Outcome of Surgical Myectomy After Unsuccessful Alcohol Septal Ablation for the Treatment of Patients With Hypertrophic Obstructive Cardiomyopathy
Sherif F. Nagueh, John M. Buergler, Miguel A. Quinones, William H. Spencer, III, and Gerald M. Lawrie
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Objectives We sought to determine the outcome of myectomy after unsuccessful alcohol ablation.

Background Alcohol septal ablation results in symptomatic improvement and a reduction in dynamic obstruction in most hypertrophic obstructive cardiomyopathy patients. However, a few patients remain with severe symptoms and obstruction and need surgery. The outcome of these cases is not well known.

Methods The medical records of 375 patients who underwent alcohol ablation at our institution were reviewed. Twenty patients (5.3%, mean age 53 ± 18 years, 17 women) subsequently needed surgical myectomy. The New York Heart Association (NYHA) functional class, angina class, exercise duration, left ventricular outflow tract (LVOT) gradient, ejection fraction, and septal thickness were tabulated. The anatomy and distribution of the septal perforator arteries were examined.

Results After ablation, NYHA functional class (3 to 2.5; p < 0.05) and LVOT gradient (93 ± 23 mm Hg to 71 ± 26 mm Hg; p < 0.05) were slightly improved, without a change in exercise duration (171 ± 124 s to 168 ± 148 s; p > 0.5). Myectomy was performed at 19 ± 15 months after ablation. There was no operative mortality, but permanent pacing was needed in 2 patients after surgery, and 3 other cases needed pacing before, or as a complication of, alcohol ablation. A significant improvement was noted, with the NYHA functional class decreasing to 1, exercise duration increasing to 423 ± 171 s, and LVOT gradient decreasing to 6 ± 11 mm Hg (all p < 0.05 versus post-alcohol ablation).

Conclusions Myectomy can be successfully performed after failed alcohol ablation, but with a higher incidence of heart block than in cases where only surgery is performed. Otherwise, alcohol ablation does not appear to adversely affect surgical outcome. (J Am Coll Cardiol 2007;50:795–8) © 2007 by the American College of Cardiology Foundation

Patients with hypertrophic obstructive cardiomyopathy (HOCM) and persistent symptoms despite adequate medical therapy, are usually considered for septal reduction therapy, by surgery or alcohol ablation (1–4). Although alcohol septal ablation results in hemodynamic and clinical improvement in most patients, a subset remains in whom the results are suboptimal. In addition, when a repeat procedure is not feasible, the treatment of these patients can be challenging. In particular, surgical myectomy may be rendered more difficult. Accordingly, we undertook the present study to determine the effect, if any, of failed alcohol ablation on the outcome of subsequent surgical myectomy.

Methods We reviewed our series of 375 patients who underwent alcohol septal ablation at the Methodist Hospital, Houston, Texas, from 1996 to 2006 to identify those patients who subsequently required surgical myectomy because of residual dynamic left ventricular outflow tract (LVOT) obstruction and advanced dyspnea and/or angina.

Patients’ records were reviewed to identify these cases and their outcome. Specifically, we noted the clinical and hemodynamic status of each of the patients before and after alcohol septal reduction therapy, the timing of surgical myectomy after the initial interventional approach, postoperative complications, and the longer-term outcome after surgery. Longer-term outcome was determined from subsequent clinic visits, written
correspondence from the referring physicians, and telephone interviews with patients or their families.

**Alcohol septal ablation.** Alcohol septal ablation was performed as previously published by our group (4). After coronary angiography, a temporary pacing wire was placed in the right ventricular apex (in patients without a permanent pacemaker) for backup pacing, should high-grade atrioventricular (AV) block develop. A 7-F guiding catheter was engaged in the ostium of the left main coronary artery, and a 9 to 10 mm × 1.5 to 3 mm balloon catheter was advanced over a guide wire into the target septal perforator artery. Myocardial contrast echocardiography (MCE) was injected through the balloon lumen to delineate the culprit septal segments. Subsequently, ethanol was injected into the artery supplying the culprit septal segments, and left in place for 5 min. A permanent pacemaker was implanted if advanced AV block was present 48 h later.

**Surgical myectomy.** In 18 patients, surgery was performed at the Methodist Hospital (Houston, Texas) by a single surgeon (G.M.L.) with extensive experience of surgical myectomy. The other 2 patients had surgery performed elsewhere. Pre-, intra- and post-operative transesophageal echocardiography was used to assess the site and extent of residual obstruction. The heart was exposed through a median sternotomy. Cardiopulmonary bypass was initiated using ascending aortic cannulation, and the aortic valve was exposed through a low transverse aortotomy. The aortic leaflets were retracted and the outflow tract inspected. The LVOT was palpated bimanually from the endocardial and epicardial surfaces of the heart. The muscular obstruction was then resected until the wall thickness of the left ventricle (LV) and septum were the same thickness as the nonobstructive LV wall and septum. This usually required a resection extending from the middle septum to the anterior edge of the anterior mitral leaflet and distally down to or beyond the level of the mitral papillary muscles toward the apex. It was unnecessary to resect the areas already ablated by the alcohol. This “extended myotomy” is a much more extensive resection than the original Morrow procedure. It is of great importance that, despite the greater difficulty in exposing the distal LV cavity and avoiding damage to the mitral valve chordae, the surgeon ensures that the most distal portion of the obstruction is resected. Temporary pacing wires were placed on the LV, and patients were kept hydrated.

**Statistics.** Data are presented as mean ± SD or median (25th to 75th percentiles). Changes in clinical and hemodynamic status in the same patients after alcohol septal reduction therapy and surgical myectomy were evaluated using repeated measures analysis of variance (ANOVA) (or ANOVA on ranks for ordinal variables), with the Holm-Sidak test (or Tukey for ordinal variables) used for subsequent pairwise comparisons. Significance was defined with a p value of ≤0.05.

**Results**

There were 20 patients (age 53 ± 18 years, 17 women) who were initially treated with alcohol septal reduction therapy and later underwent surgical myectomy because of recurrent dynamic obstruction and symptoms. At baseline, all had a dynamic gradient due to systolic anterior motion), at rest (without provocation). In this series, 4 patients had an automatic implantable cardioverter-defibrillator (AICD) at baseline. Two additional patients had an AICD implanted after alcohol ablation, and 1 patient had device implantation after surgery. All of the 7 patients had the defibrillator implanted for primary prevention because they had a high risk of sudden cardiac death.

**Acute and longer-term results of alcohol septal reduction therapy.** In the first alcohol septal ablation procedure (data summarized for all 20 patients), the mean number of septal perforator arteries injected was 1.4 ± 0.5, and the average volume of ethanol used was 2.9 ± 1 ml. Overall, peak creatine kinase (CK) averaged 1,199 ± 467 U/l, and the dynamic gradient in the catheterization laboratory as measured invasively decreased from 90 ± 20 mm Hg to 30 ± 16 mm Hg (p < 0.001). However, there were 13 patients with a residual gradient of ≥25 mm Hg.

Among these 20 patients, 9 had a repeat alcohol septal reduction procedure at 14 ± 11 months after the first procedure. In the remaining 11 patients, there were no target arteries (determined by angiography and MCE) after the first procedure had resulted in occlusion of the only artery supplying the culprit septal segments.

For the 9 patients who underwent a repeat alcohol septal reduction procedure, the mean number of septal perforator arteries injected was 1.4 ± 0.5, and the average volume of ethanol used was 2.3 ± 0.7 ml. Peak CK leak averaged 736 ± 259 U/l, and the dynamic gradient in the catheterization laboratory as measured invasively decreased from 70 ± 26 mm Hg to 35 ± 16 mm Hg (p < 0.001).

At 19 ± 15 months after alcohol septal reduction therapy (duration calculated after the second ablation for those who had a repeat procedure), all 20 patients had severe dyspnea and or angina, along with dynamic obstruction, despite adequate medical therapy and opted to proceed to surgery.

**Reasons for unsuccessful alcohol septal ablation.** In this series, there were unique aspects to the distribution of the septal perforator arteries. On examination of their angiograms, 5 patients had the target septal segments supplied by 2 vessels as determined by MCE, and ethanol was injected into only 1 artery with the expectation that this would be sufficient to induce the effective amount of septal necrosis. In the 15 other patients, the distribution of the septal...
perforator did not cover the whole septal area involved with dynamic obstruction, and residual septal thickness was present just distal to the site of alcohol ablation. This led to residual obstruction despite inducing infarction in the basal septum (Video [see Appendix]). Importantly, during surgery this septal rim was easily identified and resected.

**Results of surgery.** There was no operative mortality. All patients experienced an improvement in dyspnea and angina after surgery performed at 18 ± 6 months. An acute reduction was noted in the LVOT gradient, which was maintained at follow-up. Although a further reduction in septal thickness was observed after surgery, no significant change was noted in left ventricular ejection fraction (LVEF) (Table 1).

**Pacemaker implantation.** One of the 20 patients had a permanent pacemaker implanted before alcohol septal ablation for treatment of dynamic obstruction. After alcohol septal ablation, 2 patients needed permanent pacing for high-grade AV block. Two additional patients (10%) developed complete heart block after surgery and needed permanent pacing. Both of these patients developed right bundle branch block after alcohol septal ablation and complete AV block after surgical myectomy.

**Discussion**

This study shows that surgical myectomy can be readily performed after failed alcohol ablation with successful results that are similar to surgery as the primary treatment (1). Aside from the higher incidence of complete heart block in this situation, alcohol septal reduction therapy, in and of itself, does not appear to adversely affect the outcome of surgical myectomy.

**Unsuccessful alcohol septal ablation and the need for subsequent surgery.** Alcohol septal ablation results in symptomatic and hemodynamic improvement in the majority of patients with HOCM (2–4). However, in a subset of patients, residual obstruction and persistent symptoms necessitate surgery. In our experience, these cases are usually ones with a peak CK of <1,300 U/l after the procedure and a residual dynamic gradient in the catheterization laboratory of >25 mm Hg (5). Repeat alcohol ablation is possible in many patients. However, in the absence of septal arteries that supply the culprit septal segments, surgical myectomy becomes the only alternative. This was the case in 11 patients in whom, after the first ablation, MCE showed that the cannulated septal arteries do not supply the culprit septal segments. In addition, it is possible to entertain the possibility of mitral valve surgery in patients in whom residual septal thickness prohibits septal resection (which was not needed in the present series). Therefore, surgery can offer a number of viable options in these challenging cases.

Aside from patients who actually undergo alcohol septal ablation and subsequent myectomy, there are few other cases in whom the interventional approach is not feasible altogether. These include the very few patients (1.3% in our experience) where cannulation of the left main is challenging because of a tortuous aorta and/or peripheral vascular disease and in patients with very small septal perforators that are not amenable to intervention. These patients were not included in the present report by design, because our aim was to study the outcome of surgery after unsuccessful alcohol ablation.

**Surgical myectomy after alcohol septal ablation.** Surgery provides a direct approach to the treatment of dynamic obstruction compared with alcohol ablation. Therefore, as the primary treatment modality for HOCM, surgery in experienced centers has a higher initial success rate (≈98%) than alcohol ablation (88% in the present series).

In this particular setting, few studies have been published noting the need for surgery after an unsuccessful alcohol septal ablation (6,7). These 2 studies have reported on a total of 6 patients. Both reports noted that surgery was successful in relieving residual obstruction and in affecting symptomatic improvement. Therefore, surgery offers more options than alcohol ablation after an initial unsuccessful procedure. Depending on the patient’s preferences and physician’s experience and the unique status in any given situation, surgery may be considered in lieu of a second percutaneous approach.

However, there are potential concerns about adverse consequences in this setting with respect to LV function and also regarding dysrhythmia. In the present study, LVEF was not adversely affected by surgery after alcohol ablation, although additional studies with a larger number of patients are needed to reliably address this question.

Regarding complete AV block, the present observations are somewhat similar to the published experience in 6 patients (6,7) where pacing was needed after surgery after unsuccessful alcohol ablation. In the present series, 3 pacemakers were implanted before surgery and 2 were needed because of complete AV block after surgery. Given the development of left bundle branch block in many patients after surgery, patients who develop right bundle branch block after alcohol ablation have a higher likelihood of complete AV block after surgical myectomy. However, the rate of permanent pacing (10%) appears slightly higher than with surgery as the primary treatment modality and is close to that occurring after a first alcohol septal ablation.

**Table 1 Clinical and Echocardiographic Data After Alcohol Septal Reduction Therapy and Surgical Myectomy**

<table>
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<th>Baseline</th>
<th>After Alcohol Septal Ablation</th>
<th>After Myectomy</th>
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<tr>
<td>NYHA functional class</td>
<td>3 (3-3)*</td>
<td>2.5 (2-3)†</td>
<td>1 (1-1)</td>
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<tr>
<td>CCS angina class</td>
<td>2 (1-3)*</td>
<td>1.6 (1-2)†</td>
<td>1 (1-1)</td>
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<tr>
<td>Exercise duration (s)</td>
<td>171 ± 124†</td>
<td>166 ± 148†</td>
<td>423 ± 171</td>
</tr>
<tr>
<td>LVOT gradient (mm Hg)</td>
<td>93 ± 23*</td>
<td>71 ± 26†</td>
<td>6 ± 11</td>
</tr>
<tr>
<td>Septal thickness (cm)</td>
<td>2.3 ± 0.4*</td>
<td>1.9 ± 0.3†</td>
<td>1.3 ± 0.3</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>74 ± 6</td>
<td>71 ± 6</td>
<td>70 ± 5</td>
</tr>
<tr>
<td>Mitral regurgitation</td>
<td>1.5 (1-2)*</td>
<td>1.25 (1-1.5)†</td>
<td>0 (0-1)</td>
</tr>
</tbody>
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Data are shown as mean ± SD or median (25th to 75th percentiles); *p < 0.05 for baseline versus post-alcohol septal ablation and versus post-myectomy. †p < 0.05 versus post-myectomy.

CCS = Canadian Cardiovascular Society; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; NYHA = New York Heart Association.
potential complications should be discussed with patients when treatment plans are being considered.

Study limitations. There were only 20 patients who underwent both procedures. Therefore, this study is subject to sampling errors, and additional data with more patients are needed to have a narrow confidence interval with respect to the success and complications of surgery in this setting.

Surgical myectomy was performed mostly by a single experienced surgeon, and the management of these patients can be challenging, not only during the operation, but also in the postoperative period. Therefore, these results should only be extrapolated when the treatment of HOCM is performed at dedicated centers and may not be extrapolated to centers with limited experience.

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Reprint requests and correspondence: Dr. Sherif F. Nagueh, 6550 Fannin Street, SM-667, Houston, Texas 77030-2717. E-mail: snagueh@tmh.tmc.edu.

REFERENCES

APPENDIX

For an accompanying video, please see the online version of this article.
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