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Improvement of left ventricular diastolic function after alcohol septal ablation for obstructive hypertrophic cardiomyopathy? Yes, of course, but...: reply

We would like to thank Dr Veselka for his interest in our work and appreciate the opportunity to respond to his letter. Our article illustrated that successful alcohol septal ablation (ASA) for hypertrophic obstructive cardiomyopathy (HOCM) led to significant and sustained improvement (up to 2-year follow-up) in echocardiographic measures of diastolic function, which may contribute to improved functional status after successful ASA.

In a population of 30 patients with symptomatic HOCM treated by ASA, our study demonstrated that estimated left atrial pressure significantly decreased from baseline to 1- and 2-year follow-up. Although we estimated left atrial pressure, using the regression formula $E/E' \times 1.25 + 1.9$ proposed by Nagueh *et al.*, this equation has not been validated in a population with HOCM. However, we showed that not only did left atrial pressure improve, but also E/E' and E/V_p improved following ASA, suggesting an improvement in myocardial relaxation properties. The normalization of ventricular relaxation likely results from favourable ventricular remodelling observed following ASA, with both regression of hypertrophy and an increase in left ventricular end-diastolic volume.

Of the total population of 57 consecutive patients with HOCM who underwent ASA, 27 patients were excluded because of inability to deliver ethanol (six cases) and incomplete diastolic echocardiographic

parameters (21 cases). Of these 27 patients excluded, four patients required permanent pacing, as complete heart block is one of the major complications of ASA. In our final cohort of 30 patients, however, no subject had a permanent pacemaker. As such, our study was not affected by the fact that permanent pacing can change conventional and novel diastolic parameters.

We are interested in Dr Veselka's comments regarding sustained reduction in left atrial dimension. We would welcome the opportunity to review other data.

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Guidelines on the management of stable angina pectoris: executive summary: The Task Force on the Management of Stable Angina Pectoris of the European Society of Cardiology

Under the heading 'Hypertension, diabetes, and other disorders' on page 1358, the

guidelines¹ lean on the taskforce report on CVD prevention² suggesting 'considering a lower threshold for institution of pharmacological therapy for hypertension (130/85) for patients with established coronary heart disease (which would include patients with angina and non-invasive or invasive confirmation of coronary disease)'. Although this statement may be appropriate for the prevention of cardiovascular disease, hypertensive patients with manifest coronary artery disease need a more differentiated approach with regard to target blood pressure. On the basis of our recent findings of a subanalysis of the 22 000 patient INVEST study,³ we would like to caution about too aggressive blood pressure lowering in hypertensive patients with coronary artery disease. This holds particularly true for diastolic pressure and less so for systolic pressure. In our study, the nadir for primary outcome (all-cause death and total MI) was J-shaped, with a nadir at 119/84 mmHg.³ When diastolic pressure dropped below 70 mmHg, the adjusted hazard ratio of primary outcome doubled, and below 60 mmHg, it quadrupled. Since the coronary arteries are perfused during diastole only, it is not surprising that the coronary perfusion becomes hampered when diastolic pressure falls excessively in patients at risk, i.e. those with coronary artery disease. Thus, a cautionary statement, to the effect that low diastolic pressure should be avoided in hypertensive patients with coronary artery disease, would seem appropriate for the otherwise very thorough and comprehensive guidelines.

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cardiovascular death was inversely related to blood pressure (both systolic and diastolic) in contrast to the J-shaped relationships for cardiovascular and total mortality, leading the authors to hypothesize that poor health conditions leading to low blood pressure and an increased risk of death might in part explain the J-shaped curve.

Secondly, as discussed in the full-text version of the guidelines, there is accumulating evidence that blood pressure lowering in the 'normal' range is associated with improved cardiovascular outcomes in the population with known coronary disease. In the CAMELOT study, patients with coronary disease and mean blood pressure of 129/78 were randomized to enalapril, amlodipine, or placebo.² Blood pressure reductions were similar (5/2 mm) in both treatment groups and associated with similar relative reductions in the composite endpoint of cardiovascular death, MI, and stroke, although not statistically significant in either group because of the small sample size. An intravascular ultrasound substudy demonstrated a significant inverse correlation between progression of atherosclerosis and blood pressure reduction even in this normal blood pressure range, with the greatest benefit observed in patients whose blood pressure fell below 120/80.³

Thus, the task force has felt it important, in the absence of unequivocal evidence to the contrary, to preserve consistency between guidelines on prevention and angina with regard to targets for institution of therapy for hypertension in the presence of coronary disease. No lower limit has yet been identified as a definite cutoff beyond which blood pressure should not be lowered further, although, clearly, symptomatic hypotension or postural hypotension will limit aggressive blood pressure lowering in the lower range.

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The dangers of categorizing body mass index

Domanski *et al.* find body mass index (BMI) to be an independent-risk factor for major adverse coronary events (MACE) in men, but not in women. These results are first based on a dichotomization of BMI at 30 kg/m² and then on a further categorization of BMI into five pre-specified groups. These groupings, the first of which is based on guidelines presented by NIH¹ while the second closely mimics the categorization presented by the World Health Organization,² were developed for identifying subclasses of risk for general health concerns associated with obesity and not for any predictive ability to accurately model the relationship between BMI and the risk of the specified cardiac events. If weight categories of BMI are to be used, and there is an extensive literature suggesting that they should not,³⁻⁶ the categorization employed for the statistical analysis should reflect the nature of the association between the exposure BMI and the outcome MACE. The approach presented in Domanski *et al.*, since it is based on a pre-determined categorization, does not allow for an unrestricted assessment of the relationship between BMI and MACE. Furthermore, any categorization may find non-significant results due to low power induced by small counts in certain BMI groups. For example, the authors find a significant increase between the 'obese' and 'normal' groups of men (HR = 1.26, *P* < 0.01), while a much

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Guidelines on the management of stable angina pectoris: executive summary: The Task Force on the Management of Stable Angina Pectoris of the European Society of Cardiology: reply

We thank the authors for raising the interesting discussion regarding the treatment of hypertension in patients with concomitant coronary disease. The J-shaped association between on-treatment blood pressure and risk has been described in longitudinal cohorts of patients with treated hypertension as well as in clinical trial populations, both in on-treatment and control arms. However, it is not absolutely clear that the association is treatment-related; in fact, one meta-analysis of seven randomized controlled trials including data on more than 40 000 patients has shown that the J-shaped relationship between blood pressure and mortality was not related to antihypertensive treatment.¹ In this meta-analysis, non-