

Pressure gradient response after alcohol septal ablation in hypertrophic obstructive cardiomyopathy

Dear Editor,

Recently I read the article by Yoerger et al. titled *Time course of pressure gradient response after first alcohol septal ablation for obstructive hypertrophic cardiomyopathy* published in the American Journal of Cardiology [1]. The authors concluded that many patients who undergo ultimately successful alcohol septal ablation (ASA) for obstructive hypertrophic cardiomyopathy demonstrate triphasic pressure gradient response patterns, with a large gradient three days after the procedure. They postulated that "the acute decrease in pressure gradient reflects a loss of septal contractility caused by ischemia, necrosis, and stunning of septal myocardium and that the early increase in pressure gradient after ASA reflects the recovery of septal myocardium of stunning". However, I have a few issues with the report.

First, the phasic time course of the post-procedural outflow pressure gradient was described by our group 3 years ago. Our original study was presented at the European Congress of Cardiology in Vienna, 2003, and subsequently was published in the Polish Heart Journal [2, 3]. We hypothesized that the rapid post-procedural pressure gradient decrease is caused mainly by stunning and myocardial necrosis. The main mechanism of later continuous pressure gradient relief after ASA is the remodelling (widening) of the left ventricular outflow tract developing secondary to infarction and fibrosis of the basal septum. This process is apparent by several weeks after the procedure and in some cases develops at long-term follow-up [2, 3]. The early biphasic course (characterized by "down-up-down" changes in the pressure gradient; called "triphasic" course by Yoerger et al.) occurred in 87% of patients in our original study and 13% of patients were characterized by monophasic post-procedural haemodynamic course. Time course of the pressure gradient was characterized by initial gradient relief from 73 ± 49 to 13 ± 16 mmHg ($p < 0.01$) followed by increase to 37 ± 35 (3-5 days after procedure, $p < 0.01$) and continuous decrease to 25 ± 12 mmHg (3 weeks after procedure, $p < 0.01$) and 17 ± 14 mmHg at 3-month follow-up (NS) [3]. Thus, I am afraid that both the design of the study and the main conclusions and hypotheses demonstrated by Yoerger et al. have been published previously [2-5].

Second, in the presented study, one-year follow-up was available only for 37 patients and the authors demonstrated a "similar" pressure gradient in the monophasic and triphasic response groups (13 ± 20 vs 27 ± 24 mmHg, $p = \text{NS}$) [1]. However, the more than double pressure gradient in the triphasic group suggests that the monophasic response per se could be a predictor of successful haemodynamic effect of ASA. This hypothesis would need more prospective data because the number of patients being followed up was too small to determine the possible difference.

Third, a recent study published by Gietzen et al. suggested that repeat ASA have been performed as early as 8 to 12 days after "partially effective" first ablation to eliminate provokable pressure gradients [6]. I wish to support the conclusions of Yoerger et al. and comment on the management of early post-procedural pressure gradient increase in the majority of ASA patients. As was shown in numerous papers [1-5] and stated even in the ACC/ESC Clinical Expert Consensus Document on Hypertrophic Cardiomyopathy [7], early post-procedural haemodynamic changes are characterized by a phasic course. Thus, we believe that repeating the ASA procedure to eliminate the pressure gradient completely does not seem to be justified within the early post-procedural period. Moreover, as we demonstrated recently, the subgroup of younger patients treated by ASA is characterized by a thicker basal septum at baseline and slower haemodynamic improvement within the early post-procedural period [8, 9]. Thus, we feel that repeat of ASA should be postponed until at least 3 to 6 months after the first procedure.

Josef Veselka

References

1. Yoerger DM, Picard MH, Palacios IF, et al. Time course of pressure gradient response after first alcohol septal ablation for obstructive hypertrophic cardiomyopathy. *Am J Cardiol* 2006; 97: 1511-4.
2. Veselka J, Tesar D, Procházková S, et al. The time course of changes of left-ventricular outflow gradient after alcohol septal ablation for hypertrophic obstructive cardiomyopathy. *Eur Heart J* 2003; 24 (Suppl.): 559.
3. Veselka J, Duchonová R, Procházková S, et al. The biphasic course of changes of left ventricular outflow gradient after alcohol septal ablation for hypertrophic obstructive cardiomyopathy. *Kardiologia Polska* 2004; 60: 133-6.

4. Veselka J, Duchonová R, Procházková S, et al. Effects of varying ethanol dosing in percutaneous septal ablation for obstructive hypertrophic cardiomyopathy on early hemodynamic changes. *Am J Cardiol* 2005; 95: 675-8.
5. Veselka J, Procházková S, Duchonová R, et al. Alcohol septal ablation for hypertrophic obstructive cardiomyopathy: Lower alcohol dose reduces size of infarction and has comparable hemodynamic and clinical outcome. *Catheter Cardiovasc Interv* 2004; 63: 231-5.
6. Gietzen FH, Leuner CJ, Obergassel L, et al. Transcatheter ablation of septal hypertrophy for hypertrophic obstructive cardiomyopathy: feasibility, clinical benefit, and short term results in elderly patients. *Heart* 2004; 90: 638-44.
7. Maron BJ, McKenna WJ, Danielson GK, et al. American College of Cardiology/European Society of Cardiology Clinical Expert Consensus Document on Hypertrophic Cardiomyopathy. A report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *Eur Heart J* 2003; 24: 1965-91.
8. Veselka J, Duchonová R, Procházková S, et al. Alcohol septal ablation for obstructive hypertrophic cardiomyopathy: slower hemodynamic improvement and septum thickness reduction in younger patients. *Am J Cardiol* 2005; 95 (Suppl. 8A): 67-68A.
9. Veselka J, Duchonová R, Páleníková J, et al. Age-related hemodynamic and morphologic differences in patients undergoing alcohol septal ablation for hypertrophic obstructive cardiomyopathy. *Circ J* 2006; 70: 880-4.

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Response to the letter to the editor

We thank Dr. Veselka for his interest in our work, and would like to recognize his important contributions to the septal ablation literature. In response to issues raised by the author, we would like to point out that we presented our findings first describing this triphasic pressure gradient response after alcohol septal ablation in 2002 at the American College of Cardiology meeting in Atlanta [1]. Our observation was that there were 2 distinct types of positive responders to ablation: those with immediate and sustained reductions after ablation and those noted to have a triphasic response [2]. We have not been able to demonstrate a statistically significant difference in the degree of LVOT gradient reduction between the triphasic and monophasic response groups after their initial early divergence, but we have noted the absolute difference in mean gradients to which Dr. Veselka refers. More study is needed to understand whether the early triphasic response might predict individuals at risk for a rise in gradients or recurrence of symptoms late after ablation.

Sincerely,

Danita M. Yoerger and Michael A. Fifer

References:

1. Yoerger DM, Picard MH, Palacios IF, et al. Triphasic response to alcohol septal ablation. *J Am Coll Cardiol* 2002; 39: 157A.
2. Yoerger DM, Picard MH, Palacios IF, et al. Time course of pressure gradient response after first alcohol septal ablation for obstructive hypertrophic cardiomyopathy. *Am J Cardiol* 2006; 97: 1511-4.

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