INTRODUCTION

Hypertrophic cardiomyopathy is a common genetic disorder that can lead to significant morbidity and mortality, particularly in patients with dynamic left-ventricular outflow tract obstruction (HOCM). Alcohol septal ablation (ASA) was recently introduced for the treatment of HOCM. It was first reported in 1995 by Dr. Ulrich Sigwart, and a year later Dr. William H. Spencer, III performed the first procedure at The Methodist Hospital. Since then, several publications around the world have confirmed the procedure’s efficacy and safety, and a vast majority of patients elect to undergo ASA as an alternative to surgery. This article summarizes several aspects related to ASA ranging from case selection to its comparison with surgical myectomy.

CASE SELECTION

The usual ASA candidates are severely symptomatic patients who, despite adequate medical therapy, suffer from HOCM, as well as severe dyspnea and/or angina—with a resting gradient ≥ 30 mmHg or a provocable gradient ≥ 60 mmHg. For those able to exercise, exercise echocardiography can be used to determine the magnitude of left-ventricular outflow tract obstruction. For those unable to exercise, dobutamine echocardiography may be used to provoke obstruction (up to 20 mcg/kg/min), akin to the use of isoproterenol. It is important to identify the site and mechanism of obstruction, as well as the etiology of mitral regurgitation. This has a direct impact on the selection of therapy since patients with either valvular or congenital pathology (or both) should be referred for surgery.

TECHNICAL ASPECTS

This catheter-based treatment involves the injection of 1 to 3 ml of absolute ethanol into one or more of the septal perforator branches of the LAD coronary artery. The vessel selected is the one identified by intracoronary myocardial contrast echocardiography (MCE) to supply the area of the septum (Figures 1 and 2), causing the dynamic outflow tract obstruction. The injection of absolute ethanol results in acute occlusion of the septal perforator artery and, consequently, septal stunning and/or infarction that can be quantified by the risk area on MCE and the CK serum level. For patients without a permanent pacemaker, a temporary pacing wire is placed in the RV apex at the beginning of the procedure to provide backup pacing should a high-grade AV block occur. Patients with coexisting multi-vessel coronary artery disease are usually referred for surgery to address both CAD and HOCM. However, it is possible to successfully perform percutaneous coronary intervention in addition to ASA in selected cases. While ASA appears to be an easy procedure, there is a steep learning curve and a number of serious problems can develop. Therefore, an experienced team is essential for achieving good results with minimal complications.

ASA EFFICACY AND SAFETY

ASA results in a deterioration of basal septum function and a delay in left ventricular (LV) ejection, which lead to an acute reduction in LV outflow tract gradient and in severity of mitral regurgitation. The lowest gradients are typically recorded at six months to a year after the procedure due to the ensuing septal thinning and fibrosis that in turn lead to widening of the LV outflow tract. We have had an overall success rate of 94% with significant improvement in the severity of dyspnea and angina accompanied by an increased exercise tolerance. The improvement has been sustained at two to five years of follow up. Reducing LV dynamic outflow tract obstruction frequently produces other favorable changes including regression of LV hypertrophy, improvement of LV diastolic function and reduction in left-atrial volume and stroke work.

While the most serious procedural complications can include coronary artery dissection and advanced AV block, the complication rate in experienced centers is low. In comparison with earlier reports, the current rate of permanent pacing after ASA is lower, occurring at 5 to 10%. The determinants of AV block after ASA include the number of occluded septal arteries (> 2), bolus injection of ethanol, female gender and left bundle branch block. Concerns have been raised about the risk of ventricular dysrhythmia after ASA due to...
the development of a myocardial scar. However, published reports have demonstrated the inability to induce sustained ventricular tachycardia after ASA, and our own data do not show a higher rate of sudden cardiac death up to seven years after the procedure. Longer-term data with a larger number of patients are recommended to reliably address these concerns.

**COMPARISON WITH SURGICAL MYECTOMY**

A number of studies have been published comparing the outcomes of ASA with surgical myectomy. Only one study compared patients who were matched for age and severity of dynamic outflow tract obstruction, and it showed no significant differences in NYHA class, severity of mitral regurgitation, use of cardiac medications or exercise capacity at baseline between patients who were treated with ASA and those with surgical myectomy. For ASA performed at the Methodist DeBakey Heart Center/Baylor College of Medicine, we noted that residual gradient, symptomatic improvement and exercise tolerance at one year post-procedure were essentially similar to those of surgical myectomy performed at the Mayo Clinic. While there was no significant difference in mortality between ASA and surgery, high-grade AV block was more common after ASA. However, seven of the nine pacemakers in the ASA group were implanted prior to modification of the ethanol injection technique and the use of MCE. As previously known, atrial fibrillation developed after surgery and the incidence of aortic regurgitation was significantly higher after surgery.

**CONCLUSION**

In experienced hands, ASA can be done safely with good results and minimal complications. ASA is comparable to surgery in providing symptom relief and improving exercise tolerance in the majority of patients, with benefits persisting at long-term follow up.

**REFERENCES**