Case reports

Complete heart block after balloon dilatation for congenital pulmonary stenosis

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SUMMARY Permanent complete heart block developed in a five year old child during balloon dilatation for pulmonary valve stenosis. Damage to the atrioventricular node by pressure from the inflated balloon may have caused the conduction defect.

Percutaneous balloon dilatation has become an accepted non-surgical treatment for congenital pulmonary valve stenosis since it was introduced by Kan et al in 1982.1 Recent reports on a large number of patients have demonstrated good results and few side effects.2-4 The most important complications are systemic hypotension and arrhythmias during the dilatation process; both are transient and revert to normal spontaneously. No long term adverse sequelae have been reported. We describe a case in which permanent complete heart block developed during balloon dilatation.

Case report

A five year old girl who had had a heart murmur detected early in infancy was referred for investigation. She remained symptom free. Physical examination showed a normal girl with body weight 17 kg (50th percentile) and height 102 cm (50th percentile). The peripheral pulses were normal with a regular rate of 80/min and the blood pressure was 95/65 mm Hg. The heart was not enlarged but there was a strong cardiac impulse and a systolic thrill along the left sternal border. The first heart sound was normal, the second heart sound was split with a soft pulmonary component. A harsh grade 4/6 ejection systolic murmur was audible over the upper left sternal border. There was no ejection click. The lungs were clear and the liver was not enlarged. Chest x ray showed normal lung vascularity. Electrocardiogram showed sinus rhythm of 80 beats/min (fig 2a) and right ventricular hypertrophy. Cross sectional echocardiogram showed a thickened pulmonary valve with systolic doming. The peak systolic gradient across the pulmonary valve estimated by pulsed Doppler was about 60 mm Hg. Moderate pulmonary valve stenosis was diagnosed and written consent was obtained from her parents for cardiac catheterisation and balloon dilatation of the pulmonary valve.

The procedure was performed under general anaesthesia as recommended by some workers.3 Intramuscular trimeprazine 30 mg was given as premedication one hour before the catheterisation. Intravenous ketamine 30 mg and atropine 0.1 mg were used for induction of anaesthesia and pancuronium bromide 1.5 mg for muscle relaxation. The patient was ventilated through a 5 mm uncuffed nasotracheal tube with a 3:2 mixture of nitrous oxide and oxygen and an expiratory minute volume of 2.8 l/min. A 7F balloon tipped end hole catheter was introduced by percutaneous puncture into the right femoral vein and was advanced to the left pulmonary artery. Sequential oxygen saturations and direct pullback pressure readings were obtained in the pulmonary artery, right ventricle, right atrium, and the vena cavae. The gradient across the pulmonary valve was 55 mm Hg and the right ventricular systolic pressure was 70 mm Hg. A 22 gauge cannula was inserted into the left brachial artery to monitor the systemic blood pressure. A 6F balloon tipped angiographic catheter was then introduced via the left femoral vein into the right ventricle and a cineangiogram was taken in the anteroposterior and

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The diameter of the pulmonary valve annulus measured from videotape replay on the monitor using the external diameter of the catheter as the reference was 18 mm.

The end hole catheter was again advanced to the left lower lobe pulmonary artery and a 0.038 inch exchange guide wire was passed. The catheter and sheath were removed and a balloon dilatation catheter with a 20 x 40 mm balloon on a 9F shaft was inserted over the guide wire and advanced to the upper inferior vena cava. The balloon was flushed with a 1:1 mixture of physiological saline and contrast (meglumine iothalamate, Conray 280) to remove air bubbles. It was then advanced until its midpoint was over the position of the pulmonary valve as marked on the fluoroscopic monitor. The balloon was rapidly inflated by hand until the indentation on the balloon produced by the stenotic pulmonary valve disappeared, and was then rapidly deflated and withdrawn to the inferior vena cava. The inflation-deflation cycle took less than 15 seconds and the maximum pressure attained was 400 kPa (4 atm). The balloon position during the procedure was recorded on a cinematograph and the electrocardiogram and right ventricular and systemic arterial pressures were also recorded.

The right ventricular pressure rose to 90 mm Hg and the systemic pressure dropped to 50 mm Hg during balloon inflation. At the same time, bradycardia and extrasystoles developed. Examination of the recorded tracings showed complete heart block (fig 1b). The systemic pressure returned to the predilatation value and the right ventricular pressure fell to 50 mm Hg after deflation of the balloon. The catheter in the right ventricle was advanced to the pulmonary artery and a pullback showed a residual gradient of 25 mm Hg across the pulmonary valve.

In view of persisting complete heart block on electrocardiographic monitoring no further dilatation was attempted and the catheters and guide wire were removed. The patient was put on continuous electrocardiographic and blood pressure monitor. A tracing at four hours after the procedure still showed complete heart block with an atrial rate of 135/min and ventricular rate 75/min (fig 2b). The blood pressure was stable and the patient was symptom free. Twenty four hour Holter recording showed that the heart block was constant; the ventricular rate varied between 70 and 75 beats/min and there were no extrasystoles. A treadmill exercise test one week later showed no significant increase in ventricular rate after 15 minutes on the modified Bruce scale.
phenomenon and hence serious ventricular arrhythmias. Late deaths attributable to ventricular fibrillation have been reported after balloon angioplasty for coarctation of the aorta. The cause of the above conduction disturbances have not been determined.

The cause of the complete heart block in our patient was probably traumatic. The balloon (40 mm) may have been too long so that as it straightened during inflation its proximal segment directly impinged on the triangle of Koch and hence the atrioventricular node. The effect was more pronounced since a balloon that was relatively large for the patient's age was used. Direct pressure on the left bundle branch was thought to be the cause of the temporary left bundle branch block seen in patients undergoing balloon dilatation of an aortic stenosis. Permanent complete heart block has been produced after balloon dilatation of severely stenotic mitral valves. Though no deleterious effects were reported by Radtke et al, who used balloons that were as much as 60% larger than the pulmonary annulus, and Miller, who used uniformly large (18 mm) balloons for patients over one year old without measuring the pulmonary annulus from angiograms, our case showed that oversized balloons can be hazardous. It would be safer to use shorter balloons and the cross sectional echocardiogram or larger reference markers other than the catheter to measure the pulmonary annulus.

References