We carried out an analysis of our initial experience with using an Amplatz device for percutaneous closure of ventricular septal defects in 15 patients. The patients' ages ranged from 1 to 44 years. Seven were infants with heart failure. In 4 patients, the ventricular septal defect was located in the muscular portion of the ventricular septum; in 11 patients, it was in the perimembranous portion. The mean size of the Amplatz device selected was 11.2±3.0 mm. Successful device implantation was achieved in 12 patients. In the other 3, stable occlusion could not be achieved, and the device was retrieved without complications. Immediate complete closure occurred in 11 of the 12 patients who underwent successful implantation. No complications were observed, either immediately or during follow-up. After a mean follow-up period of 9.2±3.6 months, all patients remained free of symptoms. Follow-up Doppler echocardiography demonstrated complete closure in all patients.

Key words: Ventricular septal defect. Percutaneous closure. Congenital heart disease.
(mean weight, 7.9±1.9 kg) and sweating when feeding. Among the 8 adults, 4 presented repeated respiratory infections, one had an ischemic cerebral event, and 3 were asymptomatic. One infant had VSD and associated pulmonary stenosis, which had been successfully dilated in a prior procedure. One adult with subaortic VSD had an associated patent foramen ovale (PFO) with prior symptoms of a transient ischemic attack due to paradoxical embolism. During the same procedure, each defect was closed with an Amplatz device.

Diagnostic Catheterization

The procedure was undertaken under general anesthesia and transesophageal monitoring. Previously, a right and left hemodynamic study was performed. In addition, left ventriculography was done in 2 views: LAO 30°-craniad 40° and LAO 70°-craniad 20°. Subsequently, the defect was measured by angiography and echocardiography.

Therapeutic Phase

The diameter of the devices selected was between 2 mm and 3 mm greater than the diameter of the defect as measured by transesophageal echocardiography. An arteriovenous shunt was established with the exchange guidewire. The approach used to create the shunt varied according to the location of the defect:

- Muscular VSD (n=4): A Terumo guidewire was passed from the left to the right ventricle through the VSD and advanced to the pulmonary artery. A mammary catheter was advanced over the guidewire to this vessel, the Terumo guidewire was withdrawn, and a long (260 cm), 0.35 inch exchange guidewire was inserted. The end of this guidewire was snared with a loop inserted in the jugular vein. Subsequently the Amplatz muscular VSD occluder sheath was introduced in the jugular and advanced to the left ventricle, where the shunt was eliminated to insert the prosthesis.

- Perimembranous VSD (n=11): the same process as described was performed, with the exception that the guidewire was snared and the sheath inserted through a femoral vein route. In both cases, the sheath was inserted through a venous access to reach the left ventricle. Once the device (Amplatz membranous VSD occluder) had been connected, proper location was confirmed by angiography and echocardiography before deployment. In patients with a subaortic VSD, aortography was also performed before releasing the device to ensure that the aortic sigmoid arteries had not been compromised.

Following the procedure, all patients were treated with subcutaneous dalteparin for 1 month and aspirin for 6 months, as well as antibiotic prophylaxis to prevent endocarditis. Patients were closely monitored by frequent telephone contacts, and clinical follow-up visits were performed at 3, 6, and 12 months.

RESULTS

The main baseline and procedure-related data are shown in Table 1. Immediate results are summarized in Table 2. Proper implantation of the device was achieved in 12 of the 15 patients (80%). In the 3 remaining patients, the device could not be stabilized; hence, it was removed as a safety measure without incidents.

**TABLE 1. Baseline and Procedure Data**

<table>
<thead>
<tr>
<th>Age, y</th>
<th>14±14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women</td>
<td>3/15 (20%)</td>
</tr>
<tr>
<td>Clinical presentation</td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>7</td>
</tr>
<tr>
<td>Prior cerebral ischemic events</td>
<td>1</td>
</tr>
<tr>
<td>Repeated respiratory infections</td>
<td>4</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>3</td>
</tr>
<tr>
<td>Associated disease</td>
<td></td>
</tr>
<tr>
<td>PFO</td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td></td>
</tr>
<tr>
<td>Associated disease</td>
<td></td>
</tr>
<tr>
<td>PFO</td>
<td>1</td>
</tr>
<tr>
<td>Pulmonary stenosis</td>
<td></td>
</tr>
<tr>
<td>Associated disease</td>
<td></td>
</tr>
<tr>
<td>VSD</td>
<td>11/15 (73%)</td>
</tr>
<tr>
<td>Muscular VSD</td>
<td>4/15 (27%)</td>
</tr>
<tr>
<td>VSD with aneurysm</td>
<td>3</td>
</tr>
<tr>
<td>VSD diameter by angiography</td>
<td>7.6±4 mm</td>
</tr>
<tr>
<td>VSD diameter by TEE</td>
<td>8.9±4 mm</td>
</tr>
<tr>
<td>Mean size of occluder</td>
<td>11.2±3 mm</td>
</tr>
</tbody>
</table>

*PFO indicates patent foramen ovale; TEE, transesophageal echocardiography; VSD, ventricular septal defect.

**TABLE 2. Results**

| Successful implantation | 12/15 (80%) |
| Primary success          | 11/15 (73%) |
| Major complications (embolism, endocarditis) | 0 |
| Pulmonary/systemic flow ratio* |       |
| Baseline                 | 1.7±0.25 |
| Post-closure             | 1.1±0.27 |
| Pulmonary artery pressure* |       |
| Baseline, mm Hg          | 38±18   |
| Post-closure, mm Hg      | 27±7    |
| Residual shunt           |         |
| Angiographic (posterior in catheterization unit) | No |
| Mild                      | 4       |
| Transesophageal (posterior in catheterization unit) | No |
| Mild                      | 4       |
| Transthoracic at discharge |       |
| No                        | 11      |
| Mild                      | 1       |

*P<.05.
Complete closure of the defect was observed in 11 of the 12 patients with a satisfactory implant (Figures 1 and 2). In the majority (8/12), immediate, complete closure was documented on angiography. Aortic valve function was not affected in any of the patients with subaortic VSD receiving an implant (Figure 2). Pulmonary arterial pressure decreased significantly in the patients with elevated values. Following closure, the flow ratio approached 1 in all patients.

There were no major complications or embolism of the device. Two patients experienced some type of rhythm or conduction abnormality (nodal rhythm and left bundle branch block) following implantation, which were transient. The echocardiographic images
of a subaortic VSD in which immediate successful 
closure was achieved are shown in Figure 3.

Following closure, the clinical course was favorable 
in all patients, particularly the group of infants with 
heart failure, who showed a considerable clinical 
improvement and weight gain. The mean weight of the 
infants treated increased from 7.5±2.5 to 13.4±7 kg. 
After a mean follow-up of 9.2±3.6 months there were 
no embolic complications or cases of endocarditis, and 
all the patients treated were asymptomatic at the time 
of writing. 

In the latest transthoracic Doppler echocardiography 
study, all the patients showed complete closure. At 
medium-term, aortic valve function was not 
compromised in any of the patients with a subaortic 
implant.

DISCUSSION

Percutaneous closure of a ventricular septal defect is 
a recent technique that seems to minimize the risks 
involved in treating this entity and provides excellent 
results. In recent years the percutaneous technique has 
undergone substantial development and various series 
of patients with satisfactory percutaneous closure have 
been reported.1-16 Correction of the defect is necessary 
in symptomatic patients and percutaneous treatment 
has been shown to be a viable alternative whenever 
possible. In asymptomatic patients over three years of 
age without hemodynamic repercussions in whom 
spontaneous closure is an exception, there have been 
discrepancies about whether to apply surgical 
treatment because of the associated risks. Nevertheless, some authors have indicated that 
morbidity and mortality is higher in patients who do 
not undergo correction.17,18 This group of patients can 
be considered candidates for percutaneous closure. 
The appearance of immediate or delayed arrhythmias, 
such as conduction abnormalities or atrioventricular 
block, is a potential complication of percutaneous 
closure, as well as surgical closure (in some surgical 
series, a more than 40% incidence of right bundle 
branch block following right ventriculotomy, which is 
usually transient).19 Although larger series are 
required, the incidence of these complications seems 
to be lower following percutaneous closure.

Various types of closure devices have been used 
previously for this condition.3-16 Nevertheless, their use 
has been gradually discontinued since the introduction 
of the Amplatz device, which has shown important 
advantages with respect to the earlier ones.3-16

In our initial series, closure was achieved in 80% of 
the cases without major complications and with a 
favorable evolution at follow-up. These findings are 
similar to the reported results from other series. 
Nevertheless, the experience is still limited and the 
follow-up in months is too short to draw definitive 
conclusions. The initial results are promising and 
indicate that in the near future, many of these defects 
can be treated using a percutaneous approach.
REFERENCES


