Effects of Exercise on Transmitral Gradient and Pulmonary Artery Pressure in Patients With Mitral Stenosis or a Prosthetic Mitral Valve: A Doppler Echocardiographic Study

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Doppler echocardiography was used to determine changes in transmitral gradient and pulmonary artery pressure after exercise in 12 patients with mitral stenosis and 11 patients with a prosthetic mitral valve. The mean transmitral gradient in the mitral stenosis group was 9 ± 7 mm Hg at rest and increased to 17 ± 8 mm Hg after exercise. In patients with a prosthetic mitral valve, exercise resulted in an increase in mean transmitral gradient from 5 ± 2 to 8 ± 3 mm Hg. Calculated pulmonary artery systolic pressure increased with exercise from 41 ± 19 to 70 ± 32 mm Hg in the mitral stenosis group and from 28 ± 8 to 39 ± 15 mm Hg in patients with a prosthetic valve.

Exercise Doppler echocardiographic evaluation of changes in transmitral gradient and pulmonary artery systolic pressure was found to be technically simple and an important addition to the noninvasive evaluation of patients with mitral valve disease.

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Doppler echocardiography is an accepted method for the accurate measurement of valve gradients and calculation of valve area (1). Studies (2,3) have demonstrated a high degree of correlation between Doppler-derived pressure gradients at rest and intracardiac pressure measurements in patients with either mitral stenosis or a prosthetic mitral valve. Occasionally, symptomatic patients with mitral stenosis undergoing cardiac catheterization are found to have only mild elevation of pulmonary capillary wedge pressure at rest. In such circumstances, exercise during the cardiac catheterization procedure may reveal a marked increase in mitral gradient and pulmonary artery pressure, thereby accounting for the patient's symptoms (4).

Although feasible, exercise with groin catheters in place is cumbersome and rarely reaches levels equivalent to those of daily activities. By using Doppler echocardiography in conjunction with treadmill or bicycle exercise testing, it is frequently possible to obtain adequate images for the calculation of the postexercise transvalvular gradient. Limited studies (5,6) in patients with mitral stenosis have compared Doppler-derived and catheterization-measured valve gradients with exercise and have confirmed the accuracy of the noninvasive technique. They did not examine Doppler-derived changes in pulmonary artery pressure, although pulmonary artery systolic pressure may be accurately derived from continuous wave Doppler measurements of velocity of tricuspid valve regurgitation (7,8). In patients with chronic obstructive pulmonary disease, Doppler echocardiography has accurately demonstrated the excessive increase in pulmonary artery pressure occurring with exercise (9), thereby demonstrating the feasibility of this measurement after physical exertion. Because of the paucity of published data in patients with valve disease, the present study was designed to evaluate the utility of Doppler exercise echocardiography for measuring changes in transmitral gradient and pulmonary artery pressure in patients with mitral stenosis or a prosthetic mitral valve.

Methods

Study groups. Two groups of patients participated in the study, which consisted of a two-dimensional and Doppler echocardiographic study at rest, symptom-limited treadmill exercise and repeat Doppler examination immediately after exercise. All patients had had previous echocardiographic examinations. Patients with hemodynamically significant aortic stenosis and exertional angina and those unable to exercise were excluded from the study.

The first group (Table 1) consisted of 12 patients with mitral stenosis identified on previous echocardiograms. This group comprised nine women and three men with a mean age of 48 ± 13 years (range 28 to 69). Two of these patients had associated moderate aortic stenosis (rest gradient by Doppler examination was 36 mm Hg in each), six had mild to moderate mitral regurgitation as assessed by Doppler color flow imaging and one had associated coronary artery disease without evidence of active ischemia after coronary angioplasty 1 year previously. No patient had more than minimal

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Table 1. Clinical Characteristics of 12 Patients With Mitral Stenosis

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>Age (yr)</th>
<th>Gender</th>
<th>Valvular Dysfunction</th>
<th>Rhythm</th>
<th>NYHA Class</th>
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<tbody>
<tr>
<td>1</td>
<td>37/F</td>
<td>MS, MR</td>
<td>NSR</td>
<td>II</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>37/F</td>
<td>MS, MR</td>
<td>NSR</td>
<td>II</td>
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<td>NSR</td>
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<td>MS</td>
<td>NSR</td>
<td>III</td>
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<td>5</td>
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<td>NSR</td>
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</tr>
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<td>6</td>
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<td>NSR</td>
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<td>AF</td>
<td>III</td>
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<tr>
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<tr>
<td>11</td>
<td>49/M</td>
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<tr>
<td>12</td>
<td>69/F</td>
<td>MS, CAD</td>
<td>AF</td>
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</table>

AF = atrial fibrillation; AS = aortic stenosis; CAD = coronary artery disease; F = female; M = male; MR = mitral regurgitation; MS = mitral stenosis; NSR = normal sinus rhythm; NYHA = New York Heart Association; Pt = patient.

The second study group (Table 2) comprised 11 patients who had undergone mitral valve replacement. In addition, 3 of the 11 had a prosthetic aortic valve. There were six women and five men; their mean age of 44 ± 18 years (range: 19 to 73) did not differ significantly from that in the mitral stenosis group. Two patients were included in both the mitral stenosis and the prosthetic valve group because they were studied both before and after valve replacement. Ten of the 11 patients had a mechanical valve prosthesis (Björk-Shiley in 5, St. Jude in 3); 1 patient had a bioprosthetic Carpentier-Edwards valve. Two patients had moderate aortic regurgitation on the Doppler examination at rest and 2 had a mildly depressed ejection fraction (approximately 40%). No patient had significant mitral regurgitation. Seven of the 11 patients had normal sinus rhythm and 4 had atrial fibrillation with a well controlled ventricular response at rest.

Echocardiography. All patients underwent standard rest two-dimensional echocardiography in the left lateral decubitus position. Parasternal long- and short-axis, apical two- and four-chamber and subcostal views (the latter in the supine position) were obtained with use of a Hewlett-Packard 7702A Ultrasound System. Continuous wave Doppler examination of mitral inflow was performed with an imaging 2.5 MHz transducer in the apical four-chamber view. Tricuspid regurgitant jets were examined by continuous wave Doppler imaging in multiple views. The view producing the most complete flow envelopes and highest peak velocities at rest was used for both the rest and postexercise studies. Color flow imaging was used to help orient the Doppler beam parallel to mitral inflow and tricuspid regurgitant jets. The transducer positions where tricuspid regurgitation and mitral inflow were best obtained were marked on the patient's chest for ease in obtaining these views after exercise.

The inferior vena cava was visualized in the subcostal view to estimate right atrial pressure. The response of the inferior vena cava to inspiration was observed: ≥50% collapse was defined as a right atrial pressure of 5 mm Hg. Patients who did not exhibit inspiratory collapse of the inferior vena cava were assigned a right atrial pressure of 15 mm Hg, as previously described (9). Nine of the 12 patients in the mitral stenosis group and 9 of the 11 patients with a mitral prosthesis had inspiratory collapse of the inferior vena cava.

Exercise protocol. Patients underwent symptom-limited treadmill exercise with continuous electrocardiographic (ECG) recording (Quinton 2000 exercise treadmill). Heart rate was recorded every minute and blood pressure was recorded every 3 min during exercise. Ten of the 12 patients with mitral stenosis and 10 of the 11 with a prosthetic valve exercised according to the Bruce protocol; the remainder underwent a modified Bruce protocol. Immediately after maximal treadmill exercise was completed, patients were instructed to assume the left lateral decubitus position on the examining table and mitral inflow and tricuspid regurgitant jet velocities were obtained with use of the optimal views and transducer positions found at rest. Multiple images were obtained to ensure the best possible flow envelopes. Doppler signals were recorded both on videotape with audio output and on a strip chart recorder.

Data analysis. Mitral inflow Doppler signals on videotape were digitized and measurements for mitral mean and peak gradients, mitral pressure half-time and peak tricuspid regurgitant jet velocity were made with use of cardiac analysis software on a CineView Operating System (GTI, Freeland
Medical Group. Mitral valve area was calculated from mitral pressure half-time as described by Hallet et al. (10). Peak tricuspid valve gradient during systole was calculated from peak tricuspid regurgitant jet velocity by the modified Bernoulli equation (7.8). Right ventricular systolic pressure was calculated as the sum of the tricuspid valve gradient and estimated right atrial pressure (7.8). In the absence of a pulmonary transvalvular gradient, pulmonary artery systolic pressure is equivalent to right ventricular systolic pressure and none of the study patients exhibited an abnormal transpulmonary gradient. At least 5 cardiac cycles were averaged for each measurement for patients in sinus rhythm and at least 10 cycles were used for patients in atrial fibrillation. Exercised heart rates recorded in Table 3 represent the heart rates at the time of mitral inflow imaging immediately after exercise.

Statistical analysis. Student's paired t test analysis was used to compare values between the mitral stenosis and mitral valve replacement groups. For analysis of pulmonary artery systolic pressure, only those patients who had an adequate tricuspid regurgitant jet both at rest and during exercise were included. Statistical significance was accepted at p < 0.05.

Results

Mitral stenosis group (Table 3). The heart rate in these patients responded adequately to exercise, increasing from 76 ± 13 to 118 ± 17 beats/min (p < 0.001). The calculated mitral valve area averaged 1.4 ± 0.4 cm² at rest, increasing with exercise to 1.6 ± 0.5 cm² (p < 0.05). Mean and peak tricuspid gradients increased from 9 ± 7 and 17 ± 11 mm Hg, respectively, at rest to 17 ± 8 and 28 ± 10 mm Hg with exercise (p < 0.001 for each) (Fig. 1, left). A tricuspid

Table 3. Doppler Measurements at Rest and After Exercise in 12 Patients With Mitral Stenosis or Mitral Valve Replacement, or Both

<table>
<thead>
<tr>
<th>Pt No.</th>
<th>HR (beat/min)</th>
<th>Mitral Valve Area (cm²)</th>
<th>Peak Gradient (mm Hg)</th>
<th>Mean Gradient (mm Hg)</th>
<th>PASP (mm Hg)</th>
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<td>1.6</td>
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<tr>
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<td>17</td>
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<td>&lt;0.01</td>
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*These patients were studied both before and after mitral valve replacement (MVR). Patients 4 (top) and 6 (bottom): Patients 6 (top) and Patient 10 (bottom). Peak value based on data from 11 patients whose right ventricular systolic pressure was measured at rest. For comparison with exercise data, mean value 44.3 ± 20.2 mm Hg from nine patients in whom both rest and exercise data are available is used. Patients 1 to 7 with mitral stenosis and Patients 1 to 3, 6, to 8 and 11 with mitral valve replacement had normal sinus rhythm. Patients 8 to 12 with mitral stenosis and Patients 4, 5, 9 and 10 with mitral valve replacement had atrial fibrillation. Ex = exercise; HR = heart rate; PASP = pulmonary artery systolic pressure.
A regurgitant gradient was obtainable in 11 of the 12 patients at rest and in 9 patients after exercise. The latter nine patients (75%) had a tricuspid regurgitant gradient and thus pulmonary artery systolic pressure measurable both at rest and after exercise. Pulmonary artery systolic pressure increased from 41 ± 19 to 70 ± 32 mm Hg with exercise (p = 0.005) (Fig. 2, left). Figure 3 contains an example of the tracings from an individual patient.

Mitral valve replacement group (Table 3). Heart rate increased significantly with exercise in this group from 79 ± 9 to 104 ± 21 beats/min. Mitral valve area was measured as 2.5 ± 0.6 cm² as rest and as 2.9 ± 0.7 cm² after exercise (p < 0.01). Mean and peak mitral gradients at rest were 5 ± 2 and 12 ± 5 mm Hg, respectively, increasing to 8 ± 3 and 18 ± 7 mm Hg (p = 0.001 for each) (Fig. 1, right). A tricuspid regurgitant gradient was obtainable in 9 (75%) of the 11 patients both at rest and after exercise. Pulmonary artery systolic pressure increased from 28 ± 8 to 39 ± 15 mm Hg with exercise (p = 0.002) (Fig. 2, right).

Comparison of mitral stenosis and prosthetic mitral valve groups (Table 3). Mitral valve area measured at rest was significantly larger in the subjects with valve replacement (p < 0.001). Rest heart rate was nearly identical in the two groups, but there was a trend toward a higher heart rate with exercise in the mitral stenosis group (118 ± 17 vs. 104 ± 21 beats/min; p = NS). Mean and peak mitral gradients at rest did not differ significantly between the two groups, but those after exercise were significantly higher in the patients with mitral stenosis (p < 0.005 and p < 0.05, respectively). Pulmonary artery systolic pressure was significantly higher both at rest and after exercise in the group with mitral stenosis (p < 0.05 for each).

Two patients were studied before and after mitral valve replacement and are included in both groups. Before surgery, one had a calculated mitral valve area at rest of 1.2 cm² and mean and peak mitral gradients at rest of 18 and 30 mm Hg, respectively, increasing to 27 and 38 mm Hg with exercise. After a 23 mm St. Jude Medical mitral prosthesis was implanted, the mitral valve area calculated by pressure half-time increased to 2.5 cm² and mitral mean and peak gradients decreased to 7 and 13 mm Hg, respectively, at rest and to 9 and 16 mm Hg with exercise. After valve replacement, pulmonary artery systolic pressure decreased dramatically from 75 mm Hg at rest and 105 mm Hg with exercise before operation to 26 mm Hg at rest and 40 mm Hg with exercise. The second patient responded similarly, with mean and peak mitral gradients after surgery decreasing to <50% of preoperative rest and exercise values. Tricuspid regurgitation was not present in this patient after operation; therefore, pulmonary artery pressure could not be calculated.

**Discussion**

The present study demonstrates the feasibility of exercise Doppler echocardiography in patients with mitral stenosis and those with a prosthetic mitral valve. As anticipated, exercise resulted in a significant increase in peak and mean transmural gradients in the patients with mitral stenosis. In the patients with a prosthetic mitral valve, the pressure
increase after exercise was more moderate, representing a greater valve area.

Exercise hemodynamics in mitral stenosis. This was first studied 40 years ago by Gorlin et al. (11), using supine bicycle exercise during right heart catheterization; numerous subsequent invasive studies (12,13) have confirmed the exercise-induced increase in left atrial pressure with an associated increase in pulmonary artery pressure. Doppler echocardiography offers an accurate noninvasive method of assessing the pressure gradient across a stenotic mitral valve, and its use in patients with mitral stenosis and a prosthetic valve has been validated (1-3). It is only recently that Doppler echocardiography has been used in conjunction with exercise testing to evaluate changes in transmitral gradient. Sagar et al. (5) reported on 10 patients with mitral stenosis studied by pulsed wave Doppler echocardiography at rest and after bicycle exercise. The mean mitral gradient increased from 13 mm Hg at rest to 22 mm Hg with exercise and correlated extremely closely with invasive measurements. Tamisie et al. (6) reported on 20 patients (mean mitral area 1.0 ± 0.3 cm²) evaluated with continuous wave Doppler echocardiography in association with bicycle exercise before and after percutaneous mitral valvuloplasty. They noted a mean increase in transmitral gradient from 8 mm Hg at rest to 20 mm Hg after exercise. After valvuloplasty, the mean gradient at rest decreased considerably.

Exercise hemodynamics in patients with a prosthetic mitral valve. This has also been studied with Doppler ultrasound. In a comparison of normally functioning St. Jude and Medtronic Hall valve prostheses in the mitral position, Tatini et al. (14) found a low gradient at rest (2.5 to 3.0 mm Hg) and a minimal increase with exercise (5.1 to 7.0 mm Hg). Slightly higher rest and exercise values have been found in patients with a Bjork-Shiley or Starr-Edwards mitral prosthesis, reflecting the lower functional valve area in these patients (15). Our data on the change in transmitral gradient after exercise in patients with either mitral stenosis or valve replacement demonstrated similar changes.

Pulmonary artery pressure after exercise: role of Doppler measurement of tricuspid regurgitant jet. None of the cited studies on mitral stenosis and mitral valve replacement have examined changes in pulmonary artery pressure after exercise. In normal subjects, exercise results in a decrease in invasively measured pulmonary vascular resistance with minimal or no increase in pulmonary artery pressure (16). In patients with nonvalvular heart failure, pulmonary venous hypertension is frequently present and may worsen with exercise, with pulmonary artery systolic, diastolic and mean pressures paralleling the increase in wedge pressure. In contrast, in patients with noncardiogenic pulmonary hypertension from various causes, exercise can cause a disproportionate increase in pulmonary artery systolic pressure (16).

Noninvasive assessment of the response of pulmonary artery pressure to exercise using continuous wave Doppler measurements of the tricuspid regurgitant jet has been reported in normal subjects and patients with chronic pulmonary disease and validated by simultaneous invasive hemodynamic studies (9). By noninvasive techniques, the upper limit of right ventricular systolic pressure was accepted as 30 mm Hg at rest and as 45 mm Hg with exercise (9). In patients with pulmonary disease with normal pulmonary pressure at rest, Himelman et al. (9) found that exercise resulted in an abnormal increase to mild or moderate levels of pulmonary hypertension. In contrast, those with pulmonary hypertension at rest had a steep increase in right ventricular pressure after exercise, often to levels exceeding systemic pressure.

Our study indicates the feasibility of measuring postexercise tricuspid regurgitant jet velocities in patients with mitral valve disease and demonstrates findings similar to those seen in patients with chronic pulmonary disease. At rest, pulmonary artery systolic pressure exceeded the accepted normal maximum of 30 mm Hg in 7 of the 11 patients with mitral stenosis in whom it could be assessed and in 3 of 9 patients with mitral valve replacement. After exercise, the pressure increased in all patients in whom the tricuspid regurgitant jet could be measured and was >45 mm Hg in six of nine patients with mitral stenosis and three of nine patients with mitral valve replacement. The elevation of pressure both at rest and after exercise and the absolute changes from rest to exercise were greater in patients with mitral stenosis than in those with a prosthetic heart valve.

Five patients with mitral stenosis developed pulmonary artery systolic pressure >70 mm Hg with exercise. Three of these patients were symptomatic and subsequently underwent valve replacement. A fourth patient had marked symptomatic improvement after open commissurotomy 7 years previously for severe aortic stenosis with pulmonary hypertension but had persistent ECG evidence of pulmonary hypertension with a calculated mitral valve area of 1.5 cm². Exercise revealed an increase in pulmonary artery systolic pressure from 40 to 72 mm Hg (Fig. 3), possibly explaining the failure of resolution of the right ventricular hypertrophy on the ECG. One patient remains only mildly symptomatic (functional class II).

Although we studied only two patients before and after valve replacement, the finding that rest and exercise right-sided pressures were less in the valve replacement group suggests that successful valve replacement improves the excessive exercise-induced increase in pulmonary artery systolic pressure seen in untreated mitral stenosis—an observation in accord with the well known decrease in pulmonary hypertension at rest after corrective mitral valve surgery (17). However, correction of pulmonary hypertension at rest by commissurotomy does not necessarily correct exercise-induced abnormalities and exercise Doppler echocardiography may be a useful tool in exposing residual valve obstruction (Fig. 3).

Effect of associated lesions on transmitral gradient and pressure half-time. The calculation of transmitral gradient by Doppler echocardiography relies on mathematic assump-
Figure 3. Patient 5. Rest and exercise continuous wave Doppler tracings, showing persistence of right ventricular hypertrophy after mitral commissurotomy. The rest aortic valve (MV) and tricuspid regurgitation (TR) Doppler tracings (top and bottom left) show only a mild mean mitral gradient (GRAD) and mild pulmonary hypertension. Exercise (top and bottom right) results in a marked increase in pressures with an increase in calculated pulmonary artery systolic pressure (PASP) from 40 to 72 mm Hg (pressure measurements based on calculation of the mean of five cardiac cycles). Tracings are recorded at different scales as indicated. BPM = beats/min; HR = heart rate.

The increase in transmitral gradient underestimated the true increase in left atrial pressure.

The pressure half-time calculation for the measurement of mitral valve area was originally considered to be unaltered by exercise (10). However, subsequent studies by the same investigators (19) with validation by catheterization indicated that Doppler-derived pressure half-time was indeed affected by exercise, decreasing by an average of 30 ms. Other investigators (5,6) have confirmed this finding. We also noted a decrease in pressure half-time with exercise, resulting in a moderate increase in calculated valve area in most subjects (Table 3). Although the exact mechanism of this change in pressure half-time remains uncertain, Thomas and Weyman (20) suggested that it can be caused by changes in left atrial compliance or an increased peak transmitral gradient, or both. Because the calculation of mitral valve area after exercise was not the primary aim of our study, variations in this derived measurement are of interest but do not detract from the value of exercise for the assessment of transmitral and pulmonary artery pressures.

Tricuspid and aortic regurgitation are common in patients with mitral stenosis. Although significant aortic regurgitation may lead to overestimation of mitral valve area when calculated by pressure half-time (20), none of our patients had more than a trivial degree of regurgitation. Moderate mitral regurgitation was present in six patients with mitral stenosis, all of whom had normal ventricular function. Pressure half-time measurements in subjects with coexisting mitral stenosis and regurgitation have been shown to accurately reflect the valve area (1,21). Although significant concomitant mitral regurgitation may elevate left atrial pressure, this will be reflected in the transmitral Doppler-derived gradient at rest and with exercise, as well as in the pulmonary artery pressure. Thus, although different lesions may have different effects on Doppler-derived mitral valve area or gradient, or both, particularly during exercise, examination of the changes in pulmonary artery pressure combined with knowledge of the effects of exercise and other valve lesions may minimize errors in clinical assessment of the hemodynamic severity of mitral stenosis.

Limitations of the study. This study represents our initial experience in a relatively small group of patients with mitral valve disease. Because these patients were unselected and represent a typical group of patients with mitral stenosis or a mitral valve prosthesis, we believe that the results will be applicable to larger series.

Table 3 shows that tricuspid regurgitation was absent at rest in one patient with mitral stenosis and in two with a mitral valve prosthesis. Two additional patients with mitral stenosis (Patients 6 and 11) had trivial tricuspid regurgitation at rest that could not be adequately visualized after exercise. These findings are similar to those previously reported (22,23) and compare favorably with those observed in patients with chronic pulmonary disease, of whom only 56% at rest and 39% with exercise had adequate tricuspid regurgitant jet signals (9). In our series all patients with inadequate
tricuspid velocity signals had a mean mitral gradient at rest 
<5 mm Hg and a calculated valve area ≥1.5 cm², indicating 
only mild to moderate mitral stenosis. These patients 
are likely to have had little or no pulmonary hypertension—a 
group in whom the prevalence of tricuspid regurgitation is 
comparatively low (23).

Although we did not evaluate the technique, it is possible 
that the tricuspid regurgitant jet yield could have been 
unfurther improved by enhancement with saline solution 
(9,24). An alternative method is the use of supine bicycle 
exercise, which permits continuous measurements of Doppler 
flow (5,25). However, no increased yield would have 
been expected in the three patients who had no tricuspid 
regurgitation at rest.

A potential limitation of this study is the lack of invasive 
correlation with the noninvasive measurements. Several 
previous studies (2,6,14,15) have shown an extremely high 
correlation between invasively and noninvasively measured 
transmitral gradient in native and prosthetic valves. In 
addition, pulmonary artery pressure at rest and exercise measured 
invasively has been demonstrated to correlate accurately with 
noninvasive assessment (9). Thus, we believe that there is 
a adequate prior evidence to validate this technique.

Calculation of mitral valve area with use of pressure 
half-time resulted in a slightly higher valve area after 
exercise. This phenomenon is consistent with previous observations 
(6) and may represent an overestimation of valve area 
at a high heart rate, rather than a true increase in valve area. 
It has been suggested (18) that pressure half-time may not 
give accurate results for measuring prosthetic mitral valve 
area, although not all investigators agree with this observation. 
In our study, derivation of mitral valve and prosthetic 
valve area was a secondary consideration and, whether or 
not these calculations are valid, we believe that the gradients 
calculated across the mitral and tricuspid valves are accurate 
and give important information.

Conclusions. Our data suggest that exercise echocardiography 
provides useful information in patients with mitral 
valve disease. It may be very helpful in assessing the 
hemodynamic severity of mitral stenosis in borderline cases 
and in determining the physiologic function of prosthetic 
valves. The addition of continuous wave Doppler assessment 
of the tricuspid regurgitant jet is technically feasible in 
most cases and adds considerable information for measurement 
of the transmitral gradient. As with many studies, exercise echocardiography is probably of greatest value in 
borderline cases where symptoms are greater than anticipated 
from Doppler pressure measurements at rest. In such cases, exercise may reveal otherwise apparent abnormalities and assist decision making.

References