Assessment of Abnormal Systolic Intraventricular Flow Patterns by Doppler Imaging in Patients With Left Ventricular Dyssynergy

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Few data exist regarding the consequences of abnormalities of segmental contraction on intraventricular flow patterns. The development of color Doppler flow imaging has now permitted the visualization of intraventricular blood flow patterns. Therefore, we performed Doppler flow mapping in 41 patients (12 with normal left ventricular contraction, eight with hypokinesis or akinesis, and 21 with dyskinesis) and compared these findings with left ventriculography. Systolic blood flow by Doppler mapping in subjects with normal ventricular contraction was characterized primarily by flow through the left ventricular outflow tract and into the aorta. In patients with dyskinesis, paradoxical systolic flow toward the abnormal segment was present, and persisted for at least 50% of systole in 18 of 21 patients. Mean duration of paradoxical flow in dyskinetic patients was 77% of systole. Paradoxical flow was also observed in two of five patients with akinesis but in no patients with hypokinesis. A good correlation was observed between the duration of paradoxical systolic flow and indexes of regional wall motion (radian shortening of the involved myocardium) and global ejection fraction derived from cineangiography. The development of color Doppler imaging has made possible the evaluation of dynamic blood flow patterns within the LV cavity during systole and diastole. Thereby, flow mapping provides the potential to assess disturbances of intraventricular blood flow induced by LV dyskinesis, specifically the presence of paradoxical blood flow into dyskinetic segments during systole.

Acute myocardial infarction frequently results in the production of regional dyskinesis or frank aneurysm formation. Nevertheless, the clinical and hemodynamic consequences of left ventricular (LV) dyskinesis remain incompletely defined. Although volume expansion in systole would seem to be a fundamental criteria for dyskinesis, some studies have demonstrated actual systolic expansion of the noncontractile segment, whereas others have not.\(^1\)\(^2\) Accordingly, the hemodynamic effects of LV dyskinesis remain undefined, can vary from patient to patient, and can be an important determinant of the results of cardiac surgical procedures including coronary bypass grafting and aneurysmectomy.

Recently, color Doppler flow imaging techniques have been developed that enable noninvasive visualization of blood flow. Color Doppler flow imaging has been performed to determine the prevalence, nature, and extent of intraventricular flow disturbances associated with dyskinesis, and the relation of these flow disturbances to the magnitude of contractile abnormality and hemodynamic dysfunction as documented by cardiac catheterization and cineventriculography.

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Methods

The population for this study consisted of 41 patients who underwent cardiac catheterization for known or suspected coronary artery disease. The group comprised 26 men and 15 women in the age range of 33–79 years (mean, 53 years). Patients were selected for this study on the basis of qualitative and quantitative analysis of cineventriculography. The study population included 12 patients with normal LV wall motion on cineventriculography (all of whom had normal coronary arteriograms) and 21 patients whose ventriculograms demonstrated segmental dyskinesis. All of the latter had sustained previous myocardial infarction or manifested unstable angina. A third cohort of eight patients was identified who manifested either hypokinetic or akinetic LV segments (four with and four without previous myocardial infarction).

LV aneurysm can be defined as a localized discrete noncontractile pocket forming a definite bulge in the ventricular contour and resulting in both systolic and diastolic deformity. Based on this anatomic definition, 14 of the 21 patients with dyskinesis exhibited LV aneurysm. In patients with dyskinesis, the abnormal segment involved the apical region, including anteroapical, inferoapical, or apicolateral segments. No cases of inferobasal or posterior dyskinesis were encountered in this study. Inferobasal hypokinesis or akinesis was present in nine patients, but in each case, it coexisted with an akinetic or dyskinetic anterior or apical region.

Catheterization of the left side of the heart was performed retrograde through the femoral artery by using a fluid-filled 8F pigtail catheter, Cordis Corporation, Miami, Florida. Biplane (30° right anterior oblique and 60° left anterior oblique) LV cineangiography was performed at 60 frames per second during the injection of 36–45 ml meglumine diatrizoate. Ventriculography was technically adequate if the chamber was well opacified and no premature ventricular contractions occurred. LV silhouettes were digitized by using a hand-held cursor. LV volume was computed by using the area-length method with a regression equation for single-plane right anterior oblique ventriculograms and a correction factor derived from grids positioned at the center of the ventricle. The LV ejection fraction was calculated as end-diastolic volume minus end-systolic volume divided by end-diastolic volume.

Regional wall motion analysis was performed using an algorithm that divided the end-diastolic and end-systolic silhouettes into 24 radians by using a fixed centroid as reference (Figure 1). Systolic contraction was expressed as diastolic length minus systolic length divided by diastolic length for each of the 24 radians. Radians were numbered from 1 to 24, beginning at the posterior and ending at the anterior aortic anulus, with the apex falling between radians 12 and 13. Using this scheme, the apical segments of the left ventricle were identified as those falling between radians 9 and 16.

A variety of measurements were performed from this wall motion analysis. The number of segments exhibiting dyskinetic motion was determined, as was the maximal abnormality in shortening manifested for any radian. As an index of global function, the mean percentage shortening of all 24 radians was calculated. Because dyskinesis, when present, involved the anteroapical region in nearly all patients in this study, as an index of abnormal regional wall motion, the mean shortening of radians 9–16 was computed (Figure 1, panel B). Finally, to allow for differences in location of contractile abnormalities (anteroapical versus inferoapical), and to attempt to consider significant adjacent areas of akinesis or severe hypokinesis, the eight adjacent segments that encompassed the region manifesting the maximal contractile abnormality were identified, and the mean shortening of the radians for this area was calculated.

Due to the complex motion of the left ventricle during contraction, it is well recognized that quantitative evaluation of regional dyssynergy, whether using fixed or floating reference points, often yields data inconsistent with visual assessment. Therefore, in addition to quantitative analysis, LV cineangiograms were qualitatively assessed by one investigator (D.B.) who was blinded to the results of the echocardiographic examination. Patients were then assigned to one of five categories based on the degree of contractile failure and extent of myocardium involved in the primary wall motion disturbance present. This classification was subsequently used to assign patients to groups based on LV wall motion. In this analysis, twelve patients were designated as normal; three were designated as hypokinetic, five as akinetic, seven as mildly dyskinetic, and 14 as severely dyskinetic. Importantly, 19 of 21 patients classified as dyskinetic by visual examination demonstrated paradoxical systolic expansion by radian measurement.

The echocardiographic examination was performed on the day of or the day after catheterization in most instances. Patients were excluded from the study when significant clinical events occurred in the interval between angiography and the echo examination. The
echocardiographic examination consisted of M-mode and two-dimensional imaging, color Doppler flow mapping, and multigated color M-mode recordings. The examinations were performed using a commercially available instrument (Hewlett-Packard Corp., Andover, Massachusetts). All patients were examined with a 2.5-mHz transducer, whereas a 3.5-mHz transducer (Hewlett-Packard) was also used in those patients in whom adequate images were obtainable with this instrumentation. Recordings were performed in parasternal long-axis and short-axis views, and apical four-chamber and two-chamber views, with the patients in the left lateral decubitus position. The instrument was capable of providing mean estimates of blood flow velocity for a variable (15–60°) sector of the heart. The frame rate was determined by the sector angle, depth, and number of interrogating bursts per scan line at which the instrument operated. For the purposes of this study, we used eight bursts per scan line at the narrowest angle capable of visualizing flow in the LV apex with controls adjusted to enhance the signals emanating from low-velocity targets. By convention, velocity values were color encoded in red when moving toward the transducer, and blue when moving away. Velocity gradations were expressed in sixteen shades of red and blue respectively, whereas a variance algorithm was used to add green to the signal in the presence of turbulence.

In this study, the echocardiographic color flow images analyzed were obtained from the apical cardiac window in four- and two-chamber views. These views enabled clear visualization of flow patterns within the LV apex, the site of abnormal flow in the patients in this study. The transducer was angled until optimal anatomic and flow signals were recorded, and color gains were adjusted to the maximal level possible without the introduction of background color to the cardiac chambers or structure.

Color flow images of the left ventricle were carefully examined for the presence of abnormal intracavitary flow signals toward dysynergic segments during systole. Subsequently, multigated color M-mode recordings were obtained with the cursor positioned within such abnormal flow streams, or in the area of maximal transmitral flow in the absence of abnormal flow patterns. Two measurements were made from these recordings. First, the color M-mode record was used to calculate the percentage of systole during which apically directed blood flow was present. Specifically, the long axis of the left ventricle on color M-mode was divided into equivalent basal, mid, and apical thirds. The period of abnormal flow was measured as the time from the onset of the QRS complex to the disappearance of color signals directed toward the apex from the mid or apical left ventricle. The systolic ejection time was measured from the period between closure and opening of the mitral valve leaflets from a standard parasternal M-mode record. Both intervals were corrected for heart rate with division by the square root of the RR interval, and the duration of paradoxical flow was expressed as a percentage of systole.

As a second index of paradoxical systolic flow, the area of abnormal blood flow directed toward the apex during systole was measured from color flow images. The outer border of the apically directed flow jet was outlined by a track-ball system, and the area contained within this border was determined. Measurements were made from the echocardiographic frame that most closely coincided with the midpoint of systole. The midpoint of systole was chosen for evaluation because nearly all patients with dyskinesia manifested abnormal flow into the second half of systole, and no normal patients demonstrated apically directed flow for this duration. Multiple cardiac cycles were measured, and the maximal value for area was used in analysis.

Statistical analysis of the data was performed according to standard methods. The prevalence of abnormal flow signals in patients with dyskinesia was compared with that observed in normal subjects and in patients with hypokinesis or akinesia by analysis of variance. Subsequently, standard linear regression analysis was used to determine whether a correlation existed between either the duration or area of abnormal color Doppler flow and measurements of regional and global left ventricular function, and other clinical and hemodynamic parameters.

Results

The echocardiographic studies available for each patient were technically adequate to permit the analysis performed in this investigation. Color Doppler flow images performed with both 2.5- and 3.5-mHz transducers were available for analysis in all but three patients. Comparison of the images obtained with the two transducer frequencies revealed that the 3.5-mHz transducer registered larger areas of paradoxical mid-systolic flow than did the 2.5-mHz transducer (mean, 3.9 cm² vs. 2.5 cm²; p<0.01). In some patients, however, signal attenuation resulted in technically inadequate images with the 3.5-mHz transducer. Therefore, the measurements reported here are those obtained from the instrument that yielded the highest quality flow images because this is how the instruments are used clinically. At the depth and sector settings required to perform flow mapping in this study, the number of frames per systolic cycle was in the range of 5–8 (average, 6).

Flow Patterns in Normal Patients

In the 12 patients with normal LV wall motion, systolic intracavitary flow patterns in early systole were characterized by a red-encoded signal directed toward the apex from the posterolateral aspect of the ventricle. These red-encoded early systolic flow signals were confined to the basal and middle one third of the ventricle and had a mean duration (measured at midventricular level) that was 25% (range, 13–37%) of systole. This anteropapically directed signal in normal subjects was best identified in the apical
two-chamber image in the region immediately above the mitral ring. This early systolic signal was quickly replaced by normal ejection flow and encoded in blue within the body, apex, and LV outflow tract. None of the normal patients possessed detectable, paradoxical flow signals at midsystole. Figure 2 represents a multigated color M-mode image and a color Doppler frame taken at midsystole from a subject with normal LV wall motion. The images were obtained from the LV apex, and the cursor for the color M-mode image was directed through the maximal transmirtal flow signal.

Flow Patterns in Dyskinetic Patients

Systolic intracavitary flow in the 21 dyskinetic patients showed pronounced qualitative and quantitative differences from that seen in patients with normal LV wall motion. In dyskinetic patients, there was a prolonged persistence of red-encoded signals proceeding from the posterobasal left ventricular into midventricular and apical regions during systole (Figure 3). These signals could not be related to the timing of dyskinesis, had the appearance of a flow stream passing from the basally contracting ventricle into regions of apical dyskinesis, and coexisted with the development of blue-encoded ejection flow at midventricular and outflow tract levels. Thirteen of the 14 patients with severe dyskinesis and five of the seven patients with mild dyskinesis demonstrated this apically directed paradoxical flow extending past midsystole. The mean paradoxical flow duration was 77.4±21.7% (range, 24–100%) of systole. Although the mean paradoxical flow area at midsystole and mean duration of this flow signal was greater in patients with severe dyskinesis (n=14) than in patients with mild dyskinesis (n=7) (6.7 vs. 5.9 cm² and 81.1% vs. 70.7%, respectively), these differences did not reach statistical significance. For all 21 patients, the mean paradoxical flow area at midsystole measured 4.4±3.6 cm² (range, 0–10.9 cm²).

Ten patients with dyskinesis had experienced recent myocardial infarction (within the previous 6 weeks), and nine patients experienced remote infarctions. The remaining two patients had syndromes of unstable angina without documentation of previous infarction. Comparing the recent and remote infarct patients, there was no difference in mean global ejection fraction or mean shortening of apical radials 9–16. Patients with recent infarct, however, had some evidence supporting the presence of more extensive regional wall motion dysfunction because these patients had a greater number of dyskinetic segments (mean, 5.3 vs. 2.3, p<0.01), more pronounced systolic bulging in the worst radian (−15% vs. −6%, p<0.01), and more abnormal mean radian shortening of the eight radians encompassing the worst ventricular segment (mean, −3.7% vs. +5.1%, p<0.01) than the patients with remote infarct. Despite this, patients with recent infarct had a shorter paradoxical flow duration (mean, 69±24% vs. 90±7%, p<0.05), and midsystolic paradoxical flow areas were slightly smaller than in the patients with remote infarction (3.5±4.0 vs. 6.5±2.3 cm², p<0.1).

Fourteen of the 21 dyskinetic patients satisfied the angiographic definition of LV aneurysm. There was no significant difference in mean paradoxical flow duration or area between those who did and those who did not satisfy this definition.

Flow Patterns in Patients With Hypokinesis and Akinesis

Systolic flow patterns in hypokinetic patients resembled the patterns of normal patients with a short early systolic flow duration (35±3%) and no paradoxical flow detectable at midsystole. Mean flow duration in akinetic patients occupied an intermediate value between normal subjects and patients with dyskinesis (52.8±27.7%); however, this was not significantly different from that of the dyskinetic patients. Two patients demonstrated significant paradoxical systolic flow past midsystole. Similarly, measurement of paradoxical midsystolic flow areas in the akinetic patients yielded values that were not statistically different from those seen in dyskinetic patients (5.1±4.96 cm² vs. 4.4±3.6 cm², respectively).

Velocity and Variability of Paradoxical Systolic Flow

The paradoxical systolic flow observed in this study was invariably of low velocity. In no patient did paradoxical flow exceed the scanning Nyquist limit, which was 49 cm/sec in five patients and 36 cm/sec in the remaining patients. Pulsed Doppler recordings within areas of paradoxical flow were performed in six patients and revealed maximal velocities in the range of 10–35 cm/sec (mean, 22 cm/sec).

To evaluate the interobserver variability in assessing these flow phenomena, the echocardiographic studies of 10 patients were reviewed by two investigators. Paradoxical systolic flow was identified in seven patients and excluded in three patients by both observers. Differences in the calculated percentage of systole during which apically directed flow occurred was in the range of 0–8% for paired observation, with a mean of 4%. Differences in the area of paradoxical systolic flow in the seven patients in whom this was observed was in the range of 0.1–0.8 cm² between observers, with a mean of 0.42 cm². Correlation coefficients for measurements of both observers were r equaling 0.97 and 0.94 for duration and area, respectively.

Correlation of Color Flow Indexes With Regional Left Ventricular Wall Motion

The duration of paradoxical flow correlated well with the mean shortening of apical radials 9–16 (r=0.77; SEE, 19.2), and the mean shortening of the eight radians encompassing the maximally abnormal segment (r=0.74; SEE, 20.3) (Figure 4). Comparison of measurements of paradoxical flow area with both of these regional wall motion indexes correlated less closely, however, and yielded identical coefficients (r=0.57; SEE, 3.1). A correlation
FIGURE 2. Illustrations of a multigated color M-mode recording (upper panel) and a midsystolic color Doppler flow image from a patient with normal left ventricular wall motion (lower panel). Note that apically directed (red) color flow ceases soon after onset of the QRS complex (upper panel) and that at midsystole, all flow is directed toward the aortic valve (lower panel).
FIGURE 3. Illustrations of a multigated color M-mode recording (upper panel) and a midsystolic Doppler mapping frame from a patient with apical dyskinesis (lower panel). Note the pronounced persistence of apically directed (red) paradoxical flow during systole (upper panel) and that at midsystole, a large area of paradoxical flow disturbance is present in the apical and middle thirds of the ventricle (lower panel).
was present between color flow duration and the maximal wall motion abnormality present in the worst radian ($r=0.70$; SEE, 2.7).

**Correlation of Color Flow Indexes With Global Left Ventricular Function**

A good correlation existed between global LV ejection fraction and the duration of paradoxical color flow ($r=0.79$; SEE, 18.4) (Figure 5). Similar results were obtained from linear regression analysis of flow duration and the mean shortening of all 24 radians ($r=0.80$; SEE, 18.2). A general correlation was found to exist between ejection fraction and the area of paradoxical flow at midsystole ($r=0.68$; SEE, 2.8) and also with mean shortening of all radians ($r=0.65$; SEE, 2.9). No correlations were found between either of the color flow parameters and LV end-diastolic pressure, end-diastolic volume index, or the number of dyskinetic radians present. LV thrombus was present in only two patients in this study. One patient had suffered a recent myocardial infarction, and the other had suffered a remote event.

**Discussion**

This study was performed to define the characteristics of intracavitary LV flow patterns by using color Doppler flow imaging in patients with abnormalities of LV wall motion. Specifically, we sought to document the occurrence and assess the prevalence of paradoxical systolic flow into regions of LV dyskinesis or aneurysm formation. Further, we examined the relation of these abnormal intraventricular flow patterns to the magnitude of regional and global LV dysfunction as delineated by cineventriculography. The results of our study establish that paradoxical systolic flow into dyskinetic LV segments occurs frequently and can be correlated with the severity of regional and global contractile abnormalities in patients with coronary disease.

In patients with normal LV contraction, detailed examination of color Doppler flow images revealed a red-encoded intraventricular signal indicative of blood flow toward the apex after the onset of systole. These Doppler signals were limited to the basal
portion of the chamber and were confined to early systole, persisting only briefly (less than 50% of systole) after mitral valve closure and onset of the QRS complex. We believe that these signals result from the final phases of transmitial flow after atrial contraction and the inward motion imparted to the regional blood pool by contraction of the posterobasal and posterolateral walls. These normal signals could be readily distinguished from the paradoxical flow signals found in patients with dyskinesis. Paradoxical signals were found in the middle and apical thirds of the left ventricle and usually persisted for greater than 50% of systole. These data indicate the importance of the temporal analysis of intraventricular flow events and demonstrate the usefulness of color Doppler M-mode for this application. In fact, the duration of paradoxical intracavitary flow signals was the characteristic of greatest value not only in the identification of this abnormality but also in the quantitative correlation with LV dyssynergy.

We have documented that paradoxical systolic color flow signals can be demonstrated in the majority of patients with dyskinetic LV wall motion demonstrated by cineangiography. Of 21 patients classified as having dyskinetic segments by qualitative visual assessment of cineventriculography, 18 were found to have evidence of significant paradoxical systolic flow by Doppler mapping. This incidence (86%) is slightly greater than that described in a preliminary report by Omoto and colleagues who observed this flow pattern in 12 of 21 patients (57%) with LV aneurysms. A number of technical factors influence the ability to demonstrate these flow signals, including transducer frequency, color flow gain adjustment, sector angle, and depth of field, as well as patient variables such as chest wall thickness. Importantly, we found that imaging with the higher frequency transducer (3.5 mHz) was especially helpful in delineating this flow signal.

There were three patients (identified by cineangiography as having dyskinetic segments) in whom we were unable to document paradoxical color flow. Two of these three patients were classified in the mild dyskinesis group. Although the third patient was judged to have severe dyskinesis, the actual area of segmental dysfunction was small, involving only two apical radii by quantitative assessment. Thus, paradoxical systolic flow was absent in patients with relatively mild dyskinesis.

We have documented a good correlation between parameters of abnormal intraventricular flow by Doppler and the severity of regional wall motion disturbances, assessed by quantitative methods of wall motion analysis. The duration of paradoxical color flow was found to have a direct correlation with the mean shortening of radii in the area of dyskinesis as well as with the shortening of the radii from the most impaired segment. Measurement of color flow areas at mid systole correlated less closely with contractile abnormalities. This lesser correlation of paradoxical systolic flow area with indexes of regional dysfunction might have been due to inaccuracies in the assessment of abnormal flow imposed by the reduced frame rate of two-dimensional imaging.

Good correlations were also observed between color flow measurements and indexes of global LV function (ejection fraction and mean global radian shortening). Only coronary disease patients were included in this study; thus it is likely that these correlations between paradoxical systolic flow and global LV function are related to the magnitude of regional dyssynergy because a declining ejection fraction is itself a manifestation of increasingly severe regional wall motion abnormalities in this group of patients. Further delineation of the relation between poor LV function and abnormal patterns of color flow requires study of a group of patients with diminished global LV function without segmental dyskinesis.

We propose two explanations for these abnormal intracavitary flow patterns. The common denominator for both conditions involves contractile failure of the myocardium and the resultant propulsion of blood toward the dyskinetic segment. In one construct, the ventricular wall comprising the dyskinetic segment is poorly demarcated and noncompliant, and true systolic volume expansion does not occur. In this instance, flow occurs through the abnormal region at a slowed radial velocity and assumes a swirling nature within the dyskinetic segment. In the second circumstance, the dyskinetic ventricular myocardium is localized and capable of distention, and thus can accommodate a true increase in systolic volume. It is likely that both phenomena exist in varying degrees in many of the patients who exhibit abnormal intraventricular flow patterns.

The relative contributions of low velocity radial swirling and direct systolic expansion to the development of the abnormal intraventricular flow observed in this study remains uncertain. Some indirect evidence, however, might relate to this question. The duration and area of paradoxical systolic flow was directly related to the severity of regional and global LV dysfunction, a finding compatible with the more deleterious effect of systolic expansion of the dyskinetic segment. Further, radial swirling would be expected to produce flow both into and out of the dyskinetic segment, but our Doppler mapping examinations failed to convincingly demonstrate such bidirectional flow signals. Conversely, the finding of significant paradoxical systolic flow signals in two patients judged to have akinesis on cineangiography suggests that localized swirling played an important role in producing abnormal cavitary patterns in some patients. Also, the duration and area of paradoxical systolic flow was greater in dyskinetic patients with remote infarct than in the patients with recent infarct. This finding is consistent with the expectation that if due principally to systolic expansion, the degree of paradoxical systolic flow should be smaller in the patients with remote infarct due to the presence of less dyskinesis and the poorly distensible
nature of mature scar tissue. Further studies are required to provide a definitive resolution of this issue.

True systolic volume expansion of a dyskinetic area can impose a more severe level of ventricular dysfunction than radial swirling. Yiannikas et al.⁶ using radionuclide-gated blood-pool–scanning techniques, have documented a strong correlation between the presence of true paradoxical wall motion and improvement in ejection fraction in patients after aneurysmectomy. Similarly, experimental observations by Tyson et al.⁷ in a dog model linked depression of ventricular function to the presence of an expansile, truly paradoxical aneurysm. Nevertheless, the differentiation of true systolic expansion of dyskinetic segments from centrifugal swirling might not be of critical importance regarding the hemodynamic consequences of paradoxical systolic flow. The demonstration of intraventricular flow velocities toward a dyskinetic segment invariably indicates that some of the energy imparted to red cells by myocardial contraction is being diverted from ejection of blood into the aorta. Thus, differences in the hemodynamic effects of systolic expansion and swirling are likely quantitative rather than qualitative.

Controversy continues regarding the indications for surgical aneurysmectomy, and a variety of imaging techniques and analytical algorithms have been used to predict postoperative survival and functional capacity.⁸⁻¹¹ Whereas there is some agreement that recurrent thromboembolism and symptomatic ventricular tachycardia are indications for aneurysm resection, the response of congestive failure to aneurysmectomy is less predictable.¹¹ This is related to the difficulty in distinguishing those patients in whom congestive failure results principally from global impairment of LV function from those patients in whom failure is due to a regional wall motion disturbance. Color Doppler flow imaging allows us to visualize intraventricular flow patterns in a fundamentally new manner, and by enabling the identification and assessment of abnormal flow events might aid in the selection of patients with aneurysms for surgical resection.

In this study, we have not found paradoxical systolic flow to be strongly related to the presence of LV thrombus. This entity was clearly identified in only two of our patients, each from the categories of patients with recent and remote infarction. Delemarre et al.¹² studying patients soon after myocardial infarction with standard pulsed Doppler techniques, described a “circular” Doppler flow pattern as a risk factor for subsequent thrombus formation. It is probable that this pulsed Doppler signal represents the same phenomenon we are describing with color mapping. The relation between ventricular thrombus and paradoxical systolic flow must await further longitudinal studies after acute myocardial infarction. The presence of segmental low-velocity swirling blood flow seems an ideal circumstance for the formation of ventricular thrombus.

Several factors relating to the imaging technology and study design were capable of influencing the results observed in this study. With respect to the ultrasound examination, the lack of a standardized method of gain adjustment is a potential limitation. We performed the examinations on one machine and adjusted color flow gains to the maximal level possible without the production of background artifact. Although this technique has become standardized and is used by most echocardiographers, it does introduce a degree of subjectivity into the examination. In this study, the correlations of ventriculographic wall motion indexes were better with paradoxical flow duration than with flow area measurements. This limitation of the use of flow area relates to the temporal resolution of flow imaging in the selection of the midsystolic frame for planimetry. We believe that measurement of the duration of paradoxical flow is a more accurate assessment of the severity of the flow disturbance present. Our echo examinations were not conducted simultaneously with ventriculography, and although care was taken to ensure that intercurrent events had not occurred, we cannot absolutely control for the influence of different hemodynamic loading and ambient ischemic conditions between the two examinations.

For our quantitative assessment of wall motion, we chose a fixed-centroid algorithm.¹³ In most patients with dyskinesis, the fixed-centroid model more reliably depicts regional wall motion than does a floating-centroid model, however pronounced translation movement of the ventricle reduces the reliability of the fixed method. Translational motion did not appear to be a major factor in these study patients. We have examined a modest patient population, and additional individuals, particularly those with poor global ventricular function without significant segmental dyskinesis, require future investigation.

Color Doppler flow imaging has enabled us to examine the patterns of intraventricular blood flow. Hopefully, this imaging technique will contribute substantially to our understanding of the flow disturbance present in patients with segmental LV wall motion abnormalities. Paradoxical systolic flow appears to be an additional and easily recordable descriptor of the severity of contractile dysfunction produced by myocardial infarction. Paradoxical systolic flow might have implications regarding the use of pharmacological agents such as angiotensin converting enzyme inhibitors after infarction. The presence of systolic flow directed toward the infarct zone might identify those patients with poorly controlled congestive failure in whom aneurysmectomy can be beneficial.⁸⁻¹¹ Finally, paradoxical systolic flow might represent a precursor of thrombus formation.

References


**KEY WORDS** • echocardiography • left ventricular aneurysm • left ventricular function
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