Balloon dilatation angioplasty: nonsurgical management of coarctation of the aorta

Rubin S. Cooper, M.D., Samuel B. Ritter, M.D., and Richard J. Golinko, M.D.

ABSTRACT Balloon dilatation angioplasty was successfully performed in five patients (ages 18 months to 17 years) with discrete aortic coarctation. The catheter size was No. 8F or 9F. Selection of balloon diameter was based on angiographic measurements of the aorta determined proximal and distal to the coarctation site. A 10 sec inflation-deflation cycle at 6 to 8 atmospheres (90 to 120 psi) was performed. The systolic pressure gradients across the coarctation before balloon dilatation angioplasty ranged from 35 to 70 mm Hg. Systolic pressure gradients after balloon dilatation angioplasty ranged from 0 to 10 mm Hg. All patients had normalized blood pressure immediately. Abnormal pulsed Doppler echocardiograms were observed in all patients before balloon dilatation angioplasty; four patients had normal echocardiograms after balloon dilatation angioplasty. No serious intraprocedural complications occurred. One patient required femoral artery thrombectomy 36 hr after balloon dilatation angioplasty. One to 6 months after balloon dilatation angioplasty no patients have evidence of restenosis of coarctation. Early results suggest that balloon dilatation angioplasty may offer a safe and effective nonsurgical alternative for the treatment of discrete coarctation in older infants and children. Long-term follow-up for the incidence of restenosis and formation of aneurysms will ultimately determine the efficacy and safety of this procedure.


THE INTRODUCTION and availability of balloon dilatation catheters has led to the use of percutaneous transluminal angioplasty in infants and children with congenital heart disease. The conditions that have been treated with this technique include pulmonary valvular stenosis,1–4 aortic valvular stenosis,5–6 peripheral pulmonary artery7 and pulmonary vein stenosis,8,9 superior or inferior vena caval obstruction,10,11 coarctation of the aorta,12–17 and restenosis of coarctation.18 This report describes the results of balloon dilatation angioplasty in five patients with discrete coarctation of the aorta. In addition, pulsed Doppler echocardiography was used to assess this condition before and after balloon dilatation.

Methods

Five patients (ages 18 months to 17 years) underwent percutaneous balloon dilatation angioplasty for discrete coarctation of the aorta. All patients had clinical evidence of coarctation manifested by diminished femoral pulses, systolic hypertension of the upper extremities, and a short-grade 2/6 systolic ejection murmur at the left base and back. A chest x-ray, electrocardiogram, and a two-dimensional-pulsed Doppler echocardiogram were obtained for each patient. After informed written consent was obtained from the patient or guardian, the patients were enrolled in a research protocol approved by the hospital’s Human Investigation Committee. A thoracic surgeon was available during the angioplasty in the event of any serious complications. All patients were premedicated with a lytic solution of meperidine (2 mg/kg), chlorpromazine (1 mg/kg), and promethazine (1 mg/kg) given intramuscularly 30 min before the procedure. The right groin was anesthetized with 1% or 2% lidocaine infiltration and subsequently a percutaneous right heart catheterization was performed from the right common femoral vein. Cardiac output was determined by thermodilution. A percutaneous left heart catheterization from the right common femoral artery was then performed. Pull-back pressures were recorded across the aortic valve and the site of coarctation. A cineangiogram in the 35 degree left anterior oblique view was taken in the aortic arch just proximal to the coarctation. Measurements were made of the aortic diameter 1 cm proximal and distal to the coarctation site. A magnification factor was determined by comparing the known catheter diameter to that measured on the fluoroscopic screen. The actual dimension of the aortic diameter was equal to: measured aortic diameter × (actual catheter diameter/measured catheter diameter).

Based on the above equation, a balloon 30 or 40 mm long was selected with a maximum inflation diameter of 1 mm less than the smaller measured aortic diameter. At this time a No. 8F or 9F dilatation catheter was prepared (Meditech, Watertown, MA). To avoid air embolization in the event of balloon rupture, all air was evacuated from the balloon by inflating and deflating several times with a 50/50 mixture of saline and contrast solution. A 200 cm (diameter, 0.035 in) exchange tight J guidewire

From the Department of Pediatrics and Pediatric Cardiology, The Brookdale Hospital Medical Center, and the Division of Pediatric Cardiology, State University of New York, Brooklyn, NY.

Supported in part by research funds from the Michael Hazan Children’s Heart Fund and the Louis Caldar Foundation.

Address for correspondence: Richard J. Golinko, M.D., Department of Pediatrics and Pediatric Cardiology, The Brookdale Hospital Medical Center, Linden Blvd. at Brookdale Plaza, Brooklyn, NY 11212.

Received June 8, 1984; revision accepted Aug. 2, 1984.
COOPER et al.

FIGURE 1. Left, Before balloon dilatation angioplasty. A partially inflated balloon shows indentation caused by the coarctation ridge (waisting). Right, After balloon dilatation angioplasty. The coarctation ridge is eliminated (no waisting).

(Cook, Inc., Bloomington, IN) was positioned in the left ventricle and a balloon dilatation catheter was exchanged for the angiographic catheter. The dilatation balloon was partially inflated to identify the exact location of the coarctation ridge. The midportion of the balloon was positioned slightly proximal to this point. The balloon was progressively inflated until the waist (indentation of the balloon caused by the coarctation ridge) was obliterated. This usually required an inflation pressure of 6 to 8 atmospheres (90 to 120 psi). The inflation-deflation cycle was approximately 10 sec, minimizing the period of complete aortic obstruction. With the J guidewire still in place, an angiographic catheter was exchanged for the balloon dilatation catheter. A second cineangiogram was taken in the aortic arch. Determination of cardiac output by thermodilution was repeated and followed by a single pull-back pressure recording across the site of coarctation. The arterial and venous catheters were removed and bleeding was controlled by pressure alone. All patients were studied within 24 hr by two-dimensional pulsed Doppler echocardiography.

Results

The hemodynamic measurements before and after balloon dilatation angioplasty are summarized in table 1. Before balloon angioplasty the peak systolic pressure gradient across the coarctation ranged from 35 to 70 mm Hg (mean 52). After balloon angioplasty the peak systolic pressure gradient decreased to 0 to 10 mm Hg in all five patients. During the initial inflation, waisting of the balloon was seen in all patients. After maximal inflation the waist was eliminated (figure 1). Similar changes were seen in the aortograms obtained before and after dilatation (figure 2). Continuous pressure tracings across the site of coarctation before and after the procedure were similar in all five patients.

FIGURE 2. Left, Before balloon dilatation angioplasty. Aortogram showing coarctation ridge. Right, After balloon dilatation angioplasty. Aortogram showing elimination of the coarctation ridge.
TABLE 1
Hemodynamic data: change in peak systolic gradient in response to balloon dilatation angioplasty

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Before angioplasty</th>
<th>After angioplasty</th>
</tr>
</thead>
<tbody>
<tr>
<td>N.Z.</td>
<td>17 yr</td>
<td>35 2.8</td>
<td>0 2.1</td>
</tr>
<tr>
<td>A.P.</td>
<td>13 yr</td>
<td>45 5.5</td>
<td>0 5.5</td>
</tr>
<tr>
<td>S.T.</td>
<td>21 mo</td>
<td>70 3.0</td>
<td>0 3.0</td>
</tr>
<tr>
<td>R.F.</td>
<td>3.5 yr</td>
<td>50 2.9</td>
<td>0 2.0</td>
</tr>
<tr>
<td>C.B.</td>
<td>18 mo</td>
<td>65 4.6</td>
<td>10 5.6</td>
</tr>
</tbody>
</table>

Cl = cardiac index (by thermodilution); PSG = peak systolic gradient.

(figure 3). The pulsed Doppler flow studies after balloon dilatation demonstrated a return to a normal spectral wave form in four of the five patients (figure 4). All patients tolerated the angioplasty procedure well and there were no intraprocedural complications. Reactive hypertension and abdominal pain (postcoarctectomy syndrome) did not occur in any patient. One patient required thrombectomy 36 hr after the procedure and was discharged after 5 days. All other patients were discharged from the hospital within 48 hr of the procedure. The patient who required thrombectomy was discharged after 5 days. All patients are doing well 1 to 6 months after angioplasty and no patient has evidence of restenosis of the coarctation.

Discussion

Transluminal balloon dilatation of stenotic arteries in peripheral, 19, 20 renal, 21 and coronary 22 circulations is a well-established nonsurgical technique. Application of the balloon dilatation principle to infants and older children with congenital heart disease has only recently been described. 1-18 In 1979 a successful postmortem percutaneous transluminal dilatation of an aortic coarctation in a newborn was reported. 23 Subsequently, excised coarctation segments obtained at surgery from seven infants and children were dilated in vitro.24 Histologic studies revealed intimal and medial tears, suggesting a possible mechanism for the relief of the aortic obstruction. Early attempts to dilate aortic coarctation in very young infants were palliative at best and invariably required coarctectomy.15, 16, 25 To date there has been no report of early or late aortic dissection, although a small aneurysm at the site of balloon dilatation occurred acutely in one patient. There have been three deaths after balloon dilatation angioplasty. A 1-week-old infant died in the operating room during a pulmonary arterial banding and coarctectomy performed immediately after an unsuccessful balloon dilatation. This infant had complex intracardiac disease in addition to a juxtaductal coarctation. 17 An intraprocedural death occurred in a 1-week-old infant with ductal patency. The cause of death was aortic perforation in the juxtaductal area after attempts to advance a catheter across the previously dilated site without the aid of a guidewire.25 An additional death occurred in a 3-week-old infant when difficulties were encountered during removal of the balloon catheter. At autopsy an aortic tear was found at its junction with the right common iliac artery.

Selection of balloon size as well as positioning of the balloon are essential steps in improving the effectiveness and minimizing the risk of coarctation angioplasty. Polyethylene dilatation balloons are not distensible beyond a fixed diameter. Although excessive pressure may rupture the balloon, this results in a linear tear. Use of a balloon size 1 mm less than the smallest aortic diameter is important. After balloon dilatation, no catheter should be advanced across the coarctation site without the aid of an exchange guidewire. During the catheterization procedure sig-

FIGURE 3. Top, Before balloon dilatation angioplasty (BDA). Continuous pressure tracing across the coarctation site (arrow). Peak systolic gradient = 70 mm Hg. (Scale: each horizontal line 20 mm Hg.) Bottom, After balloon dilatation angioplasty. Continuous pressure tracing across coarctation site (arrow). Peak systolic gradient = 0 mm Hg.
significant reduction in the systolic pressure gradient across the coarctation site combined with the disappearance of the aortic ridge angiographically would seem to be the most reliable criteria for evaluating the immediate success of the balloon dilatation angioplasty. Although serial blood pressure measurements are useful for long-term follow-up, pulsed Doppler echocardiography offers an even more sensitive tool for assessment of normalized aortic blood flow. Decreased pulsatile flow in the descending aorta is seen in patients with coarctation and has been previously described. All of our patients had an abnormal aortic flow pattern before balloon dilatation. After the procedure four patients demonstrated a return to a completely normal Doppler flow pattern. One patient had incomplete normalization of descending aortic blood flow despite the elimination of the pressure gradient across the coarctation and the disappearance of the

coarctation ridge. A possible explanation for this was the continued presence of mild tubular hypoplasia of the aortic isthmus and marked poststenotic dilatation of the descending aorta. Finally, because of the femoral arterial complications we encountered, as well as the potential for emboli to migrate from the balloon, it would seem reasonable to administer heparin to patients during the angioplasty procedure.

In summary, our initial experience with balloon dilatation angioplasty in five patients is encouraging. At the present time, and particularly in older infants and children, this procedure seems to be a safe and effective nonsurgical alternative for relieving the obstruction associated with discrete coarctation of the aorta. However, the apparent hazards and technical difficulties that have been encountered in younger infants suggest that extreme caution should be exercised in treating this age group. Greater experience and long-term follow-up for late complications, including restenosis of coarctations and formation of aneurysms are still needed to determine the ultimate role of balloon dilatation angioplasty in these patients.

We are indebted to Dr. Zuhdi Lababidi for his guidance in developing this protocol and for his initial work on this project, to the staff of the Cardiac Catheterization Laboratory for their technical assistance and the excellent care our patients received, and to Lenore Gottlieb and Harriet Lustbader for assistance in preparing this manuscript.

References

Balloon dilatation angioplasty: nonsurgical management of coarctation of the aorta.
R S Cooper, S B Ritter and R J Golinko

Circulation. 1984;70:903-907
doi: 10.1161/01.CIR.70.5.903
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1984 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/70/5/903

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/