Transcatheter closure of a mid-muscular ventricular septal defect with an Amplatzer VSD occluder device

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Abstract
A 5 year old girl with a haemodynamically significant mid-muscular ventricular septal defect (VSD) had successful transcatheter closure using the Amplatzer VSD occluder. This device passes through a small diameter sheath and can be easily retrieved or repositioned. These properties may make it a suitable device for closure of large mid-muscular defects in small children.

Keywords: Amplatzer septal occluder; congenital heart defects; ventricular septal defect; transcatheter occlusion

Transcatheter occlusion of ventricular septal defects (VSD) was first reported in 1987; however, the devices employed for transcatheter occlusion have not been particularly easy to use. The Rashkind or Clamshell umbrellas require a large sheath or front loading catheter (which limits retrievability) for introduction, while the Sideris adjustable buttoned device has a two part introduction method and slow release mechanism.

We describe a case of transcatheter closure of a mid-muscular ventricular septal defect using the Amplatzer VSD occluder.

Case report
A 5 year old girl underwent transcatheter closure of a mid-muscular VSD that had been diagnosed by echocardiography in infancy. Initially she needed anticongestive treatment because of poor feeding and mild respiratory distress. Cardiac catheterisation at 9 months of age showed significantly increased pulmonary blood flow with a pulmonary to systemic flow ratio of 3:1, a pulmonary artery pressure of 36/12 mm Hg, and an aortic pressure of 90/52 mm Hg. She was treated medically until she was 2 years old. At 5 years her parents complained that she had a moderately limited exercise tolerance. Chest radiography showed a mild degree of cardiomegaly and increased pulmonary vascular markings. Electrocardiography showed a mild degree of left ventricular hypertrophy. Repeat cardiac catheterisation showed a pulmonary artery pressure of 28/14 mm Hg with a mean of 23 mm Hg, a pulmonary arteriolar resistance of 0.8 Wood units, and a pulmonary to systemic flow ratio of 2.6:1. In view of her continued symptoms and significant left to right shunt, we obtained her parents' consent to enter her into a study of transcatheter VSD closure approved by the district ethics committee.

The Amplatzer VSD occluder (AGA Medical Corp, Golden Valley, Minnesota, USA) is made from Nitinol wire mesh that has been shaped by heat treatment into a central stent with two retention discs. The VSD device is similar to the atrial septal defect device. The length of the central stent has been increased from 3 to 7 mm to allow for the thicker interventricular septum compared to the interatrial septum. The retention discs have been attenuated and project only 4 mm on each side of the device. A stainless steel microscrew with a female thread is laser welded to the right ventricular disc to provide the attachment–detachment mechanism for the delivery cable. Figure 1 shows the device currently under clinical evaluation.

Figure 1 A close up view of the Amplatzer VSD occlusion device shows the two concave retention discs projecting 4 mm beyond the 9 mm wide × 7 mm long central stent. The retention discs contain polyester patches, and the central stent is filled with polyester fibres. The right ventricular disk has a microscrew attached.
PROCEDURE
Transcatheter closure was performed under general anaesthesia to allow for continuous transoesophageal echocardiographic visualisation of the defect. Left ventricular angiography was performed in the long axis view (70° latero–anterior oblique, 25° cranial), and the size of the defect at the left ventricular surface in diastole was measured (fig 2).

A 5 F Judkins right coronary curve catheter and a long floppy exchange wire (Noodle wire; AGA Medical) was used to cross the VSD from the left ventricle. A loop of the Noodle guidewire was easily advanced into the right atrium and superior vena cava where it was initially snared out to the femoral vein with a 25 mm Amplatzer goose neck snare (Microvena, Minnesota, USA). A loop of this guidewire was formed in the apex of the left ventricle by pushing a stiff snare guide catheter to the apex of the left ventricle to keep the long sheath away from the mitral apparatus. A 7 F long sheath (AGA Medical) was then passed over the wire from the femoral vein into the apex of the left ventricle. However this sheath kinked once its dilator and guidewire were removed. The defect was crossed again and the floppy exchange wire was snared out via the right internal jugular vein and a 7 F long sheath was advanced into the apex of the left ventricle. The dilator and wire were removed.

A 9 mm diameter VSD occluder was soaked in flush solution, attached to the delivery cable and drawn into the loader. The compressed device was then introduced into the long sheath and advanced into the apex of the left ventricle. The first device did not re-expand fully and was therefore removed and replaced with a second device. The left ventricular disc was extruded and after checking that it was away from the mitral valve tension apparatus, it was pulled with the long sheath onto the left ventricular surface of the defect. While maintaining tension on the delivery cable the stent and right ventricular disc were deployed. Transoesophageal echocardiography showed the device to be well aligned with both aspects of the ventricular septum without tricuspid valve encroachment, and the device was therefore unscrewed from the delivery cable. Repeat left ventriculography showed some residual flow through the centre of the device. The total fluoroscopy time was 39.5 minutes and the procedure time was 180 minutes.

The following day she was discharged home on aspirin 75 mg once a day. At follow up clinic visits after one and three months, a very soft pansystolic murmur could be heard and her effort tolerance was reported to be normal. Chest radiography showed that the device was unchanged with no evidence of fractures of the wire mesh. Colour flow Doppler echocardiography showed a < 1 mm wide residual colour jet at the right ventricular side of the septum adjacent to the device.

Discussion
Although VSD is the most common congenital heart defect, transcatheter VSD occlusion remains an uncommon procedure in most tertiary centres. Most defects are either unsuitable for
transcatheter closure or do not require closure. Perimembranous VSD is usually too close to the aortic and atroventricular valves to permit safe transcatheter closure.1 Mid-muscular and apical VSD are usually very small and haemodynamically insignificant. Multiple muscular and apical VSD of the “Swiss cheese” type may not be easily accessible to the surgeon, and are far enough away from the valves to make transcatheter occlusion a realistic option.2 Large, relatively discrete, mid-muscular VSD are much less common than perimembranous defects. Large mid-muscular defects are still treated surgically in most centres because they cause heart failure in small infants, and transcatheter closure, to date, has required large cumbersome introduction sheaths.

Patients with haemodynamically significant muscular VSD beyond infancy are unusual and are ideal for transcatheter closure. We found the Amplatzer VSD device easy to use once the correct approach for the introduction sheath had been chosen. The first device did not open properly but was easy to withdraw without displacing the introduction sheath. The second device opened without difficulty and placement was straightforward thereafter. If this device had pulled through, it would have been a simple matter to retract it into the sheath. The ability easily to reverse the implantation procedure up until the point of release is a very significant technical advantage. The device we used was introduced through a small diameter sheath (7 F) that should permit the procedure on small infants with large mid-muscular VSD. The procedure took longer than expected because we attempted closure from the femoral vein, which provides a much more tortuous course than the jugular vein. With more practice and the use of a straighter and more direct course for the long sheath, we should be able to decrease the fluoroscopy and procedure time significantly.

In experimental studies the VSD is closed by the induction of thrombosis on the polyester fibres in the central stent and polyester patches sewn into the retention discs.3 Our patient still had a trivial residual shunt after three months that we hope will close spontaneously. We are a little concerned that a residual shunt in the presence of a prosthetic device may increase the risk of infective endocarditis. However, trivial shunts adjacent to patent ductus arteriosus umbrellas have not been found to predispose to endocarditis in an animal model.4 The other concern is device fatigability over a possible lifespan of 70 to 80 years. Other septal occlusion devices, made of stainless steel rather than Nitinol, have a high fracture rate.5 Such fractures have nearly always been without clinical consequence. Nitinol is much more fatigue resistant6 than stainless steel and even if some of the wires fractured they should remain trapped within the wire mesh. Furthermore, the device should, by then, be encased in fibrous tissue and endothelium and it is unlikely that any fractured wires would protrude or embolise.

In conclusion, we describe a case of transcatheter closure of a child’s mid-muscular VSD with the new Amplatzer VSD occluder. This device passes through a small diameter sheath and can be easily retrieved or repositioned. These properties may make it a suitable device for closure of large mid-muscular defects in small children.