Noninvasive evaluation of left ventricular performance based on peak aortic blood acceleration measured with a continuous-wave Doppler velocity meter

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ABSTRACT Peak aortic blood acceleration is recognized to be a sensitive index of global left ventricular performance. In the present study peak acceleration was assessed noninvasively in patients with a continuous-wave Doppler velocity meter. Peak aortic blood velocity and peak blood acceleration were measured by placing the ultrasonic transducer at the suprasternal notch. Measurements were obtained in 36 patients undergoing diagnostic cardiac catheterization. Peak velocity and acceleration were measured at rest just before left ventriculography. In patients with ejection fractions greater than 60%, peak acceleration was 19 ± 5 m/sec/sec. In patients with ejection fractions of 41% to 60%, peak acceleration was lower, at 12 ± 2 m/sec/sec (p < .001). In patients with ejection fractions of 40% or less, peak acceleration (8 ± 2 m/sec/sec) was markedly lower than in patients with ejection fractions greater than 60% (p < .001). Peak acceleration showed a good linear correlation with ejection fraction (r = .90), and a better power fit (r = .93). These results indicate that peak acceleration, measured noninvasively with a continuous-wave Doppler velocity meter, is a useful indicator of global left ventricular performance.


IN 1964 Rushmer suggested that the left ventricle acts as an impulse generator and that the “initial ventricular impulse” is a descriptive term for the dynamic properties of ventricular ejection. Ventricular impulse was defined as the product of force and time, where the net force (mass × acceleration) acts over time from the beginning of ejection to the attainment of peak flow. Because the peak acceleration of blood out of the ventricle occurs in early systole, it is thought to represent a manifestation of the initial ventricular impulse. Studies by Noble et al. suggested that peak acceleration was closely related to the maximum force exerted by the ventricle in early systole, and they believed that peak acceleration was also related, in some way, to the maximum initial velocity of shortening of left ventricular muscle. Studies by Stein and Sabbah showed that blood acceleration was intrinsic to the rate of change in power developed by the left ventricle and related closely to it. Peak blood acceleration was also shown to be a function of both the rate and acceleration of shortening of the radius of the left ventricle.

Studies in anesthetized dogs showed that peak acceleration of aortic blood flow was sensitive to alterations in the inotropic state but was less affected by an augmentation of preload and afterload. Peak acceleration of aortic blood flow was also found to be sensitive to alterations in the contractile state induced by temporary regional myocardial ischemia in conscious dogs. Studies by Stein and Sabbah using catheter-tip velocity sensors during cardiac catheterization in patients showed that peak acceleration was capable of differentiating patients with normal from those with abnormal left ventricular performance. In 12 patients undergoing diagnostic cardiac catheterization, Bennett et al. using catheter-tip velocity transducers, demonstrated a close relationship between peak acceleration and left ventricular ejection fraction calculated angiographically.

The above investigations establish peak blood acceleration as a good index of global left ventricular per-
formance. The routine use of this index of ventricular function in patients, however, has been limited by the need to invasively measure phasic aortic blood velocity. Recent advances in Doppler ultrasound technology have overcome this hurdle and now allow a noninvasive measurement of aortic blood velocity from which peak acceleration can be derived. The purpose of the present study was to noninvasively evaluate peak aortic blood acceleration with use of a continuous-wave Doppler velocity meter in patients undergoing diagnostic cardiac catheterization, and to relate this parameter to left ventricular performance as assessed by ejection fraction.

Methods

The Doppler system. Ascending aortic blood velocity was evaluated transcutaneously from the suprasternal notch by a conventional continuous-wave Doppler transmitter and receiver operating at 3.0 MHz (ExerDop, Quinton Instrument Co., a division of A.H. Robins, Inc., Seattle). The received Doppler-shifted blood velocity signal is processed by quadrature demodulation so that only signals representing flow toward the transducer are detected. This directional signal is bandpass filtered with 3 dB cutoff frequencies of 480 Hz and 11.5 kHz. The band-limited directional signal is then fed into a fast-attack fast-decay instrumentation, automatic gain control circuit so that its amplitude remains constant. This constant-amplitude Doppler signal is fed into a velocity circuit that tracks the instantaneous modal frequency and derives the modal peak velocity. The system samples the velocity signal every 5 msec to determine the instantaneous modal velocity (within ±10%). It then compares each instantaneous modal velocity with the previous value and retains the larger one. The largest value for instantaneous modal velocity is defined as peak modal velocity.

The system internally derives peak acceleration and the systolic velocity integral (which may also be termed stroke distance) at a high sample rate (200 Hz) to ensure that the maximum value of blood acceleration occurring during the cardiac cycle is pinpointed. It is capable of detecting peak modal velocity in the range of 0.2 to 2.5 m/sec and peak acceleration in the range of 2 to 99 m/sec/sec. The ability of this device to accurately measure peak aortic blood velocity and acceleration has been previously demonstrated in dogs. A comparison of peak velocity and peak acceleration measured with the continuous-wave Doppler velocity meter and an electromagnetic flow transducer placed at the root of the aorta resulted in correlations of .95 and .96, respectively. When the Doppler unit was used in patients to measure velocity in the ascending aorta, we attempted to align the ultrasound beam with the direction of flow. We assumed that the intercept angle of the Doppler velocity meter was 180 degrees to the direction of flow. This is consistent with previous methodology.

The continuous-wave Doppler system used in this study is capable of producing an analog signal of the velocity waveform in the ascending aorta. The analog signal of acceleration, however, if needed, must be obtained by external differentiation of the velocity waveform. The system provides an internally derived digital output, on a beat-to-beat basis, of peak acceleration, peak modal velocity, and the systolic velocity integral. A typical example of the velocity and acceleration waveforms and of the digital output from a normal subject is shown in figure 1. The accuracy and repeatability of the digital values fall within ±5% of the values derived from the analog waveforms. Once a sufficient number of beats is completed, the Doppler instrument automatically computes an arithmetic average of peak acceleration, peak modal velocity, and systolic velocity integral. This average value includes all of the beats except those that fall outside preselected limits determined by an internally installed algorithm. The algorithm excludes beats with a signal-to-noise ratio of less than 10 dB. In addition, a stack average is calculated by averaging eight data points consisting of values from the most recently obtained data set. The stack average is then compared with the current value of the parameter. All values for a given beat are excluded if any value falls outside the following empirically derived limits: peak acceleration ±40% of the stacked average, peak velocity ±25% of the stack average, and systolic velocity integral ±40% of the stack average. This algorithm, even though empirically derived, has been useful in rejecting beats with a poor signal-to-noise ratio and in rejecting extrasystolic beats and postextrasystolic beats. The values re-

![FIGURE 1. Left, Velocity waveforms and acceleration waveforms measured with the continuous-wave Doppler velocity meter in the ascending aorta of a normal adult. Right, Simultaneous beat-to-beat digital output from the Doppler system of peak acceleration (Pk A), peak velocity (Pk V), and systolic velocity integral (SD). Note the beat-to-beat variation in peak velocity and peak acceleration, which required the averaging of values over a large number of beats.](http://circ.ahajournals.org/)
ported in this study are based on the arithmetic average provided by the digital output of the Doppler instrument.

**Studies in vitro.** The accuracy of the velocity signal obtained with the Doppler instrument was evaluated in vitro in a closed-loop, pump-driven, steady-flow system. The test section consisted of an entrance region where the velocity profile was flat. Flow was calibrated with use of a beaker and stop watch. Velocity was calculated as the product of volumetric flow and the cross-sectional area of the test section that was constant. The fluid in the system was seeded with 1.5 μm silicon microspheres to allow the measurement of velocity with the ultrasound Doppler method. A range of velocities between 0.3 and 3.0 m/sec was evaluated.

**Studies in normal adults.** To determine if values of peak velocity measured with this continuous-wave Doppler meter were comparable to values reported in the literature, we measured peak modal velocity in 16 normal volunteers. None of the 16 volunteers had any symptoms or history of cardiovascular disease and none were taking any medication. Age, sex, heart rate, and blood pressure of these subjects are shown in table 1. All were studied while they were at rest in the supine position. The reported values of peak velocity, peak acceleration, and systolic velocity integral in each subject represent the average of 14 to 39 consecutive sinus beats.

**Patient population.** Doppler measurements were made in patients undergoing diagnostic left heart catheterization. The study was approved by the hospital human rights committee. Informed consent was obtained from all patients in the study. Patients with aortic valve disease or mitral insufficiency were excluded from the study. Studies were attempted in 43 consecutive patients undergoing cardiac catheterization, and reliable Doppler measurements were obtained in 36. Data reported in this study, therefore, relate only to these 36 patients. In the remaining seven, appropriate aortic velocity measurements could not be obtained. In all cases this was believed to be due to obesity. Reliable Doppler measurements, therefore, were obtained in 84% of patients. Among the 36 patients studied, 25 were men and 11 were women. The average age of the patients was 57 ± 14 years (range 28 to 78 years). Twenty-eight patients had coronary artery disease of varying severity, one patient had cardiomyopathy, and seven patients were free of any demonstrable cardiac disease.

The data were examined by separating the patients into three groups based on ejection fraction. The first group (n = 17) consisted of patients with ejection fractions greater than 60%. The second group (n = 12) consisted of patients with ejection fractions between 41% and 60%. The third group (n = 7) consisted of patients whose ejection fractions were 40% or less.

The age, sex, and ejection fraction of patients in each group are summarized in table 1. The mean age of the patients was 68 years (range 28 to 78 years). The male/female ratio was 2:1. The mean ejection fraction was 58% (range 41% to 68%).

**Table 1.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal adults</th>
<th>EF  &gt;60%</th>
<th>≤ EF  ≤60%</th>
<th>EF ≤ 40%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>31 ± 5</td>
<td>55 ± 14</td>
<td>62 ± 12</td>
<td>53 ± 4</td>
</tr>
<tr>
<td>Sex</td>
<td>8 M, 8 F</td>
<td>13 M, 4 F</td>
<td>9 M, 3 F</td>
<td>3 M, 4 F</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>69 ± 8</td>
<td>65 ± 11</td>
<td>72 ± 9</td>
<td>91 ± 15</td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>106 ± 12</td>
<td>141 ± 20</td>
<td>128 ± 27</td>
<td>118 ± 21</td>
</tr>
<tr>
<td>Diastolic pressure (mm Hg)</td>
<td>68 ± 11</td>
<td>75 ± 9</td>
<td>74 ± 13</td>
<td>72 ± 8</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>—</td>
<td>15 ± 6</td>
<td>19 ± 6</td>
<td>22 ± 6</td>
</tr>
</tbody>
</table>

EF = ejection fraction; LVEDP = left ventricular end-diastolic pressure.

**Results**

**Studies in vitro.** Studies performed in vitro showed a direct linear relationship between the measured velocity and the velocity detected by the Doppler instrument. This relationship is depicted in figure 2. The correlation coefficient was .995, the slope of the linear regression line was nearly unity (1.12), and the intercept was nearly zero (0.09).

**Studies in normal adults.** Peak aortic blood velocity measured with continuous-wave Doppler ultrasound in...
normal adults was $0.91 \pm 0.15$ m/sec (range 0.77 to 1.22 m/sec). Peak acceleration was $20 \pm 4$ m/sec/sec and ranged between 14 and 26 m/sec/sec. The systolic velocity integral was $14.3 \pm 4.0$ cm and ranged between 9.4 and 22.0 cm. Individual values for peak velocity, peak acceleration, and the systolic velocity integral in the group of normal adults are shown in figure 3.

Studies in patients

Patients with ejection fractions of greater than 60%. Among the 17 patients in whom the ejection fraction was greater than 60%, peak aortic blood velocity was $0.68 \pm 0.20$ m/sec. Peak acceleration was $19 \pm 5$ m/sec/sec and the systolic velocity integral was $10.7 \pm 5.1$ cm. Individual values for peak velocity, peak acceleration, and systolic velocity integrals in this group of patients are shown in figure 3.

Patients with ejection fractions between 41% and 60%. Among the 12 patients in whom the ejection fraction was between 41% and 60%, peak velocity was lower than in the group of patients with ejection fractions greater than 60% ($0.47 \pm 0.9$ vs $0.68 \pm 0.20$ m/sec, $p < .001$). Peak acceleration was also significantly lower ($12 \pm 2$ vs $19 \pm 5$ m/sec/sec, $p < .001$). The systolic velocity integral, however, was not different than that in patients with ejection fractions greater than 60% ($8.2 \pm 2.7$ vs $10.7 \pm 5.1$ cm). Individual values for peak velocity, peak acceleration, and systolic velocity integrals in this group of patients are shown in figure 3.

Patients with ejection fractions of 40% or less. Among the seven patients in whom the ejection fraction was abnormally low (\(\leq 40\%\)), peak velocity ($0.34 \pm 0.8$ m/sec) was significantly lower than that in patients with ejection fractions greater than 60% ($p < .001$). Similarly, peak acceleration ($8 \pm 2$ m/sec/sec) was

![Figure 2](image-url)  
**FIGURE 2.** Relationship between experimentally measured velocity and velocity measured with the continuous-wave Doppler method in a steady-flow system in vitro.

![Figure 3](image-url)  
**FIGURE 3.** Individual values for peak acceleration (top), peak velocity (middle), and systolic velocity integral (bottom) in normal adults and in three groups of patients categorized on the basis of ejection fraction (EF). Probabilities are based on comparisons with patients with EF >60%.
also markedly lower in these patients \((p < .001)\) and the systolic velocity integral \((4.4 \pm 1.6 \text{ cm})\) was diminished in comparison with that in patients with an ejection fraction of greater than 60\% \((p < .01)\). Individual values for peak velocity, peak acceleration, and systolic velocity integrals in this group of patients are shown in figure 3.

**Correlations with ejection fraction.** Among the Doppler parameters tested in the 36 patients, peak acceleration showed the best linear correlation with ejection fraction, with a correlation coefficient of .90 (figure 4). When the data were fitted to a power curve, the correlation coefficient improved slightly to .93 (figure 4). Peak velocity showed a good linear correlation \((r = .77; \text{ figure 5})\), but the systolic velocity integral showed only a fair correlation with ejection fraction \((r = .59)\).

**Discussion**

The present study shows that peak acceleration of blood in the ascending aorta, measured noninvasively by a continuous-wave Doppler method, can be used to differentiate between patients with normal and abnormal left ventricular performance as assessed by the ejection fraction. The observations made in this study were limited to a group of patients who underwent diagnostic cardiac catheterization because of documented or suspected coronary artery disease or because of cardiomyopathy. Patients with aortic valve disease or mitral regurgitation were excluded from the study. Patients with aortic valve disease were excluded because the dynamics of flow in the ascending aorta can be markedly influenced by a diseased aortic valve. In patients with mitral regurgitation the dynamics of flow across the aortic valve may also be influenced by the degree of regurgitant mitral flow during systole.

In patients who underwent diagnostic cardiac catheterization, and therefore in whom a left ventricular ejection fraction was calculated, a close correlation was observed between peak acceleration and the ejection fraction. Both of these indexes of left ventricular performance are empirically derived and each depends on different physical constraints. Nevertheless, both are ejection indexes of left ventricular performance. Their close correlation implies that both are comparatively sensitive to differences in global left ventricular function. The mathematic relationships derived in this study were considered only as a measure of the closeness of the correlation and not as a means of deriving the ejection fraction from a measurement of peak acceleration. This interpretation also applies with respect to the correlations between peak velocity, systolic velocity integral, and the ejection fraction.

The close correlation between the peak acceleration and ejection fraction observed in the patients in the present study is consistent with previous reports. Bennett et al.,\(^6\) using a catheter-tip electromagnetic velocity probe placed in the ascending aorta of 12 patients, showed that peak acceleration was related to the ejection fraction with a correlation coefficient of .88. In their study, peak acceleration ranged between 4 and 19
m/sec/sec for a range of ejection fractions between 17% and 74%. This is also consistent with our measurements of peak acceleration in the range of 6 to 32 m/sec/sec for a range of ejection fractions between 22% and 78%. A good correlation between peak acceleration and ejection fraction was also observed in patients by Chandraratna et al. using similar techniques to those used in this study. In their study, patients with ejection fractions of greater than 50% had a peak acceleration of greater than 11.6 m/sec/sec, whereas in patients with ejection fractions less than 50% peak acceleration was less than 11.6 m/sec/sec. A correlation coefficient of .83 was reported between peak acceleration and ejection fraction.

A number of investigators have measured peak acceleration using ultrasound Doppler via the suprasternal notch approach in normal subjects or patients. Innes et al. measured peak acceleration using a pulsed Doppler device applied to the suprasternal notch in six normal subjects at rest and after each of four levels of seated bicycle exercise. They found an increase in peak acceleration with an increased level of exercise. Mehta and Bennett used a continuous-wave Doppler method to evaluate peak acceleration in patients with acute myocardial infarction. They showed that peak acceleration was significantly lower in patients with an acute myocardial infarction than in age-matched normal subjects. Other investigators have also used continuous-wave Doppler techniques to evaluate peak acceleration in normal subjects in whom the inotropic state and loading conditions of the heart were altered. Loading conditions had little effect on peak acceleration, whereas an augmentation of the inotropic state produced a significant increase in peak acceleration. Another study in which a pulsed, range-gated ultrasonic Doppler method was used in normal subjects showed a decline of peak acceleration with age. Even though these studies did not correlate peak acceleration with established indexes of ventricular performance, their findings support the use of this technique for the noninvasive evaluation of global left ventricular performance.

The magnitude of peak velocity in the ascending aorta, obtained noninvasively in our study with a continuous-wave Doppler method, is consistent with values obtained invasively in patients with the use of electromagnetic catheter-tip velocity probes positioned in the ascending aorta. In patients with an ejection fraction of greater than 60%, peak velocity measured with a catheter-tip velocity probe ranged between 0.67 and 0.83 m/sec and in our Doppler study it ranged between 0.42 and 1.16 m/sec. Similarly, in patients with ejection fractions of less than 40%, peak velocity measured with a catheter-tip velocity probe ranged between 0.16 and 0.44 m/sec and in our Doppler study it ranged between 0.24 and 0.50 m/sec.

The values of peak aortic blood velocity measured in our group of normal adult volunteers (0.91 ± 0.15 m/sec) were comparable to values obtained by Gardin et al. in a similar group of normal adults. In their study of 20 normal adult subjects (age 21 to 46), the average value for peak velocity was 0.92 ± 0.11 m/sec (range 0.72 to 1.20 m/sec). Our values for peak velocity and those of Gardin et al. were somewhat lower than those reported by Hatle and Angelsen in a group of normal adults ranging in age between 18 and 72 years. The ultrasound Doppler system used in our study is a continuous-wave system designed to measure modal peak velocity. The systems used by Gardin et al. and Hatle and Angelsen were pulsed Doppler devices. Given the differences in available ultrasound Doppler instrumentation, it is reasonable to suppose that the range of normal values observed may depend on the specific instrumentation that is used.

The values for peak aortic velocity reported in the present study in patients with ejection fractions greater than 60% were lower than values observed in normal healthy adults, including those reported in this study and in studies by Gardin et al. The lower peak velocity that we observed in patients with ejection fractions in the normal range may be due to two factors. (1) The normal adults studied by us and by Gardin et al. were younger than the patients with ejection fractions in the normal range and peak velocity has been shown to decrease with increasing age. (2) A large proportion of our patients with ejection fractions in the normal range had coronary artery disease and were being treated with either calcium-channel blockers or β-blockers.

Neither peak velocity nor the systolic velocity integral related as closely to the ejection fraction as did peak acceleration. Changes in peak aortic velocity and in the systolic velocity integral, however, have been shown to provide an accurate assessment of changes in stroke volume. Both peak velocity and the systolic velocity integral, therefore, may prove to be clinically useful in estimating changes of stroke volume. A study of the sensitivity and specificity of various indexes of left ventricular performance by Lambert et al. showed that peak acceleration was far more sensitive to alterations of the inotropic state than was stroke volume and was also more sensitive than peak aortic blood velocity.

In conclusion, peak acceleration of blood in the as-
Ascending aorta of patients, measured noninvasively with a continuous-wave Doppler velocity meter, was shown to be a good indicator of global left ventricular performance. This represents a relatively simple, inexpensive, and noninvasive technique for the evaluation of left ventricular performance in patients.

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