Transcatheter Closure of Ventricular Septal Defects

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Between January and October, 1987, we attempted percutaneous transcatheter closure of seven ventricular septal defects (VSD) in six patients; none of the patients was a candidate for operative management. Patients' ages ranged from 8 months to 82 years (6.0–70 kg); diagnoses included postinfarction VSD (n = 4), congenital VSD (n = 1), and postoperative congenital VSD (n = 2). Indications for VSD closure were shock or respiratory failure (n = 5) or multiple episodes of endocarditis (n = 1). Closure was attempted with a Rashkind double umbrella: VSDs were crossed via the left ventricle and a guidewire was advanced to the right heart, snared with a venous catheter, and used to direct a long sheath (and ultimately the double umbrella) across the VSD. We crossed the VSD in all seven attempts, and a 17-mm double umbrella was successfully placed in each VSD. In the first (postinfarction) patient with the largest (12 mm) VSD, the umbrella embolized after 20 seconds to the pulmonary artery (without reducing flow). The other six umbrellas remained in position, either diminishing or abolishing the left-to-right shunts. Postinfarction patients had increasing VSD shunting over the next several days and died; at postmortem, the umbrellas remained well positioned in the septum, with other VSDs present. All three congenital VSDs had absent or diminished shunts after umbrella closure. These preliminary data indicate that transcatheter VSD closure is feasible in selected cases. (Circulation 1988;78:361–368)

Beginning with the study of Porstmann et al1 on the patent ductus arteriosus (PDA), the list of successful transcatheter closures of congenital cardiac defects has expanded to include atrial septal defects,2 aortopulmonary collaterals,3 arteriovenous fistulae,4 and Blalock-Taussig shunts.5 The Rashkind double umbrella, originally designed to close the PDA,6 has subsequently proven to be a versatile instrument to effect transcatheter closure of many other defects. With a modified double umbrella technique, we have recently closed superior vena cava–RA junction, coronary sinus septal defect, Potts anastomosis, patent foramen ovale in cyanotic heart disease, and others.7 Based on this experience, we have now devised a technique to close defects in the ventricular septum.

Patients and Methods

Patient Selection

After publication of the uses of the double umbrella to close various intracardiac defects,7 one of the authors suggested that patients with postinfarction ventricular septal defects (VSDs) who were considered inoperable might be candidates for attempted transcatheter closure. Patients were considered candidates for transcatheter VSD closure only if all of the following conditions were met: 1) VSD closure was clearly indicated for physiological or other medical reasons; 2) the patient was not a candidate for operative management (determined after consultation with a cardiothoracic surgeon); and 3) informed consent regarding the experimental nature of this procedure was obtained from the patient or responsible relative. At the Massachusetts General Hospital and the Beth Israel Hospital, transcatheter VSD closure was approved as a compassionate use on a case-by-case basis; at the Children's Hospital, a protocol for the use of double umbrellas to close non-PDA structures had previously been approved7 by the Committee on Clinical Investigation.

Hemodynamic and Angiographic Evaluation

The approximate size and location of each defect was assessed with two-dimensional echocardiogra-
phy. Each patient also underwent right and left heart cardiac catheterization before attempted closure.

The location of each VSD was determined with angled angiography: in four of seven VSDs, the defect (midmuscular in two, perimembranous in two) was seen on a 70° left anterior oblique view with cranial angulation. The distance from the VSD to the aortic valve was measured to ensure that umbrella placement would not alter aortic valve function. In patients 3 and 6, multiple VSDs and their relative sizes were difficult to demonstrate in a single view, necessitating multiple axial views.

**Catheter Closure Technique**

We reasoned that right ventricular trabeculae would make crossing the central channel of a muscular VSD difficult from a venous approach; however, our prior experience indicated that a long, tortuous retrograde arterial catheter course to a VSD would preclude delivery of the umbrella. We therefore used a double-catheter approach.

With the x-ray tube positioned in the angle that best demonstrated the location of the VSD, we attempted to cross the defect with a balloon-tipped end-hole catheter (placed via a femoral arterial sheath) from the left ventricle. The catheter was looped in the apex of the left ventricle, with the balloon inflated, and the catheter and a curved guide wire were manipulated until the balloon "seated" in the ventricular defect. Either the partially deflated balloon or a soft J-tipped wire was advanced through the VSD, into the right ventricle, and from there to either the right atrium or pulmonary artery. We then advanced the balloon catheter over the wire, through the VSD into either the right atrium or pulmonary artery. At that point, a 400-cm double-exchange 0.035 in. guide wire (Cook) with a hand-curved distal tip was advanced through the balloon-tipped catheter in preparation for snaring the long wire (Figure 1).

Based on experience in patient 1 (see below), we snared the long guide wire from a percutaneous

**FIGURE 1. Method for transcatheter muscular ventricular septal defect (VSD) closure.** A 7Fr balloon end-hole catheter is passed retrograde into the left ventricle (LV) and the inflated balloon “seats” in the VSD. The balloon and catheter are manipulated (using partial balloon inflations) across the septum into the right heart, where a 400-cm guide wire is snared via a transvenous catheter. This wire is ultimately used to advance a venous long sheath (and dilator) across the VSD, allowing placement of a double umbrella to straddle the defect.

**FIGURE 2. Venous site for percutaneous entry was determined by ventricular septal defect (VSD) location; midmuscular or apical muscular VSDs were approached from a right internal jugular approach (left panel), and perimembranous or anterior muscular VSDs were approached from the femoral vein (right panel).**
### Table 1. Hemodynamic and Angiographic Assessment of Patients Before and After Umbrella Placement

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Cardiac diagnosis</th>
<th>Other diagnoses</th>
<th>VSD location and size</th>
<th>Size of umbrella placed (mm)</th>
<th>Qp:Qs Before surgery</th>
<th>Qp:Qs After surgery</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>80 years</td>
<td>Postinfarction VSD</td>
<td>Cardiogenic shock, severe renal failure</td>
<td>Midmuscular, 13 mm</td>
<td>17</td>
<td>2:1</td>
<td>2:1</td>
<td>Umbrella embolized to left pulmonary artery, patient died 7 days later.</td>
</tr>
<tr>
<td>2</td>
<td>82 years</td>
<td>Postinfarction VSD</td>
<td>Cardiogenic shock, ventilator-dependent</td>
<td>Midmuscular, 11 mm</td>
<td>17</td>
<td>4:1</td>
<td>2:1</td>
<td>Initial improvement, shunt size. Patient died from pulmonary embolism 6 days later.</td>
</tr>
<tr>
<td>3</td>
<td>63 years</td>
<td>Postinfarction VSDs</td>
<td>1 week s/p coronary artery bypass grafting and VSD closure, cardiogenic shock, ventilator-dependent</td>
<td>Midmuscular, 11 mm; high muscular; anterior muscular, 11 mm; anterior muscular</td>
<td>17</td>
<td>1.8:1</td>
<td>2.4:1</td>
<td>Left-to-right shunt persisted, progressive shock. Patient died shortly after umbrella placement.</td>
</tr>
<tr>
<td>4</td>
<td>8 months</td>
<td>Tetralogy of Fallot, after-surgery VSD</td>
<td>Pulmonary AVM, polydactyly, cerebral atrophy, severe, recurrent aspiration, ventilator-dependent</td>
<td>Perimembranous, 4 mm</td>
<td>12</td>
<td>1.9:1</td>
<td>1.2:1</td>
<td>Extubated p umbrella. 5 weeks p umbrella, patient vomited—SVT, ventricular flutter and died.</td>
</tr>
<tr>
<td>5</td>
<td>44 years</td>
<td>Perimembranous VSD</td>
<td>Three episodes of subacute bacterial endocarditis, sensorineural deafness, hypertension, asthma</td>
<td>Perimembranous, 5 mm</td>
<td>17</td>
<td>1.4:1</td>
<td>1.0:1</td>
<td>Complete closure, 6-month follow-up.</td>
</tr>
<tr>
<td>6</td>
<td>7 years</td>
<td>Tetralogy of Fallot, pulmonary atresia, two attempts at VSD closure</td>
<td>Pressor-dependent, ventilator-dependent</td>
<td>Midmuscular, 8 mm; apical muscular, 8 mm</td>
<td>17</td>
<td>2.0:1</td>
<td>...</td>
<td>Clinical improvement with discharge 2 weeks later. Patient stable after 3 months.</td>
</tr>
</tbody>
</table>

VSD, ventricular septal defect; Qp:Qs, ratio of pulmonary flow to systemic flow; s/p, status post.

The snare was typically a 0.018 in. exchange guide wire, fashioned into a diamond loop, and advanced through a standard 7F multipurpose end-hole catheter. Once the 0.035 in. 400-cm guide wire had been retrieved out the venous sheath, a second 7F balloon end-hole catheter was advanced over the venous end of the wire to cross the VSD from the right heart. The balloon was inflated with diluted contrast in the left ventricle and withdrawn gently to test occlude the VSD (retaining both arterial and venous control of the long guide wire). Dilute contrast was drained slowly from the balloon with...
gentle traction on the venous catheter until the balloon "popped" through the VSD into the right ventricle. This maneuver not only reconfirmed the precise location of the VSD when the edges were being retracted but also allowed determination of the "stretched" diameter of the VSD. As we have previously noted, stretched defect diameters more than 10 mm may be too large to safely position a standard Rashkind 17-mm umbrella. With the VSD size and location reconfirmed, the venous sheath and catheter were removed, the skin predilated with a 12F dilator, and an 11F long sheath and dilator [US Catheter and Instruments (USCI), Billerica, Massachusetts] were advanced over the guide wire, through the VSD, and into the left ventricle; traction on the arterial end of the long wire was often helpful in passing the sheath and dilator through the VSD.

The 17-mm double umbrella was soaked in topical thrombin for 5 minutes and loaded into the delivery pod as previously described. The dilator and long wire were then removed, and the delivery pod was advanced through the venous sheath to the level of the tricuspid valve. We next advanced the umbrella out of the pod, into the sheath, through the VSD, into the left ventricle, and to the end of the sheath. The distal umbrella arms were then extruded into the left ventricular cavity, the entire sheath-umbrella system withdrawn until the distal arms flexed against the left side of the septum, the umbrella held steady, and the sheath withdrawn into the right ventricle until both sets of arms were opened, straddling the VSD. The umbrella was nudged gently to and fro to be certain that the position was correct and then released.

The infant with a residual VSD had a modified closure procedure due to small patient size (6.0 kg); the defect was crossed directly from a femoral venous approach, and an 8F sheath and 12-mm umbrella were used to close the defect.

**Evaluation After Closure**

We attempted repeat hemodynamic and angiographic assessment after umbrella closure when permitted by clinical status (see Table 1). Patients with postinfarction VSD who required intra-aortic balloon pumping before the procedure were returned to the intensive care unit with Swan-Ganz catheters in place; assessment of left-to-right shunting was made with superior vena cava and pulmonary artery oxygen saturation measurements when possible, and an attempt was made to wean from balloon pump support. Each patient underwent postumbrella two-dimensional echocardiographic evaluation, with or
without color-flow mapping, and routine chest x-rays to ensure stability of umbrella position. The patient (5) who was hemodynamically stable before the procedure was monitored overnight after VSD closure and discharged the next day.

**Results**

**Indications for Transcatheter VSD Closure**

Patients 1, 2, and 3 had postinfarction VSD with severe clinical disability. Patient 1 had cardiogenic shock and refractory renal failure (blood urea, 100 mg/dl); patient 2 had cardiogenic shock despite pressor support and intra-aortic balloon pumping; and patient 3 had undergone an operation to close postinfarction VSDs in the early postinfarction period but had evidence of significant residual left-to-right shunting and cardiogenic shock after surgery. Patient 4, the infant, had tetralogy of Fallot, chronic lung disease, and multiple congenital anomalies; she could not be weaned from ventilatory support despite intensive medical management. Her relatively small residual VSD (Qp:Qs, 1.8:1) may have contributed to chronic ventilatory dependence, but another period of cardiopulmonary bypass was thought to be inadvisable. Patient 5 had a small VSD (Qp:Qs, 1.5:1) and two previous episodes of bacterial endocarditis. Operative closure was recommended after each episode, but she steadfastly refused surgery. A third episode of endocarditis (2 years before transcatheter closure), from a resistant organism, resulted in severe sensorineural hearing loss in association with antibiotic therapy; once again, surgery was refused.

**Hemodynamic and Angiographic Evaluation**

Each patient had a restrictive VSD in that right ventricular pressure was significantly lower than left ventricular pressures. Pulmonary-to-systemic flow ratios (Qp:Qs) were increased in each patient, but pulmonary artery pressures were only slightly elevated. The location of the VSD was predicted from echocardiography in four of five patients; however, in patient 3 (who had already undergone surgery), the defect was thought to be in the midmuscular septum. At angiography, contrast did cross the midmuscular septum, but other ill-defined contrast streams also opacified the right heart: these were not seen in the posterior septum (as assessed from a 40° left anterior oblique) or the anterior apical septum (as assessed from a lateral view). The midmuscular defect was closed (see below), but a significant shunt persisted. Review of the developed films revealed one or more muscular defects in the anterior, supracristal muscular septum, as seen on a 30° right anterior oblique view. Finally, each VSD was shown to be more than 1 cm from the lower limits of the aortic valve.

**Catheter Closure Technique**

Crossing the VSD did not prove unduly difficult, although the smallest VSD (patient 5) was crossed not with the balloon catheter but with a cut-off pigtail catheter and a soft guide wire. We found guide wire retrieval simpler from the right atrium or venae cava than from the pulmonary artery.

In the first patient, with a midmuscular VSD, the long sheath was advanced from the femoral vein. The curve required to cross the right atrium, tricuspid valve, and midventricular septum from below was serpentine and quite acute; the dilator followed the wire with considerable difficulty, and the sheath buckled in the atrium each time the dilator was removed. The umbrella barely negotiated the various curves into the ventricular septum, and considerable lateral tension was therefore applied to the umbrella at release. In subsequent patients, we
chose the venous approach to the septum with the straightest possible catheter course (Figure 2).

Six of seven VSDs were 10 mm in diameter (stretched, see above) or smaller, and each had successful placement of an umbrella. In the first patient, a fully inflated (2.0 ml) balloon on the 7F catheter passed with little difficulty from left to right ventricle, indicating a stretched VSD diameter of 12–13 mm. After considerable discussion, we attempted umbrella closure, given the patient’s poor clinical status. The umbrella was released in what appeared to be the correct position, but after 20 seconds it became unstable and was carried to the left pulmonary artery. Several attempts to retrieve the umbrella were unsuccessful; when angiography demonstrated that flow to the left lung was apparently unobstructed despite the umbrella, the procedure was terminated and the patient returned to the intensive care unit in stable albeit critical condition.

**Evaluation After Closure**

In each patient with successful umbrella placement (2, 4, and 5) into a single VSD, the size of the left-to-right shunt diminished after closure (Table 1). In patients 3 and 6 (who had multiple VSDs), the change in measured left-to-right shunt was minimal. Angiography demonstrated minimal residual left-to-right shunt in patients 2 and 4 (Figure 3) and complete closure in patient 5 (Figure 4). In both patients with multiple VSDs, the channel with the umbrella appeared closed, although right ventricular opacification by other channels obviously persisted.

Patient 1 slowly deteriorated after attempted closure and died 6 days later. Patient 2 had considerable hemodynamic improvement, with a rise in blood pressure, and was weaned from balloon support. Two days after transcatheter closure, deterioration in clinical status was associated with increasing shunt size as measured from an indwelling Swan-Ganz catheter; chest film and echocardiography confirmed stable umbrella position. Deterioration continued, and the patient died 5 days after the procedure. At postmortem examination, the umbrella was well seated in the septum, with fibrin formation throughout the foam pad. However, an area of septal deficiency extended in a basal direction away from the umbrella (see Figure 5) resulting in a hole that was 8–10 mm in diameter.

Patient 3 improved after umbrella closure but deteriorated 1–2 days later. She was returned to the catheterization laboratory 2 days after the initial procedure in a state of extreme hemodynamic insta-

**FIGURE 5.** Left ventricular septal surface at postmortem 6 days after umbrella placement (patient 2). One set of umbrella arms and foam are secure against left ventricular surface, with foam fibrin coated. A separate defect was present (crossed by forceps) basal to the original defect.
bility and ongoing cardiac resuscitation. Angiography demonstrated multiple defects in the ventricular septum, near the supracristal septum. A second umbrella was placed in a ventricular defect, but she died soon after the procedure. At postmortem examination, four separate defects of 6–10 mm were found in the ventricular septum in addition to the defect closed at the initial operation; umbrellas were present in two of the defects.

Patient 4, the infant, improved after VSD closure and was weaned from the ventilator. She continued to have recurrent aspiration and feeding difficulties and unstable heart and respiratory rates of uncertain etiology. After-closure echocardiography demonstrated no significant residual hemodynamic abnormalities despite the umbrella arms being partially "closed" on x-ray and echocardiography (Figure 6); however, she died suddenly 4 months after surgery and 5 weeks after VSD closure of apparent aspiration. At postmortem, the umbrella was well positioned and uninfected, but the partially collapsed arms of the 12-mm umbrella extended slightly into the left ventricular outflow tract. There was no evidence of significant obstruction to the left ventricular outflow tract, embolic events, or valvar damage.

Patient 5 was discharged the day after the procedure with no murmur. At 6-month follow-up, she remains without clinical evidence of endocarditis or a VSD. Patient 6 had partial but significant clinical improvement after umbrella implantation. She weaned from artificial ventilation 3 days after umbrella closure of one of two muscular VSDs and was discharged 2 weeks later. She had continued clinical improvement at 3-month follow-up, although a significant left-to-right shunt persists.

Discussion

We have described a method for transcatheter placement of a double umbrella device into a variety of VSDs, indicating that percutaneous transcatheter VSD closure is quite feasible in selected cases. In addition, our experience helps to identify technique modifications that may improve the success of transcatheter closure of other defects.

Technical Considerations

Our basic approach—retrograde balloon catheter crossing of the VSD, snaring of a long guide wire from a venous approach, and the use of a double umbrella device to effect closure—appears to be a reasonable strategy. Several further observations are worth noting: the early embolization of a standard Rashkind 17-mm device out of a 12-mm defect supports our previous finding that the stretched diameter of a defect should not be larger than 50–60% of the stated diameter of a standard Rashkind double umbrella device. Early migration of such umbrellas might be due to constant hemodynamic pressure, partial inward flexion of the umbrella arms under pressure, and plasticity of myocardial tissue. Larger devices, or perhaps modification of the arm configuration, will be required to close the range of VSDs expected in clinical practice.
Next, the fact that the partially collapsed arms of a 12-mm umbrella (patient 4) protruded somewhat into the left ventricular outflow tract underscores the need for devices that will expand fully when released and thus remain flush with a septal surface.

Finally, precise anatomic definition of defects in the ventricular septum and their relation to other cardiac structures is crucial to the successful use of transcatheter VSD closure. In our limited experience, the primary cause of clinical failure was the presence of multiple separate defects or unrecognized extension of the original defect. Thus, if test occlusion of the defect with a balloon-tipped catheter does not virtually eliminate the hemodynamic or angiographic left-to-right shunt, it would appear that umbrella placement may be technically superb but clinically inconsequential.

Postinfarction VSD

The failure to improve patient status despite successful transcatheter closure of postinfarction VSDs in two cases may be due to several possibilities. One explanation is that the original septal catheter passage was made through a hemodynamically trivial hole, leaving larger defects unplugged, or may even have enlarged the initial hole. The initial hemodynamic success in the second patient supports an alternate explanation: failure may be due to VSD extension that occurs after successful closure of the initial hole. This extension may represent ongoing resorption of infarcted tissue that was allowed to continue only because the patient was stabilized. One might speculate that ongoing tissue resorption, rather than loss of suture line integrity, may also underlie many of the "residual" defects after apparently successful surgical closure.

It must be strongly emphasized that most forms of VSDs probably cannot be managed with this technique or a modification of it. Congenital perimembranous ventricular defects large enough to require closure in infancy will have defect margins that are quite close to aortic or tricuspid valves; the use of double umbrella techniques in such cases would appear likely to interfere with the function of otherwise normal valves. Congenital VSDs of the atroventricular canal type will undoubtedly be unsuited to transcatheter closure. Similarly, many postinfarction VSDs occur in the posterior muscular septum, near the tricuspid or mitral valves, where they may take serpiginous courses through the septum²; such defects may be difficult to cross and dangerous to close. Nonetheless, in patients who are poor candidates for surgery, have compromised hemodynamics, and have muscular defects removed from valvar structures, it would appear that transcatheter closure of VSDs deserves considerable further study.

References


Key Words • umbrella closure • ventricular septal defect
Transcatheter closure of ventricular septal defects.
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_Circulation._ 1988;78:361-368
doi: 10.1161/01.CIR.78.2.361
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1988 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
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