Alcohol relaxes the stiff heart

Hisham Dokainish and Nasser Lakkis*

Department of Medicine, Section of Cardiology, Baylor College of Medicine, Houston, TX, USA

Online publish-ahead-of-print 26 June 2006

This editorial refers to ‘Sustained improvement in left ventricular diastolic function after alcohol septal ablation for hypertrophic obstructive cardiomyopathy’† by D.S. Jassal et al., on page 1805

Dynamic obstruction of the left ventricular outflow tract (LVOT) has been related to symptoms of dyspnoea, angina, and syncope in patients with hypertrophic obstructive cardiomyopathy (HOCM). In 1995, Sigwart1 introduced an ingenious catheter-based technique for septal reduction by infusing a small amount of absolute ethanol into the first or second septal arteries supplying the obstructing septal bulge. Alcohol induces a well-demarcated septal base scar corresponding to, on average, 9% of the left ventricle (LV), which can be seen by SPECT imaging.2 This new technique, known as alcohol septal ablation (ASA), has gained popularity by interventional cardiologists in the USA and Europe.

Multiple reports from different parts of the world have consistently showed that, in expert hands, this technique results in significant immediate relief of LVOT obstruction, septal thinning, and regression of LV hypertrophy.2,3 These results are associated with sustained improvement in exercise tolerance (with decreased need for medications), peak oxygen consumption, cardiac index, and reduction in mean pulmonary arterial pressure. The mechanisms responsible for these favourable results are not fully elucidated yet. We and others have reported on the physical changes in septal contraction after ablation, thus reducing subaortic narrowing in systole and resulting in slower acceleration and a later peak ejection velocity.4 These changes are usually followed by geometric changes in the LV, resulting in a more parallel angle between the ejection flow and the mitral valve, with lower drag forces that lead to SAM. Other reports showed that ASA results in: (i) LV remodelling, including reduction in LV mass;5,6 (ii) increase with LV diastolic dimensions;2 and (iii) reduction in mitral regurgitation (MR) severity.2

In this issue, Jassal et al.7 report on non-invasive evaluation of LV diastolic function in 30 consecutive patients with symptomatic HOCM at baseline and at 1 and 2 years after ASA. Each patient underwent comprehensive echocardiographic examinations at baseline and on follow-up. As expected, all patients enjoyed a sustained and significant reduction in LVOT gradient and heart failure symptoms up to 2 years of follow-up. These changes were associated with significant reduction in septal thickness, increase in LV end-diastolic volumes, and decrease in MR severity.

Using previously validated, load-independent, tissue-Doppler indices for the assessment of diastolic function in HOCM,6 the authors demonstrated a significant increase in both early diastolic (E') and atrial contraction (A') at the lateral region of the mitral annulus and an increase in flow propagation velocity (Vp), as determined by the slope of peak velocity of early diastolic filling flow on the colour M-Mode image, suggesting improved myocardial relaxation at 1 and 2 years after ASA. Similarly, deceleration time and isovolumic relaxation time were prolonged at baseline and normalized after ASA, most likely due to an improvement in myocardial relaxation and not due to a rise in left atrial (filling) pressures, as evidenced by decreased left atrial volume, decreased MR, and decreased left atrial pressure after ASA. The observed changes in diastolic indices suggest improved LV relaxation and a decrease in LV and left atrial filling pressures, all of which contribute to improved symptoms and exercise tolerance. These observations regarding improvement in LV diastolic function and a decrease in symptoms at 1 and 2 years are consistent with the ones made by our group at 6 months post procedure.9

Multiple mechanisms may be responsible for the significant and sustained improvement in myocardial relaxation up to 2 years after ASA: (i) relief of the obstruction decreases systolic contraction load and improves inflow–outflow synchrony at the outflow tract, both result in improved relaxation; (ii) improved myocardial fibre lengthening early in diastole due to reduced hypertrophy and increased LV dimensions, both promote LV filling; (iii) LVOT obstruction relief results in higher aortic diastolic pressure along with a lower LV end-diastolic pressure. Both changes increase coronary perfusion pressure and blood flow, thus possibly minimizing ischaemia and increasing coronary filling, which aids relaxation during isovolumetric relaxation time; (iv) decreased collagen synthesis, which decreases LV stiffness and improves relaxation; (v) decreased afterload due to relief of LVOT obstruction resulting in decreased hypertrophy.

In conclusion, ASA ameliorates symptoms of HOCM and results in long-term improvement in myocardial diastolic parameters. It is unclear whether these positive changes occur in all patients undergoing ASA, or whether they result in improved long-term survival. It is hoped that in the near
future, we will see data on changes in systolic function after ASA. Strain analysis using echocardiographic speckle imaging and MRI tissue tagging is a particularly attractive modality for such an evaluation.

Conflict of interest: none declared.

References