



## A Reproducible Technique to Repair Tricuspid Valve Dysfunction Associated with a Chronic Ventricular Septal Defect

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### Abstract

A small subset of patients with ventricular septal defects (VSD) is noted on echocardiography to have partially occlusive ventricular septal aneurysmal (VSA) tissue. These patients are often managed medically in anticipation of spontaneous closure. However, we have found that rather than being indicative of spontaneous closures this echocardiographic aneurysmal tissue can be misinterpreted, and represents a specific pattern of damage to the tricuspid valve. If echocardiographic VSA develops in the setting of a perimembranous VSD, contrary to historical practice, we feel that consideration should be given to earlier operation to limit progressive valve injury. Various techniques are available to surgically address tricuspid valve dysfunction during repair of a VSD, but we present a simple and reproducible technique to reliably repair the valve in this unique subset of patients.

**Keywords:** Congenital heart disease, CHD; CHD, Septal defects; CHD; Valve; Heart valve repair; Tricuspid valve

### Introduction

At our institution we have encountered an infrequent, but significant clinical problem in some patients with ventricular septal defects (VSD). The majority of our patients with a VSD present early (3-6 months of age) for repair with clear indications for surgery. However, small subsets of these patients are noted on echocardiography to have ventricular septal aneurysmal (VSA) tissue that is partially occluding the VSD (Figure 1). Patients that develop restrictive VSA tissue are often managed medically in anticipation of spontaneous closure. We have recently operated on 4 consecutive patients at our institution for a VSD whose referral to surgery was delayed because they were thought to have partially occlusive VSA tissue on echocardiogram. At the time of surgery it was noted that none of these patients actually had VSA tissue. The "VSA" tissue seen on echo actually represented a much thickened septal leaflet of the tricuspid valve that was prolapsing into the VSD. Each of these patients developed a consistent pattern of damage to the tricuspid valve. The purpose of this case report is [1] to highlight the pathological changes that can occur to the tricuspid valve and be misinterpreted as VSA, and [2] present a reproducible surgical technique to address significant tricuspid valve damage in this subset of patients.

### Surgical Technique

Exposure was performed through a median sternotomy and cardiopulmonary bypass was established with central aortic cannulation and bicaval venous cannulation. Cardiac arrest was achieved with antegrade cardioplegia and the left heart was vented through the right superior pulmonary vein.

#### VSD inspection and closure

The VSD was approached through a standard right atriotomy with retraction of the septal and anterior leaflets of the tricuspid valve. As mentioned above, no VSA tissue was identified in any patient. Close inspection revealed that the anterior portion of the septal leaflet would easily prolapse down into the VSD unless manually withdrawn. Each VSD was located in the perimembranous region and standard closure was achieved with a Dacron patch.

#### Inspection of the tricuspid valve

After closure of the VSD, the right ventricle was injected with saline to test the competency of the tricuspid valve. On static testing, all 4 patients had tricuspid incompetence with prolapse of

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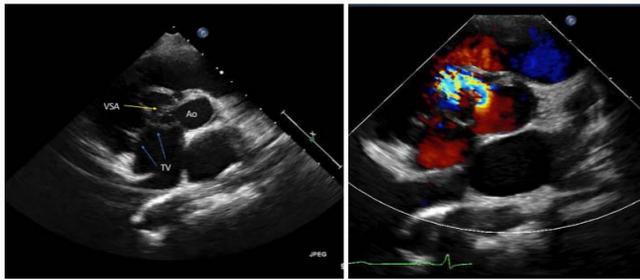
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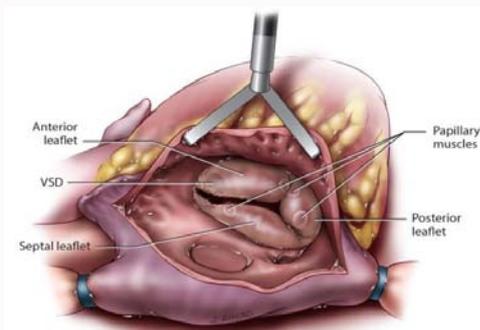
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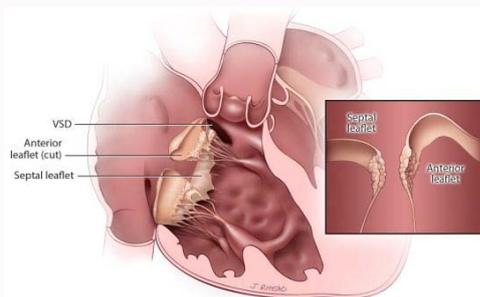
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**Figure 1:** Echocardiographic image of VSA in parasternal short axis. Ao: Aorta; TV: Tricuspid Valve Leaflets; VSA: Ventricular Septal Aneurysm.



**Figure 2A:** Surgeon's view of the tricuspid valve prior to repair.

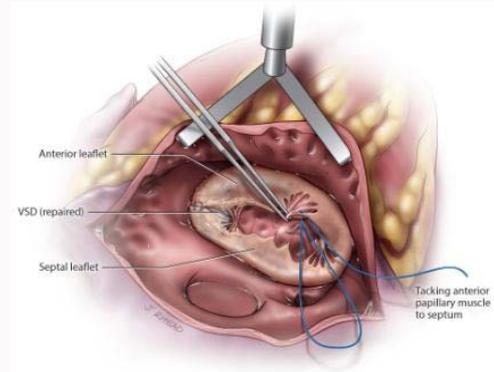


**Figure 2B:** Cross-sectional view of unrepaired VSD and tricuspid valve.

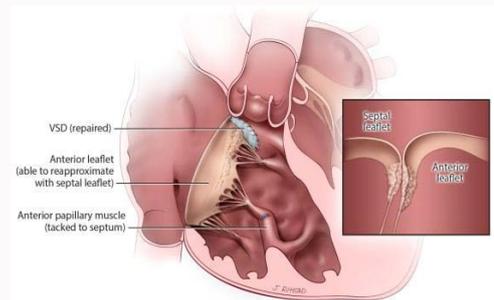
the anterior leaflet. Inspection of the valve revealed several consistent findings [1]; blunting of the commissure between the anterior and septal leaflet, significant thickening of the anterior and septal leaflet in the area of the commissure, significant attenuation and lengthening of the chordal and papillary muscle support to the anterior leaflet (Figure 2A,B). Using a blunt nerve hook, the free edge of the anterior leaflet could easily be elevated above the plane of the annulus.

**Tricuspid valve repair**

The same technique was used to repair all 4 tricuspid valves. The valvar disease was repaired by placing two horizontal mattress 5-0 polypropylene sutures in the anterosseptal commissure to recreate a sharp commissural angle and provide some support to both leaflets. The subvalvar disease was addressed at the papillary level by suturing the head of the anterior papillary muscle to the ventricular septum (Figures 3,4). The papillary muscle was translocated to the ventricular septal in the mid-portion of the septal leaflet area. The height of the translocation site was identified by using a blunt nerve hook to establish an appropriate height of the anterior leaflet in relation to the annular plane and still maintaining an adequate coaptation area.



**Figure 3:** Surgeon's view of repaired VSD and tricuspid valve.



**Figure 4:** Cross-sectional view of repaired VSD and tricuspid valve.

Once this was identified, the attenuated papillary muscle supporting the anterior leaflet was sutured to the ventricular septum using a single pledgeted 5-0 polypropylene suture in a horizontal mattress fashion. Typically, the papillary muscles are very mobile, but occasionally the base may need to be mobilized towards the apex to achieve a tension free repair.

**Results**

The first patient in the series had mild-to-moderate tricuspid regurgitation (TR) preoperatively. Initially, I only attempted a VSD closure but the intraoperative transesophageal echo showed severe TR with significant prolapse of the anterior leaflet. I returned to bypass and performed the above stated repair with a subsequent intraoperative echo showing trivial TR and no stenosis. The follow-up echo at 1 year showed a thickened tricuspid valve with mild prolapse of the anterior leaflet with trivial TR and no stenosis. The second patient preoperatively had mild TR with an intraoperative echo showing trivial TR and no stenosis. This patient has not followed up within our system. The third patient had moderate-to-severe TR preoperatively and the intraoperative echo showed trivial TR with no stenosis. One-year follow-up echo showed a thickened tricuspid valve with trivial TR and no stenosis. The fourth patient had mild TR preoperatively. The intraoperative echo showed no TR and no stenosis. Eight-month follow-up showed a thickened tricuspid valve with trivial TR and no stenosis.

**Discussion**

We report 4 consecutive patients who developed significant damage to the tricuspid valve apparatus while being followed for a VSD that was thought to be restricted by aneurysmal tissue. The assumption in each case was that the VSA was aneurysmal transformation of membranous septal tissue around the perimeter of

the VSD, and that it might facilitate spontaneous closure. However, no true aneurysmal tissue was identified at the time of surgery in any of these cases. The VSA described on echo was actually the anterior portion of the septal leaflet of the tricuspid valve prolapsing into the VSD and causing restriction.

Interestingly, the damage to the tricuspid valve in each scenario was similar; the anterior portion of the septal leaflet and septal portion of the anterior leaflet were both extremely thickened with aneurysmal enlargement of the anterior leaflet. Furthermore, the anterior papillary muscle was significantly attenuated and the chordae were elongated. Intraoperatively, in each case after VSD closure, the combination of these defects caused prolapse of the anterior leaflet producing valvar incompetence on static testing. All 4 valves were repaired in a similar fashion, with antero-septal commissuroplasty and translocation of the papillary muscle of the anterior leaflet to the ventricular septum. The translocation technique is very similar to the modified Sebening stitch used in the repair of Ebsteins anomaly [1]. Interestingly, each patient had a very similar pattern of injury upon surgical inspection that was not obvious on preoperative imaging. Only 1 patient had more than mild-moderate tricuspid valve regurgitation on preoperative imaging. All 4 patients left the operating room with trivial tricuspid valve regurgitation.

Injury to the tricuspid valve after VSD closure is a known complication and the mechanisms include direct damage to the valve, tethering or injury to the chordal support and annular distortion caused by either under- or over-sizing the patch. In these scenarios, the tricuspid leaflets are relatively normal, and the injury is surgically induced. However, in our subgroup of patients, the presence of

echocardiographic VSA was really a manifestation of significant damage to the tricuspid valve that remained clinically silent. Our theory, in three of the four cases, as to why there wasn't more preoperative tricuspid regurgitation is that the elongated portion of the anterior leaflet filled the void left by the septal leaflet prolapsing into the VSD. Certainly, not all VSDs with echocardiographic evidence of VSA will develop tricuspid dysfunction as described. However, we feel that the development or identification of VSA in a patient with a perimembranous VSD should be interpreted with caution. Rather than being a reassuring sign of spontaneous closure it may represent a pathologic adaptation of the tricuspid valve in which true tricuspid leaflet tissue causes partial VSD occlusion with resultant valve damage. If echocardiographic VSA develops in the setting of a perimembranous VSD then consideration should be given to earlier operation to limit progressive valve injury but in cases where the valve is already damaged this is a simple and reproducible technique to reliably repair the valve.

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