Perventricular device closure of muscular ventricular septal defects on beating hearts: Initial experience in eight children

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Objective: The conventional surgical repair and transcatheter closure of muscular ventricular septal defects are known to have undesirable limitations. This communication describes the short-term results of perventricular device closure of muscular ventricular septal defects with the heart beating in 8 children with 15 muscular ventricular septal defects, with or without other congenital malformations.

Methods: A subxiphoid minimally invasive incision was used in 3 children with isolated muscular ventricular septal defects whereas standard full median sternotomies were used in the other 5 children who required subsequent correction of coexisting malformations. Under the continuous guidance of transthoracic echocardiography, the free wall of the right ventricle was punctured, and a guidewire was introduced into the left ventricle through the defect. A delivery sheath was advanced over the wire and through the defect into the left ventricle. The device was released.

Results: A total of 14 muscular ventricular septal defects were successfully closed perventricularly without cardiopulmonary bypass. There was no mortality perioperatively or during the entire follow-up period. At 6-month follow-up, there was no detectable residual shunt, arrhythmia, or new mitral or tricuspid insufficiency. Other than 5 children with the coexisting malformations, none of the other children required any blood or blood products. The average hospital stay was 7.9 ± 2.2 days (range, 5–11 days).

Conclusion: Perventricular device closure of muscular ventricular septal defects with or without coexisting congenital malformations appeared to be safe and efficacious. The outcomes of short-term follow-up are acceptable.
was too close to the tricuspid annulus or the anterior rim of a large anterior defect was absent, we would not conduct a device closure. Three (37.5%) children had a singular MVSD and 1 of them had associated CHDs. All the other 5 (62.5%) children had multiple MVSDs with associated CHDs. The mean diameter of the MVSDs was 4.9 ± 2.4 mm (range, 2.0–10.0 mm). None of the children had cardiac arrhythmia before surgery. Patient demographics, the dimensions and locations of the MVSDs, and the associated CHDs are given in Table 1.

**The Devices**

There are two kinds of occluder (Shanghai Shape Memory Alloy Corporation, Shanghai, China): one is a muscular occluder and the other is a patent ductus arteriosus (PDA) occluder (Figure 1). The basic characteristics of the muscular occluder have been described before, but the flange of our device is 3 mm wider than the waist on the left ventricular (LV) side and 2 mm wider than the waist on the right ventricular (RV) side. The PDA occluder is similar to that in another study, and each occluder is designated by the diameters of both the LV and RV waist.

**Perventricular Technique**

In the child with isolated MVSDs and no CPB anticipated, a subxiphoid 3- to 4-cm skin incision was made followed by a lower partial sternotomy. The pericardium was partially and longitudinally incised and suspended for adequate exposure. In the other children with associated CHDs, conventional full median sternotomies were used. With the heart beating and under transesophageal echocardiogram (TEE) guidance, the RV free wall was gently depressed with the surgeon’s index finger. The depression on the RV free wall was easily visualized by TEE. By changing the location of the depression, we could determine a point on the RV free wall that was nearest to the defect to be occluded and yet preserve adequate distance and space to allow manipulation of the guidewire, sheath, and release of the device. The following technique was similar to that in another research report. If a PDA occluder was used, the LV disc was similarly released first, followed by the release of the waist. The appropriate device size was chosen to be 1 to 2 mm larger than the defect size. If a single device was selected to close 2 nearby defects simultaneously, the diameter of its left disc should just cover the 2 VSDs. At this point, the echocardiographer would carefully look for any residual shunt or new mitral or tricuspid regurgitation. Twelve-lead electrocardiograms were also obtained at the same time to detect any arrhythmia.

### RESULTS

Fourteen (93%) of 15 MVSDs were successfully closed without CPB. Eleven MVSDs were closed by muscular occluders and 3 MVSDs were closed by PDA occluders.

**Singular MVSD**

Three children with singular MVSD successfully underwent PVDC with 8-mm, 10-mm, and 10-mm devices for their 7-mm, 8-mm, and 8-mm defects, respectively. There was no detectable residual shunt or any new mitral or tricuspid regurgitation by TEE after the releases of the devices. The child with associated CHDs (patient 1, Table 1) also underwent uneventful repair of her CHDs with CPB.

**Multiple MVSDs**

In the child with a 10-mm anterior MVSD and a 6-mm apical MVSD (patient 4, Table 1), we closed the anterior defect with a 12-mm device uneventfully. We then used another RV free wall puncture site to establish a second pathway closer to the apex to close the apical MVSD (Figure 2). Since we anticipated that the RV apex would not have enough space to accommodate the regular occluder, we elected instead to use an 8/6-mm PDA occluder, which did not have an RV disc. The associated secundum type 6-mm atrial septal defect (ASD) was successfully closed with an 8-mm ASD occluder (Shanghai Shape Memory Alloy Corporation) via a right atrial puncture site (Figure 3). Another child (patient 5, Table 1) had 2 apical MVSDs, a large PDA, and a perimembranous VSD (PVSD). We attempted unsuccessfully to use the regular MVSD occluders to close these 2 apical MVSDs owing to the limited space at the apex, making opening the RV disc of the regular occluder impossible. We therefore switched to using two 8/6-mm and 6/4-mm PDA occluders to successfully close the 6-mm and 4-mm apical MVSDs. Immediate TEE examination did not detect any residual shunt. CPB was then established and the large PDA was ligated before cardiopulcric arrest and repair of the PVSD.

In the child with 2 relatively small MVSDs that were only 3-mm apart, as well as a nonrestrictive PVSD (patient 6, Table 1), we closed the 4-mm posterior MVSD with a 6-mm occluder relatively easily and immediate TEE examination did not show any residual shunt. The nearby 2-mm MVSD was also reduced to a 0.5-mm defect. Owing to the small size of this defect and its close proximity to the occluder of the first defect, we were unable to pass the guidewire into the LV cavity after multiple attempts. We then accurately established the spatial relationship between this small defect and the first occluder before establishing CPB and closing this defect with pledget-supported 4-0 Prolene

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**Abbreviations and Acronyms**

- ASD = atrial septal defect
- CHD = congenital heart disease
- CPB = cardiopulmonary bypass
- LV = left ventricular
- MVSD = muscular ventricular septal defect
- PDA = patent ductus arteriosus
- PVDC = perventricular device closure
- PVSD = perimembranous ventricular septal defect
- RV = right ventricular
- TEE = transesophageal echocardiogram
polypropylene suture (Ethicon, Inc, Somerville, NJ) through the tricuspid valve. The coexisting PVSD was closed in the usual fashion with a patch.

In another child (patient 7, Table 1) with two 3-mm large MVSDs that were 3-mm apart, we closed the defects with a single 6-mm occluder. The last girl (patient 8, Table 1) had 4 MVSDs and an ASD. We closed the 2 nearby anterior MVSDs with a single occluder and closed the middle and posterior MVSDs with two 5-mm occluders. Her ASD was also closed peratrially with a 12-mm ASD occluder (Shanghai Shape Memory Alloy Corporation). TEE showed all 6 MVSDs were closed successfully and no residual shunt was detected.

At discharge and 6-month follow-up, there was no detectable residual shunt or any closure-related new mitral or tricuspid regurgitation or exacerbation of any pre-existing regurgitation. All the occluders appeared to be in good positions, and no arrhythmia was noticed. There was no mortality during the hospital stay or during the follow-up period. Three children who required CPB to repair the associated CHDs and 2 children who received multiple devices needed blood transfusion; the other 3 children did not require any blood or blood product transfusion. The average hospital stay was 7.9 ± 2.2 days (range, 5–11 days).

### TABLE 1. Characteristics of the patients, MVSDs, and associated CHDs

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Gender (M/F)</th>
<th>Age (y)</th>
<th>Weight (kg)</th>
<th>MVSD1 Location (Size [mm])</th>
<th>MVSD2 Location (Size [mm])</th>
<th>MVSD3 Location (Size [mm])</th>
<th>MVSD4 Location (Size [mm])</th>
<th>Associated CHDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F 8/12</td>
<td>8</td>
<td>7/middle (8/MO)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>PAPVC, ASD (posterior, 12 mm), PVSD (12 mm), TR (moderate)</td>
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<tr>
<td>2</td>
<td>M 6/12</td>
<td>5.5</td>
<td>8/middle (10/MO)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>M 10/12</td>
<td>8</td>
<td>8/middle (10/MO)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>F 9/12</td>
<td>6</td>
<td>10/anterior (12/MO)</td>
<td>6/apical (8-6/PDAO)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>ASD (ostium secundum, 6 mm)</td>
</tr>
<tr>
<td>5</td>
<td>F 19/12</td>
<td>9.5</td>
<td>6/apical (8-6/ PDAO)</td>
<td>4/apical (6-4/PDAO)</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>PDA (10 × 8 mm), PVSD (15 mm)</td>
</tr>
<tr>
<td>6</td>
<td>M 11</td>
<td>27.5</td>
<td>4/posterior (6/MO)</td>
<td>2/posterior</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>PVSD (25 mm), TR (mild)</td>
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<tr>
<td>7</td>
<td>M 4</td>
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<tr>
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<td>3.5/anterior (7/MO)</td>
<td>3.5/anterior (7/MO)</td>
<td>3/middle (5/MO)</td>
<td>3/posterior (5/MO)</td>
<td>—</td>
<td>ASD (ostium secundum, 12 mm)</td>
</tr>
</tbody>
</table>

ASD, Atrial septal defect; CHDs, congenital heart diseases; F, female; M, male; MO, muscular occluder; MVSD, muscular ventricular septal defect; PAPVC, partial anomalous pulmonary venous connection; PDA, patent ductus arteriosus; PDAO, patent ductus arteriosus occluder; PVSD, perimembranous ventricular septal defect; TR, tricuspid regurgitation.

**FIGURE 1.** Top, The muscular occluder with the flange that is 3 mm wider than the waist on the left ventricular side and 2 mm wider than the waist on the right ventricular side. Bottom, The patent ductus arteriosus occluder with a 5-mm–tall isosceles trapezoid sagittal section and its left ventricular flange, which is 2 mm wider than the waist on the left ventricular side.

**FIGURE 2.** Another sheath was established through the apical defect after an anterior defect had been closed. LV, Left ventricle; MO, muscular occluder; RV, right ventricle; S, sheath.
increase the likelihood of arrhythmia, but it can never
ventriculotomy will impair the long-term RV function and
cavitated. Owing to the unique locations of some of the MVSDs,
are associated with other CHDs, early intervention is indi-
come hemodynamically significant, especially when they
of any residual shunt, and, if necessary, timely corrective
the effects of the devices on valve functions, the presence
measures. On the other hand, the surgeon doing a convent-
repair with CPB would have to wait until the child
is weaned from CPB before an accurate assessment of his re-
with TEE can be obtained. In the case of any residual
return to CPB is inevitable and time consuming.
Many studies have pointed out the adverse effects of CPB
in general and specifically on the development of the brain
in children. More recent studies also documented that
perioperative mortality and morbidity can be improved
if CPB is avoided. From this point of view, PVDC of
not only can eliminate the side effects of CPB alto-
gether if the child has no other associated CHDs, but can
also markedly reduce the CPB time if the child does have
other associated CHDs and also reduce the risk of postoper-
residual shunt.

Our experience with the child (patient 6, Table 1) who had
2 small nearby MVSDs might have suggested that after the
larger defect was closed with a device, the nearby smaller
defect might become even smaller, making it difficult to
pass the guidewire to the LV cavity to establish another path-
way for the deployment of the second device. This might be
due to the following: (1) the waist of the already inserted de-
vice might be compressing the muscle bridge between the 2
defects, thus effectively narrowing the diameter of the un-
closed defect, and (2) the flanges of the in-site device might
be partially covering up the nearby defect. If this occurs, it
might be easier to close it with the aid of CPB, especially
if there are other associated CHDs that require CPB for cor-
rection after an accurate assessment of the orientation of the
residual defect with respect to the device has been estab-
lished. The idea of using a bigger than necessary device to
attempt to close 2 nearby small MVSDs is certainly tempt-
ing, and our experience with 2 cases has proved the feasibil-
ity. Our initial experience also suggested that PDA ocul-
ders can be effectively used to close apical MVSDs when the
geo-
ography of the apex makes it difficult or impossible to de-
ploy the regular bi-disc occluder.

In conclusion, PVDC appeared to be a safe, effective, and
less invasive option for the treatment of either singular or
multiple MVSDs, with or without associated CHDs. The re-
results of the 6-month follow-up, in terms of stability of the de-
ces, residual shunt, closure-related valve dysfunction, and
new arrhythmia, are satisfactory. However, the conclusion
derived from our experience is limited owing to the small
size of the patients. Further studies with more patients and
longer term follow-ups will be required to truly confirm the
role of PVDC as an established form of treatment for MVSDs.

References
Surgical repair of multiple muscular ventricular septal defects: the role of re-en-
Apical right ventriculotomy for closure of apical ventricular septal defects. Ann


