Right ventricular outflow tract reconstruction for pulmonary regurgitation after repair of tetralogy of Fallot:
Preliminary results

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Abstract

Background: Pulmonary regurgitation after tetralogy of Fallot (ToF) repair is associated with right ventricular dilatation, failure and arrhythmia. Timing and technique for re-intervention remain controversial. Methods: Our recent approach is to reconstruct the dilated right ventricle outflow tract (RVOT) as a fibro-muscular sleeve to support a pulmonary homograft valve conduit in orthotopic position. Indication is based on clinical and magnetic resonance (MR) criteria. We reviewed all patients who underwent RVOT reconstruction between January 2004 and February 2005. There were seven children (mean age 14.2 ± 2 years) operated 13.2 ± 2 years after ToF repair, and 12 adults (mean age 30.15 ± 15 years) operated 23.1 ± 10 years after ToF repair. Exercise testing and MR evaluation prior to surgery and at 1 year postoperative follow-up were compared.

Results: There was no operative mortality. At 1 year, pulmonary regurgitation was mild or less in 16/19 patients. Right ventricular (RV) end-diastolic (158 ± 51 to 103 ± 36 ml/m², \(p < 0.001\)) and end-systolic volumes (85 ± 42 to 49 ± 24 ml/m², \(p = 0.001\)) fell significantly. Importantly, effective RV stroke volume (43 ± 10 to 48 ± 7 ml/m², \(p = 0.019\)) and left ventricular (LV) stroke volume (43 ± 7 to 47 ± 7 ml/m², \(p = 0.009\)) increased significantly. The mean RV/LV end-diastolic volume ratio fell markedly in both children and adults (2.22 ± 0.62 to 1.38 ± 0.52). However, no improvement in maximal VO₂ on exercise was noted in either group.

Conclusions: RVOT reconstruction restored valve function, improved RV dimensions and left and right stroke volumes. Maximal exercise capacity did not improve in either children or adults.

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Keywords: Tetralogy of Fallot; Homograft; Pulmonary regurgitation

1. Introduction

Pulmonary regurgitation (PR) after tetralogy of Fallot (ToF) repair can lead to right ventricular (RV) dilatation and failure, tricuspid regurgitation, impaired exercise performance and arrhythmias [1–3]. A timely re-operation to insert a pulmonary valve may prevent these consequences [1,4,5]. However, the precise timing for re-operation and best surgical technique remain controversial. Furthermore, assessment of the haemodynamic consequences of PR and the effects of subsequent pulmonary valve replacement is known to be difficult. The role of RVOT reconstruction has been emphasised in previous studies [6]. Our current approach is to reconstruct the dilated right ventricular outflow tract as a fibromuscular sleeve to support a homograft valve conduit in the orthotopic position with the aim of improving valve durability [7]. Should deterioration of valvar function recur, this repair provides a substrate for future percutaneous valve implantation [8,9]. Since 2004, we have prospectively evaluated all patients presenting with pulmonary regurgitation using magnetic resonance (MR) [10,11], detailed echocardiography and cardiopulmonary exercise testing. We report here the results of the cohort of patients who went on to undergo surgical RVOT reconstruction with pulmonary valve insertion.

2. Material and methods

With the approval of the Research and Development Committee, we retrospectively reviewed the records of all patients who underwent surgical RVOT reconstruction for PR after ToF repair at Great Ormond Street Hospital and the Heart Hospital, London between January 2004 and February 2005. Indication for re-intervention was based on a
controlled systemic hypothermic environment (28—32 °C) warranted femoro-femoral bypass. The latter provides a Midline conduit and haemorrhage during redo-sternotomy aortic cross clamping with cold blood cardioplegia was used. Defect or other intra-cardiac lesion requiring attention, canulation at 32 °C on the beating heart with ascending aortic and bicaval pressure monitoring and placement of external defibrillator pads, redo sternotomy was undertaken with an oscillating saw and the lower aspect of the sternum was opened with Mayo scissors. Adhesions were divided with diathermy. Most cases were performed under routine cardiopulmonary bypass on the beating heart with ascending aortic and bicaval canulation at 32 °C. In case of residual ventricular septal defect or other intra-cardiac lesion requiring attention, aortic cross clamping with cold blood cardioplegia was used. Midline conduit and haemorrhage during redo-sternotomy warranted femoro-femoral bypass. The latter provides a controlled systemic hypothermic environment (28—32 °C) at low flow for a few minutes so that surgical access into the sub-sternal space can be gained with a wider margin of safety.

Pulmonary homograft insertion was the preferred surgical option. The native main pulmonary artery was dissected out and circumferentially transected just below the main pulmonary artery bifurcation. The branch pulmonary arteries were sized, and dealt with if necessary. A longitudinal incision was made into the proximal outflow tract. Any hypertrophied muscular trabeculations in the sub-junctional region were divided to create a widely open pathway. In patients with aneurysmal RVOT patches, the akinetic thin area was excised leaving a small fibrous rim at the muscular margin, followed by plication with 4—0 Prolene to reconstruct the outflow tract with the aim of improving the distorted RVOT geometry and reducing the cavity size. The homograft was tailored in length to connect with the distal pulmonary artery using 5—0 Prolene. The proximal end of the homograft valve was implanted within the newly created muscular 'sleeve' for support. Patients with severe tricuspid regurgitation underwent concomitant valve repair. Patients with significant atrial or ventricular arrhythmia received anti-arrhythmic surgery using cryoablation.

2.2. Statistical analysis

Data are presented as mean ± standard deviation. The median is also mentioned when relevant. The preoperative and 1 year results were compared using a paired Student’s t-test. Statistical significance was reached when p was <0.05. We analysed the data using SPSS for Macintosh v.11 (SPSS, Chicago, Illinois).

3. Results

3.1. Patient characteristics

Between January 2004 and February 2005, 19 patients with previous ToF repair underwent RVOT reconstruction. Mean follow-up was 1.6 ± 0.2 years (median 1.5 years, 1.4—1.98 years). Associated conditions included: 22q11 deletion (n = 1), repaired aorto-pulmonary window (n = 1) and tracheo-oesophageal fistula (n = 1). Seven patients under 16 years of age (two males) were operated at Great Ormond Street Hospital at a mean age of 13.6 ± 1.7 years. Twelve patients older than 16 years (eight males) were operated on at the Heart Hospital at a mean age of 29.9 ± 14.5 years. All patients, but one, had undergone transannular patch repair as their ToF correction. One patient who had undergone pulmonary valvotomy followed by later RVOT revision with transannular patch insertion. Three patients had undergone previous palliation with systemic—pulmonary shunts that had been ligated at the time of repair. The time between ToF repair and RVOT reconstruction was 19.3 ± 9.1 years (12.8 ± 1.6 years in the children and 23.1 ± 9.7 years in the adults). Three patients had undergone subsequent stent implantation for branch pulmonary artery stenosis.

Eleven patients were in NYHA class II or III with the remainder in class I. All patients had severe pulmonary regurgitation on MR imaging (regurgitant fraction 40 ±%). RV/LV end-diastolic volume ratio was 2.22 ± 0.6. RV end-diastolic volume was 158 ± 51 ml/m², RV end-systolic volume was 85 ± 42 ml/m². LV end-diastolic volume was 71 ± 10 ml/m² and LV end-systolic volume was 29.6 ± 8.2 ml/m².

Fig. 1. Surgical technique for right ventricular outflow tract reconstruction.
On echocardiography, three patients had mild, two moderate and two severe tricuspid regurgitation. RVOT velocities were $1.9 \pm 0.7$ m/s.

On cardiopulmonary exercise testing, the patients achieved $70 \pm 21\%$ of predicted VO$_{2\text{max}}$ and $72 \pm 18\%$ of the predicted workload.

3.2. At operation

No patient had to go on femoral bypass. The mean cooling temperature was $30 \pm 2$ °C. All patients received a pulmonary homograft of $22 \pm 2$ mm diameter. Mean bypass time was $89 \pm 29$ min. Two patients required aortic cross-clamping (for closure of a residual atrial septal defect and tricuspid annuloplasty; cryoablation for atrial flutter and closure of a residual ventricular septal defect). Two patients had resection of obstructive RVOT muscle bundles. Three patients required reconstruction of one or both branch pulmonary arteries.

3.3. Postoperative course

There was no operative mortality. All patients were extubated within 24 h following surgery. No patient was re-operated. Two patients developed pleural effusions. One patient experienced transient renal failure and one patient had early homograft failure with significant PR and has since been re-operated. Postoperative hospital stay was of $8 \pm 5$ days.

3.4. At 1 year follow-up

There was no late mortality. All patients but one reported a clinical improvement. Only two patients were in NYHA class II with all others in class I.

On MR imaging pulmonary regurgitation was mild or less in 16 patients. The remaining three had regurgitant fractions of 30%, 31% and 40% respectively. The early PR appearing after re-operation in these three patients was due to a problem with the operative technique (kinking of the homograft) in one case and rapid homograft failure in the two other cases (one proximal stenosis with poststenotic dilatation and one cusp prolapse). In most patients, RV volumes normalized and RV ejection fraction improved. This was associated with an increase in LV end-diastolic volume and an improvement in LV stroke volume. The results are presented in Tables 1 and 2.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Preoperative and 1 year postoperative right ventricular volume and function MRI assessment</th>
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<tbody>
<tr>
<td>MRI Data (n = 19)</td>
<td>Preoperative</td>
</tr>
<tr>
<td>PRF (%)</td>
<td>$40 \pm 9$</td>
</tr>
<tr>
<td>RVEDV (ml/m$^2$)</td>
<td>$158 \pm 51$</td>
</tr>
<tr>
<td>RVEF (ml/m$^2$)</td>
<td>$85 \pm 42$</td>
</tr>
<tr>
<td>Effective RV stroke volume (ml/m$^2$)</td>
<td>$43 \pm 10$</td>
</tr>
<tr>
<td>RV ejection fraction (%)</td>
<td>$48 \pm 10$</td>
</tr>
</tbody>
</table>

PRF: pulmonary regurgitant fraction; RV: right ventricle; RVEDV: right ventricular end-diastolic volume; RVEF: right ventricular ejection fraction. Effective RV stroke volume = $(1 – PRF) \times (RVEDV – RVEF)$.

On cardiopulmonary exercise testing, obtained in 15 patients at the time of the study (no patient was excluded on the basis of his status), there was no significant improvement demonstrated in VO$_{2\text{max}}$ and anaerobic threshold. Results are shown in Table 3.

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Cardiopulmonary exercise test data</th>
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<tbody>
<tr>
<td>n = 15</td>
<td>Pre</td>
</tr>
<tr>
<td>Maximal VO$_2$ (% predicted)</td>
<td>$71.2 \pm 21.4$</td>
</tr>
<tr>
<td>Anaerobic threshold (% of maximal VO$_2$)</td>
<td>$48.6 \pm 11.9$</td>
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4. Discussion

This paper reports our recent experience of pulmonary valve replacement using RVOT reconstruction techniques involving RVOT plication and pulmonary homograft insertion for the surgical treatment of pulmonary regurgitation late after transannular patch repair of ToF. Whilst our results are consistent in terms of operative risk with that reported elsewhere, we have also been able to demonstrate a volumetric and functional improvement at 1 year following this intervention.

Previous studies report conflicting results in terms of the ability of the RV to recover following surgical pulmonary valve replacement [1,12–15]. In particular, the results of the Toronto group have caused much interest as they were unable to demonstrate significant improvements in RV volume or function following late pulmonary valve replacement [14]. The timing of operation and surgical technique were thus incriminated [12] and have led to a general preference for earlier re-operation [16] and a shift towards resection of patch aneurysms and remodelling of the RVOT. The preoperative RV volumes and the time between definitive repair and RVOT revision compare well with our population, suggesting that the surgical technique of the subsequent RVOT revision may be implicated. Their technique involved a bioprosthetic valve covered with a pericardial patch extending from the pulmonary bifurcation to the infundibulum. Their study was, however, limited by the late age of ToF repair and pulmonary valve insertion in the population (compared with our population $12.1 \pm 10.6$ years vs $4.7 \pm 7.6$ years at ToF repair, $33.9 \pm 9.2$ years vs $23.9 \pm 14$ years at pulmonary valve implantation) and the use of radionuclide angiography, which may be a less sensitive technique for measuring volume change than MR imaging. Recent work by the same group has suggested that restoration of normal RV
volumes after pulmonary valve insertion is not achievable if RV end-diastolic volume prior to intervention is greater than 170 ml/m² and RV end-systolic volume is greater than 85 ml/m². The mean preoperative RV end-diastolic volume of our population was 158 ± 51 ml/m² and RV end-systolic volume was 85 ± 42 ml/m² and most patients, demonstrated normalised RV volumes at 1 year (Figs. 2 and 3).

It has been demonstrated that sub-optimal right/left ventricular interaction is an important determinant of the clinical status in these patients [17]. The improvement in left ventricular parameters is therefore encouraging and may reflect better filling following restoration of a competent pulmonary valve. However, though the patients felt better at 1 year, and others have reported significant improvement in exercise parameters after pulmonary valve insertion, [18] we saw no improvement in objective maximal exercise capacity in our population. There may be a number of explanations for this, these patients are de-conditioned and may require re-adaptation to improve their clinical status, pulmonary function may be altered or they may have chronotropic incompetence which could impair their effort tolerance [19].

It has also been shown that diastolic RV function improves late, which could contribute to a slow recovery [20]. The relative importance of these factors is difficult to pinpoint. Most daily activities are performed at a sub-maximal level. The parameters we measured may not correspond to the reported clinical improvement. Some have suggested a 6 min walking test can be useful. This test, performed at a sub-maximal level, could be better related to the daily activities of the patients.

The importance of RVOT reconstruction has been emphasised [21]. Resection of the aneurysmal transannular patch, and RVOT plication results in optimal volume reduction of the right ventricle [11,21]. As for pulmonary valve insertion, some now implant an oversized xenograft, a Hancock conduit [22] or even mechanical valves [23]. Xenografts can provide good results, as shown in the more recent Toronto experience [16], but they require use of a ‘transannular’ patch, which may permit only limited RV volume reduction. Hancock conduits have not demonstrated superior results to homografts and are also prone to degeneration. Results with bovine jugular vein conduits remain controversial at this time [24]. Mechanical valves could be useful in those patients who could not safely undergo re-operation. No perfect conduit is available at this time for pulmonary valve insertion. In our population, we chose to implant a pulmonary homograft valve due to its known advantages [25]. Durability may be further improved by implanting in the anatomical position, such as in a Ross procedure. This was demonstrated in a study comparing paediatric Ross and non-Ross patients, with a homograft survival at 5 years of 93% in the Ross group versus 66% in the non-Ross group [7]. By reconstructing the RVOT, we aimed to implant the pulmonary homograft in the anatomic position. Another advantage of homograft conduits is the facilitation of future interventional procedures such as percutaneous pulmonary valve implantation [8]. These percutaneous valves are also prone to degeneration, but they represent a valuable therapeutic option that may reduce the number of surgeries a patient needs during his lifetime.

Our results at 1 year support our surgical strategy. However, the early PR observed in three cases was due to the operative technique in one case and to early homograft failure in the other two cases. Extreme care must be taken when implanting the homograft as any twisting or kinking due to an excessive length of the homograft can lead to early failure. The pulmonary valve function data at the time of harvest must also be available when selecting the graft.

This study was not designed to indicate the right timing of re-operation and to help define the indications for redo-surgery. With the currently used indications for PVR after ToF repair the outcome of surgery is favourable in terms of volume reduction and heart function. Longer follow-up will be necessary.

4.1. Limitations

This study is small and follow-up is limited. To understand the true role of RVOT reconstruction and its contribution to right ventricular recovery, patients should undergo prospective randomisation to this technique or conventional surgery and be subject to detailed follow-up.
4.2. Conclusion

Revision of the RVOT with RVOT reconstruction and valve insertion in the anatomic position resulted in normalisation of RV volumes and ejection fraction at 1 year. Patients reported symptomatic improvement but cardiopulmonary exercise capacity did not improve.

References


Appendix A. Conference discussion

Dr M. Hazeckamp (Leiden, The Netherlands): I fully agree with you, but I didn’t see any data on another reported parameter which is the duration of the QRS complex in the EKG. Have you looked to that also, to electrocardiographic measurements?

Dr Ghez: It has been looked at. I do not have this data here. The Q wave durations were prolonged, as you expect in these patients.

Dr Hazeckamp: Because we use more or less the same criteria as you do, apparently, for the timing of this intervention. But we also use the duration of the QRS complex. And of course, if there is a history of ventricular fibrillation or V-tach or whatever, it should be taken into account also.

Dr Ghez: Yes.

Dr D. Metras (Marseille, France): Did you mention the delay between the primary correction and the re-operation in these patients? Maybe it was too quick to see the results.

Dr Ghez: The children, which are the most recent patients, had their tetralogy of Fallot repaired before the age of 1 year and they had their RVOT reconstruction 13 years later in average. For the adults, they had their primary TOF repair at about 7 years of age. And the mean delay between Tetrology of Fallot repair and RVOT reconstruction was 23 years.

Dr Metras: So all these patients had a large transannular patch probably?

Dr Ghez: Yes, all of them.

Dr Metras: Because classically in the literature, pulmonary insufficiency could appear even if you have not a transannular patch but just after commissurotomy of a pulmonary valve. Was that the case in your patients?

Dr Ghez: All these patients had a transannular patch.

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