Treatment of restenosis of coarctation by percutaneous transluminal angioplasty

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EDWARD J. FARMLETT, M.D., JAMES S. DONAHOO, M.D., AND TIMOTHY J. GARDNER, M.D.

ABSTRACT Percutaneous transluminal angioplasty (PTA) was used successfully to treat coarctation restenosis in seven patients. The patients were 10 months to 17 years old at the time of the angioplasty, and the initial coarctation repair had been performed 10 months to 16 1/2 years previously. PTA reduced the systolic pressure gradient across the coarctation from a mean of 58 mm Hg before the procedure to a mean of 13 mm Hg immediately after PTA. Follow-up has been from 1 to 14 months and indicates that the decrease in the systolic pressure gradient is persistent.


Surgical repair of coarctation of the aorta has been performed since 1945. Recent reviews have indicated that there is a high incidence of recurrence of stenosis at the anastomotic site, particularly if the primary repair is performed in infancy. If surgical correction is delayed into later childhood, there would be a risk of persistent hypertension, although the risk of recurrent stenosis would be lessened.

The mechanism of recurrence of stenosis at the coarctation site has been considered to be the result of one or more factors: (1) incomplete relief of the obstruction at the time of the initial repair, (2) incomplete resection of "abnormal" aortic tissue that may have a tendency to proliferate, (2) failure of the anastomotic site to grow, (4) thrombus formation on the suture line, and (5) intimal and medial hyperplasia at the anastomotic site.

Surgical correction of coarctation restenosis can now be performed with an acceptable risk. However, with the availability of large, nondistensible, polyethylene, dilatation balloon catheters (Medi-Tech, Watertown, MA), percutaneous transluminal angioplasty (PTA) was considered as an alternative mode of therapy for the management of recurrent coarctation of the aorta. This article describes our experience and technique in successfully treating seven patients with coarctation restenosis.

Methods

Clinical material. Seven patients underwent percutaneous transluminal balloon angioplasty for treatment of recurrent stenosis in the aorta at the site of previous coarctation repair. The presenting data for these seven patients before the balloon angioplasty are summarized in Table 1. The seven patients were divided into four categories based on the type of primary repair performed.

Coarctation restenosis (end-to-end anastomosis, three patients)

Patient 1. An 11-year-old girl had been in congestive heart failure in infancy. When she was 6 months old, a cardiac catheterization revealed severe coarctation of the aorta with a patent ductus arteriosus (PDA). Intra-aortic pressures indicated a 50 mm Hg pressure gradient across the coarctation. At 9 months of age she had surgical resection of the coarctation with an end-to-end anastomosis. Subsequently she was asymptomatic, and when followed up in the outpatient clinic she had normal arm blood pressures and a 20 mm Hg lower pressure in her legs. At 11 years old, she had evidence of upper-extremity hypertension with diminished femoral pulses.

Patient 2. An 8-year-old boy had been in congestive heart failure in the neonatal period. When he was 11 days old, cardiac catheterization revealed severe coarctation of the aorta and PDA. When he was 12 days old the coarctation was resected and an end-to-end anastomosis was performed. After the surgical procedure he had persistent upper-extremity hypertension with decreased femoral pulses. When he was 8 years old, cardiac catheterization revealed a discrete recurrent coarctation of the aorta.

Patient 3. An 8-year-old boy had congestive heart failure and differential cyanosis at 4 days of age. Cardiac catheterization revealed severe coarctation of the aorta and PDA. The coarctation was resected, the aorta was repaired with an end-to-end anastomosis, and the PDA was ligated. Subsequently the congestive heart failure resolved, but there was persistent right arm hypertension with diminished femoral artery pulses.

Coarctation restenosis (end-to-end anastomosis, status after bypass graft, two patients)

Patient 4. A 17-year-old boy had congestive heart failure at 1 month of age. Cardiac catheterization revealed coarctation of the aorta and PDA. At 2 months of age he underwent surgical
TABLE 1
Clinical data of the seven patients undergoing PTA

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Interval since primary repair (yr)</th>
<th>Interval since graft bypass (yr)</th>
<th>Coarctation restenosis: end-to-end anastomosis</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Coarctation</td>
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<td></td>
<td></td>
<td>restenosis:</td>
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<td>end-to-end</td>
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<td>anastomosis</td>
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<td>Coarctation</td>
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<td>restenosis:</td>
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<td>anastomosis</td>
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<td></td>
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<td>S/P graft bypass</td>
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<td></td>
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<td></td>
<td>Coarctation restenosis: patch angioplasty</td>
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<td></td>
<td></td>
<td></td>
<td>Interrupted aortic arch suture line stenosis</td>
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<td></td>
<td></td>
<td></td>
<td>S/P = status after.</td>
</tr>
</tbody>
</table>

resection of the coarctation, with end-to-end anastomosis and ligation of the PDA. When he was 13½ years old, cardiac catheterization indicated recurrent stenosis with a 33 mm Hg peak systolic pressure gradient across the coarctation. Surgery was performed at age 14, in which a 10 mm Gore-tex bypass graft (polytetrafluoroethylene; W. L. Gore Associates, Inc., Elkton, MD) was inserted from the left subclavian artery to the descending aorta. The postoperative course was complicated by left phrenic nerve palsy, left recurrent laryngeal palsy, and severe asthma that required prolonged respirator support. Subsequently he developed severe hypertension with a right arm blood pressure of 170/120 and decreased femoral pulses. The hypertension was unresponsive to medical management. Because of the difficult recovery after the previous surgical procedure, he was referred for balloon angioplasty of his primary anastomotic site.

Patient 5. A 15½-year-old boy had had a surgical repair of coarctation of the aorta with end-to-end anastomosis at 9 years of age. At 15 he had upper-extremity hypertension that resulted in a blood pressure of 170/90 and leg pressures of 100/70. Cardiac catheterization revealed recurrent coarctation of the aorta. At surgery the distal aortic pressure fell to 0 mm Hg when the aorta was cross-clamped. Therefore, a 14 mm Gore-tex graft was placed from the left subclavian artery to the descending aorta. After insertion of the graft, a pressure of 140 mm Hg was measured in the right radial artery. The pressure in the transverse aortic arch was 120 mm Hg, and in the aorta distal to the bypass, pressure was 110 mm Hg. Several months after the surgery, the blood pressure in the right arm was 170/85 despite treatment with antihypertensive medication, and femoral pulses were diminished. Six months after the bypass surgery, he was referred for balloon angioplasty of the primary anastomotic stenosis.

Coarctation restenosis (patch angioplasty, one patient)

Patient 6. A 10-month-old boy underwent a patch Gore-tex angioplasty repair of coarctation of the aorta and ligation of a PDA at 5 days of age. At discharge 1 week after the surgery, he had strong femoral pulses and equal upper- and lower-extremity blood pressures.

At 7 months of age it was noted that he had a right arm pressure of 150 mm Hg and diminished femoral pulses. He remained asymptomatic with normal growth and development. At 10 months he returned from his home for reevaluation and therapy.

Interval of aortic arch (graft suture line stenosis, one patient)

Patient 7. A 6-year-old boy had presented at 2 days of age with congestive heart failure and intermittent femoral pulses. Cardiac catheterization demonstrated an interrupted aortic arch between the left carotid and left subclavian arteries, a ventricular septal defect (VSD), and a PDA. He had surgical interposition of a 5 mm Gore-tex graft from the left carotid artery to the descending aorta. When he was 2 months of age a PDA ligation and pulmonary artery banding were performed because of persistent congestive failure. When he was 2 years old the VSD was closed and the pulmonary artery band was removed. When he was 4 years old, cardiac catheterization revealed a small residual VSD and a 60 mm Hg pressure gradient across the Gore-tex graft. The 5 mm Gore-tex graft was replaced with a 10 mm Gore-tex graft. Follow-up cardiac catheterization 1 year later demonstrated only a 19 mm Hg pressure gradient across the graft. The angiogram showed a narrowing at the distal end of the graft. Proximal blood pressures were unobtainable because

TABLE 2
Hemodynamic data: initial change in SPG in response to PTA

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Before angioplasty</th>
<th>After angioplasty</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>SPG (mm Hg)</td>
<td>CO (l/min)</td>
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<tr>
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<tr>
<td>Coarctation restenosis: end-to-end anastomosis</td>
<td>1</td>
<td>60</td>
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<tr>
<td></td>
<td>2</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>64</td>
</tr>
<tr>
<td>Coarctation restenosis: end-to-end anastomosis graft bypass</td>
<td>4</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>54</td>
</tr>
<tr>
<td>Coarctation restenosis: patch angioplasty</td>
<td>6</td>
<td>68</td>
</tr>
<tr>
<td>Interrupted aortic arch suture line stenosis</td>
<td>7</td>
<td>66</td>
</tr>
</tbody>
</table>

SPG = Systolic pressure gradient across the coarctation determined from intra-aortic pressure curves; CO = cardiac output determined by thermodilution or green dye; NA = not available.
of a previous right axillary cut-down. By 6 years of age the patient complained of cramping in his feet, and he was re-evaluated by cardiac catheterization.

**Balloon angioplasty technique.** Our protocol for PTA for the treatment of coarctation restenosis was approved by the Joint Committee for Clinical Investigation. Informed consent was obtained for each patient.

In each patient the balloon angioplasty procedure was performed percutaneously. Arterial cannulas were placed in both femoral arteries, and a venous sheath was placed in one of the femoral veins. After the coarctation restenosis site was carefully crossed with a guide wire, an angiographic catheter was advanced proximal to the coarctation, and simultaneous proximal and distal aortic pressures were recorded. A venous catheter was advanced into the pulmonary artery, and cardiac output was measured by either the thermodilution or indocyanine-green method. Thoracic aortography was performed to define the dimensions of the aorta at the stenotic area.

Two observers independently measured the diameter of the aorta from the aortogram at the narrowest segment of narrowing and proximal and distal to the coarctation. A ratio of the true outer diameter of the catheter to the outer diameter measured from the angiogram was used to correct for magnification. A dilatation catheter with a polyethylene balloon diameter of 1 to 2 mm less than that of the aortic lumen above and below the narrowing was selected. An 0.035 inch exchange guide wire was advanced into the ascending aorta, and the diagnostic catheter was exchanged for the angioplasty catheter. The dilatation balloon was carefully advanced percutaneously into the femoral artery over the guide wire. As the balloon entered the artery, the catheter was gently rotated counterclockwise with the deflated balloon wrapped in a clockwise fashion. The catheter was then advanced over the guide wire and positioned with the stenosis at the center of the balloon. A 0.021 inch J guide wire was placed through a Y adapter and left in the ascending aorta. This permitted rapid and safe withdrawal and advancement of the catheter from proximal to distal to the coarctation site for pressure measurements. A single heparin dose (100 U/kg) was administered.

The balloon was inflated with dilute contrast material (30% sodium methylglucamine diatrizoate) through a pressure gauge so that the inflation pressure could be manually controlled. During the procedure proximal aortic pressures were measured through the dilatation catheter and distal pressures were measured through the femoral artery cannula. The balloon was inflated and pressure was maintained for 10 sec; the balloon was then deflated. The balloon was inflated two or three additional times in each patient. With the balloon fully deflated, the dilatation catheter was removed from the artery with the same counterclockwise rotation used for introduction. An angiographic catheter was introduced into the opposite femoral artery and proximal and distal pressures were recorded. A postdilatation aortogram was performed, and repeat cardiac outputs were measured. The dimension of the aorta was again measured directly from the angiogram by the magnification factor determined from the catheter measurements.

The heparinization was not reversed at the end of the procedure, and long-term anticoagulation was not instituted.

**Results**

The hemodynamic measurements before and after the balloon angioplasty are shown in table 2. Before the balloon angioplasty, the systolic pressure gradients across the coarctation ranged from 32 to 68 mm Hg, with a mean of $58 \pm 12$ mm Hg (SD). Immediately

![FIGURE 1](http://circ.ahajournals.org/)

**FIGURE 1.** The balloon catheter is in position across the coarctation (patient 6). A. At the beginning of the inflation the balloon is indented by the coarctation. B. With full inflation of the balloon, the "waisting" of the balloon is obliterated.

![FIGURE 2](http://circ.ahajournals.org/)

**FIGURE 2.** Simultaneous proximal and distal intra-aortic pressure tracing recorded before balloon angioplasty, during the balloon inflation, and immediately after the procedure (patient 1). The recording paper speed was slowed during the balloon inflation. Initial peak systolic gradient was 60 mm Hg and fell to 8 mm Hg after angioplasty.
after the angioplasty, systolic pressure gradients were decreased and ranged from 0 to 25 mm Hg, with a mean of $13 \pm 9$ mm Hg ($p < .001$). The cardiac output measurements before and after the angioplasty indicate that the decrease in systolic pressure gradient after the PTA was not caused by a drop in cardiac output.

Figure 1 shows the balloon catheter during dilatation. An example of the pressure tracing before, dur-
Coarctation restenosis, patch angioplasty (patient 6). The aortograms before (A) and after (B) the angioplasty demonstrate marked improvement in the constriction at the coarctation restenosis site.

Complications. One patient (No. 5) died 6 hr after a seemingly uncomplicated angioplasty. When he was being observed on the ward after the procedure, all vital signs were stable. He was unable to void in the supine position and so was being assisted to stand when he suddenly collapsed and had no obtainable pulse. The electrocardiogram revealed ventricular fibrillation. Resuscitation efforts were unsuccessful. An autopsy was performed and there were no anatomic features to explain the sudden death. No bleeding was found in the periaortic area. There was no evidence of cerebral aneurysm or bleeding. The area of stenosis showed small intimal tears without disruption of the
media. Microscopic sections revealed intimal proliferation at the site of the previous anastomosis.

**Follow-up.** Six patients have been followed clinically for 1 to 14 months after angioplasty. Systolic pressure gradients have been determined by comparing arm and leg blood pressures by use of appropriately sized cuffs. Initial and follow-up measurements indicated that the decrease in systolic pressure gradients after PTA is sustained for at least the interval of follow-up (table 4).

**Discussion**

Transluminal angioplasty techniques were first developed by Dotter and Judkins in 1964. In 1974 Grünzig introduced an improved method for angioplasty, which used a dilatation balloon catheter. Subsequently, balloon angioplasty has been used to treat vascular narrowing due to atherosclerotic plaques, fibromuscular hyperplasia, and suture line stenoses in coronary, femoral, iliac, and renal arteries, which may result from intimal and medial hyperplasia. Long-term follow-up studies show a high rate of success of angioplasty treatment of these lesions.

Histologic examination of the resected tissue removed at surgery for coarctation restenosis revealed intimal and medial proliferation and suture-line granulomas. Similarity of this tissue to that of stenotic bypass graft–artery stenosis suggests that dilatation of coarctation restenosis should also be associated with lasting results.

In 1979 Sos et al. reported successful percutaneous transluminal dilatation of a coarctation postmortem in an infant with hypoplastic left-heart syndrome. Transluminal balloon angioplasty was reported by Singer et al. to be successful in relieving the obstruction in a 7-week-old infant with early recurrent (or perhaps residual) stenosis after surgical correction by patch angioplasty in the newborn period. However, Lock et al. reported an inability to dilate a surgically resected segment of coarctation restenosis even at high pressure (8 atmospheres).

None of the patients in the series developed evidence of the postcoarctectomy syndromes of reactive hypertension or abdominal pain. Although the mechanisms of the postcoarctectomy syndromes are not completely resolved, if the risks relate to time of occlusion of the aorta at the stenotic segment, the balloon angioplasty technique should greatly decrease the incidence of postcoarctectomy syndromes, since the disturbance of flow to the descending aorta lasts only 10 sec in the PTA technique compared with about 30 min of continuous cross-clamp time in the surgical procedure. However, any responses to the increased pulse pressure in the descending aorta after relief of the obstruction would be the same after the balloon angioplasty or surgical repair.
The periaortic scarring, which is a limiting factor for surgical access to the restenosed coarctation site, would seem to be a safety factor for the transluminal approach. If a disruption of the aortic wall should occur, bleeding into the chest should be limited by the surrounding dense fibrous tissue.

Selection of balloon type and size is critical in limiting the risk of the angioplasty procedure. The polyethylene dilatation balloons are not over distensible. At excessive pressures the balloons may rupture but will not expand beyond the fixed diameter. The polyvinyl chloride balloons tested previously may overdistend and, therefore, would increase the risk of disruption of the normal aortic wall above or below the stenotic segment. The balloon size selection at less than the dimension of the aorta proximal and distal to the stenotic segment should also limit the risk of a tear in the aortic wall.

The death of one patient several hours after the angioplasty repair appeared to have been initiated by a vagal event. We have not noted any hemodynamic instability in the other patients, but because of the one death temporally associated with the procedure, all patients are monitored in the intensive care unit for 24 hr after angioplasty.

Transluminal balloon angioplasty appears to be an attractive alternative to surgical repair of coarctation restenosis. Although there are risks associated with both surgery and balloon angioplasty, the transluminal approach offers some major advantages. The technique can be combined with the diagnostic cardiac catheterization, thus limiting the number of interventions. The procedure is performed with sedation and local anesthesia rather than with general anesthesia. The hospital stay and convalescence periods are considerably shorter than after the surgery, and the patient avoids having a thoracotomy.

While long-term effectiveness of the procedure is still uncertain, the encouraging short-term results suggest that this procedure may become the treatment of choice for patients with coarctation restenosis.

### References


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**TABLE 3**

Dimensions of the stenotic segment of aorta before and after PTA, balloon size, and maximum inflation pressure

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Initial diameter of lumen at stenosis (mm)</th>
<th>Balloon diameter (mm)</th>
<th>Inflation pressure (psi)</th>
<th>Postangioplasty diameter of lumen at stenosis (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coarctation restenosis: end-to-end anastomosis</td>
<td>1</td>
<td>7</td>
<td>12</td>
<td>80</td>
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<td>2</td>
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<tr>
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<td>3</td>
<td>3</td>
<td>10</td>
<td>80</td>
</tr>
<tr>
<td>Coarctation restenosis: end-to-end anastomosis</td>
<td>4</td>
<td>2</td>
<td>15</td>
<td>45</td>
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<td>S/P graft bypass</td>
<td>5</td>
<td>7</td>
<td>18</td>
<td>55</td>
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<td>Coarctation restenosis: patch angioplasty</td>
<td>6</td>
<td>2</td>
<td>8</td>
<td>84</td>
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<tr>
<td>Interrupted aortic arch</td>
<td>7</td>
<td>5</td>
<td>10</td>
<td>Not recorded</td>
</tr>
</tbody>
</table>

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**TABLE 4**

Follow-up of results after PTA for coarctation restenosis

<table>
<thead>
<tr>
<th>SPG (mm Hg)</th>
<th>Time after PTA (mo)</th>
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<tbody>
<tr>
<td>Patient no.</td>
<td>Initially</td>
</tr>
<tr>
<td>1</td>
<td>60</td>
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<tr>
<td>2</td>
<td>32</td>
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<td>3</td>
<td>64</td>
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<td>6</td>
<td>68</td>
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<td>7</td>
<td>66</td>
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</tbody>
</table>

SPG = systolic pressure gradient.

*Arm blood pressures in patient 7 were unobtainable because of multiple cut-downs in early infancy.
15. Mitchell SE, White RI Jr, Kan JS, Tolkoj J: Improved balloons for large vessel and valvular angioplasty. (Submitted for publication)
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