Catheter Treatment for Hypertrophic Obstructive Cardiomyopathy
For Seniors Only?

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Hypertrophic cardiomyopathy is a relatively recent concept. It occurs not infrequently; \( \approx \)1 in 500 individuals within the general population is a carrier of the disease.1 The main genes responsible for hypertrophic cardiomyopathy have been characterized and can be determined with relative ease.2 Some patients develop subaortic obstruction. The original term, idiopathic hypertrophic subaortic stenosis, dates to 1964;3 since then, it has become obvious that the obstructive component has its own prognostic meaning. The obstruction occurs in \( \approx \)25% of all humans with hypertrophic cardiomyopathy,4 and the entity is now called hypertrophic obstructive cardiomyopathy.

Surgery for elimination of the obstructive element via resection of the subaortic septal bulge (myectomy) has been, since its introduction by Cleland at the Brompton in London exactly half a century ago, the only rational option for patients with clinically relevant left ventricular outflow tract obstruction that is refractory to optimal medical therapy. At experienced centers, the operation can be performed with low mortality and excellent clinical improvement.5

The concept of using transluminal techniques to reduce septal hypertrophy in patients with hypertrophic obstructive cardiomyopathy was not based on the hope of achieving better results than with surgery, but primarily to reduce morbidity. In the early 1980s, while performing coronary angiography on patients with hypertrophic obstructive cardiomyopathy, I noticed that in some patients, the first septal perforator branches appeared to irrigate primarily the area of the septal bulge responsible for outflow tract obstruction. During systole, these branches seemed to be compressed by the surrounding hyperabundant myocardium. Interfering with the proper irrigation of this territory became only logical with the advent of intracoronary manipulations, and the temporary gradient reduction observed when a regular angioplasty balloon was inflated inside the first septal perforator supported this hypothesis.

Afterward, primarily because of legal issues, the concept of inducing a septal infarct hibernated for >10 years. After this period, a patient underwent catheter-based nonsurgical septal reduction, again at the now “Royal” Brompton Hospital, on June 16, 1994.6 Before this intervention, the patient had undergone implantation of a DDD pacemaker, and she was receiving all medical therapy known at that time to reduce her impressive symptoms. This patient is still alive and is asymptomatic.7

Meanwhile, >5000 patients have submitted to catheter-based septal reduction for this disease. The number of patients who have had surgical myectomy remains between 3000 and 4000. In other words, catheter-based septal reduction has moved into the fast lane, while the “gold standard” (myectomy) has continued cruising at steady speed.

In 1995, I wrote to several centers (including the Mayo Clinic) asking whether there would be interest in a randomized trial comparing surgical myectomy with alcohol ablation. The answer was an enthusiastic yes in every instance. Unfortunately, this trial never came about, for 2 reasons: patients’ preference and, equally important, the lack of true collaboration between cardiologists and surgeons. Some registry entries that compare myectomy with catheter-ablation outcomes have been published, with divergent results.8,9 All of these registries are limping, for reasons that include bias introduced by patient preference, less-than-adequate exchange of results between competing departments, and perhaps most importantly, rather aggressive patient recruitment for catheter ablation.

In this issue of Circulation, Sorajja et al10 argue that catheter-based septal reduction may result in less-pronounced relief of gradients, and that septal ablation cannot be advocated as therapy to replace surgical myectomy, particularly in younger patients. Sorajja et al suggest that “septal ablation can be viewed as a less-invasive therapy for patients with drug refractory, severe symptoms due to obstructive hypertrophic cardiomyopathy.” I fully subscribe to that statement.

On the other hand, their suggestion that “an integrated team approach with the ability to offer either percutaneous or surgical option is essential in the managements of these patients” will probably remain hypothetical as long as very few centers obtain the surgical results published by the Mayo Clinic group and, as pointed out in the article, patient preference—for whatever good reason—remains a major decision factor.

I have a strong suspicion that many patients undergoing catheter-based septal ablation are less-than-ideal candidates for this procedure. When a patient expresses preference to avoid classic open-heart surgery, most cardiologists familiar with the catheter-based procedure will try everything to

The opinions expressed in this article are not necessarily those of the editors or of the American Heart Association.

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Circulation is available at http://circ.ahajournals.org
DOI: 10.1161/CIRCULATIONAHA.108.790865

Editorial
perform some sort of septal ablation, even if they have the impression that the final outcome will be inferior to that obtained with surgical myectomy. No one on earth can expect the results to be as good as after open-heart surgery in these patients. Nevertheless, the results of such patients continue to be introduced into the catheter arm of such registries. As Sorajja et al.10 correctly state, "unlike septal myectomy, where direct visualization guides myocardial removal, septal ablation is entirely dependent on the vascular bed of visible [and I would add, accessible] septal perforator arteries." This is the heart of the matter. Even when a patient has a strong preference to undergo a catheter procedure rather than open-heart surgery, he or she must come equipped with the appropriate arteries to make such a procedure possible. If the septal supply mainly comes from the posterior descending coronary artery, the usual procedure is useless. Sometimes multiple, small, septal branches supply the target area and require multiple individual ethanol injections. Also, procedural factors may play a role. If visible collaterals communicate with the target myocardium, a slow infusion of 1 to 3 mm of desiccated ethanol over a 3- to 5-minute period, as advocated by Sorajja et al.9 is unlikely to produce a localized infarct of the desired area. Even if the magnitude of enzyme release had been reported in this article (which it is not), the true infarct size could not accurately be estimated under such circumstances, as ethanol may contaminate other territories and create myolysis elsewhere. Also, the catheter arm of the Mayo series contains 27 patients in whom the surgical risk was believed to be significantly increased. Patients who had septal ablation had a higher prevalence of hypertension and coronary artery disease. Nevertheless, the outcome for such patients has been compared with that of myectomy patients. The higher complication rate in the catheter group can be attributed to the higher number of pacemaker implantations, which was clearly more generous than in other series, and to procedural factors that have since been corrected.

Sorajja et al.10 conclude that “in patients <65 years old, symptom relief is better after myectomy.” I beg to differ. In my mind, this sentence should instead read, “in patients with unfavorable coronary distribution, relief is better after myectomy.” Also, it remains debatable whether a retrospective analysis is the correct substrate for such a statement. Without a randomized trial whereby only patients with adequate anatomic conditions allowing effective catheter ablation are compared with patients undergoing myectomy, no definitive statement should be made.

Having seen, over the last 14 years, many patients of all ages with favorable anatomy for catheter ablation who responded extremely well to catheter ablation (and continue to do so), I cannot but advocate a true randomized trial comparing those 2 treatment options. In such a trial, only patients with favorable anatomy should be included in the catheter arm. Whether such an undertaking is realistic in view of patients’ preferences and with respect to the large number of confounding factors remains unclear.

The procedure, catheter ablation for hypertrophic obstructive cardiomyopathy, was never devised to replace surgery for symptomatic patients with hypertrophic obstructive cardiomyopathy. It was intended to provide those patients, young and old, who have favorable (and accessible) anatomy, with an alternative to open heart surgery through the induction of a meaningful septal necrosis. Plenty of evidence now exists to make us believe that this concept will hold. More-detailed statements must be postponed until they are ascertained through a true randomized trial.

Disclosures
None.

References


Key Words: Editorials ■ cardiomyopathy, hypertrophic ■ catheter ablation ■ surgery
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_Circulation_. 2008;118:107-108
doi: 10.1161/CIRCULATIONAHA.108.790865
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2008 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/118/2/107

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