

Comparison of Bare Metal Stenting and Percutaneous Pulmonary Valve Implantation for Treatment of Right Ventricular Outflow Tract Obstruction

Use of an X-Ray/Magnetic Resonance Hybrid Laboratory for Acute Physiological Assessment

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Background—Treatment of right ventricular outflow tract obstruction is possible with a bare metal stent (BMS), although this treatment causes pulmonary regurgitation. In this study, we assessed the acute physiological effects of BMS versus percutaneous pulmonary valve implantation (PPVI) using an x-ray/magnetic resonance hybrid laboratory.

Methods and Results—Fourteen consecutive children (median age, 12.9 years) with significant right ventricular outflow tract obstruction underwent BMS followed by PPVI. Magnetic resonance imaging (ventricular volumes and function and great vessel blood flow) and hemodynamic assessment (invasive pressure measurements) were performed before BMS, after BMS, and after PPVI; all were performed under general anesthesia in an x-ray/magnetic resonance hybrid laboratory. BMS significantly reduced the ratio of right ventricular to systemic pressure ($0.75 \pm 0.17\%$ versus $0.41 \pm 0.14\%$; $P < 0.001$) with no further change after PPVI (0.42 ± 0.11 ; $P = 1.0$). However, BMS resulted in free pulmonary regurgitation ($21.3 \pm 10.7\%$ versus $41.4 \pm 7.5\%$; $P < 0.001$), which was nearly abolished after PPVI ($3.6 \pm 5.6\%$; $P < 0.001$). Effective right ventricular stroke volume (right ventricular stroke volume minus pulmonary regurgitant volume) after BMS remained unchanged (33.8 ± 7.3 versus 32.6 ± 8.7 mL/m²; $P = 1.0$) but was significantly increased after revaluation with PPVI (41.0 ± 8.0 mL/m²; $P = 0.004$). These improvements after PPVI were accompanied by a significant heart rate reduction (75.5 ± 17.7 bpm after BMS versus 69.0 ± 16.9 bpm after PPVI; $P = 0.006$) at maintained cardiac output (2.5 ± 0.5 versus 2.4 ± 0.5 versus 2.7 ± 0.5 mL · min⁻¹ · m⁻²; $P = 0.14$).

Conclusion—Using an x-ray/magnetic resonance hybrid laboratory, we have demonstrated the superior acute hemodynamic effects of PPVI over BMS in patients with right ventricular outflow tract obstruction. (*Circulation*. 2009;119:2995-3001.)

Key Words: stents ■ ventricular outflow obstruction ■ heart valve prosthesis implantation ■ magnetic resonance imaging ■ pediatrics

Surgical repair of complex congenital heart disease often involves placement of a right ventricle (RV) to pulmonary artery (PA) conduit. Although surgical conduit placement can be performed with very low mortality, the lifespan of conduits is limited, reported to be <10 years.¹⁻⁴ Conduit degeneration leads to obstruction with or without pulmonary regurgitation. Thus, the majority of patients with conduits from the RV to PA undergo multiple operations during their lifetime.

Clinical Perspective on p 3001

Bare metal stenting (BMS) has been proposed as a treatment option to prolong the lifespan of conduits.⁵⁻¹⁰ This

treatment leads to the successful relief of obstruction but replaces a hemodynamically significant stenotic lesion with free pulmonary regurgitation. Although many studies have shown that BMS results in a significant reduction in the gradient across the conduit, in RV pressures, and in the ratio of RV to systemic pressure,⁵⁻¹⁰ the acute consequences of pulmonary regurgitation after BMS are not well described and cannot be fully assessed by catheter data alone.

Over the last few years, percutaneous pulmonary valve implantation (PPVI) has become a reality. With this technique, a competent valve can be implanted safely and effectively to relieve conduit obstruction without causing free

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pulmonary incompetence.^{11–15} In our institution, we are increasingly performing BMS before PPVI to provide a more suitable anchoring environment for PPVI and potentially to reduce the risk for stent fracture of the valved stent.¹⁶

More recently, combined catheterization/magnetic resonance (MR) technology has become available.^{17–20} Such catheterization/MR hybrid laboratories (x-ray/MR hybrid laboratories) have the potential to accurately assess changes in acute physiology not only with regard to pressure measurements but also by measuring ventricular dimensions/function and great vessel blood flow. In this study, we assess children undergoing sequential BMS and PPVI in the setting of an x-ray/MR hybrid laboratory to describe the acute physiological consequences of the 2 procedures.

Methods

Patients

In this prospective study, 14 consecutive children were studied between September 2007 and June 2008. Children were included when they had a clinical indication for PPVI in the context of significant RV outflow tract (RVOT) obstruction.¹¹ In symptomatic patients, this was defined as a ratio of RV to systemic pressure of >0.66 . In the absence of symptoms, this ratio had to be >0.75 . In addition, patients had to fulfill morphological requirements for PPVI, as previously published.¹¹ Briefly, the dimensions of the RVOT had to range between 14 and 22 mm as assessed by MR imaging within 1 month of the procedure. Exclusion criteria were unfavorable morphology for PPVI,^{11,21} occluded central veins, active infection, and/or contraindications to MR imaging. All investigations and interventions were performed at the Great Ormond Street Hospital for Children (London, UK). The study was approved by the institution's ethics committee. Written consent was obtained from patients and parents/guardians as appropriate.

Patients were assigned to New York Heart Association functional classes 1 to 4. Transthoracic echocardiography was performed in all patients. The RVOT gradient was calculated from the velocity across the RVOT and RV systolic pressure estimated from the tricuspid regurgitant jet.²² Systemic pressures were derived from noninvasive blood pressure measurements; the ratio of RV to systemic pressure was calculated.

Catheterization/MR Study Protocol

The impact of BMS and PPVI on biventricular physiology was assessed in a 2-staged sequential procedure, with patients serving as their own controls. All patients were mechanically ventilated via cuffed tracheal tubes. Anesthesia was induced by either intravenous propofol (10 of 14) or inhaled sevoflurane and maintained by either inhaled isoflurane (9 of 14) or sevoflurane (only 1 maintenance vapor was used in each patient). All patients were given a muscle relaxant to effect tracheal intubation and maintain immobility throughout the procedure. The concentration of anesthetic vapor was adjusted to maintain steady heart rate and blood pressure at the beginning of the study and then generally not altered thereafter. Changes in vapor concentrations, however, were made to ensure adequate depth of anesthesia according to clinical judgment. Intravenous crystalloid solutions (15 to 30 mL/kg) were infused during the study.

All interventions and MR scans were carried out under the same general anesthetic in an x-ray/MR hybrid laboratory (consisting of an x-ray Artis biplane system and a 1.5-T Avanto MR scanner, Siemens Medical Solutions, Erlangen, Germany). Patients were initially placed in the MR scanner, and biventricular volumes and great vessel blood flow were assessed. Patients were then transferred to the biplane catheter laboratory with the use of a mechanized sliding table that allows bidirectional transfer between the MR scanner and the catheterization laboratory (Miyabe, Siemens Medical Solutions). After hemodynamic assessment, BMS of the stenotic RVOT was

performed. The hemodynamic assessment was repeated, and an MR-compatible balloon wedge pressure catheter (Arrow, Reading, Pa) was placed in the pulmonary artery to avoid renegotiation of the BMS during the subsequent PPVI. All wires were removed, and the patient was transferred back into the MR scanner for repeat assessment of biventricular volumes and great vessel blood flow. A third transfer back into the catheterization laboratory was carried out, and PPVI was performed within the previously placed stent. After a post-PPVI hemodynamic assessment was performed, the patient was transferred 1 last time into the MR scanner, where biventricular volumes and great vessel blood flow were assessed (Figure).

Catheterization Protocol

Vascular access was achieved through the femoral vein and artery. Standard right heart catheterization, including pressure measurements and RVOT angiography, was undertaken. Invasive systemic pressures were monitored. Aortic root angiography was performed routinely to assess the proximity of the coronary arteries to the RVOT to avoid possible coronary compression caused by the BMS or PPVI. Simultaneous balloon inflation in the RVOT and coronary angiography were performed in patients at risk for coronary obstruction.^{6,11,23,24} Very stenotic, tortuous conduits were predilated with high-pressure Mullins balloons (NuMed, Hopkinton, NY). Balloon-expandable IntraStent Max LD stents (ev3 Intravascular, Plymouth, Minn) or covered CP stents (NuMed) were used for BMS of the RVOT. Stents were delivered and deployed with BIB catheters (NuMed). Hemodynamic measurements were repeated after BMS. PPVI was performed within the BMS. The device and technique used have been described elsewhere.^{13,25} After PPVI, pressure measurements and RVOT angiography were performed. Postdilatation with high-pressure Mullins balloons (NuMed) was performed after BMS or PPVI if appropriate.

MR Imaging Protocol

The same imaging protocol was used for MR scans before BMS, after BMS, and after PPVI. MR imaging was performed at 1.5 T (Avanto, Siemens). Retrospectively gated steady-state free-precession cine images were acquired in the vertical long-axis and 4-chamber views. They were used to plan the short-axis stack, which included the extent of both ventricles (9 to 12 slices). The cine steady-state free-precession sequence parameters were as follows: repetition time, 2.4 ms; echo time, 1.1 ms; flip angle, 60°; slice thickness, 8 to 10 mm (no gap in short-axis stack); matrix, 192×144; and field of view, 280 to 380 mm, with 25 phases per cardiac cycle.

Assessment of RV and left ventricular (LV) volumes was performed by manually defining the endocardial outline at end diastole and end systole in each of the short-axis cine images (Argus, Siemens Medical Systems). The end-diastolic volume (EDV) and end-systolic volume were calculated with Simpson's rule for each ventricle, and from these volumes, the stroke volume (SV) and ejection fraction (EF) were derived.

Flow data were acquired with a flow-sensitive gradient echo sequence (repetition time, 6.4 ms; echo time, 3.2 ms; flip angle, 30°; slice thickness, 3 mm; and matrix, 256×192, with 40 phases per cardiac cycle) during free breathing. Pulmonary blood flow was calculated from flow measurements in both branch PAs to avoid possible artifact caused by the stents in the RVOT after BMS or PPVI.^{26,27} Flow was calculated from the phase-contrast images using a semiautomatic vessel edge-detection algorithm with operator correction. Regurgitant fraction was calculated as the percent of backward flow over forward flow. When pulmonary regurgitation was present, an effective RV SV was calculated to reflect the net forward blood flow into the PAs as follows: effective RV SV = total PA forward flow – PA backward flow. All volume and flow measurements were indexed for body surface area and expressed in milliliters per square meter.

A subset of patients (11 of 14) underwent cardiac MR imaging while awake before the interventions and within 1 week after interventions. The changes in cardiac output and heart rate after the interventions were assessed.

Table 1. Patient Characteristics

Sex	Age, y	Weight, kg	Primary Diagnosis	RVOT Anatomy	Conduit Size, mm	Previous Heart Surgeries, n	Preprocedure Functional Class	Ratio of RV to Systemic Pressure
M	13.5	59	TGA, Rastelli	Homograft	15	2	2	0.90
F	15.1	69	ToF, Pulm atresia	Homograft	18	2	2	0.76
F	9.9	24.9	ToF	Homograft	Unknown	1	2	0.67
M	15.9	83	ToF, Pulm atresia	Hancock	22	3	2	0.90
F	10	31.5	Truncus arteriosus	Homograft	16	5	3	0.83
M	18	47.2	Aortic valve disease (Ross)	Homograft	19	1	2	0.79
M	17.2	65	Aortic valve disease (Ross)	Homograft	20	1	2	0.71
M	14.6	71.8	Aortic valve disease (Ross)	Homograft	Unknown	1	2	0.75
F	11.7	29.7	ToF, DORV	Homograft	17	2	2	0.68
F	11.4	33	ToF, Pulm atresia	Homograft	23	3	1	0.88
M	10.6	33.6	TGA, REV procedure	Contegra	18	2	2	0.67
M	12.3	53.4	ToF	Homograft	Unknown	1	2	0.69
M	17.8	66.9	ToF	Bioprosthesis	21	2	2	1.02
M	10.8	49.8	ToF, Pulm atresia	Carpentier Edwards	18	2	3	0.80

TGA indicates transposition of the great arteries; Rastelli, Rastelli procedure; ToF, tetralogy of Fallot; Pulm, pulmonary; Ross, Ross procedure; DORV, double-outlet RV; and REV procedure, *réparation à l'étage ventriculaire*.

Cardiopulmonary exercise testing before the interventions and within 1 month after the interventions was performed in 12 of 14 patients, and peak oxygen uptake was determined. The protocol for cardiopulmonary exercise testing has been described previously.¹²

Statistical Analysis

Data are expressed as mean±SD. MR and pressure data were compared by use of repeated-measures ANOVA and posthoc testing (Bonferroni) between the pre-BMS, post-BMS, and post-PPVI states. MR data acquired in awake patients and cardiopulmonary exercise data before and after the interventions were compared by use of the paired Student *t* test. A value of $P<0.05$ was considered statistically significant. Statistical testing and data analysis were performed with SPSS version 15.0 (SPSS Inc, Chicago, Ill).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

Results

Patient Characteristics

The patient characteristics are shown in Table 1. The median age of the patient population was 12.9 years (range, 9.9 to 17.8 years). The majority of children (9 of 14) had tetralogy of Fallot or a variant morphology. RV to PA conduits were present in all but 1 patient, who presented with a stenosed bioprosthesis in the pulmonary position. All patients except 1 were symptomatic (New York Heart Association functional class >1; see Table 1 for details). Before the procedure, a ratio of RV to systemic pressure >0.75 was present in 9 of 14.

Procedural Results

The total procedure, catheterization, and fluoroscopy times were 209±14, 92±14, and 21±12 minutes, respectively. The time frames for each of the individual steps included in the study protocol are summarized in the Figure. BMS and PPVI were successful in all patients. There were no major complications. In 1 patient, a guidewire injury caused minor lung bleeding, which was treated conservatively. In total, 16 IntraStent Max LD stents (ev3 Intravascular) were implanted

into 13 patients. In 1 patient with a small conduit (16 mm), a covered CP stent (NuMed) was used. In 2 patients with tortuous and severely obstructed conduits, predilatation was performed. High-pressure balloon dilatation after BMS was performed in 4 patients and after PPVI in 6 patients.

Hemodynamic Outcome

Invasive pressure measurements before BMS, after BMS, and after PPVI are summarized in Table 2. BMS resulted in a significant reduction in RV systolic pressure (63±14 versus 37±11 mm Hg; $P<0.001$, posthoc testing, before versus after BMS), PA to RV pullback gradient (43±11 versus 13±7 mm Hg; $P<0.001$), and ratio of RV to systemic pressure (0.75±0.17% versus 0.41±0.14%; $P<0.001$).

There was no significant further change in RV systolic pressure, PA to RV gradient, or ratio of RV to systemic pressure between after BMS and after PPVI (39±13 mm Hg, $P=0.76$; 15±8 mm Hg, $P=1.0$; 0.42±0.11, $P=1.0$, respectively). The PA diastolic pressure increased after PPVI, reflecting the restoration of pulmonary valvular competence (9±4 mm Hg before BMS versus 11±6 mm Hg after PPVI; $P=0.048$). Systemic pressures remained unchanged after BMS but increased slightly after PPVI (85±7 mm Hg before BMS versus 85±7 mm Hg after BMS versus 93±12 mm Hg after PPVI).

MR Volumes and Ventricular Function

MR parameters are summarized in Table 3. After BMS, there was a significant increase in pulmonary regurgitant fraction (21.3±10.7% versus 41.4±7.5%; $P<0.001$, posthoc testing, before versus after BMS). The reduction in afterload after BMS resulted in a decrease in RV end-systolic volume (53.1±35.4 versus 41.7±31.7 mL/m²; $P<0.001$) and in an increase in total RV SV (44.2±11.3 versus 56.6±13.8 mL/m²; $P=0.002$) and RV EF (48.7±12.2% versus 60.7±12.6%; $P<0.001$). However, these changes could not fully compensate for the induced free pulmonary regurgita-

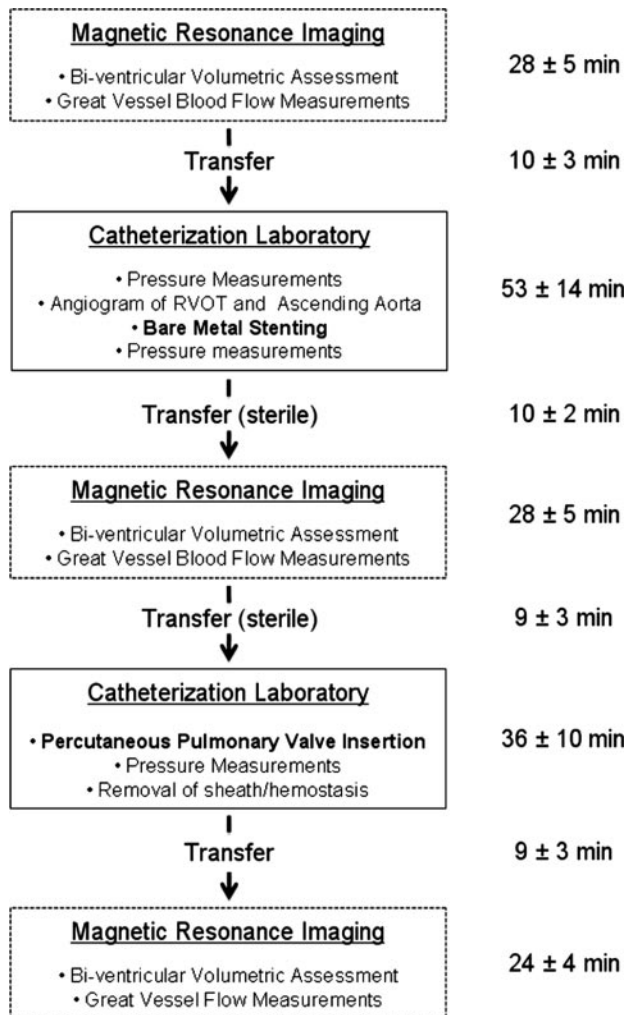


Figure. Summary of the study protocol, including the timing of the individual steps.

tion, as demonstrated by the lack of improvement in effective RV SV after BMS (33.8 ± 7.3 versus 32.6 ± 8.7 mL/m²; $P=1.0$).

Revaluation by PPVI virtually abolished pulmonary regurgitation (pulmonary regurgitation fraction, $41.4 \pm 7.5\%$ versus $3.6 \pm 5.6\%$; $P<0.001$, posthoc testing, after BMS versus after PPVI) and reduced RV EDV (98.3 ± 40.4 versus

85.3 ± 30.2 mL/m²; $P=0.021$). RV end-systolic volume (41.7 ± 31.7 versus 39.4 ± 25.4 mL/m²; $P=0.78$) remained unchanged; the RV EF ($60.7 \pm 12.6\%$ versus $56.9 \pm 11.2\%$; $P=0.08$) decreased nonsignificantly after PPVI compared with the post-BMS state. Importantly, there was a significant improvement in effective RV SV after PPVI (32.6 ± 8.7 versus 41.0 ± 8.0 mL/m²; $P=0.004$).

BMS and PPVI also had an impact on LV volumes and function. Whereas LV EDV decreased slightly after BMS, there was a significant increase in LV EDV after PPVI (69.6 ± 15.7 mL/m² before BMS versus 65.9 ± 16.7 mL/m² after BMS versus 75.4 ± 17.6 mL/m² after PPVI; $P=0.001$, posthoc testing, after BMS versus after PPVI). Similar to the effective RV SV, the effective LV SV remained unchanged after BMS (33.1 ± 6.9 versus 32.5 ± 9.4 mL/m²; $P=1$) but improved significantly after PPVI (32.5 ± 9.4 versus 40.7 ± 8.8 mL/m²; $P=0.013$).

The increase in effective RV and LV SV after PPVI was accompanied by a significant reduction in heart rate (75.5 ± 17.7 bpm after BMS versus 69.0 ± 16.9 bpm after PPVI; $P=0.006$). Consequently, there was no significant change in cardiac output after PPVI (2.5 ± 0.5 mL · min⁻¹ · m⁻² before BMS versus 2.4 ± 0.5 mL · min⁻¹ · m⁻² after BMS versus 2.7 ± 0.5 mL · min⁻¹ · m⁻² after PPVI; $P=0.14$, posthoc testing, after BMS versus after PPVI).

In the subset of patients, who underwent preintervention and postintervention cardiac MR scans while awake, there was a significant increase in cardiac output (3.2 ± 0.4 mL · min⁻¹ · m⁻² before intervention versus 3.8 ± 0.6 mL · min⁻¹ · m⁻² after intervention; $P=0.004$), whereas heart rate did not change significantly (73.0 ± 9.0 versus 75.5 ± 9.3 bpm; $P=0.042$).

On cardiopulmonary exercise testing, peak oxygen uptake (peak $\dot{V}O_2$) expressed as percentage of predicted peak $\dot{V}O_2$ increased significantly from $67.6 \pm 19.8\%$ to $75.7 \pm 15.3\%$ ($P=0.048$) after intervention.

Discussion

Over the last decade, BMS has been the nonsurgical treatment of choice for stenotic RV to PA conduits. However, this intervention has a significant drawback in that relief of stenosis leads to the creation of free pulmonary regurgitation. Cardiac catheterization studies have shown a reduction in the

Table 2. Pressure Measurements During Catheterization

Parameter	Before BMS	After BMS	Change	After PPVI	Change	ANOVA	Posthoc Analysis		
							Pre vs BMS	Pre vs PPVI	BMS vs PPVI
RV systolic pressure, mm Hg	63.1±14.4	36.6±10.9	Decrease	39.4±12.7	None	<0.001	<0.001	<0.001	0.762
RV end-diastolic pressure, mm Hg	11.7±3.9	10.1±3.8	None	8.6±3.2	Decrease	0.005	0.076	0.002	0.127
PA systolic pressure, mm Hg	19.9±4.8	23.1±7.7	Increase	23.8±7.6	None	0.049	0.199	0.066	1
PA diastolic pressure, mm Hg	9.2±3.8	8.6±3.1	None	11.2±5.7	Increase	0.041	0.316	0.262	0.048
RV to PA pullback gradient, mm Hg	43.1±14.3	13.4±7.3	Decrease	15.0±7.7	None	<0.001	<0.001	<0.001	1
RV to systemic pressure, %	74.7±17.5	40.5±14.2	Decrease	42.3±10.6	None	<0.001	<0.001	<0.001	1
LV systolic pressure, mm Hg	84.5±6.6	84.6±7.4	None	92.5±12.4	Increase	0.12	1	0.165	0.112
LV end-diastolic pressure, mm Hg	12.0±3.2	11.9±3.3	None	11.1±3.0	None	0.68	1	1	1

Table 3. MR Parameters

Parameter	Before BMS	After BMS	Change	After PPVI	Change	ANOVA	Posthoc Analysis		
							Pre vs BMS	Pre vs PPVI	BMS vs PPVI
PRF, %	21.3±10.7	41.4±7.5	Increase	3.6±5.6	Decrease	<0.001	<0.001	<0.001	<0.001
RV EDV, mL/m ²	97.4±40.13	98.3±40.4	None	85.3±30.2	Decrease	<0.001	1	0.08	0.021
RV ESV, mL/m ²	53.1±35.4	41.7±31.7	Decrease	39.4±25.4	None	<0.001	<0.001	0.002	0.776
RV SV, mL/m ²	44.2±11.3	56.6±13.8	Increase	45.9±8.8	Decrease	<0.001	0.002	1	0.009
Effective RV SV, mL/m ²	33.8±7.3	32.6±8.7	None	41.0±8.0	Increase	0.001	1	0.02	0.004
RV EF, %	48.7±12.2	60.7±12.6	Increase	56.9±11.2	None	<0.001	<0.001	0.006	0.078
LVEDV, mL/m ²	69.6±15.7	65.9±16.7	None	75.4±17.6	Increase	0.0022	0.45	0.04	0.001
LVESV, mL/m ²	32.2±12.1	29.5±12.7	None	30.5±9.9	None	0.058	0.058	1	1
Effective LV SV, mL/m ²	33.1±6.9	32.5±9.4	None	40.7±8.8	Increase	0.012	1	0.014	0.013
LV EF, %	54.7±10.4	56.4±10.7	None	60.0±7.8	Increase	0.119	0.781	0.105	0.299
Heart rate, bpm	76.9±16.8	75.5±17.7	None	69.0±16.9	Decrease	0.01	1	0.132	0.006
LV cardiac output, mL · min ⁻¹ · m ⁻²	2.5±0.5	2.4±0.5	None	2.7±0.5	None	0.121	1	0.373	0.141

PRF indicates pulmonary regurgitant fraction; ESV, end-systolic volume.

RVOT gradient and RV systolic pressure,^{5–10} which has been described as a positive procedural outcome. However, none of the previous studies have assessed the full hemodynamic consequences of BMS, particularly the impact of acute pulmonary regurgitation on RV and LV function. In this study, we show that a thorough assessment of cardiac pressures, biventricular function, and great vessel blood flow reveals the superior acute hemodynamic consequences of PPVI over BMS.

Physiological Comparison of BMS Versus PPVI

In our patients, BMS led to significant relief of the RVOT gradient and RV pressure. This reduction in RV afterload led to a lower RV end-systolic volume and therefore higher RV SV and EF. Nevertheless, as a result of the creation of free pulmonary regurgitation (pulmonary regurgitation fraction >40%), there was no improvement in effective RV SV, ie, the net pulmonary forward blood flow. Conversely, after PPVI, effective RV SV increased with no further changes in RVOT gradient or RV systolic pressure. PPVI also resulted in increases in LVEDV and effective LV SV that were not observed after BMS.

If we consider only the catheter-measured hemodynamic changes in our study, we observe that BMS and PPVI have similar beneficial effects (reduced RVOT gradient, reduced RV pressures). However, when catheter and MR data are interpreted together, it can be seen that biventricular effective SVs and efficiency improve only after PPVI. Fundamentally, inducing free pulmonary regurgitation by BMS converts the RV from a ventricle with increased pressure work to a ventricle with increased volume work. In this situation, the ratio between effective RV SV and RV external work could be considered to remain at a similar level. On the other hand, revalvulation with PPVI results in a reduction in RV SV at maintained low RV pressures. Hence, this intervention reduces the external work of the RV with increased effective RV SV, leading to an improved RV efficiency, which in turn should reduce myocardial oxygen consumption.

The increase in effective RV and LV SV after PPVI was accompanied by a significant drop in heart rate. Consequently, cardiac output did not change after BMS or PPVI. We have previously described an increase in cardiac output after PPVI when patients are assessed with MR imaging while awake (ie, no general anesthetic).^{12,28} Similarly, in the subset of patients in this study who underwent MR scans while awake before and after the interventions, cardiac output increased significantly, whereas heart rate remained unchanged. Although cardiac output and heart rate might be more susceptible to alterations in sympathetic activation when assessed in patients who are awake, these findings could indicate that these patients were slightly decompensated before intervention and therefore show a postprocedure improvement in cardiac output.

In patients under general anesthesia, we would argue that because oxygen demand is likely to be low, there is little physiological drive to increase the cardiac output. Therefore, when effective RV and LV SVs increase after a reduction in both RV pressure and volume overload, the physiological feedback is to reduce heart rate. We therefore speculate that the drop in heart rate that we observed represents an adaptation to the increase in effective RV and LV SV after PPVI at unchanged oxygen demands. This again suggests an improvement in biventricular efficiency that occurs only after PPVI, not after BMS.

Use of an X-Ray/MR Hybrid Laboratory

In this study, we have used an x-ray/MR hybrid laboratory to compare the impact of BMS and PPVI on biventricular physiology in a 2-staged sequential procedure, with patients serving as their own controls. This not only enabled accurate quantification of physiology in terms of pressure measurements, ventricular function, and great vessel blood flow but offered the potential to compare the acute hemodynamic outcome of sequential interventions (BMS and PPVI). This represents a novel utility of such hybrid laboratories in humans to assess physiological changes after interventions in

a more comprehensive way and might have the potential to influence future patient management.

Study Limitations

Because this study assessed only the acute response to BMS and PPVI, our findings cannot speak to the relative clinical impact of BMS and PPVI in children with obstructed RVOT conduits.

Pressure data and volume/flow data were not acquired simultaneously but sequentially, including several transfers between the catheterization laboratory and the MR scanner. Although probably minimized by general anesthesia, small variations in the physiological activation of the patients cannot be excluded. Similarly, because oxygen demand and consumption during the procedure were not assessed, small perturbations in the depth of the general anesthesia also cannot be excluded. Finally, when cardiac physiology is assessed under general anesthesia, it could potentially differ from assessment in awake patients.

Conclusions

The results of this study suggest the superiority of PPVI over BMS for improving biventricular efficiency in patients with RVOT obstruction in the acute setting. This was assessed by the use of an x-ray/MR hybrid laboratory, which allowed a detailed assessment of hemodynamics and, in particular, ventricular function and efficiency before and after interventions.

The results of this study should enhance our understanding of biventricular physiology after altering RV loading conditions. Future studies might include simultaneous acquisition of pressure and volume data to reconstruct pressure-volume loops of the RV and LV, which potentially could provide deeper insight into the systolic and diastolic ventricular interaction after correction of pathological RV loading conditions.

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Disclosure

Dr Bonhoeffer is a consultant to Medtronic and NuMed and has received honoraria and royalties for the device described. Drs Lurz and Khambadkone are consultants to Medtronic and have received honoraria. The other authors report no conflicts.

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CLINICAL PERSPECTIVE

In this study, we assessed children undergoing sequential bare metal stenting and percutaneous pulmonary valve implantation in the setting of an x-ray/magnetic resonance hybrid laboratory to compare the acute physiological consequences for biventricular function. Bare metal stenting has been proposed as a treatment option to prolong the lifespan of conduits and has been the gold standard for nonsurgical treatment of conduit obstruction over the last few decades. However, relief of obstruction comes at the expense of pulmonary incompetence. Recently, percutaneous pulmonary valve implantation has become a reality. This technique allows successful relief of conduit obstruction without causing valvar incompetence. Although a comparison of pressure measurements in our study showed similar beneficial effects of bare metal stenting and percutaneous pulmonary valve implantation (reduced right ventricular outflow tract gradient, reduced right ventricular pressures), combined assessment of both catheter and magnetic resonance data revealed that biventricular effective stroke volumes improve only after percutaneous pulmonary valve implantation. Therefore, at least acutely, percutaneous pulmonary valve implantation is superior to bare metal stenting in improving biventricular efficiency and should therefore represent the first nonsurgical treatment option in patients with conduit obstruction. This study also shows a novel utility of an x-ray/magnetic resonance hybrid laboratory to assess physiological changes after interventions more comprehensively. The results of this study emphasize the importance of assessing not only pressures but also biventricular efficiency after right ventricular outflow tract interventions and might have the potential to influence future patient assessment and management.