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Prolapsed Into a Ventricular Septal Defect

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日本外科宝函 50(1): 3-13

1981-01-01

http://hdl.handle.net/2433/208510
Techniques of Valvuloplastastic Surgery of the Aortic Valve Prolapsed Into a Ventricular Septal Defect

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Received for Publication, Sept. 19, 1980.

The surgical treatment of the aortic valve prolapsed into a ventricular septal defect (VSD) consists of valve repair or valve replacement. In this combined disease, pathologic changes are usually limited to only one or two of the three semilunar aortic cusps, and the remaining cusps or cusp usually maintain a normal contour and function. These valves should not be replaced by a prosthesis but repaired by valvuloplastic techniques.

Moreover, the various complications associated with anticoagulant therapy and the possible necessity for reoperation after valve replacement are hazards for both patients and physicians, because the majority of patients are in the pediatric age group.

Since 1967, we have been doing valvuloplastic surgery in all patients with aortic regurgitation (AR) and VSD whose valves should be surgically repaired. Actually, 33 patients have been treated by valvuloplastic surgery, 12 by closure of the VSD without valvular surgery and none by valve replacement. Of the 33 valvuloplasties, three needed reoperation within the next 13 years; one of these had valve replacement and two had a repeat valvuloplasty. There were no operative or late deaths in this series. A follow up study of all 45 patients proved the acceptable durability of valvuloplastic surgery.

From the technical standpoint, valvuloplastic surgery is more complicated and difficult than valve replacement, because in each case the most appropriate technique must be applied corresponding to the individual valve lesion. The purpose of this paper is to describe our recent principles and techniques of valvuloplastic surgery for aortic valves prolapsed into a VSD.

Key words: Techniques of valvuloplastic surgery, AR with VSD, Location of the VSD, Plication of the free edge, Reinforcement of the commissure.

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Principles of our valvuloplastic surgery

We have two main principles for the recovery of aortic valve function by valvuloplastic surgery for aortic valves prolapsed into a VSD. The first is to lift the prolapsed cusps up to the same level as the non-involved cusps by shortening the elongated free edge of the prolapsed cusps. The second is to strengthen commissural supports by reinforcement of the commissures of the involved cusps.

Surgical closure of the VSD may add to the effectiveness of valvuloplastic surgery in reducing AR. For instance, it is possible to diminish mild AR by surgical closure of the VSD alone in the right infracristal VSD.

Fig. 1. The upper figure shows the location of VSDs viewed from the right side. Supracristal VSDs lie just beneath the pulmonary valve. Infracristal VSDs lie just beneath the crista supraventricularis. The left lower figure shows the relationship between the aortic cusps and VSDs from the craniocaudal view. The right lower figure shows the location of VSDs viewed from the left side. Supracristal VSDs lie just beneath the right coronary cusp, and infracristal VSDs lie just beneath the right commissure.

CSV: crista supraventricularis; TV: tricuspid valve; PV: pulmonary valve; AV: aortic valve; MV: mitral valve; TV: tricuspid valve; R: right coronary cusp; N: non-coronary cusp; L: left coronary cusp.
cases of supracristal VSD, but not in cases of infracristal VSD. This fact shows the difference in surgical requirements depending on the location of the VSD. There are apparent differences in the mechanism of the prolapse of the aortic cusps between patients with supracristal VSD (Group I) and those with infracristal VSD (Group II). A defect in the supracristal region of the right ventricle is a defect just beneath the right coronary cusp of the aortic valve viewed from the left ventricle, while a VSD in the infracristal region of the right ventricle is just below the commissure of the left ventricle. VAN PRAAGH and McNAMARA noted these facts in their autopsy findings, and my clinical experiences confirm them (Fig. 1). Usually only a left to right shunt exists; however, aortic regurgitation appears in a few cases several years after birth for not completely understood reasons. In cases of supracristal VSD the right coronary cusp prolapses into defect, and this is the only cusp involved. However, in cases of infracristal VSD the deformity will frequently involve both right coronary and non-coronary cusps, but sometimes one cusp.

**Fig. 2.** The figures on the left show the mechanism by which the right coronary cusp prolapses into a supracristal VSD. The figures on the right show the mechanism by which the right coronary and non-coronary cusps and the right commissure prolapse into an infracristal VSD.

CSV: crista supraventricularis; VSD: ventricular septal defect; RV: right ventricle; LV: left ventricle.

Black arrows indicate prolapse of the cusps.
White arrows indicate resultant aortic regurgitation.
Moreover, the commissure between these two cusps is also involved (Fig. 2). Thus, the deformity of the aortic cusps in patients with infracristal VSD is more complicated than in those with supracristal VSD. This fact should always be taken into consideration in preparing for valvuloplasty.

In addition, all coexisting valvular lesions should be repaired simultaneously, such as aneurysm of the Sinus Valsalva, perforation of the cusps and adhesions between neighbouring cusps and/or between the involved cusp and the crest of the VSD, etc. Associated cardiac lesions should also be treated surgically in all cases, such as VSD, atrial septal defect, pulmonary stenosis, mitral regurgitation, coarctation of the aorta, etc.12,13

**Surgical procedures**

The patient is put on total heart-lung bypass after mid-line sternotomy. Moderate hypothermia is applied by blood cooling and a left ventricular vent is inserted from the cardiac apex.

First, during transient aortic clamping, a vertical right ventriculotomy is done, and the location of the VSD is confirmed. The aortic clamp is then released slightly to observe (through the ventriculotomy opening) the aortic cusps prolapsed into the VSD. This procedure provides accurate information about prolapsed cusps, aneurysm of Sinus Valsalva, degree of AR and adhesions between the prolapsed cusp and the crest of the VSD, etc. Moreover, the results of the valvuloplasty can be confirmed through the ventriculotomy and the VSD when the valvuloplasty has been completed. Therefore, right ventriculotomy rather than aortotomy, pulmonary arteriotomy or right atriotomy is preferred for closing the VSD. After completion of these observations, the aortic clamp is applied again and an oblique aortotomy is performed extending towards the non-coronary cusp. The myocardium is protected by injections of a cold potassium cardioplegic directly into both coronary ostia through coronary perfusion cannulae and by topical cooling. The myocardial temperature is kept between 10° and 20°C. Precise observation and analysis of the aortic cusps should be done from the aortic side before beginning valvuloplastic surgery. As mentioned before, Group II cases (infracristal VSD) should be treated somewhat differently from Group I cases (supraventricular VSD).

1. Cases with supraventricular VSD (Group I); (Figs. 3, 4)

In this group, elongation of the free edge and dilatation of the cusp are more dominant than in Group II. Discoaptation between the displaced right coronary cusp and the other two cusps is characteristic. The central portion of the free edge of the right coronary cusp is hypertrophied and thickened by the friction of the regurgitant blood stream, but peripheral portions of the free edge are usually stretched and weakened. Localized aneurysms of Sinus Valvsala (ruptured in some cases) may be found in older patients in Group I, but not in Group II. Perforation of the cusp and/or adhesions between the cusp and the crest of the VSD may be present, especially in older patients or those with a history of bacterial endocarditis. In only one case (a 38 year-old), was calcification of the cusp noted in the portion near the aortic wall.11) Fenestrations, probably of congenital origin, may be present.
GROUP I (SUPRACRISTAL VSD)

Before plication of the free edge, dissection of the adhesions between the cusp and the crest of the septal defect must be done with a knife if the adhesion restricts free movement of the cusp and coaptation of the free edges of all the cusps at the same level. Perforation of the cusp should be repaired with an autologous pericardial patch. Fenestrations of the cusps are also repaired in the same manner or by direct suture if necessary. Aneurysms of Sinus Valsalva (ruptured or not ruptured) should be repaired with a teflon felt pledget at the base of the aneurysm. When the aneurysm has a wide base, it is resected and repaired with an autologous pericardial patch.

In the first step of shortening the elongated free edge, the three cusps are apposed in the center of the valve by placing a 6-0 prolene suture in the Corpora Arantii of the three cusps (Fig. 3). With this technique, which was developed by Frater, it is possible to evaluate the degree

FIG. 3. Basic techniques of plication of the right coronary cusp in Group I (with supracristal VSD). (see text)

FIG. 4. Appositioning stitch between adjacent free edges (Group I). (see text)
of elongation of free edge of the involved cusp. By pulling the central thread, one can determine the appropriate place to plicate both ends of the elongated free edge (Fig. 3). A 5-0 suture is placed from the aortic side of the free edge at the determined site with a small piece of teflon felt. A horizontal U-stay suture is placed so as to fold over the redundant free edge of the cusp (Fig. 3). This may be sufficient as a plication, however, the threads are passed through the aortic wall exactly at the site of the commissure (Fig. 3). At this time the sutures should not be pulled through away from the commissure, but both threads should be pulled out exactly at the commissure, which is very important to reinforce the weakened commissure. The aim is to keep the plicated free edge at the same level as the adjacent normal cusps and also to give the commissure the strength to support the cusps. As the suture is pulled outside the aorta, the cusp is plicated, folded and fixed at the commissure. A teflon felt pledget is also used outside the aorta. Then plication of the same right coronary cusp is performed at the other end of the free edge near the commissure.

Accurate apposition of the free edges, especially at the Corpora Arantii, is mandatory to prevent subsequent valvular regurgitation. And this is obtained only by the plication of both commissures. These plications are also important to restore the suspending strength of the commissure. In cases of supracristal VSD, plication of the right coronary cusp alone is usually sufficient. If the commissure is still weak, as it is in some cases of severely damaged cusp, we use additional appositioning stitches between adjacent cusps near the commissure with a small teflon felt pledget, as shown in Fig. 4, to obtain accurate apposition of the free edges. However, this procedure can be omitted if one increases slightly the plication of the prolapsed cusp. When the plication is over, the central thread is removed.

Next, the VSD is closed. This is done by direct suture closure except in very large defects. U-stay sutures are placed by passing threads accurately at the pulmonary valvular annulus from inside the pulmonary artery. The same threads are passed through the lower margin of the defect, and teflon felt pledgets are placed on them. Before tying these sutures, we close the aorta with continuous sutures of 5-0 prolene. Looking at the septal defect, we release the aortic clamp and examine the resultant regurgitation. If significant regurgitation is observed, the aorta is reopened and the plication is repeated, slightly more extensively than the first time. When the valve repair is satisfactory, the threads around the septal defect are tied. Fairly large VSDs can be closed directly with safety if the threads are tied with the aorta clamped again. Very large VSDs are closed with a small patch. Direct closure of the supracristal VSD can be expected to allow suspension and reinforcement of the right coronary cusp from below.

The right ventriculotomy is closed. Meanwhile, the body temperature is returned to normal by bypass rewarming. The left ventricular vent is removed during partial perfusion. After coming off the bypass, bleeding is controlled and the median sternotomy is closed with drainage tubes left in place.

2. Cases with infracristal VSD (Group II); (Figs. 5, 6, 7)

In this group, the VSD is usually located just below the commissure of the right coronary and non-coronary cusps as viewed from the left ventricle. Therefore, the deformity frequently
Fig. 5. This figure shows prolapse of the right coronary and non-coronary cusps and the right commissure into the infracristal VSD viewed from the aortic lumen. The right commissure is stretched and weakened. Adhesions between the two cusps at different level are shown. RCC: right coronary cusp; NCC: non-coronary cusp.

includes both right coronary and non-coronary cusps and the right commissure. The free edges of the cusps near the commissure are especially elongated and weakened, and frequently adhere to each other in older patients. Infundibular muscular stenosis may be found with infracristal septal defect.

Operation and heart-lung bypass are started as described above. The location of the VSD, prolapsed aortic cusps, infundibular stenosis, etc. are confirmed through right ventriculotomy. After oblique aortotomy and myocardial protection, infundibular stenosis, if present, is released by resection of abnormal muscle. Adhesions between the aortic cusp and the crest of the VSD are dissected carefully, if necessary. Fenestration of the cusp is also closed.

GROUP II (INFRACRISTAL VSD)

Fig. 6. Basic techniques of plication and reinforcement in Group II (with infracristal VSD).
Before the start of plication of the free edges, dissection of the adhesion between the free edges of the right coronary and non-coronary cusps should be done with a knife using great care not to injure the cusps, because these cusps are very thin and adhere to each other at different levels (Fig. 5). This dissection is absolutely necessary to obtain accurate apposition of the three Corpora Arantii and reinforcement of the commissural support, because it is impossible to bring the adjacent free edges to the same level without removing the adhesions of the cusps.

Plication of the free edges is carried out in the manner already described. Plication is started at the right commissure to obtain reinforcement of the supportive strength of the commissure. The free edges of both sides of the involved commissure (the dissected portion in cases with commissural adhesion) are plicated exactly at the same level and all threads are pulled out from the aorta exactly at the site of the commissure (Fig. 6). In some cases, reinforcement of the involved commissure should be supplemented by additional appositioning stitches using teflon.
felt as shown in Fig. 7. Plication at the other end of the free edges should be done also to appose the three Corpora Arantii accurately. The other end of the free edge of the right coronary or non-coronary cusp, or both, is plicated according to the findings as the central thread is pulled up. Actually, elongation of the involved free edges is milder in Group II than in Group I, since the VSD is usually located just under the right commissure. In some cases with only one cusp (right coronary or non-coronary cusp) involved, plication is confined to both ends of the free edge of the involved cusp. Even in these cases, strict attention should be paid to the commissural support.

Infracristal VSDs are closed directly, as far as possible, with teflon felt pledgets as are supra-cristal VSDs. A very large VSD should be closed with a small patch. Before the sutures on the VSD are tied, the aortotomy is closed and regurgitation is evaluated with the aortic clamp released. Then the sutures on the VSD are tied with the aorta clamped. Heart lung bypass is discontinued after the right ventriculotomy has been closed and the left ventricular vent removed. The median sternotomy is closed with drainage tubes left in place.

The postoperative arterial pressure contour shows double pulse waves due to the diminished pulse pressure and exaggerated dicrotic notch (Fig. 8).

Discussion

A number of authors have described their valvuloplastic techniques in treating aortic valves prolapsed into a VSD.2,3,4,5,6,8,9 SPENCER et al.7 plicate the free edge of the prolapsed cusp by U-stay sutures using a teflon felt pledget in some cases and by continuous sutures in other cases. They prefer to plicate the free edge in the mobile portion and avoid fixation of the folded cusp to the aortic wall, considering it to be a cause of dehiscence of valve tissue. On the other hand, TRUSLER et al.17 fold the redundant free edge at the commissural portion and fix the fold to the aortic wall using teflon felt pledgets. In addition, they reinforce the commissure, using a thin Dacron patch just like the hood over the commissure.

The valvuloplastic techniques described in this paper may be closer to TRUSLER's than to SPENCER's. However, I would like to stress the importance of plicating the free edge at both ends near the commissures to obtain accurate apposition of the three Corpora Arantii. Commisural reinforcement is obtained by passing the threads used in U-stay sutures to plicate the free edge exactly at the commissure. Moreover, the patients with infracristal VSD (Group II) are distinguished from those with supracristal VSD (Group I) by commissural involvement. Therefore, commissural reinforcement is mandatory in eliminating aortic regurgitation in Group II. In my experience, closure of an infracristal VSD alone cannot cure even very wild AR. However, slight or even moderate AR can be diminished by closure of a supracristal VSD alone.

In treating VSD, direct suture closure is preferred, because it seems reasonable that suspension of the prolapsed and dilated cusps from below is more effective with direct than with patch closure. I do not believe that the aortic valve annulus is pulled down and AR in caused by direct suture of the VSD, as mentioned by SPENCER et al.7 Downwards displacement of the aortic valve annulus may be caused by dilatation of the Sinus Valsalva and the cusp, but it does
not cause regurgitation.

I have performed valvuloplastic surgery of aortic valves prolapsed into the VSDs since 1967. Only three reoperations were required. In the first two patients, who had supracristal VSD, plication was done in the aorta without fixation to the aortic wall and without the use of teflon felt. Severe aortic regurgitation recurred and reoperation was performed. Cusp ruptures at the site of plication were found in both patients, and plication with fixation to the aortic wall using teflon felt pledgets, as described in this paper, was done with satisfactory results. One patient with infracristal VSD was reoperated on after 3 years and 6 months. At the first operation, adhesions between the right coronary and non-coronary cusps were dissected, and plication of the non-coronary cusp was done only at the site near the commissure between the non-coronary and left coronary cusps. Fenestrations of the left coronary cusp were closed at the same time. This 26 year-old patient had a moderate AR immediately after operation, but tolerated it fairly well until he developed bacterial endocarditis with severe recurrent AR. Severe left heart failure and high fever which did not respond to digitalization and antibiotics and evidence of bacterial vegetations on UCG necessitated valve replacement with a Björk-Shiley prosthesis. In these three reoperated cases, recurrent AR was apparently caused by inappropriate valvuloplastic techniques. Therefore, AR due to prolapse of the aortic valve into a VSD can be treated safely by using only individually appropriate techniques of valvuloplastic surgery.

Summary

Recent techniques of valvuloplastic surgery of the aortic valve prolapsed into a VSD are described in detail.

The main principles are:

1) Lifting up the prolapsed cusps to the same level as the non-involved cusps by shortening the elongated free edge of the prolapsed cusps.

2) Strengthening commissural support by reinforcement of the commissures of the involved cusps.

Patients with infracristal VSD should be treated differently from patients with supracristal VSD, since involvement of right commissure and its adjacent cusps is the dominant feature in the former and involvement of the right coronary cusp in the latter.

In conclusion, this combined disease can be treated safely by using individually appropriate techniques of valvuloplastic surgery.

References


TECHNIQUES OF VALVULOPLASTIC SURGERY OF AR WITH VSD


和文抄録
心室中隔欠損に脱転せる大動脈弁に対する弁形成術手技

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著者は心室中隔欠損兼大動脈弁閉鎖不全症に対し1967年以後、弁形成術を主体とする外科的治療を行なって来た。その手術成績については既に数々発表しているが本論文においては、心室中隔欠損に脱転せる大動脈弁に対する最近の手術手技について詳細した。

著者の行なっている弁形成術の主たる原則は

(1) 脱転せる弁尖を非脱転弁尖と同一水平面に挙上させるため、脱転弁尖の伸展させる自由縁を短縮化し短縮せめる。

(2) 脱転弁尖の交通を補強することにより、交通部支持力を増強させる。

ことである。

しかしながら心室中隔欠損が室上模様下方に存在する場合には主として右交通およびその両側弁尖（右冠動脈弁および無冠動脈弁）が主たる病変部であるのに対し、心室中隔欠損が室上模様上方に存在する場合には主病変は右冠動脈弁に限局していることから、両者に対する弁形成術手技は異なるものとなる。

結論として、この複合病変における大動脈弁閉鎖不全は個々の症例に対し最も適切な手術手技を選択することにより弁形成術を行うことと安全に治療することが可能である。