Transcatheter closure of congenital and acquired septal defects

Christian Spies¹,², Qi-Ling Cao³, and Ziyad M. Hijazi³*

¹Department of Cardiovascular Diseases, The Queen’s Medical Center, Honolulu, HI, USA
²Department of Medicine, University of Hawaii, Honolulu, HI, USA
³Rush Center for Congenital and Structural Heart Disease, Rush University Medical Center, 1653 W. Congress Parkway, Jones 770, Chicago, IL 60612, USA

Transcatheter closure of congenital and acquired septal defects has been performed for over 40 years. In several circumstances, it is now considered first in-line therapy over surgical closure. In this review, we summarize the indications to close congenital and acquired septal defects and briefly review the technique and devices used to repair such defects.

KEYWORDS
Congenital heart disease; Atrial septal defect; Ventricular septal defect; Patent ductus arteriosus; Transcatheter closure

Overview

Percutaneous coronary intervention for the treatment of obstructive atherosclerotic coronary artery disease recently celebrated its 30th anniversary. Although the first balloon angioplasty performed by Andreas Gruntzig was a milestone, it does not mark the birth of interventional cardiology or transcatheter cardiac procedures. These date back to the 1950s with Rubio-Alvarez’s pulmonary valve wire valvuloplasty and the 1960s when William Rashkind first performed balloon septostomy for transposition of the great arteries. The first transcatheter closure of a congenital defect was then reported by Werner Porstmann in 1967, describing the use of his Ivalon plug for the closure of patent ductus arteriosus (PDA). Throughout the following years, transcatheter closure of atrial septal defects (ASDs) in the mid-1970s and ventricular septal defects (VSDs) in the late 1980s became possible. The Rashkind PDA occluder with its double-umbrella design was the prototype of occluder devices and had been used to close a variety of congenital and acquired defects. Several other devices have been developed over the years based on similar concept of using two umbrellas wedging the defect in between. Most of these early devices are no longer available as their use was rather cumbersome and associated with higher complication rates than conventional surgical alternatives. The development of the Amplatzer family of closure devices in the mid-1990s marked another important milestone for interventional cardiology. The design allows the use of relatively small delivery catheters. The inherent self-centering mechanism and the ability to retrieve the device if needed prior to release were among the features that have led to their wide spread use. However, for PDA closure, coils have been used that allow closure of smaller defects; although with the development of detachable coils, larger PDAs can be closed with coils as well.

Atrial septal defects

Definition and indications for closure

An ASD is a persistent communication between the two atria caused by a hole in the inter-atrial septum. Atrial septal defects present the most common congenital heart defect diagnosed in adults and occurs in approximately 4 of 100,000 newborns. The most common type is the secundum ASD, which occurs in the region of the fossa ovalis and represents ~75% of all cases. Primum ASDs, coronary sinus, and sinus venosus defects are less common. Transcatheter closure is possible only for the secundum type. This is mainly because of the lack of sufficient rims surrounding the defect to anchor
the device or due to associated defects such cleft atrio-
ventricular valves or anomalous drainage of pulmonary
veins requiring open surgical repair. An un repaired ASD
can lead to left-to-right shunt, secondary right ventricu-
lar volume overload, and pulmonary overcirculation. This
leads to right-sided heart failure and occasionally pul-
monary arterial hypertension. Patients’ symptoms may
include dyspnoea, fatigue, or palpitations, as atrial fibril-
lation and atrial flutter are common consequences of
long-standing untreated ASD.16 Symptoms commonly get
worse with increasing age, even with smaller defects,
due to an increase in left-to-right shunt caused by changes
in ventricular compliance with age.17
Transcatheter or surgical closure of an ASD is indicated
if there is evidence of right-sided volume overload as
seen by echocardiography irrespective of the patient’s
symptoms.18 Other indications for ASD closure include the
presence of significant left-to-right shunt, defined as a
pulmonary-to-systemic flow (Q_{p}/Q_{s}) ratio of >1.5:1, and
the occurrence of paradoxical embolism or documented
orthodoxia-platypnoea syndrome. Surgical closure is pre-
ferred for non-secundum ASDs or if surgical repair for associ-
ated defects is needed. Important prerequisites for ASD
closure are pulmonary arterial pressures less than two-third
ystemic and a pulmonary vascular resistance <7 Woods
units. Atrial septal defect closure in patients with severe
irreversible pulmonary arterial hypertension is contraindi-
cated, as it may shorten their life-expectancy.19

Atrial septal defect closure devices
The Amplatzer septal occluder (ASO) (AGA Medical, Ply-
mouth, MN, USA) is a double-disc device with a self-
centring mechanism. In the multicentre, non-randomized
pivotal trial including 442 patients comparing transcath-
ether closure using the ASO with surgical closure revealed
equivalent success rates.20 However, complication rates
and the length of hospital stay were better than for sur-
cegal closure. The ASO is the most widely used device for
the closure of ASDs.21,22 Defects up to 40 mm in diameter
can be closed with this occluder. Dacron patches are
embedded into the discs and connecting waist to enhance
closure. Complete endothelialization occurs between 6 and 12 months from closure.

The Helex occluder is a non-self-centring double-disc
device that consists of one single nitinol wire frame in
the shape of a coil that is covered by polytetrafluoroethy-
lene (Gore-Tex) membrane (W.L. Gore & Associates, Flag-
staff, AZ, USA). The nitinol wire is spun as two opposing
spirals that configure themselves as two separate discs
once advanced outside the delivery sheath. Following
the first human implant in 1999, the device has gained
popularity worldwide.23–25 Only defects as large as 18 mm by balloon stretch diameter can be closed effec-
tively with this device.

The CardioSEAL and its self-centring successor named
STARFlex are double-disc occluders, which were modifi-
cations to the early clamshell device (NMT Medical,
Boston, MA, USA). The device itself consists of two
square-shaped umbrellas made of Dacron, mounted on
spring coils allowing flexible positioning of each arm.

Similar to the Helex device, only defects up 20 mm by
balloon stretching can be closed effectively with these
devices. The CardioSEAL device is approved only for
VSD closure in the USA; however, many use this device
for the closure of ASDs and patent foramen ovale (PFO).26,27
A niche device for the multi-fenestrated atrial septum is the Amplatzer cribriform occluder. This
device is similar in design to the Amplatzer PFO device,
but with two equal-sized discs.28,29

Closure technique
For the most part, transcatheter ASD closure is
performed in a similar fashion with the different
devices available to date. The procedure can be
performed under fluoroscopic guidance with either simul-
taneous transoesophageal (TEE) or intracardiac echocar-
diography (ICE) monitoring.30–32 The atrial septum and
surrounding structures need to be evaluated carefully
by means of echocardiography. Sufficient rims, defined
as at least 5 mm distance from the defect to the
structure, allow for safe anchoring of the device.
A deficient anterior rim is no contraindication for the
placement of an ASO as the device wraps around the
posterior aortic wall.33 Prior to actual device deployment,
most manufacturers recommend balloon sizing of the
defect.34 Alternatively, the size of the device can be
chosen based on the measurement of the defect by
echocardiography only.15,36

Following determination of the defect size, the ASD
can be closed with the device of choice. For the ASO, a
device 0–2 mm larger than the ‘stop flow diameter’ of
the defect using a balloon is chosen. For the STARFlex/
CardioSEAL or Helex occluder, a device twice the diam-
eter of the balloon occluding the defect is chosen.
This limits the size of defects that can be closed with these
devices to 18–20 mm in diameter.

The device is loaded onto the delivery system and the
left atrial disc/umbrella is deployed inside the left
atrium. The disc/umbrella assembly is then withdrawn
until it is very close to the septum, then the connecting
waist is deployed to ‘stent’ the defect in the case of
the ASO or until the disc is flushed against the septum
in the case of the Helex/CardioSEAL devices. Then
the right atrial disc is deployed by further retraction of
the delivery sheath over the cable. Prior to release, a safe
and good position of the device has to be confirmed.
Echocardiography (TEE or ICE) is of paramount impor-
tance to determine device position and that surrounding
structures are not interfered with.37 The device is then
released. After release, the device is again evaluated
by echocardiography to ensure proper position and to
assess closure result. Figures 1–3 demonstrate the
closure steps of a secundum ASD by ICE and fluoroscopy
using the Helex device.
Patients are placed on acetyl salicylic acid 2 days prior
to procedure, and continue for 6–12 months after procedure.
Owing to migraine headaches in ~10% of patients, the
addition of 75 mg per day of Clopidogrel for 1–3 months
has been a practice over the last 5 years.38,39 Patients
undergoing uncomplicated transcatheter ASD closure can
be discharged home the following day. Transthoracic echocardiogram is done the day following closure to establish a new baseline for future surveillance evaluations. Patients are recommended to follow-up at least annually following device closure and repeat echocardiography is recommended between 3 months and 1 year after device closure and periodically thereafter.\textsuperscript{18}

Peri-procedural complications such as device embolization, thrombus formation, stroke, perforation, and tamponade are rare with near-zero procedural mortality.\textsuperscript{20,22,24,40,41} Early post-procedurally, transient atrial fibrillation may occur.\textsuperscript{42,43} Long-term complications are rare, with the erosion of the device into surrounding structures being the most serious and feared one.\textsuperscript{44–46}

**Figure 1** Intracardiac echocardiographic images in a 34 years old female with secundum ASD. (A, B) Septal view without and with color Doppler demonstrating moderate size ASD measuring about 12.5 mm defect (arrow). (C, D) Long axis view without and with color Doppler demonstrating the defect (arrow) and the superior rim (SR), inferior rim (IR). (E, F) Short axis view without and with color Doppler demonstrating very short anterior rim (AR) and posterior rim (PR) and the defect (arrow). RA, right atrium; LA, left atrium; SVC, superior vena cava; AO, aortic valve.

**Figure 2** Intracardiac echocardiographic images during deployment of a 30 mm Helex device in the same patient as in Figure 1. (A) Balloon sizing of the defect using “stop flow” technique. Arrows indicate size of the balloon. (B) Passage of the guide wire (arrow) into the left upper pulmonary vein, over which the delivery sheath was passed. (C) Deployment of the left atrial desk (arrow). (D) Deployment of the right atrial desk (arrow). (E, F) Assessment of final result by color Doppler in long (E) and short axis (F), both demonstrating good device position and no residual shunt.
The overall incidence is estimated at <0.1% for the ASO. Patients at higher risk for device erosion are those with defects associated with deficient anterior-superior rims and an oversized device. Thrombus formation on the closure device, if occurring on the left atrial side, can have catastrophic consequences. Occurrence of device-related thrombus formation seems to be related to the design of the device and appears to be the lowest with the Amplatzer and Helex occluders. If thrombus formation occurs, treatment with warfarin usually results in resolution of thrombi.

Ventricular septal defects

Definition and indications for closure

A VSD is a persistent communication between the right and left ventricles. It can occur as a congenital defect or can be acquired in the setting of an acute myocardial infarction, trauma or iatrogenic following aortic valve replacement or myomyectomy surgeries.

Congenital ventricular septal defects

Ventricular septal defects are the most common form of congenital heart defects in children. The most common type of VSD is the peri-membranous type that accounts for ~80% of all cases. The remaining 20% are muscular VSDs. Those can be further subdivided into inlet, trabecular, and infundibular defects, depending on their locations. Large, unrestricted VSDs lead to a large left-to-right shunt and congestive heart failure, which may become evident in the very first few weeks of life. If left untreated, shunting will eventually lead to pulmonary arterial hypertension with increased pulmonary vascular resistance and Eisenmenger’s physiology, at which point the shunt reverses to a predominant right-to-left shunt. Small or restrictive VSDs are usually diagnosed early in life, as they create significant murmurs. There is a good chance that both perimembranous and muscular VSDs would close spontaneously in the first few years of life. Closure of symptomatic, non-restrictive VSDs is indicated in infancy. Patients commonly present with signs and symptoms of heart failure, including poor growth, tachypnoea, tachycardia, and diaphoresis. Timing of closure, however, needs to be individualized. Infants with asymptomatic, restrictive VSDs can usually be closely followed. If the defect closes spontaneously, no further intervention is warranted.

Congenital ventricular septal defects in adults

Large, unrestricted VSDs are rarely diagnosed in adulthood. If a small VSD persists into adulthood, closure, however, is necessary for the following indications: left ventricular and/or left atrial volume overload; history of infective endocarditis or development of aortic valve insufficiency caused by the VSD. Transthoracic echocardiography is the best diagnostic tool to assess the size of the left atrium and ventricle. We do not recommend to rely on the calculation of the $Q_p/Q_s$ ratio since it is flawed with many errors. Owing to the complexity of transcatheter VSD closure and the higher incidence of complications, surgical VSD closure remains the gold standard for the treatment of most VSDs. Transcatheter VSD closure, as opposed to surgical closure, can be considered in patients with muscular defects, especially if the VSD is remote from the tricuspid and aortic valves.
Patients with residual defects after surgical closure or patients with iatrogenic defects after aortic valve replacement surgery or myomectomy\textsuperscript{5,4,5} are better served by a transcatheter closure. Closure of peri-membranous VSDs is possible with the Amplatzer peri-membranous VSD occluder; however, the risk of complete heart block is high, at ~5–6\textsuperscript{5,6}.

**Acquired ventricular septal defect**

The second group of VSDs consist of acquired defects occurring after myocardial infarction or residual defects after surgical repair. Post-myocardial infarction VSDs nowadays occur relatively rarely in \(<0.2\%\) of cases.\textsuperscript{57} The mortality and morbidity of this condition, if left untreated, is extremely high, estimated to be \(>90\%).\textsuperscript{56} Owing to the high operative mortality (~50%), transcatheter closure is an attractive alternative.\textsuperscript{57} Patient selection and timing of transcatheter VSD closure are critical and highly determine the outcome. The procedure is complicated by the patient’s haemodynamic instability and the nature of the VSDs, which are rather irregular and large with surrounding tissue necrosis, making stable device positioning difficult. Mortality after transcatheter post-myocardial infarction VSD closure is probably in the range of 25–35% and may be even higher in patients with frank cardiogenic shock.\textsuperscript{56,59} (Table 1).

**Ventricular septal defect closure devices**

The Amplatzer muscular VSD occluder (AGA Medical, Plymouth, MN, USA) is the only device specifically designed for congenital muscular VSDs (Figure 3). Since its initial use in 1998, it has become the most popular device to close muscular VSDs worldwide.\textsuperscript{58,60} Similar to the ASO for ASDs, the muscular VSD occluder is made of nitinol wire mesh in the form of a self-expandable double-disc device. The waist diameter determines the maximum size of VSD that can be treated. The largest available device for congenital defects is 18 mm in diameter and that for post-infarct VSDs is 24 mm in diameter. The mechanism of closure, again similar to the ASO, involves stenting of the actual defect by the waist and subsequent thrombus formation within the device and eventual complete endothelialization. Common contraindication to the use of this device includes a distance of \(<4\, \text{mm}\) between the VSD to any of the four valves. A second device used to close congenital, muscular VSDs, and post-myocardial infarction VSDs is the aforementioned CardioSEAL occluder.

Transcatheter closure of peri-membranous VSD has been difficult due to its close proximity to surrounding valves. Specifically designed for peri-membranous VSDs, the Amplatzer membranous VSD occluder was first implanted in 2002. Owing to its unique design, it overcame several obstacles and problems of prior devices used for this indication.\textsuperscript{56,61} It is designed differently from the muscular VSD occluder from AGA. However, similar to Amplatzer family of devices, it is made of nitinol wire and is self-expandable. The waist length is only 1.5 mm, accommodating the defect located in the thin-walled membranous septum. The left ventricular disc is asymmetric with a shorter aortic end of the left ventricular disc. This allows stable positioning, yet minimal encroachment on the aortic valve region. Following device placement, conduction abnormalities to the degree of complete atrio-ventricular block may occur and remain a concern with this device.\textsuperscript{62,63}

The Amplatzer post-infarct VSD device is very similar to the congenital muscular VSD device. However, the length of the waist is 10 mm to accommodate the thick septum in adult patients and the disks are 5 mm larger than the waist. As mentioned above, the largest size available is 24 mm.\textsuperscript{58}

Owing to the higher incidence of complete heart block after membranous VSD closure with the Amplatzer membranous VSD device, some operators prefer to use coil devices to close such defects.\textsuperscript{64,65}

**Closure technique**

Transcatheter VSD closure is technically more challenging than ASD closure. Nevertheless, success rates for transcatheter VSD closure is \(>90\%\) in experienced hands.\textsuperscript{63,64,66} Closing acquired VSDs is even more complex due to co-morbidities as well as local factors such as surrounding necrotic myocardial tissue and resulting difficulties in safely anchoring a device. To exemplify VSD closure technique in general, we briefly describe the steps of transcatheter VSD closure using the Amplatzer muscular VSD occluder (Figure 4). Prior echocardiographic assessment of the size, number, and location of the VSDs is of paramount importance. The procedure is preferentially performed under general anaesthesia with continuous TEE and fluoroscopic guidance. For single muscular VSD, one can perform the closure under fluoroscopic guidance alone with or without transthoracic echocardiography. Echocardiography is essential to evaluate the relationship of the device to the atrio-ventricular valves. Access is obtained in the femoral artery and vein. If the VSD, however, is located in the mid-ventricular septum, or in the posterior or apical septum, the right internal jugular vein should be used as the venous access for better device deployment.\textsuperscript{6} Left ventriculography is performed to define the location, size, and number of VSDs. For membranous defects, the best projection is the long axial oblique (60° left anterior oblique/20° cranial), and for mid-muscular defects, the best projection is the four-chamber view (35° left anterior oblique/35° cranial). Defect sizes are measured by either TEE or left ventriculography at end-diastole. Following device selection, the defect is crossed, usually from the left ventricular side using a Judkins right catheter, a wire is passed and exteriorized from the venous side (jugular for muscular defects/femoral for membranous defect), and the delivery sheath is advanced over this wire to the left ventricle. Placement of the Amplatzer VSD occluders is comparable with the ASO with a sequential deployment of the left-sided disc, followed by the waist and the right-sided disc. Prior to release, a stable position is confirmed by echocardiography and/or angiography. The surrounding valves are interrogated confirming normal function.
Table 1  Contemporary results of transcatheter ventricular septal defect closure

<table>
<thead>
<tr>
<th>Author</th>
<th>Device</th>
<th>Type (n)</th>
<th>Patients (n)</th>
<th>Age in years (range)</th>
<th>Success rate (%)</th>
<th>Complication rate</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chessa et al.</td>
<td>Amplatzer</td>
<td>M² (22), PM³ (18)</td>
<td>40</td>
<td>38 (18–64)</td>
<td>100</td>
<td>14.6% (2/3 arrhythmias)</td>
<td>One surgical correction</td>
</tr>
<tr>
<td>Thiele et al.</td>
<td>Amplatzer</td>
<td>PI²</td>
<td>29</td>
<td>72 (48–84)</td>
<td>86</td>
<td>41%</td>
<td>30-day survival: 35%</td>
</tr>
<tr>
<td>Qin et al.</td>
<td>Modified Amplatzer</td>
<td>PM</td>
<td>412</td>
<td>16 (3–65)</td>
<td>96.6</td>
<td>2.2%</td>
<td>No device-related complications</td>
</tr>
<tr>
<td>Nogi et al.</td>
<td>Coils</td>
<td>PM</td>
<td>41</td>
<td>13 (2–60)</td>
<td>100</td>
<td>—</td>
<td>Complete closure in all cases</td>
</tr>
<tr>
<td>Butera et al.</td>
<td>Amplatzer</td>
<td>PM</td>
<td>104</td>
<td>14 (0.6–63)</td>
<td>96.2</td>
<td>—</td>
<td>Complete closure in 99%</td>
</tr>
<tr>
<td>Carminati et al.</td>
<td>Amplatzer, Starflex, coils</td>
<td>M (119), PM (250), PS⁵ (45)</td>
<td>430</td>
<td>8 (0.4–70)</td>
<td>95</td>
<td>—</td>
<td>Four cases of late-occurring CHB-requiring pacemaker</td>
</tr>
<tr>
<td>Lim et al.</td>
<td>Cardioseal</td>
<td>M</td>
<td>55</td>
<td>8 (0.6–63)</td>
<td>92</td>
<td>8% (embolization 4%); 2% (CHB); 3% (pacemaker)</td>
<td>Complete closure at 6 months: 84%</td>
</tr>
<tr>
<td>Holzer et al.</td>
<td>Amplatzer</td>
<td>PM</td>
<td>100</td>
<td>9 (0.7–58)</td>
<td>93</td>
<td>2% (rhythm abnormality); 13% (CHB); 2% (pacemaker)</td>
<td>Two cases of late-occurring CHB-requiring pacemaker</td>
</tr>
<tr>
<td>Carminati et al.</td>
<td>Amplatzer</td>
<td>M (30), PM (87), PS (5)</td>
<td>122</td>
<td>15 (0.5–64)</td>
<td>97.5</td>
<td>2.5%; 2.5% (CHB); 2.5% (pacemaker)</td>
<td>No device-related complications</td>
</tr>
<tr>
<td>Arora et al.</td>
<td>Rashkind, Amplatzer</td>
<td>M</td>
<td>50</td>
<td>Not specified (³–²⁸)</td>
<td>100</td>
<td>—</td>
<td>30-day mortality: 28%</td>
</tr>
<tr>
<td>Holzer et al.</td>
<td>Amplatzer</td>
<td>PI</td>
<td>18</td>
<td>75 (52–86)</td>
<td>89</td>
<td>Failure rate 11%</td>
<td>Explantation rate: 10.5%</td>
</tr>
<tr>
<td>Knauth et al.</td>
<td>Starflex</td>
<td>M (92), PS (78)</td>
<td>170</td>
<td>3.9 (0.3–73)</td>
<td>99</td>
<td>Moderately serious/serious complications: 48%</td>
<td></td>
</tr>
</tbody>
</table>

¹Muscular ventricular septal defect.
²Peri-membranous ventricular septal defect.
³Post-myocardial Infarction ventricular septal defect.
⁴Post-surgical ventricular septal defect.
⁵Complete heart block.
Potential major complications include device embolization, arrhythmias, air embolism, haemolysis, valvular regurgitation, or higher degree heart blocks. In the initial US multicentre trial for muscular VSD closure, peri-procedural major complication rate was 10.7%. In more recent studies, this complication rate has decreased to 2.8% (Table 1).

Patent ductus arteriosus

Definition and indications for closure

A PDA is a persistent communication between the descending aorta and the pulmonary artery. This critical component of the foetal circulation usually closes within 72 h after birth. It is a common congenital heart defect, comprising approximately 10% of all defects. Owing to the loud murmur, diagnosis is usually made in infancy or childhood. Because of low resistance in the pulmonary vasculature, this lesion results in left-to-right shunt. If small, it usually does not lead to symptoms. However, larger PDAs are associated with significant left-to-right shunt, usually resulting in symptoms of heart failure, frequent chest infections, and failure to thrive early in life.

Owing to systemic hypertension, small-to-moderate PDAs can present with new symptoms of heart failure in adulthood. This is due to an increase in the left-to-right shunt. Another complication associated with PDAs is the risk for endarteritis. Although no data are available directly comparing closure of small, haemodynamically non-significant PDAs in asymptomatic patients vs. dental prophylaxis for the prevention of endarteritis, it is commonly accepted to close all audible PDAs to prevent the risk of endarteritis, occurrence of heart failure, and irreversible pulmonary arterial hypertension. It is controversial to close small silent PDAs that are incidentally found on echocardiography. Surgical closure has largely been replaced by transcatheter closure. Particularly in adults, surgical closure carries greater risks due to the calcifications of the PDA and surrounding friable tissue. However, if the PDA is associated with other congenital abnormalities that require surgical correction, it can be ligated during the same procedure.

Patent ductus arteriosus closure devices

Small PDAs, measuring <2–3 mm in diameter, can usually be closed using coils. Commonly used coils are Gianturco coils or Flipper coils, which are detachable. Another coil device is the Nit-Occlud PDA occluder, which is available outside the USA. Aside from the minimal diameter of the communication, the anatomy of the PDA is important to allow successful closure with coils. Commonly, the PDA has the shape of a funnel, with an ampulla on the aortic aspect of the duct and narrowest point usually close to the pulmonary arterial insertion. Those duct morphologies, if small in diameter, can be closed effectively with coils. Owing to the lack of discrete constriction, tubular ducts without a distinct ampulla are more difficult to close with coils; they carry a higher risk of coil migration. Although it is technically possible to close larger PDAs with multiple coils, most operators...
prefer to close PDAs larger than 3 mm in diameter with the Amplatzer duct occluder.\textsuperscript{13,76–78}

The Amplatzer duct occluder consists of an aortic retention disc and a plug which is 4 mm smaller in diameter than the retention disc.\textsuperscript{79} The largest device can close PDAs measuring up to 11–12 mm in diameter at the pulmonic end. PDAs larger than 12 mm in diameter can be closed with Amplatzer VSD or ASD occluders.\textsuperscript{80,81}

Closure technique

Careful procedural planning is necessary, considering several aspects of the PDA morphology such as duct size at its narrowest portion, size, and shape of the ampulla, and height and weight of the patient. Smaller PDAs, defined as <2–3 mm, are usually coiled; whereas larger PDAs are closed using the Amplatzer duct occluder. In the case of coil closure, a coil diameter of at least twice the diameter of the measured minimal duct diameter is chosen. The length of the coil is determined by the size of the ampulla. Several turns of the coil are placed in the ampulla, with less than half a turn of the coil being placed in the pulmonary arterial end of the PDA. If incomplete closure is found, a second coil should be deployed in a similar fashion. Success rate is high, with complete closure of the PDA being accomplished in nearly all cases when the device was placed successfully.\textsuperscript{72}

Inability to deliver the coils especially in small infants is one of the scenarios when surgical closure may be necessary. Peri-procedural complications are rare, coil embolization in the pulmonary artery or descending thoracic aorta may occur, although with controlled release, detachable coils, this complication is much less.\textsuperscript{82} Haemolysis is a rare complication of PDA closure and is caused by excessive shear stress destroying red blood cells. It is usually seen only in patients with large PDAs and residual flow after the procedure. Incidence is <1%.\textsuperscript{83,84} The approach for coil deployment can be either retrograde from the aortic side or antegrade from the venous side. We prefer to use the antegrade route, since the arterial catheter is used for angiography to verify coil position prior to release.

Closure of PDAs with the Amplatzer duct occluder is carried out after aortography and delineation of the exact duct morphology (Figure 5). A device at least 2 mm larger than the narrowest PDA diameter is chosen. The delivery sheath is placed from the venous side into the descending thoracic aorta. The device is deployed in a similar fashion as the other Amplatzer occluder. Angiography in the descending aorta is performed to verify device position prior to release. If position is not optimal, the device can be recaptured and the steps are repeated. Commonly, small residual shunt through the device is seen immediately after the device has been released. This shunt disappears within few days in most patients. Most common PDA morphologies are amenable to closure with the Amplatzer duct occluder. However, long, tubular ducts are difficult to close with the Amplatzer duct occluder. In such cases, Amplatzer Vascular Plug-II can be used.\textsuperscript{85} Complications occur rarely with the Amplatzer duct occluder.\textsuperscript{86} Embolization and haemolysis are described sporadically (Table 2). In some patients with pulmonary arterial hypertension (mean pulmonary artery pressure > 30 mmHg), it is better and safer to use the Amplatzer muscular VSD device. The risk of device migration with Amplatzer muscular VSD device in this position is less than the conventional PDA device.

![Figure 5](https://academic.oup.com/eurheartjsupp/article-abstract/12/suppl_E/E24/451961/12s supper_E24451961)
Table 2: Contemporary results of transcatheter patent ductus arteriosus closure

<table>
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<tbody>
<tr>
<td>Forsey et al.</td>
<td>ADO IIa</td>
<td>27</td>
<td>1.4 (0.4–76)</td>
<td>92.5</td>
<td>96%</td>
<td>96%</td>
</tr>
<tr>
<td>Huang et al.</td>
<td>ADOb, coils</td>
<td>76</td>
<td>6 (0.1–46)</td>
<td>98</td>
<td>3.2 (0.1–5)</td>
<td>98.5</td>
</tr>
<tr>
<td>Thanopoulos et al.</td>
<td>ADO II</td>
<td>25</td>
<td>3.2 (0.2–63)</td>
<td>100</td>
<td>7.7 (1–22)</td>
<td>92.3</td>
</tr>
<tr>
<td>Wang et al.</td>
<td>ADOb, coils</td>
<td>26</td>
<td>7.7 (0.5–29)</td>
<td>100</td>
<td>3.4 (0.2–71)</td>
<td>99.4</td>
</tr>
<tr>
<td>Celiker et al.</td>
<td>Nit-Occlud</td>
<td>64</td>
<td>1.8 (0.2–71)</td>
<td>99</td>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

Note: *ADO = Amplatzer Duct Occluder, *ADOb = Amplatzer Duct Occluder II*

Conclusion

Transcatheter closure of acquired and congenital defects has evolved rapidly. The majority of ASDs and PDAs can be closed safely and effectively with transcatheter techniques. Device closure of muscular VSDs is considered a very good alternative to conventional surgical closure. However, due to the higher risk of complete heart block, device closure of membranous VSDs should be reserved for selected cases.

Conflict of Interest: none declared.

References

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51. Crenshaw BS, Granger CB, Birnbaum Y, Pilper KS, Morris DC, Kleinman NS, Vahanian A, Calif RM, Topol EJ. Risk factors angiographic

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