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Images in Congenital Heart Disease

Quantitative measurements of flow in a conduit placed from the left ventricular apex to the ascending aorta by magnetic resonance imaging

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A 19-YEAR OLD MALE PATIENT HAD CONCORDANT atrioventricular with discordant ventriculo-arterial connections, a ventricular septal defect, and complex obstruction in the outflow tract from the left ventricle. At the age of eight years, he underwent a modified Rastelli operation, in which the left ventricle was connected to the aorta using a patch placed within the right ventricle, and a homograft of 21 mm diameter was placed between the right ventricle and the pulmonary arteries. During the operation, however, it was noticed that the patch obstructed the outflow from the left ventricle. During the same procedure, therefore, a Hancock bioprosthetic conduit, of 14 mm diameter, was constructed from the left ventricle apex to the ascending aorta, leaving two outlets from the left ventricle. When the patient was 13 years old, the homograft and the conduit were replaced. A new homograft, of 24 mm diameter, was placed between the right ventricle and the pulmonary arteries, and a new Hancock conduit, measuring 18 mm in diameter, was placed from the apex of the left ventricle to the ascending aorta.

Over the following years, the patient developed aortic regurgitation, and experienced two episodes of cardiac arrest from which he was successfully resuscitated. To exclude a dynamic stenosis at the anastomosis between the left ventricle and the Hancock conduit during systole, and to quantify aortic regurgitation, we carried out magnetic resonance imaging. Neither echocardiography, nor standard cardiac

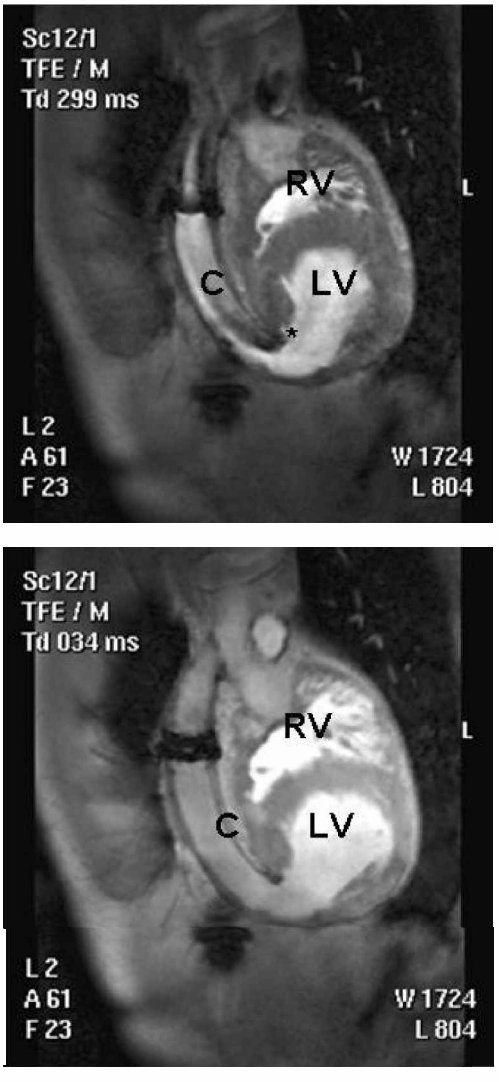

Figure 1.

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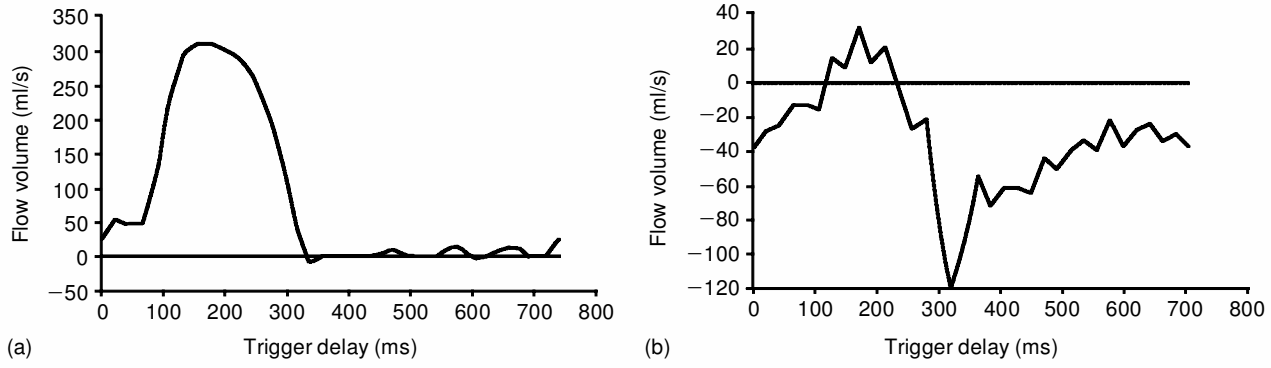

Figure 2.

catheterisation, had demonstrated conclusive evidence to answer these questions.

A multiphase gradient echo sequence covering the whole cardiac cycle was positioned in an oblique coronal projection along the Hancock conduit. Figure 1 shows flow in the Hancock conduit (C), placed between left ventricle (LV) and aorta, during systole (Fig. 1a) and diastole (Fig. 1b). The anastomosis between left ventricle and aorta is clearly depicted, and no obstruction is seen. During systole a flow void (*) in the proximal part of the conduit reflects turbulant flow. The projection shows a portion of the right ventricle (RV).

Additionally, we also analysed two flow sensitive through-plane gradient echo sequences. The first was placed distally to the Hancock valve, and the second through the ascending aorta between the native valve and the junction with the Hancock conduit. Phase shift and blood flow were quantified using Massflow® (MEDIS, Leiden, NL). Flow measurements through the Hancock conduit showed no regurgitation, with a forward flow of 60.1 ml/beat (Fig. 2a). At the native aortic valve, flow was almost exclusively regurgitant (1.9 ml/beat forward flow in contrast to 20.3 ml/beat regurgitant flow, Fig. 2b), resulting in 30 per cent regurgitation of the total stroke volume of the left ventricle. Thus, the native left ventricular outflow tract does not function as an outlet, and is solely responsible for the regurgitant flow into the left ventricle.

Because the cause of the cardiac arrests could not be found, and because we detected new disturbances of repolarisation in the absence of syncope, a defibrillator will be implanted in the patient within the next few weeks.

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