JACC: CARDIOVASCULAR INTERVENTIONS
VOL. 8, NO. 12, 2015

ISSN 1936-8798/$36.00
PUBLISHED BY ELSEVIER INC.
http://dx.doi.org/10.1016/j.jcin.2015.05.027

Fate of Hypoplastic Pulmonary Arteries After Arterial Duct Stenting in Congenital Heart Disease With Duct-Dependent Pulmonary Circulation

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ABSTRACT

OBJECTIVES This study sought to evaluate the impact of arterial duct (AD) stenting in promoting catch-up growth of hypoplastic pulmonary artery (PA) tree in congenital heart disease with duct-dependent pulmonary circulation (CHD-DPC).

BACKGROUND Significant and balanced PA growth following AD stenting has already been consistently reported in the literature. However, no data are so far available about the role of this approach in severe PA hypoplasia, which significantly impacts the risk of surgical repair.

METHODS Pre-surgical angiographic PA evaluation was performed in 45 patients with confluent PAs submitted to neonatal AD stenting as palliation of CHD-DPC. PA growth was evaluated as Nakata Index and McGoon ratio as well as individual PA *z*-score changes, both in the whole population and according to the original vessel size (Nakata Index <100 mm^2/m^2, Group I [n $=$ 15] vs. Nakata Index >100 mm^2/m^2, Group II [n $=$ 30]).

RESULTS Control angiography was performed 7.5 $\pm$ 6.5 months (median 6 months) after duct stenting, showing significant and balanced PA growth. The Nakata Index increased from 143 $\pm$ 73 mm^2/m^2 to 270 $\pm$ 88 mm^2/m^2 (124 $\pm$ 118%, $p < 0.0001$); left PA *z*-score from -0.7 ± 1.7 to 1.0 ± 1.4; right PA *z*-score from -0.6 ± 1.3 to 1.2 ± 1.3 ($p < 0.0001$ for both comparisons). Group I showed a greater increase of global PA growth (Nakata Index increase 227 $\pm$ 141% vs. 72 $\pm$ 57%, $p < 0.001$) as compared with Group II. Final PA size did not significantly differ between the groups (246 $\pm$ 105 mm^2/m^2 vs. 282 $\pm$ 78 mm^2/m^2, p $=$ NS).

CONCLUSIONS Percutaneous AD stenting is highly effective in promoting a significant and balanced catch-up growth of diminutive PAs, being therefore advisable in this subset of patients as a reliable alternative to surgical palliation.
(J Am Coll Cardiol Intv 2015;8:1626–32)

Arterial duct (AD) stenting is widely considered an effective palliation of congenital heart disease with duct-dependent pulmonary circulation (CHD-DPC) in high-risk patients unsuitable for primary repair (1-6). This option may promote significant and more balanced pulmonary artery (PA) growth as compared with surgical systemic-to-pulmonary artery shunt (7-10). However, no study has so far specifically addressed the fate of hypoplastic PAs following AD stenting, which may significantly impact the overall risk of surgical repair.

The aim of this study was to evaluate the role of AD stenting in promoting the growth potential of diminutive PAs in patients with CHD-DPC.

METHODS

PATIENT POPULATION. Between April 2003 and December 2014, 118 neonates and infants with

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Manuscript received February 18, 2015; revised manuscript received May 5, 2015, accepted May 22, 2015.

CHD-DPC underwent successful AD stenting at our institution as a cost-effective alternative to surgical palliation. Mean hospital stay for AD stenting was 12 days in a regular ward compared with 10 days (3 days in a post-surgical intensive care unit and 7 in a regular ward) for a surgical shunt, resulting in a similar global economic impact (€9,823 vs. €11,090). Among these patients, 45 needed just a short time of support to pulmonary circulation, 17 are still in follow-up before planned surgical repair, and 4 died before hospital discharge. The remaining 52 patients needed surgical repair over a mid-term follow-up and were submitted to control cardiac catheterization 7.5 ± 6.5 months (range 2 to 45 months, median 6 months) after the initial palliation. Seven patients were excluded from the analysis because the stented duct supplied disconnected PAs. The demographic and clinical data of the remaining 45 patients are reported in Table 1. At the time of AD stenting, 39 patients showed biventricular physiology, and the remaining 6 patients were supposed to be candidates for Fontan track. Thirteen patients had complete duct dependency of the pulmonary circulation due to trivial or absent additional pulmonary blood flow (PBF) (28.9%), whereas the remaining 32 patients showed mild, but clinically insignificant, accessory PBF. Eight of the 12 patients with pulmonary valve atresia with intact ventricular septum and none of the patients with pulmonary valve atresia with ventricular septal defect had been submitted to pulmonary valve perforation at the time of AD stenting. No patient showed hemodynamic significant aortopulmonary collaterals at control angiography. At the time of AD stenting, 15 patients showed very hypoplastic PAs (Nakata Index <100 mm^2/m^2, range 46 to 100, median 79 mm^2/m^2; Group I), whereas the remaining 30 patients had normal or mildly reductive PAs (Nakata Index >100 mm^2/m^2, range 110 to 399, median 164 mm^2/m^2; Group II). No difference was found between groups in terms of intracardiac anatomy, as well as complete duct dependency of the pulmonary circulation (Table 1).

INTERVENTIONAL PROCEDURE. AD stenting was performed under general anesthesia following a previously described protocol, with the aim of covering the entire AD length (4–6). Mean prostaglandin dosage at the time of AD stenting was 0.02 g/kg/min, but prostaglandin infusion had been stopped some few hours before the procedure. The stent size was individually chosen on the basis of patient size, ductal anatomy, and expected time for which palliation was needed. However, it was usually about 25% smaller than the planned surgical shunt in the belief that it acted more as a central shunt than a Blalock-Taussig shunt. After stent deployment, repeat aortic angiograms were performed in multiple views to exclude incomplete coverage of the duct as well as to evaluate the PA size and any potential stent-related PA stenosis. After the procedure, long-term acetylsalicylic acid treatment was planned at a dose of 3 to 5 mg/kg/day.

ABBREVIATIONS AND ACRONYMS

AD = arterial duct

CHD-DPC = congenital heart disease with duct-dependent pulmonary circulation

PA = pulmonary artery

PBF = pulmonary blood flow

ANGIOGRAPHIC MEASUREMENTS. Control cardiac catheterization was scheduled whenever oxygen saturation was consistently reduced over at least 2 follow-up visits or before scheduled surgical repair. Pulmonary angiography was performed in right anterior oblique and four-chamber views (Figure 1), measuring individual PAs at the site of their first branching point. The diameter of the descending

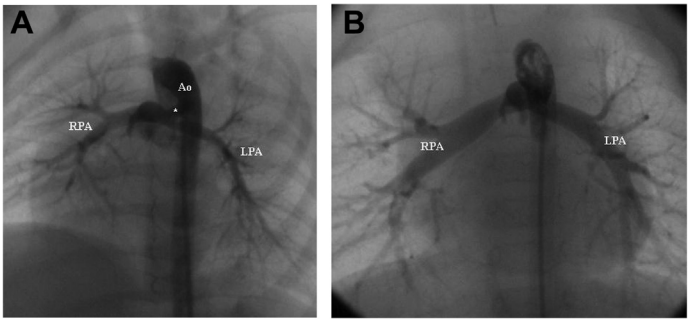
TABLE 1 Clinical, Demographic, and Angiographic Data of Patients Submitted to Control Angiography Following AD Stenting

N = 45 (complete duct-dependent pulmonary circulation 28.9%)	
Age, months	8.7 ± 7.2 (range 3–45)
Weight, kg	6.2 ± 2.6 (range 2.5–15)
Diagnosis	
Critical PS/PA-IVS	12
ToF	12
PA-VSD	9
TV Ebstein's anomaly	1
cTGA with critical PS/atresia	2
TA	1
TGA-VSD with critical PS/atresia	6
UVH with critical PS/atresia	1
Criss-cross heart with ToF	1
Group I (Nakata Index <100 mm^2/m^2, n = 15)	
Complete duct-dependency: 37.5%	
Control angiography, months: 10 ± 11 (median 6.5)	
Critical PS/PA-IVS:	3
ToF:	5
PA-VSD:	4
TV Ebstein's anomaly:	1
cTGA with critical PS/atresia:	2
Group II (Nakata Index >100 mm^2/m^2, n = 30)	
Complete duct-dependency: 24.1%	
Control angiography, months: 8 ± 5 (median 6)	
Critical PS/PA-IVS:	9
ToF:	7
PA-VSD:	5
TA:	1
TGA-VSD with critical PS/atresia:	6
UVH with critical PS/atresia:	1
Criss-cross heart with ToF:	1

Values are n unless otherwise indicated.

AD = arterial duct; cTGA = corrected transposition of great arteries; PA-IVS = pulmonary valve atresia with intact ventricular septum; PA-VSD = pulmonary valve atresia with ventricular septal defect; PS = pulmonary valve stenosis; TA = tricuspid valve atresia; TGA = transposition of great arteries; ToF = tetralogy of Fallot; TV = tricuspid valve; UVH = univentricular heart; VSD = ventricular septal defect.

FIGURE 1 PA Angiography Before and After AD Stenting



Pulmonary artery (PA) angiography in the 4-chamber view before **(A)** and 5 months after arterial duct (AD) stenting **(B)** in a newborn with originally severe PA hypoplasia (Nakata Index increase from 90 to 190 mm^2/m^2, +111%). The **asterisk** indicates the stented arterial duct. Ao = descending aorta; LPA = left pulmonary artery; RPA = right pulmonary artery.

aorta was measured at the diaphragm level. All the values were obtained during ventricular systole and, if possible, from the same angiographic frame using the angiographic catheter as a reference. Indexed PA cross-sectional area (Nakata Index) (11) and ratio of the sum of diameters of both PAs to diameter of the descending aorta (McGoon ratio) (12) were considered indicative of global vessel growth. Normative diameters of main PAs against body surface area (*z*-scores) were obtained from Internet nomograms and were deemed an index of individual PA growth. Finally, the left-to-right PA diameter ratio was considered an expression of balanced vascular growth.

TABLE 2 PA Growth Following AD Stenting in the Whole Population and According to PBF Physiology

	Pre-Stent	Post-Stent	% Change	p Value	Interaction Term (p Value)
Nakata Index, mm^2/m^2	143 ± 73	270 ± 88	124 ± 118	0.0001	
McGoon ratio	1.4 ± 0.3	2.0 ± 0.5	46 ± 36	0.0001	
LPA, mm	3.9 ± 1.2	6.9 ± 1.8	85 ± 51	0.0001	
z-score	−0.7 ± 1.7	1.0 ± 1.4		0.0001	
LPA/Ao diameter ratio	0.7 ± 0.2	1.0 ± 0.2		0.0001	
Right PA, mm	4.1 ± 1.1	7.8 ± 2.1	95 ± 52	0.0001	
z-score	−0.6 ± 1.3	1.2 ± 1.3		0.0001	
RPA/Ao diameter ratio	0.8 ± 0.2	1.1 ± 0.4		0.0001	
LPA/RPA diameter ratio	1.0 ± 0.2	0.9 ± 0.3	−5 ± 30	NS	
PA stenosis	11/45 (24.4%)				
Complete duct-dependent PBF (n = 13)					
Nakata Index, mm^2/m^2	146 ± 63	232 ± 94	76 ± 91	0.0001	0.055
McGoon ratio	1.4 ± 0.3	1.9 ± 0.3	37 ± 30	0.0001	NS
Multiple PBF sources (n = 32)					
Nakata Index, mm^2/m^2	143 ± 79	289 ± 82	145 ± 125	0.0001*	
McGoon ratio	1.5 ± 0.3	2.1 ± 0.5	48 ± 39	0.0001*	

Values are mean ± SD unless otherwise indicated. *p = NS versus complete duct-dependent PBF.

AD = arterial duct; Ao = aorta; CHD-DPC = congenital heart disease with duct-dependent pulmonary circulation; LPA = left pulmonary artery; PA = pulmonary artery; PBF = pulmonary blood flow; RPA = right pulmonary artery.

STATISTICAL ANALYSIS. All analyses were performed using SPSS for Windows version 13.0 (SPSS, Chicago, Illinois). Results are expressed as mean ± SD. Comparisons were performed using paired and unpaired Student *t* tests as well as the chi-square test. Changes between pre- and post-stenting were analyzed as repeated measures analysis of variance, with the group by pre-post interaction term included to assess the time effect on the growth changes between the groups. Significance was defined as a p value <0.05.

RESULTS

AD patency was established with stents dilated to a mean diameter of 3.6 ± 0.4 mm (range 3.2 to 4.5 mm, median 3.5 mm). The mean stent length was 13.4 ± 4.0 mm (range 8 to 24 mm, median 12 mm). AD stabilization was achieved using a single stent in the majority of patients, whereas 12 patients received 2 stents, 1 patient received 3 stents, and 1 patient received 4 stents. Ductal stenting resulted in a significant increase in oxygen saturation, from 81 ± 10% to 89 ± 7% ($p < 0.001$). No significant difference was found between the groups in terms of final duct size (3.4 ± 0.4 mm vs. 3.7 ± 0.4 mm, p = NS) and length (13.3 ± 5.0 mm vs. 16.5 ± 4.5 mm, p = NS), as well as O_2 saturation (86 ± 8% vs. 90 ± 7%, p = NS). Ductal origin was in the typical position from the aortic isthmus/upper thoracic aorta in 35 patients, whereas it was vertical in 10 patients, without any difference between the groups (26.7% vs. 20.0%, p = NS). Control angiography was performed after a median time of 6 months from the procedure, without a significant difference between groups (Table 1). Pre-surgical angiographic evaluation showed significant and balanced PA growth in the whole population, without any difference between newborns with complete duct-dependent pulmonary circulation versus multiple PBF sources (Table 2). However, Group I showed a better catch-up growth of the PA tree as compared with the group with mildly reductive PAs, without a significant difference between the groups in terms of final Nakata Index value at control angiography (Table 3, Figure 2). No significant difference was found either in the whole population or in the subgroups according to either cardiac anatomy or duct morphology and size/length. Mild-to-moderate

stenosis at the site of duct insertion was imaged in 24.4% of the patients, without a significant difference between the groups (26.7% vs. 23.3%, p = NS). However, this complication was easily addressed without incremental risk at the time of surgical repair.

TABLE 3 PA Growth According to Vessel Size at the Time of AD Stenting

	Pre-Stent	Post-Stent	p Value	Interaction Term (p Value)
Group I (Nakata Index <100 mm^2/m^2, n = 15)				
Nakata Index, mm^2/m^2	78 ± 17	246 ± 105	0.0001 NS vs. Group II	0.03
% change		227 ± 141	0.0001 vs. Group II	
McGoon ratio	1.2 ± 0.2	2.0 ± 0.4	0.0001 NS vs. Group II	NS
% change		64 ± 36	NS vs. Group II	
LPA size, mm	3.1 ± 1.3	6.4 ± 2.3	0.0001 NS vs. Group II	NS
LPA *z*-score	−2.1 ± 1.8	0.5 ± 1.8	0.0001 0.01 vs. Group II	0.01
% change		110 ± 56	0.01 vs. Group II	
LPA/Ao diameter ratio	0.6 ± 0.2	0.9 ± 0.3	0.0001 NS vs. Group II	NS
RPA size, mm	3.4 ± 1.1	7.2 ± 2.7	0.0001 NS vs. Group II	NS
RPA z-score	−1.7 ± 1.4	0.8 ± 1.8	0.0001 NS vs. Group II	NS
% change		112 ± 70	NS vs. Group II	
RPA/Ao diameter ratio	0.7 ± 0.2	1.0 ± 0.4	0.0001 NS vs. Group II	NS
LPA/RPA diameter ratio	0.9 ± 0.2	1.0 ± 0.4	NS	NS
% change		5 ± 38	NS vs. Group II	
PA stenosis	4/15 (26.7%)		NS vs. Group II	
Group II (Nakata Index >100 mm^2/m^2, n = 30)				
Nakata Index, mm^2/m^2	176 ± 69	282 ± 78	0.0001	
% change		72 ± 57		
McGoon ratio	1.5 ± 0.3	2.1 ± 0.4	0.0001	
% change		43 ± 36		
LPA, mm	4.3 ± 1.0	7.2 ± 1.5	0.0001	
z-score	0.0 ± 1.3	1.3 ± 1.3	0.0001	
% change		72 ± 44		
LPA/Ao diameter ratio	0.8 ± 0.2	1.0 ± 0.2	0.0001	
RPA, mm	4.4 ± 0.9	8.1 ± 1.7	0.0001	
z-score	−0.1 ± 0.9	1.4 ± 1.0	0.0001	
% change		86 ± 39		
RPA/Ao diameter ratio	0.8 ± 0.2	1.1 ± 0.3	0.0001	
LPA/RPA diameter ratio	1.0 ± 0.2	0.9 ± 0.2	NS	
% change		−7 ± 17		
PA stenosis	7/30 (23.3%)			

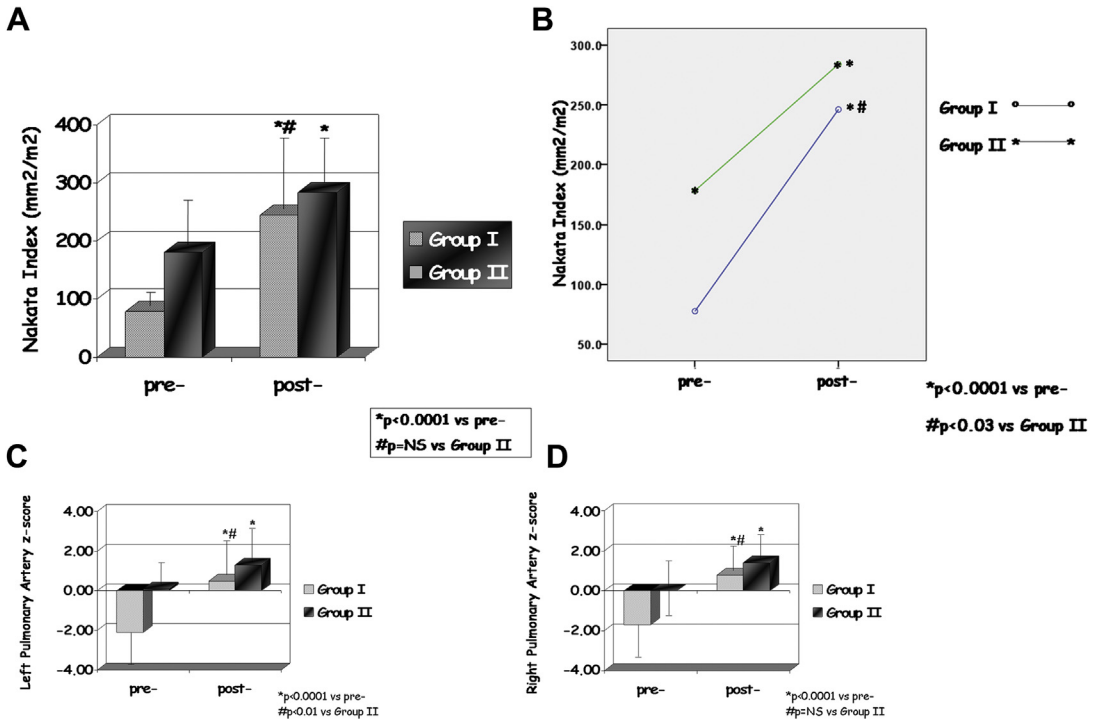
Values are mean ± SD.
Abbreviations as in Tables 1 and 2.

DISCUSSION

Failure to achieve adequate PA growth is a major obstacle to undergoing surgical repair for patients with cyanotic congenital heart disease (13–15). Indeed, PA hypoplasia was reported as a significant procedure-dependent factor in determining the Comprehensive Aristotle Score, either for surgical palliation or repair of CHD-DPC (16). Therefore, a major aim of neonatal palliation is to promote significant and balanced growth of the PA tree. Systemic-to-pulmonary shunt has been consistently found to allow significant vessel growth (17–21), although scant data are so far available in the case of hypoplastic PAs (22–24). Over time, AD stenting has gained wide acceptance as a reliable alternative to surgical palliation in patients with CHD-DPC (1–8). This option may be considered cost effective with respect to a surgical shunt in high-risk patients unsuitable for primary repair or whenever short-term PBF support is anticipated (4–6,25,26). In addition, duct patency has become an effective tool to promote significant and more balanced PA growth as compared with surgical palliation (1,5–10). To date, scant data on global and individual growth of diminutive PAs resulting from AD stenting are available in the literature. However, this anatomic arrangement is widely considered as a relative contraindication to this approach; in this case, a surgical central shunt (27) or percutaneous right ventricular outflow tract stenting (15,28) is still advocated as better therapeutic options. In our opinion, AD stenting confers most of the procedural and hemodynamic advantages of both options, being at lower risk than surgical palliation (5,6,9) and resulting in continuous (as with a surgical shunt) and balanced (as with right ventricular outflow tract stenting) PBF. In addition, conforming the stent to the size and angulations of the main PAs may theoretically maximize PA growth potential.

In this study, AD stenting confirmed its efficacy in promoting the significant and uniform PA growth already previously reported (1,7–10). Despite a trend toward better PA growth in the case of multiple PBF sources, this favorable effect was recorded even in newborns with AD as a single source of pulmonary flow, possibly due to optimal orientation of ductal flow. In addition, a better impact of AD stenting on growth potential of reductive PAs was found. In this higher-risk population, AD stenting allowed a catch-up PA growth compared with patients with mildly reductive PAs, despite similar demographic, clinical, and hemodynamic characteristics at the time of the procedure, as well as a similar diameter and length of the stented AD, and the same span of percutaneous palliation. Indeed, the "hypoplastic" group showed a faster catch-up growth, with a significantly higher percentage increase of the Nakata Index at follow-up evaluation. The final absolute PA size did not significantly differ between the groups, and also the PA tree of patients with originally diminutive branches achieved the normal range, thus decreasing their risk

FIGURE 2 Nakata Index Changes Following AD Stenting and the Time Effect on PA Growth



(A) Nakata Index changes following AD stenting in severely hypoplastic (Group I) as compared with mildly reductive (Group II) PAs. **(B)** Time effect on the PA growth in the 2 groups. Left **(C)** and right **(D)** PA *z*-scores increase after AD stenting. Abbreviations as in Figure 1.

profile for surgical repair. Finally, the growth of both PA branches in this high-risk population was balanced without any significant difference in terms of focal stenosis at the site of duct insertion as compared with the originally normal-sized PA group.

Thus, AD stenting should be considered as the first-choice palliative option in neonates with CHD-DPC and diminutive PAs, either those likely to achieve biventricular repair or destined to the Fontan circulation. In this latter subset, this approach should be even more advisable than surgical palliation in that it promotes similar, but more uniform, growth of the pulmonary vascular tree than a conventional surgical shunt (22,23), thereby hopefully favorably influencing long-term outcome of univentricular physiology.

STUDY LIMITATIONS. The study aims of comparing the mid-term effect of AD stenting in CHD-DPC with very hypoplastic versus mildly reductive PAs might be hampered by some theoretical limitations. First, the patient population is quite small, thereby precluding any reliable multifactorial analysis on the impact of patient's demographic profile, ductal anatomy, size, and length, as well as accessory PBF, on pulmonary vascular development. However, in this era of early surgical repair, this approach should be viewed as a short-term palliation, thus making less influential these variables on PA growth promotion. Second, the retrospective and nonrandomized nature of enrolment may have introduced potentially significant selection biases in evaluating the impact of this palliative option in the 2 different anatomic settings. However, there was no significant difference between the groups in terms of demographic data or pre-procedural anatomy and physiology (complete vs. partial pulmonary duct-dependency, "typical" vs. "vertical" ductal orientation, size and length of stented AD), and this option produced similar oxygen saturation over the study period. Third, this study did not evaluate the effect of surgical shunt or right ventricular outflow stenting in a matched population, thereby precluding any interpretation about the best option in this anatomic setting. However, our study did not aim to compare AD stenting versus other palliative options, but rather to evaluate the potentiality of this approach in an anatomically "borderline" population.

CONCLUSIONS

Percutaneous AD stenting with highly flexible bare-metal coronary stents may be considered an effective

tool in promoting significant and balanced PA growth in CHD-DPC over a mid-term follow-up. It seems highly effective in very diminutive PAs, thus resulting in a significant catch-up recruitment of pulmonary vessel growth potential. Thus, this option could be more strongly proposed as the first-choice approach in this subset of patients in view of lower-risk corrective surgical repair, and so modifying the selection strategy of interventional palliation.

ACKNOWLEDGMENTS The authors gratefully acknowledge Prof. Shakeel Qureshi for his invaluable help in reviewing and improving this paper. They also thank Mrs. Annunziata Orefice, chief nurse of the cardiac catheterization laboratory, and Mrs. Rosaria Riccardi, chief nurse of the clinical ward, for their tireless and loving work in assisting the small patients.

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PERSPECTIVES

WHAT IS KNOWN? PA growth is a major aim of palliation of CHD-DPC unsuitable for primary repair. AD stenting is nowadays not considered optimal palliation in the case of hypoplastic PAs, with surgical shunt or right ventricular out-flow tract stenting being advised in this setting.

WHAT IS NEW? This study shows that AD stenting is highly effective in promoting a significant catch-up growth of very diminutive PAs, significantly better than in the case of mildly reductive PAs. Thus, this option could be more strongly proposed as the first-choice approach in this subset of patients in view of a lower-risk corrective surgical repair, and so modifying the selection strategy of interventional palliation.

WHAT IS NEXT? Further studies enrolling larger populations might better clarify the role of this approach on pulmonary vascular development in terms of impact of patient's demographic profile, ductal anatomy, size and length, as well as accessory PBF.

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KEY WORDS arterial duct, congenital heart disease, cyanosis, pulmonary artery growth, stent