

# Noninvasive Evaluation of Pulmonary Capillary Wedge Pressure in Patients With Acute Myocardial Infarction by Deceleration Time of Pulmonary Venous Flow Velocity in Diastole

Atsushi Yamamuro, MD,\* Kiyoshi Yoshida, MD, FACC,\* Takeshi Hozumi, MD,\* Takashi Akasaka, MD,\* Tsutomu Takagi, MD,\* Shuichirou Kaji, MD,\* Takahiro Kawamoto, MD,\* Junichi Yoshikawa, MD, FACC†

*Kobe and Osaka, Japan*

- OBJECTIVES** This study investigates the correlation between deceleration time of diastolic pulmonary venous flow (PV-DT) and of early filling mitral flow (LV-DT), and pulmonary capillary wedge pressure (PCWP) in patients with acute myocardial infarction (AMI).
- BACKGROUND** An earlier study suggests that Doppler-derived LV-DT provides an accurate means of estimating PCWP in postinfarction patients with left ventricular systolic dysfunction. Furthermore, recent studies have suggested that PCWP correlates better with PV-DT than with LV-DT. However, the value of PV-DT and LV-DT for assessment of PCWP in patients with AMI has not been evaluated.
- METHODS** In 141 consecutive patients with AMI, we measured PV-DT and LV-DT by Doppler echocardiography, and compared these variables with PCWP measured using a Swan-Ganz catheter.
- RESULTS** There was a weak negative correlation between the LV-DT and PCWP ( $r = -0.54$ ). Although the sensitivity of  $\leq 130$  ms in LV-DT in predicting  $\geq 18$  mm Hg in PCWP was high (86%), its specificity was low (59%). On the other hand, a very close negative correlation was found between PV-DT and PCWP ( $r = -0.89$ ). The sensitivity and specificity of  $\leq 160$  ms in PV-DT in predicting  $\geq 18$  mm Hg in PCWP were 97% and 96%, respectively.
- CONCLUSIONS** In patients with AMI, Doppler-derived PV-DT showed a stronger correlation with PCWP than LV-DT. (J Am Coll Cardiol 1999;34:90-4) © 1999 by the American College of Cardiology

In patients with acute myocardial infarction (AMI), evaluation of the pulmonary capillary wedge pressure (PCWP) provides the assessment of hemodynamic severity and aids in the decision of therapeutic interventions (1). The PCWP has often been clinically evaluated by using a balloon-tipped pulmonary artery catheter (Swan-Ganz) in patients with AMI (1,2), the use of which is not without risks (3). If it becomes possible to repeatedly estimate the PCWP under noninvasive procedures without implanting a Swan-Ganz catheter, it could be useful in the management of patients with AMI. An earlier study has shown that Doppler-derived deceleration time of early filling mitral flow (LV-DT) provides a simple and accurate means of estimating the PCWP in postinfarction patients (>3 months) with left

ventricular systolic dysfunction (4). Furthermore, recent studies have suggested that the PCWP correlates better with the deceleration time of diastolic pulmonary venous flow (PV-DT) than with LV-DT (5). However, the value of PV-DT and LV-DT for assessment of PCWP has not been evaluated in patients with AMI. Accordingly, this study was undertaken to investigate the correlations of PV-DT and LV-DT with the PCWP in patients with AMI, and to compare the value of PV-DT and of LV-DT.

## METHODS

**Patients.** One hundred and seventy-five consecutive patients with AMI (within 1 week after onset;  $2.1 \pm 1.0$  days, range 1 to 7) underwent Doppler echocardiographic evaluation. The diagnosis of AMI was made if the patient had typical chest pain lasting >30 min, characteristic electrocardiographic changes and a typical creatine phosphokinase curve. Patients were excluded if they had a prior Q-wave

From the \*Division of Cardiology, Kobe General Hospital, Kobe, Japan; and †Osaka City University School of Medicine, Osaka, Japan.

Manuscript received October 30, 1998; revised manuscript received February 25, 1999, accepted March 31, 1999.

#### Abbreviations and Acronyms

AMI	= acute myocardial infarction
LV-DT	= deceleration time of early filling mitral flow
LVEDV	= left ventricular end-diastolic volume
LVESV	= left ventricular end-systolic volume
PCWP	= pulmonary capillary wedge pressure
PV-DT	= deceleration time of diastolic pulmonary venous flow
PVF	= pulmonary venous flow

myocardial infarction. Exclusion criteria were: inadequate Doppler recordings ( $n = 3$ ), nonsinus rhythms ( $n = 10$ ), merging of peak velocity during early diastole and atrial contraction mitral flow velocities ( $n = 6$ ), mitral stenosis of any degree ( $n = 1$ ), more than mild mitral regurgitation by color Doppler flow study ( $n = 7$ ), aortic stenosis with a peak velocity of  $>2$  m/s ( $n = 2$ ) and inadequate PCWP tracings ( $n = 5$ ). Finally, 141 patients (32 women, 109 men; age  $63 \pm 8$  years, range 44 to 78) were included in the study group. The study patients were grouped according to their level of PCWP, into those with a PCWP of  $<18$  mm Hg (group 1) and those with a PCWP of  $\geq 18$  mm Hg (group 2). The study protocol was approved by the Committee for the Protection of Human Subjects in Research at Kobe General Hospital. Before the hemodynamic study, all patients gave their informed consent after full explanation of the procedure.

**Hemodynamic measurements.** Pressure measurements were calibrated before and during the study and referenced to the midaxillary line. Verification of wedge position was achieved using changes in pulmonary artery pressure, waveforms and, when present, changes in mixed venous oxygen display. Pulmonary capillary wedge pressure was obtained at end-tidal apnea by an independent investigator. Arterial pressure was measured using a radial cannula or a 7-F pigtail catheter placed in the descending thoracic aorta.

**Echocardiography.** All patients were examined in the left lateral position by precordial two-dimensional and Doppler echocardiography. A Hewlett-Packard (model SONOS 2500 or SONOS 5500) ultrasound instrument with a combined 3.25-MHz imaging/2.5-MHz Doppler transducer was used. Left ventricular end-diastolic and end-systolic volumes (LVEDV and LVESV) were determined from apical two- and four-chamber views by using the Simpson biplane formula according to the recommendations of the American Society of Echocardiography (6). Tracing of the endocardial borders was performed on a digitized frame from the technically best cardiac cycle. Ejection fraction was calculated as  $(LVEDV - LVESV)/LVEDV$ .

Doppler ultrasound examinations were performed immediately before hemodynamic study. Mitral flow velocities were recorded using an apical four-chamber view, placing a

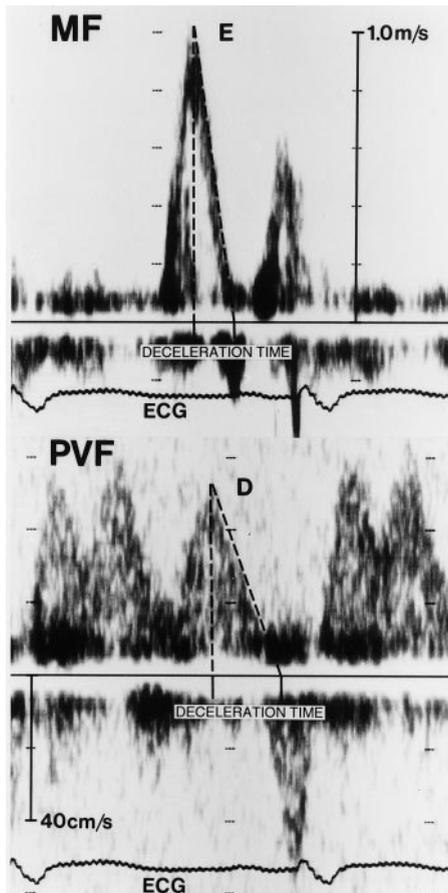
pulsed wave Doppler sample volume between the tips of the mitral leaflets, where maximal flow velocity was recorded. Pulmonary venous flow (PVF) velocities were obtained from an apical four-chamber view. Left atrial filling from the pulmonary vein is characterized by red signals along the interatrial septum in the upper part of the left atrium in the color Doppler mode. The orifice of the right pulmonary vein is imaged at the bottom of the flame-like red signals, and the pulsed Doppler sample volume was set at 0.5 to 1.0 cm into the upper right pulmonary vein. Filters were set to the minimum and gain settings were adjusted carefully at each depth to obtain an optimal spectral display. In each patient, three cardiac cycles obtained during end-tidal volume apnea with the most satisfactory signal/noise ratio were selected for analysis and averaged. The horizontal sweep speed was 100 mm/s. The following variables were derived from Doppler tracings of mitral flow and PVF. To obtain PV-DT, a line was drawn from the peak early diastolic velocity along the fall in initial velocity and extrapolated to the baseline. The PV-DT was measured from the peak early diastolic velocity to when the extrapolated line intersected the baseline (7) (Fig. 1, lower panel). From Doppler tracings of mitral flow, the LV-DT was measured in a manner similar to that of the PV-DT (Fig. 1, upper panel). All studies were recorded on an S-VHS video tape.

**Statistical analysis.** All data are expressed as mean values  $\pm$  SD. Groups were compared for clinical characteristics, echocardiographic data and hemodynamic data using the unpaired Student *t* test. Linear regression analysis was used to evaluate the correlations of PV-DT and LV-DT with PCWP and the correlation between PV-DT and LV-DT. To test intraobserver and interobserver variability, two independent observers measured PV-DT and LV-DT on videotape recordings containing selected beats (identified using the frame counter) from 20 randomly selected patients. The same beats were analyzed by one of the two observers one month later. Interobserver and intraobserver variabilities were calculated as coefficients of variation. A probability level of  $<0.05$  was considered significant. Sensitivity and specificity were calculated with standard formulas.

## RESULTS

### Hemodynamic variables and echocardiographic data.

The study patients were assigned to the following two groups: group 1, with a PCWP  $<18$  mm Hg (106 patients, 75%) and group 2, with a PCWP  $\geq 18$  mm Hg (35 patients, 25%). The clinical characteristics, hemodynamic data and echocardiographic data of the groups are presented in Table 1. Mean age and systolic blood pressure were similar in the two patient groups. Patients in group 1 had a lower mean heart rate ( $p < 0.01$ ) than patients in group 2. Patients in group 1 had a greater LV-DT ( $p < 0.001$ ) than patients in group 2. Patients in group 1 also had a greater PV-DT ( $p < 0.001$ ) than patients in group 2. Patients in group 1 had a



**Figure 1.** Recording of the mitral flow (MF) velocity curve (upper panel) and pulmonary venous flow (PVF) velocity curve (lower panel) in a patient with acute myocardial infarction. Deceleration time of mitral flow velocities in early diastole is measured from peak of early diastolic flow (E) to extrapolation of slope of velocity deceleration to baseline. Deceleration time of pulmonary vein flow velocities in diastole is measured from peak of diastolic forward flow (D) to extrapolation of slope of velocity deceleration to baseline. ECG = electrocardiogram.

lower LVEDV ( $p < 0.001$ ) with greater left ventricular ejection fraction ( $p < 0.01$ ) than patients in group 2.

**Correlation of mitral and PVF deceleration times with PCWP.** There was a weak negative correlation between LV-DT and PCWP ( $r = -0.54$ , Fig. 2). The value of 130 ms in LV-DT was the cutoff point in predicting 18 mm Hg. The sensitivity of  $\leq 130$  ms in LV-DT in predicting  $\geq 18$  mm Hg in PCWP was 86%, and specificity was 59%. There was a very close negative correlation between PV-DT and PCWP ( $r = -0.89$ , Fig. 3). The value of 160 ms in PV-DT was the cutoff point in predicting 18 mm Hg. The sensitivity and specificity of  $\leq 160$  ms in PV-DT in predicting  $\geq 18$  mm Hg in PCWP were 97% and 96%, respectively. In patients with a decreased ejection fraction ( $< 45\%$ ), the correlation between PV-DT and PCWP was slightly higher ( $r = -0.90$ ) than in patients with preserved left ventricular systolic function ( $r = -0.85$ );

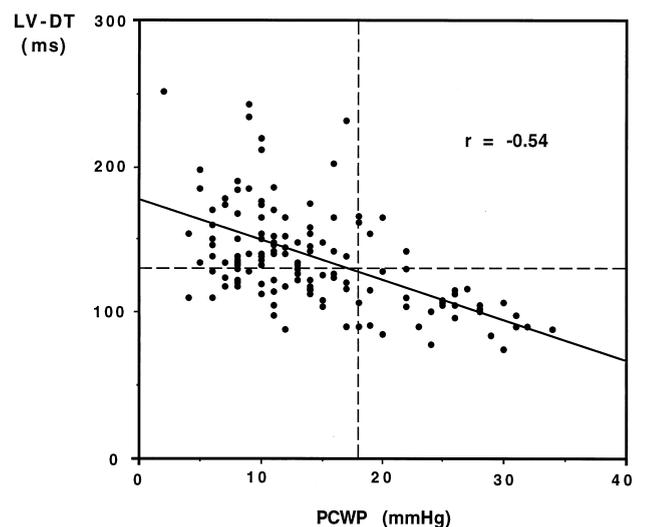
**Table 1.** Clinical, Hemodynamic and Echocardiographic Characteristics

	Pulmonary Capillary Wedge Pressure	
	<18 mm Hg (n = 106)	$\geq 18$ mm Hg (n = 35)
Age (yr)	62 $\pm$ 8	64 $\pm$ 7
Heart rate (beats/min)	79 $\pm$ 15	88 $\pm$ 16*
Pressures (mm Hg)		
Arterial systolic	119 $\pm$ 14	119 $\pm$ 12
Pulmonary capillary wedge	11 $\pm$ 4	24 $\pm$ 5†
Deceleration time of early filling mitral flow (ms)	146 $\pm$ 34	109 $\pm$ 24†
Deceleration time of diastolic pulmonary venous flow (ms)	197 $\pm$ 32	121 $\pm$ 24†
Left ventricular end-diastolic volume (ml)	111 $\pm$ 38	139 $\pm$ 29†
Left ventricular ejection fraction (%)	46 $\pm$ 12	39 $\pm$ 8*

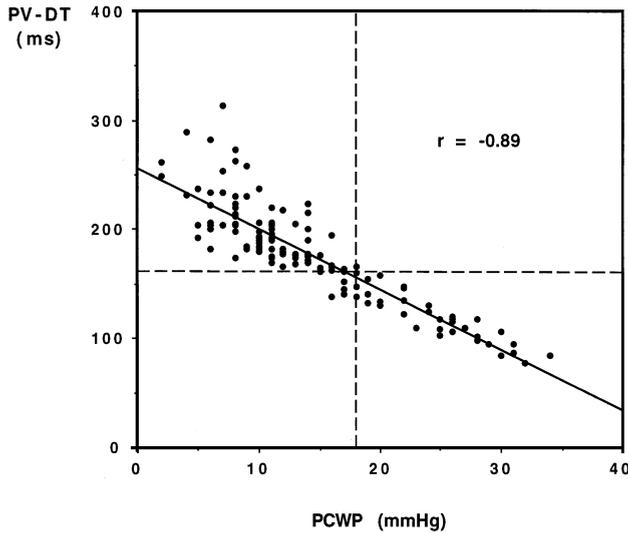
\* $p < 0.01$ , † $p < 0.001$  vs. pulmonary capillary wedge pressure  $< 18$  mm Hg.

however, the difference did not reach statistical significance. Thus, the relation between PV-DT and PCWP did not depend on the level of left ventricular systolic function.

**Correlation of mitral deceleration times with PVF deceleration times.** There was a modest correlation between the LV-DT and PV-DT in all the patients with AMI ( $r = 0.57$ , Fig. 4). A modest positive correlation ( $r = 0.66$ , Fig. 4) was also found between LV-DT and PV-DT in group 2; however, only a weak positive correlation was found in group 1 ( $r = 0.35$ , Fig. 4).



**Figure 2.** Scatterplot of the correlation between mitral deceleration time (LV-DT) and pulmonary capillary wedge pressure (PCWP). The horizontal dashed line marks the value of 130 ms in LV-DT that was found to be the cutoff point in predicting the level of 18 mm Hg in PCWP.



**Figure 3.** Scatterplot of the correlation between deceleration time of pulmonary venous flow velocities in diastole (PV-DT) and pulmonary capillary wedge pressure (PCWP). The **horizontal dashed line** marks the value of 160 ms in PV-DT that was found to be the cutoff point in predicting the level of 18 mm Hg in PCWP.

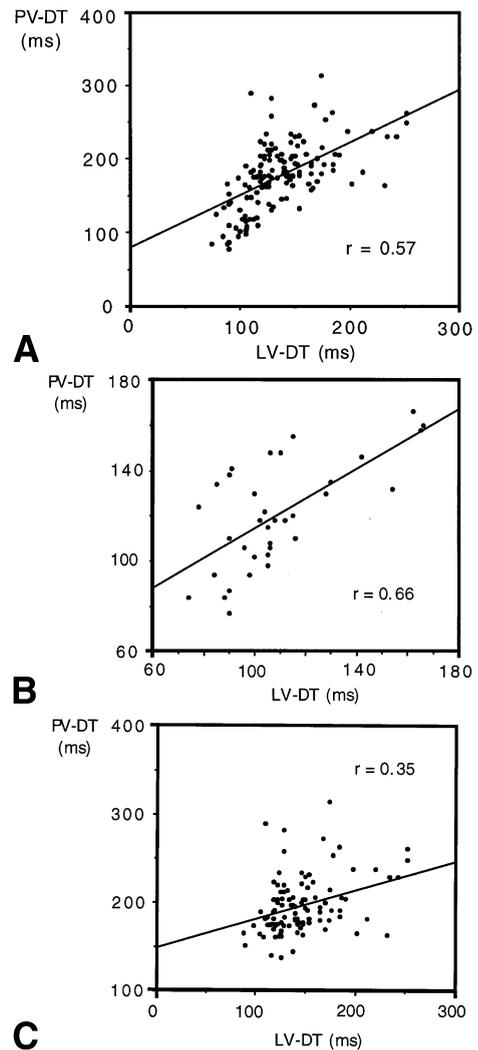
**Intraobserver and interobserver variability.** The mean percentages of interobserver and intraobserver variability for measurement of LV-DT were 2.9% and 2.5%, respectively; for measurement of PV-DT time they were 3.4% and 2.8%, respectively.

**DISCUSSION**

In the present study, we demonstrated that the PV-DT correlated more strongly with PCWP than did LV-DT in patients with AMI. To our knowledge, this is the first report demonstrating the usefulness of PV-DT in predicting PCWP in patients with AMI. Although the LV-DT provides an accurate means of estimating PCWP in postinfarction patients (>3 months) with left ventricular systolic dysfunction (4), the correlation between the LV-DT and PCWP was weak in patients with AMI.

It has been suggested that there is a direct correlation between the LV-DT and the PV-DT under different loading conditions (7). Pulmonary capillary wedge pressure has been estimated by measuring LV-DT, which is relatively easy to measure. However, increased left atrial contribution to left ventricular function by its booster pump function and reduced compliance of the left ventricle have been reported in patients with AMI (8-16). In an experimental model (17), LV-DT has been found to depend strictly on left ventricular chamber stiffness. On the other hand, PV-DT might depend mainly on the initial driving pressure of the PVF and the compliance of the left atrium and be less dependent on left ventricular compliance (5). Therefore, the correlation between PV-DT and LV-DT can be decreased in patients with AMI with reduced left

ventricular compliance and maintained left atrial compliance. In the present study, there was a weaker correlation between the LV-DT and PV-DT in patients with AMI and a PCWP of <18 mm Hg, whereas good correlations have been demonstrated in patients without AMI in previous studies (7,18). Thus, PV-DT may be more appropriate than LV-DT as an indicator of PCWP in patients with AMI. This finding is particularly important in patients with an increased left atrial contribution to left ventricular function and a reduced compliance of the left ventricle. In this context, PV-DT appears to be extremely useful in differentiating whether a patient has an elevated PCWP or not, even if a patient has a reduced left ventricular compliance.



**Figure 4.** (A) Scatterplot of the correlation between deceleration time of pulmonary venous flow velocities in diastole (PV-DT) and mitral deceleration time (LV-DT) in all patients. (B) Scatterplot of the correlation between PV-DT and LV-DT in patients with a pulmonary capillary wedge pressure (PCWP) of  $\geq 18$  mm Hg. (C) Scatterplot of the correlation between PV-DT and LV-DT in patients with a PCWP of <18 mm Hg.

**Limitations of the study.** Determination of PCWP with the proposed Doppler equation can be performed in 83% of patients with AMI from recording of PV-DT and LV-DT; criteria for most of the exclusions are patients with merging of the early diastole and atrial contraction mitral flow velocities or absence of sinus rhythm. Cardiac arrhythmias, especially atrial fibrillation, are a problem because of the loss of the mitral A wave and a decrease in pulmonary venous systolic flow (19). Nevertheless, there is a recent study which suggests that PCWP can be estimated by calculating the mean PV-DT of five consecutive heart beats even in patients with atrial fibrillation (5). Second, patients with moderate or severe mitral regurgitation were excluded on the hypothesis that this would affect the correlations of PV-DT and LV-DT with PCWP. Although it has been reported that mitral regurgitation can result in an increase in early mitral flow velocity (20), no correlation has been found between the severity of mitral regurgitation and the amplitude of either the pulmonary wedge v wave or peak early mitral flow velocity, except in patients with severe acute mitral regurgitation. Furthermore, a recent study suggests that PCWP can be reliably estimated even when mitral regurgitation is present by combining Doppler echocardiographic variables of mitral flow and PVF (21).

Because we focused on comparison of the values of LV-DT and of PV-DT in the present study patients, we did not investigate other indexes to estimate PCWP. Further investigations are necessary to confirm whether other indexes can be estimated in patients with AMI. Furthermore, we did not evaluate the changes in mitral flow and PVF patterns after the hemodynamic condition had changed in the present study. In future studies, it will be necessary to evaluate the effects of postunloading treatment and the possibility of predicting hemodynamic data improvement based on Doppler data. Finally, we did not assess the left atrial function in the present study. Further investigation would be necessary to determine whether left atrial function affected the differences between the correlations of PV-DT and LV-DT with the PCWP.

**Conclusions.** In patients with AMI, Doppler-derived PV-DT showed a stronger correlation with PCWP than LV-DT.

---

**Reprint requests and correspondence** Dr. Atsushi Yamamuro, Minatojima-nakamachi 4-6, Chuo-ku, Kobe, 650, Japan. E-mail: jse@warp-or-jp.

---

## REFERENCES

1. Forrester JS, Diamond G, Chatterjee K, Swan HJC. Medical therapy of acute myocardial infarction by application of hemodynamic subsets. *N Engl J Med* 1976;295:1356-62.

2. Swan HJC, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *N Engl J Med* 1979;283:447-51.
3. Foote GA, Schabel SI, Hodges M. Pulmonary complications of the flow-directed balloon-tipped catheter. *N Engl J Med* 1974;290:927-31.
4. Giannuzzi P, Imparato A, Temporelli PL, et al. Doppler-derived mitral deceleration time of early filling as a strong predictor of pulmonary capillary wedge pressure in postinfarction patients with left ventricular systolic dysfunction. *J Am Coll Cardiol* 1994;23:1630-7.
5. Chirillo F, Brunazzi MC, Barbiero M, et al. Estimating mean pulmonary wedge pressure in patients with chronic atrial fibrillation from transthoracic Doppler indexes of mitral and pulmonary venous flow velocity. *J Am Coll Cardiol* 1997;30:19-26.
6. Schiller N, Shah PM, Crawford M, et al. Recommendations for quantification of the left ventricle by two-dimensional echocardiography: American Society of Echocardiography Subcommittee on Standards. *J Am Soc Echocardiogr* 1989;2:358-68.
7. Nishimura RA, Abel MD, Hatle LK, Tajik AJ. Relation of pulmonary vein to mitral flow velocities by transesophageal Doppler echocardiography: effect of different loading conditions. *Circulation* 1990;81:1488-97.
8. Linden RJ, Mitchell JH. Relation between left ventricular diastolic pressure and myocardial segment length and observations on the contribution of atrial systole. *Circ Res* 1960;8:1092-9.
9. Samet P, Bernstein W, Levine S. Significance of the atrial contribution to ventricular filling. *Am J Cardiol* 1965;15:195-202.
10. Hawley RR, Dodge HT, Graham TP. Left atrial volume and its changes in heart disease. *Circulation* 1966;34:989-96.
11. Murray JA, Kennedy JW, Figley M. Quantitative angiocardio-graphy. II. The normal left atrial volume in man. *Circulation* 1968;37:800-4.
12. Rahimtoola SH, Ehsani A, Sinno MZ, Loeb HS, Rosen KM, Gunnar RM. Left atrial transport function in myocardial infarction. Importance of its booster pump function. *Am J Med* 1975;59:686-94.
13. Stott DK, Marpole DGF, Bristow JD, Kloster FE, Griswold HE. The role of left atrial transport in aortic and mitral stenosis. *Circulation* 1970;41:1031-41.
14. Matsuda Y, Toma Y, Ogawa H, et al. Importance of left atrial function in patients with myocardial infarction. *Circulation* 1983;67:566-71.
15. Kumar R, Hood, WB Jr, Joison J, Norman JC, Abelmann WH. Experimental myocardial infarction. II. Acute depression and subsequent recovery of left ventricular function. Serial measurements in intact conscious dogs. *J Clin Invest* 1970;49:55-62.
16. Diamond G, Forrester JS, Hargis J, Parmley WW, Danzig R, Swan HJC. Diastolic pressure-volume relationship in the canine left ventricle. *Circ Res* 1971;29:267-75.
17. Little WC, Ohno M, Kitzman DW, Thomas JD, Cheng CP. Determination of left ventricular chamber stiffness from the time for deceleration of early left ventricular filling. *Circulation* 1995;92:1933-9.
18. Rossvoll O, Hatle LK. Pulmonary venous flow velocities recorded by transthoracic Doppler ultrasound: relation to left ventricular diastolic pressures. *J Am Coll Cardiol* 1993;21:1687-96.
19. Keren G, Bier A, Sherez J, Miura D, Keefe D, LeJemtel T. Atrial contraction is an important determinant of pulmonary venous flow. *J Am Coll Cardiol* 1986;7:693-5.
20. Thomas JD, Weyman AE. Echocardiographic Doppler evaluation of left ventricular diastolic function. Physics and physiology. *Circulation* 1991;84:977-90.
21. Pozzoli M, Capomolla S, Pinna G, Cobelli F, Tavazzi L. Doppler echocardiography reliably predicts pulmonary artery wedge pressure in patients with chronic heart failure with and without mitral regurgitation. *J Am Coll Cardiol* 1996;27:883-93.