Right ventricular hypertrophy after arterial switch

To the editor: The recent study by Grotenhuis and colleagues demonstrated the increased peak flow velocity across the pulmonary trunk, right ventricular hypertrophy and right ventricular relaxation abnormalities in patients after an arterial switch operation.[1] The authors argued that one of the possible causes of the increased peak flow velocity was local scar tissue with loss of pulmonary artery distensibility.

I recognise the soundness of the report.

We previously analysed an input impedance spectrum of the pulmonary artery in patients after the arterial switch procedure, and reported increased pulmonary artery stiffness in these patients.[2] Many reports have demonstrated that the augmented aortic stiffness, which increases left ventricular pulsatile work, induces left ventricular hypertrophy.[3] As for pulmonary circulation, it has been reported that the increased pulmonary artery stiffness enhances the right ventricular load.[4]

Therefore, the increased stiffness of the pulmonary artery in patients after arterial switch would cause right ventricular hypertrophy. I agree with Grotenhuis and colleagues in thinking that careful observation of right ventricular function (and arrhythmia) is needed in the follow-up of patients after the arterial switch procedure.

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Competing interests: None.

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The author's reply: We appreciate Dr Murakami's comments on our recent paper in *Heart*, supporting our hypothesis that increased pulmonary artery stiffness enhances right ventricular load after the arterial switch operation.

In combination with the previously reported obstruction of right ventricular output due to systolic compression of the proximal pulmonary branches by the aorta,[1] increased right ventricular load will lead to compensatory right ventricular hypertrophy and subsequent right ventricular diastolic impairment. Close monitoring of right ventricular function after the arterial switch operation is therefore indicated during follow-up.

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Prediction of response and prognosis after cardiac resynchronisation therapy

To the editor: An accurate selection and optimal timing in instituting cardiac resynchronisation therapy (CRT) for patients are important in order to optimise the treatment. Consequently, the identification of outcome predictors is critical.

For these reasons we greatly appreciated the paper by Gradaus *et al* entitled "Diastolic filling pattern and left ventricular diameter predict response and prognosis after cardiac resynchronisation therapy".[1] This adds, on a larger scale, further evidence to the recently RESYNC results, reported at length,[2] about the relevance of the pre-CRT left ventricular dimensions (both systolic and diastolic) to functional response after CRT in patients with chronic heart failure and left bundle branch block.

In particular, an indexed left ventricular end-diastolic volume (iLVEDV)—that is, LVEDV/body surface area >142 ml/m^2 at myocardial gated single photon emission CT, has been found[3] and has been shown[4] to be a reliable predictor of functional recovery after CRT.

The RESYNC study survival branch is still continuing, and the data already collected agree with those of Gradaus *et al* about the critical role of left ventricular dimensions not only on functional outcome but also on the incidence of heart-related fatalities after CRT.

In our opinion, and according to Gradaus *et al*, left ventricular dimensions are critical prognostic measures and should be carefully taken into account before a CRT. Moreover, these data probably justify an earlier recourse to CRT in managing patients with chronic heart failure and left bundle branch block.

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The author's reply: I thank Dr Valle and colleagues for their kind comments on our paper. We agree with their comments. We found that despite treatment according to present guidelines nearly 30% of patients received no benefit from cardiac resynchronisation therapy in a clinical setting. On multivariate analyses, patients with an increased LV end-systolic diameter and concomitant diastolic dysfunction had a significantly worse outcome. The comments from Valle *et al* underline and emphasise the results of our study.

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Competing interests: None.

CORRECTION

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I M Tleyjeh, *et al*. The association between the timing of valve surgery and 6-month mortality in left-sided infective endocarditis. *Heart* 2008;**94**:892–6. The published affiliation for the fourth author of this paper, HMK Ghomrawi, was incorrect. The correct affiliation is: Division of Health Policy, Department of Public Health, Weill Medical College, Cornell University, New York, USA.