Feasibility and related outcome of intraluminal pulmonary artery banding

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Abstract

OBJECTIVES: This retrospective study evaluated the feasibility and related outcome of intraluminal pulmonary artery banding (I-PAB).

METHODS: Thirty-two children underwent I-PAB between July 2006 and April 2014. The median age and weight were 60 days (range: 5 days to 4.2 years) and 3.7 kg (range: 2.6–13.0 kg), respectively. Cardiac diagnoses included single ventricle morphology (n = 11), complex ventricular septal defects (n = 11), balanced atrioventricular septal defects (n = 3), congenitally corrected transposition of the great arteries (n = 2) and aortic arch hypoplasia with ventricular septal defects (n = 5). On cardiopulmonary bypass (CPB), 2 I-PAB modifications with either 1 (n = 24) or 2 (hour-glass-technique, n = 8) fenestrated pericardial patches were performed.

RESULTS: The median fenestration size was 5 mm (range: 4–6.5 mm). In 18 patients I-PAB was a solitary procedure; in 3 of them the decision was made intraoperatively. There was no hospital mortality. The median interval to debanding was 189 days (range: 112 days to 2.6 years). During this period, we observed a significant increase in the pressure gradient over I-PAB (P < 0.01), whereas arterial saturations remained stable. Four patients received balloon dilatation of I-PAB to prolong the palliation period. No patient experienced band occlusion, pulmonary hypertension related to I-PAB, coronary or pulmonary valve impairment. Debanding was performed in 27 patients and one of them required pulmonary patch arterioplasty due to I-PAB-associated pulmonary trunk distortion. Three patients are still awaiting further surgery. There were 2 late deaths prior to, and 3 after debanding, all not related to I-PAB.

CONCLUSIONS: I-PAB with an exactly defined internal orifice is feasible and effective. Although arterial saturations seem to remain stable, balloon dilatation of I-PAB can be performed safely and efficiently in order to prolong the palliation period. The rate of I-PAB-related complications is low, which might improve the long-term patient outcome. Therefore, despite requiring CPB, I-PAB is our institutional preference for children who require pulmonary artery banding.

Keywords: Pulmonary artery banding • Cardiopulmonary bypass • Paediatric

INTRODUCTION

The surgical management of congenital cardiac defects has evolved considerably and currently, early primary repair is favoured for most children. However, palliative pulmonary artery banding (PAB) continues to maintain its role in the management of certain cardiac defects with pulmonary overcirculation, such as selected cases of multiple VSDs [1] and functionally univentricular hearts with unrestricted pulmonary blood flow [2]. PAB is also considered in a more controversial situation such as staged surgical correction in the repair of complex aortic arch obstruction with intracardiac defect [3], or in left ventricular retraining in simple transposition of the great arteries with late referral [4] as a preparation for a subsequent arterial switch operation. Accordingly, in congenitally corrected transposition of the great arteries, PAB could be performed as a preparation of the subpulmonary, morphological left ventricle so that it can overcome the systemic resistance in the following double switch procedure or as a definitive palliation by induction of septum shift to reduce tricuspid regurgitation [5].

However, a strategy of staged surgical corrections exposes the patient to the cumulative surgical risks of both PAB and definitive repair [6]. Complication of PAB can have adverse effects on long-term outcome that demands additional management. Major drawbacks have been recognized with the use of extraluminal PAB, which is performed by placing and tightening a non-stretchable band or tape around the pulmonary artery. These include band mi-
Debanding procedure. The debanding techniques and simultaneous CPB strategies depend on the underlying cardiovascular malformation. In case of a bidirectional cavopulmonary connection, the main pulmonary trunk was divided at the band site. I-PAB was removed; the proximal pulmonary artery and pulmonary valve leaflets were over-sewn separately. To avoid branch pulmonary artery distortion, the distal pulmonary artery stump was closed by using a bovine pericardial patch. In biventricular corrections, the main pulmonary trunk was opened transversally at its anterior wall to remove the one-patch I-PAB (Fig. 1B and C) and closed with a running suture. In the two-patch technique, the 'hour glass' structure was completely removed and both ends of the pulmonary artery were directly reanastomosed. Relevant anatomical barriers resulting from I-PAB were addressed during the debanding procedure.

Outcome

Analysis included length of hospital stay as well as early, intermediate and total survival. The need and effectiveness of interventional balloon dilatation of I-PAB was noted. Early and long-term complications along with their association to I-PAB were reviewed.

Haemodynamic evaluation

Data were obtained intraoperatively, before hospital discharge and in the late inter-stage period before debanding. Haemodynamic evaluation of I-PAB was based on the patient’s clinical condition, systemic arterial saturations and echocardiographic measurement of haemodynamic data. In addition, catheterization was performed before the debanding procedure specifically to assess the degree of stenosis or anatomical distortion and the associated requirement of pulmonary artery plasty.

I-PAB in relation to Trusler’s rule

According to Trusler’s rule [11], cyanotic and cyanotic patients receive a band at a circumference of 20 mm + 1 mm/kg body weight or 24 mm + 1 mm/kg body weight, respectively. The
<table>
<thead>
<tr>
<th>Case Nr.</th>
<th>Sex</th>
<th>Age</th>
<th>Weight (kg)</th>
<th>Cardiac diagnosis</th>
<th>Extracardiac malformations</th>
<th>Performed surgery</th>
<th>I-PAB indication</th>
<th>Fenestration size (mm)</th>
<th>Postoperative complications</th>
<th>Long-term outcome</th>
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<td>1</td>
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<td>43</td>
<td>2.6</td>
<td>Hypoplastic LV, DORV, Scimitar syndrome</td>
<td>Congenital hydrocephalus</td>
<td>Hour glass I-PAB, PAPVD repair, atrioplastomy</td>
<td>Univentricular palliation</td>
<td>5</td>
<td>Low-output syndrome, CPR</td>
<td>Debanding, BCPC</td>
</tr>
<tr>
<td>2</td>
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<td>65</td>
<td>4</td>
<td>Taussig-Bing DORV</td>
<td>None</td>
<td>Hour glass I-PAB</td>
<td>Univentricular palliation</td>
<td>6</td>
<td>None</td>
<td>Debanding, BCPC, TCPC</td>
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<tr>
<td>3</td>
<td>Male</td>
<td>4 yrs</td>
<td>13</td>
<td>VSD, straddling tricuspid valve, pulmonary hypertension</td>
<td>None</td>
<td>Hour glass I-PAB, atrioplastomy</td>
<td>VSD</td>
<td>5</td>
<td>Severe cyanosis due to pulmonary hypertension, catheter-associated infection</td>
<td>Debanding, biventricular repair</td>
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<tr>
<td>4</td>
<td>Female</td>
<td>3 m</td>
<td>5.6</td>
<td>cc-TGA, severe TR, VSD, congenital heart block</td>
<td>None</td>
<td>Hour glass I-PAB, pacemaker implantation</td>
<td>cc-TGA, severe TR</td>
<td>5</td>
<td>None</td>
<td>Death before debanding (age 8.5 months; SUID)</td>
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<td>VSDs</td>
<td>6</td>
<td>Junctional ectopic tachycardia</td>
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<td>Hour glass I-PAB, aortic arch reconstruction</td>
<td>2-stage aortic arch repair</td>
<td>5</td>
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<td>Debanding, biventricular repair</td>
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<td>Male</td>
<td>6</td>
<td>3.7</td>
<td>Interrupted aortic arch type B, VSD, s/p cardiogenic shock</td>
<td>22q11.2</td>
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<td>6</td>
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<td>Trisomy 21, CHARGE syndrome</td>
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<td>Cerebral haemorrhage</td>
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<td>3.5</td>
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<td>One-patch I-PAB</td>
<td>Univentricular palliation</td>
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<td>9.1</td>
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<td>6.7</td>
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<td>Septic shock due to necrotizing enterocolitis (NEC)</td>
<td>One-patch I-PAB, aortic arch reconstruction, sigma resection and stoma (NEC)</td>
<td>2-stage aortic arch repair</td>
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<td>(Ileostomy due to NEC)</td>
<td>Debanding, biventricular repair</td>
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<td>13</td>
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<td>4</td>
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<td>Swiss cheese VSDs, PAPVD</td>
<td>None</td>
<td>One-patch I-PAB</td>
<td>VSDs</td>
<td>6</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
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<tr>
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<td>Age (days)</td>
<td>Weight (kg)</td>
<td>Diagnosis</td>
<td>Associated Conditions</td>
<td>Procedure</td>
<td>Recovery Notes</td>
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<td>Holt-Oram syndrome</td>
<td>One-patch I-PAB, pacemaker implantation</td>
<td>VSDs</td>
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<td>Postoperative E-CPR</td>
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<td>15</td>
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<td>21</td>
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<td>DILV, tricuspid atresia, TGA, pulmonary hypertension, dextrocardia</td>
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<td>One-patch I-PAB, atrioseptostomy</td>
<td>Univentricular palliation</td>
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<td>16</td>
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<td>51</td>
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<td>One-patch I-PAB</td>
<td>Univentricular palliation</td>
<td>6.5</td>
<td>Postoperative E-CPR</td>
<td>Debanding, BCPC, death (age 3 years; insufficient pulmonary blood flow/low systemic arterial saturation)</td>
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<td>17</td>
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<td>4 months</td>
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<td>Trisomy 21</td>
<td>Intracardiac inspection, one-patch I-PAB</td>
<td>Univentricular palliation</td>
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<td>None</td>
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<tr>
<td>18</td>
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<td>61</td>
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<td>AVSD</td>
<td>VACTERL association</td>
<td>One-patch I-PAB</td>
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<td>5</td>
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<tr>
<td>19</td>
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<td>50</td>
<td>3.3</td>
<td>Tricuspid atresia, pulmonary hypertension</td>
<td>None</td>
<td>One-patch I-PAB, atrioseptostomy</td>
<td>Univentricular palliation</td>
<td>5</td>
<td>None</td>
<td>Debanding, BCPC</td>
</tr>
<tr>
<td>20</td>
<td>Female</td>
<td>10</td>
<td>3.1</td>
<td>DILV, DORV, hypoplastic aortic arch</td>
<td>None</td>
<td>One-patch I-PAB, aortic arch reconstruction, atrioseptostomy</td>
<td>Univentricular palliation</td>
<td>5</td>
<td>Redo thoracotomy to control bleeding</td>
<td>Debanding, BCPC</td>
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<tr>
<td>21</td>
<td>Male</td>
<td>25</td>
<td>2.9</td>
<td>Unbalanced AVSD</td>
<td>Hirschsprung’s disease</td>
<td>One-patch I-PAB</td>
<td>Univentricular palliation</td>
<td>5</td>
<td>None</td>
<td>Death before debanding (age 6 months; SUID)</td>
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<td>22</td>
<td>Female</td>
<td>93</td>
<td>5.5</td>
<td>Swiss cheese VSDs, dextrocardia</td>
<td>None</td>
<td>One-patch I-PAB</td>
<td>VSDs</td>
<td>5.5</td>
<td>Redo thoracotomy to control bleeding</td>
<td>Waiting</td>
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<tr>
<td>23</td>
<td>Male</td>
<td>3.5 months</td>
<td>3.6</td>
<td>AVSD</td>
<td>Trisomy 21</td>
<td>One-patch I-PAB</td>
<td>AVSD, preterm birth</td>
<td>5</td>
<td>None</td>
<td>Debanding, biventricular repair, BCPC</td>
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<tr>
<td>24</td>
<td>Female</td>
<td>97</td>
<td>5</td>
<td>DORV, multiple VSDs</td>
<td>Malrotated kidney</td>
<td>One-patch I-PAB</td>
<td>VSDs</td>
<td>5.5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
</tr>
<tr>
<td>25</td>
<td>Male</td>
<td>61</td>
<td>3.3</td>
<td>VSD</td>
<td>Trisomy 21</td>
<td>One-patch I-PAB</td>
<td>VSD</td>
<td>5.5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
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<tr>
<td>26</td>
<td>Female</td>
<td>58</td>
<td>3.7</td>
<td>Swiss cheese VSDs</td>
<td>None</td>
<td>One-patch I-PAB, ASD closure</td>
<td>VSDs</td>
<td>5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
</tr>
<tr>
<td>27</td>
<td>Female</td>
<td>5</td>
<td>3</td>
<td>Simple TGA, VSD, hypoplastic aortic arch</td>
<td>None</td>
<td>Aortic arch reconstruction, arterial switch, hour glass I-PAB</td>
<td>2-stage aortic arch repair</td>
<td>5.5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
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<tr>
<td>28</td>
<td>Female</td>
<td>55</td>
<td>4.1</td>
<td>Multiple VSDs, pulmonary hypertension, cardiac failure</td>
<td>None</td>
<td>One-patch I-PAB</td>
<td>VSDs</td>
<td>5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
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</tbody>
</table>

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<table>
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<tr>
<th>Case Nr.</th>
<th>Sex</th>
<th>Age</th>
<th>Weight (kg)</th>
<th>Cardiac diagnosis</th>
<th>Extracardiac malformations</th>
<th>Performed surgery</th>
<th>I-PAB indication</th>
<th>Fenestration size (mm)</th>
<th>Postoperative complications</th>
<th>Long-term outcome</th>
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</thead>
<tbody>
<tr>
<td>29</td>
<td>Female</td>
<td>4.5 months</td>
<td>4</td>
<td>AVSD, s/p repair of aortic coarctation</td>
<td>Trisomy 21</td>
<td>One-patch I-PAB</td>
<td>AVSD</td>
<td>5</td>
<td>None</td>
<td>Debanding, AP-Shunt and BCPC take-down, death (age 1 years; electrolyte imbalance/arrhythmia)</td>
</tr>
<tr>
<td>30</td>
<td>Female</td>
<td>9 months</td>
<td>6.2</td>
<td>AVSD, suspected mitral valve straddling, pulmonary hypertension</td>
<td>Trisomy 21</td>
<td>Intracardiac inspection, one-patch I-PAB</td>
<td>VSD</td>
<td>5.5</td>
<td>None</td>
<td>Debanding, biventricular repair</td>
</tr>
<tr>
<td>31</td>
<td>Female</td>
<td>10 days</td>
<td>2.7</td>
<td>Borderline HLHS, VSD</td>
<td>Unbalanced translocation 1q/14q</td>
<td>One-patch I-PAB, aortic arch reconstruction</td>
<td>2-stage aortic arch repair</td>
<td>5.5</td>
<td>Redo thoracotomy due to symptomatic pericardial effusion</td>
<td>Waiting</td>
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<tr>
<td>32</td>
<td>Male</td>
<td>6 months</td>
<td>7.5</td>
<td>Swiss cheese VSDs</td>
<td>None</td>
<td>One-patch I-PAB, ASD closure, partial VSD closure</td>
<td>VSD</td>
<td>5</td>
<td>None</td>
<td>Waiting</td>
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</tbody>
</table>

The summarized fenestration size represents the size of the Hegar dilator, which could be passed through the central fenestration. LV: left ventricle; DORV: double outlet right ventricle; VSD: ventricular septal defect; cc-TGA: congenital corrected transposition of great arteries; TGA: transposition of great arteries; TR: tricuspid regurgitation; ASD: atrial septal defect; HLHS: hypoplastic left heart syndrome; s/p: status post; AVSD: atrioventricular septal defect; PAPVD: partial anomalous pulmonary vein drainage; DILV: double inlet left ventricle; DIRV: double inlet right ventricle; I-PAB: intraluminal pulmonary artery banding; NEC: necrotizing enterocolitis; CPR: cardiopulmonary resuscitation; E-CPR: extracorporeal cardiopulmonary resuscitation; BCPC: bidirectional cavopulmonary connection; TCPC: total cavopulmonary connection; SUID: sudden unexpected infant death; AP-Shunt: aortopulmonary shunt.
intraluminal pulmonary diameter according to Trusler’s rules was calculated as follows: intraluminal diameter (mm) = \{20 + \frac{\text{body weight (kg)}}{\pi}\} − 1 mm for biventricular and intraluminal diameter (mm) = \{24 + \frac{\text{body weight (kg)}}{\pi}\} − 1 mm for univentricular hearts. One mm was subtracted to eliminate the vessel wall thickness, and so the result represents the expected intraluminal diameter that can be compared with the central fenestration in I-PAB.

Statistical analysis

Continuous variables were expressed as means and standard deviations or as medians and ranges, according to their distribution. The continuous variables were compared with Student’s t test or the Mann–Whitney U-test, as appropriate. A P-value of <0.05 was considered statistically significant. Statistical analysis was performed with IBM® SPSS® Statistics Version 21 (IBM Corporation, Armonk, New York, NY, USA).

RESULTS

Patient demographics, morphological and procedural data, and outcome are presented in Table 1.

Patient characteristics

The study population consisted of 32 patients (20 females) with a median body weight of 3.7 kg (range: 2.6–13.0 kg). I-PAB was placed at a median age of 2 months (range: 5 days to 4 years). Eighteen patients (56%) had extracardiac malformations.

Surgery

Twenty-four patients received one-patch I-PAB, while the other 8 were managed by using the ‘hour glass’ technique. The median CPB time was 73 min (range: 20–315 min), with a median aortic
cross-clamp time of 25 min (range: 0–147 min). The median CPB time for a solitary I-PAB procedure was 54 min (range: 20–149, Case 16) and 81 min (range: 63–113) for one-patch and two-patch techniques, respectively. The lowest intraoperative body temperature was 32 ± 4°C. Concomitant cardiovascular procedures at the time of I-PAB were performed in 14 cases (44%). In other 18 patients, I-PAB was a solitary procedure: 4 of these (22%) represented an intraoperative decision after exploratory cardiotomy, while 2 patients required concomitant pacemaker implantation.

Outcome

Perioperative outcome. There was no hospital mortality. In all patients, echocardiographic control at hospital discharge demonstrated no evidence of pulmonary artery hypertension or distortion of the main pulmonary trunk. The median duration of hospital stay was 11 days (range: 5–51 days).

Two patients required venoarterial extracorporeal life support (ECLS) in association with postoperative cardiopulmonary resuscitation. In Case 16, the initially created 5.0 mm fenestration caused intraoperative systemic arterial saturation <50% after CPB discontinuation. Invasive measurement of distal pulmonary artery pressure showed 30% of the systemic arterial pressure. Thus, this patient was repeatedly put on CPB and a 6.5 mm fenestration was created. Eventually the patient was weaned from bypass but developed haemodynamic instability requiring ECLS on the first postoperative day. ECLS run and weaning after 7 days were uneventful, and he was discharged home on the 30th postoperative day. Another ECLS was implanted 8 h after surgery in Case 14 due to sudden haemodynamic instability requiring ECLS on the 21st postoperative day. ECLS run and weaning after 7 days were uneventful, and he was discharged home on the 30th postoperative day. Another ECLS was implanted 8 h after surgery in Case 14 due to sudden haemodynamic deterioration. The venoarterial ECLS was removed on postoperative day 6 and the patient was discharged home under stable conditions on postoperative day 21.

Inter-stage outcome. Two patients died before pulmonary artery debanding (Cases 4 and 21, Table 1) on postoperative days 133 and 160, respectively. Patient 4 had congenitally corrected transposition of great arteries with severe tricuspid regurgitation and congenital heart block. I-PAB was performed, along with pacemaker implantation. She died at home 45 months after surgery; a pacemaker dysfunction could not be completely ruled out. Patient 21 received I-PAB due to his unbalanced CAVSD. One month after this surgery, he required ileostomy to manage his Hirschsprung’s disease. Cardiac catheterization performed 4 months after I-PAB showed an adequate banding without pulmonary artery distortion. Unfortunately, he died unexpectedly 1 month later. In both cases, no autopsy was performed and, thus, a procedure-related cause of death could not be entirely excluded. Three patients are still with I-PAB awaiting further surgery.

Debanning. The median interval to debanding (n = 27) was 189 days (range: 112 days to 2.6 years). Definitive biventricular repair was achieved in 17 patients. In 10 patients, debanding was performed together with bidirectional cavopulmonary anastomosis, one of those needed to be taken down and replaced by a central aortopulmonary shunt (Case 29, Table 1).

One patient (Case 14) developed a mild kinking of the pulmonary artery bifurcation, as shown in the catheterization. Intraoperatively, it was observed that the I-PAB implantation was too proximal, causing a restriction in the anterior non-facing pulmonary sinus and a mild distortion of the pulmonary trunk bifurcation. The anterior sinus of the pulmonary root was then augmented using a bovine pericardial patch, resolving the associated problems as described earlier.

Another patient (Case 27) developed a stenosis in the proximal right pulmonary branch, requiring a patch augmentation in addition to the debanding procedure. This proximal branch stenosis is assumed to be closely related to the arterial switch operation with the Lecompte manoeuvre, which was simultaneously performed with ‘hour glass’ I-PAB.

Late outcome. The follow-up was completed with a median duration of 2.9 years (range: 133 days to 8.2 years). There were 3 late deaths after pulmonary artery debanding and further univentricular palliation (Cases 8, 16 and 29, Table 1). Kaplan–Meier survival rates after 1 and 5 years were 86.7 ± 6% and 81.6 ± 8%, respectively (Fig. 3). No patient experienced late pulmonary artery distortion or pulmonary valve impairment.

Balloon dilatation. Four patients (Cases 6, 8, 11 and 32) received balloon dilatation of I-PAB resulting in a decreasing pressure gradient over I-PAB. There were no substantial changes on room air saturation. Balloon dilatation was followed by biventricular repair after 2 weeks in Patient 6 and by bidirectional Glenn palliation after 2 months in Patient 8. Patient 11 received balloon dilatation of I-PAB due to suprasystemic pressure in the subpulmonary located, morphologic left ventricle. Seven months later, we proceeded with unproblematic removal of I-PAB and the definitive biventricular repair. Patient 32 is still awaiting definitive biventricular repair.

Haemodynamic evaluation

The systemic arterial saturation at the end of surgery ranged from 75 to 100%, with a trend towards lower saturation in patients with a univentricular morphology (P = 0.22). This saturation was measured after CPB discontinuation with a 50% fraction of inspired oxygen.

The pressure gradient across I-PAB increased steadily over time (P < 0.01) in uni- and biventricular patients and following the one-patch technique. However, this difference was not statistically...
significant in the two-patch technique. The mean systemic arterial saturation showed no statistically significant alteration (Fig. 4A and B).

No significant differences in both systemic arterial saturation and pressure gradient were calculated between the one-patch and two-patch techniques (Fig. 4A), and between uni- and biventricular hearts at any given time (Fig. 4B).

**Fenestration size**

There was no significant difference in fenestration size between uni- and biventricular hearts, but patients with univentricular hearts tended to have bigger fenestration for a given body weight. The mean fenestration size and the mean weight in univentricular

**Figure 4:** (A and B) Pressure gradient over I-PAB and systemic arterial saturation over time. The pressure gradient across I-PAB is typically increased steadily over time, while the mean systemic arterial saturation showed no statistically significant alteration. (A) There was no significant difference in both systemic saturation and pressure gradient between the one-patch and two-patch techniques at any given time. (B) There was no significant difference in both systemic saturation and pressure gradient between uni- and biventricular hearts at any given time.
ventricular and biventricular hearts, respectively (Fig. 1B). Additionally, I-PAB has an exactly determined implantation that causes pulmonary root impairment, but offers less diameter reduction of the previously banded pulmonary artery after debanding. Despite these efforts, a residual pressure gradient caused by the posterior fibrotic shelf might persist. I-PAB, on the other hand, does not cause a ‘snaring’ effect and offers less diameter reduction of the previously banded pulmonary artery (Fig. 1B). Additionally, I-PAB has an exactly defined orifice and is circularly sutured to the lumen of the pulmonary artery, producing practically no risk of band migration and vessel erosion. In our study, only 1 of 27 patients required I-PAB-related pulmonary patch plasty at the debanding procedure.

Pulmonary artery banding: how ‘tight’ should it be?

In tightening extraluminal PAB, Trusler’s rules [11] are used as a guide followed by finer adjustments, considering systemic saturation, pulmonary artery pressure distal to the PAB and visual cardiac pump performance [14]. However, band tightening is especially challenging in patients who have freshly been weaned from CPB. A potential benefit of I-PAB lies in its application to situations in which CPB must be used for concomitant cardiovascular corrections [9, 10]. The challenge in obtaining an appropriate size of fenestration and assessing its haemodynamic effect might be better understood by considering the Hagen–Poiseuille equation, by which the blood volume flow rate is proportionally related to the pressure gradient and to the fourth power of the vessel radius. In theory, small changes in fenestration size have a large impact on the flow and pressure gradient through the I-PAB.

Our fenestration diameter was always below the calculated intraluminal pulmonary artery diameter according to Trusler’s formulae. The difference is statistically significant, both for uni- and biventricular hearts. This might be explained from the vessel wall thickness and the thickness of vessel wall folds produced by the external band. Vessel wall folds result in smaller cross-sectional area for the same vessel circumference. However, vessel wall folds in extraluminal PAB cause a lower repetitive accuracy and, thus, it is very difficult to predict the cross-sectional area for a given reduction of the outer vessel size. The degree of cross-sectional area reduction also depends on the vessel wall thickness: the thicker the vessel wall, the more fold thickness will be acquired from extraluminal banding and, so, more reduction of cross-sectional area will be achieved. Vessel wall thickness, vessel wall folds and the use of a large width band in extraluminal PAB could cause different haemodynamic effects when compared with a central circular aperture in I-PAB.

Locke et al. [9] reported the one-patch I-PAB technique in 32 patients, all with concomitant cardiac surgery. Piluiko et al. [10] also described a similar I-PAB technique in their series of 18 patients. Early and long-term survival in those studies ranged 69–88%, and 50–61%, respectively, with low cardiac output as the most common cause of death [9, 10]. There was no early mortality in our study; however, postoperative ECLS was needed in two patients, both due to low cardiac output. When compared with previously described studies, our long-term survival rate was higher (81.6%). We assumed that this result might be related to a higher prevalence of I-PAB as a solitary procedure, which was performed in 18 patients (56%) in our cohort when compared with none [9] and 5 patients (28%) [10] in previous mentioned studies.

However, selection of fenestration size could also have influenced postoperative outcome. Along with creation of an unrestricted aortopulmonary window, Locke et al. [9] chose a central fenestration size of 2–3 mm for neonates, 3–4 mm for infants, 4–5 mm for older children and 5–8 mm for adults. Without an aortopulmonary window, I-PAB fenestration size was half of the diameter of the aortic annulus. On the other hand, Piluiko et al. [10] reported a fenestration size of 3.6 mm for patients with 3–3.5 kg body weight and 4 mm for patients weighing >3.5 kg. In both series, no difference is made between the patients with uni- or biventricular hearts [9, 10]. When compared with both studies, our I-PAB fenestration size was larger for a given body weight, especially in univentricular patients. Only 2 patients in our series received a 4 mm central fenestration (Cases 14 and 15), which was well tolerated in the latter. However, 4 mm might have been too small in Case 14, resulting in acute cardiac decompensation and low cardiac output with the requirement for ECLS. On the other hand, the low-output syndrome in Case 14 might also have been related to the perioperative pulmonary hypertensive crisis or suboptimal I-PAB implantation that causes pulmonary root impairment and pulmonary artery distortion seen at the debanding. The largest I-PAB diameter used in our study was 6.5 mm in a patient who then required ECLS due to persistent congestive heart failure. We tried to review our fenestration sizes in relation to aortic and pulmonary valve diameter; however, no correlation or ‘formula’ was identified. According to our results, we would suggest that a fenestration size between 5 and 6 mm should be appropriate for both uni- and biventricular patients.
Haemodynamics after intraluminal pulmonary artery banding

In our experience, pressure gradients over I-PAB significantly increased over time, while the alteration of systemic arterial saturation was not statistically significant, regardless of patient pathophysiology. CPB, hypothermia, mechanical ventilation, open thorax and anaesthesia are known to have impacts on intraoperatively measured pressure gradient over I-PAB. In addition, if high pulmonary vascular resistance exists, the pressure gradient over I-PAB is lower. In our study, the intraoperative pressure gradient over the I-PAB might seem low as we also avoid excessive ventricular afterload after discontinuing CPB. This pressure gradient typically increased steadily over the hospital stay as the patients recovered from surgery. The echocardiographic control before hospital discharge showed a mean pressure gradient of 65 ± 14 and 64 ± 15 mmHg for uni- and biventricular hearts, respectively. Both were considered as adequate banding. The ongoing pulmonary recovery also played a role in maintaining stable systemic saturation and reduced oxygen demand.

During the follow-up period before debanding, decrease of pulmonary vascular resistance and vessel growth can further increase the pressure gradient over I-PAB. Despite this continuously increased pressure gradient, the systemic arterial saturation still remained steady. Although not detected in catheterization, this might be partly caused by the development of small systemic-pulmonary collaterals. Therefore, we would encourage balloon dilatation not only in patients with systemic arterial desaturation but also in patients with suprasystemic right ventricular pressure and a high pressure gradient across the band.

Balloon dilatation of intraluminal pulmonary artery banding

After PAB, some authors suggested a catheter-based procedure to perform progressive band loosening. Brown et al. [15] reported a concept of dilatatable extraluminal PAB, in which the band is progressively tightened with series of surgical clips. This way, the band can be balloon dilated stepwise until complete release, if required. Still, the design of such PAB carries the risk of band migration, especially at the time of balloon dilatation. In contrast, the I-PAB fenestration could be safely and efficiently balloon dilated without the risk of migration and its related consequences.

Still, ‘tightening’ both conventional extraluminal and intraluminal PABs require reoperation. Although constriction of a two-patch I-PAB could ideally be done without CPB, ‘tightening’ of one-patch I-PAB would be a more complex undertaking requiring CPB. Corno suggested FloWatch-PAB® (EndoArt, Lausanne, Switzerland), an implantable and telemetrically adjustable, battery-free device [16]. The FloWatch-PAB® uses the concept of progressive inflation or deflation of a silicone membrane to tighten or loosen the pulmonary artery constriction. Still, despite the advantage of avoiding repeated surgeries to adjust the band, works in the literature showed some important drawbacks. Injury to the pulmonary artery wall could cause pseudoaneurysm [17] and telemetric ‘tightening’ of the band could damage the pulmonary valve [18] and pulmonary branch orifices.

The techniques of intraluminal pulmonary artery banding

At the beginning of the study period, ‘hour glass’ I-PAB was used to restrict pulmonary blood flow. The pulmonary trunk division offers a better exposure of the posterior wall without the vessel wall fold as seen in the one-patch technique. The connected part of both patches could be tightened externally without using CPB, if required. This connection area also eased the debanding procedure, reducing the risk of performing pulmonary arteriotomy at the incorrect level. A two-patch I-PAB was also preferred in patients who underwent an arterial switch procedure with Lecompte’s manoeuvre. This technique adds vessel length, minimizing tension in the pulmonary artery and within the surgically reconstructed pulmonary root. However, despite using the two-patch technique, Patient 29 required a patch augmentation of the right pulmonary branch. This might be closely related either to the surgical reconstruction technique of the pulmonary root or to the performed Lecompte manoeuvre, since stenosis of the pulmonary arteries is a common complication after the arterial switch procedure, with a reported incidence rate of 7–40% [19]. The pressure gradient across two-patch I-PAB increased steadily over time, but the difference was not reaching statistical significance at hospital discharge (Fig. 4A), which might be caused by the small patient cohort.

Since late 2008, the one-patch I-PAB technique has been favoured. This technique requires only a transverse pulmonary arteriotomy and no pulmonary trunk division. It contains only one level of running suture, and thus the I-PAB procedure could be performed faster.

Major drawbacks of intraluminal pulmonary artery banding

The requirement for CPB and the limited intraoperative fine adjustment of I-PAB are significant disadvantages of this technique, when compared with extraluminal PAB. The central fenestration might also carry a risk of thrombotic occlusion, which we never observed under daily aspirin therapy. In addition, there is a possibility of bleeding from the pulmonary arteriotomy. Iannucci et al. [20] reported a case of profound haemolytic anaemia associated to the creation of I-PAB. After reviewing our data, no evidence of severe haemolytic anaemia or I-PAB clogging was observed.

Study limitations

The present analysis shares the limitations of a retrospective review undertaken in a non-randomized uncontrolled patient population with a differing duration and intensity of the follow-up. Due to the small number of study patients and a relatively heterogeneous assortment of heart defects, postoperative courses were only partially comparable and the study population may not be large enough to pick up a statistically significant difference.

CONCLUSION

The present study shows that I-PAB can be performed in neonates and infants with low morbidity. This technique offers a precise and predictable restriction of pulmonary blood flow. Our data demonstrated a progressive increase of the pressure gradient over time and a well-maintained systemic arterial saturation. Balloon dilatation of I-PAB to reduce the pressure gradient and prolong the palliation period is safe and efficient. Therefore, despite...
requiring CPB, I-PAB is our institutional preference for children who require pulmonary artery banding.

Conflict of interest: none declared.

REFERENCES