INTERVENTIONAL CARDIOLOGY AND SURGERY

Implantation of endovascular stents for the obstructive right ventricular outflow tract

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Objectives: To evaluate the effectiveness and long term outcomes of catheter intervention for obstructive conduits between the right ventricle and pulmonary arteries.
Design: Retrospective chart review.
Setting: Tertiary care paediatric cardiology unit.
Patients and interventions: 70 procedures in 68 children (median age at intervention 6 years, median interval after conduit insertion 3.4 years) were analysed. All children had haemodynamic indications for conduit replacement. Twenty four children underwent a second intervention (stent dilatation in 17, second stent implantation in seven).
Results: Mean (SD) conduit pressure gradient decreased from 44 (18) mm Hg to 18 (12) mm Hg at the initial intervention (n = 62, $p < 0.001$) and from 39 (15) mm Hg to 23 (10) mm Hg at the second intervention (n = 16, $p < 0.001$). The percentage of the predicted right ventricular outflow area increased from 17 (9)% to 44 (22)% at the initial intervention (n = 62, $p < 0.001$) and from 24 (8)% to 29 (11)% at the second intervention (n = 21, $p < 0.001$). The conduit was subsequently replaced in 33 children. Freedom from conduit replacement from the time of stent implantation was 83%, 75%, and 47% at one, two, and five years, respectively, and from the time of the initial conduit surgery it was 87%, 64%, and 42% at five, eight, and 10 years, respectively. Body growth was maintained, no deaths were reported during follow up, and pulmonary insufficiency was well tolerated.
Conclusion: A catheter treatment strategy for obstructive conduits is safe and effective in prolonging conduit function.

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Extracardiac conduits placed between the right ventricle (RV) and pulmonary arteries (PAs) are used for a variety of congenital heart lesions. However, progressive conduit obstruction is likely and is the single most common factor limiting conduit durability. Balloon angioplasty alone has resulted in only partial relief of obstruction and frequent complications.[1–4] Almagor and colleagues[5] first reported success of stent implantation in an animal model of such obstructive conduits. Subsequent clinical studies showed that stent implantation relieved stenosis and prolonged conduit life span.[6–14] In the present study, we report medium term outcomes, effectiveness, and clinical impact of this catheter based management strategy.

METHODS

Patient characteristics

Since 1990 transcatheter stent implantation for the RV outflow obstruction has been offered to children with RV to PA conduits when surgical replacement was considered necessary for management of significant obstruction. The indication for intervention was an RV pressure $\geq$ = 3/4 of systemic arterial pressure on echocardiography, with or without the presence of symptoms. Clinical data were obtained from the computerised cardiac database at The Hospital for Sick Children, Toronto, of all children who underwent conduit stent implantation. Angiograms and haemodynamic data from studies before and after implantation were reviewed as were clinical data from the medical record. Between October 1990 and April 2002, 70 children underwent attempted transcatheter stent implantation. Of those, four children also had a second stenting procedure within a new conduit after surgical replacement of a previously stented conduit, for a total of 74 procedures. Four procedures were excluded from data analysis because of the failure of primary implantation (details described below). In six children (see below), additional pulmonary or systemic artery obstructive lesions were addressed at catheterisation, which may have affected the RV to systemic arterial pressure ratio. As such patients were few, however, they were included in the analysed cohort of 70 procedures in 68 children. Forty four children were boys and 24 were girls. Underlying cardiac diagnoses consisted of pulmonary atresia with ventricular septal defect (n = 20), tetralogy of Fallot (n = 16), common arterial trunk (n = 15), transposition of the great arteries with ventricular septal defect (n = 8), double outlet RV (n = 6), and aortic valve stenosis managed with a Ross procedure (n = 3).

Forty three children (61%) had undergone at least one previous cardiac operation before conduit insertion and 18 (26%) had had conduits replaced at least once before stent implantation. Conduit types were pulmonary (n = 27) or aortic homografts (n = 23), Polystan valved conduits (n = 8), conduits with Symbion or Hancock porcine valves (n = 6), a monocusp patch in a pulmonary homograft (n = 3), a Carpentier-Edwards valve in a previously inserted pulmonary homograft (n = 1), and simple outflow tract enlargement with a patch in a previously inserted pulmonary homograft (n = 2).

Technique

The technique of implantation has been described in detail previously.[13] Palmaz stents of various lengths (Johnson &

Abbreviations: PA, pulmonary artery; RV, right ventricle; RVOT, right ventricular outflow tract

Johnson Interventional System, Warren, New Jersey, USA) were used based on the diameter and length of the obstruction and the size of the conduit. Multiple and overlapping stents were placed for long segment stenoses. The stents were mounted on to angioplasty balloon catheters (from 8–23 mm in diameter, ⩾ 10% larger than the initial conduit diameter). Generally, the conduit was not first balloon dilated, unless the conduit was heavily calcified and then only to determine whether the lesion was expandable. Informed parental consent was obtained before all procedures.

Data collection

Haemodynamic data were reviewed before and after any catheter intervention. Angiographically, the form of conduit stenosis was classified as supravalvar, valvar, subvalvar, diffusely small, or calcified. The minimum diameter of the conduit was measured in the anteroposterior and lateral projections before and after all interventions. The perimeter and area of the conduit lesion were based on the assumption that conduit geometry was ellipsoid (perimeter $= \pi \times ((a^2 + b^2)/2)^{1/2} - (a-b)^2/8.8$; area $= \pi \times ab$). Cross sectional area was expressed as the percentage of normal RV outflow tract (RVOT) area adjusted for body surface area.[15] Technical difficulties, procedural complications, and fluoroscopy time were recorded.

Echocardiographic data (RV pressures and end diastolic dimensions) and the ratio of RV to systemic blood pressure assessed before and after the intervention and at the latest follow up or before surgical conduit replacement were recorded. The RV end diastolic dimension was expressed as a percentage of the predicted normal value and corrected for body surface area.[15]

Data analysis

Clinical and procedural characteristics are described as frequencies, median with range, and mean (SD) as appropriate. The data were analysed with StatView for Macintosh, release 5.0 (SAS Institute Inc, Cary, North Carolina, USA). Differences in clinical characteristics between children with and without a second intervention were tested with the Mann-Whitney U test. Changes in haemodynamic parameters and vascular dimensions were tested with paired or unpaired *t* tests. Freedom from surgical conduit replacement was estimated by Kaplan-Meier analysis. Differences in freedom from surgical conduit replacement between the children with and without reintervention were tested with the log rank test. Cox's proportional hazards regression model was used to assess multivariate associations. Significance was defined as $p < 0.05$.

RESULTS

Primary procedure

The median age at catheterisation was 6 years (range 6 months to 18 years), with a median interval from conduit insertion of 3.4 years (range 3 months to 14 years). The median weight was 21 kg (range 5.2–86 kg) and height (n = 69) 117 cm (range 63–177 cm). Before the procedure, 17 children had symptoms of increasing fatigue, more than moderate exercise intolerance, or chest pain.

Angiographically, conduit stenosis was caused by combined supravalvar and valvar lesions in 19 children, isolated valvar stenosis in 28, subvalvar in 5, or a diffusely small conduit in 18 children. Extensive intramural calcification was observed in 10 children.

Of the 70 procedures 65 required only a single stent, four procedures required two stents to be placed, and in one procedure three stents were implanted. The sizes of the stents were P128 and P188 in one child each, P204 in 13 procedures in 12 children, P308 in 41 procedures in 37 children, P4014 in 14 procedures in 13 children, and P5015 in six children. Multiple stents were deployed in one child to strengthen a previously stented area and overlapping stents extended lesions in four children. The median balloon diameter was 15 mm (range 8–23 mm), with the stent crossing the conduit valve in all cases. A flaring technique at the end of the stent was used in five procedures. Mean fluoroscopy time was 32 (25) minutes (range 8–79 minutes, n = 62). Additional procedures were stent insertion within the right PA in two children, within the left PA in three, and across an aortic coarctation in one child.

Haemodynamic and angiographic data

Table 1 shows haemodynamic and angiographic data before and after implantation. In univariate analysis, a younger age at surgery ($p < 0.01$), a larger percentage predicted RVOT area before catheter intervention ($p < 0.001$), and a larger ratio of balloon diameter to predicted RVOT diameter ($p < 0.001$) were significantly associated with a lower conduit gradient after intervention. Age at catheter intervention, interval from conduit insertion to catheter intervention, sex, body weight, and RV pressure before the procedure were not predictive of the pressure gradient after the intervention. In stepwise analysis, a larger percentage predicted RVOT area before catheter intervention and a larger ratio of balloon diameter to predicted RVOT diameter were independently associated with a lower gradient after the implantation. Figure 1 presents the correlation between balloon diameter (as a percentage of predicted RVOT diameter) and pressure conduit gradient after implantation. Stents implanted with balloon diameters ⩾ 100% of the predicted RVOT diameter had conduit gradients after implantation of < 15 mm Hg.

Table 1 Haemodynamic and angiographic data at primary catheter intervention

	Before stenting	After stenting	Change (%)	p Value*
Haemodynamic variables at catheterisation				
RV pressure (mm Hg)	66 (17)	45 (13)	−30 (20)	<0.001 (n=69)
PA pressure (mm Hg)	23 (7)	27 (10)	22 (35)	<0.001 (n=62)
RVOT peak to peak systolic gradient (mm Hg)	44 (18)	18 (12)	−55 (24)	<0.001 (n=62)
Ratio of RV to systemic arterial systolic pressure	0.75 (0.19)	0.47 (0.14)	−35 (18)	<0.001 (n=69)
Angiography				
Minimum diameter conduit in AP view (mm)	7.5 (2.30	11.1 (3.2)	53 (45)	<0.001 (n=66)
Minimum diameter conduit in LAT view (mm)	6.7 (2.7)	11.2 (3.4)	82 (66)	<0.001 (n=65)
Minimum perimeter of conduit (mm)	21.8 (6.5)	34.5 (8.7)	63 (37)	<0.001 (n=64)
Percentage predicted RVOT area adjusted for BSA	17 (9)	44 (22)	190 (169)	<0.001 (n=62)

Data are mean (SD).
*From paired *t* tests.
AP view, anteroposterior angiographic projection; BSA, body surface area; LAT view, lateral angiographic projection; PA, pulmonary artery; RV, right ventricle; RVOT, right ventricular outflow tract.

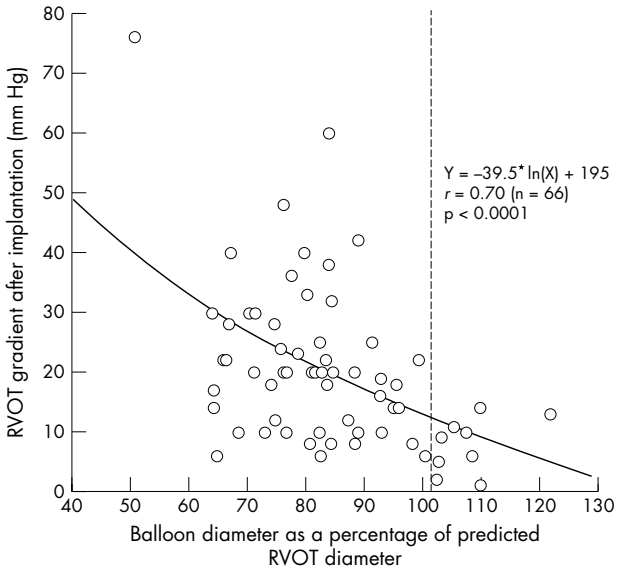

Figure 1 Relation between balloon diameter as a percentage of predicted right ventricular outflow tract (RVOT) diameter and pressure gradient across the conduit after stent implantation.

Technical difficulties and complications

Stent implantation failed in four children. In one child, the stent slipped over the catheter into the RV cavity and was surgically removed and the conduit was replaced. In another child a partially inflated stent migrated into the right PA and was retrieved and deployed within the right iliac vein. After a stent was successfully implanted in another child, a persistent stenosis was present at the proximal end of the stent and a second stent was implanted but was incompletely opened because of balloon rupture. The stent was removed and deployed within the right iliac vein. In the remaining child, the stent and partially deflated balloon were entrapped within the right PA and were surgically removed at the time of conduit replacement. In an additional five children, the delivery balloon ruptured during inflation, necessitating uneventful balloon catheter exchange.

Second catheter intervention

Twenty four of the original 70 children had a second catheter intervention because of a recurrent increase in RV pressure ($\geq$ 3/4 systemic arterial pressure). The primary mechanism of recurrent obstruction was stent compression (anterior to posterior) or reduced predicted RV outflow area in the absence of areas of intimal ingrowth.

Table 2 shows a comparison between children with and without a second catheterisation. Although the age at conduit insertion was similar between groups, children who had a second intervention where significantly younger at the initial catheter intervention ($p < 0.05$), performed at a median of 1.4 years (range 2 months to 10 years) after the initial implant, and the percentage predicted RVOT area after the initial stent implantation was significantly less ($p < 0.05$). In 17 of these children, the previously implanted stent was balloon dilated (median balloon diameter 15 mm, range 10–16 mm) and in the remainder an additional stent was implanted. There were no significant differences between the age at the first or second catheterisation, RV pressure, RVOT pressure gradient, or percentage predicted RVOT area before catheter reintervention between children who had balloon angioplasty and children with a second stent implantation. A stent was implanted within a previous stent (stent in stent) in two, at the proximal end of a previous stent (overlapping) in three, and at the distal end of a previous stent (overlapping) in two children. Mean fluoroscopy time for the procedure (n = 21) was 25 (12) minutes (range 6–46 minutes).

Haemodynamic and angiographic data at second catheterisation

Table 3 shows the haemodynamic data before and after reintervention. In univariate analysis, greater ratio of balloon to predicted RVOT diameters ($p < 0.001$), a larger percentage predicted RVOT area ($p < 0.05$), and a lower ratio of the RV to systemic arterial pressure ($p < 0.05$) before reintervention were significantly associated with a lower conduit gradient after reintervention. Age at conduit placement and catheter reintervention, sex, weight, and pressure gradient across the conduit before reintervention were not predictive of the conduit gradient after catheter reintervention. In stepwise analysis, only a larger balloon to predicted RVOT diameter was independently associated with a lower pressure gradient. There were no haemodynamic or angiographic differences between children who had a second stent and those only balloon dilated. No technical difficulties or complication were recorded.

Surgical reintervention

Over the course of this experience, the conduit was surgically replaced in 33 children. The criteria for conduit replacement were the same as for catheter intervention but also included failure of the initial or attempted catheter (re)intervention. The median interval from the initial catheter intervention to conduit replacement was 2.1 years (range 3 weeks to 10.3 years) and the median interval from the second catheter intervention to conduit replacement (n = 14) was 10 months (range 2 months to 3.8 years). No technical difficulties were reported at the time of surgery. One child underwent heart transplantation shortly after conduit replacement because of acute cardiac deterioration, one child died in the perioperative period of sepsis, and one child died suddenly four years after surgery of a presumed arrhythmia.

Table 2 Comparison between children with and without a second catheter intervention

	No reintervention (n = 46)	Reintervention (n = 24)	p Value*
Age at conduit insertion (years)	3 (0 months to 16 years)	3 (2 months to 8 years)	0.62†
Data at initial catheter intervention			
Age (years)	8 (7 months to 18 years)	6 (10 months to 13 years)	<0.05†
Balloon diameter as percentage of predicted RVOT diameter	83 (13) (n = 43)	82 (20)	0.29
Study after initial intervention			
Percentage of predicted RVOT area	49 (27) (n = 43)	36 (14) (n = 22)	<0.05
RV pressure (mm Hg)	43 (12) (n = 45)	47 (15)	0.19
RVOT peak to peak systolic gradient (mm Hg)	16 (9) (n = 43)	21 (15) (n = 21)	0.07
Ratio of RV to systemic arterial systolic pressure	0.62 (0.17) (n = 45)	0.68 (0.22)	0.34

Data are median (range) or mean (SD).
*From unpaired *t* tests; †Mann-Whitney U test.

Table 3 Haemodynamic and angiographic data at second catheter intervention

	Before reintervention	After reintervention	Change (%)	p Value
Haemodynamic variables at catheterisation				
RV pressure (mm Hg)	63 (15)	49 (11)	−19 (14)	<0.001 (n=23)
PA pressure (mm Hg)	25 (7)	25 (5)	7 (20)	0.41 (n=15)
RVOT peak to peak systolic gradient (mm Hg)	39 (15)	23 (10)	−37 (20)	<0.001 (n=16)
Ratio of RV to systemic arterial systolic pressure	0.70 (0.19)	0.52 (0.14)	−21 (16)	<0.001 (n=22)
Angiography				
Minimum diameter conduit in AP view (mm)	9.7 (2.1)	10.0 (2.3)	8 (20)	<0.01 (n=21)
Minimum diameter conduit in LAT view (mm)	7.8 (1.9)	9.0 (2.0)	20 (19)	<0.001 (n=23)
Minimum perimeter of conduit (mm)	27.1 (4.8)	29.8 (6.1)	14 (13)	<0.001 (n=21)
Percentage predicted RVOT area adjusted for BSA	24 (8)	29 (11)	31 (32)	<0.001 (n=21)

Data are mean (SD).
*From paired *t* tests.

Follow up

The median follow up for the 37 children who have not required conduit replacement was 2.6 years (range 6 months to 8.6 years). No deaths were related to the catheter procedure. Stent fracture was noted in two children, in one of whom a portion of the stent migrated to the right PA and was stabilised by balloon angioplasty.

Body growth was maintained, with a median weight gain of 7 kg (range 0.6–36 kg, n = 62) and a height increase of 14 cm (range 0–64 cm, n = 31). Mean weight velocity was 3.7 (2.8) kg/year (n = 61) and mean height velocity was 5.5 (3.0) cm/year (n = 31).

To determine the acute effect of the transcatheter intervention on RV size, RV end diastolic dimensions were compared before and after stent implantation (median 3 months after the procedure, range 0 days to 11 months, 22 (7) *v* 20 (5) mm, respectively, n = 32, p = 0.54); no differences were found. In the long term, RV end diastolic dimensions were compared before intervention and at the latest assessment before conduit replacement (median 2.3 years after the procedure, range 4 months to 10 years, 192 (52)% *v* 188 (44)%, respectively, n = 50, p = 0.54) and were not significant different. Additionally, RV dimensions at the latest echocardiographic assessment did not correlate with the interval from stent implantation (n = 50, *r* = 0.3, p = 0.50).

Freedom from conduit replacement

Kaplan-Meier freedom from conduit replacement after stent implantation was 87% at five years, 64% at eight years, 42% at 10 years, and 17% at 15 years (fig 2). Further, analysis noted freedom from conduit replacement from the time of stent implantation of 83% at one year, 75% at two years, 47% at five years, and 18% at eight years (fig 3). Cox's proportionate hazard regression modelling showed that a decreased time from the initial catheter intervention to conduit replacement was significantly associated with a higher RVOT gradient before ($p < 0.05$) and a lower percentage of predicted RVOT area after implantation ($p < 0.05$). No additional factors, whether clinical characteristics, haemodynamic function, or angiographic variables, were associated with a reduced time from initial stent implantation to conduit replacement. In stepwise analysis, a higher RVOT gradient before stent implantation was independently associated with a reduced time from initial stent implantation to conduit replacement. Freedom from conduit replacement from the time of conduit insertion did not differ between children having a second catheter intervention and children not requiring a second procedure (p = 0.18, by log rank test), although the initial stent was implanted significantly earlier in children who required reintervention than in those who did not ($p < 0.05$, Mann-Whitney U test).

Eight children had the conduit replaced < 1 year after the initial implantation. Table 4 shows a comparison between these children and those whose conduit was not replaced for > 1 year. Age at conduit insertion and haemodynamic data before stent implantation were not significantly different

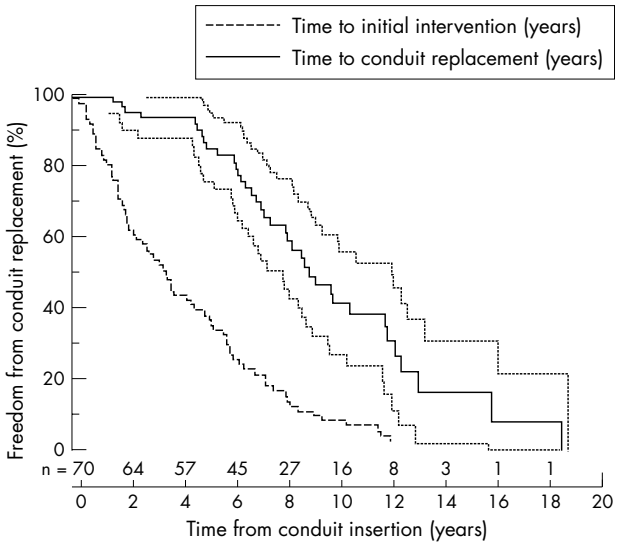

Figure 2 Kaplan-Meier plot of freedom from conduit replacement after conduit insertion. Dotted lines indicate 95% confidence interval.

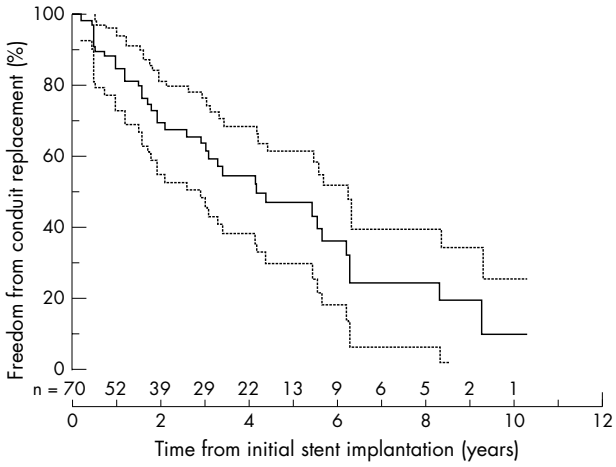

Figure 3 Kaplan-Meier plot of freedom from conduit replacement after initial stent implantation. Dotted lines indicate 95% confidence interval.

Table 4 Comparison between children who did and did not require conduit replacement <1 year after stent implantation

	Conduit replacement		
	<1 year (n=8)	>1 year (n=51)	p Value*
Age at conduit insertion (years)	4 (6 months to 15 years)	3 (1 day to 9 years)	0.19†
Data at initial catheter intervention			
Age (years)	11 (4–16 years)	6 (7 months to 19 years)	<0.05†
Balloon diameter as percentage of predicted RVOT diameter	72 (13)	85 (13) (n=49)	<0.05
Before catheter intervention			
Percentage predicted RVOT area for BSA	10 (3) (n=7)	17 (7) (n=47)	<0.05
RV pressure (mm Hg)	65 (18)	69 (17)	0.56
RVOT peak to peak systolic gradient (mm Hg)	47 (18)	47 (17) (n=46)	0.99
Ratio of RV to systemic arterial systolic pressure	0.76 (0.20)	0.77 (0.20)	0.90
After catheter intervention			
Percentage predicted RVOT area for BSA	22 (10) (n=7)	43 (18) (n=48)	<0.01
RV pressure (mm Hg)	48 (22)	46 (11)	0.64
RVOT peak to peak systolic gradient (mm Hg)	32 (21)	19 (12) (n=49)	<0.05
Ratio of RV to systemic arterial systolic pressure	0.54 (0.19)	0.47 (0.11) (n=49)	0.18

Data are median (range) or mean (SD).
*From unpaired t tests; *Mann-Whitney U test.

between groups but children who required conduit replacement were significantly older than those who did not. Additionally, before intervention the percentage predicted RVOT area and balloon diameter to predicted RVOT diameter in children with earlier conduit replacement were significantly lower. In four children, a significant waist was observed at the valve level caused by severe calcification during stent implantation, and in two children the stents could not fully expand because of diffusely narrow conduits.

DISCUSSION

These data show that stent implantation across a stenotic conduit greatly relieves the obstruction and postpones the necessity of surgical conduit replacement. Furthermore, conduit replacement was postponed to the same extent in children who had a second intervention for a previously stented conduit that had deteriorated early and in the overall group.

Our study showed that the minimum diameter of the conduit was smaller in the lateral projection than in anteroposterior projection before stent implantation due to anterior sternal compression and by cardiac structures posterior to the conduit, such as the ascending aorta. After stent implantation, diameters in the anteroposterior and lateral projections were similar. As such, the stent implantation was effective because it increased the cross sectional geometry from an ellipse to a circle, functionally removing the degenerated obstructive valve, dilating the associated anastomotic stenosis, and thickening the neointima within the conduit.

After the second procedure, the incremental increase in both RVOT perimeter and area was smaller than after the initial intervention. Dimensional area changed from ellipsoid to circular, which appears to be the mechanism of effectiveness. This was similarly observed by Schneider and colleagues,[16] who reported that 25% of second catheter procedures for restenosis after stent implantation were done because of anteroposterior external compression. In this regard, catheter reintervention appears to be useful only for children who have compressed conduits or those who had inadequate balloon diameters used at the initial catheter intervention. Additional stent implantation is also applicable in this setting if recoil occurs from external compressive forces, stenosis develops at the margins of the stent, or a fracture occurs that can be reinforced or stabilised.

The children who had a balloon diameter greater than the predicted RVOT diameter had excellent obstruction relief. However, in most cases the size of the balloon was chosen based on the original conduit diameter and effectiveness was dependent on the functional diameter of the conduit at the initial implantation. Effectiveness was reduced, as shown by early surgical conduit replacement (< 1 year), in those children who were older at their initial catheter intervention (11 years *v* 6 years) (table 4). Such children have outgrown the conduit outflow diameter, even when it is improved by stent implantation, and may not be good candidates for this management strategy.

Kaplan-Meier analysis showed that the overall freedom from conduit replacement from the time of conduit insertion for the entire cohort was significant (87% at five years, 64% at eight years). Thus, surgical conduit replacement could be postponed as shown by the rightward shift of the curve denoting the time when surgery would have been performed if the catheter intervention was not performed (fig 2). This was further underscored by the time course from stenting to eventual conduit surgery, extending conduit life in 50% of children by five years (fig 3).

Further examination of the Kaplan-Meier curve shows an increased rate of conduit failure (fig 3) within the first year after stenting. Comparing children with and without early conduit replacement, the balloon to predicted RVOT diameter was smaller and the age at catheter intervention was older.

Additionally, in six children, stents could not be sufficiently deployed because of a severely calcified valve or a diffusely small conduit. A calcified conduit in an older child is not likely to respond to a stent management strategy. In such children, balloon dilatation is performed to determine whether the conduit is distensible, the balloon will not rupture during inflation, and the achieved balloon diameter is at least 45% of the predicted RVOT diameter. One would then anticipate 50% of the children achieving a further three to four years with the conduit.

Follow up

Stent implantation results in free pulmonary regurgitation and the long term influence of pulmonary regurgitation is an obvious concern. We reported our earlier observations that free pulmonary regurgitation was clinically well tolerated, with stent reduced RV pressure overload.[8] In this longer term, larger cohort study, free pulmonary regurgitation was similarly clinically well tolerated and is supported by unchanged RV dimensions throughout follow up. However, chronic RV volume overload may result in ventricular arrhythmias or be related to cardiac dysfunction if it is longstanding.[6] This treatment strategy is not meant to replace surgical conduit management but rather to postpone conduit replacement during a time of anticipated somatic growth. In

support of this approach, these children continued to have normal body growth without symptoms. Note that the procedure can be technically difficult (four children had incomplete stent expansion and, in five, balloon rupture during the procedure required balloon exchange with the stent in situ). Nevertheless, the technical learning curve is steep, fluoroscopy times are generally short, and deployment is easier with the newer low profile flexible stents with high expansion ratios.

Recently, Bonhoeffer and colleagues[17] and Khambadkone and Bonhoeffer[18] have shown the effectiveness of a percutaneous bovine valve within a balloon expandable stent to address both pressure and volume overload. This is a very promising technology but, because of the profile of the delivery system, it is not uniformly applicable to all patients. Indeed, the median weight of our cohort was 21 kg, just below the smallest child in which this technology has been applied. Clearly, avoiding the induction of pulmonary regurgitation is most desirable but the knowledge that a naked stent will afford an acceptable clinical outcome in children too small to have (at present) a valved stent implant, as documented by these data, is reassuring. Furthermore, using a naked stent does not preclude secondary implantation (when appropriate). Our study supports stent implantation for obstruction within RVOT reconstructions as an effective and safe palliation, allowing children normal body growth before repeat intervention.

Limitations

Our results may be biased, as we did not review all children who underwent primary surgical conduit replacement during this period. Thus, the incidence of the conduit obstruction was not addressed. The impact of stent implantation on exercise performance, RV morphology by magnetic resonance imaging, or the prevalence of arrhythmias was not assessed. Additionally, as noted above, some data points were missing, although they were few and unlikely to skew the results.

Conclusions

In a child with an obstructed conduit between the RV and PA, stent implantation within the conduit appears to be safe and effective and postpones conduit replacement. Body growth should be maintained during follow up. Pulmonary insufficiency is clinically well tolerated without measurable changes in RV function. A second catheter intervention appears similarly to prolong conduit function for selected children with a conduit compressed anteroposteriorly or for children in whom a larger balloon than the predicted RVOT diameter can be used.

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