The first successful application of endovascular stents to maintain vessel patency was reported by Dotter in 1969 (1). Its use in children with congenital heart disease (CHD) was first demonstrated in the early 1990s (2,3). Stents have been used to treat pulmonary artery stenosis (congenital and acquired) (3–5), coarctation of the aorta (6,7), systemic and central venous obstructions (8), postoperative conditions, including cavo pulmonary communications and conduit/homograft obstruction (9), as well as in attempts to maintain ductal patency (10). Initially, there were concerns about the potential for stent restenosis and the ability with which subsequent balloon dilation could be performed. One study demonstrated a 3% rate of restenosis and neointimal proliferation at a mean follow-up of 13 months (11). However, the incidence of restenosis and neointimal proliferation at intermediate- to long-term follow-up remains unanswered.

**METHODS**

We retrospectively analyzed all patients who underwent redilation of endovascular stents (Palmaz Johnson & Johnson, Piscataway, New Jersey) between September 1989 and February 2000. All of the patients’ demographic data were studied (age, weight, gender, diagnosis, past surgical history), including the interval from the first stent implant to the time of redilation and subsequent redilations. Patients were classified into one of three groups according to the location of the stent implant. The cardiac catheterization data at the initial stent implant and at redilation were examined to determine the pressure gradient, lumen diameter, number and dimension of stents initially implanted, reduction in the pressure gradient (in appropriate cases expressed as the right ventricle [RV] to femoral artery [FA] pressure ratio), increase in vessel diameter and occurrence of complications.

Restenosis was defined as a reduction in the stent lumen diameter less than the adjacent nominal vessel diameter or development or increase in the pressure gradient across the stent, or both.
Abbreviations and Acronyms
ASO = arterial switch operation
CBPS = congenital branch pulmonary stenosis
CHD = congenital heart disease
FA = femoral artery
IVC = inferior vena cava
PA = pulmonary atresia
RV = right ventricle or ventricular
SVC = superior vena cava
TOF = tetralogy of Fallot
TR = tricuspid regurgitation

Neointimal proliferation was defined as a lining between the stent and the lumen, as seen on the angiogram. Mild or physiologic intimal hyperplasia was defined as <1 mm on either side of the stented vessel; moderate as 1 to 1.5 mm; and severe as >1.5 mm.

Recatheterization. Our practice has been to recatheterize patients one to three years after stent implantation, or when their weight has increased sufficiently to have hemodynamic consequences. Repeat catheterization is also indicated when the adult’s weight is reached to achieve the full dilation potential of the stented vessel.

Statistics. Comparisons of the pre-redilation and post-redilation differences in the stent’s maximal gradient, minimal vessel diameter and change in the RV/FA pressure ratio were made using a two-tailed t test. Statistical significance was set at p < 0.05.

RESULTS

Of 368 patients with a total of 752 stents implanted, 220 were recatheterized. Of those 220 patients, 103 underwent stent redilation (76 patients were redilated once, 22 patients twice and 5 patients three times). Indications for repeat catheterization in the 220 patients included patients in the initial part of the study protocol during our early experience with stent implantation (generally one year after the initial stent implant), patients who had doubled their body weight since the initial stent implant and patients who developed evidence of increased right heart pressures (clinically or echocardiographically with estimation of RV pressure from tricuspid regurgitation [TR] jet peak velocities). The patients’ median age was 9.9 years (range 0.5 to 39.8). The male to female ratio was 1.5:1; their mean weight was 36 kg (range 5.3 to 102). The mean follow-up duration was 3.8 years (range 0.1 to 10). The mean interval from stent implantation to redilation was two years (range 0.1 to 7.5). The mean duration from redilation to follow-up was two years (range from 0.1 to 7.4). Indications for stent redilation included somatic growth (n = 67), serial dilation (n = 27) and development of neointimal proliferation and restenosis (n = 9). The overall mean vessel diameter increased from 8.8 to 12.8 mm (p < 0.003). For patients with branch pulmonary stenosis (i.e., tetralogy of Fallot [TOF]/pulmonary atresia [PA], congenital branch pulmonary stenosis [CBPS], arterial switch operation [ASO]), the mean arterial gradient decreased from 20 to 6 mm Hg (p < 0.001), and the RV/FA ratio decreased from 0.5 to 0.39 (p < 0.001).

The 103 patients analyzed were classified into three groups:

1) pulmonary artery stent group (n = 94). They were further classified into four subgroups (a–d).

a) status after repair of TOF and PA subgroup (n = 72). Of 142 patients with stents placed for TOF/PA who were recatheterized, 72 were redilated (51%). The mean systolic gradient at redilation decreased from 20 (range 0 to 70) to 6 mm Hg (range 0 to 40) (p < 0.001). The mean vessel diameter increased from 9.4 (range 3 to 19) to 13.2 mm (range 5 to 24) (p < 0.001). The mean RV/FA pressure ratio decreased from 0.48 (range 0.2 to 1.1) to 0.4 (range 0.2 to 0.9) (p < 0.001).

b) CPBS subgroup (n = 9). Of 23 patients with stents placed for CPBS who were recatheterized, 9 were redilated (39%). The mean systolic gradient at redilation decreased from 19 (range 3 to 40) to 8 mm Hg (range 0 to 22) (p = 0.004), and the mean vessel diameter increased from 8.3 (range 5 to 12) to 11.2 mm (range 8 to 16) (p < 0.001). The mean RV/FA pressure ratio decreased from 0.59 (range 0.4 to 1.2) to 0.46 (range 0.3 to 0.9) (p = 0.03).

c) Status after Fontan operation subgroup (n = 6). Of 16 patients with stents placed in the Fontan operation subgroup who were recatheterized, 6 were redilated (38%). The mean systolic gradient at redilation decreased from 3 (range 0.5 to 7) to 0.5 mm Hg (range 0 to 2) (p = 0.03). The mean vessel diameter increased from 10.5 (range 7 to 13) to 14 mm (range 10 to 17) (p < 0.003). A comparison of the RV/FA pressure ratio was not applicable for this group.

d) Status after ASO subgroup (n = 7). Of 16 patients with stents placed in this subgroup who were recatheterized, 7 were redilated (43%). The mean systolic gradient at redilation decreased from 27 (range 9 to 40) to 7 mm Hg (range 2 to 11) (p = 0.001). The mean vessel diameter increased from 8.0 (range 2 to 12) to 12.6 mm (range 8 to 16) (p = 0.001). The mean RV/FA pressure ratio decreased from 0.52 (range 0.3 to 0.6) to 0.35 (range 0.3 to 0.4) (p = 0.004).

2) Iliofemoral vein occlusion group (n = 6). Of 16 patients with stents placed for iliofemoral vein occlusion who were recatheterized, 6 were redilated (38%). The mean vessel diameter at redilation increased from 2.0 (range 0 to 6) to 6.3 mm (range 2 to 12) (p = 0.04). Measurement of the RV/FA pressure ratio and pressure gradient were not applicable for this group.

3) Miscellaneous group (coarctation of the aorta, Blalock-Taussig shunt and superior vena cava [SVC] conduit) (n = 3). In the patient with coarctation of the aorta, the systolic gradient at redilation decreased from 80 to 14 mm Hg, and the vessel diameter increased from 3.6 to 7.8 mm. The vessel diameter increased at redilation from 13 to 17 mm in the patient with SVC conduit stenosis, and from 5 to 7 mm Hg.
in the patient with Blalock-Taussig shunt stenosis. Measurement of the RV/FA pressure ratio was not applicable for this group.

**Neointimal proliferation.** Of the 220 patients who underwent repeat catheterization, 208 had no significant neointimal proliferation (mild in 8 patients). Significant neointimal proliferation was present in four patients (1.8%), moderate in two and severe in two. Those with moderate neointimal proliferation were redilated, and those with severe neointimal proliferation underwent additional stent implantation. The neointimal thickness was reduced in all patients after redilation, with or without additional stent implantation.

**Restenosis.** Restenosis developed in 5 (2%) of 220 patients who were recatheterized. Specific risk factors were identified for the development of restenosis, which included:

- A lack of overlap in tandem stents (n = 1). A patient with CBPS developed restenosis in the right pulmonary artery after minimal stent separation of 1 to 2 mm. The same patient did not develop restenosis in the contralateral pulmonary artery, which had 8 to 10 mm of stent overlap (Fig. 1).
- Sharp angulation of the stent to the vessel wall (n = 1). In one of the early patients in our cohort, placement of a stent with sharp angulation to the curvilinear pulmonary artery wall resulted in development of restenosis at the distal end of the stent. This probably occurred as a reaction of the vessel wall to the sharp stent struts (Fig. 2).
- Overdilation of the stent (n = 1) (Fig. 3).
- Stent implanted in the abnormal tissue (n = 2). Two patients with presumed abnormal tissue characteristics also developed restenosis. One patient who had the ASO and Lecompte maneuver performed developed severe diffuse hypoplasia of both branch pulmonary arteries. At the time of the initial stent implant after bilateral sequential stent implantation, the stents in the right pulmonary artery had already “contracted down” (Figs. 4 and 5). At follow-up, he had developed bilateral restenoses, which were successfully redilated, with a reduction in the RV/FA pressure ratio from 0.62 to 0.34 and an increase in the stent’s vessel diameter by 20%. Another patient with Williams syndrome with severe diffuse pulmonary artery hypoplasia developed restenosis after redilation, despite a 50% increase in the vessel diameter.

**Complications.** Of 103 patients who underwent stent redilation, complications occurred in 4 (2%) of 135 procedures. Complications included:

- Pulmonary edema (n = 1). This patient with TOF and an absent left pulmonary artery previously underwent stent implantation in the right pulmonary artery. Secondary to supra-systemic RV pressure redilation of two right pul-

**Figure 1.** Angiograms of the pulmonary arteries, delineating a lack of sufficient stent overlap. a) Two stents in the right pulmonary artery nearly became separated. b) Intimal build-up noted at the site of insufficient overlap and not in the remainder of the stents.

**Figure 2.** Angiograms of the right pulmonary artery, delineating sharp angulation (right anterior oblique and cranial projections). a) Initial curvature of the vessel before stenting. b) Stenosis at recatheterization after the initial stent implant. c) Restenting with several shorter overlapping stents.
Pulmonary artery stents and balloon dilation of five distal pulmonary branches were performed, with a reduction in the RV/FA ratio from 1 to 0.7.

- Hemoptysis (n = 1). After TOF repair, hemoptysis occurred after redilation of a right pulmonary artery stent; the hemoptysis resolved without sequelae.
- Stent compression (n = 2). Stent compression occurred after redilation of the contralateral branch stent in a patient who had TOF repair and in another patient after PA/ventricular septal defect repair. The stent was removed at the time of conduit replacement in the latter patient. This contralateral stent compression complication occurred early in our experience; this complication is now avoided by simultaneous bilateral balloon inflation of branching stents.

In all stent redilation procedures, there were no transfusions, stent embolizations, emergent operations or deaths.

**DISCUSSION**

Over the last decade, the application of endovascular stents in patients with CHD has proven very successful. Stents have been particularly effective in the treatment of postoperative pulmonary artery branch stenoses and systemic venous stenoses, for which further surgical treatment is limited. Isolated balloon angioplasty of arterial and venous stenoses has been shown to be inadequate, with early follow-up demonstrating restenosis rates as high as 40% (12,13). Redilation of endovascular stents may be required to accommodate the patient’s somatic growth (not previously described), for staged, serial “further” dilation to avoid vessel overdilation during stent implant, for neointimal proliferation or for the development of restenosis. Early follow-up studies demonstrated very low rates of significant stent neointimal proliferation and/or restenosis, occurring in only 3% of patients, with a mean duration of follow-up of 13 months (11).

**Restenosis.** We defined restenosis as a reduction in the stent lumen diameter less than the nominal adjacent vessel diameter and/or the development of or increase in the pressure gradient across the stent. However, it is important to identify those cases where the stented vessel diameter has remained the same since the initial implant and the adjacent vessel to the stent has undergone further growth. This may give the appearance of restenosis on the angiogram, but with no change in the stent’s vessel diameter, it does not

---

**Figure 3.** Angiograms of the pulmonary arteries, delineating overdilation of the right pulmonary artery at the initial stent implant. a) Stretched/elongated right and left pulmonary arteries before stent placement. b) Neointimal proliferation of the left pulmonary artery at the time of recatheterization. c) Angiogram of the left pulmonary artery after redilation.

**Figure 4.** Anteroposterior projection of the pulmonary arteries, delineating abnormal tissue characteristics. a) The branch pulmonary arteries after the initial balloon dilation appear stretched and elongated. b) The proximal vessel is “contracted down” after placement of the distal stent. c) The proximal stent is placed in the same setting.
represent true restenosis. This is one of the primary reasons for repeating catheterization one to three years after the initial stent implant, so one can assess vessel growth adjacent to the stent and the need for further stent dilation to accommodate somatic growth.

Only one stent occlusion was found (0.5%) (left femoral vein/inferior vena caval stenosis after a long-term indwelling central line within the stent). Restenosis occurred in only 2% of our patients, slightly less than the rate found in another study at early follow-up in a smaller cohort of patients (11). In each case, specific risk factors were identified, including inadequate stent overlap, overdilation of the stent and abnormal underlying vascular tissue. A lack of overlap in tandem stents may result in movement of the stents relative to each other, which may traumatize the intervening vessel wall. Likewise, sharp angulation of the end of a stent results in struts penetrating into the vessel wall, which may act as a template for the development of restenosis or neointimal growth.

The identification of these factors, which predispose to stent restenosis, has enabled us to improve our technique and results. Specifically, we ensure adequate stent overlap, and, if necessary, we implant several short, overlapping stents along the vessel curvature to allow better alignment with the vessel wall. Although we have had limited experience with them, recently developed stents with increased flexibility (Intratherapeutics stents, St. Paul, Minnesota) may allow for better vessel–stent alignment. Modification of stent technique, however, in those patients with intrinsically abnormal vascular tissue is difficult, and restenosis in these patients may be less indicative of stent failure, but an unavoidable response of the underlying abnormal vascular tissue.

Neointimal proliferation. One recent study using self-expanding Wallstents in a small group (25 stents implanted in a total of 22 procedures) reported an incidence of neointimal proliferation of 28% (14). They defined neointimal proliferation as neointimal growth >30% of the vessel diameter, reporting its development in five pulmonary artery stents, one left SVC stent and one modified Blalock-Taussig shunt; one of the pulmonary artery stents was totally occluded. These results are markedly different from our experience using balloon-expandable (Palmaz) stents. Even when we used our stricter definition of neointimal proliferation, our patients developed significantly less neointimal proliferation (4%). Avoidance of initial overdilation of the stent appears to result in a reduction in the risk of neointimal proliferation, and hopefully avoidance of aneurysm formation and vessel perforation.

Somatic growth. Initially, there were concerns about the potential for stent redilation to accommodate somatic growth. Published animal studies by Grifka et al. (15) and Morrow et al. (16) unequivocally demonstrated the potential to increase the diameter of previously implanted, balloon-expandable stents using larger balloons to accommodate for somatic growth. We advocate staged, serial further dilation of arterial and venous stents, rather than attempting to gain a maximal vessel diameter at the initial stent implant, because this appears to predispose the patient to neointimal proliferation and restenosis and may increase the risk of complications. There was no significant disparity between the need for redilation between the groups, given that somatic growth was common to all groups.

Study limitations. Although the results of this study are encouraging, demonstrating improved hemodynamic data, low rates of restenosis and neointimal proliferation at intermediate-term follow-up, there are a number of study limitations. This study group represents one center’s experience and may not mirror that of the general interventional community. Catheterization data were based on retrospective analysis of the catheterization procedures. Also, we defined neointimal proliferation as significant if it was >1 mm on either side of the stent wall, an arbitrary measure that may not be agreed upon by other institutions. Finally, the mean follow-up period of our patients was 3.8 years (range 0.1 to 10), which allows us to comment only on intermediate-term outcomes.
Conclusions. Redilation of endovascular stents for patients with CHD is effective as late as 10 years after the initial stent implant. The majority of patients will require stent redilation to accommodate somatic growth. The risk of significant neointimal proliferation (1.8%) and restenosis (2%) is low and possibly avoidable, and this subset of patients may require redilation. Modification of the stent implantation technique, including avoidance of minimal stent overlap, of sharp angulation of the stent to the vessel wall and of overdilation, has reduced the incidence of restenosis. Awareness of these factors and increased operator experience has resulted in a low complication rate over the last decade. These data continue to advocate the use of endovascular stent implantation as the treatment of choice for both arterial and venous stenoses for many congenital heart defects.

Reprint requests and correspondence: Dr. Howaida G. El-Said, Division of Pediatric Cardiology, Texas Children’s Hospital, 6621 Fannin, MC 2-2280, Houston, Texas 77030. E-mail: hgelsaid@texaschildrenshospital.org.

REFERENCES