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Pathophysiologic Implications of the Helical Ventricular Myocardial Band: Considerations for Right Ventricular Restoration

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This chapter describes the structure/function relationships of the right ventricle (RV), and shows how the geometry of the helical ventricular myocardial band model defines spatial geometry of the free wall and septum that underlie dynamic action. Myocardial fiber orientation is the keynote to performance in health and disease. The transverse geometry of the RV free wall allows constriction (bellows-type motion), whereas oblique septal fiber orientation and midline septal position is essential for ventricular twisting, the vital mechanism for RV ejection against increased pulmonary vascular resistance. Therefore, the septum is considered "the lion or motor of RV performance." Distortion of such normal structure/function relationships underlies the pathophysiologic mechanisms of RV failure. Operative methods that restore normal myocardial fiber orientation are described to outline evolving surgical techniques for the surgical treatment of RV failure.
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A clear understanding of structure/function relationships for the right ventricle (RV) or left ventricle (LV) can emerge only after understanding how spatial geometry of underlying structure influences myocardial function. Advancements toward improved surgical strategies can evolve following establishment of this framework. Conversely, without this framework, clinical decisions are made from patterns of functional behavior rather than from a stepwise breakdown of the architectural underpinning that produces a structural reason for observed LV or RV activity.

This pursuit leads to the development of a "ventricular approach" to diseases that secondarily impair ventricular performance. Currently, LV restoration is the predominant clinical focus of surgical ventricular reconstruction, and LV rebuilding is based on restoring the spherically dilated LV chamber (secondary to ischemic and nonischemic diseases) to its natural elliptical form. When compared with LV restoration, surgical reconstruction and restoration of the failing RV has received less consideration and conventional treatment options for RV failure have been less favorable.[1] The infrastructure for these treatment limitations relates to restrictions in the fundamental knowledge about the geometric determinants of RV performance and how impairment of underlying structure produces dysfunction. As a consequence, the physiologic remedies of RV failure are directed toward alleviating symptoms of failure, rather than correcting the form-related components that underlie dysfunction.

The central component of this form/function relationship is the ventricular septum, which separates the LV and RV chambers. The current enigma of the septal role in defining cardiac function was initially described in 1865,[2] when the anatomist Hegar indicated that "cardiac anatomy and function will be uncertain until the structure and function of the crossed angles of fibers in the septum are defined." This report will underscore the septum's central role in determining RV function by presenting new knowledge that describes how recognition of fiber orientation in the septal structure/function relationship initiates the evolution of new treatment pathways.

Absence of understanding the vital role of the septum explains the current limitation of pulmonary valve implantation alone to correct functional abnormalities when treating pa-

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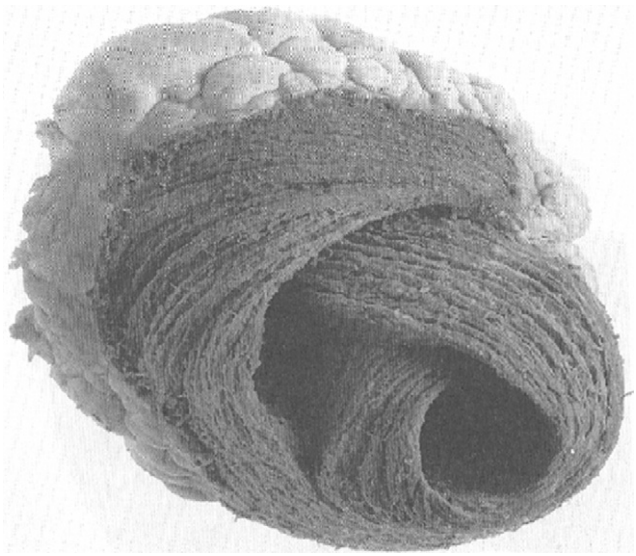

Figure 1 Fiber orientation relationship of the septum, composed of oblique fibers that arise from the descending and ascending segments of the apical loop, surrounded by the transverse muscle orientation of the basal loop that composes the free RV wall. Note the conical arrangement of the septum muscle and the basal loop wrap, forming the RV cavity.

tients with RV failure from chronic pulmonary insufficiency following repair of tetralogy of Fallot.[1] Conversely, recognition of a ventricular component introduces the potential evolution of a "valve -ventricle approach" to address chronic right heart failure. This broader approach requires deeper understanding of the impact of anatomic fiber orientation of the RV architecture (free wall and septum) on resultant performance. Such information allows the generation of a novel RV surgical restoration strategy, in a manner that will parallel guidelines currently used to reshape and resize the LV.

Anatomic and Physiologic Considerations: Structure/Function Relationships of the RV and Septum

Background

The anatomic components of the RV are the free wall and septum, and the functional role of these components requires knowledge of how fiber orientation influences performance. The septal role has been uncertain because it was formerly considered a LV structure. The helical ventricular myocardial band (HVMB) model of Torrent Guasp[3] advanced our geometric knowledge of ventricular structure because its spatial configuration shows that the heart is composed of two interconnected loops called the basal and apical components (shown in Fig. 1). The basal loop is an external structure with predominantly transverse fibers that wrap around the RVs and LVs, to resemble a turbin surrounding an inner apical loop. A central myocardial fold occurs where the right basal segment becomes the left basal segment and thereby changes the fiber orientation to an oblique direction; this creates a helical arrangement of the apical loop, comprised of a descending and ascending segment that forms the apical vortex.

The RV free wall predominantly contains the transverse basal loop that provides an external cover for the septum, which is comprised of oblique fibers of the apical loop. Some of these oblique apical loop fibers also contribute to the RV outflow tract (RVOT). A critical aspect of this spatial geometry is that the septum does not have a predominant RV or LV side, but rather the septum is the central structure between left- and right-sided cavities that is comprised of the same oblique fiber elements that form the free LV wall.[4]

Septum and Free Wall

The ventricular septum separates the LV and RV chambers, and is a crucial component of this form/function relationship because it constitutes approximately 35% of the entire ventricular myocardial mass in the adult. In 1991, Dell' Italia[5] observed that the ventricular septum is a central theme to both ventricles because it binds them together with spiral muscle bundles that encircle them in a complex interlacing fashion to form a highly interdependent functional unit that exists despite their markedly different muscle mass and chamber geometry.

The RV free wall and septum are the two architectural components of the RV, and the underlying theme of any treatment must address the concept that fiber orientation determines ejection. A vital separation between basic and clinical science exists during evaluation of the functional role of fiber orientation. Evaluation of fiber orientation per se is readily done in a Petrie dish, where only 15% muscle fiber shortening follows sarcomere stimulation. However, the intact heart contains connected fibers with varying angular orientation that depends on structure configuration. Angulation becomes an important consideration because fiber orientation defines resultant performance. The relationship of fiber orientation and function was implied in 1952 by Rushmer et al,[6] who reported that ejection fraction is approximately 30% after only transverse or circumferential muscle contraction. Subsequent studies by Ingels,[7] Sallin,[8] and Arts et al[9] showed that ejection fraction increases to 60% if there is oblique fiber orientation. These transverse and oblique angulations have direct application to RV construction because the RV basal loop wrap has predominant horizontal fibers and the septum contains reciprocally crossed oblique fibers.

Evidence for septal oblique crisscross fiber configuration is summarized by: 1) the collagen weave network of reciprocal septal spirals exposed by Lunkenheimer's air inflation studies[10]; 2) Greenbaum and Anderson's cross sections showing crisscross septal fibers[11]; and 3) the anatomic dissections of the unwrapped heart by Torrent Guasp,[3] where a right angle cross-striation of oblique fibers go to and from the conical apical tip. This oblique fiber orientation affords a greater ejection fraction for both the RVs and LVs. Furthermore, this configuration augments RV function by enhancing the septal

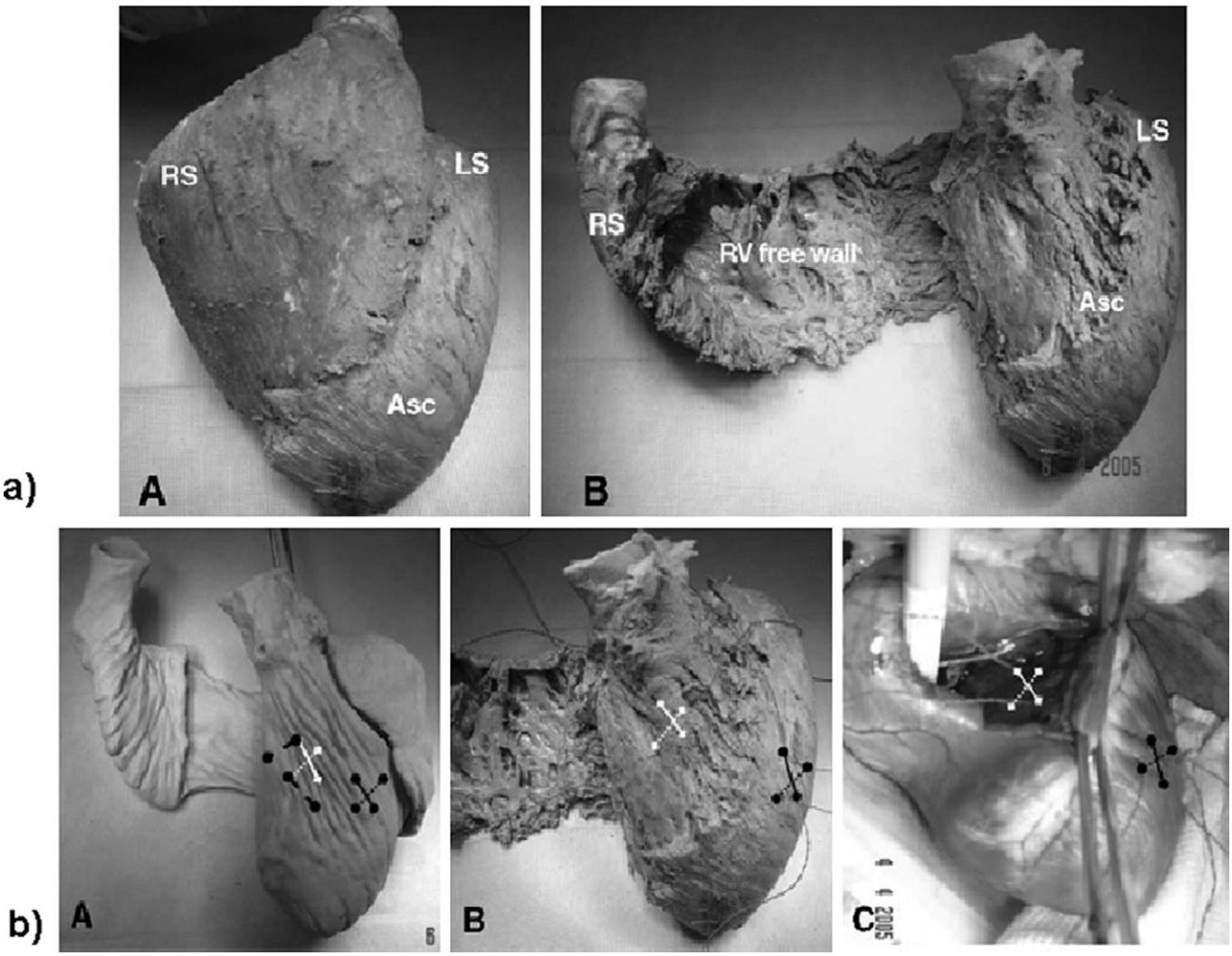

Figure 2 (a) Anatomic preparations showing the orientation of the ventricular myocardial band of the (A) intact heart and (B) after exposing the septum by unfolding of the RV free wall. Note the similar configuration of the septum and LV free wall composed of the ascending segment of the apical loop. RS, right segment of basal loop; LS, left segment of basal loop; Asc, ascending segment of apical loop. (b) Helical heart model (A), anatomic specimen (B), and experimental study (C) showing sonomicrometer crystal positioning in the descending and ascending segments of the LV free wall. Crystal orientation was either in direction of LV free wall showing maximal segmental shortening of descending and ascending segments, or placed perpendicular to maximal segmental shortening position (as shown in image b/A).

twisting motion; an action that functions in concert with the transverse forces of the free wall.

This functional hypothesis is based on the concept that the septum has reciprocally oblique fibers that should display the same functional changes as the LV free wall because both structures are formed by the descending and ascending segments of the HVMB. Functional confirmation of these anatomic studies of oblique fiber orientation of the HVMB was recently made by sonomicrometer crystal measurements (Fig. 2).[4] The predominant shortening direction of the oblique septal fibers was evaluated by crystal placement in the correct angular orientation because reduced shortening was observed if a more transverse (versus oblique) orientation was selected. The extent of septal shortening could be either accentuated or reduced by positive (dopamine, Dopamine HCL; American Regent, Inc, Shirley, NY) or negative (Esmolol HCL, Brevibloc; Baxter Health Corp, New Providence, NJ) inotropic intervention, thereby directly simulating the responsiveness of the reciprocally oblique LV free wall fibers shown previously.[12]

High-resolution echocardiography has provided further visualization of a longitudinal mid-septal line that separates layers of fiber orientation within the septum, and simultaneously documented a separate directional transverse strain pattern generated by the oblique ascending and descending segments that determine septal architecture. Identification of the underlying septum in the explanted heart is observed by detaching the overlying free wall from the septal attachment, as shown in Fig. 2. This dissection reveals that the septum is included in a continuation of the oblique layers that form the LV free wall in the helical heart configuration. Consequently, the underlying structure is the same for the LV and septum,

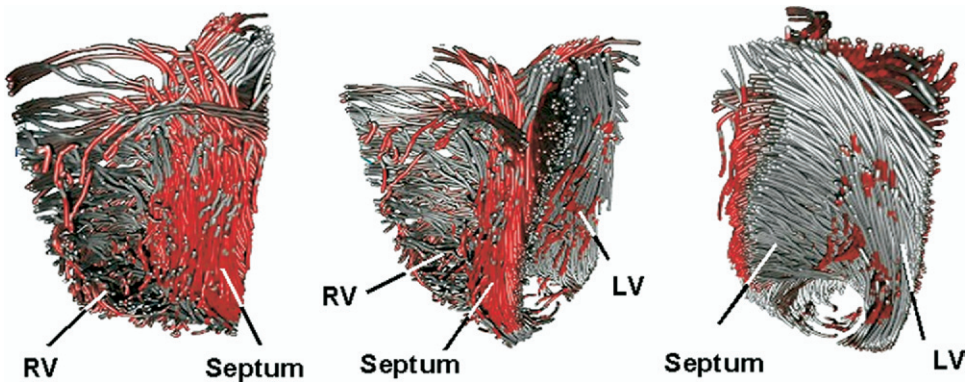

Figure 3 Fiber tracking performed in the LVs and RVs of the human heart using regularized tensor diffusion MRI visualization techniques. The geometry of the fibers of both RVs and LVs and the septum is shown and the continuity of the LV fibers with the septum can be seen. The transverse fiber orientation of the RV free wall is also evident. The left figure shows the RV and septum, the center figure shows the septum in a transverse view, and the right image shows the left side of septum. Note the fiber angulation mirrors the muscle fiber orientation in Figure 1. (Reprinted with permission from Lawrence National Berkeley Laboratory Publications.[13])

but this formation pattern escapes detection because the septum is hidden from external view by the externally overlapped RV free wall attached on its anterior and inferior surface. Consequently, the septum functions as a single unit, even though its architecture develops from two predominant oblique muscle layers. The concept of a functional left- and right-sided septum is invalidated by this structural configuration.

Recent diffusion tensor magnetic resonance fiber orientation studies further document the separation between oblique septal fibers and differently oriented fibers of the RV free wall (Fig. 3).[13] The functional interaction between the RV free wall, which has a predominant transverse basal loop component, and the oblique septum is defined by prior anatomic studies of free wall exclusion that are accomplished without direct septal injury. RV performance is not significantly impaired following either cauterization of the entire free wall[14] or replacing the free wall with patch material,[15] as long as the septum remains intact. Under these conditions the transverse shortening of the free wall is abolished, but twisting is still possible by the oblique fiber orientation of the intact septal architecture. Conversely, RV failure develops if the septum is cauterized, made ischemic by embolization, or confronted with pulmonary hypertension.[16] The elevation of RV outflow resistance creates a similar physiologic condition that mirrors increased systemic pressure for LV emptying, an action that requires a septal twisting motion to overcome afterload resistance.

Fiber orientation is a critical component of the extent of ventricular deformation that generates the strain and thickening needed for blood ejection during systole, and the rapid filling by suction in diastole. Magnetic resonance imaging (MRI)-tagged images confirm the mechanism of ejection is through twisting of fibers, followed by rapid untwisting to fill.[17] These actions reflect the "wringing a cloth" functional concept defined in 1681 by Borelli[1] for ejection, and the helical geometric design introduces a reciprocal action to twist in the other direction for rapid filling. In contrast, there are minimum external wall changes during systole and diastole because these motions are caused by RV free wall contraction that causes transverse narrowing in systole and widening during diastole.

RV MRI studies confirm the primary events involving septal muscle are chamber shortening and lengthening caused by twisting and thickening that produce ejection, and untwisting and lengthening for suction filling.[17] Recent functional reports by Klima et al[18] have confirmed these predominant septal events and confirm that the clinical significance of septal dysfunction is linked to functional performance. RV efficiency becomes impaired when the twisting capacity of the septum is altered by lesions that result in septal stretch; a pathologic event that changes the oblique fiber orientation toward a more transverse plane and limits the septal fiber vector shortening force.

The physiologic and clinical importance of these observations relates to how the efficiency of ejection is impacted by the vector forces needed for ejection into the right and left sides, because outflow efficiency relates to differences of resistance in systemic and pulmonary vascular beds. The oblique orientation of muscle fibers of the LV free wall and septum allows the wringing or twisting required to eject blood into high systemic vascular resistance. In contrast, pulmonary vascular resistance (PVR) is one sixth the amount of systemic resistance, so that the compressive force of transverse constriction or bellows-like activity from the RV free wall is sufficient to support RV ejection under normal physiologic conditions. The effectiveness of this compressive capacity following only free wall contraction is evident clinically, because postoperative RV failure is often absent, despite the frequent occurrence of early septal dysfunction (evident by echocardiographic signs of septal akinesia or hypokinesia) following many conventional myocardial protection strategies.

Despite absent septal function, the normally contracting free wall provides enough RV cavity compression by transverse circumferential narrowing to maintain normal right-

sided hemodynamic function if post operative PVR is low. Conversely, RV failure supervenes following surgical production of septal dysfunction when postoperative pulmonary hypertension results in raised PVR. Pulmonary arterial vasoconstriction or enhanced PVR from LV failure is, however, the secondary cause of this adverse right-sided hemodynamic event. The primary factor is septal stunning from inadequate septal myocardial protection. Preoperative septal twist existed before aortic cross clamping, so that postoperative impairment of the septal capacity leads to RV failure. These findings imply that the septum is the "lion of right ventricular function," because septal twisting is the predominant mechanism that ensures adequate RV performance when PVR is increased.

Sonomicrometry Studies

Sonomicrometer experiments confirm the commonality of an oblique orientation of septal fiber architecture with those of the LV free wall,[4] but crystal shortening only reflects local dimensional changes in the limited regions that were studied. However, recording of the maximal extent of regional shortening likely reflects the composite transmural ventricular twisting movement that is responsible for ejection and suction. Recognition of the importance of oblique angulations comprising the septal descending and ascending segments influences development of RV surgical restoration procedures to be subsequently described. Sonomicrometer crystal studies have shown that shortening develops before the septum fully contracts, so that the major contribution of the free wall fibers occurs during the phase of isometric contraction. In 1914, Wiggers[19] found the isometric phase was unaltered by afterloading the RV, because the pulmonary valve did not open during free wall contraction. Conversely, the pre-ejection phase was enhanced when inotropic stimulation directly affected the circumferential free wall muscle. Furthermore, information gleaned from crystal observation of delayed origin of shortening in the RVOT[4] is: 1) consistent with the aberrant oblique fibers described by Torrent Guasp,[3] and 2) confirms the clinical observations of Francis Fontan, who suggested that intraoperative RVOT pacing may impede RV ejection by prematurely obstructing the outflow tract.

Pathophysiology of RV Dysfunction

Septal and free wall structure function interaction is a key to understanding why the septal contribution to biventricular events is an important element in dealing with procedures to treat RV failure. Septal dysfunction during cardiac performance is defined by hypokinetic or akinetic motion. The abnormal change in movement alters both ventricles and develops when the septum is either stretched or becomes relatively ischemic. Impaired septal activity is observed after left-sided abnormalities (aortic insufficiency, LV outflow tract obstruction, mitral regurgitation, and so on) or following right-sided lesions such as pulmonary stenosis, pulmonary insufficiency, atrial septal defect, RV dysplasia, or septal stunning after cardiac surgery. The clinical importance of returning correct fiber orientation to restore proper septal function is evident from prior reports that documented absence of postoperative right heart failure following LV restoration in patients whose preoperative studies showed septal dysfunction from ischemic dilated cardiomyopathy.[20] Surgical LV restoration returns the bowed dysfunctional septum back into its normal anatomic central position and postoperative echocardiography confirms recovery of normal septal function.

Similar dysfunctional septal performance exists in dilated hearts following mitral insufficiency,[21] and Bernheim et al in 1910,[22] described the adverse functional consequences of left-sided septal stretch. Conversely, right-sided ventricular dilation is caused by RV events that: 1) stretch the free wall; 2) increase chamber size; and 3) bow the septum to the left side. Resultant septal hypokinesis or akinesis limits septal contribution to both left- and right-sided outputs. The septal contribution to LV failure following chronic RV failure may exist during right-sided congenital heart defects that bow the septum and include tetralogy of Fallot, pulmonary stenosis, RV dysplasia, or Ebstein's anomaly. In patients with these defects, the stretched RV septum may directly and adversely impact LV performance. An alteration of this clinical event was described by Dexter,[23] who indicated that a "reverse Bernheim effect" followed closing large atrial septal defects that caused RV volume overload; LV performance improved as the bowed septum became a functional midline structure.

A recurring functional septal dilemma occurs in patients with chronic pulmonary valve regurgitation who develop progressive RV dilatation and dysfunction many years after previous repair for tetralogy of Fallot or other forms of chronic pulmonary regurgitation and RV failure.[2] Several architectural disadvantages develop in these patients that progressively impair RV performance and include: 1) persistent pulmonary insufficiency; 2) a non-functional patch on the RV outflow tract; 3) chronic stretching of the remaining distended free wall; and 4) volume overload. Furthermore, secondary effects on the tricuspid valve follow RV dilatation and septal displacement toward the left side. The tricuspid annulus dilates and the stretched septum tethers the attached papillary muscle geometry to further impair leaflet coapatation, resulting in tricuspid insufficiency that accentuates the progressive RV failure.

The importance of the ventricular component of RV failure is emphasized by recent studies showing that correction of the inciting lesion of pulmonary insufficiency by pulmonary valve replacement during advanced stages of RV failure is often not sufficient treatment to allow RV recovery.[24] The earmark of this late stage of RV compromise is advanced RV volume increase, aneurysm or akinesia of a portion of the RVOT, and septal dysfunction. As previously noted, augmented PVR further compromises RV function.

Importantly, the septal stunning that often follows standard myocardial protection methods will accelerate RV failure, despite adequate surgical correction of congenital heart lesions. For example, sudden acute postoperative RV failure

may develop in preoperative pulmonary hypertension patients that displayed normal septal function before undergoing repair of the underlying congenital heart defect. The onset of this complication may not be evident in the operating room if the corrective procedure (such as closure of a ventricular septal defect) initially lowers pulmonary artery pressure by reducing the volume overload from shunting. Adequate free wall function generated from a large hypertrophied RV with normal pulmonary artery pressure may initially mask the importance of ongoing septal dysfunction that can be observed intra-operatively by septal hypokinesia or akinesia evident on echocardiogram. RV failure evolves when postoperative episodes of acute pulmonary vasospasm raise pulmonary artery pressure and the underlying septal injury impairs the adequate cardiac response to this change in afterload. The resultant loss of septal twisting becomes the underlying mechanism of failure and an untenable hemodynamic burden is then conferred toward the RV free wall. Unfortunately, the compressive force generated from the functioning basal loop transverse muscle may be insufficient to prevent RV failure because septal twisting is required to transmit cardiac flow beyond the high PVR.

RV Restoration: A Comprehensive Approach

The infrastructure to RV repair is linked to understanding that the RV is a crescent-shaped ventricular chamber with a triangular configuration within its inlet, body, and outflow structures under normal physiologic conditions. This geometric configuration differs from the elliptical shape of the normal conical LV chamber. RV geometry changes toward a more spherical shape following development of RV dilation and failure, as the midline septal fiber orientation stretches into a more transverse configuration, and free wall muscle tension increases following dilation. To counteract these changes, the essential components of RV restoration needed to return RV function include: free wall volume reduction, restoration of ventricular shape, and septal fiber realignment. Accordingly, these objectives closely resemble treatment options used during LV restoration.

The most common recent cohort of patients needing RV restoration include an increasing number of patients that present with RV dilatation and failure secondary to chronic pulmonary insufficiency after surgical repair of tetralogy of Fallot. In the past, pulmonary valve replacement was performed together with linear resection and plication of the RV free wall to treat outflow tract patch aneurysms. Recognition of implications of the structure/function relationships based on the HVMB may generate new RV restoration goals that: 1) reduce and limit the stretching of the free wall; 2) exclude RV aneurysm and remove previous patch and scar, 3) restore septal architecture with realignment of oblique myocardial fiber orientation.

These objectives also reduce RV cavity volume, remove residual RVOT or pulmonary artery obstruction, implant a competent valve in the pulmonary position, and may also require procedures to correct secondary tricuspid insufficiency.

The mechanical components of the surgical technique address resection and reconstruction of the free wall and outflow tract, together with rebuilding the bowed septum into a midline structure *with septal, rather than only free wall sutures*. Free wall reconstruction includes recognition that the stretched free wall reflects an "aneurysm" composed of the prior pericardial or Dacron patch and stretched residual free wall muscle. Excision of aneurysmal RV myocardium and any patch material in the outflow tract, together with identification of the residual stretched muscle, is the first maneuver. This is followed by imbrication of excessive free wall tissue during RV closure and restoration of the alignment of the oblique septal fibers. This maneuver is achieved by using either interrupted sutures between the free wall and septum (as previously described for the repair of inferior wall aneurysms,[25] or use of a circumferential U-shaped imbrication suture that is anchored to the septum, for the purpose of diminishing RV chamber size and restoring a normal septal anatomic configuration,[26] as shown in Fig. 4. Either suturing technique returns the septum to a midline location (as done with LV restoration), restores septal fiber alignment, and restores a more normal crescent-shaped RV chamber.

The RV outflow region is closed with a patch, and a Hegar dilator is used to avoid narrowing the outflow tract during the reconstruction. This method differs from the vertical resection and plication of the aneurysmal free wall as described by Del Nido and others.[27] Their longitudinal approach is an effective technique for reducing RV volume, but may fail to restore septal fiber alignment and return the septum into central position, thereby potentially limiting functional septal recovery. This non-septal approach has a common background with LV restoration because this type of linear free wall resection and plication technique was described by Cooley et al[28] in 1958 for the surgical treatment of cardiac failure from LV aneurysms. Dor[29] and Jatene[30] refined these methods of LV restoration by incorporating septum reconstruction in the procedure, thereby improving outcome by restoring septal fiber orientation. The evolution of RV restoration techniques now closely parallels the development of improved methods to achieve LV restoration.

Clinical Experience

The impact of the RV restoration using the principles and techniques described above to reconstruct the septum was evaluated by Frigiola et al[26] in a series of 16 patients with severe pulmonary regurgitation and RV dilatation. Each underwent preoperative evaluation by Doppler echocardiography, MRI, and RV myocardial acceleration during isovolumic contraction (IVA). The surgical procedure included pulmonary valve implantation and RVOT restoration achieved by removal of the aneurysm tissue, coupled with a ventriculoplasty as described to reduce volume. This ventriculoplasty was achieved by restoring the RVOT dimension using a 2-0

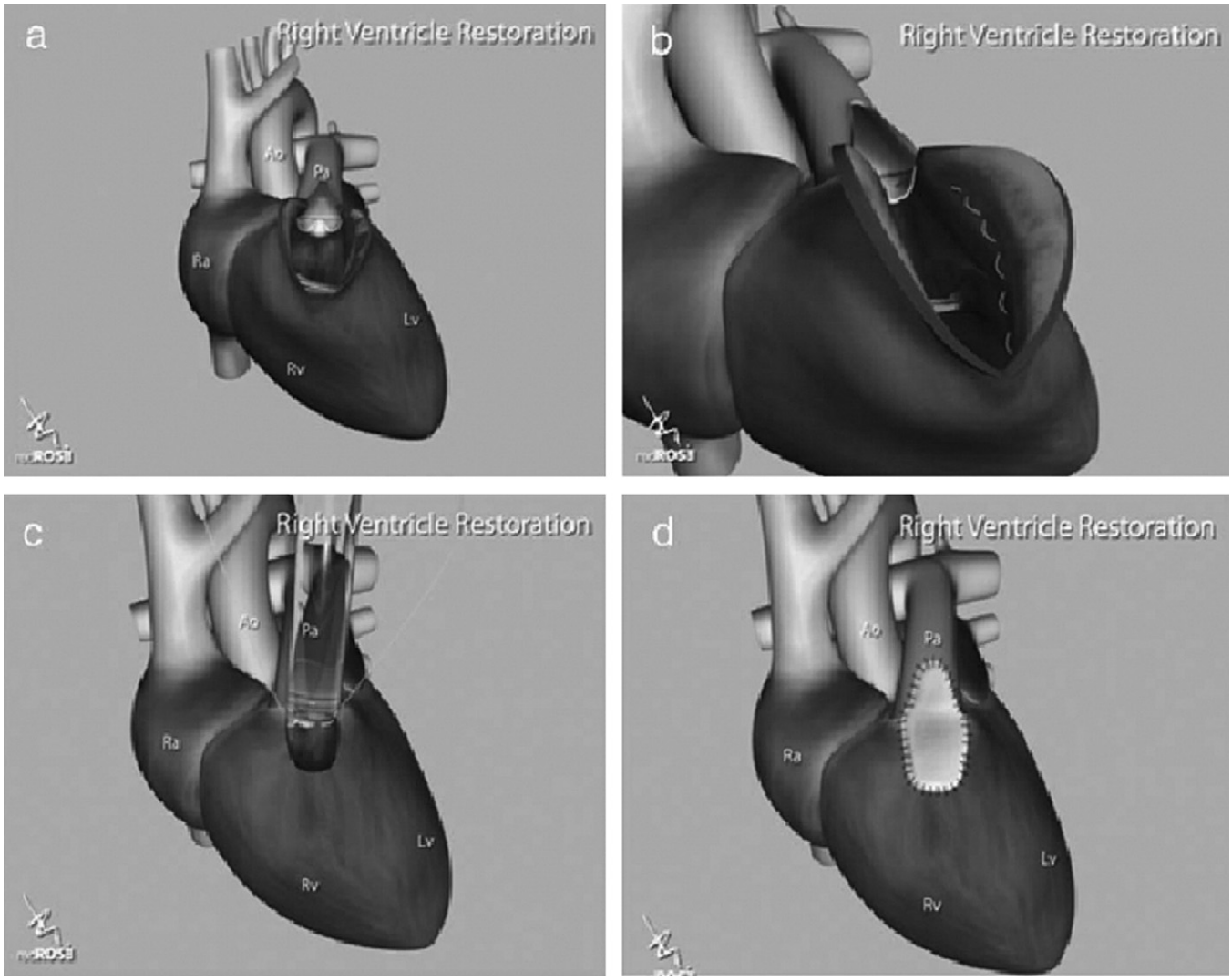

Figure 4 Schematic diagrams of the RV restoration procedure. (a) Open right ventriculotomy and pulmonary arteriotomy before restoration. (b) Placement of a continuous imbrication suture at the muscular edge between the septum and free wall. (c) Use of the Hegar dilator in RVOT to allow patch placement without restriction (note the reduction of ventricular volume by withdrawal of the imbrication suture). The suture ends are placed through the native annulus and secured. (d) Placement of the transannular patch along the imbricated ventriculotomy and over the pulmonary valve insertion site.

polytetrafluoroethylene suture (PTFE; Gore-Tex) imbrication suture anchored in the septum and free wall myocardium to achieve realignment of the oblique septal fibers and return of the septum to a midline position. This was performed using a 26 mm Hegar dilator to avoid restriction in the RVOT (as shown in Fig. 4).

A pulmonary valve was then inserted in the annulus and a Dacron transannular patch used to complete the reconstruction. To complement the approach to ventricular reconstruction, Frigiola et al[26] also described a more global method termed "VAT" (or ventricular restoration, arrhythmia surgery, and tricuspid annuloplasty), since seven patients also underwent a Maze procedure and four had tricuspid annuloplasty. All patients survived the operative procedure, and a 16-month follow-up showed reduction of cardiothoracic index and clinical improvement. Significant reduction of RV end diastolic volume and RV end systolic volume and increased RV ejection fraction was observed, and IVA changed from 0.7 ± 0.5 m/s^2 to 1.3 ± 0.6 m/s^2 in the 13 patients that underwent MRI and IVA during the preoperative control interval and 6 months after the procedure. The preliminary data from this experience confirms that the RV restoration is a simple and effective procedure, and introduces a structural component that should be added during pulmonary valve implantation in patients with severe RV dilatation and an underlying aneurysm or akinetic segment of the RV outflow tract.

RV restoration procedures that return the septum to a more central position have now been used to treat patients with RV failure and secondary arrhythmias from RV dilation in two clinical conditions including RV dysplasia[31] or persistence of right heart failure following pulmonary valve insertion to treat RV failure following chronic pulmonary insuffi-

ciency.[24] Avoidance of septal damage by inadequate intraoperative protection during the procedure is the one surgical prerequisite that must be adhered to when performing a septal procedure to return RV performance toward normal by changing RV spatial geometry. This goal was accomplished in these procedures by maintaining septal perfusion in the beating heart.[31] Similar results are possible with cardioplegia, but demonstration of consistent postoperative return of septal movement is needed before cardioplegia is routinely used. Each procedure followed the guidelines of reconstructing the RV for failure by re-establishing the normal structure/function relationships outlined by HVMB architectural considerations.

Conclusion

The RV free wall and septum are the architectural components of the RV, and the functional aspects of their anatomic fiber orientation are linked to the transverse fibers of the free wall and the oblique fibers comprising the septum. The correct angulation of these free wall and septal fiber patterns are confirmed by anatomic imaging studies and sonomicrometer crystal measurements. The predominantly oblique architecture of septal myocardium is similar for the left and right sides of the circulation, and governs the twisting cardiac action during ventricular ejection. This septal twisting action differs from the compression that results from shortening of transverse RV free wall fibers and is a necessary component for RV ejection into a pulmonary vascular bed with high resistance. These observations imply that the septum should be considered the "lion of right ventricular function."

Septal dysfunction results from lesions such as chronic pulmonary insufficiency following repair of tetralogy of Fallot, which stretch the septal fibers and alter the oblique fiber orientation and midline position of the septum. Adherence to restoring a normal oblique septal fiber orientation and regaining the septal midline position should govern operative techniques during procedures that treat RV failure. The procedures described in this report evolved from these considerations, and the early satisfactory preliminary results of restoring fiber orientation in the free wall and septum toward normal must be tested further in additional patients who present with chronic RV failure and require RV restoration.

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