Excessive pulmonary autograft dilatation causes important aortic regurgitation

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A ortic root replacement with reimplantation of the coronary arteries using the pulmonary autograft (PAG) is an accepted operative technique to treat aortic valve disease in children and adults. However, the technically demanding operative technique and lack of long term results regarding function of the PAG limit widespread use. The behaviour of the PAG in the systemic circulation raises concern because of the thinner wall of the pulmonary root and the lesser amount of collagen as compared to the aortic root.¹ Severe PAG dilatation may result in aortic regurgitation (AR) and subsequent reoperation. We reviewed our first cohort of adult patients who underwent this operation.²

METHODS

From January 1989 until May 1995, 37 adult patients (20 male; mean age 29.1 years, range 19.3–52.1 years) underwent aortic root replacement using the PAG for aortic valve disease. The aetiology was bicuspid valve (n = 28), degenerative valve disease (n = 5), cured endocarditis (n = 3), and annuloaortic ectasia (n = 1). The haemodynamic diagnosis was aortic stenosis in 14 patients, regurgitation in 16 patients, and both in seven patients. Standard procedures were applied with regard to cardiopulmonary bypass techniques. The PAG was placed on the left ventricular outflow tract and annulus. The pulmonary root was replaced by an allograft. AR was scored from colour Doppler flow studies. PAG diameters were measured from the echocardiograms after cardiopulmonary bypass, at discharge, one year postoperatively, and the last transthoracic echocardiogram (TTE) available. Measurements were performed at the annulus at the level of the leaflet hinges, and the sinus of Valsalva at the largest anteroposterior diameter. This was done in the long axis plane from a two dimensional image during systole using the inner wall distance.

RESULTS

Hospital mortality was 5.4%. The mean follow up of the 35 living patients was 8.7 years (range 6.5–13.1 years). One patient was reoperated on two years later for stenosis at the distal suture line of the allograft in the pulmonary position. Four patients underwent reoperation for moderate or severe AR with left ventricular dilatation, of whom two were asymptomatic. One patient was in New York Heart Association (NYHA) functional class II at last follow up. The remaining 29 patients were all in NYHA class I at last follow up. The four reoperated patients had moderate or severe AR at their last TTE before reoperation. The last TTE examination of the other 31 patients showed moderate AR in four patients; the remaining 27 patients had no important AR.

There was a significant increase of the PAG annulus and sinus diameters during follow up, 22% and 27%, respectively (table 1). Most of the diameter increase was already reached at hospital discharge, with diminished increase thereafter. The eight patients with moderate or severe AR at last TTE had a significantly higher increase of PAG diameters compared with the remaining 27 patients (table 2). The four reoperated patients had the highest diameter increase at annulus and sinus level (4 mm to 9 mm, and 13 mm to 21 mm, respectively). Two patients had moderate annulus (3 mm and 6 mm) and sinus diameter (both 9 mm) increases. In one patient moderate AR was already present at discharge, and during follow up there was only a small increase in annulus and sinus diameter (1 mm and 5 mm, respectively). Severe PAG annulus or sinus diameter increase (> 6 mm and > 14 mm, respectively), from discharge to last TTE, was observed in eight

Abbreviations: AR, aortic regurgitation; PAG, pulmonary autograft; NYHA, New York Heart Association; TTE, transthoracic echocardiogram

Table 1 Pulmonary autograft (PAG) annulus and sinus diameters (mm) measured with transthoracic echocardiography (TTE) during follow up					
	Annulus (SD; range)	p Value	Sinus (SD; range)	p Value	
After bypass	27 (3.9; 18 to 34)	<0.001	36 (4.6; 30 to 46)	<0.001	
Discharge	30 (1.0; 21 to 37)	<0.001	39 (1.0; 26 to 47)	<0.001	
One year	32 (0.8; 24 to 43)	0.04	43 (0.9; 32 to 57)	<0.001	
Last TTE	33 (0.9; 22 to 44)	0.04	46 (1.4; 35 to 60)	<0.001	
Increase	6 (3.7, -1 to 16) 22%		11 (5.4; 0 to 22) 27%		
Annual increase after first year	0.1 mm/year		0.4 mm/year		

A *t* test with paired variables was used for the difference of the mean PAG diameters at different time points. The relation of PAG annulus and sinus diameter increase with the severity of aortic regurgitation at last TTE was tested by a *t* test, dividing the patients into two groups according to the semiquantitative measurement of AR (none, trivial and mild versus moderate and severe).

Table 2Pulmonary autograft (PAG) annulus and sinus diameters (mm) at last transthoracic echocardiogram (TTE) and
diameter increase during follow up in 27 patients without and eight patients with important aortic regurgitation (AR)

Diameter	No important AR	Important AR	p Value
Annulus (SD; range)	32 (5.2; 25 to 44)	35 (7.1; 24 to 44)	<0.001
Sinus (SD; range)	45 (6.2; 36 to 60)	50 (8.5 (35 to 60)	<0.001
Annulus increase (SD; range)	2 (3.5; -3 to 8)	6 (2.6; 1 to 9)	<0.001
Sinus increase (SD; range)	5 (5.1; -4 to 19)	13 (4.4; 5 to 21)	<0.001
Annual annulus increase after first year in mm/year (SD; range)	0.1 (0.3; -0.5 to 0.7)	0.2 (0.3; -0.2 to 0.7)	NS
Annual sinus increase after first year in mm/year (SD; range)	0.2 (0.4; -0.4 to 1.3)	0.8 (0.6; 0.3 to 1.9)	< 0.01

patients including five patients with important AR. Large PAG annulus and sinus diameters at last TTE (> 34 mm and > 49 mm, respectively) were present in 12 patients, including six patients with important AR.

DISCUSSION

In this first cohort of operated adult patients, the increase in the PAG annulus and sinus diameters was most pronounced in the first postoperative year, as described by us and others,²⁻⁴ but extends beyond the first year, although the rate of diameter increase declines. A learning process may be suspected, regarding the amount of right ventricular tissue beneath the pulmonary valve which is transplanted to the aortic annulus. Too much of this myocardial rim may result in a loss of the firm support of the aortic annulus. Another aspect in this setting is the supra-annular versus intra-annular insertion of the PAG with more support of the latter technique. Other causal factors may be aortic annulus dilatation caused by preoperative AR, bicuspid aortic valve with a dilated ascending aorta, and mismatch between PAG and aortic annulus diameter. Several surgical techniques are used to prevent PAG dilatation: aortic annulus plication; reinforcement of PAG annulus and sino-tubular junction using pericardium or prosthetic material; reinforcement of the entire PAG with the aortic root; and removal of dilated ascending aortic wall. Short term results with these techniques have been reported with promising results.5 However, reinforcement techniques must not adversely affect geometry and compliance, and longer term results are awaited. The intra-aortic cylinder or subcoronary implantation techniques obviate concerns about diameter increase and may be a useful alternative, especially in dilated aortic roots,³ although these techniques are technically demanding and freedom from reoperation may not be better.

In conclusion, the PAG used as a freestanding root is prone to dilatation in the systemic circulation extending beyond the first postoperative year. Excessive dilatation results in important AR requiring reoperation. A widespread use of this technique should not be recommended at present until long term results of the more recently operated patients become available, including the results of PAG reinforcement techniques. However, PAG for aortic root replacement is still a valuable option in experienced centres for (young) adults since anticoagulant treatment can be avoided.

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