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Septal alcohol ablation in hypertrophic obstructive cardiomyopathy: improving cardiac function by generating a myocardial scar

We thank Efthimiadis et al. for their comments on our article concerning the improvement of systolic myocardial function in the lateral (free) wall in hypertrophic obstructive cardiomyopathy (HOCM) after alcohol septal ablation (ASA), which was studied using CMR tissue tagging and 3D strain analysis.1 In a previous work, we have demonstrated that contrast-enhanced imaging data derived in ~60% of the study-group pre-ASA after administration of gadolinium-DTPA contained only small pre-existing foci of delayed myocardial hyperenhancement, representing myocardial fibrosis and other pathological changes in the myocardial wall (e.g. disarray, inflammation, oedema, myolyxis, and necrosis).4 Compared with these hyperenhanced area-size-assessed pre-ablation, the infarct-size induced by ASA was 10-fold larger. In this respect, the induced myocardial infarct after septal ablation therapy enlarges the already existing arrhythmogenic substrate in HOCM patients. However, an electrophysiology report in high-risk patients after ASA has not indicated an increased arrhythmic substrate necessitating higher rates of implanting defibrillators.5 Although ventricular tachycardia and sudden death have been reported after ASA, these clinical features characterize the naturally course hypertrophic cardiomyopathy irrespective of therapeutic LVOT gradient reduction. Further studies are necessary to evaluate the long-term effects of ASA with respect to ventricular arrhythmias and sudden cardiac death. Our goal in the near future must be developing additional tools to identify the high-risk HOCM patients, regardless of a potential intervention for LVOT obstruction, in whom defibrillator implantation is justified.

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