INTERVENTIONAL CARDIOLOGY AND SURGERY

Stenting of hypoplastic aortic segments with mild pressure gradients and arterial hypertension

D Boshoff, W Budts, L Mertens, B Eyskens, T Delhaas, B Meyns, W Daenen, M Gewillig

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See end of article for
authors' affiliations

Correspondence to:
Dr Marc Gewillig,
Paediatric Cardiology UZ
Leuven, Herestraat 49, B
3000 Leuven, Belgium;
marc.gewillig@uzleuven.
be

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Objective: To determine the safety, feasibility and effectiveness of stent expansion of hypoplastic aortic segments with pressure gradients in patients with arterial hypertension.

Design: Non-randomised prospective clinical trial.

Setting: Tertiary referral centre, congenital cardiac unit.

Patient selection: 20 consecutive patients (median age 14.5 years, range 11.6–38.8 years) with arterial hypertension and a hypoplastic segment of the aorta. Seventeen patients had successful previous arch interventions in a coarctation site.

Interventions: Stent deployment in hypoplastic arch segments.

Main outcome measures: Gradient across the aortic arch; complications early and during follow up; residual hypertension.

Results: 23 stents were deployed: 13 in the cross and 10 in the isthmus. The mean gradient across the aortic arch decreased from 16 (SD 6) (median 17) to 3 (4) (median 1) mm Hg ($p < 0.001$). In a few patients a mild gradient persisted just distal to the left carotid artery due to residual orificial narrowing or acute angulation. No complications occurred during or after the procedure. During follow up of 2.2 years (range 0.2–4.8 years) arterial hypertension resolved in 10 patients and 10 required residual drug treatment with better control of blood pressures.

Conclusions: Pressure loss due to residual hypoplastic aortic segments can be treated effectively and safely with stent expansion. Some patients remain mildly hypertensive and require additional drug treatment.

Hypoplasia of the aortic cross or isthmus can be an isolated lesion or may persist even after adequate coarctectomy.^{1,2} Such a hypoplastic segment may cause a gradient over the arch, leading to arterial hypertension.² Even mild residual aortic arch narrowing in post-coarctectomy patients predisposes to increased daytime blood pressure and carotid intima-media thickness.³ Long-term follow-up studies after coarctectomy have shown significant morbidity and mortality beyond the fourth decade, usually secondary to or associated with arterial hypertension.^{4,5} These data suggest that the clinician should aim for an aortic arch free of any gradient in the hope of improving long-term outcome in these patients.

Surgical options to treat residual hypoplasia of the aortic cross or isthmus are aortic arch reconstruction or a crossover operation, both of which are extensive operations that can be associated with significant morbidity. Suggesting this option for mild aortic arch narrowing with residual arterial hypertension can be regarded as a very aggressive approach. Balloon angioplasty alone is mostly unsuccessful in these lesions due to elastic recoil of the vessel or to unfavourable anatomy, such as long tubular narrowing, hypoplasia, angulation or only mild obstruction.⁶

Over the past decade, endovascular stents have become available and have been shown to effectively treat vascular stenosis in patients with congenital heart defects. Several experimental studies and early clinical reports have shown the feasibility and immediate effectiveness of balloon-expandable stent implantation in patients with coarctation or re-coarctation.^{7–12} We report our early experience of stent expansion of hypoplastic aortic segments with or without previous coarctectomy.

PATIENTS AND METHODS

Between February 2001 and August 2005, 20 consecutive patients (median age 14.5 years; range 11.6–38.8 years) with

hypoplasia of the aortic cross or the isthmus and arterial hypertension were treated with stent expansion of the hypoplastic segment (table 1).

Seventeen of the 20 patients previously underwent an intervention for coarctation of the aorta at the mean age of 4.2 years (range 0.0–14.9 years, median 2.4 years). End-to-end coarctectomy had been performed in eight patients, extended end-to-end coarctectomy in one patient, Waldhausen subclavian plasty in four patients, graft interposition in one patient, repair of interrupted aortic arch in one patient, balloon dilatation in one patient and covered stent implantation in one patient. Current stents were implanted 12.9 (7.9) (range 0.3–35.0, median 13.0) years after the initial aortic arch intervention.

All patients had at referral ambulatory arterial hypertension (blood pressure measured in the right arm after 5 min of rest in the supine position; no patient had an arteria lusoria). Arterial hypertension was defined as systolic or diastolic pressure above the 95th centile for age (typically post-adolescence systolic pressure > 140 mm Hg or diastolic pressure > 90 mm Hg). Seven patients were taking long-term hypertension drugs, although some had insufficient control of blood pressure (table 1).

The stent procedure was delayed until the stent could be deployed up to a (near) adult size. Informed consent was obtained from all patients and parents where appropriate. All studies were performed in accordance with local ethics committee guidelines. The procedures were performed under general anaesthesia. Heparin 100 U/kg was given once vascular access was obtained by a femoral artery puncture. Standard left catheterisation was performed, followed by an angiogram as perpendicular to the hypoplastic segment as possible (mostly mid-left anterior oblique view). A long and stiff guidewire was advanced into the ascending aorta or the left ventricle. The diameter of the balloon was chosen to

Table 1 Patient characteristics, stent data and results

Patient number	Previous arch intervention	Age (years)	At coarctectomy		Gradient (mm Hg)		Diameter (mm)		Residual waist location (mm)	Stent length (mm)	Stent position		Before stenting		1 month after stenting		Drugs*
			At	stenting	Before	After	Before	After			Cross BT-LC	Cross LC-LSA	SBP (mm Hg)	DBP (mm Hg)	SBP (mm Hg)	DBP (mm Hg)	
1	BD	11.3	11.6	4	0	15	20			28			136	80	124	80	Atenolol 25
2	ETE + reconstruction	0.0	23.0	15	0	11	20			28	1	1	138	80	124	82	
3	WH	0.0	13	28	8	8	16	15		34	1	1	130	70	130	78	Carvedilol 6.25 candesartan 8 Atenolol 25 lisinopril 5 amlodipine 5
4	ETE, transection	0.0	14.8	14	4	10	14			28			132	68	122	76	
5			13.5	16	0	8	16			39	1	1	138	80	130	76	
6	LSA																Atenolol 50 lisinopril 10
	ETE	8.9	13.8	26	10	11	18			17	1	1	145	70	132	68	
7	ETE	2.4	13.7	18	4	8	16	11		34		1	132	92	125	80	Lisinopril 5
8	WH	0.3	15.2	15	0	11	18			22	1		130	85	126	82	
9	ETE	6.1	16.7	20	10	11	18			56	1		152	90	120	82	Propranolol 80 Bisoprolol 5
10	Graft	14.9	24.4	23	4	12	18	15		22	1		180	90	140	86	
11	ETE	3.8	38.8	3	0	9	16			36			142	70	134	74	Bisoprolol 5
12	ETE	6.2	19.2	20	13	7	14			36	1		140	90	136	84	
13	WH	0.2	12.6	9	3	8	20			39			136	72	122	82	Atenolol 25 Atenolol 50
14	WH	0.0	14.2	10	0	11	16			55	1	1	146	80	120	78	
15	Covered stent	12.9	13.3	16	0	10	18			28	1	1	130	90	130	84	Atenolol 50
16	ETE	0.2	15.9	19	0	11	16			39			150	68	130	84	
17	ETE	3.6	17.4	18	9	10	16			28+34	1	1	150	85	140	82	Atenolol 50
18			16.3	17	1	10	16			28			138	90	135	86	
19	IAA ETE	0.0	14.0	18	0	10	18	17		28	1	1	130	90	126	76	Atenolol 25
20			13.1	17	2	15	18			28			135	80	122	74	
Min		0.0	11.6	3	0	7	14			17		1	130	68	120	68	Atenolol 25
Max		14.9	38.8	28	13	15	20			56			180	92	140	86	
Mean		4.2	16.7	15.7	3.4	10.3	17.1			32.7			141	81	128	80	Atenolol 25
SD		5.1	6.2	6.2	4.3	2.1	1.8			9.8			12	9	6	5	
Median		2.4	14.5	16.5	1.5	10.0	17.0			28			138	80	128	81	Atenolol 25

*Generic name with daily dose in mg.

BD, balloon dilatation; BT, brachiocephalic trunk; DBP, diastolic blood pressure; ETE, end-to-end coarctectomy; IAA, interrupted aortic arch; LC, left carotid artery; LSA, left subclavian artery; Max, maximum; Min, minimum; SBP, systolic blood pressure; WH, Waldhausen subclavian plasty.

equal the aortic diameter proximal to the hypoplastic segment, not exceeding the diameter of the descending aorta at the level of the diaphragm. Stents were delivered through an 11 or 12 French Mullins sheath (Cook, Bjæverskov, Denmark) on a balloon-in-balloon (NuMED, Hopkinton, New York, USA). The stent was manually crimped on a vacuumised balloon. A cut-off sheath of an 11 or 12 French size was used to protect the stent while it was advanced through the haemostatic valve of the Mullins sheath. The stents used were Cheatham platinum (CP) stents (NuMED; $n = 15$) and covered CP stents ($n = 5$) (used only distal to the left subclavian artery), Genesis stents (Johnson & Johnson, USA; $n = 1$) and Intrastent (EV3, Irvine, California, USA; $n = 2$). CP stents were preferred when the ends of the stent might protrude over the origin of a neck vessel—due to the structure of this stent, the distal end struts can separately be opened or flared if necessary. All stents were inflated manually, leaving full control to the operator during expansion in deciding whether to dilate pressure-resistant waists.

Test angiograms were done through the Mullins sheath during positioning of the stent and after inflation of the inner balloon. Pressure measurements and aortic angiograms were repeated after stent implantation. Cephazolin (50 mg/kg, maximum 2 g) was given 1 h before the procedure and at eight-hourly intervals (total of three doses). Post-procedure haemostasis of the femoral artery was obtained with local compression in 15 patients and a Prostar XL (Biomedicon, Mumbai, India) occlusion device in the last five patients. Heparin sulfate was neutralised when activated clotting time exceeded 250 ms. Patients were discharged the day after the procedure after transthoracic echocardiography and chest roentgenography. No long-term drugs (coagulation or aggregation drugs) were given except hypertension drugs when indicated. Follow up at the referring outpatient clinic was scheduled after 1–2 months and thereafter every 6–12 months. Catheterisation or computed tomography was repeated only when indicated.

Statistical analysis

Continuous data are presented as mean (SD); the median is also given when the distribution is non-normal. Changes were statistically evaluated with paired Student's *t* test, and $p < 0.05$ was considered significant.

RESULTS

Immediate results at stent implantation

A total of 23 stents were implanted in 20 patients: 13 stents in the aortic cross (three between the brachiocephalic trunk and left carotid artery, 10 between the left carotid and left subclavian arteries) and 10 stents in the isthmus; three of the patients required stenting of the cross and isthmus. Figure 1 shows an aortogram before and after stent implantation in the cross. The stents were dilated up to a mean diameter of 17 (2) mm. The origin of the left subclavian artery was electively completely crossed with the stent in four patients, without any clinical complications in short-term follow up (0.6–3.3 years). The left carotid artery was partially crossed in three patients, where the stent was additionally opened and flared (fig 2). The systolic gradient across the aortic arch decreased from 16 (6) (median 17) mm Hg to 3 (4) (median 1) mm Hg ($p < 0.001$) (fig 3A) and the mean diameter of the segment increased from 10 (2) mm to 17 (2) mm ($p < 0.001$) (fig 3B). In five patients a mild gradient persisted, often just distal to the left carotid artery due to residual orificial narrowing or due to acute angulation. No major complications occurred during or after the procedure; however, two patients had a moderate groin haematoma before the use of the transpuncture closing device.

Short-term follow-up data

When reassessed after one month, systolic blood pressure had decreased from 141 (12) mm Hg to 128 (6) mm Hg ($p < 0.001$). Arterial hypertension had resolved in 12 patients (below 95th centile for age) and eight required residual drugs (β blocker with or without angiotensin converting enzyme inhibitor) (table 1). However, patients treated with drugs either had better control of their arterial pressures or were taking a significantly lower dose. The follow-up has been 2.2 (1.4) years (range 0.2–4.8 years). Two more patients started taking hypertension drugs to adequately control arterial pressures.

DISCUSSION

Coarctation of the aorta often is a generalised disease of the aorta and, some authors even suggest, of the global arterial bed. Even after successful surgical repair of a coarctation, residual aortic arch abnormalities such as a narrowed arch segment, a high cervical arch, or an abnormal winding or acute angled cross may persist. Tubular hypoplasia of the

Figure 1 (A) Aortogram 15 years after neonatal Waldhausen subclavian aortoplasty combined with open aorta valvotomy (patient no 8). The coarctation site is well repaired without residual stenosis or gradient. However, the cross between the brachiocephalic trunk and the left carotid artery is hypoplastic (11 mm) with a 15 mm Hg systolic gradient. A 22 mm eight-zig Cheatham platinum stent is ready for deployment. (B) After implantation of the 22 mm stent on an 18 mm balloon, the segment had expanded up to 18 mm without a residual pressure gradient.

Figure 2 (A) Aortogram in a 17-year-old boy after implantation of a 28 mm eight-zig Cheatham platinum (CP) stent in the cross (between the left carotid and left subclavian arteries) and a 34 mm eight-zig covered CP stent in the isthmus (patient no 17). Both stents were dilated up to 16 mm. (B) Opening and flaring of the proximal end of the CP stent in the cross, partially crossing the origin of the left carotid artery (7 mm balloon). The flow to the left carotid artery was unrestricted.

aorta is a combination of a small diameter and an abnormal length; it can involve the transverse aorta and isthmus and does not invariably produce obstruction.¹³ Long-term follow up after surgical repair of coarctation has shown that early repair during infancy optimises arch growth and that late repair increases the risk for residual hypoplasia and late arterial hypertension.⁵ The incidence of re-coarctation or arch hypoplasia after surgical repair is related to the age at repair, the type of repair and how re-coarctation is defined.^{1,5} Neonatal surgical repair now aims at treating hypoplastic segments by extended end-to-end repair.

Rationale for the stent implantation

Recent guidelines define significant (re)coarctation in adults as resting or exercise-induced hypertension and a resting arm–leg blood pressure gradient ≥ 30 mm Hg.¹⁴ Patients with mild residual gradients at rest, like those in our study, would not be considered for (re)intervention on the basis of these guidelines but may still have raised blood pressures,

exposing them to increased cardiovascular risk with premature morbidity and mortality.^{2,4,5} Coronary artery disease is the most common cause of late death after successful coarctation repair, and late systemic hypertension is largely held responsible for this premature coronary atherosclerosis.^{5,15} One can speculate that any gradient across the aortic arch will enhance arterial hypertension, among other factors such as increased stiffness of the upper vascular tree and altered pressure wave transduction.

Surgical options to treat residual hypoplasia of the aortic cross or isthmus are extensive aortic arch reconstruction under complex cardiorespiratory bypass, ascending aorta to thoracic aorta crossover or placement of an ascending aorta to abdominal aorta conduit. These surgical procedures confer some morbidity and mortality and, when compared with interventional catheterisations, longer hospitalisation, a longer rehabilitation period and relatively higher costs. Suggesting these major operations for only mild residual aortic arch narrowing may be considered excessive, and for many patients the early discomfort and risks exceed the late potential benefits. Drug treatment alone usually decreases blood pressure, but often very high doses of hypertension drugs are needed, not infrequently associated with significant side effects during long-term follow up.

In view of the high effectiveness and safety profile of transcatheter techniques and the possible benefits of complete resolution of any gradient, stenting of residual hypoplastic segments seemed to be an elegant alternative treatment option.

Results of stent implantation and follow-up

In this paper we report our early experience of stent expansion of hypoplastic aortic segments in patients with arterial hypertension, mostly after previous coarctectomy. All patients had significant and relevant gradient reduction and half had normalisation of upper limb blood pressure. Ten patients still needed hypertension treatment but with better control or at a significantly lower dose than before the intervention. It is known, however, that many patients, even without a residual gradient across the aortic arch, will develop arterial hypertension during follow-up.^{2,3} Life-long follow-up remains mandatory.

All stents were inflated manually, allowing gentle and gradual expansion not exceeding 3–5 atm. This leaves full control to the operator in deciding whether to dilate

Figure 3 (A) Systolic gradient across the aortic arch and (B) diameter of the hypoplastic segment before and after stent implantation. Mean (SD).

pressure-resistant waists. We speculated that overdilation of such pressure-resistant hypoplastic segments can severely damage the vessel wall, with a higher risk for transmural aortic tear or rupture. The segments that were treated with stent expansion were usually free of post-surgical scars or adhesions. Therefore, even a small tear can be catastrophic, which is unacceptable, especially when a procedure is done for prognostic reasons. Emergency deployment of a covered stent as a bailout is not safely applicable because of the proximity of the origin of the neck vessels. This strategy resulted in some residual narrowing with minimal gradient in some of the patients; we thought that the potential late benefits did not outweigh the possible early risks. Additional experience is required to determine whether these pressure-resistant waists can be dilated at the initial or later catheter intervention if indicated.

No major complications (stent migration or fracture, aortic wall dissection, rupture or aneurysm formation) were seen at stent placement and during follow up. Late aneurysm, forming unexpectedly and silently after stent implantation, has been described.¹⁶ Close follow up of these patients is therefore mandatory. Routine imaging techniques other than transthoracic echocardiography have been advocated after stent implantation for treatment of (re-)coarctation to avoid silent complications. However, in this series we performed in most patients only a transthoracic echocardiography at follow up, as a stent in the cross or isthmus can be adequately evaluated by echocardiography (because of the short distance, and the stent more perpendicular to echocardiograph beam).

The concern that the stent can cross important branch vessels arising from the aorta has previously been discussed.^{17, 18} Crossing a vessel may theoretically lead to thromboembolism, vessel narrowing and branch occlusion. Patency of side branches from small coronary arteries, larger pulmonary arteries and branch vessels of the aorta, even after prolonged stent placement, has been described.¹⁹ The left subclavian artery was electively crossed with the stent in four patients, without any complications in short-term follow up.

In our patient group we opted to treat mild residual hypoplasia only at an age when an (near) adult-sized stent could be implanted, thereby avoiding the necessity for later redilatation. Late redilatation may be associated with aortic dissection at the distal end of the stent,²⁰ dehiscence of the endothelium or in-stent peel into the side branch, with the risk of embolic events if any head vessel is crossed.

Increased stiffness and altered vascular relaxation reserve in the upper body vessels have been implicated in the aetiology of hypertension at rest and during exercise after coarctation repair.^{21–23} After stent placement patients may theoretically develop exercise-induced gradients across the stent, as it is a rigid, metallic structure. However, preserved overall aortic compliance has been documented in animal models after stent implantation in the aorta.²⁴ Similarly, many surgical techniques leave the vascular tree with stiff segments.

Study limitations

The final goal of these procedures is to prolong survival without vascular complications in patients with aortic arch abnormalities. Only long-term follow up in three or four decades will determine whether this objective will be met. The observation that arterial hypertension regressed or became easier to treat suggests a more favourable outcome. Ideally, future studies should have 24 h blood pressure monitoring before and at regular intervals after the procedure, comparing different treatment strategies in patients with various degrees of hypertension (ambulatory

hypertension, daytime hypertension on 24 h monitoring and exercise-induced hypertension).

Conclusion

Pressure loss due to hypoplastic aortic segments can be treated effectively and safely with stent implantation.

Many patients remain mildly hypertensive and will require additional drug treatment, with increasing incidence during follow up. The risks and benefits of intervention for even mild hypoplastic segments are favourable; the late morbidity of residual hypertension may be decreased, delayed or even avoided. Our limited experience confirms that the subclavian artery can be safely crossed with a stent, at least during short- and medium-term follow up. Lifelong follow up of these patients remains mandatory.

Authors' affiliations

D Boshoff, W Budts, I Mertens, B Eyskens, T Delhaas, B Meyns, W Daenen, M Gewillig, Paediatric and Adult Congenital Heart Unit, University Hospitals Leuven, Leuven, Belgium

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Severe hypothermia showing Osborn waves associated with transient atrial fibrillation and ST segment depression

An 80-year-old woman with no known cardiovascular risk factors was found unconscious at home with a body temperature $< 30^{\circ}\text{C}$ ($< 86^{\circ}\text{F}$). On arriving at the emergency department, her ECG (panel A) showed an atrial fibrillation (AF) with ST segment depression from V3 to V6. In leads V5 and V6 there was a terminal extra QRS deflection formed by the junction between the J point and the ST segment, consistent with the so-called Osborn waves (panel C). Serum electrolytes and cranial computed tomography (CT) were normal. After re-warming to a body temperature of 36.7°C (98°F), the patient recovered sinus rhythm (panel B) and the ST segment depression and the Osborn waves disappeared.

Hypothermia (body temperature $< 35^{\circ}\text{C}$ ($< 95^{\circ}\text{F}$)) is initially associated with sinus tachycardia, but with temperatures $< 32.2^{\circ}\text{C}$ ($< 90^{\circ}\text{F}$) sinus bradycardia supervenes, associated with a progressive prolongation of the PR interval, QRS complex, and QT interval. At temperatures $\leq 30^{\circ}\text{C}$ ($\leq 86^{\circ}\text{F}$), atrial ectopic activity can be triggered, eventually progressing into atrial fibrillation (AF). At this level of hypothermia, 80% of the patients have Osborn waves consisting of an extra deflection at the end of the QRS complex best depicted on the inferior and lateral leads. Osborn waves become more prominent as the temperature drops, and they disappear gradually with re-warming. The combination of transient Osborn waves and AF in the hypothermia setting, with the recovery of sinus rhythm after re-warming, has been rarely described. Although several ECG alterations have been described in accidental hypothermia, this is the first report of asymptomatic transient ST segment depression with recovery after re-warming.

J Benezet-Mazuecos
B Ibanez
J Farre
jbenezet@yahoo.es

(A) Emergency department 12 lead ECG obtained at core body temperature $< 30^{\circ}\text{C}$ ($< 86^{\circ}\text{F}$) showing atrial fibrillation (AF) with ST segment depression from V3 to V6. In leads V5 and V6 there was a terminal extra QRS deflection formed by the junction between the J point and the ST segment, consistent with the so-called Osborn waves. (B) 12 lead ECG obtained after patient had been re-warmed to 36.7°C (98°F); the patient recovered sinus rhythm and the ST segment depression and the Osborn waves had disappeared. (C) Detail of the Osborn wave, also known as J waves, camel-hump waves or hypothermic waves.