Diagnosis of ventricular septal rupture after myocardial infarction: value of colour flow mapping

JOHN SMYLLIE,* KEITH DAWKINS, NEVILLE CONWAY,
GEORGE R SUTHERLAND*

From the Wessex Cardiothoracic Unit, Southampton General Hospital, Southampton

SUMMARY Twenty patients with ventricular septal rupture after myocardial infarction were investigated by cross sectional echocardiography with integrated pulsed and continuous wave Doppler and colour flow mapping. Confirmatory cardiac catheterisation was performed in 12 patients. Eighteen patients had surgical repair with inspection of the defect. Six patients in whom recurrent ventricular septal rupture developed were also investigated by Doppler echocardiography and colour flow mapping. Cross sectional echocardiography correctly predicted the infarct territory in all cases but visualised the septal rupture in only seven (35%). Pulsed and continuous wave Doppler detected a disturbance of right ventricular systolic flow that was diagnostic of a ventricular septal rupture in 19 (95%), but this only accurately predicted the site in 14 (70%). Colour flow mapping studies showed a mosaic jet traversing the interventricular septum in all 20 cases, and this accurately predicted the site of rupture. In addition colour flow mapping defined three sites of ventricular septal rupture: apical, posterior, and anterior trabecular. Five of the six patients with recurrent rupture were correctly diagnosed by pulsed and continuous wave Doppler and all six were diagnosed by colour flow mapping.

Cross sectional echocardiography with colour flow mapping is a highly sensitive and rapid technique for the assessment of postinfarction ventricular septal rupture before and after operation. It was more informative about the site of the rupture than pulsed and continuous wave Doppler echocardiography.

Ventricular septal rupture occurs in about 2% of patients with acute myocardial infarction.1 Conservative treatment is associated with a high mortality2 and therefore a rapid and accurate diagnosis is essential so that early surgical repair can be performed.3-6 Because it is often difficult to distinguish between post-infarction ventricular septal rupture and acute mitral regurgitation on clinical grounds,7 a rapid and reliable non-invasive diagnostic technique such as cardiac ultrasound is needed. Cross sectional echocardiography alone, however, may fail to visualise the defect in a considerable number of patients.8-12 The addition of pulsed Doppler echocardiography increased the overall diagnostic accuracy but it was less sensitive in predicting the rupture site.8-12 Colour flow mapping has been used in the diagnosis of post-infarction ventricular septal rupture,13 but there are no published reports of studies to evaluate the potential benefits of colour flow mapping in the diagnosis and management of this condition.

We studied the diagnostic accuracy of all these ultrasound techniques in a group of patients with post-infarction ventricular septal rupture.

Patients and methods

Patients Between December 1986 and November 1987 all 20 patients referred to the Wessex Regional Cardiothoracic Unit with suspected post-infarction ventricular septal rupture were investigated by cross sectional echocardiography with integrated pulsed and continuous wave Doppler and colour flow mapping. Patients with isolated acute mitral regurgitation were excluded. The diagnosis was confirmed at operation (18 patients) or necropsy (two patients).

There were 13 men and seven women (aged 52–78
Diagnosis of ventricular septal rupture after myocardial infarction: value of colour flow mapping

Table 1 Clinical and surgical data of 20 patients with post-infarct ventricular septal rupture (VSR)

<table>
<thead>
<tr>
<th>General:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age</td>
<td>68 (range 52–78) yr</td>
</tr>
<tr>
<td>Male:Female</td>
<td>1:9:1</td>
</tr>
<tr>
<td>Anterior infarction</td>
<td>11 (55%)</td>
</tr>
<tr>
<td>Inferior infarction</td>
<td>9 (45%)</td>
</tr>
<tr>
<td>Mean time from infarct to VSR</td>
<td>3 (range 0–16) days</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnostic techniques:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>CS echo, Doppler, and CFM</td>
<td>20</td>
</tr>
<tr>
<td>Cardiac catheterisation</td>
<td>12</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Surgical:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>VSR closure alone</td>
<td>12</td>
</tr>
<tr>
<td>With associated LV aneurysm resection</td>
<td>2</td>
</tr>
<tr>
<td>With associated coronary grafts</td>
<td>6</td>
</tr>
<tr>
<td>No operation</td>
<td>2</td>
</tr>
<tr>
<td>Early death</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Recurrent VSRs</td>
<td>6 (33%)</td>
</tr>
<tr>
<td>Recurrent VSRs requiring reoperation</td>
<td>4 (22%)</td>
</tr>
</tbody>
</table>

CS echo, cross sectional echocardiography; CFM, colour flow mapping; LV, left ventricular.

(mean 68) years) (table 1). Eleven had sustained an anterior myocardial infarct and nine an inferior myocardial infarct as judged by a 12 lead electrocardiogram. Eighteen patients underwent closure of the ventricular septal rupture, two with associated left ventricular aneurysm resection and six with additional coronary artery bypass grafts. Two patients did not proceed to operation because of coexisting medical conditions; both died within days of diagnosis. In six patients (33%) recurrent ventricular septal rupture developed; these were confirmed by cardiac catheterisation, Doppler echocardiography, or both and only four (22%) required reoperation on clinical criteria.

METHODOLOGY

Echocardiography

All echocardiographic studies were performed at the bedside immediately the patient arrived on the unit and were completed within 25 minutes. Cross sectional echocardiography with integrated pulsed and continuous wave Doppler and colour flow mapping studies were performed with a Toshiba SSH 65A. Standard parasternal, apical, and subcostal views were examined in all patients. When the ventricular septal rupture was visualised the pulsed Doppler sample was placed at the right ventricular apex and moved down the interventricular septum in the apical four chamber view until systolic turbulence was detected. This latter manoeuvre was carried out in an attempt to define the area(s) of trans-myocardial flow and thus predict the defect site. We performed the colour flow mapping studies using the velocity mode with the appropriate filter and gain settings after the initial imaging and Doppler studies. The interventricular septum was then imaged, as before, until a mosaic jet signifying high velocity turbulent flow was identified traversing the septum. The position of the defect and the number of jets arising from the same defect or multiple defects were then noted. Mitral regurgitation if present was identified by continuous wave Doppler and colour flow mapping.

The same protocol was applied to postoperative patients with a suspected recurrence of a ventricular septal rupture.

Cardiac catheterisation

Left ventricular cineangiography and selective coronary arteriography were performed in 12 patients, and three had additional right heart catheterisation. Four of the six patients with recurrent defects had both left and right heart catheterisation.

RESULTS

CROSS SECTIONAL ECHOCARDIOGRAPHY

Wall motion

Cross sectional echocardiography correctly identified the infarct territory in all 20 patients by identifying areas of abnormal wall thinning and wall motion, and this correlated with the electrocardiogram and the left ventricular cineangiogram. Six of the 11 anterior myocardial infarcts had obvious dyskinetic segments with a characteristic bulge of the apical septum protruding into the right ventricle.

Visualisation of the ventricular septal rupture

The ventricular septal rupture was directly visualised in seven patients (35%) (table 2). In the remaining 13 patients complete interruption of the interventricular septum could not be demonstrated. In several patients there were localised areas of abnormal septal motion or septal thinning which, although suspicious, were not considered diagnostic of post-infarction ventricular septal rupture.

PULSED AND CONTINUOUS WAVE DOPPLER

Abnormal right ventricular flow disturbance

Pulsed Doppler echocardiography detected an abnormal right ventricular systolic flow, characteristic of a ventricular septal defect, in 19 patients (95%)
Table 2  Diagnostic accuracy of each echocardiographic technique in 20 patients with post-infarction ventricular septal rupture

<table>
<thead>
<tr>
<th></th>
<th>CS echo alone</th>
<th>PW and CW Doppler</th>
<th>Colour flow mapping</th>
</tr>
</thead>
<tbody>
<tr>
<td>Correct diagnosis</td>
<td>7 (35%)</td>
<td>19 (95%)</td>
<td>20 (100%)</td>
</tr>
<tr>
<td>Accurate site prediction</td>
<td>7 (35%)</td>
<td>14 (70%)</td>
<td>20 (100%)</td>
</tr>
</tbody>
</table>

CS echo, cross sectional echocardiography; PW and CW, pulsed and continuous wave Doppler echocardiography.

Systolic and diastolic shunting patterns

Systolic left to right ventricular shunting was shown in 19 patients by continuous wave Doppler. This typically resulted in a high velocity, systolic jet directed towards the transducer (fig 1). In six patients this systolic flow disturbance was of high velocity in all the cases studied and resulted in an aliased pulsed Doppler signal. Continuous wave Doppler also detected a high velocity jet in 95%, with peak velocities ranging from 1·9 to 4·0 m/s. Pulsed and continuous wave Doppler were non-diagnostic in an obese patient who was very ill.

In 14 patients (70%) pulsed Doppler correctly estimated the position of the ventricular septal rupture found by inspection of the defect at operation or necropsy (table 2). In the remaining six patients it did not. One patient had no detectable abnormal Doppler signal diagnostic of a post-infarction ventricular septal defect (see above). The other five patients were thought to have apical defects on the basis of the Doppler signal alone; however, at surgical inspection two had posterior and three had anterior trabecular ventricular septal rupture.

*Fig 1*  A continuous wave Doppler echocardiogram recording from a patient with a post-infarction ventricular septal rupture showing high velocity (3·8 m/s) systolic left to right shunting. In diastole there was almost pandiastolic left to right shunting (thick arrows) with a characteristic rise in velocity towards the end of diastole. In early diastole there was a short period of flow reversal (thin arrows) which is of low velocity and indicates right to left shunting (see text).
patients, however, a high velocity systolic waveform directed away from the transducer was recorded (fig 2a). This occurred in apical and anterior trabecular post-infarction ventricular septal rupture and only when the transducer was placed at the apex (fig 2b). This negative waveform closely resembled that of tricuspid regurgitation with which it could easily be confused.

Diastolic left to right ventricular shunting was detected in 15 patients. This was almost pandiastolic and of much lower velocity than the systolic shunt, with a characteristic increase in velocity towards the end of diastole (fig 1, thick arrows). In eight patients continuous wave Doppler showed a reversal of flow in early diastole, indicating a short period of right to left shunting (fig 1, thin arrows). In two of these patients, simultaneous dual catheter studies indicated a “crossover” of left and right ventricular pressures during the period of isovolumic relaxation.

**COLOUR FLOW MAPPING**
Colour flow mapping detected one or more mosaic jets (indicating high velocity, turbulent flow) traversing the interventricular septum in all 20 patients. Demonstration of colour encoded transseptal flow and the exit point of the defect accurately predicted the site of the defect and this site correlated with what was found at operation (table 2). Multiple jets were seen in three patients; they arose from the same defect in two and from two separate defects in the other. In addition colour flow mapping recognised three separate areas of flow disturbance within the interventricular septum—apical (six patients), posterior (nine patients), and anterior trabecular (five patients)—each diagnostic of a specific rupture site. Figures 3-5 show examples of these different flow patterns. In fig 3, there is a broad mosaic jet traversing the interventricular septum, passing from the left to the right ventricle that represents a large apical ventricular septal rupture. In fig 4, two mosaic jets arise from the same defect and represent a fenestrated posterior ventricular septal defect, which was confirmed at operation. In fig 5, two mosaic jets seem to originate from a single anterior trabecular defect, with one jet directed towards the apex and the other jet back towards the tricuspid valve. This was caused by a muscle band that lay across the septal surface of the right ventricular exit point of the defect and split the ventricular septal defect jet into two.

Fig 2 (a) A continuous wave Doppler echocardiogram recording and (b) a diagram from a patient with an apical post-infarction ventricular septal rupture. The continuous wave recording (a) has a negative Doppler shift indicating flow directed away from the transducer. The peak systolic velocity is 4-0 m/s. The diagram (b) represents the alignment of the continuous wave transducer to the apical defect and shows how flow from the left to the right ventricle may be directed away from a precordial transducer when it is placed at the apex.
Diagnosis of ventricular septal rupture after myocardial infarction: value of colour flow mapping

Fig 3 Colour flow map recorded in the apical four chamber view during systole showing an apical post-infarction ventricular septal rupture (mosaic jet) traversing the apical septum (see text). LV, left ventricle; RV, right ventricle; IVS, interventricular septum; MV, mitral valve; TV, tricuspid valve; RA, right atrium.

Fig 4 Colour flow map recorded from the apex with the transducer tilted to image the posterior septum during systole. This shows a posterior ventricular septal rupture (see text). See fig 3 for abbreviations.

Fig 5 Colour flow map recorded in a modified four chamber view during systole. This shows an anterior trabecular rupture (see text). See fig 3 for abbreviations.

Recurrent ventricular septal rupture (see table 3) Recurrent ventricular septal rupture developed in six patients out of the 18 who underwent surgical repair (33%). This was shown by colour flow mapping studies. In four patients the diagnosis was confirmed by cardiac catheterisation and reoperation. Cross sectional echocardiography detected the presence of only one recurrent ventricular septal rupture. Pulsed Doppler was accurate in five patients and colour flow mapping was accurate in all six. In one patient colour flow mapping detected two separate ventricular septal defect jets, one at the lower end of the patch suggesting patch dehiscence and the other away from the patch suggesting further septal rupture—this was later confirmed at operation.

Table 3 Diagnostic accuracy of each echocardiographic technique in the six patients with recurrent ventricular septal rupture

<table>
<thead>
<tr>
<th>Correct diagnosis</th>
<th>PW and CW Doppler</th>
<th>Colour flow mapping</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 (17%)</td>
<td>5 (83%)</td>
<td>6 (100%)</td>
</tr>
</tbody>
</table>

See table 2 for abbreviations.

Discussion

The results from this study suggest that colour flow mapping is both a rapid and sensitive technique for the diagnosis and preoperative assessment of patients with post-infarction ventricular septal rupture. It also gave more information than conventional imaging and Doppler. In this series cross sectional echocardiography failed to image > 50% of defects,
and this finding accords with previously reported series. The addition of pulsed Doppler increased the overall diagnostic yield but was not accurate in predicting precisely the defect site. In many patients, colour flow mapping showed a widespread disturbance of right ventricular flow and this might explain the difficulties in detecting the precise right ventricular exit point of the ventricular septal defect by pulsed Doppler. Pulsed Doppler studies were also time consuming and required a more rigorous examination technique than colour flow mapping studies.

Colour flow mapping was also able to show multiple ventricular septal defect jets, which was not possible by conventional pulsed Doppler. In this study four patients had multiple jets (three new and one recurrent). Two of these patients had jets arising from different areas of the septum and so the diagnosis of multiple defects was obvious. In the other two patients the colour jets originated from the same area of the septum and so a definitive diagnosis of multiple defects was not possible. None the less, the knowledge that multiple defects were in close proximity was of practical value to the surgeon.

Three differing, discrete, areas of transseptal flow could be identified by colour flow mapping. Correlation with the findings at operation showed these to be diagnostic of apical, anterior trabecular, or posterior ventricular septal defects (figs 3–5). Apical jets represented defects confined to the apical septum, anterior trabecular jets represented defects confined to the central and anterior septum, and posterior jets represented muscular defects in the inlet septum. This classification of the site of post-infarction ventricular septal rupture flow by colour flow mapping was easily recognisable and understood, and correlated well with the findings at operation.

To date there are little published data on the use of continuous wave Doppler in the assessment of post-infarction ventricular septal rupture. The results from this study suggest that continuous wave Doppler can be used to detect and define the high velocity right ventricular flow disturbance associated with these defects. But when it is used alone confusion may arise with the recording of the velocity waveform from apical defects where a negative waveform may be obtained (fig 2a). This contrasts with both congenital defects and other forms of post-infarction ventricular septal rupture where a positive waveform is obtained. The unwary may confuse this negative waveform with tricuspid regurgitation, which produces a similar signal. In our experience the combined use of continuous wave Doppler with colour flow mapping overcame this problem because it showed the alignment of the continuous wave beam to specific areas of colour encoded transseptal flow.

The diastolic shunting patterns associated with post-infarction ventricular septal rupture are also defined by continuous wave Doppler. Left to right ventricular shunting from early to late diastole has been well described in both congenital restrictive and post-infarction ventricular septal rupture. Early diastolic right to left shunting as shown by continuous wave Doppler has not been described before. This reversal of flow occupied a short period in early diastole that corresponded with the period of isovolumic relaxation. A postulated cause might be that a delay in right ventricular emptying would cause a crossover of left and right ventricular pressures so that the pressure in the right ventricle exceeded that of the left ventricle during this short period in early diastole. Although this early reversal of diastolic flow was of low velocity and its haemodynamic importance uncertain, it could explain the mechanism of positive contrast echoangiography, where a peripheral intravenous injection of microbubbles appears to cross from the right to the left ventricle.

Colour flow mapping also proved more valuable than pulsed Doppler in the postoperative period for the assessment of patients in whom recurrent ventricular septal rupture developed. Pulsed Doppler missed the diagnosis in one patient who had coexisting mitral regurgitation. Later a diagnosis based on colour flow mapping was correct. Colour flow mapping was not only highly accurate, but in one patient distinguished between patch dehiscence and further septal rupture. This was not possible with the conventional Doppler techniques.

The role of left heart catheterisation (in particular left ventricular cineangiography) in the management of post-infarction ventricular septal rupture has been questioned. But the insensitivity of pulsed Doppler in predicting the site of the defect has led to left ventricular cineangiography being regarded as a necessary aid to operation. Colour flow mapping has now reduced the need for left ventricular cineangiography; not only is it quicker and more sensitive but it carries less risk. Left ventricular cineangiography is no longer routinely used in our unit for the preoperative assessment of post-infarction ventricular septal rupture. The role of coronary arteriography is perhaps more contentious and is outside the scope of this paper, but the initial impression from this study was that post-infarction ventricular septal rupture caused by anterior infarcts was associated with a different distribution of coronary disease than that caused by inferior infarcts—anterior infarcts were often associated with single vessel disease whereas inferior infarcts were more frequently associated with multiple vessel disease.

In our opinion, colour flow mapping is the best
Diagnosis of ventricular septal rupture after myocardial infarction: value of colour flow mapping

 technique for the diagnosis and the preoperative and postoperative assessment of post-infarction ventricular septal rupture. It supersedes conventional pulsed Doppler echocardiography and left ventricular cineangiography, and may obviate the need for any invasive investigation in selected cases.

References


