

Discount treatment for HOCM: as good as surgery?

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This editorial refers to ‘Long-term clinical outcome after alcohol septal ablation for obstructive hypertrophic cardiomyopathy: results from the Euro-ASA registry’[†], by J. Veselka et al., on page 1517.

At a recent cardiology meeting in Geneva, one of my former colleagues quoted a remark I had made in the early 1980s during a heart-team meeting on hypertrophic obstructive cardiomyopathy (HOCM) at the University of Lausanne: ‘Wouldn’t it be much cheaper and less traumatic for the patient to create an infarct at the site of the septal bulge rather than opening the chest and removing the obstructive myocardium?’

This remark was based on the observation we had made earlier that short balloon inflations inside the major proximal septal artery resulted in disappearance of the intraventricular pressure gradient in patients with symptomatic HOCM.

More than 30 years ago, the time was not right for such a daring intervention. The project lay dormant until I made the same remark at the Royal Brompton Hospital grand rounds in London ~10 years later. It was a senior pathologist, Bob Anderson, who, after brief reflection, replied: ‘Sounds logical, why don’t you do it?’

This time, in contrast to the previous attempts, the local Ethics Committee responded favourably to my request and gave the green light for three preliminary cases of creation of septal infarction in highly symptomatic HOCM. It took another couple of years to recruit the first patient who was desperate enough to embark on such an uncharted adventure.¹

The method of alcohol septal ablation (ASA) has now replaced surgical myectomy in a significant proportion of symptomatic patients with HOCM. Numerous attempts have been made to compare the outcome of ASA with surgical myectomy.² Most publications point towards a lower requirement for permanent pacing and lower residual gradients after myectomy, suggesting a more predictable way of eliminating the outflow tract obstruction. The choice of the best septal reduction therapy, however, continues to be a

matter of debate.^{3,4} The European and American guidelines recommend, with Class IIa indication (level of evidence B), surgery as ‘first consideration for majority of patients with HOCM’. ASA is, however, also recommended as a Class IIa indication (level of evidence B) when ‘surgery is contraindicated or the risk is considered unacceptable’ because of serious co-morbidities or advanced age.^{5,6} These guidelines are based on the assumption that myectomy in lower risk surgical candidates carries a low risk of peri-operative mortality, comparable with ASA, whereas in higher risk populations myectomy might carry an unreasonably high risk. These recommendations are based on non-randomized self-reported data from four high-volume HOCM centres in the USA. The European guidelines mirror these statements.⁵

In this issue of the journal, the long-term outcome of either treatment option from seven European centres have been presented.⁷ This European prospective registry of a total of 1275 highly symptomatic, consecutive patients treated with alcohol ablation is at present the largest of its kind. Up to now, only the North American ASA Registry,⁸ which included 874 patients, comes close. It had similar survival data but very different predictors of survival (lower ejection fraction, smaller number of septal perforators, larger number of repeat procedures, greater septal thickness, and post-procedural use of beta-blockers).

Although all procedures in the European registry were performed in proficient tertiary invasive centres from seven European countries between January 1996 and February 2015, the methods were not standardized and varied considerably among operators. There seems to be a wide variety of procedural details employed by the different operators, at least within the group of patients submitted to ASA. A total of 257 patients received between 3 and 11 mL of absolute alcohol, whereas the volumes normally used nowadays do not exceed 2 mL. The speed of injection is ill defined and the choice of the septal supply vessels is vaguely analysed. On the other hand, fairly standard techniques are employed for the patients submitted to myectomy despite the relatively small numbers at each of the seven centres.

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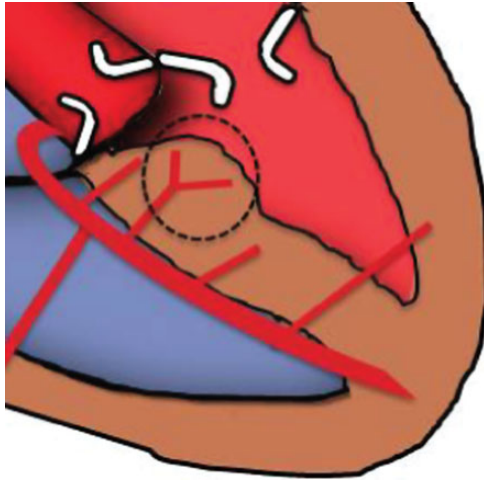


Figure 1 The result of alcohol septal ablation (ASA) depends entirely on the variable blood supply and its proper identification. Meticulous planning is essential to the success.

The most interesting finding of this study is the notion, in line with previous observations by Sorajja *et al.*⁹ and Jensen *et al.*,¹⁰ that the all-cause mortality seems to be independently associated with the magnitude of residual left ventricular outflow tract gradient. This discovery calls for efforts to try everything to optimize the outcomes. For the surgeon, such an endeavour seems straightforward; for the cardiologist, it demands clever ways of precisely identifying the supply vessels feeding the septal bulge. Such attempts require meticulous study of the different arterial structures involved in septal perfusion (Figure 1). The Liverpool group has recently been looking at this problem from different angles and has come up with intriguing suggestions.¹¹ Using computed tomography angiography (CTA) guidance prior to the procedure, a sub-branch of the artery was identified for alcohol injection. This allowed for a more precise infarct landing in the vicinity of systolic anterior movement of the mitral valve (SAM) septal contact, which in turn significantly reduced the resting gradients as compared with conventional techniques used up to now, i.e. angiography and targeted injection of echo contrast.

As the authors of the European Registry⁷ correctly state, there is still a knowledge gap with regard to post-procedural mortality and the risk/benefit ratio of ASA as compared with surgical myectomy. This multicentre, prospective registry helps to clarify the situation, although it cannot replace a randomized study. Such a trial would certainly require thousands of patients, and the logistics of such an endeavour seem literally out of range. In spite of HOCM being a relatively frequent abnormality, absolute figures are much lower when compared with the numbers of acquired heart disease patients with diagnoses such as coronary heart disease. The creation of one or two international centres of excellence able to perform ASA and myectomy equally well using standardized procedures among a highly trained group of experts seems far off, mainly due to fierce competition for the recruitment of patients.

The paper presented here is an important step in the right direction. It must be recognized, however, that HOCM is an entity with many faces requiring an individual approach to every single patient: estimation of the risk of clinically pertinent arrhythmia,¹⁰ grading of symptoms, definition of septal anatomy and its supply, implication of mitral valve cordae, co-morbidity, age, frailty, etc. The suggestion that there is one ideal way of treating such a complicated condition is an illusion, and personal ambition by individual doctors trying to get involved is not acceptable. Team work in decision-making which would be the best way of helping the individual patient is essential, and surgeons and interventional cardiologists must work hand in hand, ideally within the same team.

Although ASA is relatively cheap and seems to compete reasonably well with myectomy in many, if not most, cases, a 'discount solution' for the treatment of HOCM cannot possibly be the best bargain for all patients!

Conflict of interest: none declared.

References

1. Sigwart U. Non-surgical myocardial reduction for hypertrophic obstructive cardiomyopathy. *Lancet* 1994;**22**:346:211–214.
2. Alam M, Dokainish H, Lakkis NM. Hypertrophic obstructive cardiomyopathy-alcohol septal ablation vs. myectomy: a meta-analysis. *Eur Heart J* 2009;**30**: 1080–1087.
3. Maron MS, Olivetto I, Betocchi S, Casey SA, Lesser JR, Losi MA, Cecchi F, Maron BJ. Effect of left ventricular outflow tract obstruction on clinical outcome in hypertrophic cardiomyopathy. *N Engl J Med* 2003;**348**:295–303.
4. Maron BJ, Maron MS. Hypertrophic cardiomyopathy. *Lancet* 2013;**381**:242–255.
5. Elliott PM, Anastasakis A, Borger MA, Borggreffe M, Cecci F, Charron P, Hagege AA, Lafont A, Limogelli G, Marholdt H, McKenna WJ, Mogensen J, Nihoyannopoulos P, Nistri S, Pieper PG, Pieske G, Rapezzi C, Rutten FH, Tillmanns C, Watkins H. 2014 ESC Guidelines on diagnosis and management of hypertrophic cardiomyopathy: the Task Force for the Diagnosis and Management of Hypertrophic Cardiomyopathy of the European Society of Cardiology (ESC). *Eur Heart J* 2014;**35**:2733–2779.
6. Gersh BJ, Maron BJ, Bonow RO, Dearani JA, Fifer MA, Link MS, Naidu SS, Nishimura RA, Ommen SR, Rakowski H, Seidman CE, Towbin JA, Udelson JE, Yancy CW; American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. ACCF/AHA Guideline for the Diagnosis and Treatment of Hypertrophic Cardiomyopathy: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Developed in collaboration with the American Association for Thoracic Surgery, American Society of Echocardiography, American Society of Nuclear Cardiology, Heart Failure Society of America, Heart Rhythm Society, Society for Cardiovascular Angiography and Interventions, and Society of Thoracic Surgeons. *J Am Coll Cardiol* 2011;**58**:e212–e260.
7. Veselka J, Jensen MK, Liebrechts M, Januska J, Krejci J, Bartel T, Dabrowski M, Hansen PR, Almaas VM, Seggewiss H, Horstkotte D, Tomasov P, Adlova R, Bundgaard H, Steggerda R, Ten Berg J, Faber L. Long-term clinical outcome after alcohol septal ablation for obstructive hypertrophic cardiomyopathy: results from the Euro-ASA registry. *Eur Heart J* 2016;**37**:1517–1523.
8. Nagueh SF, Groves BM, Schwartz L, Smith KM, Wang A, Bach RG, Nielsen C, Lea F, Buegler JM, Rowe SR, Woo A, Maldonado YM, Spencer WM. Alcohol septal ablation for the treatment of hypertrophic obstructive cardiomyopathy. A multicenter North American registry. *J Am Coll Cardiol* 2011;**58**:2322–2328.
9. Sorajja P, Ommen SR, Holmes DR, Dearani JA, Gersh BJ, Lennon RJ, Nishimura RA. Survival after alcohol septal ablation for obstructive hypertrophic cardiomyopathy. *Circulation* 2012;**126**:2374–2380.
10. Jensen MK, Prinz C, Horstkotte D, van Buuren F, Bitter Th, Faaber L, Bundgaard H. Alcohol septal ablation in patients with hypertrophic obstructive cardiomyopathy: low incidence of sudden cardiac death and reduced risk profile. *Heart* 2013;**99**: 1012–1017.
11. Cooper RM, Binukrishnan S, Shahzad A, Hasleton J, Sigwart U, Stables RH. Computed tomography angiography planning improves localisation of iatrogenic infarct and procedural success in alcohol septal ablation for HOCM. *J Am Coll Cardiol* 2015; **65**:A974