Left ventricular outflow tract gradient decrease with non-surgical myocardial reduction improves exercise capacity in patients with hypertrophic obstructive cardiomyopathy

W. Rużyłło, L. Chojnowska, M. Demkow, A. Witkowski, B. Kuśmierczyk-Droszcz, W. Piotrowski, L. Rausinska, M. Karcz, L. Malecka and W. Rydlewska-Sadowska

National Institute of Cardiology, Warsaw, Poland

Objectives This study was undertaken to evaluate mid-term clinical results of non-surgical myocardial reduction in patients with hypertrophic obstructive cardiomyopathy.

Methods Twenty-five patients with left ventricular outflow tract obstruction (mean gradient of 84.54 ± 31.38 mmHg) and symptoms of dyspnoea, angina and/or syncope were treated with non-surgical myocardial reduction. The patients were followed-up for a mean period of 10.44 ± 1.8 months. In all patients clinical examination with echocardiography was repeated after every 3 months of follow-up, and a symptom-limited treadmill test was repeated at the 6 month follow-up. Eighteen patients underwent simultaneous respiratory gas analysis.

Results Clinical follow-up examinations were achieved in all 25 patients. Persistent left ventricular outflow tract gradient reduction was seen in 23 patients. Seventeen patients had a reduction of left ventricular outflow tract gradient >50% of baseline value. Twenty patients showed a clinical improvement from 2.8 ± 0.5 up to 1.2 ± 0.5 NYHA class (P<0.001). The clinical improvement was matched by an improvement in objective measures of exercise capacity in patients with significant left ventricular outflow tract gradient reduction. Exercise time increased from 571.9 ± 192.2 to 703.5 ± 175.4 s, P<0.001, and peak VO2 increased from 14.6 ± 5.2 to 20.5 ± 8.6 ml . kg⁻¹ min⁻¹, P<0.05.

Conclusion Significant left ventricular outflow tract gradient reduction with exercise capacity improvement was achieved in the majority of patients treated with non-surgical myocardial reduction. We recommend this method as an alternative to surgery for symptomatic patients with hypertrophic obstructive cardiomyopathy.

Key Words: Hypertrophic obstructive cardiomyopathy, catheter therapy, exercise capacity.

See page 704 for the Editorial comment on this article

Introduction

Hypertrophic cardiomyopathy is a disease caused by a number of mutations in the genes encoding cardiac sarcomeric proteins[1]. Twenty-five percent of patients with hypertrophic cardiomyopathy have evidence of obstruction of the left ventricular outflow tract[2]. Diastolic dysfunction and myocardial ischaemia are pathophysiological features of the disease responsible for reduced exercise capacity and functional limitation[3,4]. The physiological significance of left ventricular outflow tract obstruction is still not fully confirmed. Increased left ventricular filling pressure, documented in patients with hypertrophic obstructive cardiomyopathy during exercise[5], suggests that left ventricular outflow tract obstruction contributes to exercise limitation.

Patients with hypertrophic obstructive cardiomyopathy frequently complain of exertional dyspnoea, angina, and syncope. The therapeutic options for the treatment of obstructive hypertrophic cardiomyopathy include negative inotropic agents, DDD pacing, and myectomy. Recently, the new method of left ventricular outflow tract gradient reduction, non-surgical myocardial reduction, has been introduced as an alternative
to surgery for symptomatic patients with hypertrophic obstructive cardiomyopathy\[6\]. Published immediate and short-term results of non-surgical myocardial reduction show that this new catheter technique results in a reduction of left ventricular outflow tract gradient with an improvement in the quality of life in the majority of treated patients\[7\]. However, the improvement in exercise capacity measured by treadmill time and oxygen consumption was not clearly documented.

The aim of this study was to evaluate the mid-term clinical course after non-surgical myocardial reduction, with assessment of exercise capacity response to the procedure in a group of symptomatic patients with hypertrophic obstructive cardiomyopathy.

**Patients, methods**

The study group consisted of 25 consecutive patients with hypertrophic obstructive cardiomyopathy in whom reduction of the left ventricular outflow gradient was achieved using non-surgical myocardial reduction. One other patient was excluded from follow-up analysis because non-surgical myocardial reduction could not be performed due to failed balloon placement in the septal artery. During the intervention, a mean of 1·08 ± 0·5 (range 1 to 2) septal branches of the left anterior descending artery were occluded by injection of 3·4 ± 1·2 ml (range 1 to 6) of alcohol. Hypertrophic obstructive cardiomyopathy was diagnosed according to WHO/ISFC 1995 criteria\[13\]. All patients had echocardiographic evidence of significant left ventricular outflow tract obstruction, with prolonged mitral valve-septal contact. Mild or moderate (Sellers class 1 or 2) mitral insufficiency was found in 15 patients, and 10 patients had Sellers class 3 mitral insufficiency found during echo-Doppler examinations. None of the patients had severe mitral valve regurgitation requiring mitral valve replacement. All patients were symptomatic despite therapy with beta-blockers, verapamil or disopyramide, used either alone, or in combination in some patients. Six patients had previously undergone DDD pacemaker implantation to reduce the left ventricular outflow tract gradient. Patient characteristics at baseline are shown in Table 1.

Non-surgical myocardial reduction was approved by the Ethical Committee of the National Institute of Cardiology in Warsaw, as a method of therapy for severely symptomatic patients with hypertrophic obstructive cardiomyopathy unresponsive to medical therapy, in whom there was a left ventricular outflow tract gradient ≥ 30 mmHg at rest. All patients gave their written consent to the procedure and were informed about all alternative methods of therapy.

The patients were followed up for a mean period of 10·4 ± 1·8 months (range 6 to 13) after non-surgical myocardial reduction. Clinical examination, 12-lead ECG, echocardiography and 24 h ambulatory ECG were performed after every 3 months of follow-up. An exercise test was done after 6 months of follow-up. In the first 18 patients haemodynamic re-examination was done at 3 month follow-up. All investigations were carried out on a pre-planned schedule.

**Echocardiography**

Echocardiographic examination was performed using a Hewlett-Packard Sonos 5500 or 1500 system with a 2·5 MHz transducer. Transthoracic echocardiography was performed before and during the procedure, 1 to 2 days after, at discharge, and at 3- and at 6-month follow-up. The following parameters were measured: left ventricular end-systolic and end-diastolic dimensions — from the minor axis M-mode of the left ventricle.

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**Table 1 Clinical characteristics of 25 patients with hypertrophic obstructive cardiomyopathy who underwent non-surgical myocardial reduction**

<table>
<thead>
<tr>
<th>Age (years, mean and range)</th>
<th>49·3 ± 13·1 (20–72)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td>15 M, 10 F</td>
</tr>
<tr>
<td>Symptoms (years, mean and range)</td>
<td>6·1 ± 4·2 (1–19)</td>
</tr>
<tr>
<td>Dyspnoea NYHA class</td>
<td>2·8 ± 0·5</td>
</tr>
<tr>
<td>Angina CCS class</td>
<td>2·1 ± 0·8</td>
</tr>
<tr>
<td>Syncope</td>
<td>10/25</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>NSVT 4/25</td>
</tr>
<tr>
<td>LVOT gradient (mmHg, mean and range)</td>
<td>84·5 ± 31·3 (30–148)</td>
</tr>
<tr>
<td>Peak VO₂ (ml. kg⁻¹ min⁻¹, mean and range)</td>
<td>14·8 ± 4·1 (9·4–22·0)</td>
</tr>
<tr>
<td>Drug treatment</td>
<td>Beta-blockers 17/25</td>
</tr>
<tr>
<td></td>
<td>Verapamil 8/25</td>
</tr>
<tr>
<td></td>
<td>Disopyramide 3/25</td>
</tr>
<tr>
<td></td>
<td>Amiodarone 4/25</td>
</tr>
<tr>
<td></td>
<td>DDD pacemaker (previously implanted to relief LVOT gradient) 6/25</td>
</tr>
</tbody>
</table>

NSMR = non-surgical myocardial reduction; NYHA = New York Heart Association; CCS = Canadian Cardiovascular Society; NSVT = non-sustained ventricular tachycardia; LVOT = left ventricular outflow tract; peak VO₂ = peak oxygen consumption; DDD = dual chamber pacing.
obtained from a two-dimensional standard left parasternal view, septal thickness—from the same trace, left atrial dimension—from the M-mode at the level of the aortic valve at end-systole obtained from a two-dimensional standard left parasternal view; and left ventricular outflow gradient—measured by use of a continuous-wave Doppler probe positioned at the cardiac apex.

Quantification of mitral regurgitation (detected by colour flow Doppler), based on the size of the regurgitant jet into the left atrium was graded as follows: 0, absent; 1, mild; 2, small; 3, moderate; 4, large. The presence of systolic anterior motion of mitral valve was assessed from the M-mode and graded as follows: 0—absent; 1—present with minimum distance between mitral valve and ventricular septum during systole >10 mm, 2—without mitral-septal contact but with a distance of <10 mm between the mitral valve and septum; 3—brief mitral-septal contact (<30% of systole); 4—prolonged apposition of mitral valve leaflet to the septum.

Electrocardiography

In order to evaluate possible conduction disturbances, all patients had a standard 12-lead ECG, with a paper speed of 50 mm . s⁻¹, performed at the baseline, immediately after the procedure, and every day until discharge from hospital as well as during every follow-up visit.

24-hour ambulatory ECG monitoring

Holter registration was recorded digitally on disc with no data compression using three-channel Burdick Combo recorders (Burdick Inc., Syracuse, U.S.A.) with a sampling rate of 200 Hz for 24 h. The Holter registrations were done at baseline (1–2 days before the procedure), during the procedure, at discharge, and after 3 months and 6 months of follow-up.

Ventricular tachycardia was defined as a run of three or more consecutive ventricular beats at a rate of 120 beats . min⁻¹ or more.

Exercise testing

Twenty-five patients underwent a symptom-limited treadmill exercise test before and 6 months after the procedure. A modified Bruce protocol was used. Eighteen patients underwent simultaneous respiratory gas analysis (Vmax 29c Series Spectrometer, Sensor Medics, California, U.S.A.). Metabolic gas exchange was measured every 10 s.

Cardiac catheterization, gradient determination and ablation procedure

The right and the left femoral arteries as well as the right femoral vein were cannulated with a standard Judkins technique. A 6 F pacemaker lead was placed in the right ventricle. A 6 F pigtail catheter was positioned in the left ventricular apex and a 7 F Judkins guiding catheter in the ascending aorta. The left ventricular outflow tract gradient was determined at rest, during a Valsalva manoeuvre and after extrasystole. After an intravenous standard bolus of 10 000 U of heparin, the ostium of the left coronary artery was intubated and the first large septal branch was identified on the coronary angiogram. This was followed by insertion of a 0.014 inch guide wire into this septal branch (Stabilizer Marker Wire, Cordis or Roadrunner, COOK) and then a 2/0/2.5 mm diameter, 10 mm length balloon catheter (G-20, BARD USCI or Cobra, Boston Scientific Corp.) was placed in the proximal part of the target artery. After the balloon was inflated to 6 bar, the correct balloon position was determined by injection of contrast medium via the guiding catheter into the left coronary artery, and then by injection through the balloon catheter shaft into the septal branch. If the proximal part of the balloon was protruding into the left anterior descending artery, the balloon was deflated, pushed deeper into the septal branch, then reinflated and the position of the balloon checked again. However, at least part of the balloon was always kept in a main trunk of the septal branch to be sure that the whole supplied territory would be ablated.

When satisfactory balloon positioning was achieved, the guide wire was pulled back and 1 ml of echocardiographic contrast medium Levovist (Schering AG, Berlin) was injected through the balloon catheter shaft for transthoracic echocardiography determination of the myocardial supply area of the closed septal branch. When the area of interest, the septal myocardium forming the left ventricular outflow tract, was identified by contrast echocardiography, a slow injection of 1 to 6 ml of 96% alcohol was performed through the balloon catheter shaft. Five minutes after the completion of the alcohol injection, the balloon was deflated and removed and the morphological results of the procedure were examined by coronary angiography. The left ventricular outflow tract gradient measurements were repeated.

Statistical analysis

All values are presented as a mean ± one standard deviation. P value of <0.05 was considered significant. The paired Student t-test for continuous variables and chi-square test for discrete variables were used. All statistical analyses were performed using SAS software Ver.6.12.

Results

Complications

Technical complications

In three patients from the subset of the first 12 persons treated with non-surgical myocardial reduction, closure
of the left anterior descending segment distal to target septal branch was found during routine angiography performed immediately after the completion of the procedure. All three patients developed anterior myocardial infarction. The mean peak rise in creatinine kinase was 3914 IU (5640 to 22). At angiography performed at the 3 month follow-up the left anterior descending occlusion was found to be transient in two patients. Moderate deterioration of the left ventricular contractile function was found in one patient who remained symptomatic, as before the procedure. In two other patients left ventricular function was preserved and both reported symptomatic improvement confirmed by exercise capacity improvement. Baseline and post-procedural clinical characteristics, echocardiographic parameters and exercise capacity measurements of the patients with acute left anterior descending occlusion are shown in Table 2. We think that in all cases this complication was the result of the alcohol leakage to the left anterior descending, because of the relatively short period of the balloon occlusion (5 min) after the completion of the alcohol injection.

**Ventricular arrhythmia**

No patient developed ventricular fibrillation or sustained ventricular tachycardia including those with acute left anterior descending occlusion. In two of 21 patients, who did not have non-sustained ventricular tachycardia before non-surgical myocardial reduction, short non-sustained ventricular tachycardia runs were recorded on 24-ambulatory ECG monitoring during follow-up.

**Heart block**

Conduction abnormalities were observed after the procedure in 24 of 25 patients. In 23 patients conduction abnormalities occurred during or immediately after the procedure. In the remaining patient transient left bundle branch block developed 5 days later. Conduction abnormalities persisted in 16 of 25 patients at the 6 month follow-up. Complete heart block occurred in seven patients after ablation but in the three patients it lasted only 1 to 3 min. The other four patients required DDD pacemaker implantation before discharge. At the 6 month follow-up complete heart block was found to be persistent in only one patient. In this patient left bundle branch block was observed before the procedure.

**Symptoms**

The 6th month follow-up was available for all 25 patients. At this time, 20 patients had experienced a significant clinical improvement from $2.8 \pm 0.5$ to $1.2 \pm 0.5$ NYHA class, $P<0.001$. The other five patients showed no improvement of functional class. Seventeen of 18 patients who complained of angina showed improvement of their CCS class from $2.1 \pm 0.8$ to $0.28 \pm 0.7$, $P<0.0001$. One patient reported no change in his angina status. A recurrence of syncope was observed in only one of ten patients who complained of syncope before the procedure. Three patients who had symptomatic paroxysmal atrial fibrillation before the procedure have not been observed to have a recurrence of arrhythmia during follow-up.

**Echocardiography**

Echocardiographic results assessed at baseline, and at the 3 and at 6 month follow-up were compared in all 25 patients (Table 3). Left ventricular outflow tract gradient reduction persisted in 23 of 25 patients. In 17 patients the left ventricular outflow tract gradient diminished >50% of the baseline value, but in four patients it decreased by less than 30% of the baseline value. In two other patients the left ventricular outflow tract gradient reduced to 35% and 32% of baseline value. In two patients it did not change from baseline value, despite its reduction immediately after non-surgical myocardial reduction. Echocardiographic estimation of the left ventricular size showed the following changes: the end-diastolic dimension did not change but the end-systolic diameter increased. The left ventricular ejection fraction decreased. Interventricular septum thickness also decreased. The systolic anterior motion of mitral valve phenomenon was reduced but remained apparent. Mitral regurgitation significantly decreased, and the left atrial dimension showed a trend towards diminishing. There were no significant differences between echocardiographic measurements taken at 6 month and 3 month follow-up.

**Exercise testing**

At the 6 month follow-up examination, significant improvement in exercise capacity was found. Treadmill exercise time increased from $573.5 \pm 162.5$ (before non-surgical myocardial reduction) to $672.7 \pm 130.8$ s, $P<0.01$, and peak VO$_2$ increased from $14.8 \pm 4.1$ (before non-surgical myocardial reduction) to $18.9 \pm 6.9$ ml kg$^{-1}$ min$^{-1}$, $P<0.05$. Evaluation of exercise capacity separately for the group of 17 patients with left ventricular outflow tract gradient reduction >50% of baseline value (group 1) and for the group of eight patients in whom left ventricular outflow tract gradient decreased <50% of baseline value or remained unchanged in comparison with baseline value (group 2), showed the following results: in group 1 exercise time increased from $571.9 \pm 192.2$ (before the procedure) to $703.5 \pm 175.4$ s, $P<0.001$, and peak VO$_2$ increased from $14.6 \pm 5.2$ (before the procedure) to $20.5 \pm 8.6$ ml kg$^{-1}$ min$^{-1}$, $P<0.05$; in group 2 exercise time increased from $578.0 \pm 207.6$ (before the procedure) to $619.4 \pm 128.3$ s, ns, and peak VO$_2$ increased from $15.2 \pm 4.5$ (before the procedure) to $16.5 \pm 5.8$ ml kg$^{-1}$ min$^{-1}$, ns. Exercise capacity improved only in those patients in whom a significant gradient reduction was achieved.
Table 2  Clinical and echocardiographic characteristics of 3 patients with acute occlusion of left anterior descending artery

<table>
<thead>
<tr>
<th>Patient (initials, age, sex)</th>
<th>Study period</th>
<th>Symptoms</th>
<th>Exercise capacity</th>
<th>Echocardiographic measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Dyspnoea</td>
<td>Angina</td>
<td>Exercise time</td>
</tr>
<tr>
<td></td>
<td></td>
<td>NYHA class</td>
<td>CCS class</td>
<td>s</td>
</tr>
<tr>
<td>J-S, 53-year-old man</td>
<td>baseline</td>
<td>III</td>
<td>III</td>
<td>890</td>
</tr>
<tr>
<td></td>
<td>3 month F/U</td>
<td>I</td>
<td>I</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>6 month F/U</td>
<td>I</td>
<td>I</td>
<td>900</td>
</tr>
<tr>
<td>K-M, 39-year-old man</td>
<td>baseline</td>
<td>III</td>
<td>III</td>
<td>720</td>
</tr>
<tr>
<td></td>
<td>3 month F/U</td>
<td>II</td>
<td>III</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>6 month F/U</td>
<td>II</td>
<td>III</td>
<td>640</td>
</tr>
<tr>
<td>D-K, 28-year-old woman</td>
<td>baseline</td>
<td>III</td>
<td>II</td>
<td>660</td>
</tr>
<tr>
<td></td>
<td>3 month F/U</td>
<td>I</td>
<td>I</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>6 month F/U</td>
<td>I</td>
<td>I</td>
<td>720</td>
</tr>
</tbody>
</table>

NYHA=New York Heart Association; CCS=Canadian Cardiovascular Society; Peak VO₂=peak oxygen consumption; LVOT=left ventricular out-flow tract; LVDD=left ventricular end-diastolic diameter; LVSD=left ventricular end-systolic diameter; LA=left atrium; LVEF=left ventricular ejection fraction.
Table 3  Comparison of echocardiographic variables before non-surgical myocardial reduction and after procedure in 25 patients with hypertrophic obstructive cardiomyopathy

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>P value</th>
<th>3 month F/U</th>
<th>P value</th>
<th>6 month F/U</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVDD (mm)</td>
<td>42.1 ± 3.4</td>
<td>ns</td>
<td>45.0 ± 5.2</td>
<td>ns</td>
<td>44.6 ± 5.8</td>
</tr>
<tr>
<td>LVSD (mm)</td>
<td>23.4 ± 4.1</td>
<td>0.001</td>
<td>25.57 ± 5.2</td>
<td>ns</td>
<td>25.5 ± 8.8</td>
</tr>
<tr>
<td>IVS (mm)</td>
<td>20.0 ± 5.2</td>
<td>0.008</td>
<td>17.8 ± 5.9</td>
<td>ns</td>
<td>17.2 ± 4.9</td>
</tr>
<tr>
<td>LA (mm)</td>
<td>44.6 ± 6.1</td>
<td>0.05</td>
<td>41.9 ± 4.8</td>
<td>ns</td>
<td>38.4 ± 13.3</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>75.4 ± 10.0</td>
<td>0.04</td>
<td>71.0 ± 10.7</td>
<td>ns</td>
<td>65.6 ± 18.3</td>
</tr>
<tr>
<td>SAM</td>
<td>3.7 ± 0.5</td>
<td>0.0001</td>
<td>1.5 ± 0.9</td>
<td>ns</td>
<td>1.0 ± 0.8</td>
</tr>
<tr>
<td>Mi</td>
<td>2.4 ± 0.7</td>
<td>0.0001</td>
<td>1.1 ± 0.5</td>
<td>ns</td>
<td>1.1 ± 0.5</td>
</tr>
<tr>
<td>LVOT gradient (mmHg)</td>
<td>84.5 ± 31.3</td>
<td>0.0001</td>
<td>35.9 ± 28.1</td>
<td>ns</td>
<td>32.4 ± 25.8</td>
</tr>
</tbody>
</table>

NSMR=non-surgical myocardial reduction; LVDD=left ventricular end-diastolic diameter; LVSD=left ventricular end-systolic diameter; IVS=interventricular septum; LA=left atrium; LVEF=left ventricular ejection fraction; SAM=systolic anterior motion of mitral valve; Mi=mitral insufficiency; LVOT=left ventricular outflow tract.

Haemodynamics

Haemodynamic data showed that the left ventricular outflow tract gradient reduction persisted in 17 of 18 recatheterized patients at the 3 month follow-up. Simultaneous registration of intraventricular and aortic systolic pressures showed a resting gradient below 30 mmHg in 15 patients, and gradients of more than 30 mmHg in the remaining three patients. Provocative gradients were higher with a mean of 40.7 mmHg during the Valsalva manoeuvre and 79.2 mmHg after exercise. Comparisons of left ventricular outflow tract gradients measured invasively directly before and after the procedure as well as at the 3 month follow-up are shown in Fig. 1.

Discussion

Until recently, surgical myotomy–myectomy and atrioventricular sequential pacing were the only methods of treatment offered to patients who remained symptomatic despite medical therapy. Long-term results of myotomy–myectomy confirmed that relief of left ventricular outflow tract obstruction results in symptomatic improvement in the majority of patients[12,13,14]. However, the procedure requires extracorporeal circulation and is associated with surgical risk[13,14]. Dual-chamber pacing with shortened A-V delay reduces left ventricular outflow tract gradient and improves quality of life in a substantial percentage of patients, but it is not clear if symptomatic improvement is matched by exercise capacity increase[13,16]. A non-surgical method of septal reduction using a catheter technique aims to avoid the surgical risk. The method was introduced after observations were made on left ventricular outflow tract gradient reduction due to spontaneous myocardial infarction in hypertrophic obstructive cardiomyopathy patients[17,19], on the effect of transient balloon occlusion of coronary artery on wall motion[19], and on the left ventricular outflow tract obstruction relief by transient occlusion of the first septal branch of the left coronary artery[6,20].

The follow-up data from our patients showed that left ventricular outflow tract gradient reduction with non-surgical myocardial reduction persisted for 6 months after the procedure in most patients. However, a renewed increase in the gradient during follow-up was seen in two of 25 patients. In a substantial percentage of our patients, left ventricular outflow tract gradient reduction was associated with symptomatic improvement. Moreover, these patients, in whom a significant reduction of left ventricular outflow tract gradient was achieved, had an improvement in objective measures of exercise capacity such as exercise time and maximum oxygen uptake. In contrast, the exercise capacity did not improve in those patients in whom left ventricular outflow tract gradient reduction was not significant. These findings confirm the physiological importance of a left ventricular outflow tract obstruction.

Echocardiographic assessments, performed at 3 and at 6 months follow-up, showed an increase in the end-systolic left ventricular dimension with slightly impaired, but still normal overall contractile function. Comparing the measurements performed at 3 and at 6 months after the procedure we found neither a further increase in end-systolic left ventricular dimension nor contractile function deterioration. Progressive septal thinning was not observed either. Regarding these results, the likelihood of progression to a dilated phase of disease does not seem very high, but the left ventricular cavity dimensions and contractile function need to be evaluated during a longer follow-up. Reduction of mitral regurgitation and left atrial dimension observed in our patients during follow-up confirmed a good haemodynamic result of non-surgical myocardial reduction. Reduction of the degree of mitral insufficiency has been accepted as an important proof of surgical success in myectomy[15].

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We did not observe further left ventricular outflow tract gradient reduction when the left ventricular outflow tract gradient values found at 3 months were compared to those at the 6 month follow-up examination. This suggests that the process of left ventricular remodelling after induced septal infarction reaches a plateau after 3 months.

Reducing the left ventricular outflow tract gradient to less than 50% of baseline value did not carry benefit to the patients. We believe that the main reason for this failure was the distal occlusion of the target artery due to sub-selective alcohol injection. This resulted in only partial ablation of the target septal area. Another cause of significant gradient reduction failure was the unfavourable anatomy of the septal branches found in some of our patients. The result was that occlusion of only one of the septal arteries resulted in only partial ablation of the target septal area. In patients with insignificant left ventricular outflow tract gradient reduction, who are still symptomatic, a further non-surgical myocardial reduction procedure with occlusion of the second left anterior descending septal branch may be considered. It has been shown that in some patients a good haemodynamic and clinical result can be achieved only after a second intervention.

The most important complication of the procedure was acute occlusion of the left anterior descending observed in three of our patients. To avoid this complication we prolonged the balloon inflation time after the last alcohol injection from 5 to 10 min. Furthermore, special attention should be paid to placing the balloon in the correct position. The balloon should not be placed too proximally, but should be inflated just distal to the origin of the septal artery.

Conduction abnormalities were the most frequent complication of the procedure. A complete A-V block observed in seven patients after non-surgical myocardial reduction disappeared in several minutes in three patients and only four (16%) patients required pacemaker implantation before discharge. A similar proportion of patients in whom a permanent pacemaker was necessary after the procedure has been reported by other authors. Disappearance of complete A-V block may be explained by resolution of peri-infarction oedema. Right bundle branch block is the most frequent conduction abnormality observed after the procedure. Right bundle branch block was found after the procedure in 12 (48%) of our patients. Another complication of the procedure was non-symptomatic non-sustained ventricular tachycardia. Short runs of non-sustained ventricular tachycardia were found in two of our patients at the 3 month follow-up. Because myocardial infarction may predispose to serious arrhythmia, the ambulatory ECG needs to be monitored very closely during longer follow-up.
Also, the long-term results of the non-surgical myocardial reduction on cardiac function and prognosis need further evaluation. Since the procedure (like surgery) is a palliative treatment of the disease whose pathophysiological features also include diastolic dysfunction and ischaemia as well as arrhythmia in some patients, further medical treatment with beta-blockers and calcium antagonists or with antiarrhythmic drugs should be considered.

Conclusions

The mid-term results of non-surgical myocardial reduction confirmed immediate and short-term observations that this new catheter technique is a promising method of treatment for hypertrophic obstructive cardiomyopathy patients. Achieved haemodynamic and clinical results with documented exercise capacity improvement recommend non-surgical myocardial reduction as an alternative therapy to surgery. The risk of non-surgical myocardial reduction in terms of left ventricular remodelling and rhythm disturbances observed during mid-term follow-up appears to be acceptable. However, the long-term results of the procedure need to be carefully investigated in prospective studies.

References