SURGERY FOR CONGENITAL HEART DISEASE

ANATOMIC CORRECTION OF THE SYNDROME OF PROLAPSING RIGHT CORONARY AORTIC CUSP, DILATATION OF THE SINUS OF VALSALVA, AND VENTRICULAR SEPTAL DEFECT

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Background: Although the syndrome of ventricular septal defect and aortic regurgitation was described a long time ago, there is still no agreement about the anatomic and functional components of the syndrome and the optimal methods of management. **Objective:** Our objective was to describe a new simple technique of anatomic correction of all the components of the syndrome, based on redefining the salient anatomic and functional features of the syndrome. **Methods:** Anatomic correction of the syndrome is achieved through a transaortic approach with the placement of a series of pledget-supported mattress sutures using autogenous pericardium. The sutures are used to close the ventricular septal defect, plicate the aortic sinus, and correct the outward and downward displacement of the anulus of the aortic valve. The technique is designed to correct all the anatomic functional components including severe aortic regurgitation when present. **Results:** Between 1972 and 1996, 46 patients with this syndrome underwent surgical treatment. The current technique was used in most of the patients operated on before 1981 and in all patients since that date. There were no early or late deaths during a follow-up period varying from 3 months to 24 years (mean 8.4 years). Aortic regurgitation was abolished in 16 and improved in the remaining patients. The hemodynamic results have been maintained except in five patients operated on early in the series, in whom additional procedures on the cusps were performed. **Conclusions:** Anatomic correction of all the components of the syndrome of prolapsing right coronary cusp, dilatation of the sinus of Valsalva, and ventricular septal defect, can be achieved by a very simple technique. This technique can be applied in young children and prevents progression and secondary changes. Early correction in all patients with this syndrome is warranted. (J Thorac Cardiovasc Surg 1997;113:253-61)

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Although the syndrome of ventricular septal defect (VSD) and aortic regurgitation has been recognized as a clinical entity for a long time,¹⁻⁷ there is still no agreement about its management, particularly with regard to indications, timing, and technical details of operative treatment. We believe that the “ideal” operation should be safe, simple, reproducible, and durable and should deal with all the anatomic components of the syndrome, preferably at a young age. Achieving these goals depends on accurate characterization of the anatomic components and pathophysiology of the syndrome. The purpose of this article is to define the components of the syndrome and describe a simple technique of complete anatomic correction of this anomaly,

Fig. 1. A section in the aortic root and RVOT illustrating the dilatation of the aortic sinus and the discontinuity between the aortic media (*black line*) and anulus of aortic valve (*dotted area* at the base of the cusp), the lack of support to the anulus and the sinus of Valsalva, the downward and outward displacement of the anulus with the right ventricle, and the lack of coaptation between the aortic cusps.

which we have developed and used over the past 24 years.

Methods and patients

Anatomic features. Although prolapse of the right or noncoronary aortic cusp can occur through a VSD, which has been reported to vary in position, in our experience the syndrome involves the right coronary cusp in the vast majority of cases and consists of four anatomic components:

1. A moderately large VSD situated just below the right coronary cusp (Fig. 1). When viewed through the left ventricle, the defect is oval and extends from just below the commissure between the right and noncoronary cusps, along the muscular septum to approximately the midpoint of the right coronary cusp (Fig. 2, *A*). The membranous septum is usually absent. The lower border of the defect is formed by the crest of the ventricular septum, which is usually muscular but could be covered by fibroelastic tissue. The conducting tissue is located on the left ventricular aspect 4 to 5 mm below the edge of the defect (Fig. 2, *A* and *B*). The upper margin of the defect is made by the right coronary cusp and part of the dilated sinus of Valsalva with no intervening muscular tissue (Fig. 1). When viewed from the right ventricle, the VSD is in the perimembranous/outlet position or entirely in the outlet septum. The position of the VSD in relation to the aortic

valve is fairly constant (below the right coronary sinus) regardless of its relation to the rest of the interventricular septum (perimembranous or outlet). Only very occasionally is the VSD related to the noncoronary sinus (2/46 patients in our series). The salient feature of the syndrome is the lack of continuity between the ventricular septum on the one hand and the aortic sinus on the other (Fig. 1). The “anulus” is defined as the site of attachment of the aortic cusps to the aortic root (Fig. 3). Even though the word “anulus” suggests a circular ring, the aortic anulus is a crown-shaped structure and in cross section is represented by a definite triangular fibrous structure (Fig. 3). In the normal aortic root, the anulus marks the junction of the aortic sinus, cusp, and ventricular septum (Fig. 3). In contrast, in this syndrome the anulus loses its connection to the septum and represents the junction between the thinned out aortic sinus and the cusp (Fig. 1) and is clearly visible when viewed from above through the aortic sinus or from below through the ventricular aspect. In addition, the anulus is usually clearly visible echocardiographically (Fig. 4) and provides important diagnostic features.

2. Dilatation of the right coronary sinus of Valsalva with loss of continuity between the media and anulus of the aortic valve. This results in sagging of the aortic anulus into the VSD with varying degrees of aneurysmal bulge or rupture of the sinus into the right ventricular outflow tract (RVOT) (Fig. 1).

3. Aortic valve abnormalities, which include downward and outward displacement of the anulus in the region of the right coronary cusp into the right ventricle (Fig. 1) with progressive sagging and enlargement of the body of the cusp. The result is failure of the coapting surface of the cusp to meet the other two cusps and progressive elongation of the free margin in the right coronary cusp. In some patients a slight degree of rotation of the whole of the aortic root in a counterclockwise direction is present. Occasionally the aortic valve is bicuspid. Although the aortic valve abnormalities are usually localized to the right coronary cusp and sinus in patients with long-standing aortic regurgitation, secondary changes in the form of thickening and retraction of the free borders of all three cusps can be present.

4. Varying degrees of RVOT obstruction, produced by bulging of the dilated right coronary sinus of Valsalva into the RVOT (Figs. 1 and 5), with or without dynamic obstruction caused by hypertrophied muscle bands and occasionally endocardial fibrous thickening. The obstruction is usually mild.

Echocardiographic features. Although the diagnosis is usually made by the presence of an immediate diastolic murmur in a patient known to have a VSD, recent refinement in echocardiographic techniques⁸ allows accurate recognition and quantification of the various components of the syndrome, even before the development of an audible diastolic murmur. The characteristic dilatation of the right coronary sinus and sagging of the anulus, sinus, and cusp into the RVOT can be recognized in the parasternal long-axis, short-axis and four-chamber views (Fig. 6, *A* to *C*). In addition, the degree of aortic regurgitation can be assessed by color flow Doppler analysis and the gradient across the RVOT estimated by Doppler interrogation.

Pathophysiology. A rational approach to the management of these patients depends, to a large extent, on

Fig. 2. The location of the VSD and the sagging aortic cusp as viewed from the left side through a transaortic approach. The position of the conducting tissue and the pledget-supported sutures used for the repair is also shown both before (**A**) and after (**B**) the sutures are tied. Note the elevation of the ventricular crest and base of the cusp produced by the sutures that plicate the sinus of Valsalva.

thorough understanding of the factors that are responsible for the progressive nature of the functional and anatomic abnormalities in this syndrome. As mentioned earlier, the basic abnormality in this syndrome is the “discontinuity” of the aortic media from the aortic anulus and ventricular septum in the region of the right coronary sinus (see Fig. 3). This results in inherent weakness of this area, with a tendency for progressive dilatation of the sinus and downward and outward displacement of the aortic valve anulus in this region. These progressive changes are brought about by hemodynamic factors that operate throughout the whole of the cardiac cycle. During early systole the fast jet produced by the left-to-right shunt, through the slitlike VSD, results in a Venturi effect, tending to displace the unsupported anulus outward and downward into the right ventricle (Fig. 7, *A*). Later in systole the aortic cusp prolapses into the VSD and is acted on by direct pressure from the cavity of the left ventricle, which tends to displace both the cusp and the anulus further into the right ventricle (Fig. 7, *B*). During diastole the high pressure in the aortic root distends the dilated sinus with further displacement of the aortic anulus toward the right ventricular cavity (Fig. 7, *C*). As the sinus dilates, the distending force becomes greater, owing to the increase in wall tension. The increasing aortic regurgitation and the existing left-to-right shunt cause left ventricular volume overload, which could cause potentially irreversible changes in the left ventricular wall structure and function.

Similarly, structural changes in the aortic valve cusps can develop, which could reduce the chances of effective surgical repair.

Surgical technique. All the anatomic components can be corrected by a very simple transaortic repair. The operation is performed with the use of cardiopulmonary bypass, moderate hypothermia, and crystalloid cardioplegia. The ascending aorta is opened through an oblique incision, starting in the midline and curving down toward

the center of the noncoronary sinus, similar to the incision used for homograft replacement of the aortic valve. The anatomic features are confirmed by inspecting the aortic sinuses and elevating the right coronary cusp (see Fig. 2). The extent of dilatation of the right coronary sinus and the exact definition of the thin area of the sinus resulting from discontinuity between the aortic valve anulus and the media of the aorta are accurately defined. A series of interrupted Ethibond 4-0 mattress sutures (Ethicon, Inc., Somerville, N.J.) supported by small pericardial pledgets are then inserted through the crest of the ventricular septum, slightly toward the right ventricular aspect, to avoid the conducting tissue, which is on the left ventricular aspect 4 to 5 mm below the edge of the crest (see Fig. 2). The sutures are then passed through the anulus of the aortic cusp into the sinus of Valsalva.

The same sutures are used to plicate the thin portion of the aortic sinus and thus have the effect of reattaching the ventricular septum and anulus of the valve to the media of the aorta. This results in closing the VSD, elevating the right coronary anulus and cusp, and reducing the size of the right coronary sinus and RVOT (Fig. 8, *A* and *B*). The sutures are tied in the aortic sinus, avoiding encroaching on the right coronary orifice (Fig. 8). Although, ideally, the knots should be tied outside the aorta, this is not possible in this region because of the close proximity of the RVOT. The knots, however, are situated in the sinus, which is usually washed by vortices during diastole. The presence of any residual defects between the mattress sutures is checked (Fig. 2) and additional sutures are inserted, if necessary. Direct suture of the VSD without exerting undue tension is feasible regardless of the size of the defect. This is rendered possible by the presence of “redundant” tissues (septum and aortic sinus) in the vertical plane (Fig. 4). The effect of the repair is to plicate the redundant tissues toward the media of the aortic sinus (Figs. 2 and 4), thus elevating the anulus and cusp and

Fig. 3. Section in a normal human aortic root showing the continuity between the aortic media and anulus.

displacing them centrally toward the lumen of the aorta. This has the effect of increasing cusp coaptation and reducing or abolishing aortic regurgitation.

The valve is then inspected and tested for competence by stretching the body of the cusp with a blunt instrument to evaluate the presence, or absence, of prolapse resulting from elongation of the free border and failure of coaptation between the right coronary cusp and the rest of the valve apparatus. Usually the prolapse is completely corrected by the elevation of the base of the cusp, particularly in patients operated on at the appropriate time (early in the course of the disease before the development of the gross secondary changes in the valve cusps). If the cusp continues to prolapse after insertion of the mattress sutures, the free border is shortened by plicating it near one or both commissures as described by Spencer,⁹ Trusler,¹⁰ and their associates. Alter-

Fig. 4. Echocardiogram in the parasternal long-axis view after anatomic correction of the syndrome showing the plicated sinus, with closure of the VSD, elevation of the cusp, and restoration of competence of the aortic valve. RV, Right ventricle; LV, left ventricle; LA, left atrium; MV, mitral valve.

Fig. 5. RVOT obstruction produced by bulging of the dilated right coronary sinus of Valsalva and a degree of hypertrophy of the RVOT myocardium.

natively, if the central part of the border is thick enough to hold sutures, the plication is performed near the corpus aurantii. The efficacy of the repair can be readily appreciated echocardiographically in the immediate postoperative period (Fig. 4).

Fig. 6. Two-dimensional echocardiograms illustrating all the features of the syndrome. **A**, Parasternal long-axis view during ventricular systole showing dilatation of the right coronary sinus of Valsalva and prolapse of the aortic sinus, anulus, and cusp with the right ventricle. **B**, Parasternal long-axis view during diastole showing further prolapse of cusp/sinus and anulus with failure of coaptation of cusps. **C**, Apical four-chamber view showing the relation of the prolapsed sinus/cusp to the crest of the ventricular septum. *LV*, Left ventricle; *RV*, right ventricle; *LA*, left atrium; *MV*, mitral valve; *LVOT*, left ventricular outflow tract; *LC*, left coronary artery.

Clinical experience. Between 1972 and 1996, 46 patients were operated on by one of us for this condition. During the early period of this experience this technique was used for an increasing proportion of the patients. Since 1981 all patients with this anomaly underwent repair by means of the current technique. In all, 38 patients had the transaortic repair described herein. There were 23 male and 15 female patients whose ages ranged between 18 months and 57 years (mean 10.9 years). All patients had notable symptoms except for four children who had minimal or no symptoms. Two patients had a history of bacterial endocarditis. The degree of preoperative aortic regurgitation was judged to be trivial in three, moderate in 18, and severe in six patients. Echocardiographic features of left ventricular diastolic overload were present in most patients. Pulmonary artery pressure was within normal limits in all patients. Early in our experience the anatomic and functional changes in the aortic root and valve were defined by angiography (Fig. 9); more recently, echocardiography alone was used for that purpose (Fig. 6).

In all the patients in this series the VSD was located underneath the right coronary cusp, except in one in whom part of the VSD was under the noncoronary sinus. At the time of the operation, anatomic correction by the transaortic mattress sutures, described earlier, was sufficient to restore competence of the aortic valve in 25 of 38 (65%) patients; in the remaining patients the free border of the right coronary cusp was shortened by plicating the free border or excision of triangular segments of the body of the cusp.

The use of these additional procedures was common early in our experience, especially in older patients with long-standing (neglected) disease.

There have been no early or late deaths during a period of follow-up varying from 3 months to 24 years (mean 8.4 years). In the immediate postoperative period aortic regurgitation (as assessed by clinical and echocardiographic criteria) was abolished in 16 and improved in the remaining patients. The initial result was maintained in all patients except for five patients who underwent successful

Fig. 7. Hemodynamic forces that influence progression of the syndrome during early systole (A), late systole (B), and diastole (C).

valve replacement (8 to 11 years after the operation); a homograft valve was used in three and a pulmonary autograft in two. These patients were operated on early in the experience and had additional procedures involving the cusp at the first operation (excision of triangular pieces in three and plication of the free border in two). At the latest follow-up all patients are free of symptoms with evidence of good correction of the hemodynamic lesion. Echocardiography (see Fig. 4) confirmed correction of both the anatomic and functional abnormalities.

Discussion

This study has served to define further the anatomic and functional components of the syndrome of VSD and aortic regurgitation and to describe a very simple technique for correction of all the components of the syndrome.

The association between aortic regurgitation and VSD in a specific subset of patients was recognized by Breccia¹ in 1906. Since then many excellent pathologic and clinical studies^{2-8, 11, 12} have served to unravel most of the features of the syndrome. Yet the precise anatomy and cause, or causes, of the development and progression of the aortic regurgitation are still the subject of controversy; as a consequence, there is no agreement about an optimal plan for the treatment of these patients.

We, here, propose that the basic abnormality in this syndrome is the lack of continuity between the aortic media, anulus, and ventricular septum, which, under normal conditions, provides good support to the aortic valve apparatus (see Fig. 3). This abnor-

malty is similar to that described in patients with aneurysms of the sinus of Valsalva¹³⁻¹⁶ in whom the media of the aortic sinus fails to reach the aortic anulus. In the syndrome of VSD and prolapsing aortic sinus and cusp, additional failure of the anulus to attach to the ventricular septum is present. These basic abnormalities render the aortic sinus and anulus in this region to be weakened and unsupported (Fig. 1). With the passage of time several forces, detailed earlier in this article under the heading of pathophysiology, aggravate the anatomic and functional abnormalities principally by progressively displacing the aortic anulus downward and outward. Initially the hemodynamic lesions are not severe, because the aortic regurgitation is absent or mild and the left-to-right shunt is small owing to partial obstruction of the VSD by the prolapsing sinus and cusp and the presence of a degree of RVOT obstruction. Turbulence around the aortic valve predisposes to infective endocarditis,^{11, 12} which was encountered after the operation in two of our patients.

Although all the structural and functional abnormalities in this syndrome are essentially progressive, the rate of increase in aortic regurgitation varies considerably in different patients. This, and the perceived lack of a safe simple method of correction, has prompted many clinicians to treat these patients conservatively for prolonged periods of time. This policy can result in severe secondary changes in the valve, which could make it unsuitable for repair, as well as left ventricular dysfunction,

Fig. 8, A and B. Section in the aortic root illustrating the plicating sutures used to close the VSD, elevate the anulus, reduce the size of the sinus, and restore competence of the aortic valve.

which can be irreversible in some patients. In this article we describe a simple technique for anatomic correction of all the components of the anomaly which, when applied relatively early, can correct both the anatomic and functional abnormalities including severe aortic regurgitation if present. The technique consists in placing the sutures through the ventricular crest, aortic anulus, and sinus of Valsalva and therefore restoring the continuity between these structures and correcting the downward and outward displacement of the aortic anulus (Fig. 8), which we believe to be the basic abnormality in this disease. The technique is simple, reproducible, and safe and can be applied in young children if necessary. Previous techniques involved transatrial or transventricular repair of the VSD, usually combined with repair or replacement of the aortic valve.

Garamella and his colleagues¹⁷ reported using a transaortic approach for repair of VSD and aortic regurgitation, which were done as separate procedures, using a patch. In our experience the transaortic approach gives excellent exposure of the VSD, which allows for secure closure as part of the

Fig. 9. Aortogram showing prolapse of both the right coronary sinus and cusp into the right ventricle with fairly severe aortic regurgitation.

anatomic repair, without encroaching on the conducting tissue. This approach has the additional advantages of avoiding myocardial injury resulting from a ventriculotomy or atriotomy. The aortic valve is usually rendered competent by elevating the anulus and plicating the sinuses by the same stitches. Patients with advanced secondary changes affecting the aortic cusp require additional operative procedures on the thin cusp tissue with shortening of the free margin or excision of triangular pieces of cusp tissue (or both). They are thus at risk for recurrence of regurgitation caused by tearing of cusp tissue. In contrast, the repair at the base of the cusp and sinus, described in this article, involves thicker tissue, which eliminates the risk of recurrence caused by tearing. We therefore believe that repair should be performed before the onset of changes in the cusp.

It is concluded that the syndrome of VSD, prolapsing right coronary cusp, dilated aortic sinus, and RVOT constitutes a clinicopathologic entity that can be diagnosed confidently by echocardiography. The syndrome can be anatomically corrected by means of a simple technique that gives good early and long-term results. In view of this, the progressive nature of the disease, and the risk of endocarditis, we believe that early correction in all patients with this syndrome is warranted.

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Discussion

Dr. Yasunaru Kawashima (Osaka, Japan). I congratulate Professor Yacoub for his excellent result with this unique technique of repairing VSD associated with aortic regurgitation. I am also very much interested in this anomaly and reported in *Circulation* an anatomic classification and method of operation for VSD associated with aortic regurgitation in 1973. In this article I also mentioned that direct closure of VSD without patching is a more effective way to push the herniated right coronary cusp back into the left ventricle and to reduce the degree of aortic regurgitation without a direct procedure on aortic valve. I have done this from the right ventricular side through the pulmonic valve, although I did not plicate the Valsalva sinus, and Professor Yacoub did it from the left ventricular side through the aortic valve. I presume Professor Yacoub's method is more effective in reducing aortic regurgitation because the plication itself functions as a kind of valvuloplasty. However, as I have reported previously, there is a group of patients in whom there is no conal muscular rim between the pulmonic valve and VSD. In these cases, the aortic and pulmonic valves are contiguous and there is no firm muscular tissue to which the lower edge of VSD is to be approximated without a patch, even with plicated Valsalva sinus or aortic cusp between them.

In this group of patients, I think it is mandatory to close the VSD with a patch, inserting the sutures from inside the pulmonic valve cusp; and in most of the patients, valvuloplasty is necessary because the associated aortic regurgitation is usually severe. In our series of 95 patients operated on since 1969, 66 fit the category Professor

Yacoub has mentioned. In most of them, the VSD was closed with a patch.

Professor Yacoub, there may be a racial difference between white and Asian people with regard to the anatomy of the conal septum in this group of patients as in the frequency of outlet VSD. While using your new technique in 46 patients, how many patients did you encounter in whom there was no conal muscular rim between the semilunar valves and in whom both semilunar valves were contiguous? What kind of procedure have you used for those patients without a muscular rim? Do you think you can use your new technique even for such patients?

Mr. Yacoub. Professor Kawashima, thank you for your comments. We believe that by repairing the VSD through the aortic valve one can see this anatomy predictably in the majority (in our series it was all the patients), regardless of whether the VSD is doubly committed or a perimembranous outlet defect. However, I do recognize the frequency of outlet septal defects in Asian patients; perhaps that variation is more prevalent than in our experience. I think, however, that the plication could be performed as viewed from the aortic valve, particularly if one does not need muscular tissue, conus tissue, for the plicating sutures. These are tied to the media of the aorta on the left side below the right coronary orifice, which usually is quite satisfactory for suture placement.

Dr. Erle H. Austin (*Louisville, Ky.*). Dr. Yacoub, I am impressed with your technique. If it is as simple as you describe, many of us will want to try it.

I am concerned that tension might result in the sutures pulling through the muscle at the crest of the VSD. Did you have any recurrent VSDs in your group? I am also concerned about the conduction tissue. You noted that the conduction tissue should be avoidable, but did any of your patients have heart block?

Mr. Yacoub. Thank you, Dr. Austin. Like you, we were worried about undue tension resulting from direct suture placement. However, there is a redundancy of tissue in the ventricular aspect. The ventricular septum itself is quite long. In addition, the sinus is aneurysmal with a lot of excess tissue. We have had pinpoint shunts. And we explored the gaps between the mattress sutures and not

infrequently added additional sutures. These extra sutures were added, not to alleviate undue tension, but just to close gaps between the sutures themselves.

We have had no problems with conduction abnormalities. Indeed, what is so impressive about this technique is that the electrocardiogram afterward is entirely normal. The PR interval is normal and, more impressive, there is no right bundle branch block. The QRS interval is normal with no conduction abnormalities.

Dr. Richard A. Jonas (*Boston, Mass.*). Professor Yacoub, I would like to add my congratulations. I would also like to explore with you the indications for this technique. Would you agree with routine closure of a subpulmonary VSD as prophylaxis to prevent aortic valve prolapse?

Mr. Yacoub. Yes, indeed, Richard. Follow-up echocardiograms of these patients show the very characteristic dilatation of the sinus that occurs before the prolapse.

Dr. Jonas. May I assume that if you are operating prophylactically, you would not use this technique for a subpulmonary VSD before prolapse has occurred?

Mr. Yacoub. Actually, the subpulmonary VSD is not the problem. It is the subaortic VSD with a prolapsing sinus and the aortic root that worries me. I evaluate the echocardiogram to see whether there is any degree of aortic regurgitation. Surprisingly, this technique sometimes abolishes even moderate to severe aortic regurgitation. However, I am eager to correct regurgitation before there are secondary changes in the cusp. The appearance of the dilated right coronary sinus is the indication for operation.

Dr. Jonas. That is really my question. With a membranous VSD we are often presented with a child who has some echocardiographic evidence of very early prolapse and yet does not have any regurgitation. Under those circumstances, would you do this operation? At what point do you need to add plication of the free edge of the cusp, which I note you did have to do in a number of the patients?

Mr. Yacoub. Even in the absence of regurgitation the aortic sinus and anulus are displaced, and this can be corrected by exactly the same technique of plication without the need for touching the cusps.