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Evaluation of dilated cardiomyopathy by pulsed Doppler echocardiography

The ability of pulsed Doppler echocardiography to identify patients with left ventricular systolic dysfunction was evaluated in 12 patients with dilated (congestive) cardiomyopathy. A range-gated, spectrum analyzer–based Doppler velocimeter was used to record blood flow velocity in the ascending aorta and main pulmonary artery. The following blood flow velocity parameters were measured or derived: peak flow velocity, acceleration time, average acceleration, deceleration time, average deceleration, ejection time, and aortic flow velocity integral. Doppler blood flow velocity data in the cardiomyopathy patients were compared to data from 20 normal subjects. Measurements from the ascending aorta revealed that peak aortic flow velocity discriminated between cardiomyopathy patients (mean 47 cm/sec, range 35 to 62) and normal subjects (mean 92 cm/sec, range 72 to 120) with no overlap in data ($p < 0.001$). Aortic flow velocity integral was also able to separate the patients with dilated cardiomyopathy (mean 6.7 cm, range 3.5 to 9.1) from normal subjects (mean 15.7 cm, range 12.6 to 22.5) with no overlap in data ($p < 0.001$). Although mean values for average aortic acceleration and aortic ejection time were also significantly different (both $p < 0.005$), there was some overlap between the two groups. Pulmonary artery blood flow studies demonstrated significantly increased average acceleration, as well as decreased ejection time (both $p < 0.05$), but no difference in average deceleration or peak flow velocity in cardiomyopathy patients compared to normals. Compared to pulmonary flow measurements, aortic Doppler flow velocity measurements allowed better separation of cardiomyopathy and normal groups. In addition, aortic peak flow velocity appeared to correlate well ($r = 0.83$) with M-mode echocardiographic measurement of left ventricular percent fractional shortening; both parameters were equally useful in discriminating patients with normal left ventricular function from those with global dysfunction (dilated cardiomyopathy). Thus pulsed Doppler echocardiography appears to be a useful addition to M-mode and two-dimensional echocardiography in the quantitative noninvasive assessment of left ventricular systolic function. (Am Heart J 106:1057, 1983.)

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Doppler echocardiography has been shown to be a useful noninvasive technique for evaluating beat-to-beat variations in stroke volume in dogs[1-3] and humans,[4-7] and in characterizing the normal flow velocity patterns in the great arteries in humans.[8-12] A potentially important clinical application of this technique would be the noninvasive evaluation of global ventricular function in patients with known or suspected heart disease. To investigate this potential clinical application, we used pulsed Doppler echocardiography to measure aortic and pulmonary artery blood flow velocities in 12 patients known to have dilated cardiomyopathy and compare these to data from 20 normal subjects.

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METHODS

Patient population. The cardiomyopathy study group consisted of 12 patients (nine men and three women, age range 36 to 80 years). This protocol was approved by the Institutional Human Subjects Review Committee and all individuals gave their informed consent to the study. All patients had clinical findings characteristic of dilated (congestive) cardiomyopathy and M-mode echocardiograms typical of this disease[13,14]—including a left ventricular diastolic dimension greater than 25% above the normal value predicted on the basis of age and body surface area,[15-17] a left ventricular percentage fractional shortening less than 25% (normal range 28% to 44%[16])

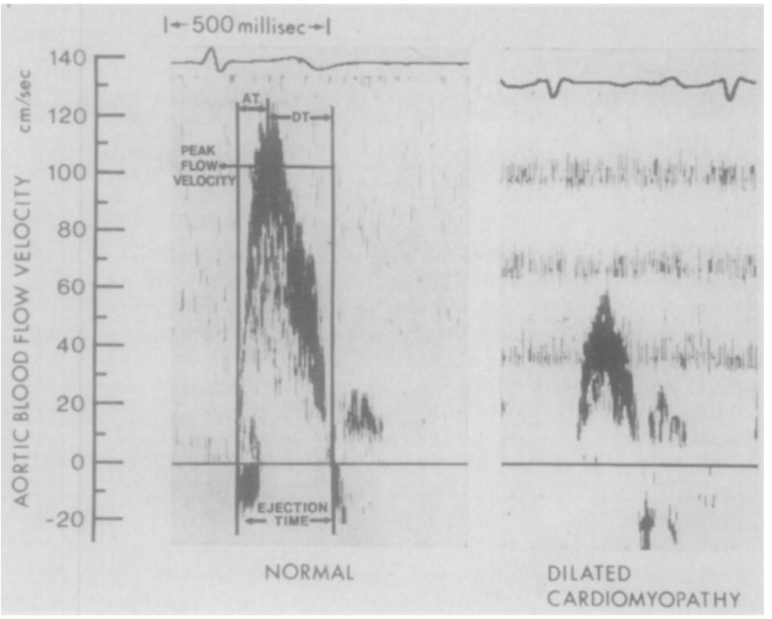

Fig. 1. Doppler blood flow velocity recordings from the ascending aorta in a normal subject *(left panel)* and in a patient with dilated cardiomyopathy *(right panel)*. Aortic blood flow velocity in centimeters per second is displayed on the *vertical axis* and time is shown on the *horizontal axis*. The normal Doppler aortic flow velocity recording is labeled to show how measurements are made of peak flow velocity, acceleration time (AT), deceleration time (DT), and ejection time. Note that peak aortic flow velocity, flow velocity integral, average acceleration, and ejection time are greater in the normal subject than in the cardiomyopathy patient. See text for details.

and increased E-point septal separation.[18] Two-dimensional echocardiograms revealed a dilated, nonhypertrophic left ventricle in all patients—with diffuse hypokinesis in all 12 and additional areas of mild dyskinesis in four patients. Four of the 12 patients had chest pain and underwent cardiac catheterization including coronary arteriography and left ventricular angiography. Three of these patients were demonstrated to have at least one coronary artery lesion with greater than 50% luminal narrowing. None had left ventriculograms suggestive of a discrete left ventricular aneurysm. The presumptive etiology of the cardiomyopathy was alcoholic in four, ischemic in four, and viral or idiopathic in four patients.

At the time of the study, 10 patients were taking digoxin or furosemide, three were taking oral isosorbide dinitrate and/or nitroglycerin ointment, three were taking hydralazine, and one was taking no medication. Studies were performed at least 6 hours after the last dose of hydralazine, isosorbide dinitrate, or nitroglycerin ointment.

Normal subjects. Blood flow velocity was recorded in the ascending aorta and main pulmonary artery of 20 normal subjects.[12] There were 12 men and 8 women, age range 21 to 46 years. All 20 had normal cardiovascular histories, physical examinations, and M-mode echocardiograms. None of the normal subjects was a trained competitive athlete and all had body weight within 20% of that predicted for height on the basis of the Metropolitan Life Insurance Company tables.[19]

Instrumentation and recording technique. Doppler echocardiographic studies were performed utilizing an echocardiographic unit that incorporates a spectrum analyzer-based, range-gated, pulsed Doppler velocimeter interfaced with a mechanical sector scanner to produce M-mode and real-time two-dimensional echocardiographic images as well as blood flow velocity recordings (Ultra-Imager, Electronics for Medicine/Honeywell Corp., Denver, Colo.).[11, 12] In this instrument, the Doppler frequency shifts were detected by a dual-channel spectrum analyzer, converted into the corresponding flow velocity and displayed at 5 msec intervals on a strip chart. The frequency shifts that occurred during any given 5 msec sampling interval were brightness-modulated and displayed so that the frequency at which the greatest number of red blood cells were moving was indicated as the brightest signal (i.e., the darkest point on the light-sensitive paper) with frequency shifts reflecting smaller numbers of blood cells being displayed as progressively less bright signals. Frequency shifts corresponding to flow velocities of less than 10 cm/sec in either direction were attenuated by high-frequency, band-pass filters and were displayed as a relatively signal-free area around the zero flow velocity line. Flow velocity patterns were displayed at 100 mm/sec sweep speed on an oscilloscope and were selectively stop-framed utilizing a switch to freeze the image. The frozen image was then recorded on glossy black-on-white electrostatic paper at a paper speed of 50 mm/sec. The net result was to obtain high contrast images recorded at the equivalent of 100 mm/sec paper speed without running the strip recorder at this very rapid paper speed (Fig. 1).

By convention, blood flowing in the normal direction within the circulatory system was indicated by a positive, or upward, deflection on the tracing. Therefore, in the ascending aorta, flow out of the heart and toward the transducer (located in the suprasternal notch) was displayed as a positive deflection and flow away from the transducer was displayed as a negative deflection. In the main pulmonary artery, flow away from the transducer in the parasternal position was displayed as positive and flow towards the transducer was displayed as negative.[12] This convention facilitated a direct comparison between flow velocity records obtained from the ascending aorta and from the main pulmonary artery.

Pulmonary artery flow velocity recordings were obtained using the two-dimensional Doppler echocardiographic approach. Specifically, a 3.5 MHz sector scanner transducer was used to image the main pulmonary artery in the parasternal short-axis view. The Doppler sample volume—i.e., a rectangle representing the cylindrical region from which the Doppler frequency shift was measured—was then electronically superimposed on the two-dimensional image of the proximal main pulmonary artery in a near-parallel orientation to the long axis of blood flow.[11,12] As previously described, if the angle of incidence of the ultrasound beam is within 20 degrees of the long axis of blood flow, the error introduced into the blood flow velocity measurement is less than 6% of peak blood flow velocity.[2,9]

Ascending aortic flow velocity recordings were obtained using a 2.25 MHz, right-angle M-mode echocardiographic transducer placed in the suprasternal notch position.[12] Since the ascending aorta was not imaged directly, special care was taken to record the maximum ascending aortic blood flow velocity by mapping the ascending aorta at various depths from the transducer. Specifically, the distance from the transducer to the nearest boundary of the sample volume was varied at 1 cm intervals from a depth of 3 to 9 cm. At each sample volume depth, the transducer was angulated to obtain the peak flow velocity as detected audibly and recorded graphically on an oscilloscope screen and a strip chart. The sample volume depth used for measurement of aortic flow velocity was the one that produced the maximal aortic flow velocity recorded by this mapping technique.[12]

In both aortic and pulmonary artery flow velocity measurements, the sample volume was a cylinder approximately 15 mm in axial length and 3 to 4 mm in diameter.[12]

Blood flow velocity measurements. Measurements of blood flow velocity parameters form the ascending aorta and main pulmonary artery strip chart records were made in a similar manner. The parameters measured included peak flow velocity (centimeters per second), acceleration time (milliseconds), deceleration time (milliseconds), and ejection time (milliseconds)[12] (Fig. 1). Because of a dispersion of flow velocities at peak flow and during deceleration, peak flow velocity was measured at the center of the Doppler flow spectrum at the time that maximum blood flow velocity was recorded. Acceleration time was measured from the onset of ejection to the point of peak flow velocity. Similarly, deceleration time was measured from the point of peak flow velocity to the end of systolic ejection. Average acceleration and deceleration of blood flow were calculated by dividing the peak flow velocity by the acceleration and deceleration times, respectively. Ejection time was measured from the onset of ejection to the end of systolic flow.

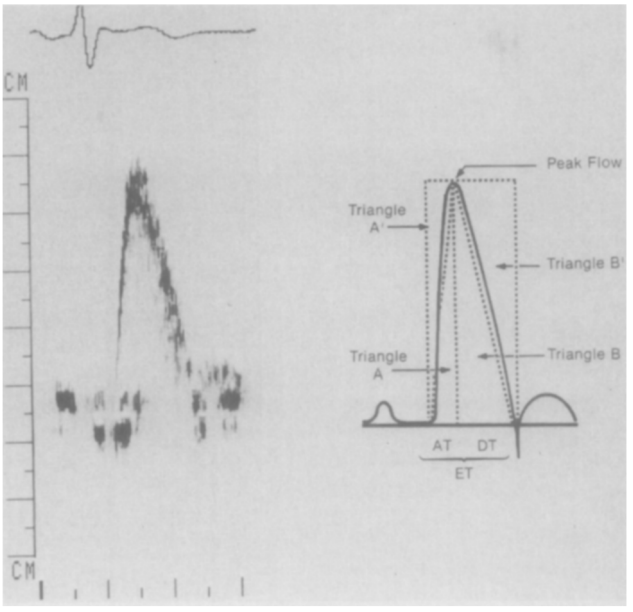

Fig. 2. Mathematical model for approximating flow velocity integral (FVI, i.e., the area under the flow velocity curve). Aortic flow velocity tracing is shown on the *left* and method for deriving the approximation, FVI = 1/2 PFV × ET, is depicted on the *right*. See text for details. Abbreviations: *ET* = ejection time; *AT* = acceleration time, and *DT* = deceleration time, as in Fig. 1.

An additional parameter, the aortic flow velocity integral or the area under the aortic flow velocity curve (in centimeters), was planimetered from the beat demonstrating the greatest peak flow velocity in each of the 20 normal subjects and in 12 cardiomyopathy patients. In addition, a mathematical approximation to the planimetered area under the flow velocity curve, based on multiplying peak flow velocity (PFV in centimeters per second) times one half the ejection time (ET in seconds),[4] was compared to the planimetered flow velocity integral. The derivation of this mathematical model, depicted in Fig. 2, is as follows (with TR = triangle): TR A + TR B + TR A′ + TR B′ = PFV × ET. Since TR A = TR A′ and TR B = TR B′, then 2 TR A + 2 TR B = PFV × ET. Therefore, TR A + TR B = 1/2(PFV × ET). Because of beat-to-beat variations in flow velocity parameters related to respiration, pulsus alternans, etc., we utilized for our measurements the beat displaying the greatest peak flow velocity. Patients with atrial fibrillation were excluded from our study population.

Statistical comparisons. Doppler flow velocity measurements in the aorta and pulmonary artery in the

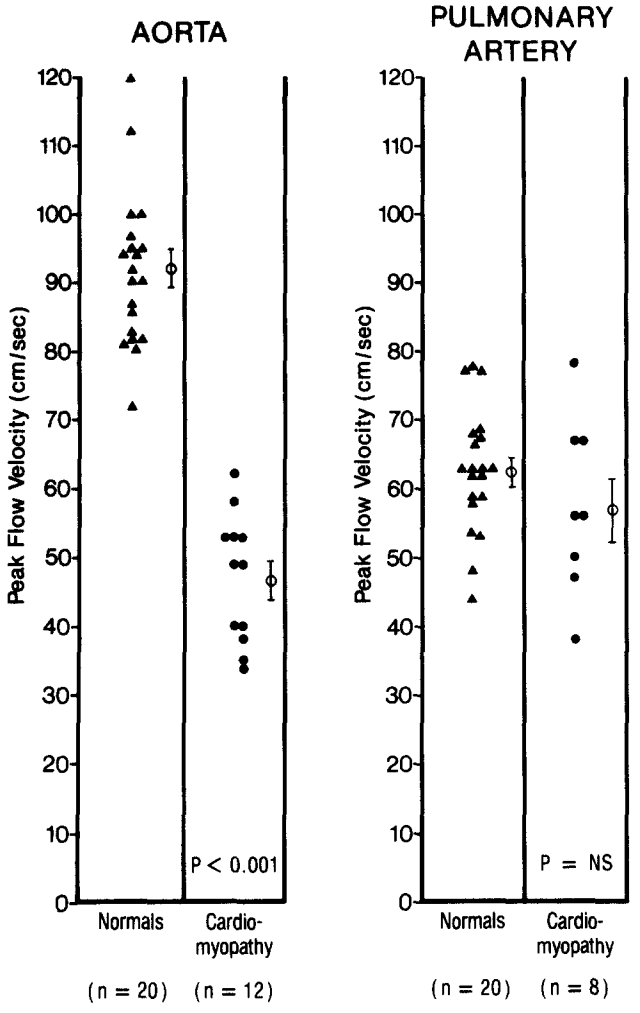

Fig. 3. Peak flow velocity in the ascending aorta *(left panel)* and main pulmonary artery *(right panel)* are plotted for 20 normal subjects and 12 cardiomyopathy patients. Pulmonary artery data are available in eight of the cardiomyopathy patients. The mean in each group is expressed by an *open circle* bisected by a horizontal line. The standard error of the mean is depicted on *either side* of the mean symbol. The *p* values reflect the comparison between the two groups in each panel.

cardiomyopathy patients were compared to data obtained in 20 normal subjects[12] utilizing Student's *t* test. In addition, tests of normality of sample distribution and nonparametric statistical tests were performed because of the relatively small population sample sizes. The nonparametric tests did not yield different statistical conclusions from the parametric tests. Consequently, the two-tailed *p* values reported reflect those calculated from Student's *t* test analysis. Standard linear regression analysis was utilized to calculate the correlations between aortic peak flow velocity and left ventricular percent fractional shortening, as well as between the flow velocity integral as measured by planimetry and as estimated from the mathematical model.

RESULTS

Blood flow velocity data from the ascending aorta and main pulmonary artery were recorded in all 20 normal subjects. Ascending aortic flow velocity patterns could be recorded in all 12 cardiomyopathy patients. However, since the two-dimensional transducer was not available when four of the studies were performed, pulmonary flow velocity was recorded in only 8 of the 12 cardiomyopathy patients (67%). Fig. 1 depicts typical ascending aortic flow velocity recordings from a normal subject and from a patient with dilated cardiomyopathy. By graphical inspection, it can be seen that peak aortic flow velocity and flow velocity integral are lower and ejection time shorter in the cardiomyopathy patient. The average acceleration is also lower in the cardiomyopathy patient, but this is the least obvious on casual inspection, since the peak flow velocity attained in the cardiomyopathy patient is less than in the normal subject.

Peak flow velocity. Aortic and pulmonary flow velocity data in the normal subjects and in cardiomyopathy patients are displayed in Figs. 3 to 6. Peak flow velocities in the ascending aorta and main pulmonary artery are plotted for normal subjects and cardiomyopathy patients in Fig. 3. In the aorta, peak flow velocities range from 35 to 62 cm/sec (mean 47) in cardiomyopathy patients and from 72 to 120 cm/sec (mean 92) in normals, a significant difference ($p < 0.001$). Furthermore, there is no overlap in the data points between the two groups. In the main pulmonary artery, peak flow velocities range from 38 to 78 cm/sec (mean 57) in cardiomyopathy patients versus 44 to 77 cm/sec (mean 62) in normals. This difference is not statistically significant and there is considerable overlap of data in the two groups.

Acceleration time. Acceleration time in the aorta ranged from 55 to 98 msec (mean 73) in the cardiomyopathy patients and from 83 to 118 msec (mean 98) in normals. Although there is overlap in the data from the two groups, mean acceleration time is significantly decreased ($p < 0.001$) in the cardiomyopathy patients. Data for average acceleration in the aorta are plotted in Fig. 4. In the cardiomyopathy patients, average acceleration (range 389 to 921 cm/sec/sec, mean 659) is significantly less ($p < 0.001$) than in the normal subjects (range 735 to 1318 cm/sec/sec, mean 955). However, there is moderate overlap of the average acceleration data between the two groups.

In the main pulmonary artery, acceleration time ranged from 55 to 128 msec (mean 101) in cardiomyopathy patients and from 130 to 185 (mean 159

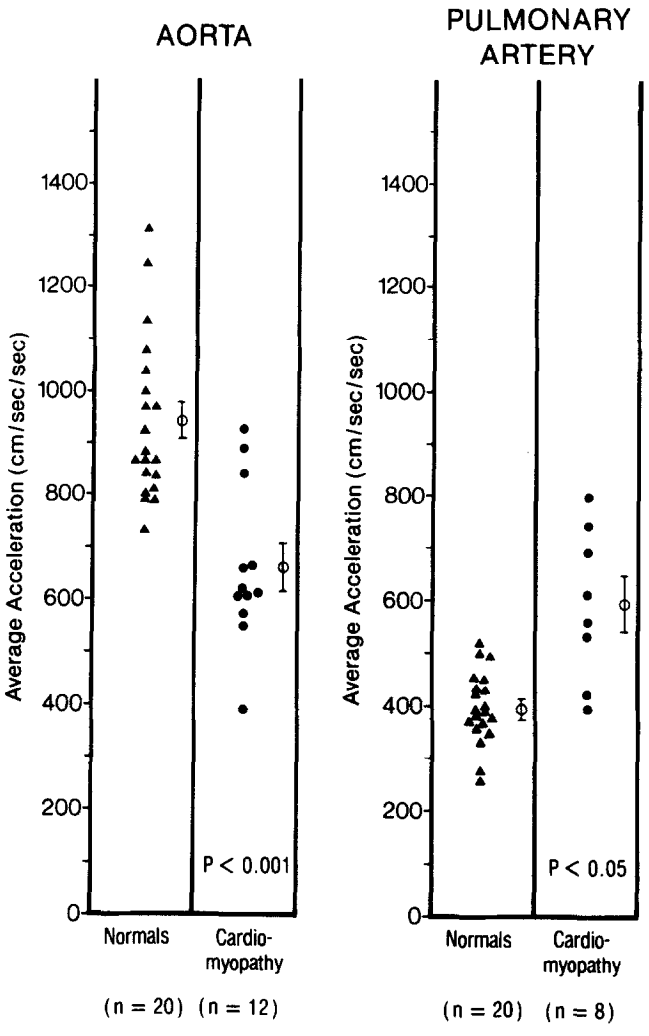

Fig. 4. Average acceleration in normal subjects and cardiomyopathy patients are plotted for the ascending aorta *(left panel)* and the main pulmonary artery *(right panel)*. Symbols are as in Fig. 3.

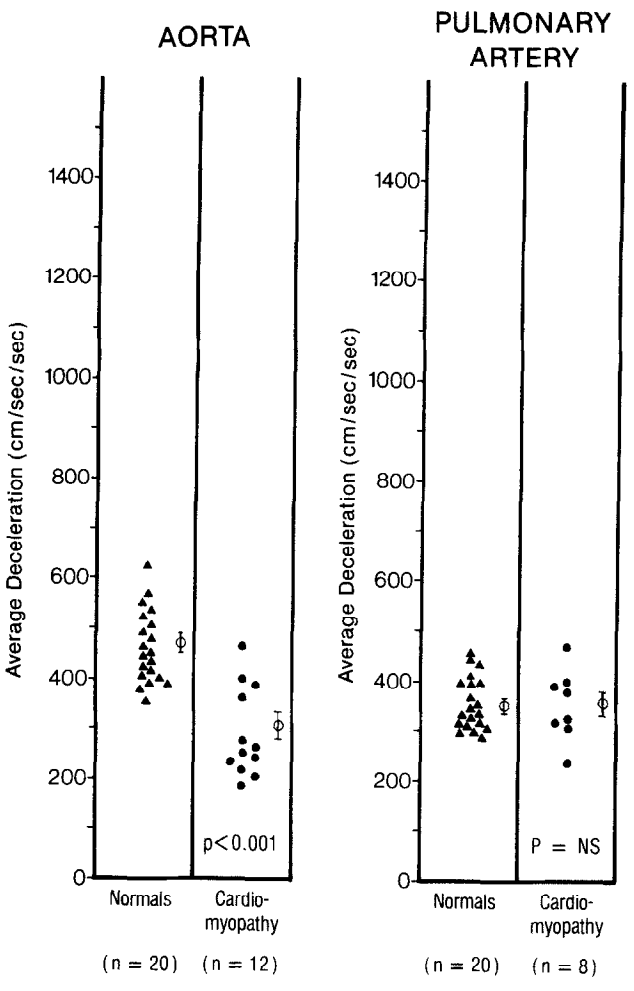

Fig. 5. Average deceleration data in the ascending aorta *(left panel)* and main pulmonary artery *(right panel)* are plotted for normal subjects and cardiomyopathy patients. Symbols are as in Fig. 3.

msec) in normals. Thus, mean pulmonary acceleration time is significantly decreased in the cardiomyopathy group ($p < 0.01$). Data for average acceleration in the main pulmonary artery are also plotted in Fig. 4. Because pulmonary artery peak flow velocity is similar whereas mean acceleration time is decreased in the cardiomyopathy patients versus normals, average acceleration in the main pulmonary artery is significantly greater ($p < 0.05$) in cardiomyopathy patients than in normals. There is, however, overlap in the data from the two groups with average acceleration ranging from 392 to 800 cm/sec/sec (mean 594) in the cardiomyopathy patients and from 270 to 515 cm/sec/sec (mean 396) in normals.

Deceleration time. Deceleration time in the ascending aorta ranged from 93 to 220 msec (mean 164) in the cardiomyopathy patients and from 170 to 230 msec (mean 197) in normals. There is considerable overlap in the data for deceleration time, the difference between the two groups being of borderline statistical significance ($p = 0.05$). Data for average deceleration in the aorta are plotted in Fig. 5. In the cardiomyopathy group, data for average deceleration (mean 301 cm/sec/sec, range 194-477) overlap with data in normals (mean 473 cm/sec/sec, range 355 to 630), but there is a statistically significant difference ($p < 0.001$).

In the main pulmonary artery, deceleration time ranged from 95 to 230 msec (mean 164) in cardiomyopathy patients and from 148 to 208 msec (mean 172) in normals. The data in these two groups overlap considerably and are statistically similar. Likewise, data for average deceleration in the pul-

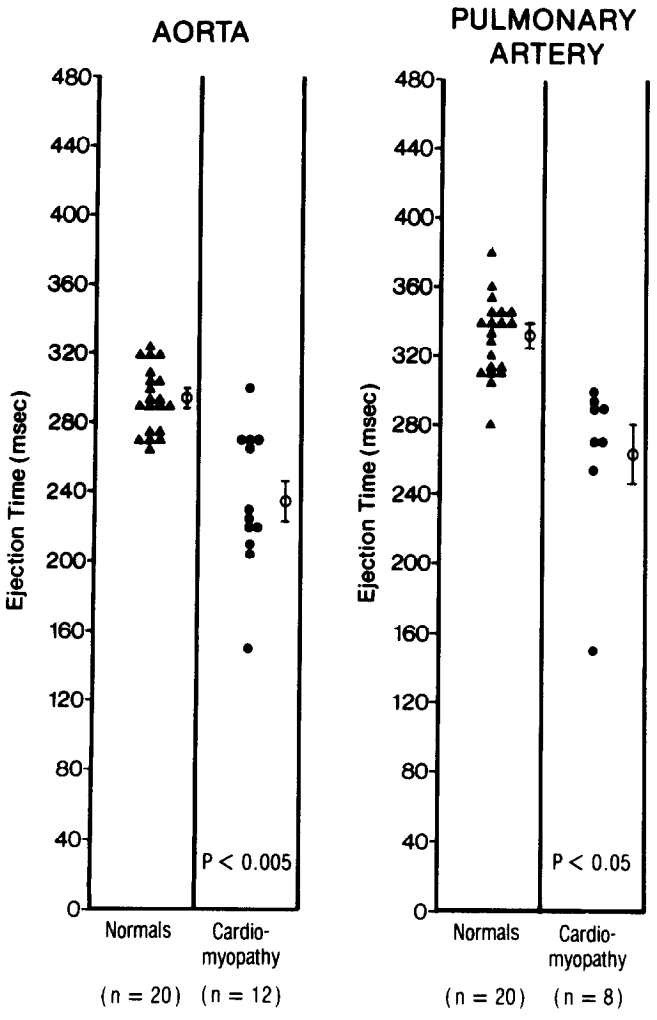

Fig. 6. Ejection time measured in the ascending aorta *(left panel)* and main pulmonary artery *(right panel)* are plotted for normal subjects and cardiomyopathy patients. Symbols are as in Fig. 3.

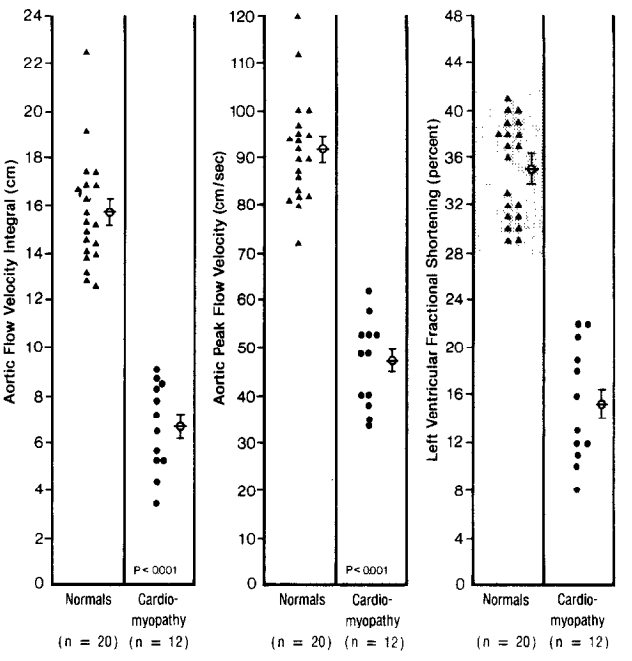

Fig. 7. Aortic flow velocity integral *(left panel)*, aortic peak flow velocity *(middle panel)*, and left ventricular percent fractional shortening *(right panel)* are compared in normal subjects and in patients with cardiomyopathy. Note that the data for all three measurements separate normal subjects from patients with cardiomyopathy to a similar degree. *Shaded area* represents normal range for left ventricular percent fractional shortening.[16]

monary artery (Fig. 5) are statistically similar and show considerable overlap between cardiomyopathy patients (range 243 to 479 cm/sec/sec, mean 357) and normal subjects (range 297 to 460 cm/sec/sec, and mean 356).

Ejection time. Data for ejection times measured in the ascending aorta and main pulmonary artery are plotted in Fig. 6. In the aorta, ejection time ranged from 150 to 300 msec (mean 236) in cardiomyopathy patients and from 265 to 325 msec (mean 294) in normals. Although there is moderate overlap in the data, there is a significant difference ($p < 0.005$) between the two groups. Although mean heart rate in the cardiomyopathy patients (79 bpm) is 16 bpm greater than in the normal subjects, correction of the ejection times for heart rate utilizing the formula of Weissler et al.[20] still results in a statistically significant difference ($p < 0.05$). In the main pulmonary artery, ejection time is also significantly shorter ($p < 0.05$) in cardiomyopathy patients (range 150 to 300 msec, mean 265) than in normals (range 280 to 380 msec, mean 331), with moderate overlap of data between the two groups. In both cardiomyopathy and normal groups, mean ejection time is longer in the pulmonary than in the ascending aorta.

Aortic flow velocity and LV fractional shortening. In the normals, mean aortic flow velocity integral (determined by planimetry of the area under the flow velocity curve) was 15.7 cm with a range of 12.6 to 22.5, whereas in the cardiomyopathy patients, mean flow velocity integral was 6.7 cm with a range of 3.5 to 9.1 (Fig. 7, *left panel*). The flow velocity integral was significantly different in the two groups ($p < 0.001$), with no overlap in the data. The mathematical approximation for flow velocity integral, FVI (EST), correlated well ($r = 0.97$) with the planimetered area under the flow velocity curve, FVI (PLAN). The following regression equation describes their relationship: FVI (PLAN) = 1.14 FVI(EST) + 0.3, where FVI(EST) = PFV × 1/2 ET.

Fig. 7 displays a comparison of data for aortic flow velocity integral, peak aortic flow velocity, and left

ventricular percent fractional shortening in the normal and cardiomyopathy patients. The two groups, it should be noted, were defined in such a manner that there was no overlap in data for left ventricular percent fractional shortening. Aortic peak flow velocity (PFV) and left ventricular percent fractional shortening (LV%FS) in the normal subjects and cardiomyopathy patients were related by the regression equation PFV = 2.8 (LV%FS) − 0.9, with a good correlation ($r = 0.83$).

There was an excellent correlation between aortic peak flow velocity and aortic flow velocity integral ($r = 0.98$) in the normal and cardiomyopathy groups. Consequently, the regression equation relating planimetered aortic flow velocity integral [FVI (PLAN)] and left ventricular percent fractional shortening, FVI (PLAN) = 0.39 (LV%FS) + 1.6, also demonstrated a good correlation ($r = 0.84$).

DISCUSSION

Aortic PFV. Our study demonstrates that Doppler blood flow measurements are useful in differentiating normal subjects from patients with poor left ventricular systolic function due to dilated (congestive) cardiomyopathy. Aortic PFV proved to be an excellent discriminator between the two groups. In the present study, aortic PFV averaged 92 cm/sec (range 72 to 120) in normal subjects but was significantly reduced to an average of 47 cm/sec (range 35 to 62) in patients with dilated cardiomyopathy. There was no overlap in measurements from the two groups. Although the mechanism for the reduction in aortic flow velocity in patients with dilated cardiomyopathy is not entirely known, our data suggest that it may be directly related to a decrease in left ventricular systolic function. This suggestion is based on the observation that there is good correlation ($r = 0.83$) between LV%FS and aortic PFV.

Aortic flow velocity integral. Another measurement that allowed excellent separation of the normal subjects and cardiomyopathy patients was the aortic flow velocity integral (FVI). The FVI (in centimeters), when multiplied by the cross-sectional area of the vessel (in square centimeters), should theoretically give a noninvasive estimate of stroke volume (in cubic centimeters). Thus the FVI is an approximation of aortic stroke volume. That the aortic flow velocity integral and peak flow velocity were both good discriminators of reduced left ventricular systolic function is not surprising, because several studies have suggested a relationship between aortic PFV and left ventricular stroke volume. Colocousis et al.[1] demonstrated during experimental exsanguination and fluid infusion in dogs that Doppler aortic PFV is linearly related to stroke volume—except at higher cardiac outputs, where stroke volume is augmented by a prolongation of ejection time while peak velocity reaches a plateau. Spence[21] has shown in hypertensive patients that aortic PFV is increased after nadalol treatment. This finding was felt to be secondary to a decrease in heart rate with a related increase in stroke volume. Moreover, recent data from our laboratory[22] have shown a good correlation between percent changes in PFV (and FVI) and percent changes in invasively measured stroke volume and systemic vascular resistance following therapeutic interventions.

Aortic acceleration. In the present study, average aortic acceleration was found to be reduced in patients with dilated cardiomyopathy compared to normals. Although the reductions were highly statistically significant, there was some overlap between measurements from cardiomyopathy patients and normal subjects. The observation that there is a relationship between average aortic blood flow acceleration and left ventricular systolic function reinforces the preliminary findings of Chandraratna et al.,[23] who found a positive correlation between maximum blood flow acceleration in the transverse aorta and left ventricular ejection fraction in patients with coronary artery disease. However, our data indicate that both aortic PFV and aortic flow velocity integral appear to better discriminate between the cardiomyopathy and normal groups than does average aortic blood flow acceleration. One explanation for this finding may be that small errors in measurement of the acceleration time result in relatively large errors in calculation of average acceleration. That this may indeed be a problem is suggested by another study from our laboratory[12] in which we evaluated reproducibility of Doppler aortic flow velocity measurements recorded in 10 normal subjects by two technicians within 30 minutes of each other. In this study,[12] we found much larger variability in acceleration time and average acceleration measurements than in PFV or flow velocity integral measurements.

Ejection time. The mean ejection time in both the ascending aorta and main pulmonary artery of the cardiomyopathy patients also was significantly decreased relative to normal, a finding that previously has been well documented in the presence of reduced left ventricular function and low stroke volume. In the present study, ascending aortic ejection time measured by the Doppler technique, whether uncorrected or corrected for heart rate, was a good discriminator between normals and cardio-

myopathy patients. A similar finding was previously reported by Weissler et al.[20] using corrected left ventricular ejection time data derived from external carotid pulse tracings. However, in our study we found much more overlap in the two groups for ejection time measurements than for aortic flow velocity or flow velocity integral measurements.

Pulmonary artery versus ascending aorta. Doppler flow studies in the main pulmonary artery demonstrated significantly decreased acceleration time, increased average acceleration, and decreased ejection time in the cardiomyopathy patients compared to the normal subjects. Deceleration time, average deceleration, and PFV in the main pulmonary artery were not significantly different in the cardiomyopathy and normal groups.

The findings of decreased acceleration time and increased average acceleration in the pulmonary artery of the cardiomyopathy patients differs from the aortic blood flow velocity measurements in these patients. Although our data do not directly explain the differences, one might speculate that the cardiomyopathy patients have a higher pulmonary artery resistance compared to normals and that this may play some role in producing an increased acceleration of blood into the pulmonary artery, perhaps by stimulating right ventricular thickening. Support for this speculation includes the observation that in normals, average acceleration is almost three times greater in the aorta, despite an approximately five times higher resistance in the aortic than in the pulmonary arterial circulation.[24]

Review of our data indicates that the ascending aortic flow measurements appear to be more useful than pulmonary artery flow measurements in identifying and following patients with dilated cardiomyopathy and global left ventricular systolic dysfunction. Moreover, the ascending aortic flow measurements can be obtained without the necessity of two-dimensional echocardiographic imaging. Therefore, it is possible to adequately evaluate and follow these patients—e.g., in the intensive care unit[4, 22]—by obtaining data solely from the ascending aorta using an M-mode transducer in the suprasternal notch. If one wishes to measure stroke volume accurately, however, it will be necessary to accurately measure the cross-sectional area of the vessel through which the blood is flowing—possibly by utilizing two-dimensional echocardiography.[25]

Limitations. There are several potential sources of error in measurements of ascending aortic and pulmonary artery blood flow velocity. One source of error could result from placing the sample volume nonparallel to the long axis of blood flow. Another source of error could be related to dispersion of blood flow velocities during a given sampling interval, making it difficult to determine the exact PFV measurement. We attempted to minimize the former source of error by utilizing the two-dimensional transducer to place the sample volume parallel to the long axis of blood flow in the pulmonary artery and by the mapping technique to obtain the maximal PFV in the ascending aorta. We attempted to minimize the latter source of error by approximating the midpoint of the spectral dispersion at PFV and considering this point as the average value for PFV.

Conclusions. Our study has demonstrated some important potential applications of Doppler aortic blood flow velocity measurements in the evaluation of left ventricular function. The information derived from Doppler measurements is similar in some respects to data that can be obtained from M-mode echocardiography. For example, LV%FS, as measured by M-mode echocardiography, appears to correlate well ($r = 0.83$) with both aortic PFV and flow velocity integral, as measured by Doppler echocardiography. Both M-mode and Doppler measurements are useful in discriminating patients with normal left ventricular function from those with global left ventricular dysfunction (e.g., dilated cardiomyopathy). Although both techniques are noninvasive and can be performed repetitively on human subjects, pulsed Doppler echocardiography has one advantage that may make it an important addition to M-mode echocardiography in the quantitative assessment of left ventricular systolic function—i.e., Doppler aortic flow velocity measurements reflect function of the entire left ventricle rather than of a limited region at the base of the heart. This advantage of Doppler flow velocity measurement over M-mode echocardiography may prove to be particularly useful in patients with significant left ventricular wall motion abnormalities.

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