A New Noninvasive Method for the Estimation of Peak dP/dt

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Background. Peak dP/dt is a good index of ventricular performance that is not influenced by afterload, wall motion abnormalities, or the variations in ventricular anatomy and morphology commonly encountered among patients with congenital heart disease. Unfortunately, the clinical utility of peak dP/dt has been limited by the fact that its measurement generally requires an intraventricular catheter. Hence, the measurement is subject to changes in contractility, insensitive to changes in afterload, and only mildly affected by changes in preload. Furthermore, unlike many of the commonly used noninvasive indexes of ventricular performance, its measurement and interpretation are subject to distortion by wall motion abnormalities or the variations in ventricular anatomy and morphology commonly encountered among patients with congenital heart disease. The clinical utility of peak dP/dt has been limited, however, by the fact that its measurement conventionally requires insertion of an intraventricular catheter.

Hence, there exists a need for a reliable and widely applicable method of noninvasively measuring peak dP/dt. Features of the ventricular pressure curve suggest a means by which this may be accomplished. Peak dP/dt occurs during isovolumetric contraction, and the pressure rise during isovolumetric contraction is almost linear. The mean dP/dt during isovolumetric contraction (mean dP/dt), ie, the ratio of the rise in pressure during isovolumetric contraction (aortic diastolic pressure minus the systemic ventricular end-diastolic pressure (VEDP)) over the isovolumetric contraction time, should provide a good estimate of peak dP/dt that could be generated noninvasively.

Methods and Results. Echo/phonocardiography was used to measure the isovolumetric contraction time and a blood pressure cuff to estimate aortic diastolic pressure of 27 patients (age, 1 day to 77 years) with congenital or acquired heart disease. VEDP was determined by three methods: (1) intraventricular catheter, (2) assumed VEDP of 10 mm Hg, and (3) assignment of a normal or elevated value on the basis of clinical history. The three estimates of mean dP/dt, thus generated were compared with simultaneous measurements of peak dP/dt obtained during cardiac catheterization. Invasively measured peak dP/dt correlated well with the indirect determinations (r=.95, .89, and .92 for methods 1, 2, and 3, respectively; P < .0001).

Conclusions. Echo/phonocardiography can be used in conjunction with a blood pressure cuff and indirect estimates of VEDP to generate mean dP/dt, an index of ventricular function that approximates and closely correlates with peak dP/dt. This noninvasive measurement can be obtained in almost any patient and may be useful in the assessment of ventricular performance in a variety of cardiovascular disorders. (Circulation. 1993;88:2693-2699.)

Key Words • echocardiography • diagnostic techniques • defects, heart

The maximal rate of pressure rise during ventricular contraction (peak dP/dt) is a good index of ventricular performance.1-3 Peak dP/dt is sensitive to changes in contractility, insensitive to changes in afterload, and only mildly affected by changes in preload.1-6 Furthermore, unlike many of the commonly used noninvasive indexes of ventricular performance, its measurement and interpretation are not subject to distortion by wall motion abnormalities or the variations in ventricular anatomy and morphology commonly encountered among patients with congenital heart disease. The clinical utility of peak dP/dt has been limited, however, by the fact that its measurement conventionally requires insertion of an intraventricular catheter.

Hence, there exists a need for a reliable and widely applicable method of noninvasively measuring peak dP/dt. Features of the ventricular pressure curve suggest a means by which this may be accomplished. Peak dP/dt occurs during isovolumetric contraction, and the pressure rise during isovolumetric contraction is almost linear.1,8 The mean dP/dt during isovolumetric contraction (mean dP/dt), ie, the pressure rise during isovolumetric contraction divided by the isovolumetric contraction time, should therefore closely approximate peak dP/dt (Fig 1). Furthermore, it may be possible to obtain the data necessary for the calculation of mean dP/dt from noninvasive echo/phonocardiographic measurements. The purpose of this study was to compare noninvasive estimates of mean dP/dt, generated using the concepts outlined above, with simultaneous catheter-derived measurements of peak dP/dt.

Methods

Patient Population

Between April 1992 and October 1992, 27 patients undergoing clinically indicated cardiac catheterizations were enrolled in the study. The patients ranged in age from 1 day to 77 years (median, 10 years); 16 had congenital cardiac malformations; the remainder had acquired cardiovascular disorders (Table 1). Only patients who were in a regular rhythm were included in the study. The protocol for the study was approved by the
human investigation review committee of Tufts New England Medical Center, and informed consent was obtained from the subjects and/or their parents before participation in the study.

**Study Protocol**

Systemic ventricular pressure was recorded from intraventricular catheters at a paper speed of 200 mm/s. (The term "systemic ventricle" is meant to refer to the ventricle that supplies the majority of the aortic blood flow, e.g., the left ventricle in normal patients, the right ventricle in patients with transposition of the great arteries.) In 10 patients, a 5F micromanometer-tipped catheter (Millar Instruments, Houston, Tex) was used; in the remainder, standard fluid-filled catheters were used. All catheters were zeroed and calibrated before recording. Care was also taken to ensure that all air bubbles were removed from the fluid-filled catheter systems.

While the catheter-derived data were being obtained, simultaneous apical phonocardiograms and two-dimensionally guided M-mode echocardiograms of the aortic and systemic atrioventricular valves were recorded with a Hitachi EUB 165 echocardiography machine with a phonocardiogram attachment (Hitachi Medical Corp, Tokyo) at maximal sweep speed (150 mm/s), using standard techniques. The blood pressure in the right upper extremity was also determined with an appropriate-sized blood pressure cuff (Critikon Inc, Tampa, Fla).

**Measurements**

Peak dP/dt was derived from the ventricular pressure tracings using a Dextra off-line analysis system (Dextra Medical Systems, Inc, Lakewood, Calif). The mean of at least five consecutive beats was used for each measurement.

Isovolumetric contraction time was measured as the time period between systemic atrioventricular valve closure and aortic valve opening. The timing of atrioventricular valve closure was determined from the onset of the first heart sound on the phonocardiogram. The M-mode tracing of the systemic atrioventricular valve was used to confirm that the onset of the first heart sound accurately corresponded to atrioventricular valve closure. Aortic valve opening was determined from the M-mode tracing of the aortic valve. The time between the first heart sound and the opening of the aortic valve was measured using an off-line analysis package (Hitachi Medical Corp, Tokyo). The mean of at least five beats was used for each measurement.
Table 1. Patient Population: Clinical Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>60 y</td>
<td>CAD</td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>61 y</td>
<td>CAD, S/P non-Q-wave MI</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>75 y</td>
<td>CAD, S/P inferior MI, CABG</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>62 y</td>
<td>CAD</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>77 y</td>
<td>CAD, S/P non-Q-wave MI</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>1 y</td>
<td>Partial AVC</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>72 y</td>
<td>CAD</td>
</tr>
<tr>
<td>8</td>
<td>M</td>
<td>43 y</td>
<td>CAD, S/P non-Q-wave MI</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>1 y</td>
<td>S/P truncus arteriosus repair</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>18 y</td>
<td>DCRV</td>
</tr>
<tr>
<td>11</td>
<td>M</td>
<td>10 y</td>
<td>Subaortic membrane</td>
</tr>
<tr>
<td>12</td>
<td>M</td>
<td>16 y</td>
<td>KD, giant aneurysms</td>
</tr>
<tr>
<td>13</td>
<td>M</td>
<td>55 y</td>
<td>CAD</td>
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<td>14</td>
<td>M</td>
<td>0.75 y</td>
<td>Hypoplastic RV, S/P Glenn</td>
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<tr>
<td>15</td>
<td>M</td>
<td>8 y</td>
<td>S/P AVC repair, moderate MR</td>
</tr>
<tr>
<td>16</td>
<td>M</td>
<td>8 y</td>
<td>d-TGA, S/P Senning</td>
</tr>
<tr>
<td>17</td>
<td>F</td>
<td>7 y</td>
<td>TOF</td>
</tr>
<tr>
<td>18</td>
<td>F</td>
<td>3 y</td>
<td>PA/IVS, S/P shunt and RVOT patch</td>
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<tr>
<td>19</td>
<td>M</td>
<td>1.5 y</td>
<td>TAPVR to coronary sinus</td>
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<tr>
<td>20</td>
<td>F</td>
<td>0.4 y</td>
<td>VSD</td>
</tr>
<tr>
<td>21</td>
<td>M</td>
<td>1 d</td>
<td>d-TGA</td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>6 y</td>
<td>I-TGA, S/P VSD repair, moderate TR</td>
</tr>
<tr>
<td>23</td>
<td>M</td>
<td>8 y</td>
<td>S/P TOF/AVC repair, severe MR</td>
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<tr>
<td>24</td>
<td>F</td>
<td>16 y</td>
<td>CoA, subaortic stenosis</td>
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<tr>
<td>25</td>
<td>F</td>
<td>75 y</td>
<td>CAD, S/P non-Q-wave MI</td>
</tr>
<tr>
<td>26</td>
<td>F</td>
<td>43 y</td>
<td>CAD, S/P non-Q-wave MI, DM</td>
</tr>
<tr>
<td>27</td>
<td>M</td>
<td>10 y</td>
<td>I-TGA, AVC, PS</td>
</tr>
</tbody>
</table>

CAD indicates coronary artery disease; MI, myocardial infarction; CABG, coronary artery bypass graft; AVC, atrioventricular canal; DCRV, double-chamber right ventricle; KD, Kawasaki's disease; RV, right ventricle; MR, mitral regurgitation; TGA, transposition of the great arteries; TOF, tetralogy of Fallot; PA/IVS, pulmonary atresia with intact ventricular septum; RVOT, right ventricular outflow tract; S/P, status post; TAPVR, total anomalous pulmonary venous return; VSD, ventricular septal defect; TR, tricuspid regurgitation; CoA, coarctation of the aorta; DM, diabetes mellitus; and PS, pulmonary stenosis.

Aortic diastolic pressure was determined from the blood pressure cuff diastolic reading. The average of at least two measurements was used for each determination. Ventricular end-diastolic pressure was determined by three methods: (1) direct measurement from the catheter-derived ventricular pressure tracings (average of at least five beats), (2) assumed ventricular end-diastolic pressure of 10 mm Hg, and (3) assignment of a normal or elevated value on the basis of clinical history. Specifically, adult patients who had had a Q-wave myocardial infarction or had undergone ventriculography just before the study were assigned a ventricular end-diastolic pressure of 20 mm Hg; all other adult patients were assigned a value of 10 mm Hg. Similarly, pediatric patients who had received contrast material immediately before the study were assigned a ventricular end-diastolic pressure of 12 mm Hg; all other pediatric patients were assigned a value of 5 mm Hg.

Hence, three estimates of ventricular end-diastolic pressure were generated for each patient. Correspondingly, three different echo/phonocardiography-based estimates of mean dP/dt_e were calculated for each patient using the equation

\[
\text{mean } \frac{dP}{dt_e} = \frac{(\text{aortic diastolic pressure}) - (\text{ventricular end-diastolic pressure})}{\text{isovolumetric contraction time}}
\]

Statistical Analysis

The STATVIEW statistics program (Abacus Concepts, Morgan Hill, Calif) was used to compare the catheter-measured peak dP/dt with the three echo/phonocardiography-based estimates of mean dP/dt_e and to generate and compare the corresponding regression equations.
In addition to univariate regression analysis, peak dP/dt and the estimates of mean dP/dt were compared using the methods of Bland and Altman.13 The level of statistical significance was defined at P≤.05. Results are expressed as mean±SD.

Reproducibility of Measurements

The echo/phonocardiographically derived estimates of dP/dt were generated independently by two observers and twice by the same observer. Excellent interobserver (r=.94, SEE=91) and intraobserver (r=.95, SEE=70) correlations were obtained. The mean difference between observations did not differ significantly from zero and was not related to the magnitude of peak dP/dt.13

Results

Data from individual patients are presented in Table 2. The catheter-derived peak dP/dt averaged 1540±480 mm Hg/s (range, 438 to 2439). The echo/phonocardiography-based estimates of mean dP/dt averaged 1322±414 mm Hg/s (range, 556 to 2041) (method 1), 1381±403 mm Hg/s (range, 556 to 2192) (method 2), and 1332±420 mm Hg/s (range, 511 to 2150) (method 3). Excellent correlations existed between the catheter-derived peak dP/dt and each of the echo/phonocardiography-based estimates, with correlation coefficients of .95, .89, and .92, respectively; P<.0001 (Fig 2). However, the slopes of the regression equations (1.1, 1.06, and 1.05, respectively) were slightly greater than 1 (P<.05), and the noninvasively derived values for mean dP/dt, tended to underestimate peak dP/dt by approximately 10% to 15% (Fig 3).

For method 1, mean dP/dt underestimated peak dP/dt by 218±153 mm Hg/s. For method 2, the underestimation averaged 159±220 mm Hg/s and for method 3, 208±188 mm Hg/s (all P<.0001). The mag-
mitude of peak dP/dt had little effect on the degree of underestimation (Fig 3).

Results from patients with congenital heart disease did not differ from those obtained from patients with acquired heart disease, nor did the results from patients studied with micromanometer-tipped catheters differ from those obtained with fluid-filled catheters (Figs 2 and 3).

Discussion

The results of this study indicate that echo/phonocardiography-generated estimates of mean dP/dt of ventricular function for a wide range of ventricular functions. As one might expect, estimation method 1, which used direct measurements of ventricular end-diastolic pressure to calculate the change in pressure during isovolumetric contraction (a method potentially applicable to patients with Swan-Ganz or left atrial catheters), correlated best with peak dP/dt. However, because ventricular end-diastolic pressure is usually small relative to aortic diastolic pressure, errors generated in the measurement or estimation of this variable introduce relatively small errors into the ultimate estimation of mean dP/dt. Hence, even when all patients were assumed to have ventricular end-diastolic pressures of 10 mm Hg (method 2), the resulting completely noninvasive estimate of mean dP/dt still correlated well with the catheter-measured peak dP/dt. A rough refinement of the ventricular end-diastolic pressure estimate (method 3) improved the correlation still further.

In general, the echo/phonocardiographic methods tended to slightly underestimate the catheter-derived peak dP/dt. It therefore would be incorrect to consider mean dP/dt to be equivalent to peak dP/dt unless, perhaps, the values for mean dP/dt were first corrected using the appropriate regression equations. However, because of the strong similarities and excellent correlation that exist between these two indexes of ventricular function, it is reasonable to expect that mean dP/dt could provide physicians with clinically useful information regarding a patient's hemodynamic status.

Previous investigators have used Doppler echocardiography to estimate peak dP/dt in patients with mitral insufficiency (the rate of pressure rise, or RPR method). Previous investigators relied on mathematical reasoning somewhat analogous to that used in the present study, ie, they too, constructed a ratio of pressure over time. The denominator of the ratio was the time required for the Doppler velocity of the mitral insufficiency jet to increase from 1 to 3 m/s. The numerator of the ratio was pressure difference corresponding to these velocities according to the modified Bernoulli equation, ie, 36-4=32 mm Hg. The resulting estimate of peak dP/dt has been found to correlate well with simultaneous catheter-derived measures of peak dP/dt, with correlation coefficients ranging from .84 to .94. However, Chen et al., in an elegant analysis of this technique, found that the Doppler RPR method tends to underestimate peak dP/dt by an average of 30±197 mm Hg/s (approximately 25%), and the degree of this underestimation is dependent on the magnitude of peak dP/dt. The reason for this underestimation may relate to the fact that the Doppler RPR method actually measures the mean dP/dt during the early portion of isovolumetric contraction, whereas
peak dP/dt usually occurs near the midpoint of isovolumetric contraction or even later. In contrast, our method (which measures the mean dP/dt during the entire isovolumetric contraction period) underestimates peak dP/dt by only approximately 10% to 15%.

Despite its shortcomings, the clinical utility of the Doppler RPR method has been demonstrated by Pai et al., who found preoperative Doppler-derived peak dP/dt to be one of the best predictors of postoperative ejection fraction in patients undergoing surgery for severe mitral insufficiency. We speculate that similar clinically useful applications will be found for echocardiographically derived mean dP/dt. Furthermore, because echo/phonocardiography can be used to measure mean dP/dt in virtually any patient (and, unlike the Doppler technique, is not limited to patients with mitral insufficiency), the echo/phonocardiographic method described in this study may be much more widely applicable than the Doppler-based method.

The method of estimating mean dP/dt presented in this study also offers advantages over most other non-invasive indexes of ventricular performance. It can be measured easily and quickly at a patient’s bedside using equipment commonly available to most cardiologists.

Phonocardiographic attachments are available for most echocardiography machines. Alternatively, in the absence of a phonocardiogram, isovolumetric contraction time can be measured from sequential M-mode echocardiograms of the mitral and aortic valves. Furthermore, because mean dP/dt is a function only of pressure and time, its measurement is not hampered or confounded by variations in ventricular wall motion, anatomy, or morphology. It therefore may be a particularly appropriate index of ventricular function for patients with segmental wall motion abnormalities secondary to coronary artery disease and patients with various forms of congenital heart disease. As was demonstrated by this study, the presence of systemic right ventricles, functional single ventricles, ventricular septal defects (either open or surgically closed), right ventricular volume overload lesions, etc, did not adversely affect the accuracy of the echo/phonocardiographic-based estimates of mean dP/dt.

We also speculate that mean dP/dt, because of its similarity to peak dP/dt, may be relatively insensitive to alterations in afterload. It therefore may, in some circumstances, reflect acute changes in contractility more accurately than afterload-sensitive noninvasive indexes of ventricular function such as shortening frac-

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**Figure 3.** Graphs depicting the accuracy and variability of the echo/phonocardiographic methods of estimating peak dP/dt. A, Estimate 1; B, estimate 2; and C, estimate 3. The thick solid line represents the mean difference between the catheter-derived peak dP/dt and the echo/phonocardiographic estimates; the dotted lines represent the 95% confidence limits. Solid symbols represent data from transducer-tipped catheters; open symbols, fluid-filled catheters. Triangles represent patients with congenital heart disease; squares, patients with acquired cardiovascular disorders. The echo/phonocardiographic methods significantly underestimated peak dP/dt; the degree of underestimation tended to be slightly greater at higher levels of peak dP/dt.
tion, ejection fraction, velocity of circumferential fiber shortening,19 systolic time intervals,20 or aortic Doppler blood flow indexes.21

Limitations of the Study

Calculation of mean dP/dt requires the measurement of extremely brief time periods (ie, the isovolumetric contraction time) and, in the absence of invasive lines, indirect measurements of central aortic diastolic pressure and rough estimation of left ventricular end-diastolic pressure. Errors may be introduced during the estimation of each of these variables. For example, a ±5-millisecond error in the measurement of the isovolumetric contraction time would introduce a −11% to +14% error into the calculation of the average subject’s mean dP/dt. Similarly, a ±5 mm Hg error in the aortic or left ventricular end-diastolic pressure would introduce a ±9% error into the calculations. Standard error analysis using the delta method22 indicated that the standard errors observed in this study would increase by only 14% if the measurement errors could be limited to the values measured above. It is clear, however, that the reliability of mean dP/dt can be greatly influenced by the accuracy of measurements. Consequently, we feel that it is important to attempt to minimize measurement error as much as possible (eg, by recording data at fast paper speeds and by using the average of several measurements for each calculation) when applying these methods to the clinical setting.

One must also recognize that the reliability of the echo/phonocardiographic methods described in this study may be diminished among patients in whom the aortic diastolic pressure is not significantly greater than the ventricular end-diastolic pressure, eg, patients with severe aortic insufficiency. Under these circumstances, the change in pressure during isovolumetric contraction will be small and the duration of isovolumetric contraction brief. Consequently, accurate measurement of these variables would be more difficult and the estimation of peak dP/dt prone to error. Estimates of peak dP/dt obtained from these patients should therefore be interpreted with caution.

Finally, because of the influence of preload on peak dP/dt, it may be inappropriate to use mean dP/dt as an index of contractility under conditions where preload varies greatly or in the setting of an irregular rhythm. Under these circumstances, it may be preferable to use one of the more preload-independent (albeit more complex) indexes of ventricular function such as Vm/[(dP/dt)/(ventricular pressure)], or Vmax, i.e., the value of (dP/dt)/(ventricular pressure) extrapolated to a ventricular pressure of zero.1,2

Conclusions

Echo/phonocardiography can be used to generate an index of ventricular function (mean dP/dt) that approximates and closely correlates with peak dP/dt. This noninvasive measurement can be readily obtained in almost any patient and may be useful in the assessment of ventricular performance in a variety of cardiovascular disorders.

References

A new noninvasive method for the estimation of peak $dP/dt$.
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