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Utility of Continuous Wave Doppler Echocardiography in the Noninvasive Assessment of Left Ventricular Outflow Tract Pressure Gradient in Patients With Hypertrophic Cardiomyopathy

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Subaortic obstruction is an important determinant of the clinical presentation of and therapeutic approach to patients with hypertrophic cardiomyopathy. Therefore, assessment of the presence and magnitude of the intraventricular pressure gradient is paramount in the clinical evaluation of these patients. To establish the utility of continuous wave Doppler echocardiography in assessing the pressure gradient in hypertrophic cardiomyopathy, 28 patients representing the wide hemodynamic spectrum of this disease underwent simultaneous determination of the subaortic gradient by continuous wave Doppler ultrasound and cardiac catheterization.

With use of the modified Bernoulli equation, the Doppler-estimated gradient showed a strong correlation with the maximal instantaneous pressure difference measured at catheterization, both under basal conditions ($r = 0.93$; $p < 0.0001$) and during provocative maneuvers ($r = 0.89$; $p < 0.0001$). In 26 of the 28 patients, all assessments of the subaortic gradient were in agreement within 15 mm Hg (average difference 5 ± 3 mm Hg). In the other two patients there were substantial differences between these measurements (under basal conditions in one patient and after provocation in another), although the Doppler technique predicted the presence of marked subaortic obstruction in each. In both patients the erroneous interpretation was due to superimposition of the mitral regurgitation signal on that of left ventricular outflow.

Doppler waveforms from the left ventricular outflow tract showed variability in contour among different patients and in individual patients. Hence, continuous wave Doppler echocardiography is a useful noninvasive method for estimating the subaortic gradient in patients with hypertrophic cardiomyopathy. However, technical factors such as contamination of the outflow tract jet with that of mitral regurgitation and variability in waveform configuration may importantly influence such assessments of the subaortic gradient.

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A substantial proportion of patients with hypertrophic cardiomyopathy demonstrate a left ventricular outflow tract pressure gradient under basal conditions or with provocative maneuvers (1,2). Such subaortic gradients are characteristically dynamic and produced by mid-systolic contact between the mitral valve and ventricular septum (2–7). Marked elevation in left ventricular systolic pressure due to outflow obstruction may be an important determinant of symptoms and clinical course (2,8). Furthermore, the magnitude of the subaortic gradient may show important lability and change either spontaneously or after therapeutic interventions, such as drug treatment or operation (1,2,9–12). Consequently, identification and serial assessment of the outflow gradient may be an important facet of the longitudinal evaluation of patients with hypertrophic cardiomyopathy. To obviate performing serial invasive cardiac catheterizations for this purpose, it is desirable to acquire a noninvasive and easily accessible capability for reliably estimating the subaortic gradient in patients with this disease.

Continuous wave Doppler echocardiography has achieved widespread application for the noninvasive estimation of gradients in patients with valvular heart disease (13–16). This technique has also been assessed in hypertrophic cardiomyopathy, although thus far studies have been restricted to a small number of patients (17) and selected investigations performed in the operating room (18). Therefore, the present study was undertaken in a sizable group of patients with hypertrophic cardiomyopathy demonstrating a wide range of gradients measured at cardiac catheterization to investigate the utility and limitations of continuous wave Doppler ultrasound in the noninvasive assessment of the subaortic gradient in this disease.

Methods

Selection of patients. From January to May 1988, 56 patients who had not had surgery were admitted to the

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Cardiology Branch, National Heart, Lung, and Blood Institute, for evaluation of hypertrophic cardiomyopathy and cardiac catheterization. Before cardiac catheterization, complete echocardiographic studies were performed in each patient. This examination included a continuous wave Doppler interrogation for the purpose of identifying peak left ventricular outflow tract velocity (and estimating the subaortic gradient), as well as prospectively determining the optimal transducer position and angulation necessary to achieve definition of this signal in advance of the catheterization study.

In 9 of the 56 patients the Doppler examination did not yield a left ventricular outflow tract signal of sufficient technical quality and these patients were excluded. The remaining 47 patients underwent simultaneous continuous wave Doppler studies and intracardiac pressure measurements in the catheterization laboratory. However, in 19 of these 47 patients, although Doppler interrogation of outflow velocities in the echocardiography laboratory had yielded a satisfactory signal, the Doppler examination obtained during cardiac catheterization was unsatisfactory largely because of restrictions of the catheterization procedure itself, that is, inability to adequately modify the patient's body position on the catheterization table to permit proper identification of the left ventricular outflow tract signal. Usually this involved patients who could not be readily placed in the left lateral decubitus position during the catheterization procedure and in whom a technically adequate Doppler study could not be achieved in the supine position. Consequently, the remaining 28 patients who underwent cardiac catheterization with simultaneous and technically adequate continuous wave Doppler examination constitute the present study group.

Characterization of patients. The diagnosis of hypertrophic cardiomyopathy was confirmed in each of the 28 patients by the echocardiographic demonstration of a hypertrophied nondilated left ventricle in the absence of any other cardiac or systemic disease that could itself produce the magnitude of left ventricular hypertrophy observed (19). Study patients ranged in age from 18 to 72 years (mean 43); 17 (60%) were male. In each patient cardioactive medications were withdrawn at least 3 days before the catheterization study. All patients had normal sinus rhythm at the time of cardiac catheterization. Patients gave informed consent for all procedures.

Echocardiography. Echocardiographic examinations were performed with use of commercially available instruments. Images were obtained in several cross-sectional planes with use of standard transducer positions (20). With use of previously described methods (21), the distribution of left ventricular hypertrophy was assessed primarily in the parasternal short-axis plane, although parasternal long-axis and apical views were also used to integrate the information obtained from the short-axis images.

In the short-axis plane, the left ventricle was divided into four segments that constituted the anterior and posterior ventricular septum and anterolateral and posterior left ventricular free walls (22). The presence and extent of left ventricular hypertrophy in these four left ventricular regions were evaluated in diastole directly from the television monitor with the aid of calipers. Wall thickness was measured at the levels of both the mitral valve and the papillary muscles (21,22). For each region of the left ventricle, the portion that showed the greatest thickness (whether situated basally or apically) was considered as the maximal thickness of that segment.

M-mode echocardiograms were derived from the two-dimensional image under direct anatomic visualization. Cardiac dimensions were assessed according to the criteria of the American Society of Echocardiography (23).

Continuous wave Doppler examination. The examinations were obtained with a Hewlett-Packard ultrasound system utilizing a 1.9-MHz nonimaging transducer. Studies were performed from the apical window with the patient in the supine or left lateral decubitus position during quiet respiration. This transducer position was selected with regard to our prior experience in patients with hypertrophic cardiomyopathy, that Doppler interrogation of the left ventricular outflow tract from the apex yields a satisfactory signal more consistently than do examinations performed from the second right intercostal space or suprasternal notch.

Each study was performed by the same technologist who was instructed to angulate the transducer to obtain the maximal velocity signal from the left ventricular outflow tract. In particular, an effort was made to isolate a spectral profile showing a relatively slow increase in velocity culminating in a delayed peak velocity in mid-systole associated with the characteristic high pitched acoustic properties of the left ventricular outflow tract jet. In our experience and that of other laboratories (24,25), such waveforms are typical of patients with hypertrophic cardiomyopathy.

Gain and filter settings were then adjusted to obtain the signal with the highest audible frequency, the maximal peak velocity and the optimal signal to noise ratio. Also, particular care was taken to separate the outflow tract signal from that of mitral regurgitation by orienting the transducer more medially and anteriorly whenever a regurgitant jet was identified. The signal of mitral regurgitation was characterized by earlier onset, more abrupt initial increase in velocity and higher peak velocity than that of the outflow tract signal. To minimize any potential bias on the part of the technologist with regard to selection of beats (that is, waveforms) for recording, other participants in the cardiac catheterization procedure were instructed to avoid disclosing the value or approximate magnitude of the measured pressure gradient.

Measurement of pressure gradient. The pressure gradient across the left ventricular outflow was measured by recording pressures obtained simultaneously from fluid-filled catheters placed within the body of left ventricle and femoral artery. Left ventricular pressure was measured with an end-hole catheter and the femoral artery pressure with a 24-in. (10.2-cm) Teflon catheter. Care was taken to ensure that the elevated left ventricular systolic pressure was not

artifactually produced by entrapment of the catheter within left ventricular trabeculations, based on previously described criteria (9,10). Also, in each patient an aortic valve gradient was excluded by pullback recordings of the left ventricular catheter. In 22 of the 28 patients (all with a basal outflow gradient <100 mm Hg), Doppler waveforms from the left ventricular outflow tract of sufficient technical quality were obtained during the simultaneous recording of left ventricular and femoral artery pressures with a provocative maneuver, that is, intravenous infusion of isoproterenol (until a heart rate ≥120 beats/min was achieved) or a catheter-induced premature ventricular contraction, or both.

The systolic left ventricular outflow pressure gradient was measured as the maximal instantaneous pressure difference, as well as the peak to peak difference, between the left ventricular and femoral artery pressures. In calculating the maximal instantaneous pressure gradient, small signal delays inherently present between left ventricular and distal arterial pressure recording sites, as well as the delay intrinsic to fluid-filled catheter systems (about 10 ms), were compensated for by shifting the arterial pressure tracing leftward so that its initial pressure increase coincided with that of the left ventricular tracing.

Simultaneous Doppler and hemodynamic measurements. Once the optimal Doppler signal from the left ventricular outflow tract was achieved in the catheterization laboratory, Doppler and intracavitary pressure recordings were initiated simultaneously. Separate strip-chart recordings of the continuous wave Doppler spectral profiles and pressure tracings were both obtained at 100 mm/s for 60 to 90 s, sufficient to include about 60 to 120 beats. An electrocardiographic (ECG) tracing was recorded on both the Doppler and the pressure tracings. Induced premature ventricular contractions were utilized to match the Doppler and pressure tracings in time.

Analysis of Doppler recordings. Continuous wave Doppler spectral recordings and pressure tracings were interpreted independently by two investigators. The observer who interpreted the Doppler recordings did not have prior knowledge of the patient's clinical and hemodynamic findings and was not present in the cardiac catheterization laboratory when the simultaneous Doppler and catheterization studies were performed.

The reader analyzed only those beats having a Doppler spectral signal that was judged to represent the velocities across the left ventricular outflow tract and for which there was sharp and unambiguous definition of the entire waveform contour, so that peak velocity could be ascertained and measured with a high degree of accuracy. Measurements were made of the beats with the highest peak velocity, although those judged to represent mitral regurgitation were disregarded.

Orientation of the Doppler beam was assumed to be virtually parallel to the direction of the systolic flow (<20° angle of incidence) and therefore no routine angle correction was incorporated in the calculation of outflow gradient from the continuous wave Doppler waveform. Left ventricular outflow pressure gradient was estimated by utilizing the modified Bernoulli equation (13,16): $G = 4V^2$, where G = gradient (in mm Hg) and V = maximal flow velocity (in m/s).

Reproducibility. Interobserver variability in the measurement of peak left ventricular outflow tract velocity from the Doppler spectral profile was assessed independently by two investigators in a separate group of 34 consecutive patients with hypertrophic cardiomyopathy who had undergone continuous wave Doppler examination in the echocardiography laboratory but were not part of the primary study group.

These 34 patients were selected to ensure that the investigators participating in the interobserver variability analysis were not biased. Because only one of the investigators involved in this study was unaware of the pressure gradients recorded during cardiac catheterization, the Doppler tracings obtained in the catheterization laboratory could not be included in the assessment of interobserver variability.

In each of the 34 patients, three consecutive beats were selected for analysis (by a third observer); both investigators measured peak velocity in each beat and these values were then averaged. The mean values obtained by the two investigators were then compared and their relation analyzed statistically.

Statistical analysis. Relations between different variables were assessed by means of Pearson's correlation coefficient and linear regression analysis. A p value < 0.05 was considered to be statistically significant. All values reported are mean values ± SD.

Results

Echocardiography

An asymmetric pattern of left ventricular hypertrophy was identified in each patient. Wall thickening was confined to the anterior ventricular septum in 3 patients, involved both the anterior and the posterior ventricular septum (but not the left ventricular free wall) in 5 and diffusely involved substantial portions of both the septum and the anterolateral free wall in 19. The remaining patient showed a more unusual pattern of hypertrophy with thickening of the posterior septum and the anterolateral free wall (26). Maximal left ventricular wall thickness (usually of the anterior septum) ranged from 15 to 40 mm (mean 23).

Left ventricular end-diastolic cavity dimensions were 32 to 54 mm (mean 41) and end-systolic dimensions were 10 to 38 mm (mean 25). Left atrial dimension was 29 to 60 mm (mean 47) and was >45 mm in 10 patients. Systolic anterior motion of the mitral valve was present in 24 patients but resulted in mitral-septal apposition in only 16.

Correlation of Doppler and Hemodynamic Measurements of Outflow Gradient

Basal gradient. In each of the 28 study patients, 5 to 25 beats recorded at cardiac catheterization under basal

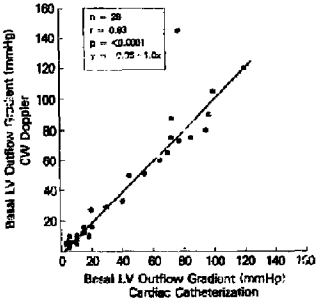


Figure 1. Correlation between the peak instantaneous left ventricular (LV) outflow gradient measured at cardiac catheterization and the simultaneously obtained gradient estimated by continuous wave (CW) Doppler ultrasound under basal conditions in 28 patients with hypertrophic cardiomyopathy.

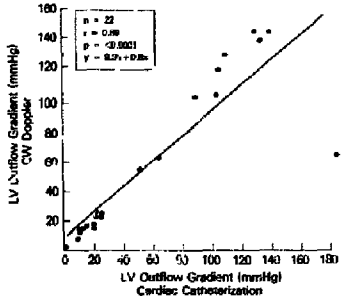


Figure 2. Correlation between the peak instantaneous left ventricular (LV) outflow gradient measured at cardiac catheterization and the simultaneously obtained gradient estimated by continuous wave (CW) Doppler ultrasound after provocation maneuvers in 22 patients with hypertrophic cardiomyopathy.

conditions were analyzed (mean 13 ± 5 beats/patient); a total of 355 beats were analyzed in the overall study group. Maximal instantaneous pressure gradient across the left ventricular outflow tract ranged from 5 to 120 mm Hg (average 45); peak flow velocity ranged from 1.2 to 6 m/s (mean 3.4). There was a highly significant correlation between the maximal instantaneous pressure gradient measured at catheterization and the gradient estimated by Doppler recording ($r = 0.93$; $p < 0.0001$) (Fig. 1). In 27 of the 28 patients, Doppler and catheterization assessments of the basal outflow gradient agreed within 15 mm Hg (average difference 5.5 ± 3 mm Hg). In the remaining patient, Doppler assessment overestimated the recorded gradient by 68 mm Hg (146 vs. 78 mm Hg).

Peak to peak basal left ventricular outflow tract pressure gradient ranged from 0 to 104 mm Hg (mean 35). A highly significant correlation was also identified between the peak to peak and Doppler-estimated gradients ($r = 0.91$; $p < 0.0001$). When patients were analyzed individually, the Doppler-estimated gradient exceeded the catheterization gradient in each. In 27 of the 28 patients, Doppler and catheterization gradients agreed within 25 mm Hg (average difference 10 ± 6 mm Hg); in the remaining patient Doppler ultrasound overestimated the recorded gradient by 80 mm Hg (146 vs. 66 mm Hg).

Provokable gradient. In 22 of the 28 patients, a technically satisfactory Doppler signal was recorded from the left ventricular outflow tract during the infusion of isoproterenol (15 patients) or in the beat after a premature ventricular contraction (7 patients). In these 22 patients, the maximal provokable instantaneous pressure gradient ranged from 8 to 185 mm Hg (mean 65); peak outflow velocity ranged from 1.5 to 6.2 m/s (mean 3.9). There was a highly significant correlation between the measured maximal instantaneous pressure gradient and the Doppler-estimated gradient ($r = 0.89$; $p < 0.0001$) (Fig. 2). In 21 of the 22 patients, Doppler and catheterization assessments of instantaneous outflow gradient agreed within 15 mm Hg (average difference 8 ± 4 mm Hg); in the remaining patient, Doppler assessment underestimated the gradient by 120 mm Hg (65 vs. 185 mm Hg).

Patients with a variable gradient. In 6 of the 28 patients, a particularly wide range of subaortic pressure gradients was recorded during the cardiac catheterization procedure (either under basal conditions or with provocation). These patients showed a difference of 40 to 120 mm Hg (mean 78 ± 26) between the minimal and maximal peak instantaneous gradient recorded. Correlation between the measured and Doppler-estimated gradients was very strong in each of these patients; correlation coefficients for individual patients ranged from 0.87 to 0.98 (Fig. 3).

Interobserver variability. Peak flow velocities measured by the first observer ranged from 1.2 to 6.1 m/s (mean 3.4 ± 1.1) and those measured by the second observer were 1.2 to 6 m/s (mean 3.4 ± 1.1). Mean interobserver differences were 0.06 ± 0.05 m/s (range 0 to 0.2). Correlation of peak flow velocity measurements between the two independent observers was very strong ($r = 0.99$; $p < 0.0001$) (Fig. 4).

Retrospective Analysis of Doppler Studies

Doppler studies were also reviewed in a retrospective fashion to derive explanations for certain observed discrepancies between the gradients estimated by Doppler ultra-

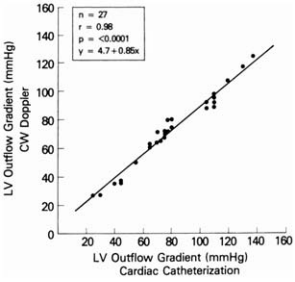
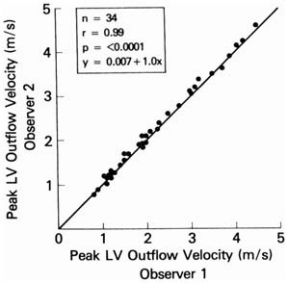


Figure 3. Correlation between the peak instantaneous left ventricular (LV) outflow gradient measured at cardiac catheterization and the simultaneously obtained gradient estimated by continuous wave (CW) Doppler ultrasound in a 24-year old patient with hypertrophic cardiomyopathy who demonstrated a particularly variable subaortic gradient. Depicted are 27 beats with a wide range of gradients obtained under basal conditions and during provocation maneuvers.

sound and those recorded in the catheterization laboratory. This analysis showed that the major discrepancies were the consequence of a superimposition of the signals from the mitral regurgitation and left ventricular outflow tract jets. Thus, in 26 of the 28 patients either no mitral regurgitation signal was identified on the tracing or the outflow tract signal could be separated distinctly from a signal of mitral regurgitation (Fig. 5). However, in two patients (one assessed under basal conditions and one with provocation), the signal corresponding to the mitral regurgitant jet was either erroneously identified as representing part of the outflow tract signal or overlapped with the outflow spectral profile, so that it was not possible to accurately assess the peak outflow

Figure 4. Correlation between Doppler left ventricular (LV) outflow tract peak velocity measurements made by two independent observers in 34 patients with hypertrophic cardiomyopathy.



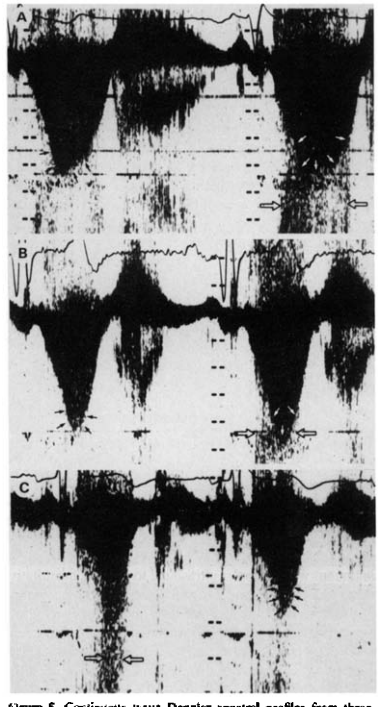


Figure 5. Continuous wave Doppler spectral profiles from three patients (A, B and C) with obstructive hypertrophic cardiomyopathy showing examples of superimposition of a mitral regurgitation jet on that of left ventricular outflow tract. For each patient the two waveforms shown correspond to consecutive beats. In each patient the "true" outflow tract signal (which correlated with the subaortic gradient measured at cardiac catheterization) (**small black arrows**) was successfully isolated from the signal of mitral regurgitation (**large white arrows**). It was often possible to identify the outflow waveform (**small white arrows in the second beat**) within the mitral regurgitation spectral profile. Even minimal changes in orientation of the Doppler beam (including those secondary to the heart motion) can result in superimposition of the two signals because of the particularly close proximity between the outflow tract and mitral regurgitant jets. In this and other figures showing continuous wave Doppler tracings calibration marks are 1 m/s apart.

tract velocity. Failure to recognize these distinctions resulted in overestimation or underestimation of the outflow gradient in these two patients.

Clinical Findings

Patients experienced a wide variety of symptoms, including exertional dyspnea (n = 18), chest pain (n = 15), fatigue (n = 13), palpitation (n = 12) and syncope (n = 10). Twelve patients were either asymptomatic or mildly symptomatic at the time of evaluation; in these patients cardiac catheterization was performed as part of an electrophysiologic study for evaluation of syncope (n = 8) or nonsustained ventricular tachycardia during ambulatory Holter ECG monitoring (n = 4). The remaining 16 patients were severely symptomatic despite appropriate medical therapy and underwent cardiac catheterization as part of a preoperative evaluation before ventricular septal myotomy-myectomy.

Genetic transmission of hypertrophic cardiomyopathy to first-degree relatives (27) could be substantiated in 16 of the 28 study patients. Although there was no family history of hypertrophic cardiomyopathy in the remaining 12 patients, systematic echocardiographic studies were not performed in these pedigrees.

Discussion

Doppler assessment of the outflow tract gradient. Continuous wave Doppler echocardiography has proved to be useful in quantitatively assessing the degree of fixed left ventricular outflow tract obstruction in patients with aortic valve stenosis (15,16). The results of the present investigation demonstrate that in a large group of patients with hypertrophic cardiomyopathy it is also possible to utilize continuous wave Doppler ultrasound to derive a quantitative estimate of the dynamic left ventricular outflow tract gradient characteristic of these patients. In our study group of 28 patients who represent the wide hemodynamic spectrum of hypertrophic cardiomyopathy, we found a strong correlation between outflow gradient estimated by Doppler ultrasound (and calculated with use of a modification of the Bernoulli equation) (13,16) and the peak instantaneous, as well as peak to peak, gradient recorded simultaneously in the catheterization laboratory. Furthermore, this strong relation was observed for outflow gradients assessed both under basal conditions and with provocative maneuvers. These findings show that it is possible to utilize continuous wave Doppler ultrasound in patients with hypertrophic cardiomyopathy to estimate outflow gradients noninvasively in an ambulatory setting, obviating the necessity of performing multiple cardiac catheterizations over the longitudinal follow-up period.

Previous reports. Two previous studies (17,18) analyzed simultaneously obtained Doppler-estimated and catheter-measured outflow gradients in patients with obstructive hypertrophic cardiomyopathy and showed good correlations between both measurements. However, each of these studies has certain limitations with respect to the selection of patients and application to the clinical setting. For example, Sasson et al. (17) analyzed the multiple recordings obtained with cardiac catheterization and simultaneous transthoracic Doppler echocardiography in a small group of only five selected patients. The study of Stewart et al. (18) was confined to measurements obtained in the operating room at the time of ventricular septal myotomy-myectomy in 10 patients, with the Doppler probe positioned directly on the ascending aorta to measure outflow tract velocity. In contrast, the present investigation utilized transthoracic echocardiography to study a large number of consecutively evaluated patients with hypertrophic cardiomyopathy representative of the broad hemodynamic and morphologic spectrum of the disease (1,2). This study design permitted us to assess both the strengths and the limitations of the continuous wave Doppler technique for clinical use in the noninvasive assessment of the left ventricular outflow gradient in patients with hypertrophic cardiomyopathy.

Clinical applicability of the Doppler method: limitations. It has been our experience that continuous wave Doppler echocardiography is not applicable to the estimation of subaortic gradient in all patients with hypertrophic cardiomyopathy. In an important minority of patients studied (9 of 56, 16%), it was not possible to obtain an optimal discernible signal from the left ventricular outflow tract under standard conditions in the echocardiography laboratory because of an inadequate ultrasound window or distortion of left ventricular geometry. For example, in many patients the markedly thickened anterobasal ventricular septum bulges into the left ventricular outflow tract (21,28) in such a fashion as to make an unimpeded and reliable interrogation of outflow velocity by continuous wave Doppler ultrasound difficult or virtually impossible. In addition, 19 patients were excluded from this study because a satisfactory left ventricular outflow signal could not be obtained at the time of cardiac catheterization. However, these latter patients do not represent an intrinsic limitation of the Doppler technique, but rather reflect the difficulties and restrictions inherent in positioning patients for optimal transthoracic echocardiographic studies in the catheterization laboratory.

Discrimination of the outflow tract jet from that of mitral regurgitation. Even in the absence of such limitations, it may be difficult to distinguish the high velocity systolic signal of the outflow tract (due to systolic anterior motion of the mitral valve) from the jet of mitral regurgitation that is usually present in patients with the obstructive form of hypertrophic cardiomyopathy (2). This diagnostic dilemma may exist even though the outflow and mitral regurgitation signals usually show characteristic differences in timing and contour (24,29). In patients with hypertrophic cardiomyopathy, the high velocity blood flow jets associated with mitral systolic anterior motion and with mitral regurgitation occur in particularly close anatomic proximity (2). Indeed, in those patients with the smallest left ventricular outflow tract dimension (and therefore greater magnitude of subaortic obstruction) (28), it may be particularly difficult to accurately identify and isolate the true outflow tract signal. Thus, "contamination" of the outflow tract signal by mitral regur-

gitation is a potential source of error in the quantitative estimation of subaortic gradient in many patients with hypertrophic cardiomyopathy and, in fact, accounted for discrepancies between Doppler- and catheterization-assessed aortic gradient in two of our study patients (one measured under basal conditions and one during provocative maneuvers). Consequently, in patients with hypertrophic cardiomyopathy, caution is advised in interpreting jets of more than about 5.5 m/s (estimated gradient >120 mm Hg) as representative of the left ventricular outflow tract signal because a velocity of such magnitude is most consistent with mitral regurgitation. However, it should be emphasized that while the Doppler technique significantly overestimated or underestimated the subaortic gradient in two of our study patients, the nature and magnitude of these discrepancies proved to be of little clinical importance.

In this study all continuous wave Doppler examinations were obtained with a nonimaging transducer. An alternative approach would be the use of an imaging continuous wave Doppler probe that would allow the examiner to align the Doppler beam under direct visual inspection according to anatomic landmarks and the direction of blood flow (as documented by color flow imaging). However, despite the theoretic advantages of the imaging approach, it has been our experience that the nonimaging technique yields satisfactory signals more consistently and therefore has been the method of choice in our laboratory.

Variations in waveform of the outflow tract jet. In the course of this investigation, we encountered certain variability in waveform configuration. First, in different patients outflow tract signals that correlated with (and therefore represented) the pressure gradient recorded at cardiac catheterization frequently showed differences in waveform shape and timing of peak velocity (Fig. 6). In most such patients outflow velocities increased relatively slowly in early systole but then rose abruptly and peaked in mid-systole, resulting in the concave and asymmetrically shaped waveform previously described as characteristic of patients with obstructive hypertrophic cardiomyopathy (24,25). However, in other patients the rate of increase in early systolic flow velocities was more rapid, with the peak velocity achieved earlier in mid-systole, producing a more symmetrically shaped waveform. Both these waveform shapes are distinctly different in appearance from that of patients with fixed valvular aortic stenosis, in whom peak velocity is apparent early in systole (15,16). Also, it has been our experience that in some patients with hypertrophic cardiomyopathy systolic waveforms may be recorded in addition to the "true" signal representing the spectral velocity profile of left ventricular outflow (Fig. 7). These spurious waveforms usually have a lower peak velocity occurring very late in systole and are characterized by a narrow and pointed peak; they clearly differ in appearance from those waveforms in which the peak velocity correlates with the subaortic pressure gradient measured at catheterization. Such signals probably originate

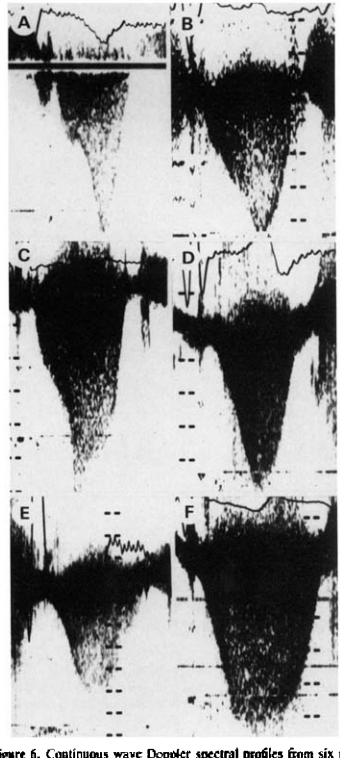


Figure 6. Continuous wave Doppler spectral profiles from six patients with obstructive hypertrophic cardiomyopathy demonstrating the spectrum of variability in waveform contour among different patients. Each of these signals was obtained at cardiac catheterization and the gradient estimated from the peak velocity correlated closely with the actual pressure gradient. **A** and **C**, The brief initial rise in velocity is relatively rapid but is followed by a more gradual increase in velocity to achieve the peak in mid-systole, resulting in an asymmetric leftward concave shape. **B**, Similar to **A** and **C** but with less pronounced asymmetry. **D**, **E** and **F**, The rate of increase in velocity is relatively slow and the waveform shows a more symmetric configuration.

from regions of the left ventricle other than the outflow tract, including portions that obliterate at end-systole (29).

Awareness of the variability in waveform configuration in patients with hypertrophic cardiomyopathy is obviously

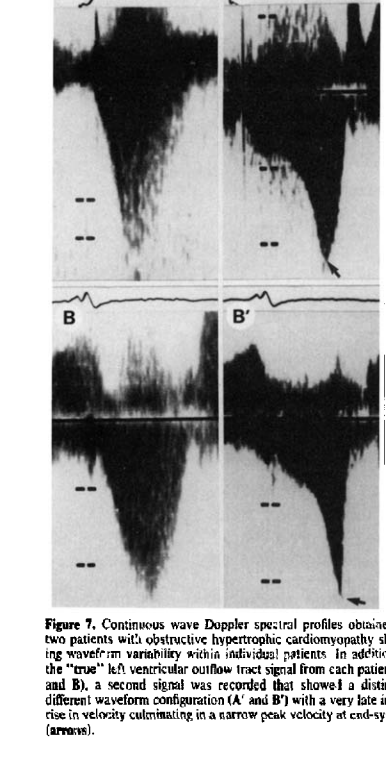


Figure 7. Continuous wave Doppler spectral profiles obtained in two patients with obstructive hypertrophic cardiomyopathy showing waveform variability within individual patients. In addition to the "true" left ventricular outflow tract signal from each patient (**A** and **B**), a second signal was recorded that showed a distinctly different waveform configuration (**A'** and **B'**) with a very late initial rise in velocity culminating in a narrow peak velocity at end-systole (**arrows**).

critical for proper performance of the continuous wave Doppler examination because of its potential impact on estimation of outflow tract gradient. Furthermore, although it was not the purpose of our study to relate the Doppler findings to pathophysiologic mechanisms, the variability in systolic waveform shape encountered could conceivably reflect different patterns of systolic anterior motion (7) and mechanisms of dynamic subaortic obstruction in hypertrophic cardiomyopathy.

Conclusions. Simultaneous echocardiographic and hemodynamic studies in patients with hypertrophic cardiomyopathy showed that Doppler echocardiography is a useful means of noninvasively assessing the magnitude of left ventricular outflow obstruction under basal conditions or with provocative maneuvers. Consequently, the left ventricular outflow tract gradient can be assessed serially and noninvasively in patients with hypertrophic cardiomyopathy in an ambulatory setting in lieu of cardiac catheterization. However, a variety of factors may affect utilization of the Doppler technique in this regard, including distorted left ventricular geometry, potential for contamination of the left ventricular outflow tract signal with that of mitral regurgitation and variability in the contour of the outflow tract waveform among patients or in individual patients. These factors should be considered during the performance and interpretation of Doppler studies in patients with hypertrophic cardiomyopathy and emphasize the importance of experience on the part of the operator and interpreter in achieving meaningful results.

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