Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy
Results With Respect to Intraprocedural Myocardial Contrast Echocardiography

Lothar Faber, MD; Hubert Seggewiss, MD; Ulrich Gleichmann, MD

Background—Percutaneous transluminal septal myocardial ablation (PTSMA) has been introduced as an alternative procedure for reducing the left ventricular outflow tract gradient (LVOTG) in hypertrophic obstructive cardiomyopathy. We report on the acute and mid-term results in 91 symptomatic patients with respect to intraprocedural myocardial contrast echocardiography (MCE).

Methods and Results—PTSMA was intended for 46 women and 45 men (54.1 ± 15.5 years). In 2 patients, the intervention could not be completed. In the first 30 patients the target vessel was determined by probatory balloon occlusion alone and in the remainder by additional intraprocedural MCE. Resting LVOTG was reduced from 73.8 ± 35.4 to 16.6 ± 18.1 and nostextrasytolic LVOTG from 149.3 ± 42.5 to 61.9 ± 43.0 mm Hg (P < 0.0001 each). In 10 (11%) patients, permanent DDD pacemaker implantation was necessary. Two (2%) patients died, 1 from ventricular fibrillation associated with treatment for chronic obstructive pulmonary disease after 9 days and 1 from fulminant pulmonary embolism after 2 days. After 3 months, mean New York Heart Association class was reduced from 2.8 ± 0.6 to 1.1 ± 1.0 (P < 0.0001). The LVOTG remained reduced to 14.6 ± 25.5 mm Hg at rest and 49.1 ± 48.7 mm Hg (P < 0.0001 each). Four patients underwent successful repeat PTSMA. Determination of the target vessel by MCE was associated with a higher rate of acute (92% vs 70%; P < 0.01) and mid-term (94% vs 64%; P < 0.01) success.

Conclusions—PTSMA is a promising nonsurgical technique for reduction of symptoms and LVOTG in hypertrophic obstructive cardiomyopathy. MCE has been shown to be a useful addition to probatory balloon occlusion for target vessel selection. Prospective, long-term observations of larger populations and a comparison with the established forms of therapy are necessary to determine the definitive significance of PTSMA.

Key Words: hypertrophy • cardiomyopathy • ablation • contrast media • echocardiography

Non surgical catheter treatment of hypertrophic obstructive cardiomyopathy (HOCM) was introduced in 1994. In patients considered to be candidates for surgical myectomy, alcohol-induced percutaneous transluminal septal myocardial ablation (PTSMA) reduces symptoms and left ventricular (LV) outflow tract gradients (LVOTG). Intraprocedural myocardial contrast echocardiography (MCE) as an imaging technique has been integrated into the procedure by our group. We report on acute and mid-term results in the first 91 patients treated with PTSMA with respect to this technical modification.

Methods

Study Population
Between January 1996 and December 1997, 182 patients with HOCM were referred to our center for further evaluation. Interventional treatment was intended in 91 of these who were seen with the following inclusion criteria: New York Heart Association/Canadian Cardiovascular Class (NYHA/CCS) functional class III or IV, LVOTG > 50 mm Hg at rest or > 100 mm Hg at provocation (Valsalva maneuver, isoproterenol, or postextrasystole) and ≥1 septal branch suitable for intervention. Patients with class II symptoms were accepted for intervention if medical treatment was not tolerated or if a high LVOTG was combined with the presence of multiple risk factors for sudden cardiac death. Patients with coexistent cardiac abnormalities requiring surgery were excluded. Myectomy had been performed in 5 (6%) patients 8.5 ± 2.5 years (range 4 to 11) before PTSMA; 5 (6%) patients had not responded to previous DDD pacemaker implantation. Further detailed baseline data are displayed in Table 1.

Written informed consent was given before intervention, after intensive discussion of the various treatment options, with special attention to the novelty of PTSMA and the absence of long-term experience.

Preinterventional Studies
The study protocol with preinterventional and postinterventional workup is shown in Table 2. Echocardiographic measurements were obtained following ASE guidelines. LVOTG was assessed by continuous wave Doppler echocardiography (CWDE) at rest and at Valsalva maneuver. Mitral regurgitation and systolic anterior move-
TABLE 1. Clinical Baseline Data of 91 Patients With Symptomatic HOCM

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean±SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>54.1±15.5</td>
<td>16–88</td>
</tr>
<tr>
<td>Time since HOCM diagnosis, y</td>
<td>6.1±4.8</td>
<td>0.1–27.0</td>
</tr>
<tr>
<td>Body height, cm</td>
<td>169±11</td>
<td>147–195</td>
</tr>
<tr>
<td>Body weight, kg</td>
<td>76±15</td>
<td>42–126</td>
</tr>
<tr>
<td>Body mass index kg/m²</td>
<td>26.7±3.8</td>
<td>17–40</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td>2.8±0.6</td>
<td>2–4</td>
</tr>
</tbody>
</table>

TABLE 2. Study Protocol

<table>
<thead>
<tr>
<th>PTSMA After PTSMA</th>
<th>3 Months After PTSMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>History</td>
</tr>
<tr>
<td>Clinical status</td>
<td>Clinical status</td>
</tr>
<tr>
<td>ECG</td>
<td>ECG</td>
</tr>
<tr>
<td>Lung function test</td>
<td></td>
</tr>
<tr>
<td>Bicycle stress test</td>
<td></td>
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<tr>
<td>Pulmonary artery catheter</td>
<td></td>
</tr>
<tr>
<td>Echocardiography</td>
<td>Echocardiography</td>
</tr>
<tr>
<td>Diagnostic cardiac catheterization</td>
<td></td>
</tr>
</tbody>
</table>

Intervention

Before PTSMA, a diagnostic left heart catheterization was performed. The first 30 patients underwent PTSMA as previously described by Sigwart and by our own group with measurement of the LV inflow tract pressure by a Brockenbrough catheter introduced transseptally. From patient 31 onward, LV inflow tract pressure was measured with a 5F multipurpose or modified –4 sideholes only at the pigtail segment—5F pigtail catheter (Cordis) by a retrograde approach and with careful placement of the catheter tip to exclude entrapment. Aortic pressure was monitored by the percutaneous transmural coronary angioplasty guiding catheter after exclusion of an aortic valve gradient. The LVOTG was assessed at rest and after a premature ventricular beat. All patients received a 4F transfemoral pacemaker lead (Cordis) introduced into the right ventricular apical region and 10 000 IU IV heparin as antithrombotic prophylaxis.

The presumed target vessel was then selectively intubated with a 0.014-inch guide wire through a 7F or 8F percutaneous transluminal coronary angioplasty guiding catheter (Figure 1). A short, slightly oversized over-the-wire balloon (1.5 to 3.0 mm) was introduced and inflated, and the distal vessel bed was contrasted. After exclusion of dye reflux into the left anterior descending coronary artery (LAD), and if proobatory vessel closure by the inflated balloon had resulted in significant LVOTG reduction, the alcohol was injected in fractions of ≥1 mL in the first 30 patients.

In the remaining patients, additional intraprocedural MCE was performed to determine the target vessel. After verification of the correct balloon position and the hemodynamic effect of probatory balloon occlusion, 1 to 2 mL of the echo contrast agent (Levovist, concentration 350 mg/mL; Schering) was injected through the inflated balloon catheter under continuous transthoracic echocardiographic imaging. Alcohol was given only when the area of maximum flow acceleration, that is, gradient formation, and opacified septal mismatch, the diagnostic coronary angiogram was revised and the procedure repeated with another septal branch.

After definitive identification of the target vessel, patients received 0.15 to 0.3 mg IV buprenorphine just before the alcohol injection. The balloon remained inflated for 10 minutes after the alcohol administration to enhance tissue contact and to exclude alcohol reflux into the LAD. Finally, hemodynamic measurements were repeated.

Follow-Up Studies

All patients were monitored on the coronary care unit (CCU) for ≥48 hours. The vascular sheaths were removed after normalization of the coagulation measurements. Cardiac enzymes and ECG controls were done every 4 hours; echo and CWDE studies were done once per day. Before discharge, noninvasive follow-up was performed as shown in Table 2. If possible, medical treatment was continued with a cardioselective β-blocker or, in case of contraindications, with verapamil. After 3 months, all patients underwent clinical and noninvasive follow-up: 70 (80%) patients had additional recatheterization.

Statistics

Patient data were collected in a relational database (Filemaker 3.0, Claris Corp) and analyzed with the Statview 4.5 (Abacus Concepts) and Winstat 3.1 (Kalma Co Inc) statistical software packages. Results of continuous variables are displayed as mean±SD. Student’s t tests for paired and unpaired samples were used for group comparisons (baseline measurements, in-hospital follow-up, and 3-month follow-up as well as comparisons between the patients with and those without MCE for target vessel selection). Frequency distributions were assessed with the χ² test. ANOVA was performed when comparing more than 2 groups. Differences were considered significant if the 2-tailed P value was <0.05.

Results

Technical Aspects

PTSMA was completed in 89 patients, with a mean procedural time of 98±33 minutes (46 to 190) and a fluoroscopy time of 14±10 minutes (3 to 45). The amount of contrast dye was 262±127 mL (50 to 680), the amount of alcohol injected 3.4±1.7 mL (1.5 to 11), with 1.1±0.3 target vessels occlud-
Since introduction of MCE, only 1 vessel was occluded per session. In 5 (8.5%) out of 59 patients, the target vessel was found originating from a diagonal or intermediate branch of the left coronary artery. Despite MCE introduction, intervention time decreased with growing experience (Table 4). In 2 patients, alcohol ablation was not performed: The septal branch could not be reached with a guide wire in 1 patient; in another patient with repeat PTSMA, a stable balloon position could not be achieved. These patients underwent surgery later on.

Complications

In the Catheterization Laboratory
Chest pain induced by alcohol injection could be managed by central analgesics in all patients. In 1 patient with significant pericardial effusion induced by a penetrating pacer lead, emergency percutaneous pericardiocentesis was performed and PTSMA successfully continued. Complete heart block developed in a total of 62 (70%) patients. At CCU admission, complete heart block was still present in 31 patients (35%). All patients left the catheterization laboratory in stable hemodynamic condition.

During In-Hospital Course
After failed compression therapy, 1 patient needed surgery for a false aneurysm at the puncture site. Two patients required blood transfusions because of groin hematoma. Pericardial effusion without hemodynamic compromise was seen in 5 patients. In 3 patients with pulmonary comorbidity, respiratory problems developed, mechanical ventilation being necessary in 1 of these. In another patient with severe pulmonary obstructive disease, exacerbation and intensive (topical and intravenous) treatment with β-agonists was associated with the first death of our series caused by intractable ventricular fibrillation 9 days after a successful intervention. A femoral vein thrombosis, probably induced by the indwelling pacemaker lead, leading to fulminant pulmonary embolism and refractory shock 36 hours after PTSMA, was diagnosed by postmortem examination only.

Ten (11%) patients required permanent DDD pacemaker implantation: because of sustained trifascicular block in 4 and intermittent conduction problems in 6 patients, in 1 of these 11 days after intervention. Introduction of MCE was associated with a higher rate of rapid recovery of the atrioventricular (AV) conduction and a reduction of the pacemaker implantation rate from 17% to 7% (Table 4).

Until Follow-Up
One patient underwent thrombectomy of a symptomatic femoral vein thrombosis 3 weeks after discharge and a normal postoperative course. Clinically relevant rhythm dis-

Figure 1. Coronary angiograms. A, Identification of target vessel in right anterior oblique view (arrows). B, Balloon inflation in proximal part of target vessel. C, Injection of contrast dye to define perfusion area and to exclude reflux into LAD. D, Final visualization of vessel stump after completed PTSMA.
turbances or other HOCM complications were not seen in the follow-up period after PTSMA.

Cardiac Enzyme Changes
The creatine kinase (CK) peak was $676 \pm 347$ U/L (201 to 1940) after 10.6 $\pm$ 4.9 hours (4 to 24) with an MB fraction of $85 \pm 49$ U/L (18 to 281). The GOT peak was $110 \pm 64$ U/L (21 to 446). Maximum enzyme rise correlated with the amount of alcohol injected, not with the hemodynamic efficacy of PTSMA. The CK-MB peak was significantly lower in the patients with MCE for target vessel selection (Table 4).

ECG Changes
In 52 (58%) patients, a new bundle branch block was present after PTSMA. The right branch was predominantly affected (43%). After 3 months, 9 of these bundle branch blocks had disappeared. Two patients with DDD pacemaker implantation after PTSMA showed complete LVOTG elimination and stable recovery of the AV conduction at follow-up.

Symptoms and Exercise Tolerance
A number of patients reported symptomatic improvement in the catheterization laboratory. After 3 months, 82 (94%) out of the returning 87 patients reported significant improvement of symptoms and exercise tolerance, with a mean NYHA class reduction from $2.8 \pm 0.6$ to $1.1 \pm 1.0$ and a maximum tolerated workload improvement from $87.5 \pm 59.4$ to $110.3 \pm 9.5$ W ($P<0.05$). Complete elimination of symptoms was reported by 29 (33%) patients. Clinical improvement was more frequent and more pronounced in the patients with an MCE-guided intervention (Table 4).

Hemodynamic Effects: LVOT Obstruction
LVOTG at rest was reduced from $73.8 \pm 35.4$ to $36.4 \pm 29.3$ mm Hg ($P<0.001$) after probatory balloon occlusion and to $16.6 \pm 18.1$ mm Hg ($P<0.0001$) after alcohol injection. Postextrasystolic gradients and the obstruction-associated phenomena of SAM and mitral regurgitation were reduced in a parallel way (Table 3). In 75 (84%) patients, a short-term hemodynamic success as defined by a complete elimination of LVOTG (Figure 3) or a reduction of >50% was achieved in 21 (70%) of 30 patients without and in 54 (92%) of 59 patients with intraprocedural echo monitoring ($P<0.01$).

After 3 months, in 59 (84%) of 70 patients with recatheterization, sustained LVOTG reduction was seen; in 30 (43%) of these the LVOTG showed further regression as compared with the acute results. Hemodynamic mid-term success rate (>50% LVOTG reduction) again was higher in the MCE group (45 of 48 [94%] vs 14 of 22 [64%]; $P<0.01$). Further on, the rate of LVOTG recurrence was lower (1 of 48 [2%] vs 5 of 22 [23%]; $P<0.05$; Table 4). The CWDE measurements of the LVOTG as well as the echo data concerning SAM and mitral regurgitation, available in all 87 patients, paralleled the invasive data (Table 3). In all 4 (5%) patients who underwent repeat PTSMA after 3 months, this led to complete LVOTG elimination.

Systolic and Diastolic LV Function and LV Hypertrophy
Until discharge, all patients developed a circumscript akinesia of the subaortic septum. In the patients with MCE guiding, this area matched with the region opacified intraoperatively (Figure 2). Global LV function remained unchanged. Late diastolic mitral inflow was accentuated together with a prolonged deceleration time of the early mitral inflow wave (Table 3). Until follow-up, left atrial diameter significantly decreased as well as LV end-diastolic pressure and mean pulmonary artery pressures. Both septal and LV posterior wall thickness showed significant regression (Table 3).
Obstruction of LV outflow, diastolic dysfunction, and rhythm disturbances including sudden cardiac death are the main problems in patients with HOCM. Medical therapy, surgical myectomy, and AV sequential pacing predominantly aim for LVOTG reduction. Although the effect of medical treatment and AV sequential pacing on symptoms and LVOT obstruction is often limited, the good symptomatic and hemodynamic results of surgery are overshadowed by considerable perioperative morbidity and mortality rates. After successful surgery, however, patients also tend to have improved markers of diastolic LV function and a favorable prognosis.

### TABLE 3. LV Outflow Tract Obstruction, LV Systolic and Diastolic Function, and LV Hypertrophy

<table>
<thead>
<tr>
<th>Variable/Method</th>
<th>Baseline</th>
<th>Acute Result</th>
<th>3 Months After PTSMA</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Invasive study</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVOT gradient, mm Hg</td>
<td>73.8±35.4</td>
<td>16.6±18.1</td>
<td>14.6±25.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>At rest</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postextrasystole</td>
<td>149.3±42.5</td>
<td>61.9±43.0</td>
<td>49.1±48.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LV end-diastolic pressure, mm Hg</td>
<td>21.9±7.6</td>
<td>17.4±6.9</td>
<td>14.6±25.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>79.2±9.2</td>
<td>76.6±11.3</td>
<td>14.6±25.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pulmonary artery mean pressure, mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>At rest</td>
<td>19.2±6.7</td>
<td>16.9±5.8</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>With maximum exercise</td>
<td>45.0±11.0</td>
<td>39.6±9.6</td>
<td>&lt;0.05</td>
<td></td>
</tr>
<tr>
<td><strong>M-mode echocardiography</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left atrial diameter, mm</td>
<td>47.0±7.3</td>
<td>45.3±6.4</td>
<td>44.4±7.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV end-diastolic diameter, mm</td>
<td>47.3±5.3</td>
<td>45.7±5.3</td>
<td>48.6±5.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fractional shortening, %</td>
<td>41.8±6.8</td>
<td>39.8±7.7</td>
<td>38.2±6.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Septal thickness, mm</td>
<td>21.1±3.3</td>
<td>17.1±3.6</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Posterior wall thickness, mm</td>
<td>13.9±2.6</td>
<td>13.1±2.1</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>SAM, grade</td>
<td>2.4±0.7</td>
<td>1.3±0.7</td>
<td>&lt;0.0001</td>
<td>0.8±0.8</td>
</tr>
<tr>
<td><strong>Pulsed wave, continuous wave, or color Doppler</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV outflow tract gradient, mm Hg</td>
<td>59.6±38.5</td>
<td>29.1±25.3</td>
<td>17.8±17.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Mitral regurgitation, grade</td>
<td>143.6±53.8</td>
<td>73.1±49.2</td>
<td>54.5±44.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>e/a Ratio of mitral inflow</td>
<td>0.8±0.5</td>
<td>0.6±0.5</td>
<td>0.4±0.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Deceleration time: e wave, ms</td>
<td>1.17±0.38</td>
<td>0.96±0.31</td>
<td>0.98±0.31</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>296±116</td>
<td>318±106</td>
<td>392±157</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

All P values compared with baseline measurements.

**Discussion**

Obstruction of LV outflow, diastolic dysfunction, and rhythm disturbances including sudden cardiac death are the main problems in patients with HOCM. Medical therapy, surgical myectomy, and AV sequential pacing predominantly aim for LVOTG reduction. Although the effect of medical treatment and AV sequential pacing on symptoms and LVOTG obstruction is often limited, the good symptomatic and hemodynamic results of surgery are overshadowed by considerable perioperative morbidity and mortality rates. After successful surgery, however, patients also tend to have improved markers of diastolic LV function and a favorable prognosis.

**Figure 3.** Hemodynamic treatment result of PTSMA. Pressure curves of LV inflow tract (LVIT) and aorta (Ao) before (a) and after (b) intervention with complete elimination of LVOTG and reduction of LV end-diastolic pressure.
Forty years after the first descriptions, interventional cardiology is now developing a new treatment option for HOCM. The induction of a limited “therapeutic” infarction within the hypertrophied septal myocardium leads to localized thinning and contractile dysfunction, expands the LV outflow tract, and thus reduces LVOTG and depending symptoms. The first preliminary studies leading to this catheter-based imitation of surgical myectomy date from the 1980s. In 1994, probatory balloon occlusion of septal branches of the left coronary artery, leading to transient ischemia-induced LVOTG reduction, was reported. In 1995, Sigwart published the first report on definitive alcohol-induced septal reduction in 3 severely symptomatic patients. The first small series showed promising results concerning symptoms and LVOTG reduction, with acceptable complication rates.

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Our experience with 91 patients is in line with these reports. Baseline data show that the clinical spectrum of HOCM in terms of disease severity and complications is well represented in our patient group. Notably, patients with prior surgical myectomy and DDD pacemaker implantation could also be treated effectively. The previous experience concerning ongoing LVOTG reduction and absence of complications until mid-term follow-up was confirmed as well as our strategy of awaiting the remodeling process before considering a repeat PTSMA or other nonmedical treatment options.

An important improvement of the new method in our opinion has been gained by the integration of echocardiographic monitoring. Analysis of the first patient had shown that probatory balloon occlusion of the presumed target vessel did not reliably predict the definitive treatment result. In some of these patients, up to 3 vessels had to be occluded for satisfactory LVOTG reduction. In view of the cases of HOCM with spontaneous LV dilatation and LVOTG reduction, carrying a particularly bad prognosis, we tried to optimize the amount of ablated myocardium.

With intracoronary injection of the echo contrast agent, opacification of the strategic septal area involved in LVOTG formation and thus a definition of the extent and localization of the induced necrosis was possible in all cases.

In the future, the reduction of sustained AV conduction disturbances may also translate into a reduced rate of pacemaker implantations. Currently, the pacemaker implantation rate after MCE-guided PTSMA (7%) is comparable to that associated with surgical myectomy.

As far as diastolic and global systolic LV function is concerned, our hemodynamic follow-up data demonstrate improvement of LV end-diastolic pressure as well as a reduction of pulmonary artery pressure both at rest and with exercise. The changes of the mitral inflow pattern should therefore not be interpreted as an aggravation of diastolic...
dysfunction. In contrast, it seems more likely that a preexisting pseudonormalization of the mitral inflow pattern regressed as a consequence of a reduced transmural driving pressure. The reduction of left atrial dimensions lends further support to this interpretation.25

Surprisingly, not only septal hypertrophy decreased as a consequence of the therapeutic infarction, but also LV posterior wall thickness. This may be due to relief of the pressure overload and may also have influenced diastolic function parameters.

Taking into account the fact that a learning curve is still present in this series, PTSMA seems to be a quite safe procedure. Complications, however, must be considered as previously reported, that is, thoracic discomfort, AV conduction disturbances, ventricular dysrhythmias, and death.1,3,6,7

The first death,6 associated with severe chronic obstructive pulmonary disease, led us to include routine preinterventional assessment of lung function in the study protocol and to prolong CCU monitoring in affected patients. The second death from pulmonary thromboembolism underlines the importance of timely sheath removal from the femoral vessels. In the case of sustained AV conduction disturbances, a transjugular pacing lead should be inserted. Ventricular septal defects and cerebral events, also possible from a theoretic point of view, have not been observed to date.

Limitations
Several limitations must be considered in this report. We did not perform invasive electrophysiological studies in our patients to assess the risk of ventricular tachyarrhythmias or AV conduction disturbances. The echo indexes used are rough markers of diastolic LV function. Considering the effect of MCE on target vessel selection and treatment results, learning curve effects cannot be ruled out. Finally, the patients were not randomly assigned to interventional treatment but by individual indication. A comparison with established forms of nonmedical treatment of HOCM is thus not possible.

Conclusions
PTSMA is a promising interventional treatment modality for patients with HOCM who do not show satisfactory response to medical therapy. Without the operative trauma including cardiac-pulmonary bypass, in >90% of the patients elimination or a substantial reduction of the LVOT gradient is possible. Intraprocedural echocardiographic monitoring has proven to be an important improvement of the new method. Future work should be directed at reducing the rate of AV conduction system lesions and definitive pacemaker implantations on the one hand and at directed at reducing the rate of AV conduction system lesions and definitive pacemaker implantations on the other hand. Furthermore, the long-term effect of PTSMA on global systolic and diastolic function and on prognosis remains to be assessed. A prospective registry of all interventionally treated patients with HOCM will be very helpful in clarifying these issues. The definitive value of PTSMA as compared with other treatment modalities remains to be studied in a prospective, randomized trial.

References


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