Balloon dilatation of a Waterston aortopulmonary anastomosis

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SUMMARY Percutaneous balloon dilatation of a Waterston anastomosis was performed in a child who had had four shunt operations. Initially, the procedure was apparently successful, but pulmonary oedema developed and he died 12 hours after the procedure.

Balloon dilatation of a Waterston anastomosis is technically possible and may prove to be an alternative to reoperation in certain patients. But further experience with the technique is necessary to determine the optimum size of the inflated balloon, and to avoid the risk of producing an excessive increase in pulmonary blood supply.

Percutaneous balloon dilatation is useful for the treatment of a wide variety of stenotic lesions associated with congenital heart disease. The technique may prove to be valuable for the dilatation of surgical aortopulmonary anastomoses; however, published data seem to be restricted to a single report of balloon angioplasty of a stenosed Blalock-Taussig shunt.¹ We report the case of a patient who had balloon angioplasty of a Waterston anastomosis after four shunt operations. The blood supply to the right lung was increased to such an extent that pulmonary oedema developed and he died 12 hours later.

Case report

A thirteen year old boy with pulmonary atresia and ventricular septal defect had had a Waterston shunt constructed in the neonatal period. Later he had a left Blalock-Taussig shunt when he was two and revision of this shunt when he was five. When he was nine he had an ascending aorta to left pulmonary artery Goretex shunt. The last operation was complicated by severe haemorrhage from multiple small collateral vessels. Four years after his last operation his cyanosis and exercise tolerance had again worsened. Reinvestigation showed that his principal pulmonary blood supply was to the right pulmonary artery from the 13 year old Waterston shunt. The central and main pulmonary arteries were occluded and there was only a scant collateral supply to the left lung. No true pulmonary arteries were identified on the left and the Blalock-Taussig and Goretex shunts were

occluded. The diameter of the Waterston shunt was approximately 3 mm.

Under general anaesthetic and mechanical ventilation with 30% oxygen the right pulmonary artery was catheterised by a retrograde percutaneous approach from the right femoral artery with a 7 French Gensini catheter. A double length guide wire was positioned in the pulmonary artery supplying the right lower lobe and the Gensini catheter was replaced by an 8 mm Meditech balloon catheter. No waist was seen when the balloon was fully inflated. A 12 mm balloon catheter was substituted, and a clear waist was seen which abruptly disappeared on full balloon inflation. Repeat aortography showed that the size of the Waterston anastomosis had increased considerably. The pulmonary artery pressure abruptly rose to 42/30 mm Hg from 15/11 mm Hg, the aortic diastolic pressure fell to 46 mm Hg from 65 mm Hg, and the aortic oxygen saturation rose from 79% to 96%. He recovered rapidly from the general anaesthetic and seemed to be well; however, he had a collapsing pulse. Shortly afterwards clinical signs of frank pulmonary oedema developed and he was promptly reventilated. A chest radiograph showed pulmonary oedema and a pleural effusion on the right and considerable oligoemia on the left. Despite intensive supportive therapy he died 12 hours after dilatation.

Necropsy showed gross congestion and oedema of the right lung with a surrounding pleural effusion and confirmed the angiographic findings of pulmonary atresia with ventricular septal defect and occluded central and Blalock-Taussig shunts. There were dense mediastinal and pericardial adhesions and the heart was not enlarged. There was no extravas-

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Figure The orifice of the Waterston anastomosis viewed from the aorta. Dilatation was largely the result of disruption of the wall of the anastomosis, producing a V shaped tear in its inferior wall.

cular evidence of trauma or haemorrhage surrounding the ascending aorta or the right pulmonary artery. The Waterston anastomosis had been enlarged to a maximum diameter of approximately 10 mm by a tear on its inferior aspect (figure), extending from the aorta into the proximal right lower lobe pulmonary artery. The tear included the intima and elastic and fibrous tissue of the aortic and pulmonary arterial walls, but did not extend into the dense scar tissue surrounding the anastomosis. Microscopy of the aortic wall at the anastomosis showed considerable fibroelastic intimal hyperplasia.

Discussion

Balloon angioplasty may be valuable for the relief of both branch pulmonary artery stenosis^{2,3} and coarctation of the aorta.^{4,5} The choice of the optimum size of the inflated balloon may be difficult because this depends not only on the severity of the stenosis but also on its site—that is, the elasticity of the lesion to be dilated. Balloon diameters ≥ 2.5 times that of the stenosis may be needed for the successful dilatation of narrowings of the branch pulmonary arteries or coarctation of the aorta²⁻⁵ and balloon dilatation of the pulmonary valve may require balloon diameters that are up to 40% greater than the diameter of the valve annulus.⁶ Postoperative narrowings of venous channels may be very compliant, and may require inflated balloon diameters of up to four or five times the diameter of the stenosis.⁷ In a report of dilatation of a stenosed Blalock-Taussig shunt successful dilatation (three times the diameter of the stenosis) was eventually achieved with an inflated balloon with a diameter six times that of the stenosis,¹ suggesting that stenotic lesions involving the surrounding scar tissue may be the most elastic of all. Dense fibroelas-

tic tissue was found within the aortic wall at the site of anastomosis in our patient.

It has been suggested that an increase of 2 mm in balloon size is warranted if no clear waist is seen on the initial balloon inflation when a stenosis of a branch pulmonary artery is being dilated.³ This recommendation implies that the absence of a waist may predict unsuccessful dilatation. Because we thought that dilatation of the Waterston anastomosis in this case would be difficult to achieve, we chose to increase the balloon size by 4 mm and this resulted in overdilatation, with a final anastomosis diameter of about 80% of the inflated balloon diameter. In retrospect, it seems possible that despite the absence of a balloon waist our initial dilatation might have been successful. This experience prompts us to recommend detailed haemodynamic reappraisal and measurement of systemic arterial oxygen saturation after each balloon inflation irrespective of the radiographic appearances during balloon inflation.

The absence of postmortem evidence of an increase in heart size in our patient is also of note. Multiple thoracotomies may be followed by extensive mediastinal fibrosis, and the dense pericardial adhesions seen in this case may well have compromised the ability of the heart to dilate and increase its stroke volume to cope with a sudden increase in pulmonary blood flow.

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