Long-Term Outcome of Alcohol Septal Ablation in Patients With Obstructive Hypertrophic Cardiomyopathy
A Word of Caution

Folkert J. ten Cate, MD, PhD; Osama I.I. Soliman, MD, PhD; Michelle Michels, MD; Dominic A.M.J. Theuns, PhD; Peter L. de Jong, MD; Marcel L. Geleijnse, MD, PhD; Patrick W. Serruys, MD, PhD

Background—The impact of alcohol septal ablation (ASA)-induced scar is not known. This study sought to examine the long-term outcome of ASA among patients with obstructive hypertrophic cardiomyopathy.

Methods and Results—Ninety-one consecutive patients (aged 54±15 years) with obstructive hypertrophic cardiomyopathy underwent ASA. Primary study end point was a composite of cardiac death and aborted sudden cardiac death including appropriate cardioverter-defibrillator discharges for fast ventricular tachycardia/ventricular fibrillation. Secondary end points were noncardiac death and other nonfatal complications. Outcomes of ASA patients were compared with 40 patients with hypertrophic cardiomyopathy who underwent septal myectomy. During 5.4±2.5 years, primary and/or secondary end points were seen in 35 (38%) ASA patients of whom 19 (21%) patients met the primary end point. The 1-, 5-, and 8-year survival-free from the primary end point was 96%, 86%, and 67%, respectively in ASA patients versus 100%, 96%, and 96%, respectively in myectomy patients during 6.6±2.7 years (log-rank, P=0.01). ASA patients had a 5-fold increase in the estimated annual primary end point rate (4.4% versus 0.9%) compared with myectomy patients. In a multivariable model including a propensity score, ASA was an independent predictor of the primary end point (unadjusted hazard ratio, 5.2; 95% CI, 1.2 to 22.1; P=0.02 and propensity score-adjusted hazard ratio, 6.1; 95% CI, 1.4 to 27.1; P=0.02).

Conclusions—This study shows that ASA has potentially unwanted long-term effects. This poses special precaution, given the fact that ASA is practiced worldwide at increasing rate. We recommend myectomy as the preferred intervention in patients with obstructive hypertrophic cardiomyopathy. (Circ Heart Fail. 2010;3:362-369.)

Key Words: hypertrophic cardiomyopathy ■ ablation ■ infarction ■ mortality ■ myectomy ■ defibrillators ■ survival

Obstructive hypertrophic cardiomyopathy (HCM) is characterized by asymmetrical septal hypertrophy and dynamic left ventricular (LV) outflow tract obstruction.1 In patients with symptomatic obstructive HCM, surgical resection (myectomy) of the basal septum relieves LV outflow obstruction with improvement in long-term outcome.2,3 Alcohol septal ablation (ASA) is an alternative technique in which ethanol is injected into 1 or more septal perforator branches of the left anterior descending coronary artery. The efficacy of ASA has been proven with hemodynamic results mimicking those of myectomy.3-7 The septal morphological appearance post-ASA is one of the myocardial infarction.8 It is not well known whether this may produce an arrhythmic substrate and possibly trigger sudden cardiac death (SCD).9,10 Therefore, the present study sought to describe long-term post-ASA outcome.
Diagnostic Evaluation
HCM was defined as a hypertrophied and nondilated LV in the absence of other cardiac or systemic disease that could explain hypertrophy. All patients underwent a standard 2-dimensional echo-Doppler ultrasound examination and clinical risk assessment including Holter monitoring and treadmill exercise testing.

ASA Procedure
ASA was performed as described previously. In brief, using the standard Judkins technique, a 6F pacemaker lead was placed in the right ventricle, a 6F pigtail catheter was positioned into the LV, and a 7F Judkins guiding catheter was placed in the ascending aorta. LV outflow tract peak systolic gradient was continuously monitored throughout the whole procedure. After initial coronary angiography for localizing the origin of the septal perforating arteries, a 1.5 to 2.5×10 mm balloon catheter was introduced over a 0.014-inch guide wire into the target perforator artery and inflated. ASA was performed with the assistance of myocardial contrast (1 mL SonoVue, Bracco, Geneva, Switzerland) echocardiography to identify the septal region by the selected septal branch. If no contrast was seen outside the thickened basal septum, 0.5 mL alcohol was injected over 30 seconds followed by saline flush under continuous hemodynamic and ECG surveillance. A successful procedure was defined as absence of a residual invasive dynamic gradient >25 mm Hg. If the target reduction in pressure gradient was not achieved, alcohol injections were repeated after 5 minutes (maximum 2.5 mL) within the same perforator branch. If not successful, the procedure was repeated in a second perforator branch. Once success was achieved, the balloon was deflated, and coronary angiography was repeated to confirm the occlusion of the septal branch and patency of the left anterior descending coronary artery. A temporary pacemaker lead was kept in place for at least 24 hours. A permanent pacemaker was used to treat a persistent high-grade atrioventricular block and an implantable cardioverter-defibrillator (ICD) implantation was considered at the discretion of the HCM cardiovascular specialist (F.J.t.C.).

ICD
ICD implantation for secondary prevention was considered for patients with HCM who survived cardiac arrest and for primary prevention for patients with HCM who had ≥2 conventional risk factors for HCM-related SCD. In addition, ICD was used as an alternative to conventional pacemaker in a few patients with post-ASA persistent high-grade atrioventricular block. Device implantations were performed according to customary practice, with defibrillation thresholds routinely tested to document successful termination of lethal arrhythmias. For all patients, ICD programming was intended to avoid inappropriate therapy and tailored according to the clinical presentation. Mean ventricular tachyarrhythmia detection rate was 349±18 ms, and the mean fibrillation detection rate was 283±15 ms.

Device interrogation was performed on a 3-month basis and otherwise following patients’ symptoms. Arrhythmias responsible for triggering defibrillator therapy were identified from the stored intracardiac electrogams. Defibrillator therapy was considered appropriate when triggered by ventricular fibrillation (VF) or ventricular tachycardia (VT). Device therapy was considered inappropriate when triggered by “benign rhythms” with rates exceeding the lower antitachycardia therapy limit and ECG surveillance. A successful procedure was defined as residual LV outflow tract peak systolic gradient ≤10 mm Hg and no other complications due to residual LV outflow tract gradient.

Myectomy Patients
Outcome of ASA patients were compared with outcome of 40 consecutive patients with obstructive HCM who underwent modified septal myectomy during the same period of enrollment of the ASA patients.

Follow-Up
Two investigators (F.J.t.C. and M.M.) examined all patients at 3, 6, and 12 months and then yearly with complete follow-up to 9 years. Cause of death was documented from hospital records, general practitioners records, and civil registries. SCD was defined as instantaneous and unexpected death within 1 hour after a witnessed collapse in patients who previously were in stable clinical condition or nocturnal death with no antecedent history of worsening symptoms.

Statistical Analyses
Qualitative variables were expressed as percentages and quantitative variables as mean (SD). The normality distribution for continuous data were examined with the Shapiro-Wilk test. Comparison of numeric variables was performed using the 2-sided Student t test or Wilcoxon rank-sum test, and the χ² or Fisher exact tests were used to compare qualitative variables. A continuous propensity score analysis was performed to adjust for the intergroup (ASA versus myectomy) differences in baseline characteristics caused by the selection bias inherent to the nonrandomized nature of the study. A propensity score representing the likelihood of having ASA as opposed to myectomy was calculated for each patient by using a logistic regression analysis that identified variables independently associated with the type of procedure. All variables listed in Table 1 were included in a univariate regression analysis, and variables exhibiting a P value <.20 were included in a multivariate model. Those variables (P < 0.020) were age, positive risk factors for HCM-related SCD, LV outflow tract peak gradient, ventricular septum thickness, and LV end-systolic diameter. Kaplan-Meier curves were used to delineate freedom from death or aborted SCD and compared with log-rank test. In addition, the incidence of postprocedural primary end point was further evaluated between groups as adjusted, for propensity score in a multivariable regression analysis. The propensity score did not emerge as an independent predictor of the primary end point, suggesting that differences attributed to the type of intervention by the initial Cox regression analyses were not explained by bias in patient selection on the basis of their baseline characteristics. Results were expressed as hazard ratios with 95% CIs. A P value <.05 was considered significant. SPSS version 15.0 (SPSS Inc, Chicago, IL) was used for statistical analysis.

Results
Baseline Characteristics
Table 1 lists baseline data of all patients. In 81 (89%) patients, ASA was based solely on patient preference, and 10 (11%) patients had a high surgical risk (Table 2).

Procedural Data
Mean number of septal perforator arteries used for ASA was 1.1±0.3. Mean ethanol volume was 3.5±1.5 mL with a larger volume in the early (first 25 patients) versus late (4.5±1.2 mL versus 2.4±1.0 mL) experience. LV outflow tract peak pressure gradient reduced from 92±25 mm Hg at baseline to 8±17 mm Hg (P<0.001) immediately post-ASA.
Echocardiographic data
do not reveal a specific cause. Four (4%) patients survived in-hospital cardiac arrest. One patient had VF on the day post-ASA; the patient refused to receive an ICD and suddenly died 2.3 years post-ASA. One patient had intraprocedural VF, and follow-up went uneventful over 7.4 years. One patient survived 2 episodes of in-hospital VF (day 4) and sustained VT 5.6 years post-ASA. One patient survived in-hospital sustained VT with hemodynamic instability on the day post-ASA. In these latter 3 patients, an ICD was implanted for secondary prevention. Four (4%) patients survived in-hospital cardiac arrest. One patient had VF on the day post-ASA; the patient refused to receive an ICD and suddenly died 2.3 years post-ASA. One patient had intraprocedural VF, and follow-up went uneventful over 7.4 years. One patient survived 2 episodes of in-hospital VF (day 4) and sustained VT 5.6 years post-ASA. One patient survived in-hospital sustained VT with hemodynamic instability on the day post-ASA. In these latter 3 patients, an ICD was implanted for secondary prevention.

**In-Hospital and 30-Day Primary End Point**
Procedure-related mortality was seen in 2 patients (2%) due to cardiac tamponade and intractable VF, respectively. This latter patient died during the ASA procedure after the first 0.5 mL ethanol injection. Autopsy did not reveal a specific cause. Four (4%) patients survived in-hospital cardiac arrest. One patient had VF on the day post-ASA; the patient refused to receive an ICD and suddenly died 2.3 years post-ASA. One patient had intraprocedural VF, and follow-up went uneventful over 7.4 years. One patient survived 2 episodes of in-hospital VF (day 4) and sustained VT 5.6 years post-ASA. One patient survived in-hospital sustained VT with hemodynamic instability on the day post-ASA. In these latter 3 patients, an ICD was implanted for secondary prevention.

**Long-Term (>30 Days) Primary End Point**
During follow-up, 6 patients (7%) died due to SCD and 1 patient (1%) died 5.8 years post-ASA because of end-stage

**Table 3. Study End Points Among ASA Patients (n=91)**

<table>
<thead>
<tr>
<th>Primary end point (n=19)</th>
<th>Early &lt;30 Days</th>
<th>Late ≥30 Days</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac mortality</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>2 (2)</td>
<td>7 (8)</td>
<td>9 (10)</td>
</tr>
<tr>
<td>SCD</td>
<td>1 (1)</td>
<td>6 (7)</td>
<td>7 (8)</td>
</tr>
<tr>
<td>Other cardiac death</td>
<td>1 (1)</td>
<td>1 (1)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Arrhythmogenic complications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>4 (4)</td>
<td>7 (8)</td>
<td>11 (12)</td>
</tr>
<tr>
<td>Resuscitated SCD</td>
<td>4 (4)</td>
<td>3 (3)</td>
<td>7 (8)</td>
</tr>
<tr>
<td>Appropriate ICD shocks</td>
<td>0</td>
<td>4 (4)</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Secondary end points (n=28)*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-cardiac death</td>
<td>0</td>
<td>2 (2)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>ICD implantation</td>
<td>8 (9)</td>
<td>8 (9)</td>
<td>16 (18)</td>
</tr>
<tr>
<td>Inappropriate ICD therapy</td>
<td>0</td>
<td>2 (2)</td>
<td>2 (2)</td>
</tr>
<tr>
<td>Acute myocardial infarction</td>
<td>2 (2)</td>
<td>0</td>
<td>2 (2)</td>
</tr>
<tr>
<td>ASA failure</td>
<td>3 (3)</td>
<td>7 (8)</td>
<td>10 (11)</td>
</tr>
<tr>
<td>Permanent pacemaker dependency</td>
<td>4 (4)</td>
<td>0</td>
<td>4 (4)</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>1 (1)</td>
<td>0</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>1 (1)</td>
<td>10 (11)</td>
<td>11 (12)</td>
</tr>
</tbody>
</table>

Values are presented as n (%).

*Some patients had >1 end point.

Primary End Point
During 5.4±2.5 years, 1 or more events were seen in 35 (38%) patients post-ASA (Table 3), of whom 19 (21%) met the primary end point. Using Kaplan-Meier survival analysis, the estimated annual rate of the primary end point was 4.4% over 8 years (Figure 1). During follow-up 11 (12%) patients died due to cardiac causes (9) and noncardiac causes (2).
heart failure. Eleven (12%) patients survived 1 or more episodes of ventricular tachyarrhythmia. In 4 patients, the arrhythmia was successfully terminated by ICD discharge, for fast VT/VF 0.3, 0.6, 1.3, and 2.6 years after ICD implanta-
tion. Cycle length was 200, 230, 230, and 330 ms, respectively (Figure 2). In the remaining 7 patients without ICD, cardiopulmonary resuscitation was needed. Of those 7 patients, 1 later died during follow-up and 3 patients who
survived sustained VT with hemodynamic instability 3.2, 4.4, and 5.6 years, respectively post-ASA, received an ICD.

Secondary End Points
Twenty-eight patients (31%) had 1 or more secondary end points.

In-Hospital and 30-Day Secondary End Points
Eight (9%) patients had an ICD implantation. None of these patients had inappropriate discharge during the 30-day follow-up post-ASA. Two patients had nonfatal acute myocardial infarction due to coronary artery dissection and ethanol spill into the left anterior descending coronary artery. These 2 patients were successfully managed and underwent myectomy at a later date. As mentioned before, ASA failure was seen in 3 patients due to technical causes. Four (4%) patients had a high-grade atrioventricular block and received a permanent pacemaker. One patient subsequently had extended hospitalization for pericardial effusion due to right ventricular perforation from temporary pacemaker placement. This patient underwent myectomy 2 months later because of unsuccessful reduction of LV outflow pressure gradient (Table 3).

Long-Term (>30 Days) Secondary End Points
Two patients (2%) died because of noncardiac causes (suicide and cancer 2 years post-ASA). Eight (9%) patients had an ICD implantation, of whom 2 patients had inappropriate discharges because of atrial arrhythmias. At the most recent follow-up, 10 (11%) patients had failed ASA, of whom 7 (8%) patients had early successful reduction but experienced a progressive increase in LV outflow gradient associated with recurrence of symptoms. Five patients had repeat ASA, and 5 patients underwent myectomy. One or more episodes of atrial fibrillation were reported in 10 (11%) patients (Table 3).

Conventional Risk Factors for HCM-Related SCD and the Primary End Point
Forty ASA patients (44%) had at least 1 conventional risk factor for HCM-related SCD, of whom 13 (14%) had 2 risk factors. No patient had a history of aborted SCD before ASA. In addition, there was no difference in the number of risk factors between patients with and without the primary end point (0.79±0.79 versus 0.54±0.72; P=0.21). Likewise, as seen in Figure 3, incidence of primary or secondary end points was not related to the number of conventional risk factors for HCM-related SCD.

Outcome of Myectomy Versus ASA Patients
As seen in Table 1, patients who underwent myectomy had similar baseline characteristics as ASA patients. Also, comparable immediate reductions of LV outflow gradient were seen. There was 98% procedural success in myectomy patients and the in-hospital and 30-day postoperative clinical course went uneventful. During a mean follow-up of 6.6±2.7 years, 2 (5%) patients died and none had aborted SCD. The 1-, 5-, and 8-year survival free from the primary end point was 100%, 96%, and 96%, respectively, which was better than seen in the ASA patients (96%, 86%, and 67%, respectively; \( \chi^2 \) = 5.9; log-rank \( P=0.01; \) Figure 1). ASA patients had \( \approx \) 5-fold annual rate of the primary end point (4.4% versus 0.9%) compared with myectomy patients. A total of 5 (6%) patients had an ICD implantation in the myectomy group compared with a total of 16 (18%) in the ASA patients and 1 patient had permanent pacemaker for complete atrioventricular block. ICD implantation was for primary prevention indication before (4 patients) and after (1 patient) myectomy. As mentioned previously, ICD appropriate shocks were recorded in 4 (25%) of the patients who underwent ICD implantation in the ASA group. None of the myectomy patients had an ICD shock during follow-up. Myectomy was repeated in 1 patient because of recurrent obstruction 8 days postoperative and in 1 patient due to dehiscence of the mitral valve patch 6 months postoperative. In both patients, postoperative clinical course went uneventful with a follow-up of 8.6 and 3.8 years, respectively.

Cox proportional-hazards regression analysis among the total intervention group (n=131) including baseline variables that are listed in Table 1, type of intervention (ASA versus myectomy), ethanol volume, and propensity score showed that only ASA was an independent predictor of the primary end point with an unadjusted hazard ratio \( \beta \) : 5.2: 95% CI 1.2 to 22.1, \( P=0.02 \) and propensity score-adjusted hazard ratio 6.1: 95% CI 1.4 to 27.1, \( P=0.02 \). Of note large (\( \approx \) 2 mL) ethanol volumes were not associated with the primary end point (\( P=0.72 \)) (Figure 4). Importantly, only 1 of 10 (10%) ASA patients in whom myectomy was considered a high-risk procedure met the primary end point compared with 18 of the remaining 81 (22%) low-to-moderate risk patients. The combined rate of postprocedural primary and secondary end points in myectomy patients was 15% compared with 38% among ASA patients, \( \chi^2 = 5.8; \) adjusted \( P<0.02 \).
Discussion

The main finding of our study is that $\approx 1$ of 3 patients with HCM who underwent ASA had major cardiovascular complications during the procedure and follow-up, including cardiac death or resuscitated SCD in $\approx 1$ of 5 patients. Compared with the myectomy patients, ASA patients had a 5-fold increase in the estimated annual primary end point.

Of note, the early (30-day) complication rate of ASA patients in our study was similar to the complication rate reported in a meta-analysis in $\approx 3000$ ASA patients with respect to mortality (2.2% versus 1.5%), VF rate (3.3% versus 2.2%), permanent pacemaker dependency (11.0% versus 10.5%), pericardial effusion (1.1% versus 0.6%), and coronary artery dissection (1.1% versus 1.8%); whereas procedural failure occurred less often (3.3% versus 11.1%).

Importantly, most serious adverse events were late, and therefore, ASA carries a long-term risk. Of note, all ASA patients who died suddenly had no ICD, where there was no death among the patients who received an ICD. Since its introduction in 1994, the number of ASA procedures has exceeded the number of surgical procedures by 10- to 35-fold during the same time period worldwide. The notion of our study is to provide a word of caution to the increased number of ASA procedures done.

Arrhythmogenic ASA-Induced Scar

The potential risk of arrhythmic events due to post-ASA healed myocardial infarction in patients prone to arrhythmias has been a matter of concern since early practice of ASA. Our group described post-ASA regional hyperenhancement on contrast-enhanced MRI in the basal septum in all patients. Mean infarction size was $20 \pm 9 \text{ g}$, corresponding to $10 \pm 5\%$ and $31 \pm 16\%$ of LV and septal mass, respectively.

Frequent episodes of nonsustained VT and VF have been reported in several series early post-ASA. The high arrhythmogenic susceptibility of patients with HCM could be attributed to myocardial fiber disarray.

In a large cohort of patients with HCM, ICD interventions appropriately terminated VT/VF in 20% of patients with a 10.6% annual intervention rate for secondary prevention after aborted SCD and 3.6% per year for primary prevention of SCD. In a recent study, annual ICD intervention rate was 2.8% over a 3-year period in patients with HCM who underwent ASA and ICD implantation for primary prevention of SCD.

The relation between the size of post-ASA infarction and arrhythmogenic risk is not clearly understood. In a recent article, a lower ethanol volume was an independent predictor of improved long-term survival after ASA, a finding that we could not reproduce. Boekstegers et al reported that electrophysiological testing before and 4 to 6 months post-ASA did not suggest enhanced arrhythmogenesis. However, most of the arrhythmic complications in our series occurred after 4 years post-ASA. Of note, the size of the septal infarction in our series was not different from other series.

ASA Versus Surgical Treatment of Patients With Obstructive HCM

Ideally, a randomized trial comparing myectomy and ASA should be performed to examine whether they are true equivalent therapeutic options. However, such a trial is unlikely to be undertaken. Alternatively, comparison of ASA and myectomy outcomes can be performed by analyzing data from single center registries. In a small series of ASA patients, Maron et al found a 4-fold increase in appropriate ICD intervention rate (10.3% versus 2.6% per year, respectively) compared with patients who had previously undergone surgical septal myectomy. In another report, myectomy patients with an ICD had over 10-fold fewer appropriate ICD discharges (0.2% versus 4.3% annually, respectively) compared with patients with nonoperated HCM with an ICD.

In another report by Sorajja et al surgical patients had significantly better symptom-free survival compared with ASA (89% versus 71%) over a 4-year follow-up. In our HCM cohort, ASA induced relief of LV outflow obstruction with subsequent reduction in LV hypertrophy and improvement...
in clinical status, LV function, and microcirculation. However, the incidence of arrhythmic complications and cardiac death was ~5-fold as compared with myectomy patients.

In contrast to the findings described previously, Kwon et al. did not suggest excess mortality on long-term follow-up in ASA versus myectomy patients when statistically corrected for the higher risk profile of ASA patients. However, their primary end point did not include arrhythmic complications, which occurred in approximately half of the patients with the primary end point in our series. In addition, mortality rate was 3-fold increased in the ASA patients compared with myectomy patients (24% versus 8%). Most of the ASA mortality occurred in patients older than 65 years. However, a log-rank test between the ASA and myectomy in 28 propensity-matched patients was negative, obviously because ASA patients older than 65 years were excluded in this analysis. In addition, 6 (11%) of the ASA patients underwent myectomy after failed ASA, despite being high-risk candidates. It is not clear to what extent these crossed over to patients to myectomy contributed to the mortality in that group. Fernandes et al. recently reported a 1.1% rate of SCD on long-term follow-up post-ASA. However, contrary to our study, 8% of patients were lost to follow-up. In addition, no data on arrhythmic complications were reported.

Risk Factors of HCM-Related SCD and Post-ASA Outcome

There is uncertainty as to precisely identify patients with HCM at greatest risk of SCD. In some studies, a single risk factor for SCD was sufficient to justify prophylactic ICD, whereas in other studies well-known risk factors were not predictive of ICD therapy. In our study, the number of risk factors for SCD in ASA patients was very low and was not different between patients with and without the primary end point. Moreover, the 9 patients who died because of cardiac cause without ICD had no major or minor risk factors for SCD, except for 1 patient who had a family history of SCD and had recurrent episodes of nonsustained VT. A logical inference of our risk factor data is that ASA itself is a risk factor for SCD independent of the amount of ethanol, age, and comorbidities.

Of note, appropriate ICD discharges are not equivalent to SCD. In addition, the small sample size, might not rule out different between patients with and without the primary end point. Moreover, the 9 patients who died because of cardiac cause without ICD had no major or minor risk factors for SCD, except for 1 patient who had a family history of SCD and had recurrent episodes of nonsustained VT. A logical inference of our risk factor data is that ASA itself is a risk factor for SCD independent of the amount of ethanol, age, and comorbidities.

Conclusions

The therapeutic choice for ASA is limited by the lack of long-term safety data as compared with myectomy. Because a randomized study between ASA and myectomy is unlikely to be performed, we can only rely on registries in single centers. Our data show that ASA is effective but has potentially adverse long-term effects. This poses special precaution, given the fact that ASA is practiced worldwide at increasing rates. We recommend myectomy as the preferred treatment of choice in patients with symptomatic obstructive HCM.

Acknowledgments

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Disclosures

None.

References


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**CLINICAL PERSPECTIVE**

The safety and efficacy of alcohol septal ablation has been a matter of long-lasting debate, particularly when compared with the surgical gold standard in patients with symptomatic obstructive hypertrophic cardiomyopathy (HCM). The safety concerns are related to the potential arrhythmogenic effects of septal ablation induced scar. Despite the lack of long-term safety data, number of septal ablation procedures exceeded more than 35-fold the number of all surgical procedures worldwide. In this study, registry of symptomatic patients with obstructive HCM who underwent septal ablation and surgical treatment at HCM tertiary referral center were analyzed. The overall estimated annual incidence of a composite primary end point (cardiac death and aborted sudden cardiac death including defibrillator shocks for ventricular tachyarrhythmia) after septal ablation was 5-fold of that in the surgical HCM cohort. Importantly, early success and complications rates were not different from mean rates in a published meta-analysis of ~3000 patients after septal ablation. Most of postablation complications were late sudden cardiac death or aborted sudden cardiac death. Generally, in experienced specialized HCM centers, septal ablation is an efficacious procedure that may improve symptoms in most patients with obstructive HCM. Despite this, our results provide solid evidence that at long-term evaluation, septal ablation is not as safe as surgery, and therefore, surgery should remain the preferred therapeutic option for septal reduction therapy.
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