Septal myectomy: cut, coil, or boil?

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This editorial refers to ‘Non-surgical septal myocardial reduction by coil embolization for hypertrophic obstructive cardiomyopathy: early and 6 months follow-up’ by E. Durand et al.,† on page 348

Hypertrophic cardiomyopathy (HOCM) is characterized by asymmetric septal hypertrophy with outflow tract obstruction in approximately one-third of patients. However, recent studies suggest that obstruction may be present in up to 70% of patients with enhanced sympathetic activation under exercise conditions.1 Clinical features are dyspnoea on exertion, angina pectoris, and atrial and ventricular arrhythmias, which mainly are due to diastolic dysfunction, whereas syncope and presyncope often are due to outflow tract obstruction and represent an ominous sign of ventricular arrhythmias and sudden cardiac death.

The treatment strategy is 3-fold: (i) medical therapy for targeting diastolic dysfunction; (ii) myectomy (interventional or surgical) for relief of obstruction; and (iii) implantable defibrillators for prevention of sudden cardiac death.2

For decades, the gold standard for septal myectomy has been surgical treatment, with excellent short- and long-term results.3 In 1995, a new interventional technique (alcohol ablation of the septum) was put forward for percutaneous treatment of HOCM.4 In their study, Durand and co-workers5 propose a new technique for septal ablation of the myocardium, namely coiling of septal branches. One to five coils were deployed into the first or second septal branch of the left anterior descending artery in 20 patients with HOCM, thereby inducing septal ischaemia with myocardial necrosis and a consecutive creatine kinase (CK) rise of 386 U/L. As a result, the pressure gradient decreased significantly from 80 to 35 mmHg at the 6 months follow-up examination. Clinical symptoms and exercise capacity improved, as reflected by a significant increase in peak oxygen consumption (from 14.8 ± 4.5 to 18.5 ± 4.5 ml/kg/min) and a prolongation of exercise duration (from 7.1 ± 3.5 to 9.0 ± 3.8 min). The procedure was well tolerated, and no AV blocks were observed. However, in one patient, septal perforation occurred, which was surgically treated; however, the patient died postoperatively. Other complications were not reported, and the authors claim from this series that in contrast to alcohol ablation or surgical myectomy, no AV blocks occur with coil embolization. Nevertheless, they suggest that larger studies, ideally employing a randomized comparison between coil embolization and alcohol septal ablation, are warranted.

The authors are to be congratulated for these results and this new technique. A major limitation of alcohol ablation is, indeed, an AV block, which occurs in 27% of all patients (transient); ~10% need pacemaker implantation (Table 1). However, previous data suggest that infarct size is larger with alcohol ablation (10–15 g) compared with 3 g with coil embolization. This finding is paralleled by a larger CK rise with alcohol ablation (1038 U/L) when compared with coil embolization (386 U/L). Alcohol appears to penetrate more deeply into the septal region and induces a larger area of necrosis, whereas coil embolization leads primarily to ischaemia and only secondarily to necrosis. However, in some rare cases, alcohol dissipation (spill over) to non-target myocardial areas (right ventricle or apex of the left ventricle) may occur.

In Durand et al.’s pilot study, a quarter of the patients had unsuccessful treatment with a resting gradient >50 mmHg at 6 months. This reflects the learning curve, as stated by the authors, or indicates the less aggressive nature of the technique. Alcohol ablation has been reported to be successful in 90–95% of all patients.

As an alternative, radiofrequency ablation has been recommended for reduction of septal hypertrophy in children with HOCM, because alcohol ablation in this patient group is strongly discouraged due to the induction of potential arrhythmias.6 From reviewing the literature and comparing alcohol ablation with coil embolization (Table 1), there are clear differences in the release of CK and the decrease in outflow tract gradient, suggesting a larger infarct with alcohol ablation than with coil embolization. The absence of AV block with coil embolization could be explained by the smaller infarct size and the modest decrease in pressure gradient.

Interestingly enough, a Polish group7 performing coil embolization in patients with HOCM reported a transient AV block in 43% of patients (Table 1). Thus, Durand’s study may represent a highly selected group that does not show AV block during coil embolization. Larger samples may answer this question.

Only a randomized trial will allow for a fair comparison of the two techniques. A contraindication for alcohol ablation (and an...
indication for coil embolization) may represent pre-existing left bundle branch block (LBBB). A complete AV block may occur in these patients when alcohol ablation induces right bundle branch block (RBBB) in up to 50% of patients. Amiodarone treatment is another contraindication due to associated AV conduction prolongation. A third contraindication to alcohol ablation is the pre-existing AV block I or II (Figure 1).

In summary, coil embolization represents a new and promising method for reducing septal hypertrophy in patients with HOCM. The first results suggest a lower risk for the occurrence of AV block during septal ablation, but haemodynamic success may be limited, with less reduction in outflow tract gradient.

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### References


