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ORIGINAL INVESTIGATIONS

Survival After Alcohol Septal Ablation in Patients With Hypertrophic Obstructive Cardiomyopathy

Angelika Batzner, MD,[a,b] Barbara Pfeiffer, MD,[b] Anna Neugebauer, MD,[b] Diaa Aicha, MD,[b] Christoph Blank, MD,[b] Hubert Seggewiss, MD[a,b]

ABSTRACT

BACKGROUND Alcohol-induced infarction for treatment of symptomatic hypertrophic obstructive cardiomyopathy (HOCM) was discussed as a risk factor for increased cardiac mortality during follow-up.

OBJECTIVES This study sought to report on long-term survival after echo-guided alcohol septal ablation (percutaneous transluminal septal myocardial ablation [PTSMA]) in symptomatic patients with HOCM.

METHODS Between May 2000 and June 2017, PTSMA with alcohol injection was performed in 952 patients (age 55.7 ± 14.9 years; 59.2% men; 73.3% New York Heart Association functional class III or IV; 50.3% syncope; 10.3% sudden cardiac death in family). Clinical follow-up after 6.0 ± 5.0 years was achieved in all patients.

RESULTS We injected 2.1 ± 0.4 cc of alcohol. Maximal creatine kinase rise was 872 ± 489 U/l. Two (0.21%) patients died 3 and 33 days after ablation. Permanent pacemaker was implanted in 100 (10.50%) patients. Echo gradients were acutely reduced from 63.9 ± 38.2 mm Hg to 33.6 ± 29.8 mm Hg at rest and from 104.6 ± 44.0 mm Hg to 56.5 ± 41.0 mm Hg at Valsalva ($p < 0.0001$, each). During follow-up, 164 (17.2%) patients underwent reablation due to the planned staged procedure, 18 (1.9%) patients underwent surgical myectomy, and 49 (5.10%) patients underwent cardioverter-defibrillator implantation. Seventy patients died: causes of death were identified as noncardiovascular in 50, stroke related in 6, and cardiac in 14 patients. Estimated 5-year survival was 95.8%, estimated 5-year survival free of cardiovascular events was 98.6%, and an estimated 5-year survival free of cardiac events was 98.9%. Corresponding values at 10 years were 88.3%, 96.5%, and 97.0%, and at 15 years were 79.7%, 92.3%, and 96.5%.

CONCLUSIONS In this study, PTSMA could be proofed as a safe procedure with ongoing symptomatic improvement and excellent long-term survival. Therefore, PTSMA is a reasonable alternative to surgical myectomy in HOCM. (J Am Coll Cardiol 2018;72:3087–94) © 2018 by the American College of Cardiology Foundation.

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Hypertrophic cardiomyopathy is the most common genetic heart disease with an estimated incidence between 0.2% and 0.6% (1). Left ventricular obstruction is an important pathophysiologic finding, with significant impact on symptoms and prognosis of the patients. For more than 50 years, surgical myectomy was the gold standard for symptomatic treatment in patients with high outflow tract gradients (2,3). More than 2 decades ago, alcohol septal ablation was introduced as a new treatment option in symptomatic patients with hypertrophic obstructive cardiomyopathy (HOCM) (4–6).

From the [a]Department of Kardiologie, Klinikum Würzburg Mitte, Standort Juliusspital, Würzburg, Germany; and the [b]Medizinische Klinik 1, Leopoldina-Krankenhaus, Schweinfurt, Germany. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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ABBREVIATIONS AND ACRONYMS

CI = confidence interval

HOCM = hypertrophic obstructive cardiomyopathy

ICD = implantable cardioverter-defibrillator

PTSMA = percutaneous transluminal septal myocardial ablation

SCD = sudden cardiac death

Echo-guided septal ablation (percutaneous transluminal septal myocardial ablation [PTSMA]) resulted in better acute and midterm follow-up results (7,8). But, from the very beginning concerns about long-term effect had been made and alcohol-induced infarction was discussed as risk for elevated cardiac mortality during long-term follow-up (9–11). We report on long-term mortality after PTSMA in a large single-center series.

METHODS

PATIENT SELECTION. Between May 2000 and June 2017, PTSMA was intended in 1,014 patients who met the following clinical and hemodynamic inclusion criteria: dyspnea or angina of New York Heart Association/Canadian Cardiovascular Society functional class III or IV, recurrent exercise-induced presyncope or syncope, and left ventricular gradients ≥30 mm Hg at rest or ≥50 mm Hg at provocation (Valsalva maneuver or post-extrasystolic potentiation). In 62 patients, a target septal branch could not be identified by echo guidance, resulting in abortion of the intervention without injection of alcohol. The study population includes the other 952 patients in whom alcohol was injected after identification of a target septal branch without echocardiographic detected potential misplacement.

All patients gave written informed consent before intervention after intensive discussion of the various treatment options. In the beginning of the study, special attention was drawn to the novelty of PTSMA and the absence of long-term experience.

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PTSMA TECHNIQUE. The PTSMA procedure has been described elsewhere (7,8). In brief, initial diagnostic catheterization excluded additional cardiac disease requiring cardiac surgery. Simultaneous measurement of left ventricular inflow tract and aortic pressures were mandatory for estimation of left ventricular outflow tract gradient at rest, Valsalva, and post-extrasystolic. Because of the risk of the development of complete heart block, a temporary pacemaker lead was inserted in patients without a permanent pacemaker or cardioverter-defibrillator (ICD) in place. The estimated target septal artery was wired with a standard percutaneous coronary intervention wire. An "over-the-wire" balloon was then advanced and inflated to avoid backflow of alcohol into the left anterior descending artery, thereby avoiding infarction of nontarget myocardial areas. After the guidewire was withdrawn echocardiographic contrast agent was injected through the balloon catheter with simultaneous transthoracic echocardiography. After stop of the production of Levovist (Schering, Berlin, Germany) in 2011 cooled agitated Gelafundin 4% (B. Braun, Melsungen, Germany) was used as contrast agent (12). Selective angiography of the target septal branch through the inflated balloon catheter documented the adequate sealing of the septal branch and excluded filling of any other coronary artery through septal collaterals (13). Up to 3 cc (1 cc/1 cm septal thickness) of absolute alcohol were then injected slowly through the central lumen of the balloon catheter under continuous fluoroscopic, hemodynamic, and electrocardiographic monitoring. Ten minutes after the last alcohol injection, the balloon catheter was withdrawn and a final angiogram documented complete occlusion of the septal branch and normal flow in the left anterior descending artery. Measurements of the outflow tract pressure gradient at rest and with provocation were repeated. Independent of the acute hemodynamic result we followed a staged ablation with occlusion of only 1 septal branch per session to await the hemodynamic effect of remodeling after induction of the therapeutic infarction. Exceptions with ablation of >1 branch were only planned and performed in 11 patients with long subaortic and additional midventricular obstruction. At least 48 to 72 h hemodynamic and electrocardiographic surveillance at the intensive care unit was performed. Cardiac enzymes were measured every 6 to 8 h. The temporary pacemaker was left in place until the next morning. The earliest 48 h after alcohol injection, a decision was made regarding implantation of a permanent pacemaker or a defibrillator if there was an increased risk of sudden cardiac death (SCD) according to the clinical risk stratification models (14,15). Hospital discharge was intended after a minimum of 7 days.

FOLLOW-UP. The patients underwent a first noninvasive follow-up control after 3 months. Afterward, annual cardiac examinations had been performed either in our institution or by the referring cardiologists. In 2017, a questionnaire was sent to the patients analyzing vital status, cardiac events in the meantime, and symptomatic change compared with the time before ablation. In doubtful answers and nonreturned questionnaires direct telephone contact to the patients (13.30%) or general practitioner or referring cardiologist or clinic (11.90%) was performed.

DEFINITIONS. Echocardiographic measurements were obtained following American Society of Echocardiography guidelines. Left ventricular outflow

TABLE 1 Baseline Characteristics of 952 With Alcohol Injection During Alcohol Septal Ablation (PTSMA)

Age, yrs	55.7 ± 14.9
Male	564 (59.2)
Height, cm	170.3 ± 13.8
Body weight, kg	82.8 ± 17.1
Dyspnea	
NYHA functional class III/IV	698 (73.3)
Angina pectoris	
CCS class II	354 (34.9)
CCS class III	166 (16.4)
Syncope	
Unexplained	93 (9.6)
Exercise-induced	388 (40.7)
Presyncope	341 (35.8)
Palpitations	293 (30.8)
Medication	
Beta-blocker	662 (69.5)
Verapamil	246 (25.8)
Disopyramide	13 (1.4)
Prior septal reduction	
PTSMA	44 (4.6)
Myectomy	20 (2.1)
Family history	
HCM positive	227 (23.8)
SCD positive	98 (10.3)
Prior pacemaker	52 (5.6)
Prior ICD	90 (9.7)
Cardiovascular diseases	
Hypertension	516 (54.3)
Coronary artery disease	123 (12.9)
Atrial fibrillation	133 (14.0)
Paroxysmal	113 (11.9)
Permanent	20 (2.1)
Echocardiography	
Maximal septal thickness, mm	21.0 ± 4.3
Subaortic septal thickness, mm	19.6 ± 3.9
Left ventricular posterior wall thickness, mm	13.2 ± 2.9
Left ventricular end-diastolic diameter, mm	43.4 ± 6.5
Left ventricular end-systolic diameter, mm	23.3 ± 5.7
Left atrial diameter, mm	46.1 ± 6.8
Doppler gradient, mm Hg	
Rest	63.9 ± 38.2
Valsalva	104.6 ± 44.0
Gradient at rest <30 mm Hg	237 (24.9)
ECG, including Holter	
Sinus rhythm	834 (88.2)
Left bundle branch block	73 (7.7)
Atrial fibrillation	20 (2.1)
Paroxysmal supraventricular tachycardia	113 (12.1)
Nonsustained ventricular tachycardia	98 (10.5)
Ergospirometry	
Exercise capacity, W	113.3 ± 44.7
Peak V_{O_2}, l/min/kg	19.8 ± 5.6
V_{O_2} at anaerobic threshold, l/min/kg	13.7 ± 4.2
Abnormal blood pressure response	105 (12.7)

Values are mean ± SD or n (%).
CCS = Canadian Cardiovascular Society; ECG = electrocardiography; HCM = hypertrophic cardiomyopathy; ICD = implantable cardioverter-defibrillator; NYHA = New York Heart Association; PTSMA = percutaneous transluminal septal myocardial ablation; SCD = sudden cardiac death; V_{O_2} = oxygen consumption.

tract gradients was assessed by continuous wave Doppler echocardiography at rest and at Valsalva maneuver. All-cause mortality was defined as death due to any cause. Cardiovascular death was defined as death related to any cardiovascular disease, including stroke. Cardiac death was defined as death related to any cardiac disease including SCD. SCD was defined as sudden and unexpected death within 1 h after a witnessed collapse in a previously stable patient or death that occurred during sleep.

STATISTICS. Data were collected in a SQL database. Statistical analysis were performed using Stata 15 (StataCorp, College Station, Texas). Continuous variables were expressed as mean ± SD. Frequencies were given for discrete variables. Comparison of continuous variables was carried out using the paired Student's *t*-test. A p value <0.05 was considered statistically significant. Survival estimations including 95% confidence interval (CI) were analyzed with Kaplan-Meier curves.

RESULTS

BASELINE CHARACTERISTICS. Between May 2000 and June 2017, 952 patients underwent first PTSMA with alcohol injection. This group included patients with insufficient results after prior alcohol septal ablation (n = 44 [4.6%]) and surgical myectomy (n = 20 [2.1%]) in other institutions (Table 1).

Mean age of the patients was 55.7 ± 14.9 years, with a range between 14.9 and 85.1 years, and 59.2% were men. Leading clinical symptoms were dyspnea (New York Heart Association functional class III or IV) in 73.3%, angina (Canadian Cardiovascular Society class III) in 16.4%, and exercise-induced syncope in 40.7% of the patients. At hospital admission, 96.7% were under antiobstructive medication. Significant coronary artery disease was diagnosed in 12.9% and atrial fibrillation in 14.0%. A permanent pacemaker had been implanted in 5.6% and an ICD in 9.7% before hospital admission for septal ablation.

Echocardiographically estimated maximal septal thickness was 21.0 ± 4.3 mm, left atrial diameter was 46.1 ± 6.8 mm, and left ventricular outflow tract gradient was 63.9 ± 38.2 mm Hg at rest and 104.6 ± 44.0 mm Hg at Valsalva maneuver.

TABLE 2 Hospital Course and Follow-Up Events of 952 Patients With PTSMA

Invasive gradient baseline, mm Hg	
Rest	51.3 ± 38.1
Valsalva	94.9 ± 38.6
Post-extrasystolic beat	134.8 ± 48.8
PTSMA	
Branches tested by contrast echo	1.2 ± 0.5
>1 branch tested	154 (16.2)
Branches treated with alcohol injection	1.01 ± 0.12
>1 branch treated*	11 (1.2)
Alcohol injected, cc	2.1 ± 0.4
Maximal CK rise, U/l	872 ± 489
ECG including Holter	
Sinus rhythm	804 (87.0)
Atrial fibrillation	40 (4.3)
Right bundle branch block	436 (46.1)
Left bundle branch block	137 (14.5)
Nonsustained ventricular tachycardia	77 (8.3)
Paroxysmal supraventricular tachycardia	107 (11.6)
Complications	
Cath lab	
Temporary AV block III	377 (39.6)
Nonsustained ventricular tachycardia	8 (0.9)
Left main dissection	1 (0.1)
Hospital course	
Permanent pacemaker implantation	100 (10.5)
Pericardial effusion	24 (2.5)
Tamponade requiring pericardiocentesis	5 (0.5)
Death	2 (0.2)
Echo Doppler gradients at discharge, mm Hg	
Rest	33.6 ± 29.8†
Valsalva	56.5 ± 41.0†
Events during follow-up	
Myectomy	18 (1.9)
Re-PTSMA,	164 (18.1)
Permanent pacemaker	25 (2.6)
ICD	49 (5.1)
Permanent atrial fibrillation	50 (5.2)
Stroke	14 (1.5)
Death	70 (7.4)
Causes of death during follow-up	
Cardiovascular	20 (2.1)
Stroke	6 (0.6)
Cardiac	14 (1.5)
Sudden	4 (0.4)
Heart failure	8 (0.9)
Acute myocardial infarction	2 (0.2)
Other	50 (5.3)

Values are mean ± SD or n (%). *Patients with subaortic and midventricular obstruction. †p < 0.0001 versus baseline measurements.
AV = atrioventricular; CK = creatine kinase; other abbreviations as in Table 1.

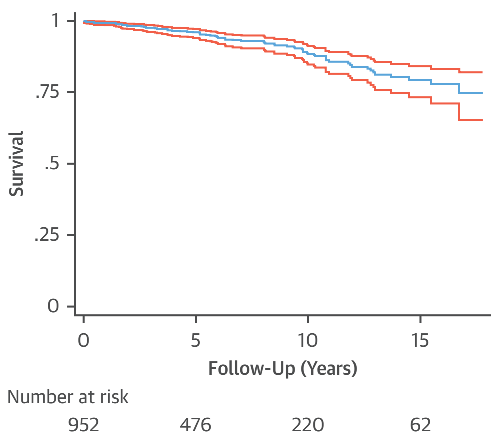
ACUTE RESULTS. Injection of an average of 2.1 ± 0.4 cc alcohol was performed in 952 patients (Table 2). Up to 5 branches had to be tested by contrast echocardiography to identify the target septal branch without potential alcohol misplacement. The primarily chosen first septal branch could be confirmed as target branch in 83% of the patients. In 32 (3.4%) patients, alcohol injection was only possible in a second intervention as a target branch could be clearly identified only after careful reading of the angiographic sequence of the first session. Maximal creatine kinase rise was 872 ± 489 U/l. Echo-Doppler gradients at hospital discharge were reduced to 33.6 ± 29.8 mm Hg at rest and 56.5 ± 41.0 mm Hg at Valsalva ($p < 0.0001$ vs. baseline, each).

During hospital stay, 2 (0.2%) patients died due to pulmonary embolism and multiorgan failure after pneumonia 3 and 33 days after ablation. Including all ablations in this patient group, procedure-related mortality was 0.17%. A total of 377 (39.6%) patients developed total heart block during alcohol injection, but implantation of permanent pacemaker was necessary in only 100 (10.5%) patients. Sixteen of 73 (22.0%) patients with pre-existing left bundle branch block developed permanent heart block requiring permanent pacemaker. A total of 436 (46.1%) patients developed right bundle branch block. In 1 (0.1%) patient, left main dissection occurred during attempt to wire the left anterior descending artery, which could be fixed by stent implantation. Six months later, PTSMA could be successfully performed after no restenosis of the left main stent was documented.

LONG-TERM FOLLOW-UP. After 6.0 ± 5.0 years clinical information was achieved either by questionnaire or direct communication with the patient or the last treating physician (Table 2). A total of 898 (94.3%) patients reported on improved clinical symptoms and 34 (3.6%) patients felt unchanged compared with the time before ablation. Due to our general strategy of ablation of only 1 branch per ablation, reablation was necessary in 164 (18.1%) patients, with hemodynamically and clinically insufficient follow-up result after the first procedure. Eighteen (1.9%) patients had to undergo surgical myectomy. Forty-nine (5.1%) patients underwent ICD implantation due to changing risk-stratification models for prevention of SCD (14,15). Fifty (5.3%) patients developed permanent atrial fibrillation and 14 (1.5%) patients suffered from ischemic stroke.

During follow-up, 70 patients died. Main causes were noncardiac in 50 patients—mostly malignant diseases. Cardiovascular deaths had been observed in 20 patients: 6 patients died after stroke and 14 patients due to cardiac reasons. Cardiac deaths occurred in 2 patients with additional coronary artery disease at an acute myocardial infarction, in 8 patients at end-stage heart failure, and in 4 patients as SCD. Patients who died during follow-up were older than survivors were (65.7 ± 12.1 years of age vs. 54.9 ± 14.8 years of age; $p < 0.0001$) at the time

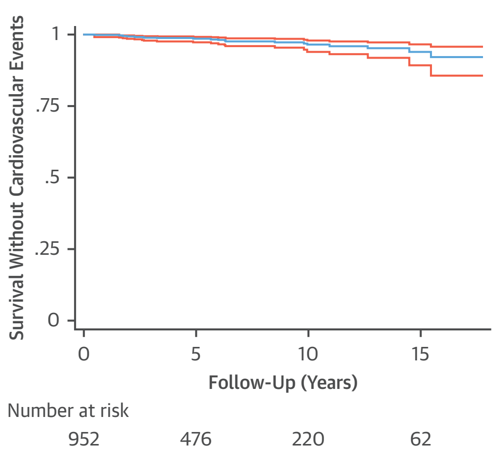
FIGURE 1 Kaplan-Meier Curve of Estimated Overall Survival With 95% CI After PTSMA in 952 Patients



Kaplan-Meier survival analysis after percutaneous transluminal septal myocardial ablation (PTSMA) estimated a 5-year survival of 95.8% (95% confidence interval [CI]: 94.1% to 97.2%), a 10-year survival of 88.3% (95% CI: 84.8% to 91.2%), and a 15-year survival of 79.7% (95% CI: 73.9% to 84.4%). **Orange lines** represent 95% CI.

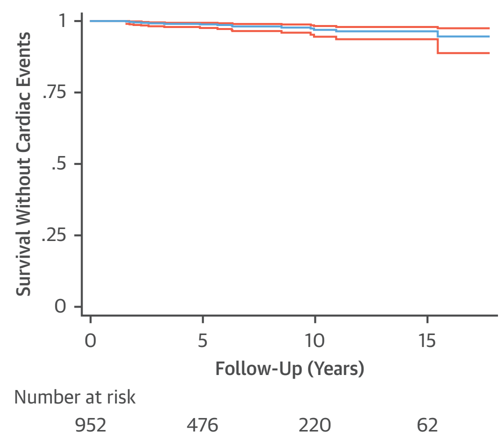
of PTSMA. Patients with noncardiac death were older (67.9 ± 12.1 years of age) compared with patients with cardiovascular death (60.2 ± 10.4 years of age), cardiac death (57.9 ± 10.8 years of age), and SCD (53.1 ± 14.6 years of age; $p < 0.0001$, each). The intervals between PTSMA and death were 6.7 ± 4.3 years in all patients, without significant differences between patients with noncardiac (6.8 ± 4.3 years), cardiovascular (6.4 ± 4.6 years), cardiac (5.7 ± 4.1 years), and sudden death (4.7 ± 4.0 years).

FIGURE 2 Kaplan-Meier Curve of Estimated Survival Free of Cardiovascular Events With 95% CI After PTSMA in 952 Patients



Kaplan-Meier survival analysis after PTSMA in 952 patients resulted in survival free of cardiovascular events after 5 years in 98.6% (95% CI: 97.4% to 99.3%), after 10 years in 96.5% (95% CI: 94.2% to 98.0%), and after 15 years in 92.3% (95% CI: 85.8% to 95.9%). **Orange lines** represent 95% CI. Abbreviations as in Figure 1.

FIGURE 3 Kaplan-Meier Curve of Estimated Survival Free of Cardiac Events With 95% CI After PTSMA in 952 Patients



Kaplan-Meier survival analysis after PTSMA in 952 patients resulted in survival free of cardiac events after 5 years in 98.9% (95% CI: 97.7% to 99.5%), after 10 years in 97.0% (95% CI: 94.7% to 98.3%), and after 15 years in 96.5% (95% CI: 93.9% to 98.0%). **Orange lines** represent 95% CI. Abbreviations as in Figure 1.

Kaplan-Meier survival analysis (Figures 1 to 3) resulted in an estimated 5-year survival of 95.8% (95% CI: 94.1% to 97.2%), an estimated 5-year survival free of cardiovascular events of 98.6% (95% CI: 97.4% to 99.3%), and an estimated 5-year survival free of cardiac events of 98.9% (95% CI: 97.7% to 99.5%). Corresponding values at 10 years were 88.3% (95% CI: 84.8% to 91.2%) for overall survival, 96.5% (95% CI: 94.2% to 98.0%) for survival free of cardiovascular events, and 97.0% (95% CI: 94.7% to 98.3%) survival free of cardiac events. After 15 years, estimated overall survival rate was 79.7% (95% CI: 73.9% to 84.4%), estimated survival free of cardiovascular events 92.3% (95% CI: 85.8% to 95.9%), and estimated survival free of cardiac events 96.5% (95% CI: 93.9% to 98.0%).

DISCUSSION

Hypertrophic cardiomyopathy is the most common genetic cardiac disease. Left ventricular outflow tract obstruction is an important pathophysiologic finding which has great impact on mortality (16,17). Since the 1960s, surgical myectomy was known to improve

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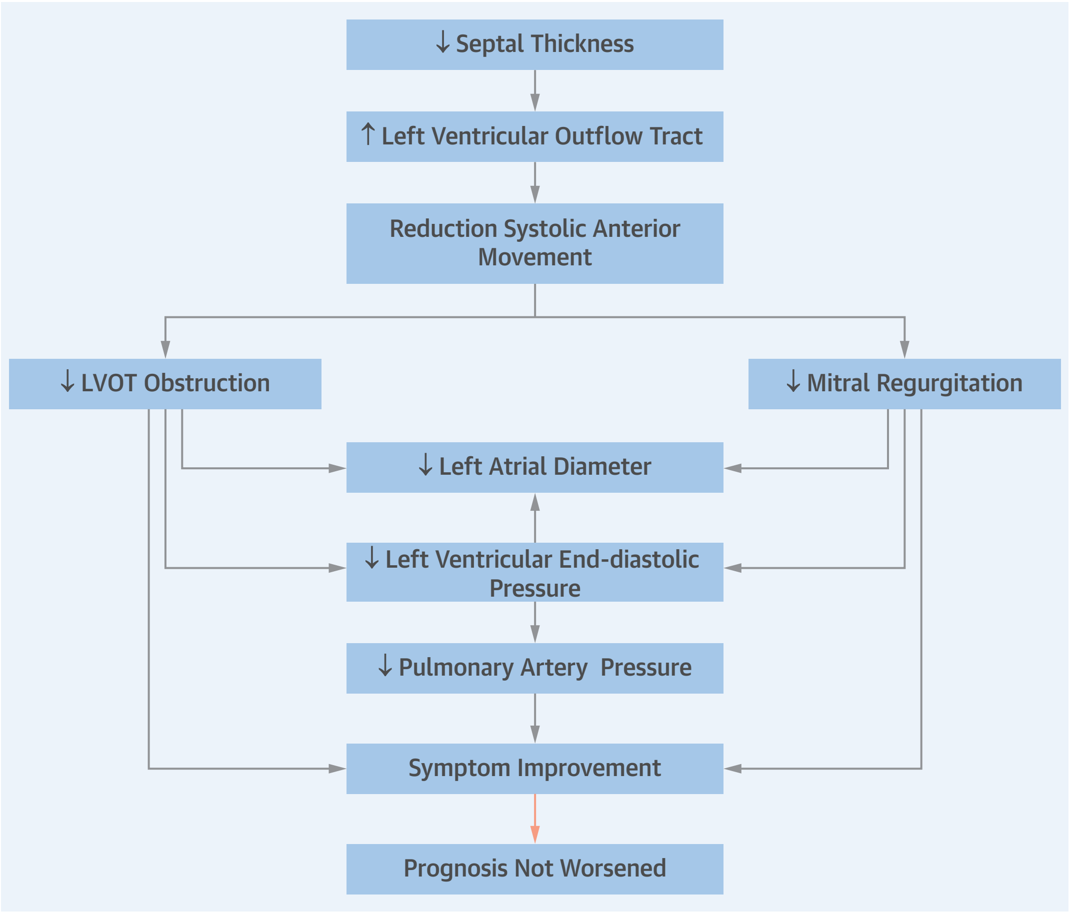
CENTRAL ILLUSTRATION Clinical, Hemodynamic, and Morphologic Changes After Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy



Batzner, A. et al. J Am Coll Cardiol. 2018;72(24):3087-94.

Demonstrates clinical, hemodynamic, and morphologic changes (3,7) after successful percutaneous transluminal septal myocardial ablation (PTSMA) in patients with hypertrophic obstructive cardiomyopathy. The **downward arrows** indicate reduction and the **upward arrows** indicate widening. LVOT = left ventricular outflow tract.

quality of life and survival, although randomized trials are lacking (3).

Since the late 1980s, the idea of interventional treatment of outflow tract obstruction by alcohol-induced circumscribed infarction with consecutive thinning of the septum and widening the left ventricular outflow tract had been described (Gunnar Berghöfer, March 1989, personal communication) after studies of temporary balloon occlusion in coronary arteries had shown regional wall motion abnormality (18) and injection of alcohol into septal branches had shown to be able to influence ventricular tachycardia (19).

First reports of successful alcohol septal ablation (4-6,20) raised concerns about long-term effect, and alcohol-induced infarction was discussed as risk factor or at least arbitrator for elevated cardiac mortality during long-term follow-up, even more than a decade after its introduction (9-11,21). An important step toward lower complication rates of alcohol septal ablation was the introduction of echo guidance as standard procedure in 1996 (7).

The data from our large single-center and single-operator experience show low in-hospital complication rates, with a patient-related hospital mortality of 0.21%. The comparison with other studies underlines the importance of a high-volume operator and center (22,23). Sorajja et al. (24) reported on quantity-related acute and follow-up results. Furthermore, it should be stressed that in our experience hospital mortality occurred only during the post-interventional period, which underlines the importance of intensive care unit monitoring for at least 48 h and in-hospital observation for 1 week.

One possible criticism of our data is the number of necessary reablations (18.10%). Our approach of a single-vessel ablation per session is related to the early known effect of remodeling after ablation (7). This supported us to keep the potential damage of alcohol ablation as small as possible. Furthermore, it must be taken into account that the first septal branch supplies the basal septum incomplete in a large number of patients (25). We think that the low number of the reported complications is related to our careful staged procedure and supports our stepwise approach. Therefore, the potential need of a low risk reablation should not be rated as a complication but rather as a potential part of the procedure.

Undoubted is the positive effect of PTSMA on improvement of clinical symptoms (**Central Illustration**). Similar to this observational study, previous single and follow-up observations with smaller study populations or shorter follow-up periods have also shown no increased morbidity and mortality after septal ablation (8,22,26,27). It should be underlined that patients who died during follow-up were older than the surviving patients at the time of PTSMA. The oldest subgroup were patients with noncardiac deaths.

Some of the earlier studies have observed a reduction of changeable risk factors for SCD, which may explain these findings (8,27). A very small subpopulation of our cohort with ICD implantation for primary prevention of sudden death before alcohol ablation had shown no risk of adverse events during follow-up (28).

This observational study was not designed for comparison of alcohol septal ablation with natural history or surgical myectomy, but rather to show that PTSMA performed in an experienced center is not followed by increased mortality. This was clearly approved by our data in a sufficient large number of patients and with sufficient long-term follow-up period.

Nonrandomized comparisons have shown age-independent comparable survival of HOCM patients after alcohol septal ablation and an age-matched nonobstructive hypertrophic cardiomyopathy cohort (29). Survival after alcohol septal ablation and surgical myectomy had comparable results of both invasively gradient reduction treatment options (30). Therefore, PTSMA should be stated as comparably effective with surgical myectomy for symptom relief without increased procedural and long-term risk.

From the principal standpoint a randomized trial comparing PTSMA and myectomy in symptomatic HOCM patients should be performed. But, due to the low prevalence of HOCM and low rate of hard endpoints during follow-up, especially cardiac mortality, it seems to be unlikely to perform such a study. Therefore, a team of experts in both treatments should clarify the best individual optimal therapy in an individual HOCM patient with respect to anatomical findings, concomitant cardiac and noncardiac morbidities, and patient choice (3). It should be underlined that operator's expertise and availability is one of the most important criteria to achieve a good result in both PTSMA and myectomy.

STUDY LIMITATIONS. This study is an observational single-center analysis of survival after alcohol septal ablation in symptomatic patients with HOCM. Despite the large number of all treated patients with complete follow-up, the report has some limitations. Observational studies always include a potential selection bias. Furthermore, the absence of a control group should be considered before concluding that PTSMA is superior to other treatment options. This could only be done in a prospective randomized trial comparing septal ablation and myectomy in symptomatic HOCM patients. Finally, it should be mentioned that we reported on mortality not including adequate ICD interventions.

CONCLUSIONS

In this study, PTSMA could be proofed as a safe procedure with ongoing symptomatic improvement and excellent long-term survival. Therefore, PTSMA is a reasonable alternative to surgical myectomy in HOCM.

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ADDRESS FOR CORRESPONDENCE: Dr. Hubert Seggewiss, Kardiologie, Klinikum Würzburg Mitte, Standort Juliusspital, Juliuspromenade 19, 97070 Würzburg, Germany. E-mail: seggewiss.hubert@t-online.de.

PERSPECTIVES

COMPETENCY IN PATIENT CARE AND PROCEDURAL SKILLS: In symptomatic patients with HOCM, echo-guided percutaneous alcohol septal ablation is a relatively safe alternative to surgical myectomy and associated with symptomatic improvement and long-term survival.

TRANSLATIONAL OUTLOOK: Randomized trials are needed to compare long-term outcomes of percutaneous septal ablation with surgical myectomy in patients with HOCM.

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