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Letter to the Editor

Ramipril for left ventricular diastolic function in patients with pulmonary regurgitation after repair of tetralogy of Fallot

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We are grateful to the authors for their interest in our recent publication reporting stabilisation of diastolic and systolic left-ventricular (LV) function in repaired tetralogy of Fallot (TOF) patients with pulmonary regurgitation receiving 6 months of ramipril treatment [1].

We agree aortic dilatation is relevant in TOF. It is already present in infants before repair, and is contributed to by both haemodynamic and intrinsic aortic wall stiffness [2]. With current earlier surgical repair its importance may decrease. Aortic dilatation though common in older adults, has slow rates of progression [3].

The development of myocardial fibrosis has been assumed to reflect irreversible change, yet more recent emerging evidence contradicts this. A study investigating reverse myocardial remodelling one year following valve replacement in patients with aortic stenosis discovered, that not only myocardial cell volume decreased but also extracellular matrix volume, implying diffuse LV fibrosis is plastic; thus it could potentially reverse and be a therapeutic target [4]. Diffuse fibrosis CMR was not available at the time of the trial.

We welcome the comments that many unanswered questions remain including exploring the clinical effects of targeting renin-angiotensin-aldosterone-system in symptomatic patients with more severe RV and/or LV failure or in pulmonary stenosis patients. However, these groups do not represent the vast majority of contemporary adult patients with pulmonary regurgitation and milder dysfunction such as those we studied [1]. We also need to ascertain the effects of ramipril and other drugs given over a longer period to inhibit the process of slow deterioration which has already started in childhood.

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Conflicts of interest

The authors report no relationships that could be construed as a conflict of interest.

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