wave velocity curve; D = pulmonary venous diastolic forward flow velocity curve; E = mitral E wave velocity curve; ECG = electrocardiogram; S = pulmonary venous systolic forward flow velocity curve.

calculated using a zero pressure asymptote from peak rate of rise in LV pressure (dP/dt) to 5 mm Hg above LV end-diastolic pressure, as described previously (22,23).

Averaged values of three or more consecutive beats were used for statistical analysis.

Intraobserver and interobserver variabilities. The intraobserver and interobserver variabilities for Doppler tracings were assessed using 11 random Doppler recordings. The mean (±SD) values of variability were 0.04 ± 0.03 m/s (intraobserver) and 0.04 ± 0.04 m/s (interobserver) for measurement of peak mitral E wave velocity; 0.03 ± 0.03 m/s (intraobserver) and 0.02 ± 0.01 m/s (interobserver) for measurement of peak mitral A wave velocity; 6.5 ± 3.4 ms (intraobserver) and 6.9 ± 5.1 ms (interobserver) for measurement of mitral A duration; 0.02 ± 0.02 m/s (intraobserver) and 0.02 ± 0.02 m/s (interobserver) for measurement of peak pulmonary venous systolic forward flow velocity; 0.02 ± 0.02 m/s (intraobserver) and 0.02 ± 0.03 m/s (interobserver) for measurement of peak pulmonary venous diastolic forward flow velocity; and 11.5 ± 5.6 ms (intraobserver) and 10.0 ± 8.4 ms (interobserver) for measurement of pulmonary venous A wave duration.

Statistical analysis. Data are expressed as mean values ± SD. Bivariate correlation between Doppler indexes and LV end-diastolic pressure was performed with simple least-squares linear regression analysis. The correlation study was performed for patients with an EF ≤50% and for those with an EF >50% to assess the influence of systolic function on the validity of these methods. The statistical significance of the difference between the values of the two groups was tested with analysis of variance and the Scheffé *F* test. Results were considered significant at $p < 0.05$.

We assessed the ability of the combined analysis of transmitral and pulmonary venous flow velocity curves to identify LV end-diastolic pressures ≥15 mm Hg and ≥20 mm Hg by receiver operating characteristic (ROC) analysis. To assess whether the combined analysis has any informational content, we compared the areas under the ROC curves with 0.5 (area under the line of no information) using a Wilcoxon rank-sum statistic, as previously described (24). Results were considered significant at $p < 0.05$.

Table 1. Comparison of Clinical Characteristics Between Patients With an Ejection Fraction ≤50% and Those With an Ejection Fraction >50%

Characteristic	EF ≤50% (n = 23)	EF >50% (n = 59)
Age (yr)	64 ± 10	65 ± 10
No. of diseased coronary arteries with >70% stenosis	2.2 ± 0.9	1.8 ± 0.9
EF (%)	32 ± 10	61 ± 6*
LVEDD (mm)	61 ± 9	50 ± 5*
LV mass index (g/m^2)	133 ± 40	97 ± 22*
LA diameter (mm)	45 ± 9	40 ± 5*

*$p < 0.05$ compared with patients with an ejection fraction (EF) ≤50%. Data are expressed as mean value ± SD. LA = left atrial; LV = left ventricular; LVEDD = left ventricular end-diastolic diameter.

Results

Patient characteristics. One patient was excluded from analysis because the systolic blood pressure at cardiac catheterization was >40 mm Hg above that during echocardiography. Thus, data from 82 patients (55 men and 27 women, mean age 64 years) were analyzed. In these 82 patients, the mean differences between systolic and diastolic arterial pressures and heart rates at catheterization and echocardiography were 6 ± 19 mm Hg, −2 ± 11 mm Hg and 1 ± 7 beats/min, respectively. All the patients had interpretable Doppler recordings of transmitral flow velocity curves. In 14% of the patients, Doppler recordings of pulmonary venous flow velocity curves could not be obtained because of technical limitations. The clinical characteristics of patients with an EF ≤50% and those with an EF >50% are outlined in Table 1. Patients with an EF ≤50% had more LV remodeling with dilation and hypertrophy and a more dilated left atrium.

Assessment of LV end-diastolic pressure with transmitral or pulmonary venous flow velocity curves alone: early diastolic velocity indexes. Correlations between the early diastolic Doppler indexes and LV end-diastolic pressure were determined. The deceleration time correlated with LV end-diastolic pressure in patients with an EF ≤50% ($r = -0.85$, $p < 0.01$), but not in patients with an EF >50% ($r = -0.15$) (Fig. 2). The E/A ratio correlated with LV end-diastolic pressure in patients

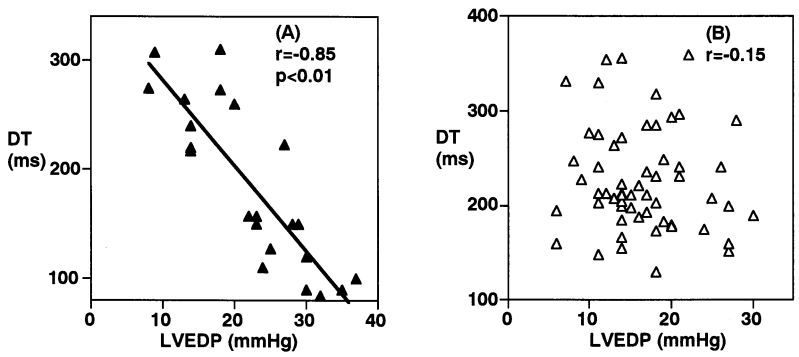

Figure 2. Correlation between the deceleration time (DT) and left ventricular end-diastolic pressure (LVEDP) (**A**) in patients with an EF ≤50% and (**B**) in those with an EF >50%. **Solid line** = regression line.

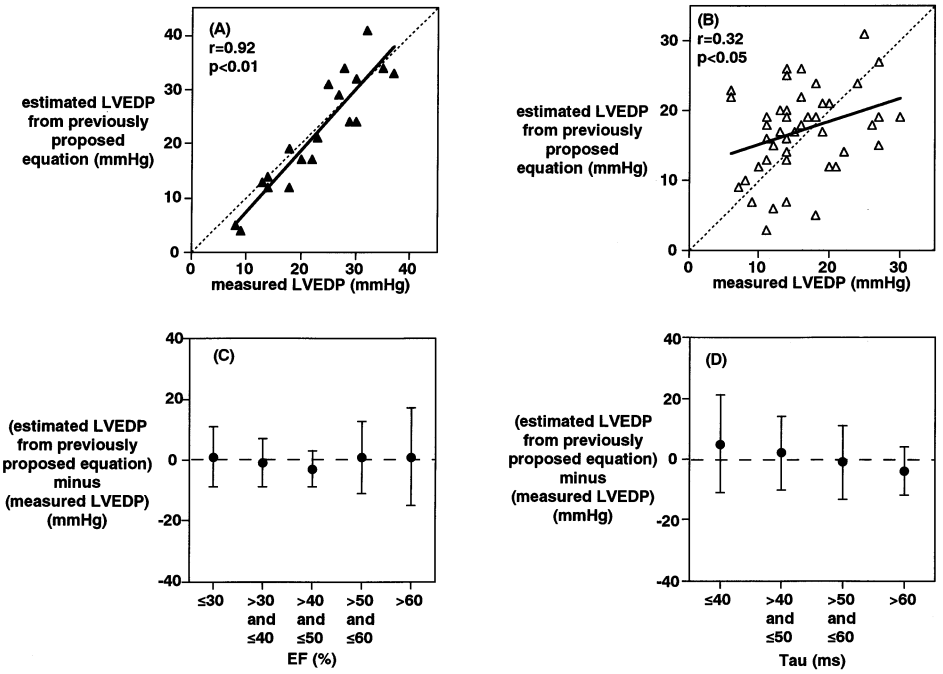

Figure 3. Correlation between the estimated left ventricular end-diastolic pressure (LVEDP) from a previously proposed equation (18) and measured LVEDP in patients with an EF ≤50% (**A**) and those with an EF >50% (**B**). **Dotted line** = line of identity; **solid line** = regression line. Mean (±2 SD) of the difference between the estimated (21) and measured LVEDP in a subgroup of patients classified according to their EF (**C**) or the time constant of LV relaxation (Tau) (**D**). **Horizontal dotted line** = the difference is equal to 0 mm Hg.

with an EF ≤50% ($r = 0.61$, $p < 0.01$). The correlation was weaker in patients with an EF >50% ($r = 0.33$, $p < 0.05$). The pulmonary venous systolic fraction (a ratio of the time–velocity integral of pulmonary venous systolic forward flow wave to the sum of the time–velocity integrals of systolic and diastolic forward flow waves) correlated with LV end-diastolic pressure in patients with an EF ≤50% ($r = -0.69$, $p < 0.01$), but the correlation was weaker in those with an EF >50% ($r = -0.48$, $p < 0.01$).

Similar results were obtained using LV pre–A wave pressure as an index for LV filling pressure. In patients with an EF ≤50%, the correlation coefficients of the deceleration time, the E/A ratio and the pulmonary venous systolic fraction with LV pre–A wave pressure were −0.74 ($p < 0.01$), 0.45 and −0.62 ($p < 0.05$), respectively. Those in patients with an EF >50% were −0.006, 0.38 and −0.29, respectively.

The Doppler-derived LV end-diastolic pressure, based on a previously proposed equation (21), correlated with measured LV ventricular end-diastolic pressure in patients with an EF ≤50% ($r = -0.92$, $p < 0.01$), but the correlation was weaker in those with an EF >50% ($r = 0.32$, $p < 0.05$) (Fig. 3, A and B). The relation between the estimated LV end-diastolic pressure using a previously proposed equation (21) minus the measured LV end-diastolic pressure and EF or the time constant of LV relaxation is shown in Figure 3, C and D. The scatter between the estimated and the measured LV end-diastolic pressures was greater in patients with a higher EF and a shorter time constant.

Assessment with combined analysis of transmitral and pulmonary venous flow velocity curves at atrial contraction. The peak pulmonary venous A wave velocity correlated with LV end-diastolic pressure in patients with an EF ≤50% ($r = 0.66$, $p < 0.01$), but did not show a correlation in patients with an EF >50% ($r = 0.17$). The difference in the durations at atrial contraction between pulmonary venous and transmitral flow velocity curves (pulmonary venous A duration minus mitral A duration) correlated with LV end-diastolic pressure in patients with an EF ≤50% ($r = 0.80$, $p < 0.01$) and in those with an EF >50% ($r = 0.69$, $p < 0.01$). In all patients combined, this correlation was also significant ($r = 0.71$, $p < 0.01$) (Fig. 4). The 95% confidence intervals are indicated by the stippled lines in Figure 4. When LV pre–A wave pressure was used as an index for LV filling pressure, the difference in the durations had a significant correlation in patients with an EF ≤50% ($r = 0.73$, $p < 0.01$), as well as in patients with an EF >50% ($r = 0.43$, $p < 0.05$).

A ROC curve plotting sensitivity against specificity for the differences in the A duration of the mitral versus pulmonary venous velocities is shown in Figure 5. The areas under the ROC curve for detecting LV end-diastolic pressure ≥15 mm Hg (0.866) and ≥20 mm Hg (0.945) were significantly larger than 0.5 (an area under a line of no information).

Left ventricular relaxation in patients with preserved and impaired systolic function. For patients with systolic dysfunction in whom the time constant of LV relaxation could be calculated (n = 16), the time constant was uniformly prolonged and >40 ms. For patients with preserved systolic function in whom the time constant could be calculated (n = 40), the time constant ranged from 27 to 75 ms.

Discussion

In the present study, the early diastolic Doppler indexes derived from transmitral or pulmonary venous flow velocity

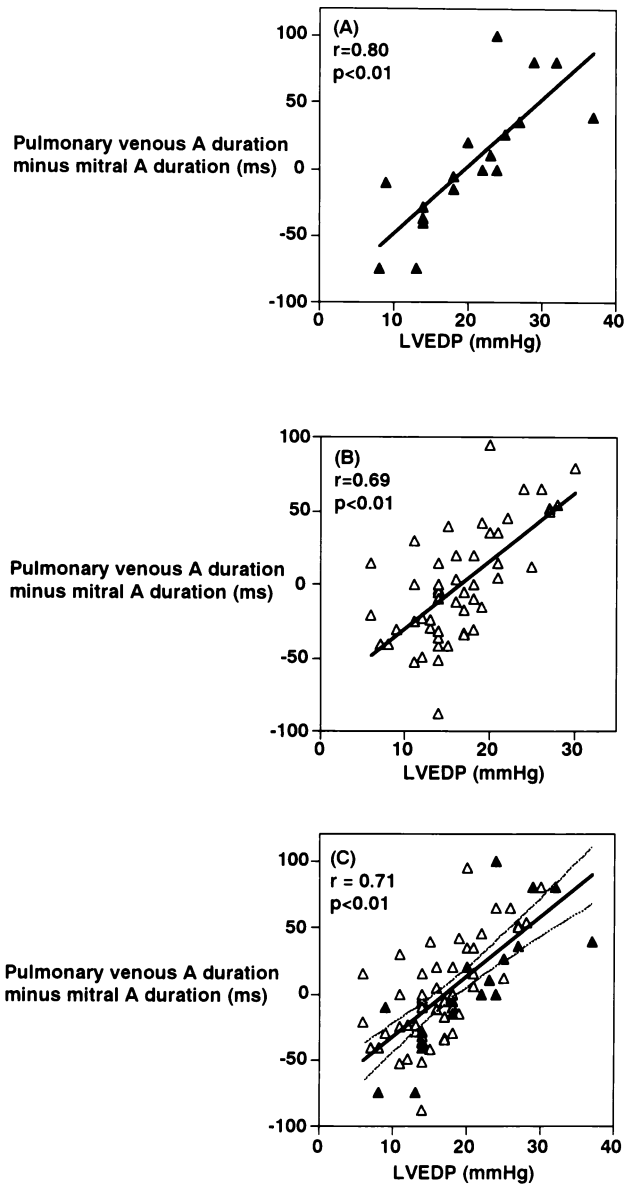

Figure 4. Correlation between the difference in pulmonary venous and mitral A durations and left ventricular end-diastolic pressure (LVEDP) (**A**) in patients with an EF ≤50% (**solid triangles**); (**B**) in those with an EF >50% (**open triangles**); and (**C**) in all the patients. **Solid line** = regression line; **dashed lines** = 95% confidence interval.

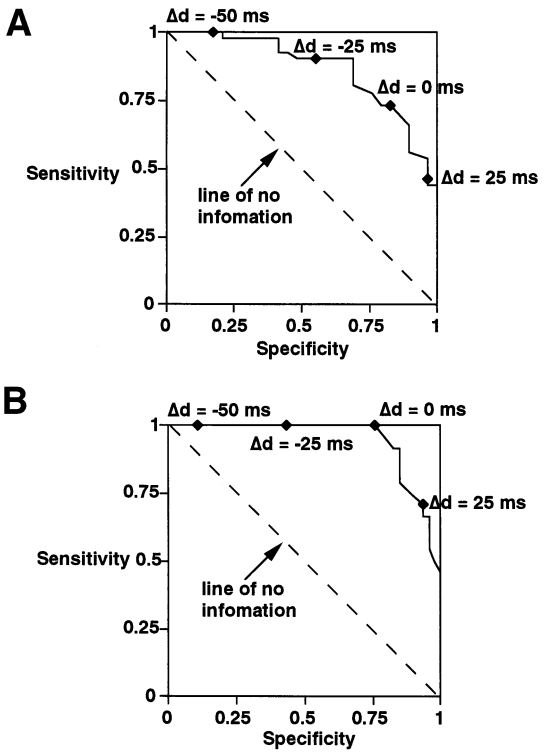

Figure 5. Receiver operating characteristic (ROC) curves for the difference in pulmonary venous and mitral A durations (Δd) in predicting LV end-diastolic pressure ≥15 mm Hg (**A**) and ≥20 mm Hg (**B**). The curves demonstrate that with a greater difference in the pulmonary vein minus mitral A duration, there will be a lower sensitivity but higher specificity for detecting an elevated LV end-diastolic pressure. Sensitivity and specificity of the arbitrarily chosen partition values of the difference in the durations for detecting LV end-diastolic pressure ≥15 mm Hg are, respectively, 1.0 and 0.17 for the difference of −50 ms; 0.90 and 0.55 for the difference of −25 ms; 0.73 and 0.83 for the difference of 0 ms; and 0.46 and 0.97 for the difference of 25 ms. Sensitivity and specificity of the same partition values for detecting LV end-diastolic pressure ≥20 mm Hg are, respectively, 1.0 and 0.11 for the difference of −50 ms; 1.0 and 0.43 for the difference of −25 ms; 1.0 and 0.76 for the difference of 0 ms; and 0.71 and 0.93 for the difference of 25 ms. These points are shown on the ROC curves as **diamond-shaped points.**

curves alone correlated with LV filling pressures in patients with systolic dysfunction, but the correlation was weak for patients with preserved systolic function. These early diastolic indexes—deceleration time, peak mitral E wave velocity and E/A ratio—are routinely obtained in most echocardiographic laboratories. In contrast, the difference in pulmonary venous and transmitral flow durations at atrial contraction correlated well with LV filling pressure, regardless of systolic function.

Assessment with transmitral or pulmonary venous flow velocity curves alone: early diastolic indexes. Recent studies have shown an excellent correlation between LV filling pressure and the early diastolic Doppler indexes in a large number of patients. All the subjects had systolic dysfunction due to either myocardial infarction (6) or dilated cardiomyopathy (5), and the results are similar to those for our patients with a reduced EF. Previous studies of the assessment of filling pressures using early diastolic Doppler indexes did not specifically evaluate the effect of systolic function on the accuracy of these measurements (21,25,26). In the present study, the estimated LV filling pressure showed a poor correlation if patients with preserved systolic function were selected, despite a good correlation in the group as a whole. Analysis of the relation between EF or the time constant of LV relaxation and the difference between the estimated and measured LV end-diastolic pressures (Fig. 3, C and D) demonstrated that the difference becomes larger in patients with a higher EF and a shorter time constant.

The early diastolic Doppler indexes of both transmitral and pulmonary venous flow velocity curves respond in a certain manner to changes in preload (12,26–29). However, they are dependent on many other interrelated factors. The peak mitral E wave velocity, the deceleration time and the isovolumic relaxation time are affected by LV relaxation, compliance and systolic function and left atrial compliance (14,27,30–33). Pulmonary venous systolic fraction depends on left atrial compliance, relaxation and contractility as well as LV compliance, relaxation and contractility (34–36). Thus, a direct relation between filling pressures and these early diastolic indexes would not be expected in all patients.

It has been hypothesized (37) and subsequently shown in an experimental model (38) that a certain progression of these early diastolic indexes occurs in individual patients with advancing disease. From the normal pattern, the deceleration time is prolonged, the E/A ratio is decreased, the isovolumic relaxation time is prolonged and the pulmonary venous systolic fraction is increased in the early stage of diastolic dysfunction when LV relaxation is impaired with little increase in filling pressures. Further progression of ventricular diastolic dysfunction is associated with altered compliance and increased LV filling pressures, which induces "normalization" of transmitral indexes. In end-stage disease, increased LV filling pressures produce a "restrictive" transmitral pattern, with shortened deceleration and isovolumic relaxation times, an increased E/A ratio and a decreased pulmonary venous systolic fraction.

Patient groups with coronary artery disease and preserved systolic function will include patients with normal as well as impaired diastolic function, as shown in the present study by the wide range of filling pressures and time constant of LV relaxation observed in these patients. In such patients, a "normal-appearing" transmitral flow velocity curve may be either normal with normal filling pressures or the result of an increase in LV filling pressure associated with diastolic dysfunction (pseudonormalization). In the former, the peak mitral E wave velocity and deceleration time are affected by rapid ventricular relaxation and diastolic suction, whereas in the latter the increased left atrial driving pressure increases the peak mitral E wave velocity and decreases deceleration time. Figure 2B suggests that the presence of patients with a shorter deceleration time (<180 ms) and lower LV end-diastolic pressure (<20 mm Hg) (i.e., true normal subjects) precludes correlation between these two variables. The previously proposed estimation of LV end-diastolic pressure using early diastolic Doppler indexes (21) overestimates LV end-diastolic pressure in patients with intact LV relaxation who have a normal transmitral flow velocity curve and normal LV filling pressure (Fig. 3D). In contrast to the patients with normal systolic function, all patients with LV systolic dysfunction had diastolic dysfunction (39), as evidenced by the prolongation of the time constant found in the present study. In these patients who have abnormal systolic and diastolic function, there is a relation between the early diastolic Doppler indexes and LV filling pressures.

Assessment with combined analysis of transmitral and pulmonary venous flow velocity curves at atrial contraction. The difference between pulmonary venous and mitral A durations can detect increased LV end-diastolic pressure both in patients with systolic dysfunction and in those with preserved systolic function, in contrast to the poor ability of the other Doppler indexes in patients with preserved systolic function. Rossvoll and Hatle (8) proposed that the difference in the durations is a reliable index for assessing LV end-diastolic pressure on the basis of the previous finding that left atrial A wave pressure is widened in association with an increase in LV diastolic pressure (40). The present study confirms and extends these previous studies by demonstrating that this index is reliable in assessing LV end-diastolic pressure, irrespective of systolic function. Furthermore, the present study emphasizes the clinical usefulness of these measurements in individual patients by demonstrating little overlap of the values of this index between patients with and without increased LV end-diastolic pressure, irrespective of systolic function. Indexes recorded during atrial contraction may be influenced less by LV relaxation and elastic recoil, which occur in early diastole, are related to systolic function (39,41) and affect the conventional Doppler indexes derived from early diastolic transmitral or pulmonary venous flow velocity curves (14,31–35,42).

The measurement of peak velocities by Doppler echocardiography is affected more by errors due to the angle of interrogation than are time intervals (8), and peak pulmonary venous A wave velocity is underestimated by transthoracic Doppler estimation (43). The peak pulmonary venous A wave velocity did not correlate with LV end-diastolic pressure in patients with preserved systolic function in the current study, in part because of the presence of patients with a normal filling pressure and high peak pulmonary venous A wave velocity. The absolute value of the peak velocity is affected by left atrial contractility (44). In contrast, the pulmonary venous and mitral A durations may be affected equally by left atrial contractility; thus, effects of left atrial contractility on each index may be canceled by the difference in the durations.

The acceptable results afforded by the transthoracic approach are gratifying because transesophageal echocardiography is indicated in a minority of patients; if transesophageal echocardiography were required to obtain these signals, the clinical usefulness of this index would be limited.

Study limitations. In the present study, LV pressure was recorded with either high fidelity, manometer-tipped catheters or fluid-filled catheters. Doppler echocardiography and measurement of LV pressure were not done simultaneously, but we confirmed that blood pressure and heart rate did not change significantly with catheterization in comparison with echocardiographic recordings in the 82 patients whose data were used for analysis. Also, we recorded pulsed wave Doppler transmitral flow velocity curves simultaneously with catheterization pressure measurements in 15 patients. The absolute (±SD) value of the difference in peak mitral E wave velocity between the precatheterization echocardiographic study and that performed at the

time of catheterization was 0.05 ± 0.04 m/s; the absolute value of the difference in peak mitral A wave velocity was 0.07 ± 0.06 m/s; and the absolute value of the difference in mitral A wave duration was 6.7 ± 4.5 ms. These differences were small and also suggested that simultaneous measurement would not have resulted in significantly different values for the Doppler indexes.

Conclusions. Although the search for the perfect noninvasive means to assess LV diastolic pressures continues, the findings of the present study confirm and extend those of other investigators and emphasize that analysis of transmitral or pulmonary venous early diastolic Doppler indexes alone provides only limited information about LV diastolic pressure in patients. In the patients with systolic dysfunction, these indexes can be used to predict increased LV filling pressures but are not accurate for patients with normal systolic function. Although technically more demanding, the determination of the difference in pulmonary venous and mitral A durations is feasible and accurate in detecting increased LV diastolic pressure, irrespective of systolic function.

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