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Right ventricular restoration during pulmonary valve implantation in adults with congenital heart disease☆

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Abstract

Objective: Pulmonary regurgitation may cause progressive right ventricular dilatation and dysfunction in adult patients previously repaired for right ventricular outflow tract obstruction (RVOTO), and who require subsequent valve implantation for relief of these symptoms. Right ventricular recovery after pulmonary valve implantation (PVI) may be closely linked to the functional importance of the structural presence of an aneurysm or akinetic segment in the RVOT area. To test this concept, the impact of the right ventricular restoration with a new surgical ventriculoplasty technique is evaluated following pulmonary valve implantation in patients with severe pulmonary regurgitation and right ventricular dilatation. **Methods:** Sixteen patients with severe pulmonary valve regurgitation (PVR) and right ventricular dilatation with RVOT aneurysm underwent right ventricular remodelling since January 2002. Each underwent preoperative evaluation by Doppler echocardiography, magnetic resonance imaging (MRI), and right ventricular myocardial acceleration during isovolumic contraction (IVC). The surgical procedure included pulmonary valve implantation and RVOT restoration achieved by removal of the aneurysm tissue, coupled with a ventriculoplasty to reduce volume, accomplished by creating a satisfactory RVOT dimension by placing with 2-0 Gortex suture to allow acceptance of a 26 Hegar dilator to avoid restriction. Thirteen associate surgical procedures were added in nine patients. **Results:** All patients survived the operative procedure and underwent a 16-month follow-up interval. A reduction of cardio thoracic index and a clinical improvement occurred in each patient. Significant reduction of RVEDV and RVESV and increased right ventricular ejection fraction was observed, and IVC 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 IVC during the preoperative control interval and 6 months after the procedure. **Conclusions:** This preliminary database implies that the right ventricular restoration is a simple and effective procedure, and introduces a structural component that should be added during pulmonary valve implantation in patients with severe right ventricular dilatation and underlying aneurysm or akinesia of the right ventricular outflow tract.

Keywords: Right ventricular dysfunction; RVOT repair; Pulmonary insufficiency; Surgical right ventricular restoration

1. Introduction

Pulmonary valve regurgitation (PVR) is the principal reason of reoperation in adult patients that previously underwent correction of right ventricular outflow tract obstruction (RVOTO) when they were children. The underlying prior lesions include tetralogy of Fallot (TOF) or pulmonary valve stenosis [1—3]. The physiologic consequences of PVR include progressive right ventricular dilatation and dysfunction [4—7], and the typical treatment has traditionally been pulmonary valve implantation (PVI) [1—8]. However, correction of the inciting lesion of pulmonary insufficiency may not be sufficient treatment to allow right ventricular recovery, when pulmonary valve implantation is performed at either an advanced stage of RV failure and/or when done in the presence of the structural defect of either aneurysm or akinesia of a portion of the right ventricular outflow tract (RVOT) [5,7,8].

The implications of these aneurysmal or akinetic lesions are that they disrupt right ventricle structure and thereby produce functional alterations of the physiologic peristaltic RV contraction pattern that proceeds from RV inlet toward the outlet, a contractile sequence that is heavily dependent upon RV geometric configuration. Recognition that either aneurysm or akinetic lesions of RVOT may impose extremely

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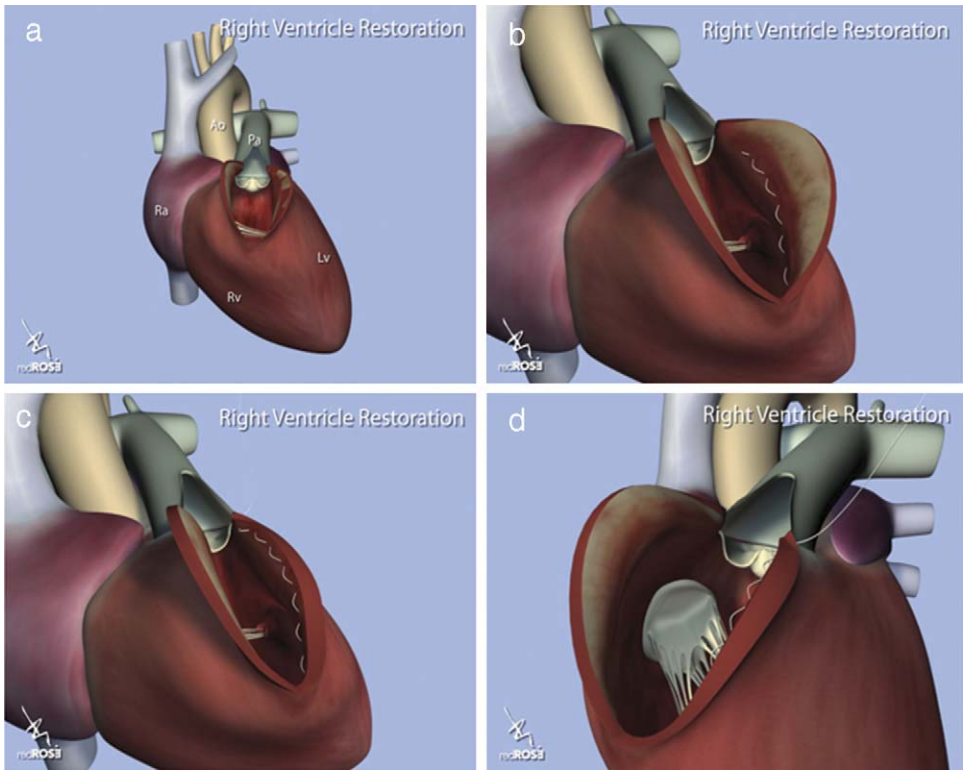

Fig. 1. Schematic drawings of right ventricular restoration. (a) Open ventriculotomy and pulmonary arteriotomy prior to restoration; (b) placement of continuous imbrication suture at muscular edge between septum and free wall; (c) excision of redundant free wall tissue; and (d) exposure of free wall for imbrication suture.

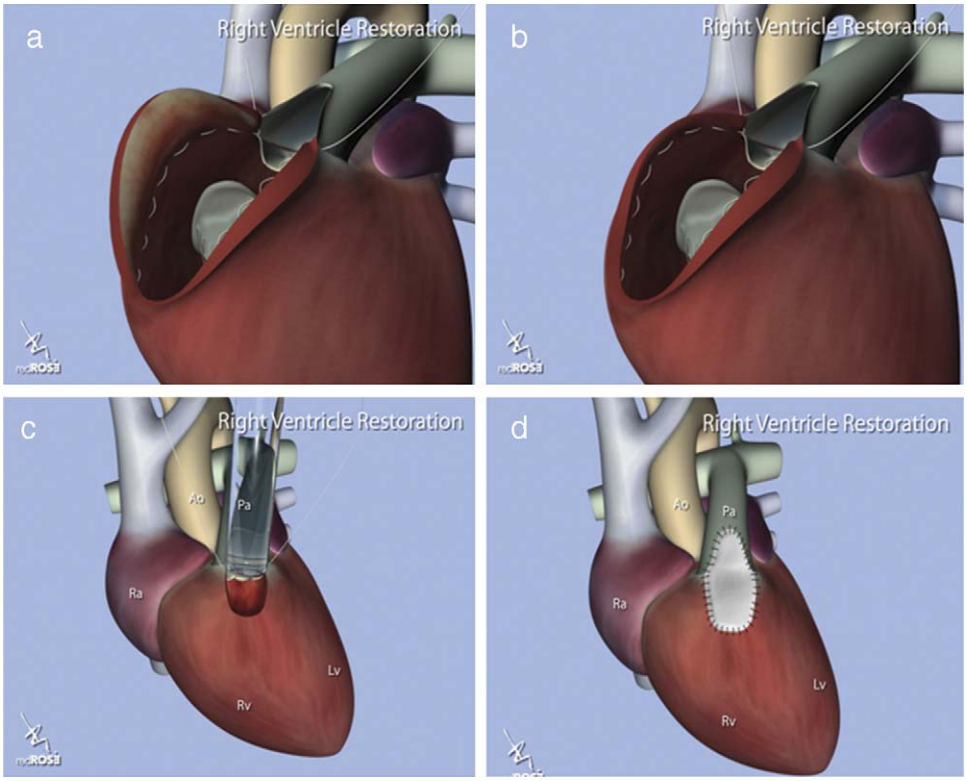

Fig. 2. Schematic drawings of right ventricular restoration. (a) Continuation of imbrication suture along muscular edge of free wall; (b) excision of redundant free wall tissue; (c) placement of Hegar dilator in RV outflow tract to allow patch placement without restriction; note, reduction of ventricular volume by withdrawal of imbrication suture; the suture ends are placed through the annulus and secured; and (d) placement of Haemashield outflow patch along imbricated ventriculotomy and over pulmonary arteriotomy site.

deleterious functional consequences led us to include surgical treatment of these ventricular wall defects at the time of PVI in patients with RV failure.

The aim of this preliminary study is to (a) evaluate the functional impact of right ventricular restoration during PVI in patients with severe PVR and right ventricular dilatation, and (b) to describe a new simple surgical technique of ventriculoplasty that imposes a 'valve–ventricle' treatment of RVOTO in the adult population with late complications of pulmonary valve regurgitation.

2. Materials and methods

Sixteen adult patients that had correction of tetralogy of Fallot repair ($n = 14$) or pulmonary valve stenosis relief ($n = 2$) during childhood underwent PVI between June 2002 and June 2005. Each patient presented with dilated right ventricle and aneurysm/akinesia RVOT, and also underwent right ventricular restoration with ventriculoplasty at the time of PVI.

There were nine males and seven females, and the mean age was 38 years (range 20–65 years). At the previous operation, 11 patients had been repaired with a transannular pericardial patch, 2 had a limited infundibular patch, 2 had a prosthetic valved conduit between the right ventricle and the pulmonary arteries, and 1 had a biological valve implanted in pulmonary position that had become stenotic.

The mean time interval from first repair and right ventricular remodelling and PVI was 18 years (range 9–35 years). Mean preoperative cardiothoracic index was 0.69 ± 0.09. Thirteen patients suffered from severe pulmonary valve regurgitation and three had combined pulmonary valve stenosis and insufficiency.

Four patients had previously episodes of arrhythmias: three ventricular tachycardia and one supraventricular tachycardia. Seven patients had moderate to severe tricuspid valve regurgitation, one had a residual ventricular septal defect, and one had aortic valve regurgitation. Four patients were in NYHA functional class IV, nine patients in class III, and three in class II.

All patients were preoperatively studied by Doppler echocardiography, magnetic resonance imaging (MRI), and right ventricular myocardial acceleration was measured during isovolumic contraction (IVC) during tissue Doppler imaging (TDI).

On echocardiography, all patients had a dilated right ventricle with cavity volumes greater than those measured in the left ventricle. At MRI, the mean RVEDV was 125 ± 32 ml/m^2; the mean RVESV was 63.5 ± 20 ml/m^2; and the mean RVEF was $50.1 \pm 9.2\%$. Mean IVA was 0.7 ± 0.5 m/s^2 at TDI measurement.

2.1. Surgical technique

After institution of cardiopulmonary bypass, the techniques of protection for accomplishing these procedures included the beating heart in 10 patients, while 6 patients received blood cardioplegia during aortic cross-clamping.

The procedure started by removing the previous transannular or infundibular patch in 14 patients and excising the prosthetic valved conduit in 2 patients. The annular prosthesis was then applied. The implanted pulmonary valves were determined by the surgeons' preference and included a mechanical valve in five cases, biological valve in five cases, prosthetic valved conduit in four cases, and pulmonary homograft in two cases.

All the aneurysmatic tissue in the RVOT was removed (Figs. 1 and 2; Videos 1 and 2, see Appendix A); the junction point was identification of the functioning edges of adjacent muscle. A ventriculoplasty was then done to reduce RV volume, and this was accomplished by placing a continuous 2-0 Gortex suture all around the previously identified still functioning muscular edges (including the septal area) and by pulling the two ends of the suture towards the pulmonary annulus, in order to create a RVOT dimension that was able to accept a 26 Hegar dilator to insure absence of restriction. The two ends of the Gortex suture were then fixed at level of the pulmonary valve annulus, with the dilator in place. After removing the dilator, a Dacron patch is placed over the reduced ventriculoplasty and pulmonary arteriotomy to close these openings (Figs. 1 and 2; Videos 1 and 2).

Thirteen associated surgical procedures were added in nine patients: seven tricuspid valve repairs, four arrhythmias surgery, one residual VSD closure, and one aortic valve replacement. In four patients, we addressed the ventricle, arrhythmias, and the tricuspid valve in a procedure we termed the complete 'VAT approach,' whereby V = ventricular remodelling, A = arrhythmia surgery, and T = tricuspid valve repair. The arrhythmia surgery consisted of right ventricular intraoperative radiofrequency ablation in three cases, and right-side Maze with radiofrequency ablation in one patient.

3. Results

All patients survived the surgical procedure. The median ICU stay was 3 days (range 1–12 days). One patient required reoperation on the second postoperative day for bleeding.

At 16 months mean follow-up, all patients showed a reduction of cardiothoracic index (mean 0.58 ± 0.07) and a clinical improvement. Whereas, preoperatively, 4 patients were in NYHA functional class IV, 9 patients in class III, and 3 in class II, the postoperative analysis showed 10 patients in NYHA functional class II, 6 in class III, and none in class IV.

Thirteen patients underwent MRI and IVA studies during the control preoperative interval and had repeat analysis 6 months after the procedure. There was significant reduction ($p < 0.05$) of RVEDV (from 125 ± 32 ml/m^2 to 96.0 ± 14.1 ml/m^2) and RVESV (from 63.5 ± 20 ml/m^2 to 38.8 ± 12.1 ml/m^2), increase in right ventricular ejection fraction (from $50.1 \pm 9.2\%$ to $58.0 \pm 10.0\%$) and improvement of IVA from 0.7 ± 0.5 m/s^2 to 1.3 ± 0.6 m/s^2. Fig. 3 shows pre- and postoperative CMR study in four-chamber and short-axis views.

Each patient submitted to arrhythmia surgery underwent Holter monitoring 3 and 12 months postoperatively, and the three patients who underwent right ventricular ablation had programmed ventricular tachycardia stimulation in the cardiac catheterization laboratory 6 months later. All

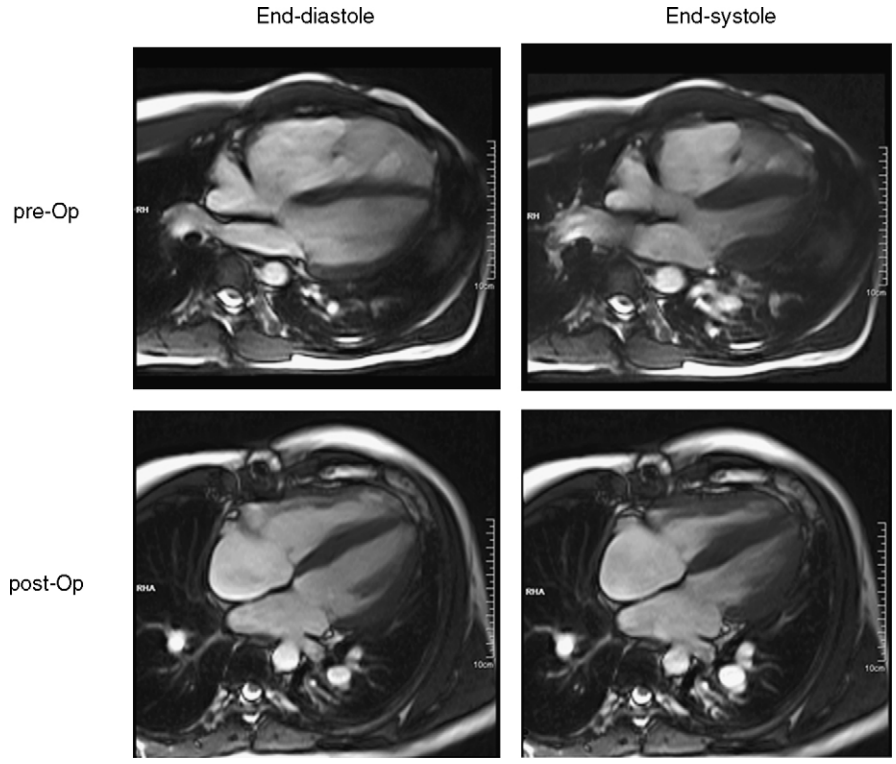

Fig. 3. Pre- and postoperative CMR study of patient undergoing right ventricular restoration. Note, (a) preoperative enlarged end-systolic and diastolic volume; and (b) postoperative study shows that restoration produces a significant reduction in RV endsystolic and diastolic volumes compared to baseline.

patients remained in sinus rhythm, and no ventricular arrhythmias were detected at the follow-up examination.

4. Discussion

Pulmonary valve regurgitation is a recognized complication that follows treatment of several congenital heart disease with RVOTO that include tetralogy of Fallot, pulmonary stenosis, pulmonary atresia with intact ventricular septum, and pulmonary atresia with VSD [1—8]. The consequences of chronic regurgitation include an increased volume load that becomes the principal hemodynamic substrate responsible for right ventricular dilatation and dysfunction, exercise intolerance, arrhythmias, and sudden death [1—4].

To offset this burden, pulmonary valve implantation has been considered the operation of choice, since this procedure carries a low operative risk and its beneficial effects include clinical improvement, decreased right ventricle size, and improvement in right ventricular function [1—8]. However, when the stage of the disease is too advanced, PVI alone may not sufficiently improve the right ventricular functional recovery, especially when the structural defect of an aneurysm or akinetic area exists in the RV outflow tract [5,7,8].

The secondary dilation of the right ventricular cavity that exists with aneurysm/akinesia of RVOT is extremely deleterious to both RV and LV function [6]. Furthermore, Therrien et al. [9] and Harrison et al. [10] identified RV enlargement as cause of development of arrhythmia and sudden death. This observation of deleterious volume-related effects on arrhythtmia generation is consistent with the reports of Babuty and Lab [11] who consider stretch the Okkum's razor reason for ventricular ectopy. More importantly, Davlouros et al. [12] confirmed the negative effect of aneurysmatic/akinetic RVOT on the RV function and demonstrated that this lesion also impairs left ventricular function due to an adverse interventricular interaction, and implied that the ventricular presence of this defect may lead to possible failure to recover RV function following PVI. Despite these observations, exclusion of this structural defect during PVI has received limited attention in the literature.

5. Underlying physiology

The right ventricle has a crescent-shaped geometry that contains a transverse outer wall which is folded around the cone-shaped left ventricle, whereby the septum is obliquely oriented [13]. The right ventricular ejection depends on a peristaltic contraction that proceeds from the inlet toward the outlet. The transversely oriented free wall constricts and the oblique septum twists when it has normal positional orientation. However, with either volume overload or right ventricular stretch, as occurs with pulmonary regurgitation or pulmonary hypertension, the septum becomes hypocontractile and bulges, thereby showing limited contribution to cardiac output [14].

In normal conditions, the septum is thought to be responsible for 50% of the ejection workload of the right ventricle and the left ventricular contraction contributes from 24 to 35% of right ventricular output [15]. The interaction of the septum to biventricular performance is evident from the

studies of explained ventricular interdependence, whereby one ventricle affects the other. Bernheim [16], in 1910, described right ventricular compression after left ventricular hypertrophy, and Dexter [17], in 1956, described a 'reverse Bernheim event' after large atrial septal defects and right-sided volume overload. Surgical correction of the ASD restored normal septal geometry and improved left-sided function by allowing more normal septal function through correction of this architectural disadvantage.

Functional abnormalities are sometimes expanded following repair of tetralogy of Fallot, especially in patients with pulmonary insufficiency and right ventricular dilation, as part of the septum is a non-contractile patch. Left ventricular function is often not normal and the left ventricular contribution to the right ventricular contraction decreases by septal bulging [12]. In this context, the role of the peristaltic contraction of the right ventricular free wall becomes a fundamental determinant of RV performance. At the same time, the integrity of the free wall becomes compromised because an aneurysm or akinetic area of the repaired RVOT causes a loss of energy and sphinteric action furthers the detrimental role in the right ventricular function.

6. Outflow tract pathology and management

Several factors account for the aneurysm or akinesia of the RV outflow tract [12] after repair of tetralogy of Fallot. The anatomic contributers include prior placement of a large transannular or large infundibular patch, as well as aggressive myomectomy during infundibular resection. There may also be an ischemic insult resulting from conal arterial branch interruption during suture placement area around the patch [12]. We believe that the aneurysmal/akinetic RVOT lesion should be surgically treated at the time of PVI; this stance is taken independent from the precise etiology of why the aneurysm is present.

The implications of restoring pulmonary valve function and avoiding RVOT aneurysm/akinesia were stressed by Davlouros in a review of late follow-up of 85 patients after TOF repair. D'Udekem [8] was the first to understand that the failure of RV recovery in Fallot patients after PVI was in part secondary to the delay of reoperation, and in part secondary to limitations of the surgical technique. D'Udekem made a logical parallel between the right and left ventricle that contained a non-functional area, and thereby addressed principals of left ventricular plication as LV aneurysms are treated. This background led him to suggest adding a pulmonary infundibuloplasty. This procedure is accomplished at the completion of homograft implantation, by narrowing the dimensions of ventriculotomy site, by employing two strips of felt pledgets during ventricle closure [7]. As with the left side, exclusion of the non-functional region will improve function the remote viable muscle whose architecture is changed by this geometric approach [18].

The restoration method used in this report involves a simpler technique that was developed to rebuild the natural components of RV geometry. Three principles governed this action and include (a) reduction of RV volume, (b) restoration of free wall and septal architecture, and (c) avoidance of outflow tract restriction. Exclusion of the prior transannular or infundibular patch and marking the new site by identifying the site of suture plication at the identified edges of functioning muscle were the first steps in accomplishing the first two goals. Construction of the reductive ventriculoplasty by imbricating the continuous 2-0 Gortex suture to plicate this segment simultaneously brought the septum into a more midline position and decreased RV volume by withdrawing and shrinking the free wall toward the annular site around the site of pulmonary valve implantation. Simultaneously, restriction of the outflow tract area was avoided by using the 26 Hegar dilator so that the overlying patch for closure avoided any restriction to RV emptying across the newly placed pulmonary valve.

7. A more global approach

The described technique for ventricular restoration only addressed one aspect of the long-standing complications, and in some patients we also addressed the secondary events of RV dilation that (a) compromises tricuspid valve function (via annular dilation and adverse leaflet tethering due to left-sided septal bulging), and (b) causes arrhythmias due to atrial or ventricular stretch [11]. These components characterize the complete 'VAT approach,' whereby V = ventricular restoration, A = arrhythmia surgery, and T = tricuspid valve repair.

Our aggressive approach to treat tricuspid regurgitation includes placing a tricuspid valve ring (TVR), an action that is consistent with the suggestions of other authors [4,6,7] who similarly recommend tricuspid valve repair at the time of pulmonary valve implantation. This correction directly deals with the deleterious systolic consequences of tricuspid insufficiency, a dysfunctional event that is detrimental and beyond adverse alterations due to RV outflow tract problems.

We have adopted the same policy for approaching atrial and ventricular arrhythmias. The preoperative incidence of atrial arrhythmias in repaired tetralogy of Fallot ranges from 2 to 23%, and is between 14 and 65% for ventricular arrhythmias [19,20]. Sustained ventricular tachycardia is present in more than 20% of these patients, with a risk of late sudden death of 2—6% over 18.5 ± 9.6 years [18—20]. These findings of the arrhythmogenetic effects of RV dilation mirror those we recently reported for dilated left ventricles treated by LV restoration [21].

The current approach for patients that will undergo PVI includes preoperative assessment for atrial and ventricular arrhythmias according to the regimen of (a) 24 h of electrocardiogram monitoring, (b) exercise testing, and (c) electrophysiologic study with programmed ventricular stimulation; this evaluation scheme parallels prior LV approaches. Treatment options include intraoperative right-side Maze ablation for atrial arrhythmias. Sustained ventricular tachycardia may be treated at the time of reoperation with direct endocardial resection or ablation, in addition to ventricular restoration that successfully ablates ventricular arrhythmias without endocardial resection and cryoablation [21].

Several associated procedures were done, as the global operative objective is correction of all problems (including VSD repair and aortic insufficiency). Consequently, we cannot

precisely distinguish which aspect of surgical correction is most important. Additionally, timing for reoperation in these patients remains a crucial point, and will be more accurately developed as increasing experience is accumulated.

8. Markers for recovery

A robust marker in assessment of RV function/dysfunction is required to facilitate decision-making. The currently available methods include angiography, MRI, and various echocardiographic techniques. Non-invasive assessment of RV function has been considered difficult because of the geometry of RV shape and prior lack of knowledge about how parts of this architecture influence performance. For example, some investigators use RV ejection-phase indices (MRI or 3D echocardiography) to determine RV function, but the load dependency of ejection fraction limits their utility [20,22]. There is great potential benefit from MRI, as it overcomes limitations of other methods and PVR has been recently shown to be of great advantage when RVEDVI is $>150\ ml/m^2$. Of greater importance, delay of PVR in advanced disease characterized by RVEDVI $>200\ ml/m^2$ was associated with failure to improve [23]. From the vantage point of geometric correction of RV failure, our findings suggest that attention should be directed toward the 'valve—ventricle solution,' rather than the current 'valve only approach,' since a ventriculotomy is a basic component of the surgical procedure, and the suggested geometric closure simultaneously addresses the 'valve—ventricle' at the time of intervention.

Others have indicated that IVA on TDI can be used in long-term monitoring of these patients, since these measures were experimentally [24] and clinically [25] shown to be rapidly obtained and provide reliable non-invasive reproducible measurements of RV systolic function. We used this method (together with MRI) in 13 of 16 patients (involving the more recent ones) and demonstrated a significant reduction of RVEDV and RVESV, increasing RVEF and IVA from $0.7 \pm 0.5\ m/s^2$ to $1.3 \pm 0.6\ m/s^2$. These markers document the improved NYHA classification recorded in each of the 16 survivors of this preliminary report. In the future, we shall study pre- and postoperative septal function by MRI, as we presume restoration of the midline septal position will return the twisting action that is an important determinant of septal function.

9. Conclusions

This early report affirms that RV restoration is a safe, simple, and effective surgical procedure that should be added during PVI in patients with severe RV dilatation and aneurysmal/akinetic RVOT. The concept is that a 'valve—ventricle' approach is needed in dilated RV failure. A novel ventricular procedure is described that addresses a technique that combines the principals of reduction, restoration, and avoidance of restriction in its planning and execution phases. Furthermore, we introduce the 'complete VAT (ventricle, tricuspid valve, and arrhythmia) approach' to define a global view of addressing the secondary complications of RV dilation. The enclosed preliminary data is encouraging, but a larger number of cases and a longer follow-up will be necessary to confirm the validity of our approach.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ejcts.2006.03.007.