Results of Stenting for Aortic Coarctation

Mariano Ledesma, MD, Carlos Alva, MD, Felipe David Gómez, MD, Agustín Sánchez-Soberanis, MD, Enrique Díaz y Díaz, MD, Carlos Benítez-Pérez, MD, Rodolfo Herrera-Franco, MD, Rubén Arguero, MD, and Ted Feldman, MD

Endovascular stents have been widely used in peripheral and coronary arteries, postsurgical stenosis of the pulmonary arteries, and in the superior vena cava and conduits in congenital heart diseases.[1–8] Several reports support the use of balloon-expandable stents for aortic coarctation (AC) in humans.[9–13] This study reports the results obtained with stent implantation in young and adult patients with AC.

• • •

Between September 1996 and August 2000, 56 stents were implanted in 54 patients with AC (35 male, 19 female; age range 8 to 49 years, mean 22 ± 9). AC was defined as a stenosis with a peak-to-peak gradient of ≥20 mm Hg at rest. Fourteen patients had associated malformations: subaortic stenosis (2), ventricular septal defect (2), patent ductus arteriosus (1), coronary disease (2), and aortic valve disease (7). Four patients had undergone balloon angioplasty 2.5 to 7 years before the study. Two had restenosis and 2 had small saccular dilations. One case had surgical end-to-end repair with restenosis. All patients had localized AC, but 1 had a long tubular stenosis. Clinical success was defined as a peak-to-peak pressure gradient of ≤20 mm Hg after stenting with no major complications. Technical success was defined as successful stent deployment without complications. Clinical and Doppler examinations were performed at 1 month and every 6 months. Pressure gradients before and after stenting were compared using Student's *t* test (paired, 2-tail). A p value of <0.05 was considered significant.

All cases were sedated. Antibiotic prophylaxis was not used. After arterial access, all patients received 100 U/kg of heparin. Activated clotting time values were not measured. Gradient and arch angiography were assessed. We used 55 Johnson & Johnson stents (41 P-308, 12 P-4014, and 2 P-5014, Warren, New Jersey) and one 60-mm-long Wallstent (Boston Scientific Corporation, Natick, Massachusetts). Stents were selected for the diameter of the proximal aorta. The Wallstent was used for a long stenosis. Predilation was performed in only 3 patients. A stiff Amplatz guidewire was used with a 80-cm-long transseptal 9Fr sheath (Cook, Bloomington, Indiana) with a P-308 stent, or a 11Fr sheath with the P-4014 or P-5014 stents. A dilator with a sheath was advanced across the AC. The dilator was removed, leaving the sheath and wire. Afterward, the stent was manually crimped on a balloon with a balloon-to-isthmus ratio of approximately 1.0 to 1.2. Maximum balloon size was 25 mm. The stent was advanced to the stenosis and the sheath was withdrawn, exposing the stent. The balloon was inflated to 3 to 6 atm using a 20-ml inflator. Pressures and angiography were repeated. Heparin was restarted 4 hours after sheath removal and infused for 24 hours. Postprocedure heparin infusions were used to minimize femoral thrombotic complications, not for stent anticoagulation.

Successful deployment was achieved in 53 patients (98%). Fifty-two patients (96%) had clinical success. The 1 failure was due to a residual gradient of 30 mm Hg after stenting for restenosis after prior surgical repair. Although no high-pressure balloon was available to us, there was no fluoroscopic calcification of the AC. This patient underwent a successful reoperation. Mean pressure gradient significantly decreased, from 50 ± 20 mm Hg (range 11 to 110) to 5 ± 8 mm Hg (range 0 to 30, $p<0.001$). In 29 patients there was no gradient after stent placement (Figure 1). In the patient with long tubular AC (17 years old, 57 kg), the stenotic site was predilated with an 8-mm balloon to place a Wallstent (16 × 60 mm length). The stent did not totally expand, moving to the distal side of the AC. It was thus necessary to place a P-308 stent, which resulted in expansion of the Wallstent. Fourteen months later, a small aneurysm was seen around the Wallstent. The aneurysm did not involve the stent ends, and may have been due to overdilation of the balloon (Figure 2). Magnetic resonance images showed no growth of the aneurysm 9 months later. In the 2 cases in which an aneurysm occurred after previous balloon angioplasty, stent implantation was enough to resolve the problem (Figure 3).

One patient experienced vagal bradycardia that required atropine. Two patients had paradoxical hypertension immediately after positioning the stent, which was treated with oral β blockers for 3 weeks. In 1 case the stent was not delivered because balloon inflation moved the stent distally before the stent expanded. In this patient, the stent was trapped in the femoral artery when pulled back to the sheath. It was removed by surgery a few days later, and the AC was surgically corrected.

Two cases had stent migration during balloon inflation. In the first, the stent deployed 2 mm below the AC site. There was complete resolution of the AC as a result of the balloon dilation and nothing further was done. This patient developed an aneurysm of the unstented site. In the second patient, we implanted a second stent to cover the dilation site after the first stent moved distally. There were no cases where balloon rupture caused stent migration.

From the Cardiology Hospital, National Medical Center 21st Century, Mexican Institute of Social Security, Mexico City, Mexico; and University of Chicago Hospitals, Chicago, Illinois. Dr. Ledesma's address is: Privada de Corregidora #5, San Jerónimo Lídice. C.P. 10200, Mexico D.F. E-mail: ledesmav@prodigy.net.mx. Manuscript received December 4, 2000; revised manuscript received March 13, 2001.

0002-9149/01/$–see front matter
PII S0002-9149(01)01705-2

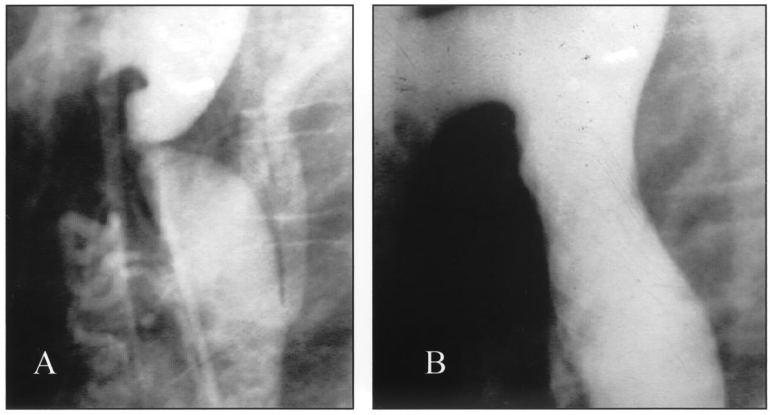

FIGURE 1. ***A,*** **the lateral view of discrete AC.** ***B,*** **lateral view after implantation with a P-308 Johnson & Johnson stent. Satisfactory anatomic correction was achieved.**

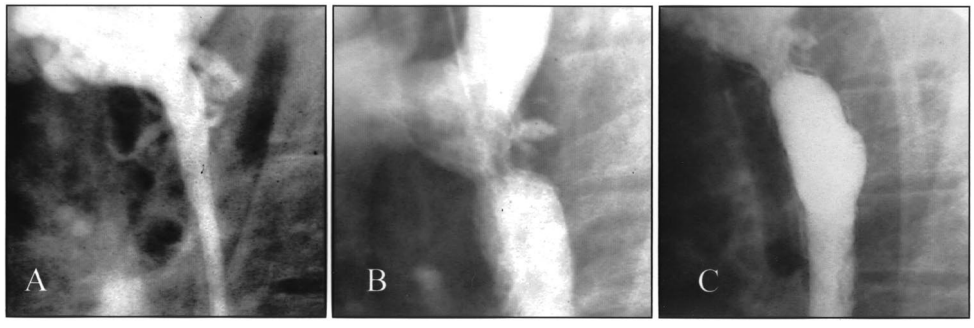

FIGURE 2. ***A,*** **the lateral view before stenting. This patient had severe tubular and hypoplastic AC.** ***B,*** **the lateral view after implantation of a Palmaz P-308 stent proximal to the Wallstent. There was a persistent waist with a 20 mm Hg gradient. The diameter of the tubular segment increased from 2.5 to 15 mm.** ***C,*** **the lateral view 14 months later shows an aneurysm around the Wallstent. The Palmaz stent was redilated and the gradient decreased from 25 to 15 mm Hg. The stents separated, leaving a small gap.**

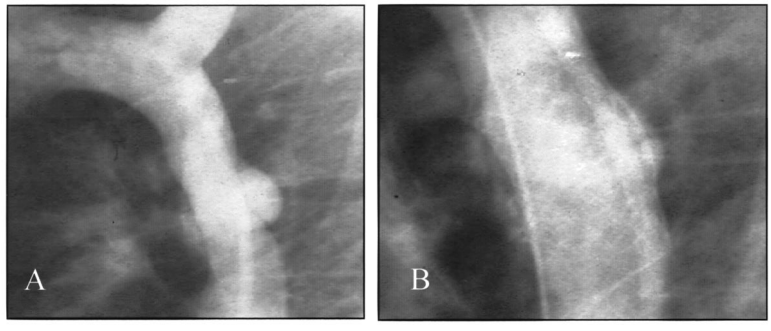

FIGURE 3. ***A,*** **the lateral view showing a small aneurysm in posterior aortic wall (balloon angioplasty 5 years before).** ***B,*** **the lateral view after implantation of a Palmaz P-308 stent, with satisfactory correction of the aneurysmatic dilation.**

At a mean of 25 months (2 to 52), we decreased the doses of antihypertensive drugs, and in 30 patients drugs were withdrawn. All patients remained asymptomatic with normal femoral pulses. Maximal follow-up gradients measured by Doppler or catheterization in 36 patients were between 0 and 41 mm Hg (mean 7 $\pm$ 10). Fifteen patients had a 0 mm Hg gradient. Eleven were reevaluated 11 to 37 months later (mean 22) with intravascular ultrasound, demonstrating minimal intimal tissue growth (Figure 4). The patient with distal migration of the stent developed aortic dilatation proximal to it 2 years later. The patient with long tubular coarctation and a Wallstent developed recoarctation and a small aneurysm around the Wallstent 14 months later and was managed medically (Figure 2). Two stents were redilated at follow-up. No stent strut fractures have been seen on follow-up chest x-rays.

• • •

This analysis in 53 patients with AC who were treated with stenting demonstrates good clinical and hemodynamic results. In most patients direct stenting was used. Direct stent placement without predilation decreases the risk for stent migration.[14] Predilation makes it more difficult to make contact with the stenosis segment, so stent movement before it is fixed in position may occur. Despite this, 1 stent embolized distally in this experience. Although it is possible to deploy embolized stents in the iliac vessels, we were unable to do this and had to resort to surgical removal. The only patient with re-AC had a long tubular coarctation. The aorta was dilated from 2.5 to 15 mm with dissection of a long area and, consequently, formation of an aneurysm. Recatheterization showed that the Wallstent and adjacent P-308 stent had separated, leaving a small gap between them. Thus, with a large difference in lumen diameter between the coarcted segment and the adjacent aorta, a possible strategy is to treat the AC in stages, first with a stent of diameter from 8 to 10 mm, and then in 3 months to redilate with a bigger balloon. Surgery is another alternative.

A potential advantage of stent implantation over using balloons alone is that stents may decrease the incidence of restenosis. This technique is probably not applicable in infants when the aorta is still growing. The possibility of reexpanding a stent several years after implantation has been described, but the risks have not been fully elucidated.[15–18]

In summary, in selected patients with AC, stent implantation is a safe alternative to balloon angioplasty alone, with minimal morbidity and no mor-

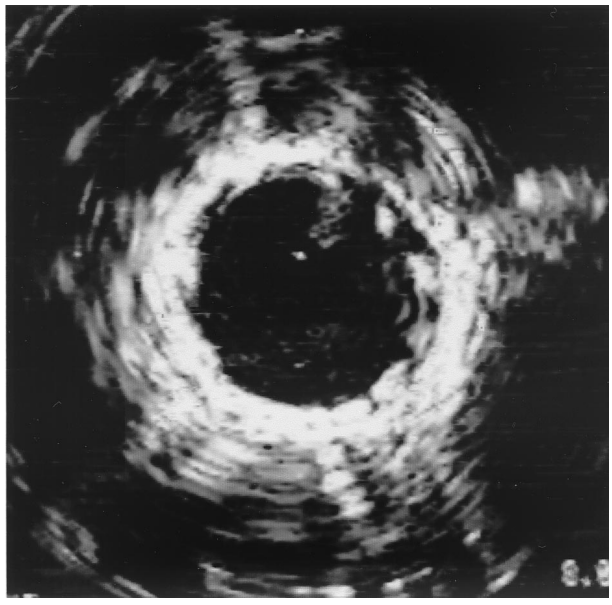

FIGURE 4. Intravascular ultrasound imaging. The Palmaz stent P-308 shows no intimal growth, 22 months after stent implant. Some tissue prolapse is noted at the 1 o'clock position.

tality. Stents eliminate or reduce the gradient and avoid surgical correction.

1. Hellenbrand WE, Allen HD, Golinko RJ, Hagler DJ, Lutin W, Kan J. Balloon angioplasty for aortic re-coarctation. Results of valvuloplasty and angioplasty of congenital anomalies registry. *Am J Cardiol* 1990;65:793–797.
2. Hijazi ZM, Fahey JT, Kleinman CS, Hellenbrand WE. Balloon angioplasty for recurrent coarctation of aorta: immediate and long-term results. *Circulation* 1991;84:1150–1156.
3. Morrow WR, Vick GW III, Nihill MR, Rokey R, Johnston DL, Hedrick TD, Mullins CE. Balloon dilatation of unoperated coarctation of the aorta-short and intermediate-term results. *J Am Coll Cardiol* 1988;11:133–138.
4. Lababidi ZA, Daskalopoulos DA, Stoeckle H. Transluminal balloon coarctation angioplasty. Experience with 27 patients. *Am J Cardiol* 1984;54:1288–1291.
5. De Lezo JS, Sancho M, Pan M, Romero M, Olivera C, Luque M. Angiographic follow-up after balloon angioplasty for coarctation of the aorta. *J Am Coll Cardiol* 1989;13:689–695.
6. Palmaz JC, García O, Schatz RA, Rees CR, Roeren T, Richter GM, Noeldge G, Gardiner GA Jr, Becker GJ, Walker C, et al. Placement of balloon-expandable intraluminal stents in iliac arteries. First 171 procedures. *Radiology* 1990;174: 969–975.
7. Levine MJ, Leonard BM, Burke JA, Nash ID, Safian RD, Diver DJ, Baim DS. Clinical and angiographic results of balloon-expandable intracoronary stents in right coronary stenosis. *J Am Coll Cardiol* 1990;16:332–339.
8. O'Laughlin MP, Perry SB, Lock JE, Mullins CE. Use of endovascular stents in congenital heart disease. *Circulation* 1991;83:1923–1939.
9. Morrow WR, Smith VC, Ehler WJ, Van Dellen AF, Mullins CE. Balloon angioplasty with stent implantation in experimental coarctation of the aorta. *Circulation* 1994;89:2677–2683.
10. O'Laughlin MP, Perry SB, Lock JE, Mullins CE. Use of endovascular stents in congenital heart disease. *Circulation* 1991;83:1923–1939.
11. Redington AN, Hayes AM, Ho SY. Transcatheter stent implantation to treat aortic coarctation in infancy. *Br Heart J* 1993;69:80–82.
12. De Lezo JS, Pan M, Romero M, Medina A, Segura J, Pavlovic D, Martínez C, Tejero I, Navero JP, Torres F, et al. Balloon expandable stent repair of severe coarctation of the aorta. *Am Heart J* 1995;129:1002–1008.
13. Bulbul ZR, Bruckheimer E, Love JC, Fahey JT, Hellenbrand WE. Implantation of balloon-expandable stents for coarctation of the aorta: implantation data and short-term results. *Cathet Cardiovasc Diagn* 1996;39:36–42.
14. Fellows KE, Radtke W, Keane JF, Lock JE. Acute complications of catheter therapy for congenital heart disease. *Am J Cardiol* 1987;60:679–683.
15. Tynan M, Finley JP, Fontes V, Hess J, Kan J. Balloon angioplasty for treatment of native coarctation. Results of valvuloplasty and angioplasty of congenital anomalies registry. *Am J Cardiol* 1990;65:790–792.
16. Mendelsohn AM, Dorostkar PC, Morehead CP, Lupinetti FM, Reynolds PI, Ludomirsky A, Lloyd TR, Heidelberger K, Beekman RH. Stent redilatation in canine models of congenital heart disease: pulmonary artery stenosis and coarctation of the aorta. *Cathet Cardiovasc Diagn* 1996;38:430–440.
17. Morrow WR, Palmaz JC, Tio FO, Ehler WJ, Van Dellen AF, Mullins CE. Re-expansion of balloon expandable stents after growth. *J Am Coll Cardiol* 1993;22:2007–2013.
18. Shaffer KM, Mullins CE, Grifka RG, O'Laughlin MP, McMahon W, Ing FF, Nihill MR. Intravascular stents in congenital heart disease: short- and long-term results from a large single-center experience. *J Am Coll Cardiol* 1998;31:661–667.

Congential Heart Disease in the Medicaid Population of Southern Arizona

Alyse M. Baron, MD, Richard L. Donnerstein, MD, Eric Kanter, MD, F. John Meaney, PhD, and Stanley J. Goldberg, MD

Birth prevalence of congenital heart disease (CHD) in the general population is approximately 8/1,000 live births.[1–9] Approximately 4 million children are born each year in the United States, and of these, almost 1.3 million are born into poverty.[10] Assuming the same rate of CHD among children in poverty as in the general population, we would expect that approximately 10,400 infants with CHD would be born into families in poverty each year. Our clinical observations in Southern Arizona suggested that the prevalence of CHD may be increased among our Medicaid patients. Previous studies have not shown this to be true.[2,11] However, these data were collected in 1968 and from 1971 to 1984 and therefore may not apply today under different socioeconomic and health care circumstances. This study was thus designed to determine the risk of CHD in patients without chromosomal disorders whose mothers were on Medicaid at the time of their conception and/or birth compared with the CHD prevalence in patients whose mothers were not on Medicaid during either time period. It is important to indicate that Medicaid in Arizona is offered only to families or persons who fall at or below 30% of the federal poverty level. Therefore, this study will assess the occurrence of CHD in the

From the Department of Pediatrics, Steele Memorial Children's Research Center, Diamond Research Laboratory, and University Heart Center, University of Arizona College of Medicine, Tucson, Arizona.

This study was supported in part by donations on behalf of the Sean Coleman family, Tucson, Arizona.

Dr. Goldberg's address is: 1501 North Campbell Avenue, Tucson, Arizona 85724-5073. E-mail: sgoldber@peds.arizona.edu.

Manuscript received December 13, 2000; revised manuscript received and accepted March 15, 2001.

0002-9149/01/$–see front matter
PII S0002-9149(01)01706-4