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Echocardiography for Hemodynamic Assessment of Patients With Advanced Heart Failure and Potential Heart Transplant Recipients

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Objectives. **This study sought to assess the accuracy of Doppler echocardiographic techniques for the determination of right heart catheterization hemodynamic variables in patients with advanced heart failure and in potential heart transplant recipients.**

Background. **Doppler echocardiographic techniques permit the noninvasive acquisition of hemodynamic variables traditionally used for the assessment of patients with advanced heart failure and potential heart transplant candidates. However, the accuracy of these techniques has not been sufficiently well documented for clinical application in individual patients.**

Methods. **Echocardiographic data required for estimation of mean right atrial, pulmonary artery and mean left atrial pressures and cardiac output were obtained. Right heart catheterization was performed immediately after Doppler echocardiographic data were acquired, before any intervention that might have altered the subject's hemodynamic status.**

Results. **A complete Doppler echocardiographic hemodynamic data set was acquired in 21 (84%) of 25 subjects. For all variables, invasive and noninvasive hemodynamic values were highly correlated ($p < 0.001$), with minimal bias and narrow 95% confidence limits. An algorithm constructed from the noninvasive hemodynamic variable values identified all patients with adverse pulmonary vascular hemodynamic variables (i.e., transpulmonary gradient ≥12 mm Hg, pulmonary vascular resistance ≥3 Wood units or pulmonary vascular resistance index ≥6 Wood units × m^2). This algorithm identified 12 (71%) of 19 patients for whom right heart catheterization was unnecessary.**

Conclusions. **Doppler echocardiographic estimates of hemodynamic variables in patients with advanced heart failure are accurate and reproducible. This noninvasive methodology may assist with monitoring and optimization of medical therapy in patients with advanced heart failure and may obviate the need for routine right heart catheterization in potential heart transplant candidates.**

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Assessment of pulmonary vascular hemodynamic variables by right heart catheterization is an essential component of orthotopic heart transplantation recipient evaluation, and threshold values for selected hemodynamic variables have been identified that predict poor outcomes both early and late after heart transplantation (1–6). These variables include a transpulmonary gradient (TPG) ≥12 mm Hg, pulmonary vascular resistance (PVR) ≥3 Wood units and pulmonary vascular resistance index (PVRI) ≥6 Wood units × m^2 (1–6). In practice, the presence of any one of these adverse hemodynamic predictors is an indication for serial right heart catheterization and assessment of pulmonary vascular reactivity before heart transplantation (1). However, right heart catheterization is an invasive technique, associated with complications such as pneumothorax, infection, arrhythmia and bleeding. Serial right heart catheterization is logistically difficult in outpatients receiving anticoagulant therapy, and data obtained from this procedure are susceptible to interpretive errors, especially in patients with mitral valve and pericardial disease.

For the hemodynamic assessment of patients with advanced heart failure and potential heart transplant recipients, Doppler echocardiographic techniques allow acquisition of similar hemodynamic data but are noninvasive, facilitate serial evaluation in outpatients and provide additional physiologic information that may reduce interpretive error, including ventricular function, valvular integrity and pericardial disease. Several investigators (7–29) have demonstrated that Doppler echocardiographic and invasive hemodynamic measurements are highly correlated in population studies, but the accuracy of these noninvasive techniques has not been sufficiently well documented for clinical application in individual patients. In this context, validation of Doppler echocardiographic hemodynamic techniques for clinical use requires the demonstration of narrow limits of agreement between these methods when applied to individual patients. The purpose of the present

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Abbreviations and Acronyms

CO = cardiac output
LAP = left atrial pressure
PADP = pulmonary artery diastolic pressure
PAMP = pulmonary artery mean pressure
PASP = pulmonary artery systolic pressure
PVR = pulmonary vascular resistance
PVRI = pulmonary vascular resistance index
RAP = right atrial pressure
TPG = transpulmonary gradient
VTI = velocity time integral

study was to assess the accuracy of Doppler echocardiographic techniques for the determination of right heart catheterization hemodynamic variables in individual patients with advanced heart failure undergoing evaluation for heart transplantation.

Methods

Patients. This prospective study included consecutive patients with severe congestive heart failure (New York Heart Association functional class III or IV) undergoing right heart catheterization for determination of hemodynamic suitability for heart transplantation. Patients with a mechanical valve prosthesis, stenotic valvular heart disease or congenital heart disease were excluded.

Protocol summary. After obtaining written informed consent, two-dimensional echocardiographic images were recorded of 1) the inferior vena cava during inspiration and expiration, from the subcostal transducer position; and 2) the left ventricular outflow tract, from the parasternal long-axis position. Continuous and pulsed wave spectral Doppler images of the tricuspid regurgitation, pulmonic regurgitation, mitral regurgitation and transaortic systolic flow signals were obtained from multiple transducer positions. Blood pressure was measured with an oscillometric cuff at 1-min intervals during echocardiographic data acquisition. Right heart catheterization was performed immediately (within 30 min) after the echocardiographic data were acquired, before interventions that might alter hemodynamic status (i.e., administration of intravenous fluids, diuretic drugs, vasodilators or sedatives).

Echocardiographic protocol. Echocardiographic studies were performed in the left lateral decubitus or supine position with a Hewlett-Packard ultrasound system (Sonos 2500) using a 2.5-MHz transducer or a dedicated nonimaging Doppler transducer. Imaging from multiple transthoracic windows and use of several ultrasound transducers facilitated alignment of the Doppler ultrasound beam parallel to blood flow. All echocardiographic images were stored on super-VHS videotape. Strip chart recordings of all spectral Doppler images were obtained for off-line analysis. For each Doppler-based measurement, estimates were obtained from three or more representative cardiac cycles and averaged. Only Doppler signals with easily visualized spectral envelopes were chosen for analysis. Doppler echocardiographic data were acquired and analyzed as follows:

Mean right atrial pressure. Images of the inferior vena cava were obtained from the subcostal transducer position. The diameter of the inferior vena cava was measured using digital calipers, ~2 cm before its insertion into the right atrium, during quiet inspiration and expiration. Mean right atrial pressure (RAP) was estimated from the inferior vena cava diameter and its degree of respirophasic change (7–9).

Pulmonary artery systolic pressure (Fig. 1). Pulmonary artery systolic pressure (PASP) was calculated as the sum of the estimated RAP and the gradient between the peak right ventricular systolic pressure and the estimated RAP. This gradient was estimated by application of the modified Bernoulli equation to the peak velocity of the continuous wave Doppler tricuspid regurgitation signal ($V_{TR\ Max}$): PASP = RAP + $4V^2_{TR\ Max}$ (10–13).

Pulmonary artery diastolic pressure (Fig. 2). Pulmonary artery diastolic pressure (PADP) was calculated as the sum of the estimated RAP and the gradient between the pulmonary artery end-diastolic pressure and the right ventricular end-diastolic pressure, which is equal to the RAP at this time in the cardiac cycle (9,14,15). This gradient was estimated from the end-diastolic velocity of the continuous wave Doppler pulmonic regurgitation signal ($V^2_{PR\ end\text{-}diastolic}$): PADP = RAP + $4V^2_{PR\ end\text{-}diastolic}$.

Pulmonary artery mean pressure. Pulmonary artery mean pressure (PAMP) was calculated by adding one-third of the pulmonary artery pulse pressure to the estimated PADP: PAMP = PADP + 1/3(PASP − PADP).

Left atrial pressure (Fig. 3). Left atrial pressure (LAP) at the time of aortic valve opening, an estimate of mean LAP, was calculated as the difference between the aortic diastolic pressure (AoDP) and the pressure gradient between the left ventricle and left atrium at the time of aortic valve opening. This pressure gradient was determined from the peak velocity of the continuous wave Doppler mitral regurgitation signal at the time of aortic valve opening ($V^2_{MR\text{-}AVO}$): LAP = AoDP − $4V^2_{MR\text{-}AVO}$ (21).

The timing of aortic valve opening was measured as the distance from the peak of the electrocardiographic R wave to the leading edge of the pulsed wave Doppler aortic valve opening click. This distance was used to identify the peak mitral regurgitation velocity at the time of aortic valve opening, as measured from the R wave at the time the mitral regurgitation Doppler signal was recorded (21). Aortic diastolic pressure was determined by oscillometric sphygmomanometry (21,30) (Dinamap Vital Signs Monitor, 1846SX, Critikon, Inc.).

Cardiac output. Cardiac output (CO) was calculated as the product of the heart rate, aortic velocity time integral (VTI) and the area of the left ventricular outflow tract. The aortic VTI was determined using the Hewlett-Packard Sonos 2500 analysis package. The outflow tract diameter was measured with digital calipers in the parasternal long-axis view: CO =

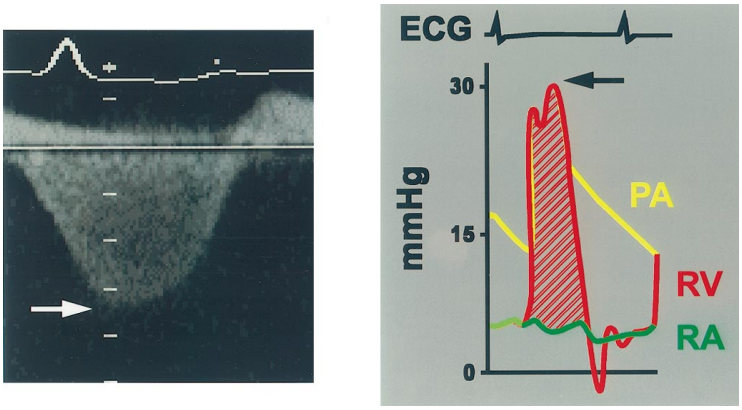

Figure 1. Doppler echocardiographic determination of PASP. **Left panel,** Spectral continuous wave Doppler signal of tricuspid regurgitation corresponding to the right ventricular (RV)–right atrial (RA) pressure gradient denoted by the **red shading** in the **right panel. Right panel,** Stylized right-sided hemodynamic waveforms. PASP was calculated as the sum of the estimated RAP and the gradient between the peak right ventricular systolic pressure and the right atrial pressure **(arrow),** as estimated by application of the modified Bernoulli equation to peak velocity represented by the tricuspid regurgitation Doppler signal **(left panel, arrow)**. ECG = electrocardiogram; PA = pulmonary artery.

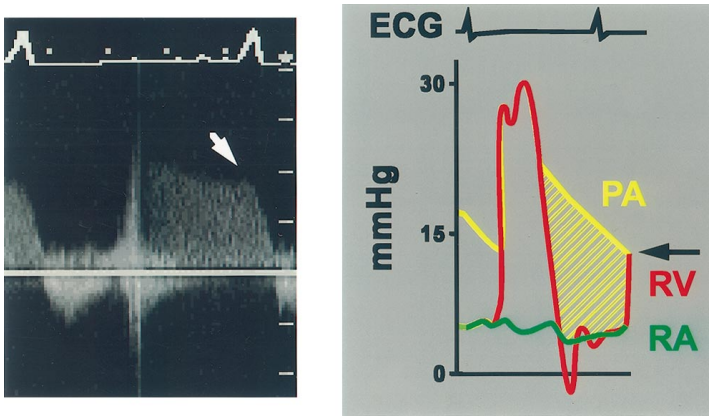

Figure 2. Doppler echocardiographic determination of PADP. **Left panel,** Spectral continuous wave Doppler signal of pulmonic regurgitation corresponding to the pulmonary artery (PA)–right ventricular (RV) pressure gradient denoted by the **yellow shading** in the **right panel. Right panel,** Stylized right-sided hemodynamic waveforms. PADP was calculated as the sum of the RAP and the gradient between the pulmonary artery end-diastolic pressure and the right ventricular end-diastolic pressure **(arrow)** by application of the modified Bernoulli equation to the end-diastolic velocity of the pulmonary regurgitation Doppler signal **(left panel, arrow)**. ECG = electrocardiogram.

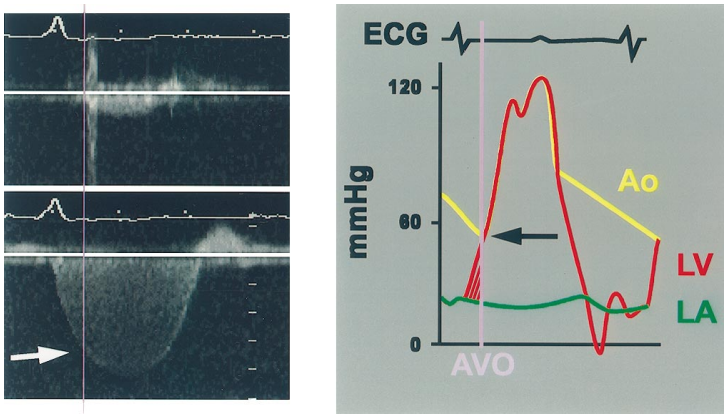

Figure 3. Doppler echocardiographic determination of LAP. **Left panel,** Spectral continuous wave Doppler signal of the aortic valve opening (AVO) click **(top)** and mitral regurgitation **(bottom)** corresponding to the left ventricular (LV)–left atrial (LA) pressure gradient denoted by the **red shading** in the **right panel. Purple vertical lines** denote the time in the cardiac cycle of aortic valve opening. **Right panel,** Stylized left-sided hemodynamic waveforms. LAP at aortic valve opening was calculated as the difference between aortic diastolic pressure (estimated by oscillometric sphygmomanometry [see text]) and the left ventricular–left atrial pressure gradient at aortic valve opening, as determined by application of the modified Bernoulli equation to the peak velocity of the spectral Doppler mitral regurgitation signal at the time of aortic valve opening **(left panel, arrow)**. Ao = aorta; ECG = electrocardiogram.

[Heart rate][Aortic VTI][π][Outflow tract diameter/2]2 (31–34).

Right heart catheterization. Right heart catheterization was performed with fluid-filled Swan-Ganz catheters (Baxter Healthcare Corp., Edwards Critical Care Division) using standard techniques. After calibration of disposable pressure transducers, an internal jugular or subclavian vein was cannulated using the Seldinger technique. The Swan-Ganz catheter was advanced under fluoroscopic guidance and by observation of transduced pressure waveforms. RAP, PASP, PADP and pulmonary capillary occlusion pressure (an estimate of mean LAP) were measured from strip chart recordings at end-expiration. The wedged position of the tip of the right heart catheter during recording of the occlusion waveform was verified fluoroscopically. PAMP was measured as the digital average of the PASP and PADP. CO was determined by the cold saline thermodilution technique.

Derived hemodynamic variables. Cardiac index was calculated as CO divided by body surface area. The transpulmonary gradient (TPG) was calculated as PAMP minus LAP, or the pulmonary artery occlusion pressure. Pulmonary vascular resistance (PVR) was calculated as TPG divided by CO. The PVR index (PVRI) was calculated as TPG divided by the cardiac index.

Table 1. Invasive Hemodynamic Variables

	Mean ± SD	Range
RAP (mm Hg)	12.2 ± 6.3	5–29
PASP (mm Hg)	49.9 ± 17.1	20–80
PADP (mm Hg)	25.1 ± 10.1	9–42
PAMP (mm Hg)	33.3 ± 11.5	13–51
PAOP (mm Hg)	23.5 ± 9.5	7–42
CO (liters/min)	4.7 ± 1.4	3.1–8.3
CI (liters/min)	2.3 ± 0.7	1.5–3.6
TPG (mm Hg)	9.9 ± 3.6	3–18
PVR (Wood U)	2.3 ± 1.0	0.7–4.8
PVRI (Wood U × m^2)	4.7 ± 2.2	1.3–10.9

CI = cardiac index; CO = cardiac output; PADP = pulmonary artery diastolic pressure; PAMP = pulmonary artery mean pressure; PAOP = pulmonary artery occlusion pressure; PASP = pulmonary artery systolic pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; RAP = right atrial pressure; TPG = transpulmonary gradient.

Data analysis. Demographic and hemodynamic data are expressed as mean value ± SD of subject values. Invasive and noninvasive variables were determined in blinded manner. The Pearson r coefficient was used to compare the population values of the invasive and noninvasive hemodynamic variables. The method of Bland and Altman (35) was used to assess methodologic agreement in individual patients. Interobserver variability is expressed as mean value ± SD of the differences between the measurements of two observers (J.H.S., A.N.).

Results

Patients. The study included 25 patients (22 men, 3 women) with a mean age of 48 ± 12 years (range 18 to 68) and a mean body surface area of 2.09 ± 0.29 m^2 (range 1.53 to 2.69). The etiology of heart failure was ischemic cardiomyopathy in 12 patients and idiopathic cardiomyopathy in 13. The average left ventricular ejection fraction was 19 ± 4% (range 12% to 28%). Invasive hemodynamic variables are shown in Table 1.

Accessibility of Doppler echocardiographic data. RAP, PADP and cardiac index could be determined noninvasively in all 25 patients (100%). PASP and PAMP could be determined noninvasively in 23 (92%) of 25 patients. LAP could be determined noninvasively in 22 (88%) of 25 patients. Acquisition of a complete Doppler echocardiographic hemodynamic data set was possible in 21 (84%) of 25 patients.

Invasive versus noninvasive variables. Correlations and limits of agreement between the two methods are shown in Table 2, and selected variables are represented graphically in Figure 4. For all variables, invasive and noninvasive hemodynamic values were highly correlated ($p < 0.001$), biases were small, and the 95% confidence limits were narrow. The differences between invasive and noninvasive hemodynamic variables were not significant for any variable. Interobserver variability for noninvasively determined variables was small (RAP 0.81 ± 3.25 mm Hg, PASP 0.50 ± 5.90 mm Hg, PADP 0.33 ± 3.17 mm Hg, LAP 2.58 ± 3.31 mm Hg).

Table 2. Agreement Between Invasive and Noninvasive Determinations of Hemodynamic Variables

	r Coeff	Bias	95% CL
RAP (mm Hg)	0.93	0.3	5.2
PASP (mm Hg)	0.97	0.1	8.2
PADP (mm Hg)	0.97	0.9	5.2
PAMP (mm Hg)	0.98	0.7	4.6
LAP (mm Hg)	0.97	3.1	4.3
CO (liters/min)	0.97	0.1	0.7
CI (liters/min)	0.97	0.03	0.1
TPG (mm Hg)	0.77	2.4	4.8
PVR (Wood U)	0.84	0.6	1.4
PVRI (Wood U × m^2)	0.83	1.2	2.9

CL = confidence limit; Coeff = coefficient; LAP = left atrial pressure; other abbreviations as in Table 1.

Noninvasive pulmonary vascular hemodynamic thresholds. An algorithm was constructed from the noninvasive hemodynamic variables that would identify all patients with any adverse invasively measured pulmonary vascular hemodynamic variable (i.e., TPG ≥12 mm Hg; PVR ≥3 Wood units; or PVRI ≥6 Wood units × m^2). Scatterplots showing the relations between invasive and noninvasive measurements of the derived hemodynamic variables and the associated noninvasive hemodynamic variable thresholds that corresponded to traditional invasive thresholds are shown in Figure 5; 72% of the data points were concordant. By design, Doppler echocardiographic thresholds (TPG ≥13 mm Hg; PVR ≥3.5 Wood units; and PVRI ≥6.5 Wood units × m^2) identified all patients with high risk pulmonary vascular hemodynamic variables. Application of an algorithm based on the noninvasively determined thresholds identified 12 (71%) of 19 patients in whom right heart catheterization was unnecessary.

Discussion

In potential candidates for heart transplantation, fixed pulmonary hypertension is a powerful predictor of mortality after operation (1–6). Fixed pulmonary hypertension is a contraindication to heart transplantation, and invasive determination of pulmonary vascular hemodynamic variables is the standard of care (1). Furthermore, patients on waiting lists for heart transplantation frequently require serial right heart catheterization procedures to exclude the development or progression of pulmonary hypertension.

Doppler echocardiographic techniques for evaluation of cardiac hemodynamic variables. *RAP.* Accurate estimation of RAP is crucial to the accurate Doppler echocardiographic assessment of right heart hemodynamic variables because it is a component of the equations for calculating PASP, PADP and PAMP. Several noninvasive techniques for estimating mean RAP have been described, including measurement of the height of the jugular venous pulse and regression equations that incorporate right heart Doppler echocardiographic variable values. These techniques may be invalid in patients with heart failure or have not been validated sufficiently for clinical

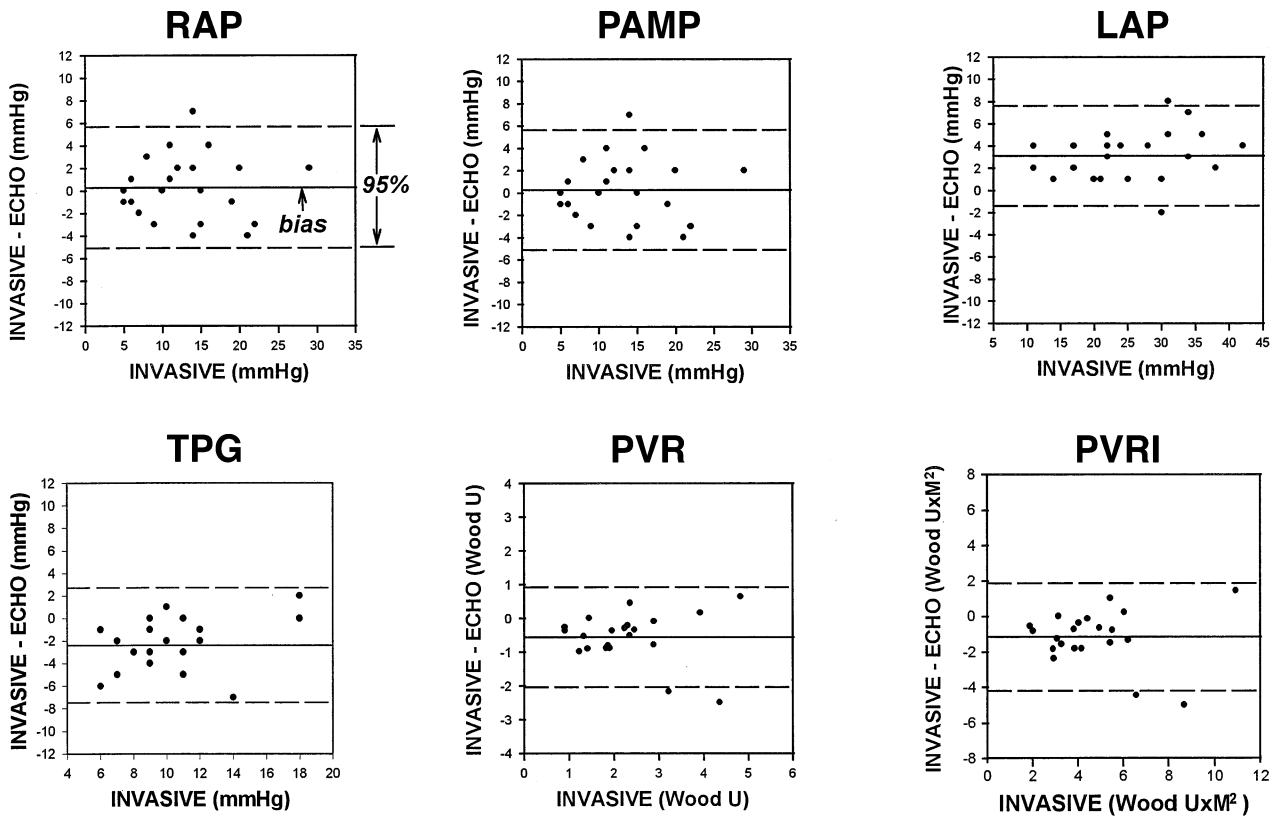

Figure 4. Bland-Altman plots of selected hemodynamic variables. ECHO = echocardiographic.

use (12,36,37). The technique used in the present study relies on the assessment of the size and respirophasic reactivity of the inferior vena cava (7–9). The accuracy of RAP values described in the present study is similar to that previously reported (7–9).

Pulmonary artery pressures. The accuracy of the technique used to estimate PASP is well established, and this technique is used in many clinical echocardiography laboratories (9–13). The technique to estimate PADP also has been validated (9,14,15). Doppler echocardiographic estimates of PAMP from the early diastolic velocity of the pulmonary regurgitation signal or from regression equations related to the pulmonary systolic flow acceleration time have been described (13,16,17), but we have not found them to be sufficiently accurate or reproducible for clinical use. We applied an equation commonly used to estimate the mean systemic arterial pressure to the pulmonary arterial circulation. The correlation, bias and 95% confidence limits for the relation between invasive and noninvasive estimates of PAMP were similar to those of its components, PASP and PADP. The errors inherent in these techniques did not appear to summate, despite similar directions of bias. This may have resulted from cancellation of errors in the calculations from individual patients.

LAP. A variety of techniques for the noninvasive estimation of LAP, including subtraction of the end-diastolic pressure gradient between the aorta and left ventricle from the diastolic blood pressure or subtraction of the peak mitral regurgitation spectral Doppler velocity from the systolic blood pressure have been described (17–20). In the absence of moderate or greater degrees of mitral or aortic regurgitation, these data are usually inaccessible. Regression equations incorporating left heart and pulmonary vein echocardiographic variable values, although promising, are not sufficiently accurate for estimation of specific pulmonary vascular hemodynamic values (23–29).

The technique used to estimate LAP in the present study relies on the initial component of the continuous wave mitral regurgitation spectral Doppler signal and assumes that LAP at the time of aortic valve opening can be used as a surrogate for mean LAP (21). That LAP does not change significantly in early systole, even in the presence of significant mitral regurgitation, has been demonstrated (20). The accuracy of the modified Bernoulli equation for determining instantaneous systolic pressure differences between the left ventricle and atrium has been validated and is used clinically for estimation of left ventricular dP/dt and tau (38–46). Frequent measurement of diastolic blood pressure at different phases of the respiratory cycle overcomes problems with respirophasic variation in LAP. The accuracy of the oscillometric technique for measuring diastolic blood pressure has been validated against central aortic pressures (30). This technique critically depends on proper cuff placement and cuff size, both of which were carefully monitored in the present study.

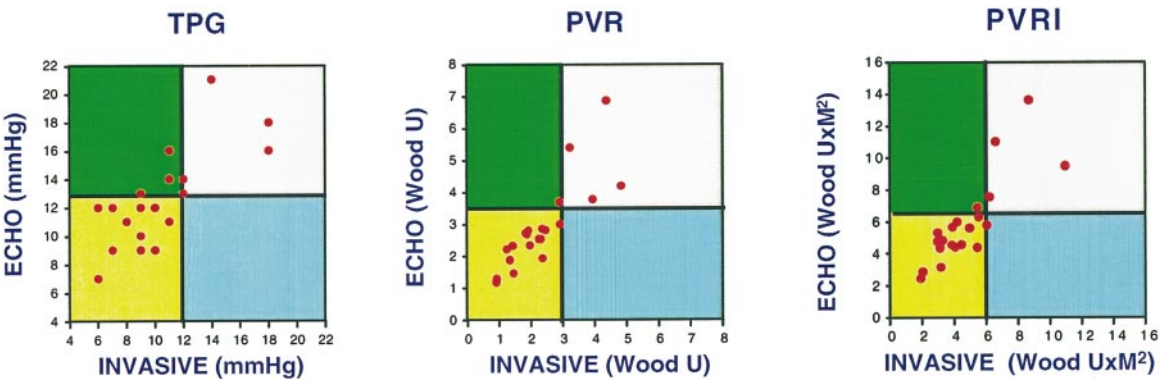

Figure 5. Scatterplots of invasive and noninvasive pulmonary vascular hemodynamic data sets. Each scatterplot is divided into four quadrants by the invasive and noninvasive hemodynamic thresholds (see text). **Yellow areas** = right heart catheterization not required, correctly predicted by echocardiography; **green areas** = right heart catheterization not required, incorrectly predicted by echocardiography; **white areas** = right heart catheterization required, correctly predicted by echocardiography; **blue areas** = need for right heart catheterization missed by echocardiography.

CO. The technique used to estimate CO has been validated and is used in many clinical echocardiography laboratories (31–34).

Accessibility of Doppler echocardiographic data. Because patients with congestive heart failure frequently have valvular regurgitation, acquisition of a complete Doppler echocardiographic data set was possible in 84% of patients. Because the inferior vena cava is easily visualized by two-dimensional echocardiography, RAP could be estimated in all patients. Cardiac index and CO also were accessible in all patients. The observation that the pulmonary regurgitation spectral Doppler velocity signal was accessible and sufficient to estimate PADP in each subject is in agreement with previously reported findings (9,14). Tricuspid regurgitation is common in patients with heart failure, and its spectral Doppler signal is easily accessed from multiple transducer locations (9). The variable that limited acquisition of a complete data set usually was LAP, noninvasive determination of which required detection of the initial portion of the spectral Doppler mitral regurgitation signal in the present study. Because mitral regurgitation is common in patients with heart failure, it is not surprising that this signal could be detected in most patients with careful interrogation.

Although echocardiographic contrast agents were not administered in our study, their use may increase the accessibility of both right- and left-sided Doppler signals, potentially increasing the number of patients to whom these Doppler-based techniques could be applied.

Application to orthotopic heart transplantation evaluation. Because of the high mortality associated with heart transplantation in patients with pulmonary hypertension, it is necessary that any method proposed as a substitute for preoperative right heart catheterization be very sensitive for the identification of patients with an adverse pulmonary vascular hemodynamic status. Specificity is a less critical issue because patients with suspected pulmonary hypertension by echocardiography would be referred for right heart catheterization to confirm this diagnosis and to assess pulmonary vascular reactivity. Invasive assessment of pulmonary vascular hemodynamic status was considered indicated if any invasive predictor value exceeded the threshold value; hence, noninvasive thresholds were chosen such that the Doppler echocardiographic algorithm was 100% sensitive. That is, the thresholds for the noninvasive pulmonary vascular hemodynamic variables were chosen to identify all patients with adverse invasive pulmonary vascular variable values. By design, they did not erroneously categorize any patient with pulmonary hypertension as normal. In the present study, almost 75% of all right heart catheterizations could potentially have been avoided.

Limitations of the study. Invasive and noninvasive data sets were not acquired simultaneously, but invasive measurements were obtained within 30 min of the Doppler echocardiographic study, without interventions that might affect intracardiac filling pressures (i.e., administration of intravenous fluid, diuretic drugs, vasodilators or sedatives). Although fluid-filled Swan-Ganz catheters were used rather than micromanometers, this was considered appropriate because they are routinely used for clinical decision making. The potential that thermodilution CO measurements may have been altered by tricuspid regurgitation was not a significant problem in this study because significant tricuspid regurgitation was uncommon.

The learning curve for accurate acquisition and interpretation of Doppler echocardiographic data requires that each of these noninvasive techniques be validated in individual laboratories before application to patient care. The Doppler echocardiographic technique for estimation of LAP requires meticulous evaluation of hard-copy printouts of spectral Doppler signals from multiple cardiac cycles to minimize beat to beat variability and the effects of respiration. Currently, this technique is laborious and critically dependent on identification of the leading edge of the aortic valve opening click (21). Software for computer-assisted LAP determination has been developed and may make this technique easier to use, faster to perform and easier to teach (21). Finally, the ability of Doppler

echocardiographic measurements to track interventions has not yet been demonstrated, and the noninvasive algorithm for determining hemodynamic suitability for heart transplantation has not been prospectively evaluated.

Conclusions. Doppler echocardiographic estimates of hemodynamic variables in patients with advanced heart failure are accurate and reproducible. These techniques depend on meticulous acquisition and interpretation of two-dimensional echocardiographic images and spectral Doppler signals that are readily accessible in patients with advanced heart failure. This noninvasive methodology may obviate the need for routine right heart catheterization in many potential heart transplantation candidates and could assist with monitoring and optimization of medical therapy in patients with severe congestive heart failure.

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