

Clinical Research

Echocardiography-Guided Percutaneous Septal Ablation in Patients with Hypertrophic Obstructive Cardiomyopathy: One Year Follow-Up

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Introduction: Percutaneous transluminal septal myocardial ablation (PTSMA) by alcohol-induced septal branch occlusion has been introduced as a new treatment option in symptomatic patients with hypertrophic obstructive cardiomyopathy. Echocardiographic monitoring of the procedure with injection of echo-contrast agent in the balloon-occluded target septal branch through the central lumen of the balloon catheter has resulted in improvement of acute results. We report on the late results after 1-year follow-up.

Methods: One hundred consecutive symptomatic (NYHA class 2.8 ± 0.6) patients underwent percutaneous septal ablation with echocardiographic monitoring. Two of the patients had prior surgical myectomy, 5 had prior DDD pacemaker implantation and 2 had non-echo-guided septal ablation, all of which were haemodynamically and clinically unsuccessful. Three patients with LAD lesions underwent PTSMA and PTCA in one session. An additional 3 patients underwent PTCA of an RCA lesion and PTSMA after 6 months due to ongoing symptoms, despite a good angioplasty follow-up result. All patients had clinical and non-invasive (echocardiography, exercise stress test) follow-up after 3 months and 1 year.

Results: The intervention was performed in 1 septal branch in each of the 100 patients by injection of 3.2 ± 0.7 ml alcohol. In 3 patients we had to change the branch after echocardiography had shown misplacement of echo-contrast. In an additional 5 patients, septal branches originating atypically from a diagonal branch could be identified as target vessels. Acute reduction of the left ventricular outflow tract gradient was achieved in 99 patients from 76 ± 37 to 19 ± 21 mm Hg at rest, from 104 ± 34 to 43 ± 31 mm Hg during Valsalva manoeuvre, and from 146 ± 45 to 59 ± 42 mm Hg post extrasystole ($p < 0.0001$ for each). Mean CK increase was 570 ± 236 U/l. One patient died at day 2 due to massive pulmonary embolism following deep venous thrombosis. Permanent DDD pacing due to post-interventional complete atrioventricular block was required in 8 patients. No other patient died during follow-up. All 99 living patients showed clinical improvement in NYHA class: 1.4 ± 0.6 after 3 months and 1.5 ± 0.6 after 1 year ($p < 0.0001$, each versus baseline). Exercise capacity also improved from 90 ± 49 W before the procedure to 114 ± 42 W at 3 months and 121 ± 37 W at 1 year ($p < 0.0001$, each versus baseline). Twenty-two out of 23 patients with an abnormal exercise blood pressure response before PTSMA showed normalisation of this finding after 1 year, while only 1 of 31 patients with exercise-induced syncope before PTSMA reported this symptom during the one-year follow-up period. Echocardiography revealed thinning of the ablated septal area, similar to the post myectomy result. No septal perforations were observed. Significant reduction of the left ventricular posterior wall thickness was also identified after 3 months (from 13.9 ± 2.5 mm to 12.8 ± 2.0 mm, $p < 0.0001$) with ongoing significant decrease after 1 year (12.0 ± 1.8 mm, $p < 0.0001$ versus baseline and 3 months). Left ventricular end-diastolic dimensions showed only a slight increase from 45.6 ± 5.5 mm to 46.6 ± 5.5 mm at 3 months ($p < 0.05$) with no further significant increase at 1 year, whereas left atrial dimension decreased significantly. Furthermore, Doppler-estimated mitral regurgitation as well as the grade of SAM significantly decreased ($p < 0.0001$ versus baseline, at both 3 months and 1 year).

Conclusion: Percutaneous septal ablation is an effective treatment of symptomatic patients with hypertrophic obstructive cardiomyopathy. Left ventricular remodelling after the alcohol induced therapeutic septal infarction resulted in improvement of acute gradient, reduction during one-year follow-up with ongoing symptomatic and objective improvement and without significant complications and side effects.

Hypertrophic obstructive cardiomyopathy (HOCM) is defined as a primary, frequently familial and genetically determined condition characterized by myocardial hypertrophy and dynamic left ventricular outflow tract obstruction¹⁻³. It is also accompanied by diastolic dysfunction of varying degree. In addition to hypertrophy of the ventricular myocardium, primarily the interventricular septum, morphologic changes of the papillary muscles and mitral valve leaflets may also be observed, imposing different treatment options²⁻⁴.

Surgical myotomy/myectomy has traditionally been the treatment of choice for drug-refractory symptomatic patients with outflow tract obstruction⁵⁻⁸. The high postoperative mortality of up to 10% has been reduced to <1-2% in experienced centres. Recently, the potential therapeutic options for symptomatic patients have been dramatically changed by the introduction of dual-chamber pacemaker implantation⁹⁻¹¹ and percutaneous transluminal septal myocardial ablation (PTSMA) by alcohol-induced septal branch occlusion¹²⁻¹⁸. We report on the acute and 1 year follow-up results of echocardiography-guided septal ablation¹⁹, which has been established as the standard technique of PTSMA.

Methods

The study population consisted of 100 consecutive patients (Table 1) with HOCM and drug-refractory symptoms (n=98) under optimal treatment or severe side effects from medical treatment (n=2) who were treated with echocardiography-guided PTSMA. The centres in which this therapeutic modality was performed are referral centres for patients with HOCM and indications for invasive therapy. The patients of the study were treated consecutively in a time period of 27 months and were selected from a cohort of 205 patients with HOCM, who had been referred for further evaluation and management. The indications for PTSMA were symptoms of dyspnoea NYHA III-IV and/or angina CCS III-IV with a high outflow tract gradient (≥ 50 mmHg at rest, or ≥ 30 mmHg at rest and ≥ 100 mmHg during Valsalva manoeuvre). Patients with less severe symptoms were only treated if they had documented individual risk factors for sudden cardiac death, such as abnormal blood pressure response on exercise, exercise-induced syncope, paroxysmal atrial fibrillation or supraventricular tachycardia, and left atrial enlargement. Two of the patients had had previous surgical

Table 1: Clinical baseline data in 100 consecutive patients with symptomatic hypertrophic obstructive cardiomyopathy and echocardiographic guided percutaneous transluminal septal myocardial ablation (PTSMA).

Men (n)	50
Age (years)	52.7±15.7
Family history (n)	30
NYHA class	2.8±0.6
CCS class	1.9±1.3
Syncope (n)	31
History of disease (years)	5.8±5.5
Prior myectomy (n)	2
Prior DDD-pacemaker (n)	5
Prior, non-echo-guided PTSMA (n)	2
<u>Drug treatment before PTSMA:</u>	
Beta-blocker (n)	36
Verapamil (n)	62
Atrial fibrillation (n)	4
Prior cardiac decompensation (n)	13
<u>Holter monitoring:</u>	
Non-sustained ventricular tachycardia (n)	19
Supraventricular tachycardia (n)	24
Left atrial dimension ≥ 45 mm (n)	60
Abnormal exercise blood pressure response (n)	23

NYHA: New York Heart Association, CCS: Canadian Cardiological Society.

myectomy, 5 patients had had DDD pacemaker implantation, while 2 patients had had non-echo-guided septal ablation. Patients with concomitant coronary artery disease were included only if they possessed focal single vessel disease amenable to angioplasty. Three patients with LAD lesions underwent PTSMA and PTCA in one session. An additional 3 patients underwent PTCA of an RCA lesion initially and percutaneous septal ablation after 6 months, because of ongoing symptoms despite a good angioplasty follow-up result. Patients with concomitant cardiac disease requiring surgery, e.g. extensive coronary artery disease, valvular disease, and marked morphologic abnormalities of the mitral valve or the papillary muscles responsible for gradient formation or mitral regurgitation, were excluded. Furthermore, hypertrophic cardiomyopathy without a significant resting or provokable outflow tract gradient was deemed a clear contraindication for PTSMA. Moreover, planned ablation was not performed when echocardiographic identification of a target septal branch failed, or upon observing opacification of any cardiac structure other than the target septal area by echo-contrast medium.

Echocardiographic measurements of cardiac anatomy and function were obtained following ASE

guidelines²⁰. Left ventricular outflow tract gradient (LVOTG) was measured by continuous wave Doppler echocardiography at rest and during Valsalva manoeuvre. Mitral regurgitation and systolic anterior motion (SAM) of the mitral valve apparatus were graded semiquantitatively (0=absent to 3=severe/with complete septal contact)²¹. Symptom-limited bicycle exercise tests were performed in upright position, starting with 25 Watts and increasing by 25 Watts every 2 minutes, in patients with NYHA functional class <IV and left ventricular outflow tract (LVOT) resting gradients <100 mm Hg. Abnormal blood pressure response was defined as systolic blood pressure decrease or increase <20 mm Hg during exercise. All patients received 24-hour Holter monitoring before and 3 months after PTSMA.

Written informed consent was given prior to intervention after extensive discussion of the various treatment options, with special attention to the novelty of PTSMA and the absence of long-term experience. In patients with unknown coronary anatomy the PTSMA procedure was directly preceded by coronary angiography. In all patients without prior permanent pacemaker implantation, a 4F temporary pacemaker lead was inserted in the apex of the right ventricle via the left femoral vein. A 5F specially designed pigtail catheter with a hole only at its distal end (Cordis Europa, Roden, The Netherlands) was inserted via the left femoral artery and positioned in the left ventricular outflow tract. An aortic valve gradient was excluded after placement of a 7F PTCA guiding catheter in the ascending aorta via the right femoral artery. After advancing the pigtail catheter to the ventricular apex, the LVOTG was measured by simultaneous pressure recordings at rest, following an extrasystolic contraction and during Valsalva manoeuvre. The extrasystolic contraction was easily produced by slightly stirring the pigtail catheter. The estimated target septal branch presumed to be responsible for the blood supply to the hypertrophied septal area involved in obstruction was next identified by coronary angiography (Figure 1a). Heparin (10.000 IU) was administered intravenously to help avoid thromboembolic complications, and analgesic medication (10 mg morphine iv) was given before alcohol injection. The target septal branch was catheterised with a 0.014" angioplasty guidewire, followed by a short (10 mm) over-the-wire balloon catheter (1.5-2.5 mm Concerto[®], Occam, Eindhoven, Netherlands). The balloon selected was slightly oversized compared to the septal branch di-

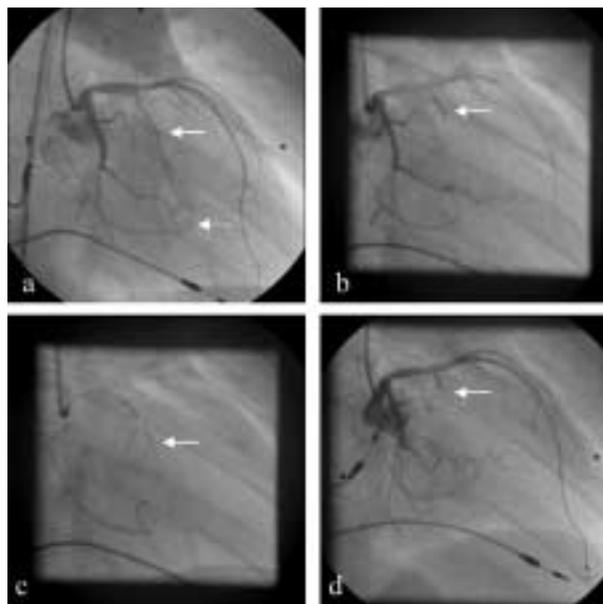


Figure 1. Left coronary angiography shows the target septal branch (arrow) in RAO (a). Occlusion of the septal branch after balloon inflation (b). Injection of dye through the central balloon lumen of the inflated balloon determines the supply area of the septal branch (c; arrow) and excludes leakage into the LAD. Final visualization of the occluded septal branch (arrow) after alcohol injection (d), (Dotted arrow: special pigtail catheter).

ameter estimated by on-line quantitative coronary angiography using the HICOR system (Siemens, Erlangen, Germany). Balloon inflation was performed using 6 bars pressure (Figure 1b).

After excluding reflux of contrast medium (and thus alcohol) into the LAD by injection of a small amount of radiological contrast dye (1-2 ml) through the guidewire lumen of the catheter (Figure 1c), we administered 1-2 ml of echo contrast medium (Levovist, Schering, Berlin, Germany; 350 mg/ml) through the central lumen of the balloon catheter during echocardiographic monitoring (Figure 2b). The echocardiographic criteria for correct vessel selection were 1) complete coverage of the echo-contrast marked septal area encompassing the area of SAM-septal contact and the colour Doppler estimated area of maximal flow acceleration and 2) no opacification of any other cardiac structure (Figure 3a, 3b). Only if these criteria were fulfilled, we slowly injected 2-4 ml of 96% alcohol in 1 ml increments through the central lumen of the over-the-wire balloon catheter. It should be mentioned that neither the target artery nor the alcohol dose was derived from the haemodynamic result after initial balloon occlu-

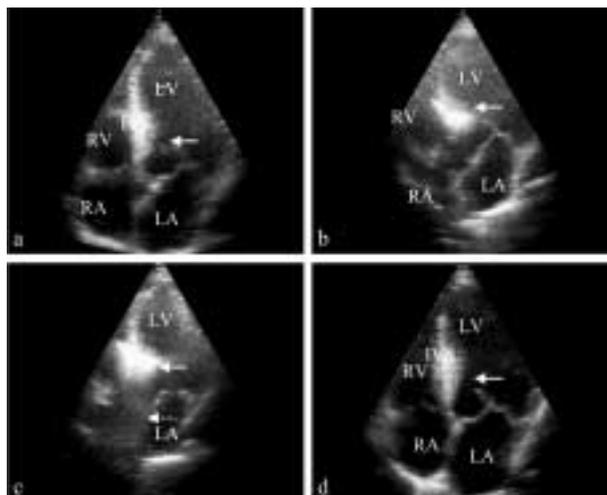


Figure 2. Echocardiographic study during PTSMA. Baseline view, with an angulated apical 4-chamber view (a), where the point of SAM-septal contact can be easily identified (arrow). Injection of echo-contrast agent (b) with satisfactory opacification of the target area (arrow). Visualisation after the alcohol injection (c), with the characteristic echo-shadow (dotted arrow). Final result after 1 year (d). (IVS: interventricular septum, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle).

sion. The amount of alcohol injected depends on the acute haemodynamic effect observed and the echocardiographically estimated size of the contrasted septal area.

Ten minutes after the last injection of alcohol, the balloon was deflated and removed during continuous aspiration through the central lumen of the catheter,

thus ensuring that no alcohol could spill into the LAD. After a final angiographic visualisation of the left coronary artery, to verify complete occlusion of the septal branch and exclude coronary lesions (Figure 1d), the haemodynamic measurements were repeated (Figure 4) and the intervention ended.

Statistics: Continuous variables are expressed as mean values \pm standard deviations. Frequencies are given for discrete variables. Comparison of continuous variables was carried out using the paired Student T-test. Frequencies were tested with the chi-square test. A p-value <0.05 was considered statistically significant. Statistical analysis was performed using Winstat 3.1 (Kalmia Co, Cambridge, MA).

Results

A) Technical and acute haemodynamic results

The intervention was performed in 1 septal branch in each of the 100 patients by injection of 3.2 ± 0.7 ml alcohol. In 3 patients we had to change the branch after echocardiography had shown misplacement of echo-contrast. In additional 5 patients, septal branches originating atypically from a diagonal branch could be identified as target vessels.

Haemodynamic measurements showed an acute gradient elimination at rest and at provocation in only 26 patients. 60 patients had gradient reductions $\geq 50\%$ compared to baseline values, whereas 13 patients had 20-49% reductions and one patient had no change in baseline gradient. Mean gradient was

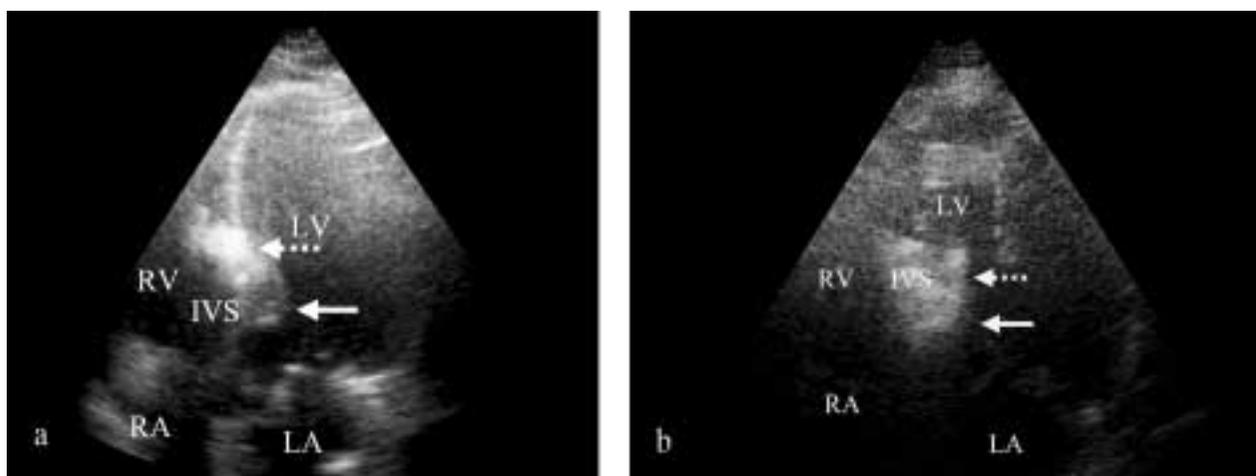


Figure 3. Angulated apical 4-chamber view (a) with echo-contrast opacification of the interventricular septum, albeit towards the right ventricular side and far from the point of SAM-septum contact (arrow). After change of the target vessel (b) optimal echo-contrast opacification of the subaortic septum with coverage (dotted arrow) of the SAM-septum contact point and 1 cm below (arrow). (IVS: interventricular septum, LA: left atrium, LV: left ventricle, RA: right atrium, RV: right ventricle).

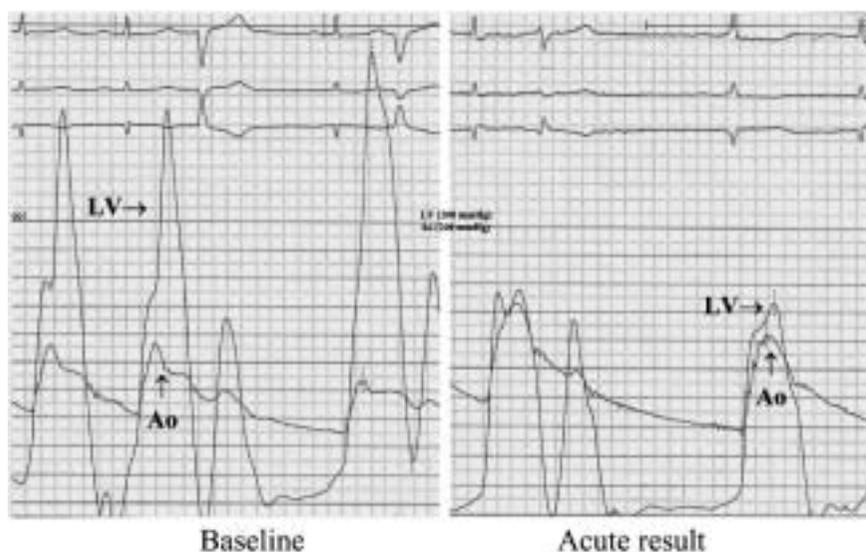


Figure 4. Optimal acute result after PTSMA with resting and post-extrasystolic elimination of left ventricular outflow tract gradient after alcohol-induced occlusion of the septal branch. (Ao: aortic pressure, LV: left ventricular pressure).

reduced from 76 ± 37 to 19 ± 21 mm Hg at rest, from 104 ± 34 to 43 ± 31 mm Hg during Valsalva manoeuvre, and from 146 ± 45 to 59 ± 42 mm Hg post extrasystole ($p < 0.0001$ for each comparison). Doppler studies showed further gradient reduction during follow-up (Figure 4), resulting in complete elimination of outflow tract gradient in 34 patients after 3 months and in 58 patients after 1 year ($p < 0.01$). After 1 year, an additional 37 patients had $\geq 50\%$ and 4 patients had $< 50\%$ gradient reduction compared to baseline values. Three patients developed mid-cavitary gradients after relief of outflow tract obstruction. Due to adequacy of symptomatic improvement reintervention was not required.

B) Complications

No patient developed complications at the time of the Levovist injection. After therapeutic septal infarction, the mean CK peak value was 571 ± 236 U/l, 10.4 ± 4.8 hours after alcohol injection.

Complete atrioventricular block developed during the procedure in 64 patients, with recovery of AV conduction in 42 patients (65.6%) in the catheterisation laboratory, while only 8 patients (12.5%) required permanent DDD pacemaker implantation. After dislocation of the atrial lead, re-operation was required in one of these 8 patients.

One 74-year-old woman experienced ventricular fibrillation on day 2, due to dislocation of the temporary pacer lead, and underwent successful defibrillation. One 64-year-old man died from pulmonary embolism on day 2. Autopsy detected a deep venous thrombosis at the venous insertion site of the tempo-

rary pacemaker lead. One patient required elective surgical repair of pseudoaneurysm at the arterial puncture site. Haemodynamically insignificant pericardial effusions were observed in 4 patients with complete disappearance within one week.

During follow-up there were no additional deaths. Within 3 months after PTSMA one patient experienced a period of atrial fibrillation, which was successfully cardioverted to sinus rhythm. At 1-year follow-up, 2 additional patients reported successfully treated periods of atrial fibrillation.

Two patients developed short periods of 2nd degree AV-block 4 and 8 months after PTSMA respectively, without syncope, and underwent DDD-pacemaker implantation.

Only 7 of 19 patients with asymptomatic non-sustained ventricular tachycardia (NSVT) before PTSMA had NSVT at 3 months follow-up Holter monitoring.

C) Clinical symptoms and exercise tests

At 3 months follow-up all patients reported clinical improvement, with decrease of NYHA functional class from 2.8 ± 0.6 to 1.4 ± 0.6 and CCS angina class from 1.9 ± 1.3 to 0.5 ± 0.8 ($p < 0.0001$ for each comparison). Ongoing significant symptomatic improvement was reported in 93 patients after 1 year to NYHA class 1.5 ± 0.6 and CCS class 0.5 ± 0.7 .

Only one of 31 patients with exercise-induced syncope before PTSMA reported this symptom during the one-year follow-up period.

Exercise testing showed increased exercise capacity from 90 ± 49 to 114 ± 42 Watts after 3 months ($p < 0.0001$) and to 121 ± 37 Watts after 1 year

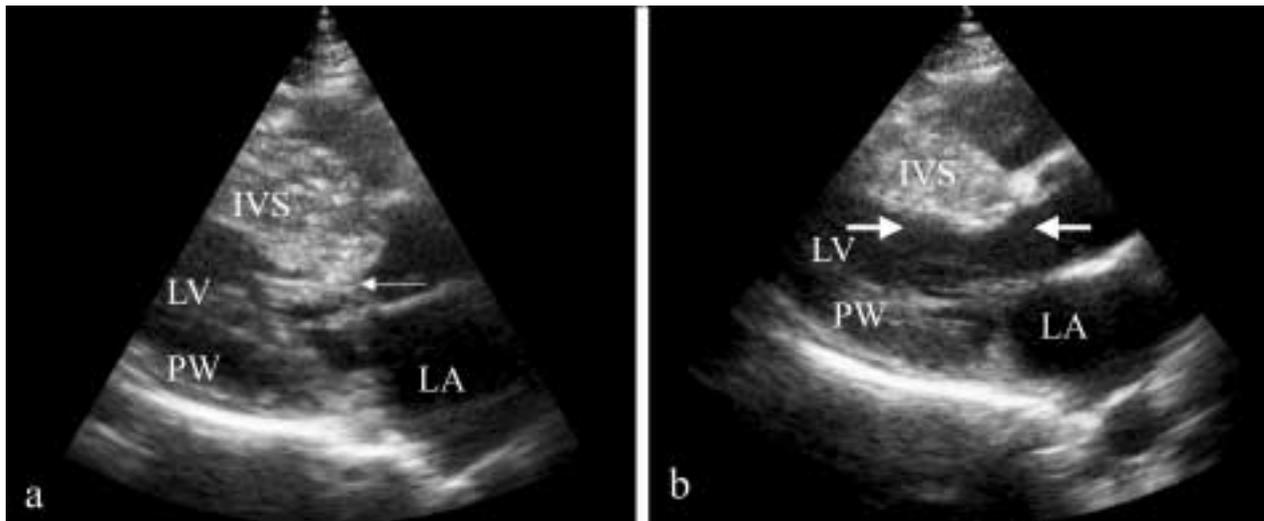


Figure 5. Parasternal long axis view at systole before (a) and 1 year after (b) PTSMA. Fig. a shows complete SAM-septal contact (arrow) at the friction area of the septum. One year after PTSMA widening of the left ventricular outflow tract and thinning of the subaortic septum are observed. (IVS: interventricular septum, LA: left atrium, LV: left ventricle, PW: left ventricular posterior wall).

($p < 0.0001$ vs. baseline; NS vs. 3 months). Increased exercise time from 6.1 ± 3.3 to 8.2 ± 3.4 minutes was documented after 3 months ($p < 0.0001$) and to 9.4 ± 2.9 minutes after 1 year ($p < 0.0001$ vs. baseline; $p < 0.001$ vs. 3 months). Twenty-two of 23 patients with an abnormal blood pressure response to exercise before PTSMA showed a normal result for this finding after 1 year.

D) Echocardiographic follow-up results

Echocardiographic studies frequently revealed thinning of the ablated septal area that resulted in myectomy-like subaortic channels (Figure 5). Septal perforations were not observed. Furthermore, significant reduction of the left ventricular posterior wall

thickness was observed after 3 months with ongoing significant decrease after 1 year (Table 2). Left ventricular end-diastolic dimensions showed only a slight increase, whereas left atrial dimension significantly decreased: 22 out of 60 patients with left atrial size >45 mm at baseline showed decrease below that value at 1 year follow-up ($p < 0.001$). Moreover, Doppler estimated mitral regurgitation as well as the grade of SAM significantly decreased (Table 2). These findings are in accordance with the observed ongoing gradient reduction after induced septal infarction.

Discussion

Since the early 1960s widening of the outflow tract by myotomy/myectomy has been a well-established

Table 2: Results of echocardiographic measurements at baseline and during follow-up in patients with percutaneous transluminal septal myocardial ablation (PTSMA).

Variable	Baseline	P Baseline vs. 3 months	3 months	P 3 months vs. 1 year	1 year	P 1 year vs. baseline
Left atrial diameter (mm)	47.5 ± 7.1	<0.0001	44.8 ± 6.2	NS	45.2 ± 7.0	<0.01
LV end-diastolic dimension (mm)	45.6 ± 5.5	<0.05	46.6 ± 5.5	NS	46.1 ± 5.8	NS
Septal thickness (mm)	21.1 ± 3.3	<0.0001	16.5 ± 3.9	0.01	15.1 ± 4.2	<0.0001
LV posterior wall thickness (mm)	13.9 ± 2.5	<0.0001	12.8 ± 2.0	<0.0001	12.0 ± 1.8	<0.0001
Mitral regurgitation (1+ to 3+)	1.0 ± 0.6	<0.0001	0.5 ± 0.5	NS	0.5 ± 0.5	<0.0001
SAM (1+ to 3+)	2.5 ± 0.7	<0.0001	0.9 ± 0.9	<0.01	0.6 ± 0.8	<0.0001

(LV = left ventricular; SAM = systolic anterior movement of the mitral valve).

surgical treatment for symptomatic, drug-refractory patients with HOCM⁵⁻⁸. Surgery substantially reduces the outflow tract gradient in 90% of patients and results in good clinical long-term improvement. Surgical results improved with perioperative echocardiographic guidance. The reported perioperative mortality of up to 10% in the early years could be reduced by growing surgical experience as evidenced by the <1-2% mortality shown in the large surgical series of Schulte⁵. An analysis of perioperative mortality identified older patients, patients requiring perioperative amiodarone therapy, patients in functional class IV, and patients with additional surgical procedures as high-risk patients⁵⁻⁷. Furthermore, there is a risk of about 5% of post-surgical complete AV-block requiring pacemaker implantation.

Recently, randomised trials of permanent dual-chamber pacing could show little impact on haemodynamic and clinical improvement in patients with hypertrophic cardiomyopathy and outflow tract gradient, with any benefits observed confined to elderly patients⁹⁻¹¹. Therefore, DDD-pacing cannot be recommended as the first choice treatment in drug-refractory patients with HOCM.

In the mid 1990's, percutaneous septal ablation was introduced as a new treatment option in patients with hypertrophic cardiomyopathy and left ventricular outflow tract obstruction¹². Alcohol induced septal branch occlusion resulted in a localised septal infarction with thinning and akinesia of the infarcted area and reduction of the left ventricular outflow tract gradient. Prior studies have shown a high rate of re-interventions and pacemaker implantations in haemodynamically guided septal ablation^{14,18}. In order to optimise the ablated septal area echocardiographic guidance of PTSMA was introduced¹⁹. This technique is now in common use after studies have shown improvement of acute haemodynamic results and, even more important, reduction of complications, e.g. the need of permanent pacemaker implantation¹⁵.

In this consecutive series of 100 patients treated with the echo-guided technique, the in-hospital complication rate was at least comparable to the known surgical results. In-hospital mortality occurs during post-interventional monitoring, an experience reported by other groups as well, with in-hospital mortality up to 4%¹⁸. Therefore, intensive monitoring is an important part of the treatment and should continue for at least 48 hours post procedure. The lower complication rate reported in this study could be

achieved by echo-guided change of the septal artery considered for ablation, which was necessary in 8% of the patients with identification of atypical target branches in 5 patients. In addition, the risk of acute mitral or tricuspid regurgitation could be avoided by changing the septal artery after identification of papillary muscle opacification by echo-contrast-medium injection (Figure 3a, 3b). The most important finding of this study was the lack of adverse clinical side effects such as ventricular fibrillation and clinically significant ventricular tachycardia during the post-hospital course. This finding needs to be confirmed by longer-term follow-up studies.

Besides the potential risk of life-threatening rhythm disorders, the potential for the development of left ventricular dilatation, which can sometimes be seen in the end-stage of this disease, was one of the most pointed out criticisms of this treatment. Therefore, an optimisation of the infarcted septal area was one of the reasons to introduce echocardiographic guidance. Comparisons with different techniques have documented a reduction of infarcted area with improvement of acute and short-term follow-up results¹⁵. This report confirms previous observations that percutaneous septal ablation does not result in significant left ventricular dilatation during mid-term follow-up¹⁶. Further long-term observations should verify this lack of significant left ventricular functional impairment. As in patients with surgical myectomy, previous studies^{14-15,18} have shown improvement of diastolic function, which is one of the reasons for symptomatic restriction.

The most important finding is the ongoing symptomatic improvement after PTSMA. In addition to the subjective reduction of functional class, we observed objective increases in mean exercise capacity, thus effectively contradicting prior criticisms²². Furthermore, the observed normalisation of abnormal blood pressure response during exercise in most patients may suggest that, at least in patients with the obstructive form of hypertrophic cardiomyopathy, gradients play an important role in the pathophysiology of this risk factor for sudden death²³. Further studies should investigate the influence of gradient reduction on the potential pathophysiologic mechanism of abnormal exercise blood pressure response as well as exercise-induced syncope.

The induced circumscribed septal infarction resulted in progressive reduction of outflow tract gradients over time, which should primarily be seen as a result of postinfarction remodelling, with further

shrinkage of the infarcted area demonstrated by the reduction of septal thickness in the infarcted area, often resulting in a myectomy like subaortic channel. Furthermore, reduction of left ventricular posterior wall thickness could be observed after both percutaneous and surgical septal reduction²⁴. It could be interpreted as the result of relief –or at least reduction– of pressure overload due to outflow tract obstruction, analogous to the case in patients with aortic valve stenosis after valve replacement.

Despite the lack of randomised studies of percutaneous and surgical septal ablation, it appears that both treatment options result in comparable haemodynamic and clinical results²⁵⁻²⁶. This report shows that in experienced centres PTSMA does not result in higher complication rates compared to published surgical results. Nevertheless, it must be pointed out that pre-interventional echocardiographic diagnosis with a clear definition of the site and mechanism of obstruction is of critical importance for the different therapeutic options of septal thickness reduction. Patients with structural mitral valve disease responsible for mitral regurgitation should be referred for surgery, as well as patients with additional coronary and non-coronary indications. A randomised trial of both ablation techniques seems to be difficult to design and perform because of the rarity of the disease and the relatively low mortality risk in adult patients.

In conclusion, PTSMA is a promising treatment option in symptomatic patients with hypertrophic obstructive cardiomyopathy. Echocardiographic monitoring with optimisation of the ablated area results in improvement of haemodynamic as well as clinical results and reduction of complications. However, limited knowledge of the long-term effects of PTSMA mandates careful patient selection. Furthermore, we should underscore the importance of restricting PTSMA to few centres with large interventional experience and knowledge of this particularly heterogeneous disease. In order to improve PTSMA results, the influence of the different ventricular and valvular morphologies as well as the influence of different genetic subtypes should be investigated in the future.

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