Am J Physiol Heart Circ Physiol 293: H120–H125, 2007.
First published February 23, 2007; doi:10.1152/ajpheart.00097.2007.

Relationship between tissue Doppler-derived RV systolic function and invasive hemodynamic measurements

Yasutomo Hori, Takuto Kano, Fumio Hoshi, and Sei-ichi Higuchi

Department of Small Animal Internal Medicine, School of Veterinary Medicine, Kitasato University, Towada, Aomori, Japan

Submitted 24 January 2007; accepted in final form 23 February 2007

Hori Y, Kano T, Hoshi F, Higuchi S-i. Relationship between tissue Doppler-derived RV systolic function and invasive hemodynamic measurements. *Am J Physiol Heart Circ Physiol* 293: H120–H125, 2007. First published February 23, 2007; doi:10.1152/ajpheart.00097.2007.— Tissue Doppler imaging (TDI) is effective in assessing right ventricular (RV) function, but the relationship between invasive measurements and RV-TDI remains unclear. We investigated the RV systolic function by using the TDI-derived systolic myocardial (Sa) velocity and myocardial performance index (MPI). Beagles ($n = 7$) were anesthetized in the right lateral recumbent position. A 3.5-Fr micromanometer-tipped catheter was placed in the RV to determine the hemodynamic changes. Dobutamine (5.0 and 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and esmolol (50 and 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$) were infused intravenously. Pulsed Doppler (PD) and TDI measurements were performed in the apical four-chamber view. Compared with baseline, the PD-MPI decreased significantly with the dobutamine infusion at 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$). Both dobutamine infusions significantly decreased the TDI-MPI ($P < 0.01$, $P < 0.05$). Esmolol increased the PD- and TDI-MPI but not significantly. Dobutamine significantly increased the Sa velocity (both $P < 0.001$), whereas esmolol had no effect. The Sa velocity was strongly correlated with the peak positive derivative of the RV pressure (+dP/dt; $r = 0.93$). The negative correlation between the +dP/dt and TDI-MPI ($r = -086$) was greater that with the PD-MPI ($r = -0.54$). Stepwise regression analysis showed that the Sa velocity and PD-derived isovolumic contraction time were identified to predict the +dP/dt ($r = 0.94$, $r^2 = 0.89$; $P < 0.001$). We determined that the systolic myocardial velocity and TDI-MPI were strongly correlated with the RV contractility. These results suggest that the TDI-derived systolic myocardial velocity and MPI predict RV systolic function.

dog; right ventricle; systolic myocardial velocity; tissue Doppler imaging

GIVEN THE DIFFICULTY IN DETERMINING the right ventricular (RV) endocardial surface and the complexity of its shape, conventional Doppler echocardiographic evaluation of RV systolic or diastolic function is inaccurate. Several recent reports have shown the utility of tissue Doppler imaging (TDI) for examining left ventricular diastolic function (6, 16). TDI of the tricuspid annulus is also useful for assessing systolic and diastolic function (22). Clinical studies have reported that the systolic myocardial velocity is related to RV systolic function, as determined by using noninvasive examinations, including magnetic resonance imaging (MRI) and two-dimensional echocardiography (12, 18). In addition, the myocardial performance index (MPI) is a noninvasive pulsed Doppler (PD) measurement that can evaluate ventricular systolic and diastolic function (7, 13). The MPI indicates the severity of and provides prognostic information about heart disease, such as aortic valve stenosis, dilated cardiomyopathy, and mitral valve regurgitation (3–5, 10). In addition, a modified MPI derived from the TDI has been used to evaluate left ventricular function in clinical studies (6, 8, 11).

However, the relationship between invasive measurements of RV systolic function and the TDI-derived systolic myocardial velocity remains unclear. In addition, the relationship between the PD-MPI and TDI-MPI derived from the tricuspid annulus as RV systolic function is also unclear. We therefore examined the accuracy of systolic myocardial velocity and the MPI recorded from the tricuspid annulus as measures of RV systolic function.

MATERIALS AND METHODS

Animals. Male beagles ($n = 7$), weighing 8–12 kg, aged 1–2 years, were used for this study. All dogs were housed individually in cages and were fed commercial dry food. The dogs had free access to water. This study followed the Guidelines for Institutional Laboratory Animal Care and Use of the School of Veterinary Medicine at Kitasato University.

After being sedated with butorphanol (0.2 mg/kg iv) and atropine (0.025 mg/kg sc), the dogs were anesthetized with propofol (6.0 mg/kg iv) and intubated. Anesthesia was maintained with 2.0% isoflurane in oxygen. The dogs were positioned in the left lateral recumbent position. The respiratory rate was maintained with an artificial ventilator (KV-1a; Kimura Medical Instrument, Tokyo, Japan). The end-tidal P_aCO_2 was monitored and maintained between 35 and 45 mmHg, and the heart rate was monitored by using an electrocardiogram (COLIN BP-608; Nihon Kohden, Tokyo, Japan). Under fluoroscopic guidance, a high-fidelity 3.5-Fr micromanometer-tipped catheter (Millar Instruments, Houston, TX) was placed through the right jugular vein into the right ventricle. The peak systolic RV pressure and end-diastolic RV pressure were measured; the peak positive (+dP/d*t*) and negative (−dP/d*t*) first derivatives of the RV pressure were calculated from the RV pressure profiles recorded with a micromanometer catheter. After the procedures were completed, a 20- to 30-min stabilization period was allowed to establish a stable baseline condition for the hemodynamic and echocardiographic measurements. All measurements were recorded initially to establish baselines.

Drug administration. Dobutamine was infused at rates of 5.0 and 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ for 5 min via the cephalic vein (16, 17). Measurements were made at both doses of dobutamine. Similarly, esmolol was infused at 50 and 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ (6). After the first procedure was completed, a stabilization period of at least 30 min was provided to allow a return to the stable baseline condition.

Echocardiography. PD echocardiography was used to measure the tricuspid valve inflow velocity in the four-chamber view with the sample volume positioned at the tips of the tricuspid valve leaflets.

Address for reprint requests and other correspondence: Y. Hori, Dept. of Small Animal Internal Medicine, School of Veterinary Medicine, Kitasato Univ., 23-35-1 Higashi, Towada, Aomori 034-8628, Japan (e-mail: hori@vmas.kitasato-u.ac.jp).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

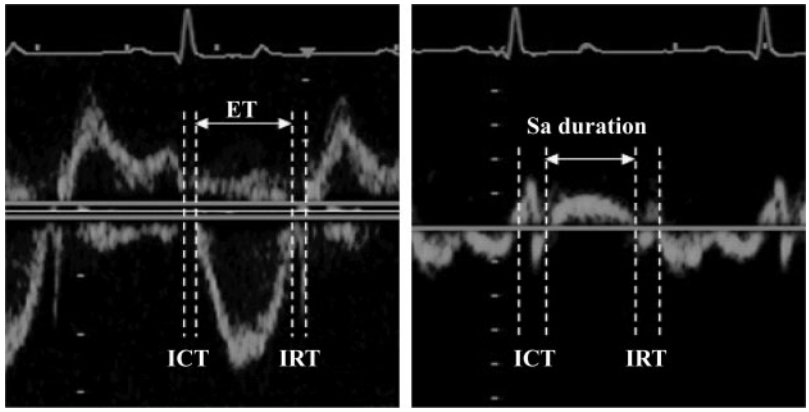

Fig. 1. Pulsed Doppler (PD)-derived myocardial performance index (MPI; *left*) and tissue Doppler-derived modified MPI (*right*). ET, ejection time; ICT, isovolumic contraction time; IRT, isovolumic relaxation time; Sa, ventricular systolic velocity.

Then the peak early (E wave) and late (A wave) diastolic inflow velocities and the ratio of the E wave to the A wave (E/A ratio) were measured. Pulmonary outflow Doppler measurements were made with the sample volume placed just below the pulmonary valve in an apical long-axis view. The ejection time (ET) was measured from the onset to the end of the pulmonary outflow. Time-interval measurements were made by using the internal analysis package of the ultrasound unit. The intervals were determined by using two carefully placed vertical cursors that were moved with a trackball. From the PD recordings, the MPI index was calculated in the usual way; the total isovolumic (concentration and relaxation) time was divided by the ET (7, 13). The time from the cessation of the tricuspid valve A wave to the onset of the tricuspid valve E wave of the next cardiac cycle (*a*) is equal to the total isovolumic time plus the ET (*b*). The MPI was calculated by using the formula $(a - b)/b$ (Fig. 1). The isovolumic relaxation time (IRT) was calculated by subtracting the interval between the peak of the R wave and the end of the ET from the interval between the peak of the R wave and the onset of the E wave. The isovolumic contraction time (ICT) was calculated by subtracting the ET and IRT from the interval between the cessation of the tricuspid valve A wave and the onset of the tricuspid valve E wave of the next cardiac cycle.

The TDI program was set to pulse-wave Doppler mode. High-pass filtering was removed, and the system settings were adjusted to optimize the TDI data. Gains were minimized to allow for a clear tissue signal with minimal background noise. The TDI velocities were obtained from the four-chamber view. The size of the Doppler sample volume was set at an axial length of 2 mm, and the sample volume was placed at the free wall of the tricuspid valve annulus (22). The peak myocardial velocity was measured during systole (Sa), early diastole (Ea), and late diastole (Aa) (Fig. 1). The ratios of the Ea wave to the Aa wave (Ea/Aa ratio) and of the E wave to the Ea wave (E/Ea ratio) were calculated. From the TDI recordings, the duration of the Sa wave was measured from the onset to the end of the Sa wave. The ICT was measured from the end of the Aa wave to the onset of the Sa wave. The IRT was measured from the end of the Sa wave to the onset of the Ea wave. The modified MPI obtained by using the TDI was calculated as (ICT + IRT)/Sa wave duration (6).

Transthoracic echocardiography was performed by using a SONOS 5500 (Hewlett Packard) and a 12-MHz probe. Echocardiographic data were obtained simultaneously during end-expiratory apnea. The echocardiograms were analyzed by using the commercial analysis software package supplied with the system. The average of three cardiac cycles was calculated. The data were stored digitally and were analyzed off-line by a single observer.

Statistical analysis. The data are given as means ± SD. The changes in the hemodynamic and echocardiographic measurements were compared with baseline at the different states by using one-factor repeated-measures ANOVA. The significance of the differences between the mean values at baseline and each condition was tested by using the post hoc Tukey multiple-comparison test. The MPI derived from the PD was compared with the TDI in each inotropic state by using a paired *t*-test. Single linear regression analysis was used to compare the changes in the echocardiographic measurements with the changes in $+dP/dt$ and $-dP/dt$. A value of $P < 0.05$ was considered statistically significant. Stepwise regression analysis was used to determine the hemodynamic parameters that correlated best with the individual Doppler variables. A value of $F > 2.0$ was considered statistically significant.

RESULTS

The changes in the hemodynamic measurements with the administration of the inotropic agents are summarized in Table 1. Compared with baseline, the heart rate was increased significantly with dobutamine at 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$), whereas it was decreased significantly with esmolol at each dose (both $P < 0.05$). With dobutamine administration, the peak systolic RV pressures were increased significantly from baseline at each dose ($P < 0.05$ and $P < 0.001$, respectively), whereas the end-diastolic RV pressure was decreased signifi-

Table 1. *Changes in hemodynamic measurements during inotrope administration*

	Dobutamine			Esmolol		
	Baseline	5 $\mu g \cdot kg^{-1} \cdot min^{-1}$	10 $\mu g \cdot kg^{-1} \cdot min^{-1}$	Baseline	50 $\mu g \cdot kg^{-1} \cdot min^{-1}$	100 $\mu g \cdot kg^{-1} \cdot min^{-1}$
HR, beats/min	108±13	111±15	133±19*	111±13	105±11*	103±8*
RVP, mmHg	20±2	33±4*	47±13‡	20±2	20±1	20±1
RVEDP, mmHg	2.8±1.4	1.5±1.0	0.8±2.2*	2.3±2.2	3.1±1.5	3.6±1.9
$+dP/dt$, mmHg/s	310±33	812±128‡	1043±146‡	318±66	275±20	260±28*
$-dP/dt$, mmHg/s	−212±21	−432±98*	−767±255‡	−230±41	−188±24	−157±50*

Data are given as means ± SD. HR, heart rate; RVP, right ventricular (RV) systolic pressure; RVEDP, RV end-diastolic pressure; $+dP/dt$, peak positive derivative of RV pressure; $-dP/dt$, peak negative derivative of RV pressure. *$P < 0.05$ vs. baseline; †$P < 0.01$ vs. baseline; ‡$P < 0.001$ vs. baseline.

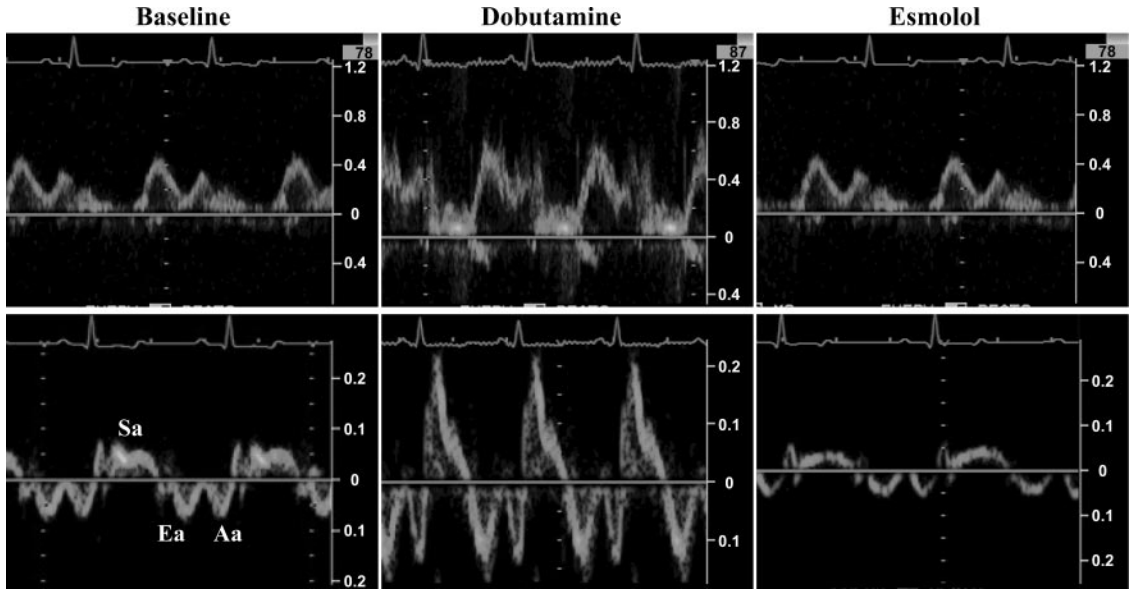

Fig. 2. Representative recordings of the lateral tricuspid annular motion velocity using tissue Doppler imaging (TDI) at baseline (*left*), with dobutamine (*middle*), and with esmolol (*right*). Ea, ventricular early diastolic velocity; Aa, ventricular late diastolic velocity.

cantly at 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$). Esmolol tended to elevate the end-diastolic RV pressure, but the difference was not significant. Compared with baseline, +dP/d*t* was increased significantly with dobutamine at each dose (both $P < 0.001$) but was decreased significantly with esmolol at 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$). Similarly, −dP/d*t* was increased significantly with dobutamine at each dose ($P < 0.05$ and $P < 0.001$, respectively) but was decreased significantly with esmolol at 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$).

The inotropic agent-evoked echocardiographic changes are shown in Fig. 2. The changes in the PD measurements with the inotropic agents are described in Table 2. Although dobutamine evoked an increase in the E wave at each dose ($P < 0.001$) and in the A wave at 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$), esmolol did not change either wave measurement. Consequently, neither dobutamine nor esmolol changed the E/A ratio. The ICT was significantly shortened during dobutamine administration (both $P < 0.001$) compared with baseline, whereas the IRT was not changed. By contrast, the IRT was prolonged significantly with esmolol at 100 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.01$), whereas the ICT was not changed. Compared with baseline, the PD-MPI decreased significantly with dobutamine at 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$). The ET was decreased significantly with dobutamine at 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.001$), and thus no significant changes were seen in the PD-MPI. During esmolol administration, the PD-MPI increased from baseline, but not significantly.

The changes in the TDI measurements with the inotropic agents are described in Table 3. Although dobutamine evoked significant increases in the Sa (both $P < 0.001$), Ea (both $P < 0.001$), and Aa velocities ($P < 0.05$ and $P < 0.01$, respectively), esmolol significantly decreased the Ea velocity (100 $\mu g \cdot kg^{-1} \cdot min^{-1}$; $P < 0.05$) and did not change the Sa or Aa velocity. Consequently, the Ea/Aa ratio was increased significantly with dobutamine at 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ($P < 0.05$) but was not changed with esmolol. During dobutamine administration, ICT and IRT were significantly shortened from baseline ($P < 0.001$ and $P < 0.05$, respectively), whereas they were not changed with esmolol. The duration of the Sa wave was significantly shortened from baseline with dobutamine (both $P < 0.001$) but was not changed with esmolol. As a result, dobutamine significantly decreased the TDI-MPI from baseline ($P < 0.01$ and $P < 0.05$, respectively), whereas esmolol increased it, but not significantly.

On comparing the MPI between the PD and TDI methods, the TDI-MPI was significantly higher than the PD-MPI in each

Table 2. *Changes in left ventricular pulsed Doppler measurements during inotrope administration*

	Dobutamine			Esmolol		
	Baseline	5 $\mu g \cdot kg^{-1} \cdot min^{-1}$	10 $\mu g \cdot kg^{-1} \cdot min^{-1}$	Baseline	50 $\mu g \cdot kg^{-1} \cdot min^{-1}$	100 $\mu g \cdot kg^{-1} \cdot min^{-1}$
E wave, cm/s	35.6±8.5	69.8±8.0‡	76.1±11.3‡	36.9±4.8	37.1±5.0	30.9±8.0
A wave, cm/s	34.0±8.9	47.1±15.1	54.7±9.6*	35.7±8.1	33.7±2.3	31.7±3.4
E/A ratio	1.1±0.4	1.6±0.5	1.4±0.3	1.1±0.2	1.1±0.2	1.0±0.3
ICT, ms	25±13	−6±10‡	−13±9‡	22±10	16±15	20±23
IRT, ms	25±12	10±10	26±29	31±5	30±14	48±12†
ET, ms	218±8	194±18	158±20‡	217±11	219±13	213±23
MPI	0.24±0.07	0.03±0.09*	0.10±0.22	0.25±0.05	0.21±0.09	0.33±0.17

Data are given as means ± SD. E wave, peak early diastolic velocity; A wave, peak late diastolic velocity; ICT, isovolumic contraction time; IRT, isovolumic relaxation time; ET, ejection time; MPI, myocardial performance index. *$P < 0.05$ vs. baseline; †$P < 0.01$ vs. baseline; ‡$P < 0.001$ vs. baseline.

Table 3. *Changes in left ventricular tissue Doppler measurements during inotrope administration*

	Dobutamine			Esmolol		
	Baseline	5 μg·kg^{-1}·min^{-1}	10 μg·kg^{-1}·min^{-1}	Baseline	50 μg·kg^{-1}·min^{-1}	100 μg·kg^{-1}·min^{-1}
Sa, cm/s	5.9±0.9	19.0±3.6‡	22.0±3.3‡	6.1±1.4	6.3±1.3	5.8±1.3
Ea, cm/s	5.9±1.0	14.3±3.3‡	12.4±4.2‡	6.2±1.4	5.6±1.1	5.2±0.5*
Aa, cm/s	6.4±2.5	9.9±3.7*	12.4±2.6†	6.5±1.7	7.3±1.8	7.0±1.5
Ea/Aa ratio	1.0±0.3	1.6±0.5*	1.0±0.4	1.0±0.3	0.8±0.2	0.8±0.1
E/Ea ratio	6.2±1.7	5.2±1.6	6.8±3.0	6.2±1.2	6.8±1.5	6.0±1.4
ICT, ms	45±8	18±9‡	17±10‡	45±3	44±7	49±6
IRT, ms	49±9	30±14*	31±19*	48±9	56±15	58±16
Sa duration, ms	205±13	175±12‡	159±18‡	208±14	208±12	213±13
MPI	0.46±0.05	0.27±0.12†	0.3±0.12*	0.45±0.03	0.48±0.05	0.50±0.08

Data are given as means ± SD. Sa, systolic myocardial velocity; Ea, early diastolic myocardial velocity; Aa, late diastolic myocardial velocity. *$P < 0.05$ vs. baseline; †$P < 0.01$ vs. baseline; ‡$P < 0.001$ vs. baseline.

situation (Fig. 3). The results of the single regression analysis of dP/dt and the echocardiographic measurements are described in Table 4. The Sa velocity was strongly positively correlated with +dP/dt ($r = 0.93$, Fig. 4), whereas there was a negative correlation between +dP/dt and the TDI-MPI ($r = -0.86$, Fig. 5*A*), which was greater than that between +dP/dt and the PD-MPI ($r = -0.54$, Fig. 5*B*). Similarly, there was a positive correlation between −dP/dt and the TDI-MPI ($r = 0.74$), whereas the regression coefficient was reduced for the PD-MPI ($r = 0.36$). The −dP/dt was significantly correlated with the TDI-IRT ($r = 0.59$, $P < 0.001$) but not with the PD-IRT ($r = 0.11$).

Stepwise regression analysis showed that the Sa velocity and PD-ICT could predict +dP/dt ($r = 0.94$, $r^2 = 0.89$; $P < 0.001$). By contrast, the PD-derived E wave velocity, E/A ratio, and IRT and the TDI-derived Aa velocity and Ea/Aa ratio had identical abilities to predict −dP/dt ($r = 0.95$, $r^2 = 0.9$; $P < 0.001$, Table 5).

DISCUSSION

Our study demonstrated that the systolic myocardial velocity derived from the tricuspid annulus is strongly correlated with +dP/dt. Furthermore, stepwise regression analysis showed that the systolic myocardial velocity derived from the tricuspid annulus is a strong predictor of RV systolic function. Recently, the need to assess RV systolic and diastolic function has increased in patients with heart disease. RV function predicts the prognosis and severity in patients with RV dysfunction, such as pulmonary stenosis and tetralogy of Fallot (2, 22). There are several methods for assessing RV function: catheterization, nuclear MRI, and echocardiography (12, 14, 18). The main limitations of these methods are their relatively high cost, demand for time, and invasive nature of catheterization. Recently, several authors showed that TDI-derived myocardial velocities were related to systolic ventricular parameters, as well as to diastolic ventricular motion. The systolic myocardial velocity in the left ventricle is correlated with +dP/dt as a measure of systolic function, which is decreased in patients with heart failure (1, 19, 21). TDI-derived myocardial motion is a noninvasive assessment of RV function that provides clinical and prognostic information on various functional parameters in patients with heart failure (2, 9, 13, 15). A few reports demonstrated that the systolic myocardial velocity of the tricuspid annulus is a sensitive indicator of global RV contractility, which is decreased in patients with RV dysfunction compared with normal subjects (20, 22). In addition, the systolic myocardial velocity was correlated with the ejection fraction measured by using MRI and two-dimensional echocardiography (14, 20). However, data on the relationship between the systolic myocardial velocity derived from the tricuspid annulus and invasive measurements of RV function are rare. Therefore, our results, supported by previous data, suggest that an assessment of RV function using the TDI-derived

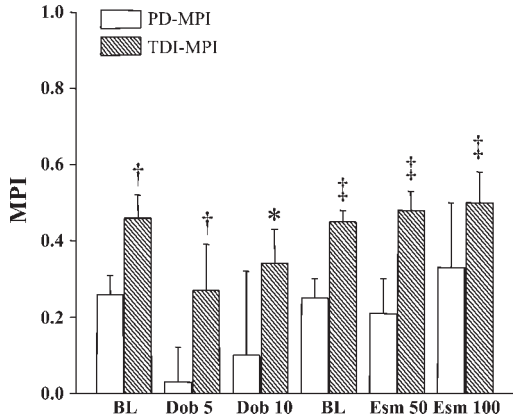

Fig. 3. Comparison between the PD-MPI and TDI-MPI. *$P < 0.05$ vs. baseline; †$P < 0.01$ vs. baseline; ‡$P < 0.001$ vs. baseline.

Table 4. *Single-regression analysis of dP/dt and echocardiographic measurements*

	Pulsed Doppler, r	Tissue Doppler, r
+dP/dt		
Sa wave		0.93‡
ICT	−0.73‡	−0.68‡
MPI	−0.54‡	−0.86‡
−dP/dt		
E wave	−0.82‡	
A wave	−0.71‡	
E/A ratio	−0.34*	
Ea wave		−0.64‡
Aa wave		−0.71‡
Ea/Aa ratio		−0.19
E/Ea ratio		−0.16
IRT	0.11	0.59‡
MPI	0.36*	0.74‡

*$P < 0.05$; ‡$P < 0.001$.

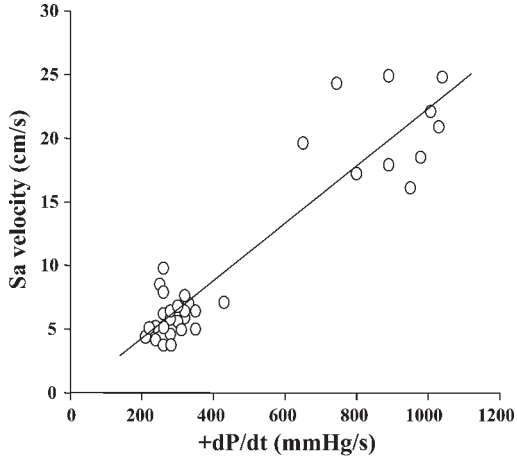

Fig. 4. Relationship between positive derivative of the right ventricular pressure (+dP/d*t*) and Sa wave velocity. These parameters were significantly correlated ($r = 0.93$, $P < 0.001$).

Table 5. *Stepwise regression analysis to determine dP/dt* and individual Doppler variables

	β	*F* Value
+dP/d*t*		
Sa wave pulsed Doppler	0.76	98.1
ICT	−0.26	11.0
−dP/d*t*		
E/A ratio	0.76	39.4
E wave pulsed Doppler	0.67	22.7
IRT	0.75	36.3
Aa wave	0.40	12.8
Ea/Aa ratio	0.27	7.8

β, standard regression coefficient.

tricuspid annulus motion provides functional information on longitudinally oriented RV myocardial fibers (12).

The MPI is a useful clinical index of global ventricular function for evaluating both systolic and diastolic function (7, 13). The PD-MPI has gained acceptance as a clinical examination for assessing cardiac function and is particularly useful as a predictor of clinical outcome in patients with cardiac disease (3–5, 10). The main advantage of this index is that it appears to be independent of ventricular geometry and heart rate (5). However, one of the main limitations of the PD-MPI is that it cannot be calculated over a single cardiac cycle because the interval between the end and onset of mitral inflow and the ET are measured sequentially. By contrast, the TDI can provide the timing elements necessary to calculate the MPI on a beat-to-beat basis (6). In addition, the TDI derived from the mitral annulus velocities allows the determination of the isovolumic contraction and relaxation times and the ET over a single cardiac cycle in normal conditions (6). However, the relationship between invasive measurements and TDI-derived tricuspid annulus velocities has long been unclear. In this study, dobutamine significantly decreased the MPI, whereas esmolol increased the MPI, although not significantly. By comparison, the TDI-MPI was higher than the PD-MPI in each situation. These results indicate that the MPI derived by using different methods should be interpreted with caution. Furthermore, our data show that both +dP/d*t* and −dP/d*t* are more strongly correlated with the TDI-MPI than with the PD-MPI. Yasuoka et al. (22) reported that the PD-MPI did not differ between patients with congenital heart disease and normal subjects, whereas the TDI-MPI was significantly greater in patients with heart disease than in normal subjects. These results suggest that the PD-MPI may be misinterpreted because it cannot be calculated over a single cardiac cycle, whereas the TDI-MPI should indicate RV global function.

Limitations. This study investigated the response of the RV TDI to inotrope administration in normal dogs. We cannot exclude the possibility that general anesthesia might have modulated the echocardiographic measurements. In addition, complete autonomic blockade was not used in this study, so reflex autonomic changes might have affected the filling variables of the heart. Since measurements of +dP/d*t* and −dP/d*t* are dependent on the loading conditions, the relationship between these derivatives and the echocardiographic data should be interpreted with caution. Furthermore, chronic heart disease might give rise to different responses. Therefore, the TDI should be validated for evaluating the entire range of responses in conscious dogs with heart disease and in healthy controls.

Conclusion. Our data demonstrated that the systolic myocardial velocity derived from the tricuspid annulus was strongly correlated with +dP/d*t* for the RV, implying that the systolic myocardial velocity provides accurate information on RV contractility. Furthermore, the TDI-MPI was highly correlated with +dP/d*t* and −dP/d*t* as an assessment of global RV

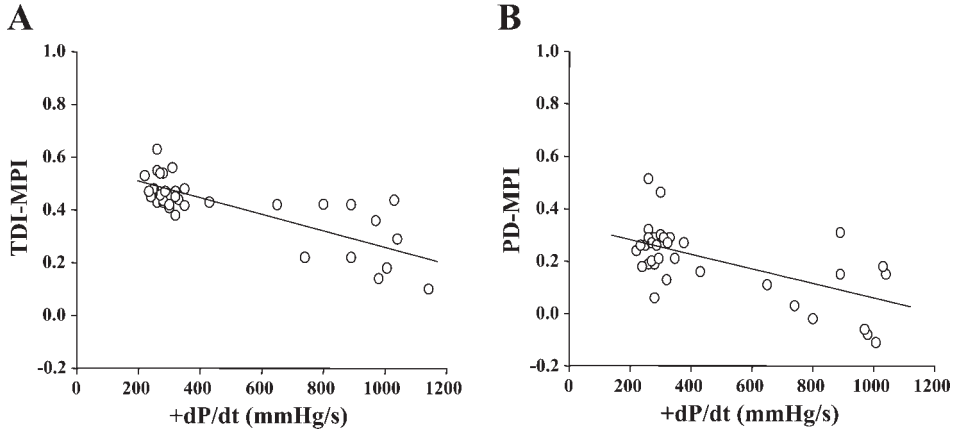

Fig. 5. Relationship between negative derivative of the right ventricular pressure (−dP/d*t*) and MPI. Significant correlations were observed between +dP/d*t* and the TDI-MPI ($r = -0.86$; *A*) and PD-MPI ($r = -0.54$; *B*).

function. Although the PD-MPI might have limited ability to assess RV function, the TDI-MPI is a sensitive indicator of RV function. These results suggest that the TDI-derived systolic myocardial velocity and MPI provide additional information related to RV systolic function.

REFERENCES

1. **Ama R, Segers P, Roosens C, Claessens T, Verdonck P, Poelaert J.** The effects of load on systolic mitral annular velocity by tissue Doppler imaging. *Anesth Analg* 99: 332–338, 2004.
2. **Apostolopoulou SC, Laskari CV, Tsoutsinos A, Rammos S.** Doppler tissue imaging evaluation of right ventricular function at rest and during dobutamine infusion in patients after repair of tetralogy of Fallot. *Int J Cardiovasc Imaging* 23: 25–31, 2007
3. **Arnlov J, Ingelsson E, Riserus U, Andren B, Lind L.** Myocardial performance index, a Doppler-derived index of global left ventricular function, predicts congestive heart failure in elderly men. *Eur Heart J* 25: 2220–2225, 2004.
4. **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* 15: 869–876, 2002.
5. **Bruch C, Schmermund A, Marin D, Katz M, Bartel T, Schaar J, Erbel R.** Tei-index in patients with mild-to-moderate congestive heart failure. *Eur Heart J* 21: 1888–1895, 2000.
6. **Cannesson M, Jacques D, Pinsky MR, Gorcsan J 3rd.** Effects of modulation of left ventricular contractile state and loading conditions on tissue Doppler myocardial performance index. *Am J Physiol Heart Circ Physiol* 290: H1952–H1959, 2006.
7. **Cheung MM, Smallhorn JF, Redington AN, Vogel M.** The effects of changes in loading conditions and modulation of inotropic state on the myocardial performance index: comparison with conductance catheter measurements. *Eur Heart J* 25: 2238–2242, 2004.
8. **Eidem BW, McMahon CJ, Ayres NA, Kovalchin JP, Denfield SW, Altman CA, Bezold LI, Pignatelli RH.** Impact of chronic left ventricular preload and afterload on Doppler tissue imaging velocities: a study in congenital heart disease. *J Am Soc Echocardiogr* 18: 830–838, 2005.
9. **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* 86: 654–658, 2000.
10. **Eto G, Ishii M, Tei C, Tsutsumi T, Akagi T, Kato H.** Assessment of global left ventricular function in normal children and in children with dilated cardiomyopathy. *J Am Soc Echocardiogr* 12: 1058–1064, 1999.
11. **Farias CA, Rodriguez L, Garcia MJ, Sun JP, Klein AL, Thomas JD.** Assessment of diastolic function by tissue Doppler echocardiography: comparison with standard transmitral and pulmonary venous flow. *J Am Soc Echocardiogr* 12: 609–617, 1999.
12. **Geva T, Powell AJ, Crawford EC, Chung T, Colan SD.** Evaluation of regional differences in right ventricular systolic function by acoustic quantification echocardiography and cine magnetic resonance imaging. *Circulation* 98: 339–345, 1998.
13. **Harada K, Tamura M, Toyono M, Yasuoka K.** Effect of dobutamine on a Doppler echocardiographic index of combined systolic and diastolic performance. *Pediatr Cardiol* 23: 613–617, 2002.
14. **Helbing WA, Bosch HG, Maliepaard C, Rebergen SA, van der Geest RJ, Hansen B, Ottenkamp J, Reiber JH, de Roos A.** Comparison of echocardiographic methods with magnetic resonance imaging for assessment of right ventricular function in children. *Am J Cardiol* 76: 589–594, 1995.
15. **Meluzin J, Spinarova L, Dusek L, Toman J, Hude P, Krejci J.** Prognostic importance of the right ventricular function assessed by Doppler tissue imaging. *Eur J Echocardiogr* 4: 262–271, 2003.
16. **Nagueh SF, Sun H, Kopelen HA, Middleton KJ, Khoury DS.** Hemodynamic determinants of the mitral annulus diastolic velocities by tissue Doppler. *J Am Coll Cardiol* 37: 278–285, 2001.
17. **Ruan Q, Rao L, Middleton KJ, Khoury DS, Nagueh SF.** Assessment of left ventricular diastolic function by early diastolic mitral annulus peak acceleration rate: experimental studies and clinical application. *J Appl Physiol* 100: 679–684, 2006.
18. **Silverman NH, Hudson S.** Evaluation of right ventricular volume and ejection fraction in children by two-dimensional echocardiography. *Pediatr Cardiol* 4: 197–203, 1983.
19. **Tabata T, Cardon LA, Armstrong GP, Fukamach K, Takagaki M, Ochiai Y, McCarthy PM, Thomas JD.** An evaluation of the use of new Doppler methods for detecting longitudinal function abnormalities in a pacing-induced heart failure model. *J Am Soc Echocardiogr* 16: 424–431, 2003.
20. **Tuller D, Steiner M, Wahl A, Kabok M, Seiler C.** Systolic right ventricular function assessment by pulsed wave tissue Doppler imaging of the tricuspid annulus. *Swiss Med Wkly* 135: 461–468, 2005.
21. **Yamada H, Oki T, Tabata T, Iuchi A, Ito S.** Assessment of left ventricular systolic wall motion velocity with pulsed tissue Doppler imaging: comparison with peak dP/d*t* of the left ventricular pressure curve. *J Am Soc Echocardiogr* 11: 442–449, 1998.
22. **Yasuoka K, Harada K, Toyono M, Tamura M, Yamamoto F.** Tei index determined by tissue Doppler imaging in patients with pulmonary regurgitation after repair of tetralogy of Fallot. *Pediatr Cardiol* 25: 131–136, 2004.