Progress in
Cardiovascular Diseases
VOL XXXII, NO 1 JULY/AUGUST

Transcatheter Treatment of Congenital Heart Disease

Robert H. Beekman and Albert P. Rocchini

DURING THE 1980s the pediatric cardiac catheterization laboratory has undergone a transition from a diagnostic laboratory to a therapeutic suite where congenital cardiac abnormalities are not only evaluated but often can be treated. Therapeutic transcatheter procedures have been developed that can provide palliative or definitive therapy to children with a variety of congenital cardiovascular disorders. Nonsurgical transcatheter treatment is now available for obstructive lesions including pulmonary valve stenosis, pulmonary artery stenosis, aortic valve stenosis, coarctation of the aorta, and mitral stenosis. Some left-to-right shunt lesions can also be treated in the catheterization laboratory. Embolization techniques are available to deal with aortopulmonary collateral vessels or surgical shunts. Further, new approaches are being developed which promise effective transcatheter treatment of a secundum atrial septal defect or patent ductus arteriosus. Clearly, a pediatric cardiac catheterization is no longer complete once a hemodynamic and angiographic evaluation is performed. The pediatric cardiologist must go further, and ask whether the lesion at hand can be effectively treated in the catheterization laboratory without resorting to a more invasive surgical approach.

At C.S. Mott Children's Hospital, transcatheter treatment of congenital heart disease has been performed since 1981, and has steadily increased in importance. Figure 1 demonstrates the increasing number of therapeutic transcatheter procedures performed at this institution during the period from 1981 through 1987. In 1987 therapeutic catheterizations accounted for approximately 15% of all our pediatric cardiac catheterizations. These procedures provided palliative or definitive treatment for children with a variety of structural cardiac abnormalities. The figures underestimate the current significance of catheter therapy as they do not include other interventional procedures such as balloon atrial septostomy, blade septostomy, foreign body retrieval, endomyocardial biopsy, or transcatheter ablation of an accessory pathway. Further, these figures do not include transcatheter patent ductus arteriosus (PDA) occlusions, which are planned, but have not yet been performed at this institution. The data presented in Fig 1 clearly demonstrate the current importance of transcatheter treatment of congenital heart disease. Further, from the increasing trend one can infer that therapeutic catheterization will account for an even larger percentage of total pediatric cardiac catheterizations in the near future.

In this article we discuss therapeutic transcatheter approaches to a variety of congenital cardiovascular abnormalities. Some procedures have become the treatment of choice (eg, valvuloplasty for pulmonary stenosis) while others remain investigational (eg, angioplasty for native coarctation). Formal cost-benefit analyses of these procedures including a comparison to accepted surgical alternatives have not yet been performed, and long-term follow-up data are generally lacking. Nevertheless, we will discuss the current role played at our institution by therapeu-

From the Division of Pediatric Cardiology, C.S. Mott Children's Hospital, University of Michigan, Ann Arbor.

Supported by the NIH General Clinical Research Center, Grant 5MO1-RR00042.

Address reprint requests to Robert H. Beekman, MD, Box 0204, F1116, C.S. Mott Children's Hospital, Ann Arbor, MI 48109.

0033-0620/89/3201-0001$5.00/0

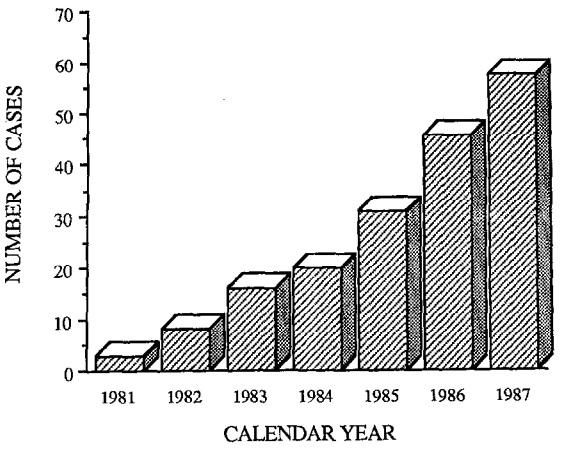

Fig 1. Number of therapeutic transcatheter procedures performed in children at C.S. Mott Children's Hospital during the calendar years 1981 to 1987. In 1987 these cases accounted for 15% of all cardiac catheterizations performed at this institution.

tic catheterization as an alternative to surgery for the child with congenital heart disease.

PULMONARY VALVE STENOSIS

Until recently, surgical valvotomy had been the only treatment available for valvar pulmonary stenosis. The first nonsurgical approach to this disease was described in 1979 by Semb et al.[1] They relieved pulmonary valve stenosis in an infant by inflating a balloon catheter in the pulmonary artery and withdrawing it back through the malformed valve. Subsequently in 1982, Kan et al[2] described the technique of percutaneous transluminal balloon valvuloplasty. They reported an 8-year-old child with valvar pulmonary stenosis in whom valvuloplasty acutely reduced the peak gradient from 48 mmHg to 14 mmHg. The procedure was then performed on four other children with similar effectiveness. Since that, others have had marked success using balloon valvuloplasty to treat isolated pulmonary valve stenosis.[3-11] Lababidi and Wu,[4] for example, described the effects of balloon valvuloplasty in 18 patients with valvar pulmonary stenosis. In these patients, valvuloplasty reduced the peak stenosis gradient from 81 mmHg to 23 mmHg and the right ventricular systolic pressure from 106 mmHg to 50 mmHg, without producing significant pulmonary regurgitation.

There are two groups of children in whom balloon pulmonary valvuloplasty has not been uniformly successful: (1) neonates with critical pulmonary stenosis and (2) children with dysplastic pulmonary valves. The major problems encountered with critical pulmonary stenosis in the neonate relate to the difficult task of positioning a balloon catheter across the critically narrowed valve, and to the frequently associated annular hypoplasia and tricuspid valve stenosis. With careful attention to technique and the use of smaller catheters and guidewires (see below) we feel that the success rate will improve in this patient subgroup. Recently, Zeevi et al.[12] reported successful dilation of 4/5 neonates with critical pulmonary valve stenosis. In children with a dysplastic pulmonary valve, hypoplasia of the valve annulus is almost always present,[13,14] thus diminishing the likelihood that balloon valvuloplasty will provide satisfactory relief of stenosis. DiSessa et al[15] have suggested that the presence of a dysplastic pulmonary valve is a contraindication to pulmonary valvuloplasty. The authors and others[16] have found, however, that valvuloplasty may be effective in some children with this condition.

The mechanism of balloon pulmonary valvuloplasty is not known. Most likely, valvuloplasty reduces right ventricular outflow obstruction by separating fused valve commissures, or tearing or stretching valve leaflets. Disruption of the valve annulus seems unlikely when balloons smaller than 140% of the annulus diameter are used.[17,18] In one child with tetralogy of Fallot, Lababidi described intraoperative observations of pulmonary valve morphology after balloon valvuloplasty.[4] The child's bicuspid pulmonary valve was found to have a tear in the anterior valve raphe, thus documenting that valvuloplasty can in fact tear pulmonary valve leaflet tissue. More recently Walls et al,[19] from the same institution, reported additional observations of the morphologic effects of balloon pulmonary valvuloplasty. At the time of elective operative repair of other congenital cardiac anomalies, the pulmonary valves of five children who had undergone valvuloplasty were inspected. Balloon valvuloplasty was found to have caused commissural splitting in one child, a cusp tear in one, and the combination of commissural splitting and cusp avulsion in two.

Technique

Balloon pulmonary valvuloplasty, as with the other interventional procedures described in this review, is performed in the cardiac catheterization lab with the patient sedated but without the use of general anesthesia. A complete cardiac catheterization and a right ventricular angiogram are obtained for confirmation of the diagnosis and measurement of the valve annulus. The valve annulus diameter is measured from the angiogram with the aid of a calibrated marker catheter to adjust for image magnification. We have used criteria for performing pulmonary valvuloplasty similar to those used in the past to indicate the need for a surgical valvotomy. In general, we have performed balloon valvuloplasty in children with valvar pulmonary stenosis and (1) a resting peak-to-peak gradient of 40 mmHg or more or (2) in an infant with right-to-left atrial shunt regardless of gradient.

The technique of percutaneous balloon pulmonary valvuloplasty is relatively straightforward. A 7 French end-hole catheter is usually advanced from the right femoral vein into the left pulmonary artery. A 260 cm exchange guidewire is then passed through the catheter into the left lower pulmonary artery. The catheter and venous sheath are removed, leaving the guidewire in place. The valvuloplasty catheter consists of an 8 or 9 French shaft upon which is mounted a polyethylene balloon (to date, we have used angioplasty catheters manufactured by the Mansfield Corporation). The balloon diameter is usually selected to be 1.2 to 1.3 times the diameter of the pulmonary valve annulus.[20] If the annulus exceeds 18 mm in diameter, or if a single balloon is deemed too large for the child's femoral vein, then the double balloon technique may be used (Fig 2). The sum of the two balloon diameters should be about 1.6 to 1.7 times the diameter of the pulmonary valve annulus.[21] The guidewire remains in the left pulmonary artery during the valvuloplasty in order to stabilize the dilation catheter. After the balloon is positioned across the valve, it is inflated by hand to 3 to 6 atm of pressure with dilute contrast until the waist produced on the balloon by the valve disappears. Generally, several inflations are performed with minor adjustments in catheter position to assure optimal valve dilation. The deflation-inflation

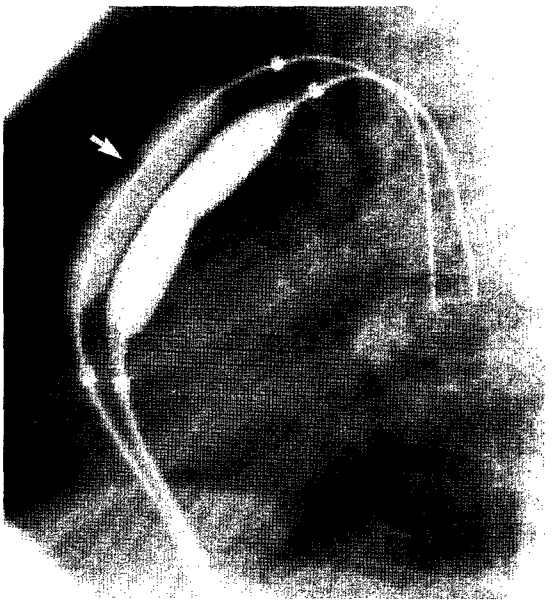

Fig 2. Double balloon pulmonary valvuloplasty in a 21-month-old child with pulmonary valve stenosis. The arrow points to the waist created on the balloons by the valve annulus.

cycle should be as short as possible, lasting no longer than 20 seconds, with less than ten seconds of complete occlusion of the right ventricular outflow tract. Right ventricular and aortic pressure and heart rate should be monitored while the balloon is inflated. The balloon catheter is then exchanged for another hemodynamic catheter and postvalvuloplasty hemodynamics are recorded.

When pulmonary valvuloplasty is performed in the neonate with critical pulmonary stenosis the valvuloplasty technique is modified in several ways. First, in order to maintain ductal patency and assure adequate oxygenation, the neonate should be maintained on prostaglandin E_1 throughout the procedure. Second, since it is impossible to safely use a 7 French end-hole catheter to cross the pulmonary valve of a neonate with critical stenosis, we have found it useful to cross the valve with either a 4-5 French end-hole catheter. A 0.014 to 0.017 inch teflon-coated guidewire is then advanced through the ductus into the descending aorta. The valve is progressively dilated, starting with a 4 to 5 mm low profile coronary angioplasty catheter, and then proceeding to a balloon approximately 1.2 times the diameter of the pulmonary valve annu-

lus. It should be noted that valvuloplasty effectiveness cannot be assessed acutely from gradient and right ventricular pressure alone in a neonate whose ductus is open. In these infants, a successful dilation should decrease right ventricular pressure to near systemic levels and allow for safe discontinuation of the prostaglandin infusion. Once the ductus has closed, the degree of residual right ventricular outflow obstruction can be assessed clinically, by Doppler echocardiogram and/or by follow-up catheterization if indicated.

Results

Acute effects. The acute results of balloon pulmonary valvuloplasty at our institution have been excellent. Between July 1982 and February 1988, 48 children, ranging in age from one day to 23 years (mean 4.2 years), have undergone percutaneous balloon valvuloplasty for treatment of congenital pulmonary valve stenosis at the University of Michigan. In these children, the peak-to-peak right ventricular outflow gradient has been reduced by 53% from 76 mmHg to 36 mmHg ($P < .0001$). The right ventricular systolic pressure decreased from a mean of 95 mmHg to 56 mmHg ($P < .0001$). These acute results are similar to those published from other institutions that have generally found immediate postvalvuloplasty gradients below 40 mmHg.[2,4-11,18,20]

Our experience with neonates who have critical pulmonary stenosis and children with dysplastic pulmonary valves should be noted. We have attempted valvuloplasty in four severely ill, cyanotic newborns with critical pulmonary valve stenosis. In only two have we been able to successfully cross and dilate the valve. Using the technique modifications described above, however, we feel that the success rate in this age group will improve. In children with a dysplastic pulmonary valve our experience suggests that, if the valve annulus is near normal size (diameter >75% of normal for body surface area) valvuloplasty will usually be effective. We have performed valvuloplasty in eight children with a dysplastic pulmonary valve and successfully reduced the right ventricular outflow tract gradient in five. Six of the eight children had Noonan syndrome and the remaining two had pulmonary atresia with an intact ventricular septum and had undergone a pulmonary valvotomy and systemic-to-pulmonary artery shunt at one to three days of age. In the five successful cases, the right ventricular outflow tract gradient decreased from 84 mmHg to 59 mmHg immediately after valvuloplasty; over the ensuing year the average gradient has decreased further to 29 mmHg. In our series of children with pulmonary valve stenosis, those with a dysplastic pulmonary valve have had the greatest additional decrease in right ventricular outflow tract gradient 1 year after valvuloplasty. The reason for this phenomenon is unknown.

Complications. Neither we nor others have encountered serious short- or long-term complications after pulmonary valvuloplasty. Premature ventricular contractions are frequently seen in most patients during the valvuloplasty procedure; however, no long-term ventricular or atrial ectopy has been reported. In one of our 12 patients who underwent an elective follow-up catheterization, the right femoral vein was found to be occluded. Doppler evidence of pulmonary valve insufficiency is frequently present after balloon valvuloplasty; however, in no patient has pulmonary valve insufficiency been reported to cause clinical problems.

Follow-up. We[21,22] like others[23] have observed excellent long-term relief of pulmonary stenosis following balloon valvuloplasty. Figure 3 depicts the serial right ventricular outflow tract gradient measurements obtained 12 months or more after valvuloplasty in the first 28 children who underwent pulmonary valvuloplasty at our institution. In ten the right ventricular outflow gradients were documented at cardiac catheterization 1 year after valvuloplasty. In the remaining 18 children, and in all subjects who underwent 2-, 3-, 4-, and 5-year follow-up, the residual gradients were estimated by Doppler echocardiography. The residual outflow tract gradients underwent a significant decline ($P < .001$) during the first year following valvuloplasty. This decrease in outflow gradient probably reflects regression of hypertrophic infundibular muscle. Others have also documented the development of an infundibular gradient after balloon valvuloplasty.[6,7,16,23] In fact, severe infundibular obstruction after valvuloplasty has been reported, on one occasion, actually to increase the right ventricular outflow obstruction.[24] Data from our series and others suggest that after a decrease during

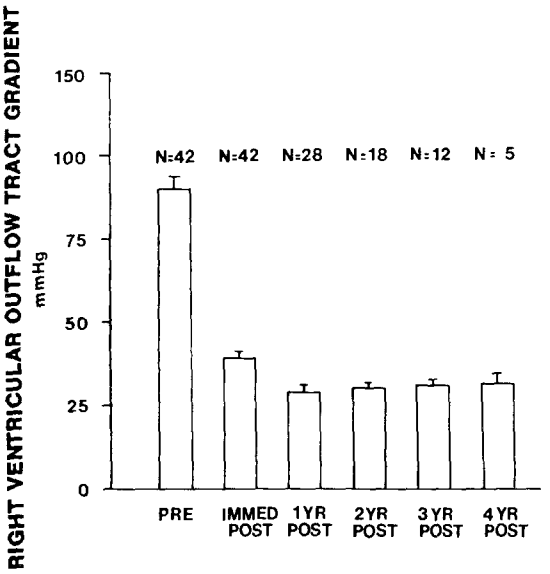

Fig 3. Serial measurements of right ventricular outflow gradient in the first 28 children followed for >12 months after balloon pulmonary valvuloplasty at C.S. Mott Children's Hospital. The gradients measured at 1 year were significantly less ($P < .001$) than the gradients immediately after valvuloplasty. (Reprinted with permission.[21])

the first year, the residual right ventricular outflow tract gradients remain stable for up to 5 years of follow-up.

We have had to repeat a pulmonary valvuloplasty in two children. In both of these cases, we initially used a valvuloplasty balloon whose diameter was 2 to 3 mm smaller than the pulmonary valve annulus. For the repeat valvuloplasty, we used a balloon whose diameter was 4 to 5 mm larger than the annulus. In both instances the second procedure was successful, and gradient reduction has been maintained for 36 and 48 months, respectively.

Conclusions

Our results and those of others suggest that balloon valvuloplasty is a safe, effective method of treating isolated congenital pulmonary valve stenosis in childhood. Since the results compare favorably with those of surgical valvotomy, we believe that balloon valvuloplasty is the treatment of choice for isolated valvar pulmonary stenosis. In the case of the neonate with critical pulmonary stenosis, more experience is needed before valvuloplasty can be recommended as the best initial treatment for these patients. We feel that there are two relative contraindications to the use of pulmonary valvuloplasty: The first is the presence of pulmonary valve annulus hypoplasia. If the annulus is <75% of the normal annulus diameter for the patient's body surface area, valvuloplasty does not appear to provide significant relief of right ventricular outflow obstruction. It is our impression that annulus size is more critical to predicting valvuloplasty success than is the morphology of the pulmonary valve itself (ie, so-called dysplastic valve). The second relative contraindication to valvuloplasty is the presence of associated congenital heart disease requiring surgical intervention. The most common situation encountered is a patient with pulmonary valve stenosis and a large atrial septal defect. We believe that valvuloplasty should not be performed in this situation, since it will not eliminate the need for an open-heart operation.

PULMONARY ARTERY STENOSIS

Peripheral pulmonary arterial stenosis, or hypoplasia, can occur as an isolated lesion[25] or with tetralogy of Fallot,[26,27] postrubella syndrome,[28] William syndrome,[29] and Alagille syndrome.[30] Pulmonary artery stenosis also occurs as a complication of surgical aortopulmonary shunts and conduits. Despite the poor prognosis with isolated congenital pulmonary arterial stenosis,[31] and the association of branch pulmonary arterial stenosis with a poor outcome after surgical repair of tetralogy of Fallot, surgical correction of stenotic pulmonary arteries remains difficult and at times impossible.[31,32] Because surgical treatment of peripheral pulmonary artery stenosis is often unsatisfactory, balloon dilation angioplasty has been attempted for this lesion. In 1980, Martin et al[33] described the first trial of percutaneous transluminal angioplasty for peripheral pulmonary arterial stenosis. Following that report, Lock et al[34] described the use of transvenous angioplasty in newborn lambs with experimentally produced branch pulmonary arterial stenosis. These investigators were able to successfully dilate experimental pulmonary artery stenoses using a modified Gruntzig balloon catheter. In the lamb, balloon dilation was associated with a significant reduction in the systolic gradient across the stenosis, and an increase in the diameter of the site and the amount of blood flow through the vessel. Histologic evaluations demonstrated that balloon angioplasty enlarged the

stenotic areas by stretching and often tearing of the intima and media of the pulmonary artery.[35]

As a result of these experiments, clinical trials of balloon dilation angioplasty of peripheral pulmonary artery stenosis have been initiated.[36-39] For example, Ring et al[39] have reported successful angioplasty in 26/52 (50%) dilations performed in 24 children. The majority of these children had tetralogy of Fallot, with or without pulmonary atresia. These investigators felt that angioplasty failure was often related to patient age, and that nondilatable pulmonary artery stenoses were more common in children over 2 years of age.

Technique

The protocol for angioplasty of peripheral pulmonary arteries is similar to that for pulmonary valve stenosis. After baseline hemodynamic measurements and a selective pulmonary artery angiogram are performed, an end-hole catheter is advanced across the peripheral pulmonary artery stenosis. Since it is frequently difficult to enter stenotic, hypoplastic branch pulmonary arteries, no single catheter type will always work. Initially, we use a 7 French balloon-tipped wedge catheter, but if it fails to traverse the stenosis we will then try any one of the following: a right Judkins coronary catheter, an Amplatz coronary catheter, a cobra-curved catheter, or a sidewinder-curved catheter. Once the catheter is advanced to the distal pulmonary artery, a balloon angioplasty catheter is positioned across the stenosis over an 0.038 inch Teflon-coated guidewire. We and others recommend that the balloon diameter be 3 to 4 times the diameter of the narrowest segment of pulmonary artery. The balloon is inflated under low pressure (one or two atmospheres) with dilute contrast material, and its position is adjusted so that the pulmonary artery stenosis, located by the waist produced on the balloon, is at the midportion of the balloon. Under continuous fluoroscopic monitoring the balloon is further inflated until either the waist deformity disappears, or until the balloon's maximal dilation pressure (usually 4 to 7 atm) is achieved. Balloon inflation should be maintained for 10 to 60 seconds as tolerated by the patient. Systemic arterial pressure and heart rate should be constantly monitored during the dilation. After dilation, the balloon catheter is removed, an angiographic catheter is carefully exchanged over the guidewire, and a repeat angiogram and pressure pull-back is performed. As with angioplasty of coarctation of the aorta, if the exchange guidewire becomes dislodged the angioplasty site should not be recrossed by an unguided catheter because of the risk of vessel perforation; in this situation, a repeat angiogram should be performed in the main pulmonary artery. For safety (in case of pulmonary artery perforation or thrombosis), we usually perform angioplasty in only one major branch pulmonary artery per session.

Results

Acute results. Results of percutaneous balloon angioplasty for peripheral pulmonary artery stenosis have been less impressive than those for valvar pulmonary stenosis. To date, we have attempted balloon dilation of 37 pulmonary arteries in 25 patients. Since successful relief of vascular obstruction should increase the diameter of the obstructing segment, decrease the pressure gradient and increase blood flow across the site, we consider angioplasty of a peripheral pulmonary artery to be successful if two or more more of the following criteria are met: (1) $\geq$75% increase in pulmonary artery stenosis diameter; (2) $\geq$50% reduction in the systolic pressure gradient between the distal and main pulmonary arteries; (3) $\geq$50% reduction in either peak right ventricular systolic pressure or in the ratio of right to left ventricular systolic pressures; (4) $\geq$25% increase in total pulmonary blood flow. Using these criteria, pulmonary artery angioplasty has been successful in 20/37 vessels that we have dilated (54% success rate). Angioplasty has been successful regardless of the location of the stenosis, or whether the stenosis is discrete or diffuse in nature. In general, it has been very effective in children with pulmonary artery stenosis associated with tetralogy of Fallot or pulmonary atresia with ventricular septal defect (except when the stenosis is related to a surgical shunt). Figure 4A demonstrates a discrete stenosis of the right pulmonary artery at the insertion of a valved conduit in a 9-year-old boy with pulmonary atresia and a ventricular septal defect. After balloon angioplasty the diameter of the stenotic site had nearly doubled (Fig 4C), and the systolic pressure gradient from the con-

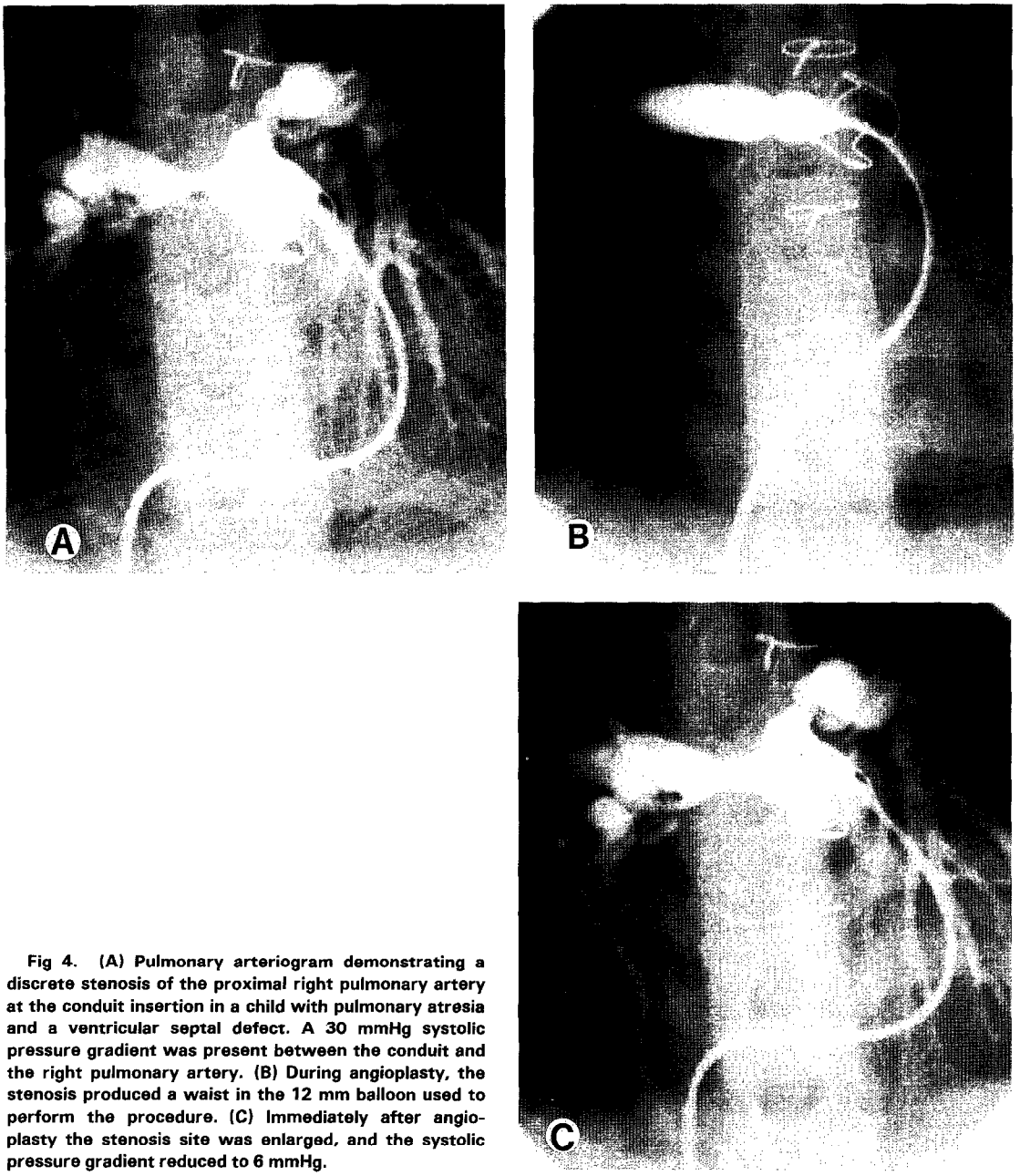

Fig 4. (A) Pulmonary arteriogram demonstrating a discrete stenosis of the proximal right pulmonary artery at the conduit insertion in a child with pulmonary atresia and a ventricular septal defect. A 30 mmHg systolic pressure gradient was present between the conduit and the right pulmonary artery. (B) During angioplasty, the stenosis produced a waist in the 12 mm balloon used to perform the procedure. (C) Immediately after angioplasty the stenosis site was enlarged, and the systolic pressure gradient reduced to 6 mmHg.

duit to the right pulmonary artery had decreased from 30 to 6 mmHg. Figure 5 illustrates the improvement in right pulmonary artery size after angioplasty in a 10-year-old girl with severe, diffuse, pulmonary artery hypoplasia.

In those cases where angioplasty was judged unsuccessful the procedure failed either because of technical difficulties positioning an adequate balloon across the stenosis, or because of the presence of a refractory stenosis. In contrast to the report of Ring et al,[39] we have observed no relationship between angioplasty success and patient age. The first reason for an unsuccessful angioplasty relates to difficulties in technique. We were unable to advance the angioplasty catheter across the stenotic segment in three of our first 13 patients (23%). With experience and the availability of small angioplasty catheters and stiff guidewires, however, the inability to traverse pulmonary artery stenoses has been nearly overcome. In situations where the pulmonary arteries cannot be entered during cardiac

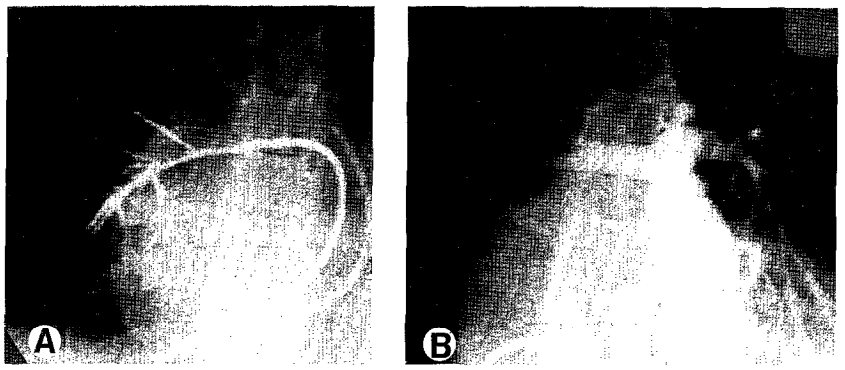

Fig 5. Right pulmonary arteriogram demonstrating severe diffuse hypoplasia of the entire right pulmonary artery in a 10-year-old girl (A) After a series of angioplasty procedures the sizes of the proximal right and left pulmonary arteries are significantly improved (B) and right ventricular systolic pressure was reduced to 70% of systemic.

catheterization, balloon angioplasty has been used successfully in the operating room.[40]

The second, and often most important, cause of an unsuccessful angioplasty relates to the refractory nature of some pulmonary artery stenoses. In our experience the following factors have been associated with stenoses that are less amenable to angioplasty: (1) stenosis at the site of a previous Blalock-Taussig, Waterston-Cooley or Potts shunt; (2) stenosis at the origin of the left pulmonary artery associated with a right ventricle to pulmonary artery conduit; (3) pulmonary artery stenosis after the arterial switch repair of D-transposition of the great arteries; and (4) pulmonary artery stenosis associated with either William syndrome or Alagille syndrome. Of nine pulmonary artery stenoses due to a previous aortopulmonary shunt (Blalock-Taussig [n = 6], Waterston-Cooley [n = 2], Potts [n = 1]), only 3/9 (33%) were successfuly dilated. Despite the use of large balloons and inflation pressures of up to 6 atm, the balloon waist could not be eliminated in these cases. We believe that failure of angioplasty in this setting may be related to the fact that stenosis at the site of a prior surgical shunt is caused by external fibrosis, rather than an anatomic defect of the pulmonary artery wall itself. A similar mechanism may also explain the difficulty we encountered with angioplasty of a proximal left pulmonary artery stenosis caused by a right ventricle to pulmonary artery conduit (n = 1), and a main pulmonary artery stenosis following the arterial switch repair of D-transposition (n = 1). Of the seven children in our series with either Alagille syndrome (n = 3) or William syndrome (n = 4), only one (a boy with Alagille syndrome who developed an aneurysm at the angioplasty site) had a satisfactory result of angioplasty for peripheral pulmonary artery stenosis. In most of these cases a large balloon was inflated until the waist was nearly eliminated, yet the stenosis returned immediately upon deflation of the balloon. On the basis of this experience we feel that balloon angioplasty is unlikely to succeed in children with pulmonary artery stenosis related to either the William or Alagille syndrome.

Complications. Significant complications have occurred following percutaneous balloon angioplasty for peripheral pulmonary artery stenosis. Other institutions have reported exsanguination from pulmonary artery rupture, transient arrhythmias, cyanosis, hypotension, and iliac vein obstruction.[36,39,41] In the 37 angioplasty procedures in our series, we have had four (11%) major complications. In one child with right pulmonary artery stenosis after repair of tetralogy of Fallot, the pulmonary artery was perforated by the guidewire. The patient required multiple transfusions and underwent emergency surgery at which time a small circular perforation of a distal branch pulmonary artery was repaired.[37] One patient had balloon angioplasty performed in the operating room at the time of surgical placement of a right ventricle to pulmonary artery conduit, but developed hemoptysis and severe hypoxemia and died 20 hours after surgery. At postmortem examination the lungs were congested with blood but no pulmonary artery perforation was found.[37] Another child developed asymptomatic pulmonary edema two hours after a successful right pulmonary artery angioplasty. The x-ray evidence of edema resolved over the following month. Finally, a child with Alagille syndrome developed a left pulmonary artery aneurysm after a successful angioplasty. No progression or regression of the aneu-

rysm was found at repeat catheterization 10 months later.

Follow-up. We have followed the children in our series with a successful pulmonary artery angioplasty for up to 6 years. Each child has remained well and without signs of subsequent deterioration. Seven children have undergone repeat cardiac catheterization. In all but one patient, the diameter of the stenosis and the systolic pressure gradient across the stenosis have remained similar to their immediate postangioplasty values. Others have also demonstrated that pulmonary artery angioplasty, when effective, appears to provide long-lasting relief of stenosis.[36,39]

Conclusions

In summary, balloon angioplasty of peripheral pulmonary artery stenosis can provide significant hemodynamic relief to a group of patients in whom surgical management is often difficult and unsuccessful. The best results in our series were generally obtained with naturally occurring stenoses (as opposed to stenosis related to a surgical shunt) in children with tetralogy of Fallot. In contrast, our data suggest that angioplasty is less likely to succeed if the pulmonary artery stenosis is the result of a previous aortopulmonary shunt or is related to the William or Alagille syndrome.

AORTIC VALVE STENOSIS

Conventional treatment for the child with aortic valve stenosis has been surgical. Typically, the first surgical procedure has consisted of an open aortic valvotomy. Postoperative recurrence of stenosis has been a problem, however, and subsequent surgery has often required an aortic valve replacement.[42] In a recent review of the long-term results of surgical valvotomy for congenital aortic stenosis, Hsieh et al found a 44% actuarial probability of reoperation (for restenosis or insufficiency) by 22 years.[42] Aortic valve replacement was required in 20/21 patients in this series. Because the surgical approach to congenital aortic stenosis is clearly palliative, a number of centers have begun to assess the effectiveness of percutaneous balloon valvuloplasty for this lesion.

In 1984 Lababidi first reported the acute effects of percutaneous balloon valvuloplasty in children with congenital valvar aortic stenosis.[43,44] The procedure was found to reduce acutely the transvalvar gradient in each of the 27 patients evaluated. Overall, balloon valvuloplasty reduced the systolic gradient from 108 mmHg to 32 mmHg. Following the procedure, mild aortic insufficiency was noted in seven patients and moderate insufficiency in one patient. Subsequently, a number of centers have reported similar acute results in childhood.[45-48] Percutaneous balloon valvuloplasty has been found to reduce acutely the peak systolic gradient of aortic valve stenosis by 50% to 60%. Mild aortic insufficiency is often produced, but severe regurgitation has been uncommon. Vascular complications, primarily consisting of hemorrhage and/or pulse loss, have occurred frequently. These problems have generally been managed conservatively and have caused few short-term serious effects. The mortality rate for the procedure is probably in the range of 1% to 2%. Several deaths have been reported, all in infants less than 6 months of age, and the majority in infants under 1 month of age with critical stenosis.[49] In these children, laceration of the aortic arch or avulsion of a valve cusp were felt to be the causes of death.

The mechanism by which percutaneous balloon valvuloplasty reduces the aortic valve gradient has been elucidated in clinical and autopsy studies. McKay evaluated the morphological effects of balloon dilation in post mortem and intraoperative studies of adults with calcific aortic stenosis.[50] In valves with commissural fusion, valvuloplasty separated the leaflets by splitting the fusion lines. Valve mobility was also improved by cracking of the nodular calcium deposits within the leaflets. Lababidi et al[43] and Walls et al[44] reported intraoperative observations of two aortic valves following unsuccessful balloon dilation. In each valve, tears along the lines of commissural fusion were observed. The evidence suggests, therefore, that in congenital valvar aortic stenosis balloon valvuloplasty relieves obstruction by producing tears along the lines of commissural fusion. The use of excessively large balloons has been shown to damage the left ventricular outflow tract, however. In a study of 16 normal lambs Helgason[46] produced tears of the ventricular septum, aortic valve leaflets, and ascending aorta with oversized balloons with

diameters from 1.2 to 1.5 times the diameter of the aortic valve annulus. No significant damage to the left ventricular outflow tract was noted in lambs following inflation of balloons 1.1 times the annulus diameter or smaller.

Technique

At our institution, balloon aortic valvuloplasty is performed percutaneously using a transarterial retrograde approach. Both femoral arteries are catheterized, without the use of a sheath, and the aortic valve is crossed in a retrograde fashion. The patient is then heparinized with 100 units/kg (maximum dose 3,000 units). The peak-to-peak systolic ejection gradient is determined from simultaneous recordings of left ventricular and ascending aortic pressures, and cardiac output is measured using the thermodilution or Fick technique. Before valvuloplasty, angiography is performed in the left ventricle and ascending aorta. Criteria for performing balloon aortic valvuloplasty have generally been those used in the past to indicate the need for a surgical valvotomy. Valvuloplasty is performed in the presence of: (1) a resting gradient ≥70 mmHg; (2) a resting gradient of 50-69 mmHg with symptoms or ischemic changes on resting or exercise ECG; (3) in the presence of congestive heart failure and low cardiac output regardless of gradient. Patients with a hypoplastic annulus or more than moderate aortic insufficiency have not been regarded as candidates for balloon valvuloplasty.

Both the single and double balloon techniques have been used at our institution. In either case, the balloon catheter is inserted percutaneously over an 0.035 inch exchange wire whose tip is curled in the left ventricular apex. With the single balloon technique, a balloon is chosen whose diameter is approximately equal to the diameter of the aortic valve annulus. With the double balloon technique (Fig 6), two balloons of similar size are chosen so that the sum of their diameters is approximately 1.3 times the diameter of the annulus. The deflated balloon(s) is advanced over the exchange wire and positioned across the aortic valve. The balloon(s) is then inflated by hand with a dilute mixture of contrast and saline. Care is taken to assure that the visible waist created by the valve is in the mid portion of the balloon(s). The balloon is rapidly deflated,

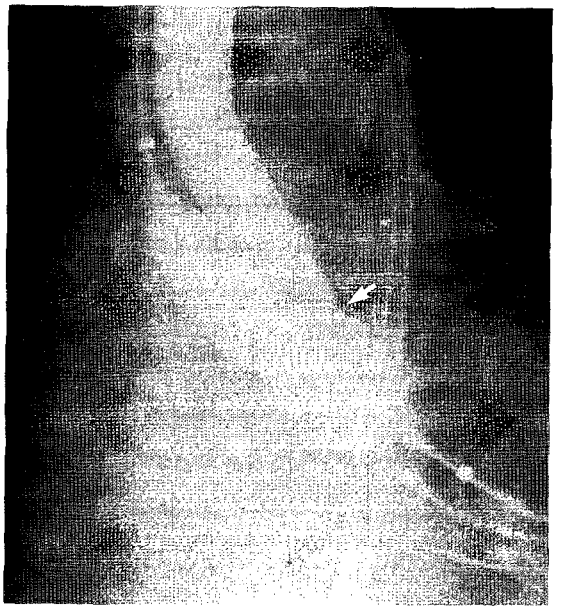

Fig 6. Double balloon aortic valvuloplasty in a teenage boy with aortic valve stenosis. The arrow points to the waist created on the balloons by the valve annulus.

attempting to limit inflation to less than ten seconds. Several repeat inflations are performed with minor changes in balloon position to assure proper location in the left ventricular outflow tract. After valvuloplasty the balloon catheter(s) is removed and a pigtail catheter advanced over the wire to the left ventricle. Measurements of left ventricular and ascending aortic pressures and cardiac output are repeated to document valvuloplasty effectiveness. After the catheters are withdrawn from the left ventricle, an aortic cineangiogram is filmed to assess aortic insufficiency that may have been induced. The catheters are then removed, hemostasis is achieved with digital pressure, and pressure dressings are applied. The children are observed overnight and generally discharged the next morning.

Results

Acute effects. Percutaneous balloon valvuloplasty has been performed at our institution in 27 children and adolescents with valvar aortic stenosis since July, 1985. All children have had congenitally bicuspid aortic valves. The patients ranged in age from 3 months to 21 years and in weight from 5 kg to 78 kg. The aortic valve annulus diameters ranged from 7 to 30 mm. Twenty children had native (unoperated) aortic stenosis, and seven had recurrent stenosis after a

previous surgical valvotomy. Overall, percutaneous balloon valvuloplasty acutely reduced the peak systolic gradient by 53%. The aortic valve gradient averaged 80 mmHg before valvuloplasty and 38 mmHg after the procedure ($P < .001$; Figs 7,8). Left ventricular systolic pressure decreased from 179 mmHg to 143 mmHg ($P < .002$), and left ventricular end-diastolic pressure decreased from 16 mmHg to 12 mmHg ($P < .01$). There was no difference between children with native stenosis or recurrent postoperative stenosis in the acute gradient reduction afforded by valvuloplasty. Heart rate and cardiac output did not change significantly after valvuloplasty. Catheter induced ventricular arrhythmias were common during the procedure, but no patient had significant ectopy after wires and catheters were removed from the left ventricle.

In our hands, the double balloon technique has provided more effective acute gradient relief than the single balloon technique in children with valvar aortic stenosis.[51] To compare the effectiveness of the two valvuloplasty techniques, the single and double balloon approaches were used in two consecutive groups of children. In the first 16 procedures a single balloon with an average balloon to annulus diameter ratio of 0.96 was used. In the subsequent 11 procedures, the double balloon technique was used in which two balloons are positioned across the valve and inflated simultaneously. In these procedures the ratio of the balloon diameter sum to valve annulus diameter averaged 1.32. The single balloon approach reduced the average peak systolic gradient by 43% from 82 mmHg to 46 mmHg, while the double balloon approach reduced the gradient by 67% from 76 mmHg to 26 mmHg. The peak systolic gradient after valvuloplasty was significantly lower when the double balloon approach was used ($P < .01$). Although this study was not randomized and therefore is vulnerable to a learning curve effect, we feel the data suggest the double balloon technique may achieve superior acute results compared with the single balloon technique in this patient population. The double balloon approach may have other advantages.[47] Smaller balloon catheters are required, which may be less traumatic to the femoral arteries. In addition, the use of two balloons extends the applicability of the technique to larger children and adolescents whose annulus diameter often exceeds 20 mm.

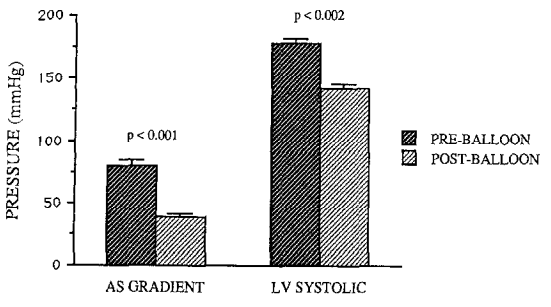

Fig 7. Acute effects of balloon valvuloplasty on peak-to-peak AS gradient, and the LV systolic pressure in 27 children with valvar AS. Valvuloplasty reduced the AS gradient by 53%, and the LV systolic pressure by 20%. AS, aortic stenosis; LV, left ventricular.

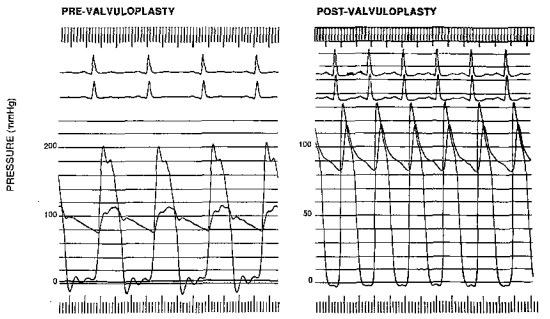

Fig 8. Simultaneous LV and aortic pressure tracings before and after balloon valvuloplasty in a 19-year-old male with valvar AS. The peak-to-peak pressure gradient was reduced acutely from 83 mmHg to 19 mmHg, with only a trivial increase in aortic regurgitation (Note the different pressure scales on the two panels.)

Complications. The complications of aortic balloon valvuloplasty encountered in this series relate to the development of aortic insufficiency and to vascular injury; there has been no mortality in this series. Valvuloplasty produced new aortic insufficiency in 6/27 children (22%). Aortic insufficiency increased by 1+ in 3, 2+ in 2, and 3+ in one patient. No patient has required medical or surgical therapy for valvuloplasty-induced aortic insufficiency. Bleeding from the puncture site was sufficient to require transfusion in two children. Femoral artery pulse loss occurred in four children, and responded to 24 to 48 hours of heparin therapy. A 14-year-old boy developed mild to moderate mitral insufficiency 2 months after aortic valvuloplasty, possibly related to trauma to the mitral valve apparatus during the procedure.[45]

Conclusions

Conventional surgical treatment for a child with valvar aortic stenosis, whether it be valvotomy or aortic valve replacement is palliative. Surgical aortic valvotomy typically reduces the systolic gradient to approximately 20 to 40 mmHg, and mild to moderate aortic insufficiency may occur in up to 60% of patients.[52-55] Late reoperation is often required after either surgical valvotomy or aortic valve replacement.[42] The early results of percutaneous balloon valvuloplasty in children with aortic stenosis, therefore, compare very favorably to the surgical experience. The available data indicate that balloon valvuloplasty can be expected to reduce the peak systolic gradient of valvar aortic stenosis by approximately 50% with minimal mortality, and with generally only minor increases in aortic insufficiency. The procedure is effective in children with unoperated aortic stenosis, and in children with recurrent stenosis following a prior surgical valvotomy. Balloon valvuloplasty should be considered as an alternative to surgical valvotomy in children with aortic stenosis who meet traditional criteria for intervention. For children with unoperated aortic stenosis, balloon valvuloplasty should remain an investigational procedure until follow-up studies indicate that the rate of recurrent stenosis is not unacceptably high. For children with recurrent aortic stenosis following a previous surgical valvotomy, we feel that balloon valvuloplasty should be attempted before reoperation since the surgical alternative is often an aortic valve replacement.[42] Long-term follow-up studies are needed, both for native and recurrent postoperative aortic stenosis, to document that the benefits of percutaneous balloon valvuloplasty are long lasting.

COARCTATION OF THE AORTA

For nearly 40 years the conventional treatment of coarctation of the aorta has been surgical. Follow-up studies have shown, however, that the long-term prognosis after surgical repair of coarctation is not entirely benign. The clinical outcome following surgical repair may be affected by residual or recurrent stenosis, aneurysm formation at the repair site, resting or exercise hypertension, and by associated lesions such as aortic stenosis. A 25% to 60% incidence of recurrent coarctation following resection and end-to-end anastomosis in infancy has been reported,[56-58] and we have been unable to show that the use of the subclavian angioplasty has affected this appreciably.[59] Reoperation for a recurrent coarctation may be difficult, and has carried a 4% to 5% mortality rate at our institution.[60] Finally, after a prosthetic patch repair of coarctation we have found evidence for aneurysmal dilatation at the repair site in 24% of patients, an average of 5.6 years following repair.[61] It is clear that surgical coarctation repair is no panacea. Therefore, angioplasty has been attempted at a number of institutions as an alternative to surgical repair of coarctation of the aorta.

In 1979 Sos et al[62] demonstrated the feasibility of coarctation angioplasty in segments of coarcted aorta that had been surgically excised. Subsequently, a number of reports have documented the effects of percutaneous balloon angioplasty in children with coarctation of the aorta; the procedure has been used in children with a native (unoperated) coarctation and children with a recurrent coarctation following surgical repair. In 1988 angioplasty remains controversial for native coarctation, but has gained wide acceptance for recurrent coarctation.

Native Coarctation

A number of institutions have reported that balloon angioplasty is effective in reducing or eliminating the aortic obstruction in many children with a native coarctation.[63-71] Morrow et al[64] recently reported short- and intermediate-term results of angioplasty in 33 children and young adults with a native coarctation. The procedure decreased the systolic gradient from an average of 46 mmHg to 8 mmHg, and was felt to be successful in 31 of 33 patients. Follow-up data in 20 patients an average of 16 months after angioplasty found no significant restenosis. Two children were found to have a small aneurysm at the dilation site. Lababidi et al[65] described similar results in 20 children. Dilation acutely decreased the coarctation gradient from 53 mmHg to 10 mmHg without mortality or serious morbidity. Nine children underwent repeat catheterization an average of 8.6 months after dilation, and were found to have a mean residual gradient of 18 mmHg (range 0 to 32 mmHg). Only two children had an appreciable increase in the residual gradient at follow-up, and both were

neonates at the time of dilation. No aneurysms were encountered.

Balloon angioplasty for native coarctation remains controversial primarily because of concern over restenosis and late aneurysm formation. Restenosis appears to be a particular problem when dilation is performed in early infancy. A number of centers have described early restenosis in small numbers of neonates and young infants following balloon angioplasty.[63,66,68,69] A number of these children have been effectively palliated, however, as documented by improvement or resolution of hypertension or heart failure despite the presence of a residual gradient. Although based on very few patients, these results suggest that balloon angioplasty may provide a palliative approach to coarctation in early infancy allowing surgical repair to be delayed to an age when the long-term surgical results are more favorable.[60]

Late aneurysm formation is also a major concern after balloon dilation of a native coarctation. In 1986 Marvin et al[72] reported angiographic evidence of aneurysm formation at the dilation site in six of 11 children following angioplasty of a native coarctation. Histologically, the aneurysms were found to have an intact adventitia, with localized medial thinning and linear medial tears. The incidence rate of aneurysm formation after native coarctation angioplasty has varied widely in published reports, from 0%[65,71] to as high as 55%.[72] The two largest follow-up series both found small aneurysms in two of 20 patients evaluated by angiography or magnetic resonance imaging 1 to 2 years after angioplasty[63,64]; these data suggest that the true incidence of this complication may be approximately 10%. Careful long-term studies clearly are needed to define better the incidence and natural history of late aneurysms following balloon angioplasty of native coarctation.

Recurrent Postoperative Coarctation

Percutaneous balloon angioplasty has also been shown to be effective in patients with recurrent stenosis following previous surgical repair of coarctation of the aorta.[21,65,66,71,73,74] Angioplasty was introduced for recurrent coarctation in 1983 by Lock et al,[66] who described the procedure in five children, and by Kan et al,[73] who described the procedure in seven children and adolescents. Angioplasty provides acute relief of obstruction in the majority of children with recurrent stenosis following surgical repair, provided the stenosis is not long-segment in nature or associated with significant hypoplasia of the transverse arch or isthmus. Typically, reduction of the systolic gradient below 20 mmHg can be expected. The procedure appears equally effective regardless of the type of prior surgical repair.[21,74] There have been few reports of the long-term outcome after angioplasty for recurrent coarctation, and no systematic angiographic follow-up studies have been performed. Soulen et al[75] reported follow-up evaluation by magnetic resonance imaging in eight patients an average of 40 months after angioplasty of recurrent coarctation. Mild residual narrowing was imaged in most patients, but no aneurysm formation was found. Residual pressure gradients ranged from 0 to 12 mmHg. Saul et al[74] have described a small aneurysm at the dilation site in two children after angioplasty for recurrent arch stenosis following repair of an interrupted aortic arch. Overall, the available data suggest that balloon angioplasty provides effective nonsurgical treatment for recurrent postoperative coarctation, although more longitudinal data are necessary to define the true incidence of recurrent stenosis and aneurysm formation.

Technique

Balloon coarctation angioplasty typically is performed transarterially using a retrograde approach. Both femoral arteries are entered percutaneously and the patient is fully heparinized with 100 units/kg (maximum dose 3,000 units). The coarctation is crossed in a retrograde fashion and the peak systolic gradient is determined from simultaneous recordings in the ascending and descending aorta. An anterior-posterior (AP) and lateral aortogram are filmed, and the diameter of the aortic isthmus measured just beyond the left subclavian artery using a calibrated marker catheter. As with other lesions, we have used criteria for coarctation angioplasty similar to those used to indicate the need for surgical repair. Angioplasty has been performed in the presence of (1) a resting gradient $\geq$30 mmHg; (2) resting upper extremity hypertension;(3) in the presence of congestive heart failure regardless of gradient. Patients with a long-segment

coarctation or associated severe tubular hypoplasia of the aortic arch have been excluded.

An angioplasty catheter is used whose balloon diameter is approximately equal to the diameter of the aortic isthmus just beyond the left subclavian artery. We have not used the double balloon technique for coarctation, although this has been described.[76] The deflated balloon is advanced over an exchange wire until the balloon is positioned across the coarctation. The balloon is then inflated by hand until the waist produced on the balloon by the coarctation is seen to disappear. Usually several inflations are performed with minor changes in balloon position. The balloon catheter is then removed and a pigtail catheter is advanced over the exchange wire to the ascending aorta. We never advance the catheter through a freshly dilated portion of aorta unless it is guided by a wire, because of the risk of aortic perforation. Measurements of pressure in the ascending and descending aorta and thermodilution cardiac output are repeated to assess the acute hemodynamic results. An ascending aortogram is filmed to image the coarctation site and exclude the presence of aortic tears or aneurysms. The catheters are removed and hemostasis is achieved with direct pressure. The patients are observed overnight in a moderate care setting with careful monitoring of upper and lower extremity BPs and pulses.

Results: Native Coarctation

Acute effects. Since January, 1983 percutaneous balloon angioplasty has been performed at our institution in 49 children and adolescents with coarctation of the aorta. In 31 children angioplasty was performed as initial treatment of a native coarctation, that is one which had not undergone prior surgical repair. These 31 children ranged in age from five weeks to 14.7 years (mean 6.0 years), and in weight from 4.4. kg to 81.5 kg (mean 23.8 kg). Associated cardiovascular lesions were present in nine children. Four had a small PDA, two mild to moderate aortic stenosis, one a small ventricular septal defect, one mitral stenosis, and one infant had an atrioventricular septal defect with pulmonic stenosis, dextrocardia and a right aortic arch (Fig 9). Five girls also had Turner syndrome. Balloon angioplasty was performed with balloons ranging in diameter from 5 to 20 mm. The diameter of the isthmus ranged from 5 to 21.5 mm in these children, and the ratio of balloon to isthmus diameter averaged 1.04. Angioplasty acutely reduced the peak systolic coarctation gradient in every child. For the group as a whole the systolic coarctation gradient decreased by 73% from 49 mmHg to 13 mmHg ($P < .001$). The systolic pressure in the ascending aorta also decreased, from 141 mmHg to 126 mmHg ($P < .001$). There was no significant change after angioplasty and heart rate or cardiac output.

Complications. Acute complications of angioplasty most commonly involved injury to the femoral artery. Pulse loss occurred in six children, but was transient and responded to 24 to 48 hours of heparin therapy. Despite return of the pulse, one child was found to have an occluded right femoral artery at an elective follow-up catheterization. Significant hemorrhage from the angioplasty site occurred in one child and required transfusion. A cerebrovascular accident occurred in an 8-year-old boy early in our experience, most likely a result of thromboembolism of clot from the exchange wire. We have subsequently modified our technique to include thorough heparinization of each patient and use of new clean exchange wires during every catheter exchange. There was no paradoxical hypertension[77] and no angioplasty-related deaths in our series.

Follow-up. It has been our protocol to assess the longer-term results with a follow-up cardiac catheterization in all children 12 months or more after balloon angioplasty of a native coarctation.[63] Twenty children have now undergone this follow-up study, an average of 14.2 months after angioplasty (Fig 10). In these children the residual coarctation gradient ranged from -5 mmHg to 36 mmHg (mean 14 mmHg), and 14 of 20 had a residual gradient less than 20 mmHg. For the group as a whole, there was no significant increase in coarctation gradient during this 14 month follow-up period. The residual gradient immediately after angioplasty and that measured at follow-up were virtually identical (13 mmHg *v* 14 mmHg, $P = .62$). The systolic pressure in the ascending aorta, however, had decreased at follow-up from 131 mmHg to 117 mmHg ($P < .01$). A small aneurysm at the angioplasty site was present in two children. Neither child was symptomatic and in neither

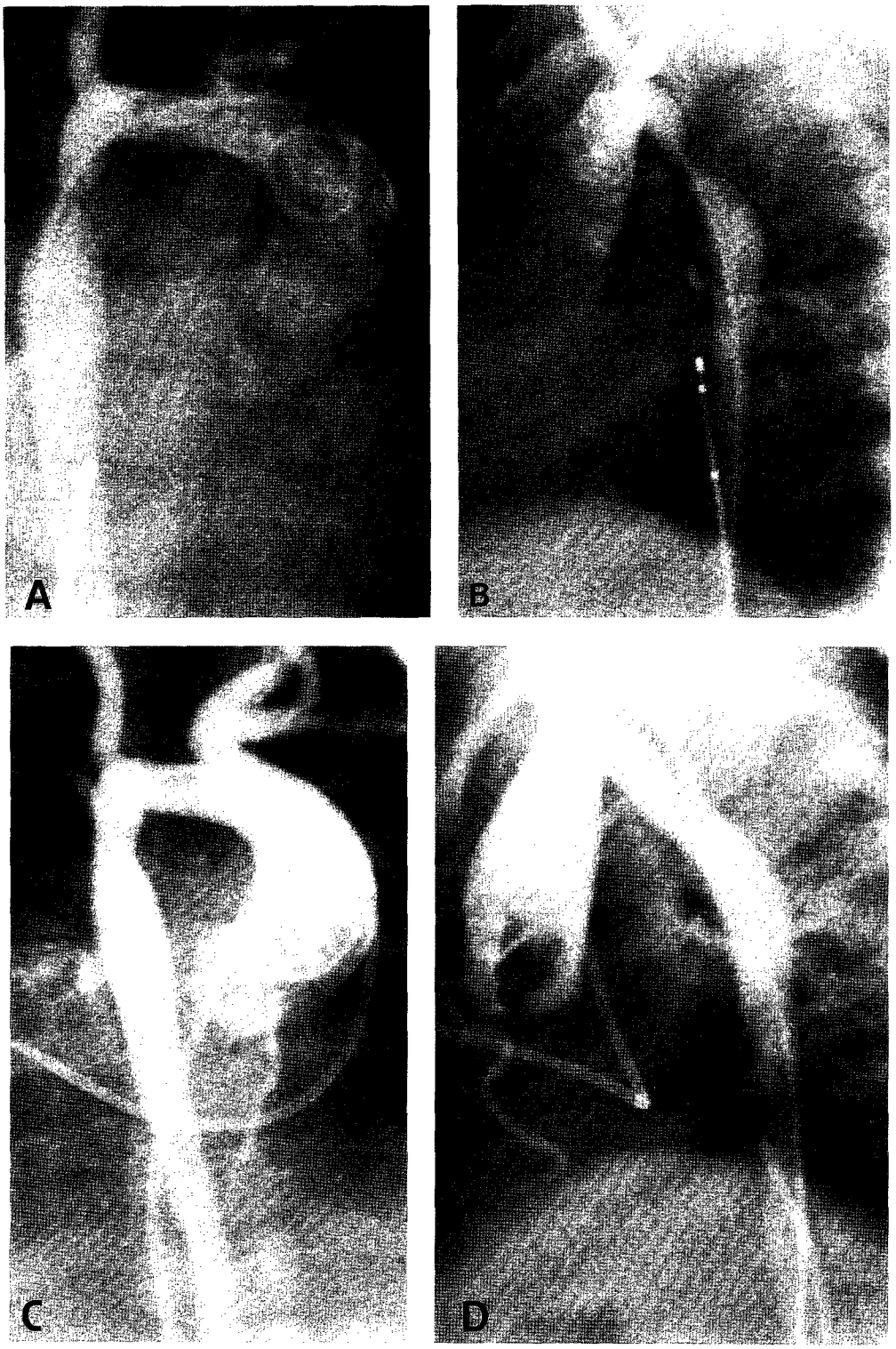

Fig 9. (A,B) Anteroposterior and lateral aortogram demonstrating a thoracic coarctation in a 10-month-old infant with complex cyanotic heart disease, dextrocardia, and a right aortic aortic (Table 1, case 5). The peak systolic pressure gradient measured 54 mmHg. An electrode catheter (with a known interelectrode distance) is filmed to allow adjustment for image magnification when measuring a vessel or annulus size. (C,D) Repeat aortogram 1 year after angioplasty shows virtually no evidence of the coarctation and no aneurysm formation. There was no residual pressure gradient between the ascending and descending aorta.

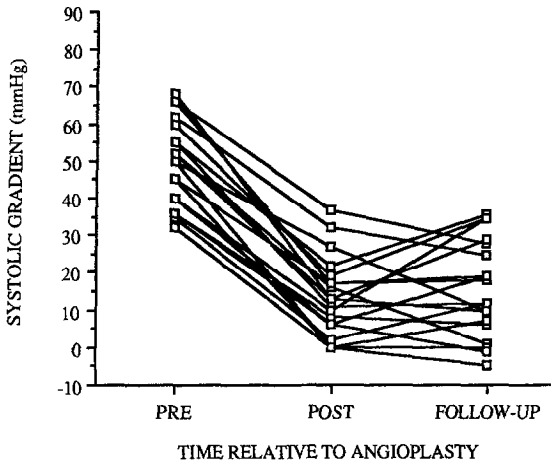

Fig 10. Peak systolic pressure gradients in 20 children with a native coarctation, measured at cardiac catheterization before and immediately after angioplasty, and at follow-up (mean 14.2 months after angioplasty). In this group, the systolic coarctation gradient averaged 51 mmHg before angioplasty, 14 mmHg immediately after angioplasty, and 13 mmHg at follow-up.

was the aneurysm detected by chest x-ray or echocardiography. One child, whose small aneurysm was associated with a 25 mmHg residual coarctation gradient, has undergone successful surgical repair (Fig 11). The second child is being followed carefully, and has shown no progression in aneurysm size over a 2-year period. Our follow-up data, therefore, document a 10% incidence of aneurysm formation following angioplasty of a native coarctation in childhood, a rate identical to that reported by Morrow.[64] Based on our follow-up catheterization data, angioplasty has achieved a good result (ie, gradient < 20 mmHg and no aneurysm) in 13 of 20 children (65%) with a native coarctation.

Our limited experience with coarctation angioplasty in infancy confirms previous reports suggesting that angioplasty may be less effective in infants.[63,66,68,69] In our series, five children underwent angioplasty of a native coarctation before 12 months of age (Table 1). Four infants had congestive heart failure, and a fifth had severe upper extremity hypertension. These children have been followed for up to 24 months, and three have undergone elective follow-up catheterization and angiography (Fig 9). Despite the presence of a residual gradient in 4/5 children (ranging from 28 to 42 mmHg), the procedure has provided effective palliation in four infants. Congestive heart failure resolved in 3/4 children and hypertension improved in the fourth. In one child (case 3, Table 1) angioplasty acutely diminished the coarctation gradient from 62 to 19 mmHg, but a substantial gradient recurred on the third day following the procedure. There was little change in the patient's congestive heart failure, and he therefore underwent surgical coarctation resection and has done well.

Results: Recurrent Postoperative Coarctation

Acute effects. Percutaneous balloon angioplasty has been performed at our institution in 18 children with recurrent coarctation following a previous surgical repair. These 18 patients ranged in age from 4 months to 22 years (mean 7.2 years) and in weight from 4.9 kg to 88.5 kg. Previous surgery consisted of resection and end-to-end anastomosis (n = 9), left subclavian angioplasty (n = 4), prosthetic patch aortoplasty (n = 4), and repair of an interrupted aortic arch in (n = 1). Associated cardiovascular lesions included a ventricular septal defect in five children, aortic stenosis in five children, and Shone's complex in one child. None of the children had Turner syndrome. All children had a discrete residual or recurrent coarctation documented at the site of surgical repair (Fig 12). Angioplasty was performed from 3.5 months to 22 years after surgical coarctation repair, using balloons ranging from 6 to 20 mm in diameter. The ratio of balloon to isthmus diameter averaged 1.01. Angioplasty acutely reduced the systolic coarctation gradient in these 18 children by an average of 81%, from 42 mmHg to 8 mmHg ($P < .001$). The procedure was equally effective regardless of the type of prior surgical repair. Only two children had a residual gradient exceeding 20 mmHg after angioplasty. In one child this was due to the use of an undersized balloon. In the second child the residual gradient was due to diffuse hypoplasia of the aortic arch. Both children subsequently underwent successful reoperation. In a third child angioplasty acutely reduced the gradient to 10 mmHg, but the gradient recurred within days and reoperation was required.

Complications. Acute complications occurred in several patients undergoing angioplasty for a recurrent coarctation, but no deaths were attributable to the procedure. A 14-year-old boy was found to have an aortic tear immediately after angioplasty which required emergent surgi-

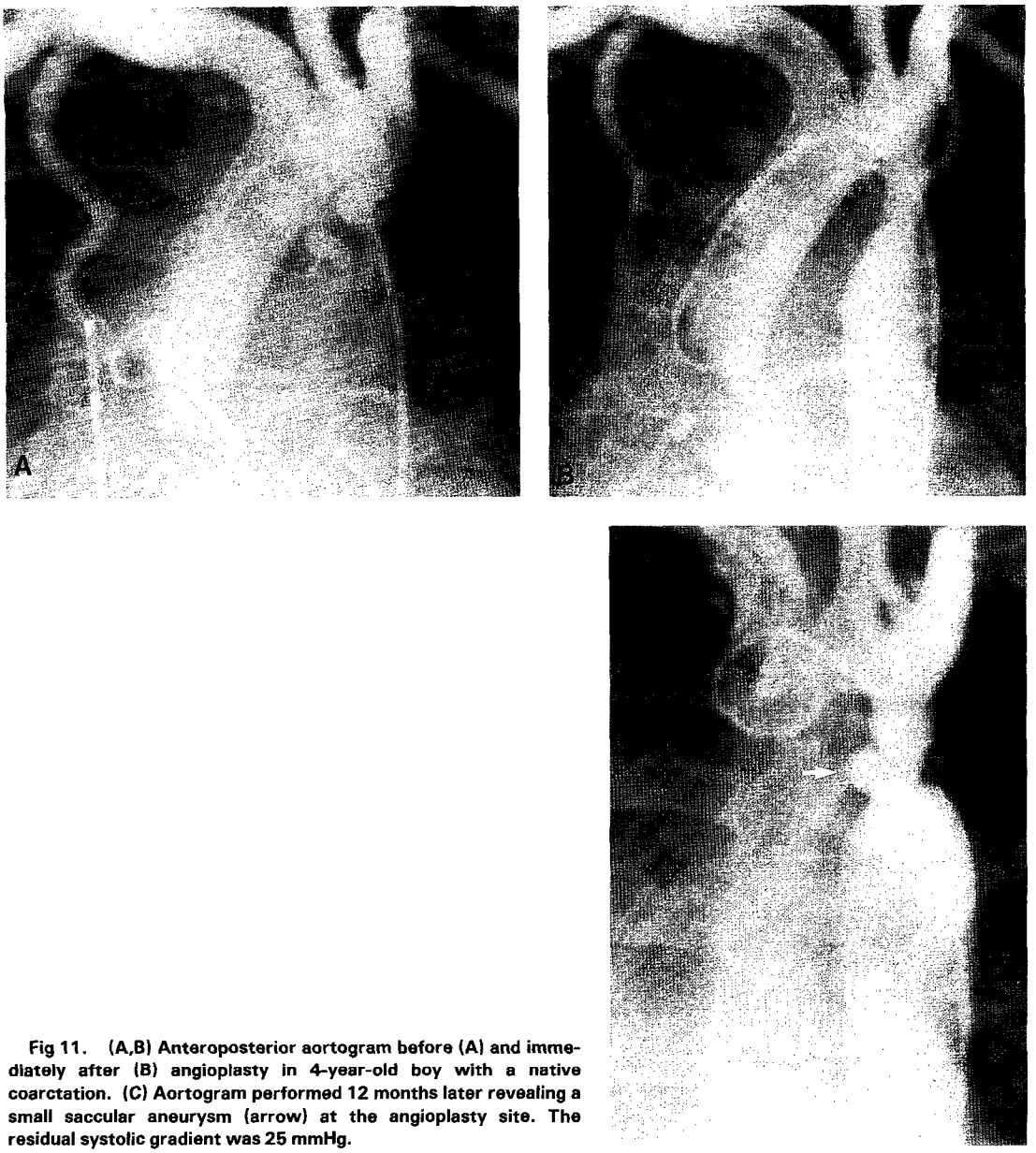

Fig 11. (A,B) Anteroposterior aortogram before (A) and immediately after (B) angioplasty in 4-year-old boy with a native coarctation. (C) Aortogram performed 12 months later revealing a small saccular aneurysm (arrow) at the angioplasty site. The residual systolic gradient was 25 mmHg.

cal repair. In the operating room a small linear tear was observed at the site of a prior end-to-end anastomosis, and fresh hemorrhage was contained by adventitia and surrounding scar tissue. In two other patients, femoral artery injury occurred consisting of pulse loss in one and hematoma in another.

Follow-up. Follow-up cardiac catheterizations have not been routinely performed in these patients. Clinical evaluation, however, has been carefully performed in all including arm and leg BPs and two-dimensional echocardiograms. Repeat cardiac catheterization has been performed in two children and magnetic resonance imaging in three. Other than the three children noted above who required surgical repair, angioplasty has provided effective and long-lasting relief of stenosis. There have been no other children in whom significant stenosis has recurred following angioplasty. None of the children has evidence of aneurysm formation following balloon angioplasty of a recurrent coarctation.

Conclusions

Percutaneous balloon angioplasty has been shown to reduce acutely the pressure gradient in

Table 1. Pertinent Clinical and Hemodynamic Data Regarding Five Infants (<12 mo) Who Underwent Balloon Angioplasty of a Native Coarctation

Case	Age	Weight (kg)	Balloon Size (mm)	Gradient (mm Hg)			AO Systolic (mm Hg)			Heart Failure	
				Pre	Post	F/u*	Pre	Post	F/u*	Pre	F/u*
1	5 wk	5.1	7	45	10	35	145	108	115	–	–
2	6 wk	4.5	5	74	17	32	172	117	122	+	–
3	7 wk	4.7	5	62	19	42	115	104	122	+	+
4	6 mo	7.5	7	66	37	28	148	152	120	+	–
5	10 mo	4.4	5	54	9	0	120	110	102	+	–

Abbreviations: Ao, aortic; F/u, follow-up; Pre, immediately before angioplasty; Post, immediately after angioplasty.

*F/u of one week in case 3, who had early recurrence of gradient and required early surgical repair. F/u of 10 to 24 months in remaining cases. F/u pressures obtained at cardiac catheterization in cases 1,4,5; by cuff measurements in cases 2,3.

children with a native coarctation or with recurrent stenosis following previous surgical repair. For children with a native coarctation, we consider balloon angioplasty to be an investigational procedure. Surgical repair remains the conventional therapy for these children, but because of the morbidity and mortality associated with surgery, as well as recurrent postoperative stenosis and aneurysm formation,[56-61] angioplasty has been used as an alternative nonsurgical form of treatment. In our experience balloon angioplasty of a native coarctation has yielded good results, that is, a residual gradient <20 mmHg and no aneurysm, in approximately 2/3 of the children. Our follow-up data suggest that restenosis is unlikely to occur during a 1 to 2 year follow-up period. The majority of children with a poor long-term result have been so defined because of a residual gradient >20 mmHg. A significant residual gradient appears more likely when balloon angioplasty is performed in infancy. Nevertheless, in our experience, the majority of these infants will be well palliated with relief of heart failure and hypertension, and surgical repair can be postponed to an older age when the long-term results of surgery are more favorable. Aneurysmal aortic dilatation occurs in approximately 10% of children following balloon angioplasty of a native coarctation. We identified a small aneurysm at the dilation site in 2 of 20 children. More follow-up data are required to document the long-term relief of obstruction and the natural

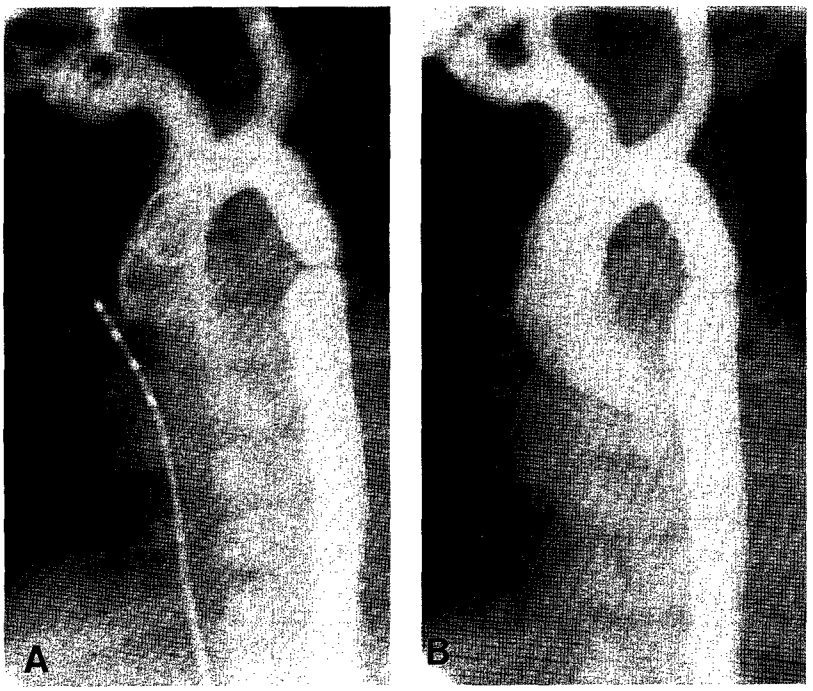

Fig 12. (A) Anteroposterior aortogram in a 20-month-old boy with subaortic stenosis and a residual coarctation, 18 months after a subclavian angioplasty repair. There is a discrete residual coarctation present, with a peak systolic pressure gradient of 30 mmHg. (B) Aortogram immediately after angioplasty demonstrating marked improvement in the residual coarctation. The systolic pressure gradient was reduced to 0 mmHg.

history of the aortic aneurysms following balloon dilation of a native coarctation. We would suggest, however, that balloon angioplasty may prove to be a rational first step in a treatment strategy which would include subsequent surgery should angioplasty be ineffective. Such a strategy may allow 60% to 70% of children to avoid surgical repair.

Percutaneous balloon angioplasty has been considered by some to be the treatment of choice for recurrent coarctation following previous surgical repair.[78] We would concur, because the procedure is effective if the recurrent stenosis is discrete and not associated with aortic arch hypoplasia, and because many of these children have already undergone multiple surgical procedures. However, balloon angioplasty is not without its risks in this patient population. Our most serious complication, an aortic tear requiring emergent surgical repair, occurred in a teenage boy with recurrent stenosis following a resection and end-to-end anastomosis. Furthermore, the long-term effectiveness of balloon angioplasty for recurrent postoperative coarctation has not been well documented, nor has the incidence of angioplasty-induced aneurysms been determined. Thus, although we feel that percutaneous balloon angioplasty is the preferable treatment for children with a recurrent postoperative coarctation, follow-up data are needed to document the long-term effectiveness of the procedure in this patient population.

MITRAL VALVE STENOSIS

Although congenital mitral stenosis is a rare lesion, rheumatic mitral stenosis remains a common cardiac disorder in much of the world. The surgical options available for the management of rheumatic mitral stenosis include closed commissurotomy, open commissurotomy, and mitral valve replacement. Congenital mitral stenosis presents a more difficult surgical challenge which may require splitting of fused papillary muscles, splitting or excision of fused chordae, and often mitral valve replacement. In 1984, Inoue reported the successful use of a specially designed balloon valvuloplasty catheter to treat rheumatic mitral stenosis in six adults.[79] Subsequently, we and others have used a similar technique to perform percutaneous balloon mitral valvuloplasty in children with congenital or rheumatic mitral stenosis.[80-82] Lock et al, for example, performed balloon valvuloplasty in eight children and young adults with rheumatic mitral valve stenosis.[81] Using the single balloon technique from a transseptal approach, they were able to nearly double the indexed mitral valve area, from a mean of 0.73 cm^2/m^2 to 1.34 cm^2/m^2. Short-term follow-up of two to eight weeks obtained by cardiac catheterization in six patients, demonstrated sustained hemodynamic improvement in four and some degree of restenosis in two. In total, balloon mitral valvuloplasty has been reported in only 16 children and adolescents (15 with rheumatic and one with congenital mitral stenosis).[47,80-82] The combined hemodynamic results in these published cases documents a reduction in left atrial "a" wave to left ventricular pressure gradient from 22.6 mmHg to 8.2 mmHg ($n = 16$), an increase in mitral valve area from 0.77 cm^2/m^2 to 1.29 cm^2/m^2 ($n = 11$), and a reduction in mean pulmonary artery pressure from 34.5 mmHg to 27 mmHg ($n = 11$). There is very little experience with balloon valvuloplasty in children with congenital mitral stenosis, and the results of the procedure may be expected to differ from that described in rheumatic disease since mitral valve pathology is quite different in the two disorders.

Technique

Before performing mitral valvuloplasty a complete hemodynamic and angiographic assessment should be obtained, including measurement of left atrial and left ventricular end-diastolic pressures, cardiac output, and a left ventricular angiogram. If the patient has been in chronic atrial flutter or fibrillation, a 2-dimensional echocardiogram should be obtained to rule out the presence of a left atrial thrombus.

The valvuloplasty procedure can be performed using one or two balloons. If the single balloon technique is to be used, valvuloplasty is performed as follows. The left atrium is entered with an 8-12 French Mullins transseptal sheath. After entry into the left atrium is accomplished, the patient is anticoagulated with heparin (100 units/kg, maximum dose of 3,000 units). A 7 French balloon end-hole catheter is advanced from the left atrium through the mitral and aortic valves to the descending aorta. A teflon coated 0.035 inch exchange guidewire is passed through the

catheter and positioned in the descending aorta. The sheath and balloon catheter are then withdrawn, and the valvuloplasty catheter is advanced over the guidewire across the atrial septum and mitral valve to the apex of the left ventricle. We have found that if a transseptal sheath of 9 French or larger is used, then it is not necessary to dilate the atrial septum in order to advance a valvuloplasty balloon smaller than 20 mm in diameter into the left atrium. Otherwise, the atrial septum is dilated with an 8 mm angioplasty balloon before the valvuloplasty catheter is advanced across the atrial septum. We choose a catheter whose balloon diameter is equal to the maximal estimated mitral annulus diameter using two-dimensional echocardiography. Once the valvuloplasty catheter is positioned across the mitral annulus, the balloon is inflated with dilute contrast until the waist in the balloon created by the stenotic mitral valve disappears. The balloon is inflated two or three times, with the duration of each inflation limited to ten seconds or less. After valvuloplasty is performed the hemodynamic measurements and a left ventricular angiogram are repeated.

Our preferred method is to perform mitral valvuloplasty using the double balloon technique, in which the mitral valve is dilated with two balloons inflated simultaneously (Fig 13). The advantage of the two balloon technique is that smaller catheters are used, and dilation of the atrial septum is often not necessary. We choose two balloons whose diameters sum to approximately 1.3 times the diameter of the mitral annulus. The two balloon technique can be accomplished in one of two ways. The first approach is to perform two transseptal punctures. Two 8-12 French Mullins sheaths are advanced into the left atrium, and two 7 French end-hold catheters are passed through the mitral and aortic valves to the descending aorta. Two exchange wires are then positioned in the aorta and two valvuloplasty catheters advanced across the mitral valve. The second approach requires only one transseptal catheter. Mansfield Scientific (Mansfield, MA) has developed a double-lumen catheter through which two guidewires can be advanced to the left heart through a single transseptal sheath. Using this catheter, two exchange guidewires are positioned across the mitral valve. The transseptal catheter is removed and the two valvuloplasty catheters are advanced, one at a time, through the same venous entry site and the same atrial septal puncture.

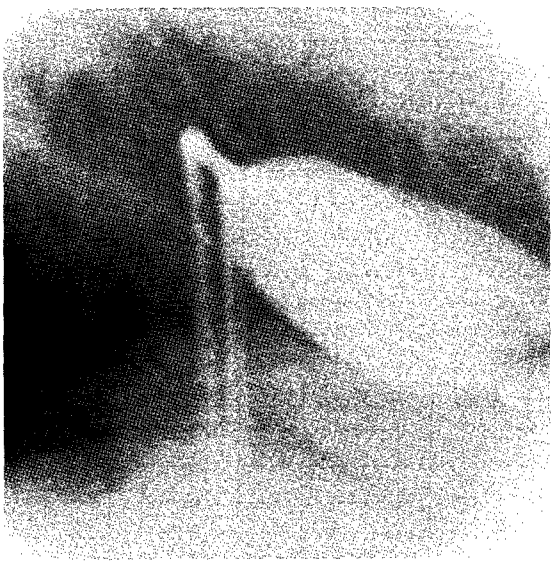

Fig 13. Lateral angiogram demonstrating a double balloon mitral valvuloplasty procedure.

Results

Acute effects. We have attempted mitral valvuloplasty in five children, three with congenital mitral stenosis and two with rheumatic mitral stenosis. In two children with congenital mitral stenosis we were unable to cross the mitral valve and valvuloplasty could not be performed.[80] In the other three children, balloon valvuloplasty acutely reduced the gradient between the left atrial "a" wave and left ventricular end-diastolic pressures from a mean of 28 mmHg to 12 mmHg, increased the mitral valve area from 0.87 cm^2/m^2 to 1.19 cm^2/m^2, and reduced the mean pulmonary artery pressure from 42 mmHg to 29 mmHg.

Complications. We have experienced few acute complications with balloon mitral valvuloplasty. In the two children with rheumatic mitral stenosis, the procedure caused a trivial increase in the degree of mitral insufficiency. No patient has developed hemoptysis, pulmonary edema, or evidence of thromboembolic events. Although not encountered in our series, a small left-to-right atrial shunt has been reported as a complication of the procedure.[81,82]

Follow-up. Long-term follow-up of our three patients after successful valvuloplasty has documented the best outcome in the two children with rheumatic mitral stenosis. These two children have been followed for 3.5 and 2 years, respectively, and both have reported persistent symptomatic relief. Repeat catheterization in one 1.5 years after valvuloplasty demonstrated sustained hemodynamic improvement. The one child who underwent valvuloplasty for congenital mitral stenosis had symptomatic improvement for 6 months. Symptoms returned, however; therefore, surgery was performed. At surgery there was evidence of valvuloplasty-induced separation of the anterior, but not the posterior commissure.

Conclusions

At present, balloon valvuloplasty remains an experimental form of treatment for children with mitral stenosis. Based on the adult experience and the limited published experience in children, it appears that balloon valvuloplasty may have an important role in the management of children with rheumatic mitral stenosis. However, before mitral valvuloplasty becomes the treatment of choice for children with rheumatic mitral stenosis, more follow-up data are necessary to define the extent and duration of clinical improvement provided by this technique. There is too little experience to speculate on the ultimate role of balloon valvuloplasty in the management of children with congenital mitral stenosis

ATRIAL SEPTAL DEFECT (SECUNDUM)

When a secundum atrial septal defect (ASD) is large enough to cause right ventricular volume overload, conventional treatment has been and still is open surgical repair. Surgical repair of a secundum ASD is usually quite successful, but the procedure is associated with some morbidity and mortality related to the use of general anesthesia, a sternotomy or thoracotomy, cardiopulmonary bypass, and an atriotomy incision. In 518 patients undergoing repair of a secundum ASD between 1976 and 1983, Kirklin reported a 1% mortality rate, a 1.5% rate of reoperation for postoperative bleeding, and a 2% rate of reoperation for a persistent or recurrent ASD.[83] Additional surgical morbidity may relate to embolic stroke (early or late), supraventricular tachyarrhythmias and/or sick sinus syndrome, and the postpericardiotomy syndrome.[84-87] Thus, although ASD repair is one of the most effective open heart operations, there appears to be justification for pursuing nonsurgical techniques of ASD closure.

In 1976 Mills and King[88] reported the first transcatheter closure of a secundum ASD in humans using a double-umbrella device. Transvenous umbrella ASD closure was performed at cardiac catheterization in five adolescents and adults, and completely relieved the left-to-right shunt in four patients. In the fifth patient, known to have three defects, the left-to-right shunt was diminished. Persistent closure of the ASD was documented at a follow-up cardiac catheterization in two patients. In no instance was evidence of significant arrhythmia, hemolysis, or endocarditis found. Rashkind, paralleling the work of Mills and King, also developed a transcatheter closure device for nonsurgical treatment of patients with a secundum ASD.[89,90] During the past decade this device has evolved from a dumbbell shaped double-disk prosthesis to the current single-disk, hook fastened occluder. The most recent device consists of six stainless steel ribs, three of which terminate with a small barbed hook, which support a disk of polyurethane foam material. There have also been refinements in the delivery system, including an improved release mechanism and centering arms to increase the likelihood that the occluder device will be properly seated on the ASD. Based on Rashkind's data in animal and human experiments, the Food and Drug Administration (FDA) has recently approved the Rashkind Occluder (USCI, Tewksbury, MA) for clinical trials of transcatheter ASD closure. These trials are currently underway in several pediatric centers.

Technique

The procedure is performed in the cardiac catheterization lab without general anesthesia. Once the catheters are inserted the patient is heparinized (100 units/kg, maximum dose 3,000 units). A complete right and left heart catheterization is performed to document the presence and size of the secundum ASD, and to exclude possible associated defects including pulmonary stenosis and pulmonary hypertension. A left atrial cineangiogram is filmed in a 30-degree left

anterior oblique position to confirm the ASD size and location. To meet criteria for transcatheter closure, a patient must have an isolated secundum ASD, ≤18 mm in diameter, without associated lesions requiring surgical repair or causing elevated right ventricular or right atrial pressure. Informed consent is obtained to perform the ASD closure as an investigational procedure, and we proceed only with surgical backup available.

The Rashkind ASD Occluder is currently manufactured in three sizes: 25 mm, 30 mm, and 35 mm in diameter. We prefer to use an occluder whose diameter is approximately twice the largest diameter of the ASD. The delivery system, with a collapsed prosthesis within its pod, is inserted into a 16 French delivery sheath that has been introduced percutaneously into the right femoral vein and whose tip is positioned in the left atrium. The delivery catheter is then advanced across the ASD to the tip of the venous sheath. We use biplane fluoroscopy and echocardiography to assure that the tip of the delivery system is positioned in the center of the left atrium. The venous sheath and pod are then slowly retracted and the prosthesis is advanced slightly until it exits the pod and springs open within the left atrium. The delivery catheter is further retracted to allow the centering arms to open widely. Rapid traction on the delivery catheter allows the centering mechanism to guide the occluding prosthesis over the ASD. Firm traction is then exerted on the catheter to embed the anchoring hooks of the prosthesis firmly into the atrial septum. When all three hooks are properly seated, the occluder device is released from the catheter. The pod is advanced over the centering arms and the catheter is withdrawn into the venous sheath. The entire delivery system is then removed from the femoral vein, and an 8-10 French sheath is inserted. A repeat right heart catheterization is performed to detect and quantitate any residual shunt which may be present. A pulmonary arteriogram is filmed to visualize the presence or absence of an atrial shunt on the levophase. The patient is observed for 24 hours and is discharged home the second day following transcatheter ASD closure.

Results

Clinical experience with transcatheter closure of secundum ASD is limited. Rashkind has published his experience with the single-disc occluder in 23 children and adults with a small to moderate secundum ASD.[89,90] This group included children as young as 4 years of age. Satisfactory closure was achieved in 14 children (61%), but in nine the closure was considered unsatisfactory. Six of these nine children underwent surgical ASD repair, four emergently. Surgical retrieval of the device and ASD closure were reportedly uncomplicated.

At the University of Michigan, we have attempted transcatheter ASD closure with a 25 mm Rashkind Occluder in three children since August, 1987. The children ranged in age from 3 to 8 years, and in weight from 11.1 to 22 kilograms. Each had evidence of significant right ventricular volume overload, with an isolated secundum ASD ranging in size from 7 to 13 mm in diameter. Satisfactory closure has been obtained in one child, an 8-year-old boy with a 13 mm ASD (Fig 14). In this child the procedure took approximately 30 minutes and was uncomplicated. The pulmonary to systemic flow ratio decreased from 1.8 to 1.1, with angiography documenting a tiny residual shunt at the margin of the occluder. The child's physical exam normalized, right ventricular dilatation and paradoxical septal motion on echocardiogram resolved immediately, and diminished heart size and pulmonary vascularity were evident on chest x-ray (Fig 15). The child was discharged home 24 hours after closure and has done very well during 6 months of follow-up.

The remaining two children required surgical repair after transcatheter ASD closure was unsuccessful. In our first attempt, a 3-year-old with a 13 mm ASD, the occluding device was not properly seated on the ASD. The patient underwent uncomplicated surgical repair and retrieval of the device from the lateral right atrial wall. In the third subject, a 6-year-old with a 7 mm ASD, difficulty was encountered seating the device on the atrial septum. It appeared that one anchoring hook did not attach properly and may have been pulled through the ASD to the right atrial side of the septum. Because the ASD was in large part occluded, and the pulmonary to systemic flow ratio decreased from 1.6 to 1.1, the child was observed with serial exams and echocardiograms. Two days later a second anchoring hook appeared to detach from the atrial septum, and

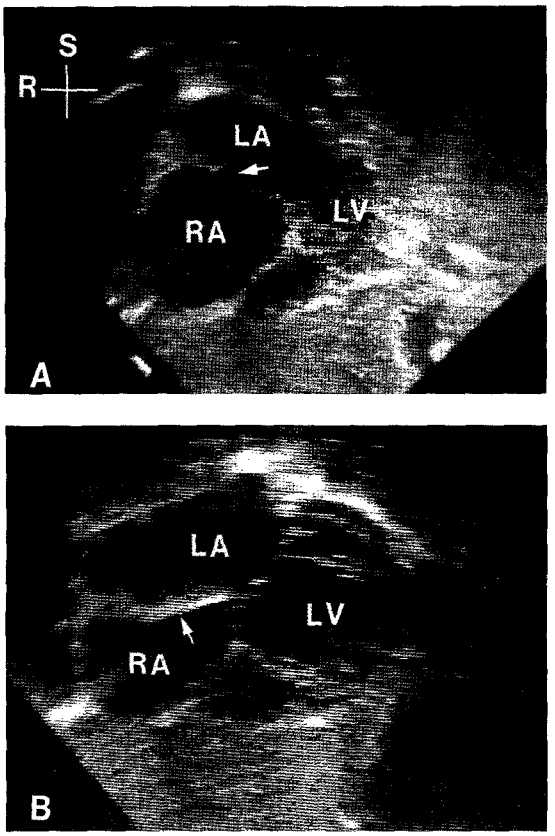

Fig 14. Two-dimensional echocardiogram (four chamber view) in an 8-year-old boy with a moderate secundum atrial septal defect before (A) and after (B) transcatheter closure of the defect. Before closure a centrally located 13 mm defect is present (upper arrow). After transcatheter closure the 25 mm Rashkind device is seen occluding the defect. A very small residual shunt was detected by Doppler at the right margin of the device, but was no longer present 6 months later.

the device rotated into the left atrium above the mitral valve. The child was taken immediately to surgery where the device was removed, the mitral valve inspected, and the ASD repaired. He is currently doing well with clinical evidence of mild mitral regurgitation.

Conclusions

Transcatheter ASD closure is clearly an experimental procedure. Results to date have been mixed, with approximately half of all attempted closures being successful. Although the case can be made that transcatheter ASD closure followed by surgery if necessary is a reasonable treatment strategy, since half of the children may be spared surgery, we feel that the high failure rate can be reduced. Revisions in the delivery system to orient the device more parallel to the atrial septum, and revisions in the Rashkind Occluder itself to assure better attachment to the septum are being actively explored. Such refinements should, in the near future, improve the safety and efficacy of transcatheter ASD closure.

PATENT DUCTUS ARTERIOSUS

Surgical division of a PDA is a safe and highly successful procedure. It does, however, require the use of general anesthesia and a thoracotomy incision, and carries a low risk of perioperative complications such as bleeding, injury to the recurrent laryngeal nerve, injury to the left phrenic nerve, and chylothorax.[91] Further, the two largest published surgical series have together reported a 1.5% incidence of recurrent postoperative ductal patency.[92,93]

In 1967, Porstmann et al described a nonsurgical technique for closure of a PDA in the cardiac catheterization laboratory.[94] Subsequently, they reported a 5-year experience with the procedure in 62 children and adults.[95] Based on the angiographic appearance of the PDA, a plug, made of Ivalon foam, was tailored to an appropriate size and shape. A 3-meter long guidewire was then inserted into the femoral artery, advanced across the PDA into the right heart, retrieved and withdrawn from the femoral vein to create an arteriotransductal-venous loop. The Ivalon plug was then advanced transarterially over the wire and packed into the PDA. When ductal closure was accomplished, the guidewire was removed transvenously in order to avoid dislodging the plug. Porstmann reported complete ductal closure in 56 of 62 patients, with no evidence of recurrent patency or plug migration during follow-up of up to 4 years. In seven patients the procedure resulted in stenosis or occlusion of the external iliac artery, and in three vascular surgery was necessary. No mortality was reported.

In 1976, Rashkind developed a transcatheter technique for PDA closure based on a single disk occluding device similar to that described above for ASD closure.[96] Through animal and clinical trials, the prosthesis has been refined and currently consists of a hookless double-disk device.[97] The two opposing disks allow the device to become lodged with the ductus without the use of anchoring hooks, and therefore the device can be withdrawn back into its delivery pod if necessary.

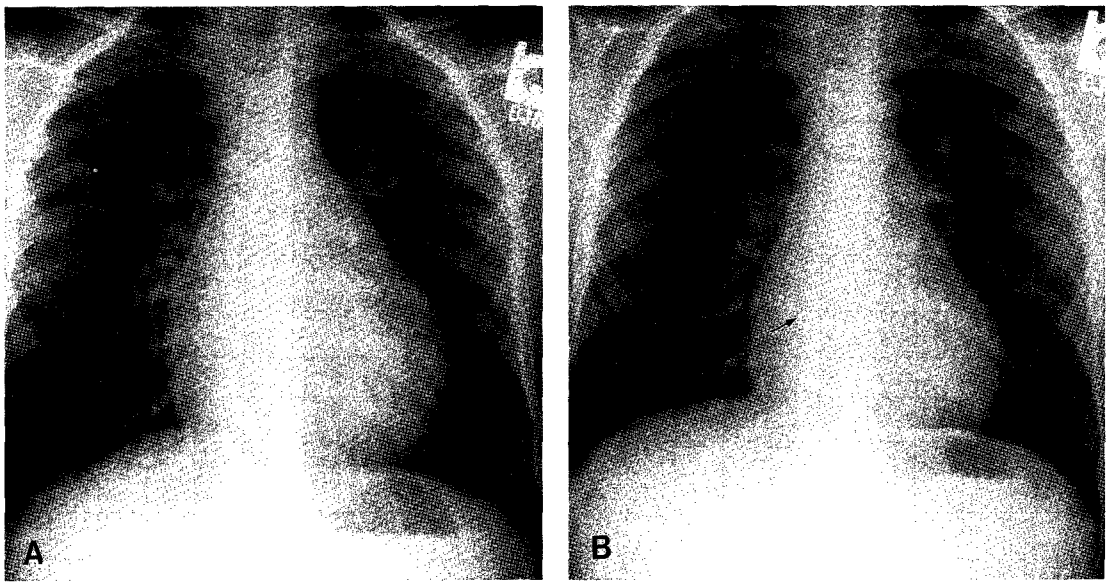

Fig 15. (A,B) Chest x-ray before (A) and one day after (B) transcatheter closure of a moderate secundum ASD in an 8-year-old boy (see Fig 14). After ASD closure the heart size returned to normal and the pulmonary vascular markings were diminished. The Rashkind Occluder device is evident to the right of the spine (B, black arrow).

Clinical trials of the double-disk occluder have subsequently been conducted since 1981 at several pediatric centers.

Technique

Although initially designed for transarterial delivery, recent trials have used a transvenous approach for delivery of the Rashkind PDA occluder. Bash and Mullins[98] have described a modification of the technique that uses a long venous sheath to guide the device to the ductus. The procedure has been described in detail elsewhere[97,98] and will only be briefly summarized here. A 12 mm or 17 mm diameter double-disk PDA occluding device is used depending on the size of the ductus (the larger device for a PDA >5 mm in diameter). After a complete hemodynamic study, PDA size and location are carefully assessed using biplane angiography. An 8 French balloon-tipped catheter is advanced from the femoral vein, through the right heart, across the PDA to the descending aorta. A long 8 or 11 French Mullins sheath is advanced over the catheter, which is then withdrawn leaving the sheath in place. The delivery catheter, with the PDA occluder loaded in its pod, is advanced through the sheath to the level of the tricuspid valve. The occluder is then pushed out of the pod and advanced to the tip of the sheath (which serves as a functional extension of the pod). With the tip of the sheath positioned at the aortic end of the PDA, the occluder device is further advanced until the distal disk opens in the aorta. The sheath, delivery catheter and occluder device are then withdrawn until resistance is felt, and the distal disk is observed to flex in the aortic end of the ductus. The sheath is then retracted to allow the proximal disk to open in the pulmonary artery end of the ductus. Once the device has been properly seated, it is released from the delivery catheter and the entire delivery system is removed. A aortogram is performed to document PDA occlusion.

Results

Transcatheter PDA occlusion has not yet been performed at the University of Michigan. A recent multicenter trial, however, has reported encouraging results in 146 patients treated since 1976.[97] The patients ranged in age from 1 month to 42 years, and in weight from 2.4 to 100 kg. Overall, transcatheter PDA occlusion was successful in 96 patients (66%). Since 1984 when device and delivery modifications (including the Mullins transvenous approach) were adopted, however, the procedure has been successful in 64 of 79 attempted cases (81%). In the remaining 15 patients the occluder device either could not be

delivered properly (n = 3), embolized to a pulmonary artery or the descending aorta (n = 7), or resulted in an incomplete closure (n = 5). These patients either underwent elective surgical repair or have chosen to wait for a repeat attempt at transcatheter closure.

Complications. Complications of transcatheter PDA occlusion have primarily involved embolization of the occluder device or incomplete PDA closure. In the published trials,[97] occluder embolization has generally occurred to a pulmonary artery although embolization to the descending aorta has also been described. In the majority of these cases, the embolized occluder device has been retrieved uneventfully with a catheter. In one patient, a device became entangled in the tricuspid valve apparatus and had to be surgically removed. There were no deaths reported during these trials.

Conclusions

Transcatheter PDA occlusion using the Rashkind occluder device appears to promise a safe and effective nonsurgical method of treating many children with a PDA. In the most recent trials the technique has resulted in an 81% rate of complete closure, and the incidence of embolization has diminished. It is of interest that Rashkind et al[97] concluded that "the greatest complication of the nonsurgical technique is that the patient could require subsequent standard operative repair" (p 591). Currently, the procedure remains investigational pending approval by the FDA.

AORTOPULMONARY COLLATERAL VESSELS AND SURGICAL SHUNTS

Aortopulmonary arterial communications often occur as collateral vessels in association with other congenital anomalies, such as tetralogy of Fallot or pulmonary atresia with ventricular septal defect, or as the result of a surgical shunt procedure. In either case these vessels typically arise from the descending aorta or the brachiocephalic arteries. Their hemodynamic importance is quite variable; however, those that warrant occlusion are often difficult to manage surgically. As an alternative to surgical ligation, these aortopulmonary collateral vessels can often be occluded using transcatheter embolization techniques. A wide variety of thrombogenic materials and occlusion devices have been used to occlude such vessels including: bucrylate adhesive,[99] Gelfoam or Ivalon particles,[100,101] detachable silicone balloons,[102-104] steel Gianturco coils (Cook: Bloomington, IN),[105-108] and Rashkind umbrella devices.[109] In our experience, most such vessels can be successfully occluded with Gianturco coils. A large pediatric experience has been reported by Radtke et al,[110] wwo attempted coil embolization of 79 vessels in 50 patients. Total occlusion was achieved in 60/79 (79%) vessels, subtotal occlusion in 17/79 (22%) vessels, and no occlusion in only 1/79 vessels. Inadvertent coil embolization occurred in 4/79 procedures.

Technique

We prefer to occlude aortopulmonary collateral vessels and surgical Blalock-Taussig shunts by transcatheter embolization using Gianturco coils. After vascular access is obtained by percutaneous entry into a femoral artery, the collateral vessel is selectively catheterized usually with a 7 French balloon-tipped end-hole catheter. The precise size and location of the vessel is determined by selective angiography. Multiple angled views are obtained to optimally profile the vessel. We then inflate the balloon and occlude flow in order to answer the following questions: (1) Does the vessel dilate when it is obstructed? Most collaterals are compliant to varying degrees and a vessel that measures 3 to 4 mm on the initial angiogram may dilate to 5 to 6 mm when occluded. It is the diameter of the vessel during balloon occlusion that is used to determine the size of Gianturco coil to be used. (2) Are the patient's hemodynamics altered by occlusion of the vessel? When a collateral vessel or shunt is to be occluded in a patient with cyanotic heart disease one must be certain that the occlusion will not produce excessive hypoxemia. (3) Is this vessel the sole vascular supply to a given segment of the lung? We usually do not embolize a vessel if it is the only vascular supply to a portion of the lung, since pulmonary infarction may occur.

Once it is determined that the vessel can be safely occluded, we chose a coil whose diameter is 40% to 50% larger than the vessel diameter measured during balloon occlusion. A coil 0.025 or 0.038 inch in diameter is used depending on the size of the child. The straightened coil is

advanced through the end-hole catheter using a flexible guidewire, and emerges in the collateral vessel as a coil 3 to 15 mm in diameter. Extremely flexible guidewires are used in order to avoid straightening and displacement of the catheter tip during coil placement. Whenever possible we also inflate the balloon when delivering the coil through a wedge catheter. Balloon inflation helps to stabilize the catheter during coil placement, and also decreases the risk of coil migration to the distal pulmonary artery by diminishing blood flow through the vessel. After coil embolization, vessel occlusion by thrombosis generally occurs within 10 to 15 minutes. During this period of time, a balloon may be inflated in the orifice of the vessel to slow flow and thus to promote thrombus formation at the coil. If complete occlusion is not apparent on a repeat angiogram 10 to 15 minutes after the initial coil placement, then another coil (or coils) is inserted to further obstruct the vessel. We have found that as many as six or seven Gianturco coils may be necessary to completely occlude a large aortopulmonary collateral.

Results

We have attempted coil embolization of 12 vessels in seven children. These children have ranged in age from 6 months to 19.5 years. The purpose of embolization was to occlude an aortopulmonary collateral vessel before definitive surgical repair of pulmonary atresia with a ventricular septal defect (n = 3), to occlude a patent Blalock-Taussig shunt (n = 2; one classic and one modified shunt), to occlude large aortopulmonary collateral vessels causing heart failure in an infant with pulmonary atresia and a ventricular septal defect (n = 1), and to occlude an aortopulmonary collateral causing hemoptysis in a young adult with cyanotic congenital heart disease (n = 1).

A total of 31 Gianturco coils were used to successfully occlude 11 of the 12 vessels (Fig 16). One collateral vessel that remained partially

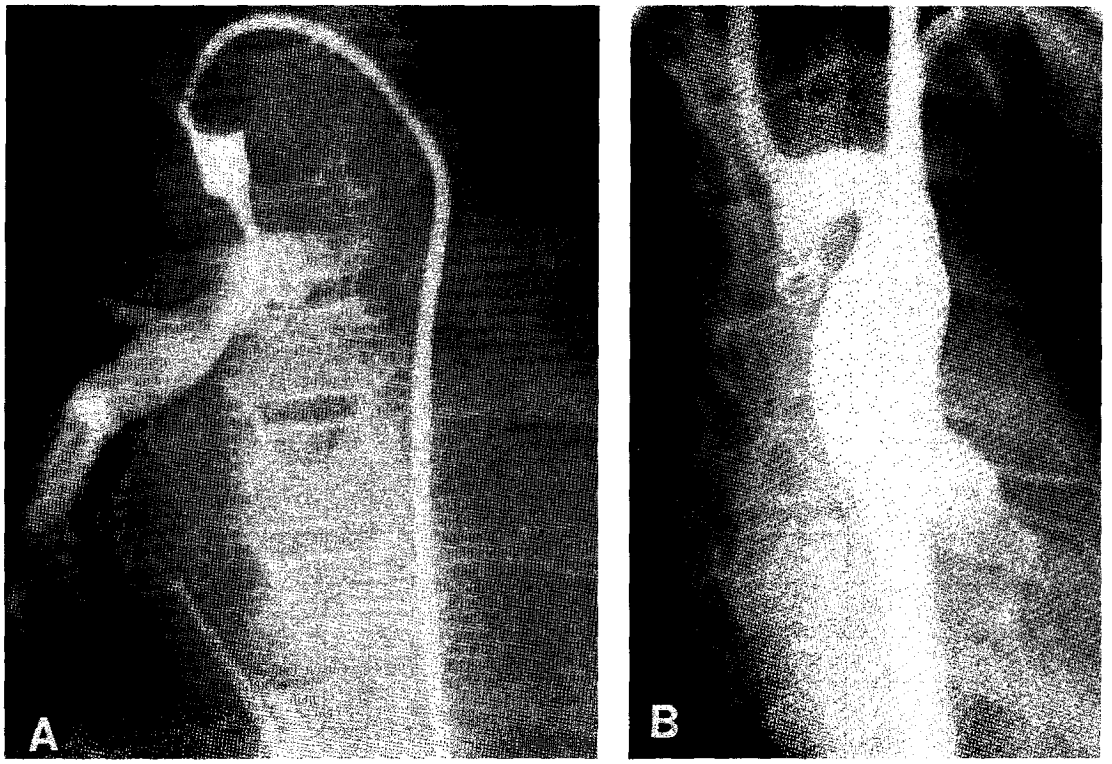

Fig 16. (A) Selective arteriogram performed in a classic right Blalock-Taussig shunt demonstrating shunt patency, and a stenosis at its insertion into the pulmonary artery. (B) Aortogram performed 15 minutes after coil embolization of the Blalock-Taussig shunt documents successful shunt occlusion.

patent at the end of the initial study was found to be completely occluded at a follow-up catheterization 3 months later. Despite two attempts, we were unable to successfully embolize a 5 mm modified left Blalock-Taussig shunt in a 21-month-old child. During the first attempt the coil failed to lodge in the shunt and embolized to a distal pulmonary artery where it was retrieved. A second coil was then delivered while a large balloon was inflated in the left pulmonary artery at the insertion of the shunt in order to temporarily occlude shunt flow. Immediately upon deflation of the balloon, however, the coil again embolized to the distal left pulmonary artery. It was retrieved uneventfully at the time of surgical shunt ligation.

Complications. The only complications that we have encountered have been inadvertent embolization of a coil to either a systemic artery or peripheral pulmonary artery. In three cases a coil embolized to an iliac or femoral artery, and in all three it was successfully retrieved using a 5 French bioptome catheter. Twice in one child, described above, a coil embolized to the left pulmonary artery and was retrieved by catheter on one occasion, and then in the operating room when it became clear that surgical shunt ligation was necessary.

Conclusions

Transcatheter embolization provides a safe and effective nonsurgical method of closing aortopulmonary collateral vessels and surgical shunts. We have found that it is particularly useful in dealing with aortopulmonary collaterals (preoperatively or postoperatively) in children with tetralogy of Fallot with or without pulmonary atresia. In these patients, embolization of collateral vessels is an effective method of controlling pulmonary blood flow or treating hemoptysis in selected cases. Coil embolization also provides a nonsurgical method of occluding a Blalock-Taussig shunt when indicated, although the technique may not succeed in some children with a modified Goretex shunt.

REFERENCES

1. Semb BHK, Tjönneland S, Stake G, et al: "Balloon valvuotome" of congenital pulmonary valve stenosis with tricuspid valve insufficiency. Cardiovasc Radiol 2:239-241, 1979
2. Kan JS, White RI, Mitchell SE, et al: Percutaneous balloon valvuloplasty: A new method for treatment of congenital pulmonary-valve stenosis. N Engl J Med 307:540-542, 1982
3. Rocchini AP, Kveselis DA, Crowley D, et al: Percutaneous balloon valvuloplasty for treatment of congenital pulmonary valvular stenosis in children. J Am Coll Cardiol 3: 1005-1012, 1984
4. Lababidi Z, Wu Jr: Percutaneous balloon pulmonary valvuloplasty. Am J Cardiol 52:560-562, 1983
5. Rees PG, Bull C, Deanfield JE: Percutaneous balloon valvuloplasty for pulmonary valve stenosis in infants and children. Br Heart J 54:435-441, 1985
6. Pepine CJ, Gessner IH, Feldman RL: Percutaneous balloon valvuloplasty for pulmonary valve stenosis in the adult. Am J Cardiol 50:1442-1445, 1982
7. Shuck JW, McCormick DJ, Cohen IS, et al: Percutaneous balloon valvuloplasty of the pulmonary valve: role of right to left shunting through a patent foramen ovale. J Am Coll Cardiol 4:132-135, 1984
8. Brodsky SJ: Percutaneous balloon angioplasty: Treatment for congenital valvular pulmonic stenosis. Am J Dis Child 138:851-854, 1984
9. Rao PS, Mardini MK: Pulmonary valvotomy without thoracotomy: The experience with percutaneous balloon pulmonary valvuloplasty. Ann Saudi Med 5:149-154, 1985
10. Miller CAH: Balloon valvuloplasty and angioplasty in congenital heart disease. Br Heart J 54:285-289, 1985
11. Tynan M, Baker EJ, Rohmor J, et al: Percutaneous balloon pulmonary valvuloplasty. Br Heart J 53:520-524, 1985
12. Zeevi B, Keene JF, Fellows K, et al: Balloon dilation of critical pulmonary stenosis in the first week of life. Circulation 76:IV-265, 1987 (Suppl IV)
13. Jeffery RF, Moller JH, Amplatz K: The dysplastic pulmonary valve: A new roentgenographic entity. Am J Roentgenol Ther Radium Nucl Med 114:322-339, 1972
14. Koretzky ED, Moller JH, Korns ME, et al: Congenital pulmonary stenosis resulting from dysplasia of the valve. Circulation 60:43-53, 1969
15. DiSessa TG, Alpert BS, Chase NA, et al: Balloon valvuloplasty in children with dysplastic pulmonary valves. Am J Cardiol 60:405-407, 1987
16. Rey C, Marache P, Francart C, et al: Percutaneous transluminal balloon valvuloplasty of congenital pulmonary valve stenosis, with a special report on infants and neonates. J Am Coll Cardiol 11:815-820, 1988
17. Ring JC, Kulek TJ, Burke BA, et al: Morphologic changes induced by dilation of the pulmonary valve anulus with overlarge balloons in normal newborn lambs. Am J Cardiol 55:210-214, 1985
18. Radtke W, Keane JF, Fellows KE, et al: Percutaneous balloon valvotomy of congenital pulmonary stenosis using oversized balloons. J Am Coll Cardiol 8:909-915, 1986
19. Walls JT, Lababidi Z, Curtis JJ: Morphologic effects

of percutaneous balloon pulmonary valvuloplasty. South Med J 80:475-477, 1987

20. Rao PS: Influence of balloon size on short-term and long-term results of balloon pulmonary valvuloplasty. Tex Heart Inst J 14:57-61, 1987

21. Rocchini AP, Beekman RH: Balloon angioplasty in the treatment of pulmonary valve stenosis and coarctation of the aorta. Tex Heart Inst J 13:377-385, 1986

22. Kveselis DA, Rocchini AP, Snider AR, et al: Results of balloon valvuloplasty in the treatment of congenital valvar pulmonary stenosis in children. Am J Cardiol 56:527-532, 1985

23. Kan JS, White RI Jr, Mitchell E, et al: Percutaneous transluminal balloon valvuloplasty for pulmonary valve stenosis. Circulation 69:554-560, 1984

24. Ben-Shachar G, Cohen MH, Sivakoff MC, et al: Development of infundibular obstruction after percutaneous pulmonary balloon valvuloplasty. J Am Coll CArdiol 5: 754-756, 1985

25. Orell SR, Kaineil J, Wahlgren F: Malformation and multiple stenosis of the pulmonary arteries with pulmonary hypertension. Acta Radiol 54:449-520, 1960

26. Blackstone EH, Kirklin JW, Bertranou EG, et al: Preoperative prediction from cineangiograms of post repair right ventricular pressure in tetralogy of Fallot. J Thorac Cardiovasc Surg 73:542-553, 1979

27. Wilson JM, Mack JW, Turley K, et al: Persistent stenosis and deformity of the right pulmonary artery after correction of the Waterston anastomosis. J Thorac Cardiovas Surg 82:169-175, 1981

28. Hastreeiter AR, Joorabchi B, Pujatti G, et al: Cardiovascular lesions associated with congenital rubella. J Pediatr 71:59-65, 1967

29. Beuren AJ, Schulze C, Eberle P, et al: The syndrome of supravalvular aortic stenosis, peripheral pulmonary stenosis, mental retardation and similar facial appearance. Am J Cardiol 13:471-475, 1964

30. Greenwood RD, Rosenthal A, Crocker AAC: Syndrome of intra-hepatic biliary dysgenesis and cardiovascular malformations. Pediatrics 58:243-247, 1976

31. Gill CC, Moodie DS, McGoon DC: Staged surgical management of pulmonary atresia with diminutive pulmonary arteries. J Thorac Cardiovas Surg 73:436-448, 1977

32. Haworth SG, Rees PG, Taylor JRN, et al: Pulmonary atresia with ventricular septal defect and major aortopulmonary collateral arteries: effect of systemic pulmonary anastomosis. Br Heart J 45:133-142, 1981

33. Martin EC, Diamond NG, Casarella WJ: Percutaneous transluminal angioplasty in non-atherosclerotic disease. Radiology 135:27-33, 1980

34. Lock JE, Niemi T, Einzig S, et al: Transvenous angioplasty of hypoplastic and stenotic pulmonary arteries in newborn lambs. Circulation 64:886-893, 1981

35. Edwards BS, Lucas RV Jr, Lock JE, et al: Morphologic changes in the pulmonary arteries following percutaneous balloon angioplasty for pulmonary arterial stenosis. Circulation 74:135-143, 1986

36. Lock JE, Castaneda-Zuniga WR, Fuhrman BP, et al: Balloon dilatation angioplasty of hypoplastic and stenotic pulmonary arteries. Circulation 67:962-967, 1983

37. Rocchini AP, Kveselis D, Dick M, et al: Use of balloon angioplasty to treat peripheral pulmonary stenosis. Am J Cardiol 54:1069-1073, 1984

38. Kveselis D, Rocchini AP: Percutaneous transluminal angioplasty of peripheral pulmonary arterial stenosis, coarctation of the aorta, superior vena caval and pulmonary venous stenosis, and other great-artery stenosis. Semin Interventional Radiol 1:201-214, 1984

39. Ring JC, Bass JL, Marvin W, et al: Management of congenital stenosis of a branch pulmonary artery with balloon dilation angioplasty. Report of 52 procedures. J Thorac Cardiovasc Surg 90:35-44, 1985

40. Foker JE, Turley K, Lock JE, et al: Intraoperative balloon dilation of stenotic and hypoplastic pulmonary arteries. Circulation 68:213, 1983 (abstr, suppl II)

41. Fellows KE, Radtke W, Keane JF, et al: Acute complications of catheter therapy for congenital heart disease. Am J Cardiol 60:679-683, 1987

42. Hsieh K, Keane JF, Nadas AS, et al: Long-term follow-up of valvotomy before 1968 for congenital aortic stenosis. Am J Cardiol 58:338-341, 1986

43. Lababidi Z, Wu J, Walls JT: Percutaneous balloon aortic valvuloplasty: Results in 23 patients. Am J Cardiol 53:194-197, 1984

44. Walls JT, Lababidi Z, Curtis JJ, et al: Assessment of percutaneous balloon pulmonary and aortic valvuloplasty. J Thorac Cardiovasc Surg 88:352-356, 1984

45. Choy M, Beekman RH, Rocchini AP, et al: Percutaneous balloon valvuloplasty for valvar aortic stenosis in infants and children. Am J Cardiol 59:1010-1013, 1987

46. Helgason H, Keane JF, Fellows KE, et al: Balloon dilation of the aortic valve: studies in normal lambs and in children with aortic stenosis. J Am Coll Cardiol 9:816-822, 1987

47. Mullins CE, Nihill MR, Vick GW, et al: Double balloon technique for dilation of valvular or vessel stenosis in congenital and acquired heart disease. J Am Coll Cardiol 10:107-114, 1987

48. Rupprath G, Neuhaus KL: Percutaneous balloon aortic valvuloplasty in infancy and childhood. Am J Cardiol 55:1855-1856, 1985

49. Waller BF, Girod DA, Dillon JC: Transverse aortic wall tears in infants after balloon angioplasty for aortic valve stenosis: Relation of aortic wall damage to diameter of inflated angioplasty balloon and aortic lumen in seven necropsy cases. J Am Coll Cardiol 4:1235-1241, 1984

50. McKay RG, Safian RD, Lock JE, et al: Balloon dilatation of calcific aortic stenosis in elderly patients: Postmortem, intraoperative, and percutaneous valvuloplasty studies. Circulation 74:119-125, 1986

51. Beekman RH, Rocchini AP, Crowley DC, et al: Comparison of single and double balloon valvuloplasty in children with aortic stenosis. J Am Coll Cardiol 12:480-485, 1988

52. Jones M, Barnhart GR, Morrow AG: Late results after operations for left ventricular outflow tract obstruction. Am J Cardiol 50:569-579, 1982

53. Conkle DM, Jones M, Morrow AG: Treatment of congenital aortic stenosis. Arch Surg 107:649-651, 1973

54. Jack WD, Kelly DT: Long-term follow-up of valvulot-

omy for congenital aortic stenosis. Am J Cardiol 38:231-234, 1976

55. Wagner HR, Ellison RC, Keane JF, et al: Clinical course in aortic stenosis. Circulation 56:147-157, 1977 (suppl I)

56. Patel R, Sinigh SP, Abrams L, et al: Coarctation of aorta with special reference to infants. Br Heart J 39: 1246-1253, 1977

57. Khoury GH, Hawes CR: Recurrent coarctation of the aorta in infancy and childhood. J Pediatrics 72:801-806, 1968

58. Eshaghpour E, Olley PM: Recoarctation of the aorta following coarctectomy in the first year of life: A follow-up study. J Pediatrics 80:809-814, 1972

59. Beekman RH, Rocchini AP, Behrendt DM, et al: Long-term outcome after repair of coarctation in infancy: Subclavian angioplasty does not reduce the need for reoperation. J Am Coll Cardiol 8:1406-1411, 1986

60. Beekman RH, Rocchini AP, Behrendt D, et al: Reoperation for coarctation of the aorta. Am J Cardiol 48: 1108-1114, 1981

61. Bromberg BI, Beekman RH, Bank ER, et al: Aneurysms following prosthetic patch repair of coarctation: An evaluation of prevalence and screening techniques. Circulation 74:467, 1986 (abstr)

62. Sos T, Sniderman KW, Rettek-Sos B, et al: Percutaneous transluminal dilatation of coarctation of thoracic aorta post mortem. Lancet 2:970-971, 1979

63. Beekman RH, Rocchini AP, Dick M, et al: Percutaneous balloon angioplasty for native coarctation of the aorta. J Am Coll Cardiol 10:1078-1084, 1987

64. Morrow WR, Vick GW, Nihill MR, et al: Balloon dilation of unoperated coarctation of the aorta: Short and intermediate-term results. J Am Coll Cardiol 11:133-138, 1988

65. Lababidi ZA, Daskalopoulos DA, Stoeckle H: Transluminal balloon coarctation angioplasty: Experience with 27 patients. Am J Cardiol 54:1288-1291, 1984

66. Lock JE, Bass JL, Amplatz K, et al: Balloon dilation angioplasty of aortic coarctations in infants and children. Circulation 68:109-116, 1983

67. Singer MI, Rowen M, Dorsey TJ: Transluminal aortic balloon angioplasty for coarctation of the aorta in the newborn. Am Heart J 103:131-132, 1982

68. Sperling DR, Dorsey TJ, Rowen M, et al: Percutaneous transluminal angioplasty of congenital coarctation of the aorta. Am J Cardiol 51:562-564, 1983

69. Finley JP, Beaulieu RG, Nanton MA, et al: Balloon catheter dilatation of coarctation of the aorta in young infants. Br Heart J 50:411-415, 1983

70. Cooper RS, Ritter SB, Golinko RJ: Balloon dilatation angioplasty: Nonsurgical management of coarctation of the aorta. Circulation 70:903-907, 1984

71. Allen HD, Marx GR, Ovitt TW, et al: Balloon dilation angioplasty for coarctation of the aorta. Am J Cardiol 57:828-832, 1986

72. Marvin WJ, Mahoney LT, Rose EF: Pathologic sequelae of balloon dilation angioplasty for unoperated coarctation of the aorta in children. J Am Coll Cardiol 7:117A, 1986 (abstr)

73. Kan JS, White RI, Mitchell SE, et al: Treatment of restenosis of coarctation by percutaneous transluminal angioplasty. Circulation 68:1087-1094, 1983

74. Saul JP, Keane JF, Fellows KE, et al: Balloon dilation angioplasty of postoperative aortic obstructions. Am J Cardiol 59:943-948, 1987

75. Soulen RL, Kan J, Mitchell S, et al: Evaluation of balloon angioplasty of coarctation restenosis by magnetic resonance imaging. Am J Cardiol 60:343-345, 1987

76. Moore JW, Pearson CE, Lee DH, et al: Dual-balloon angioplasty of recoarctation of the aorta. Tex Heart Inst J 14:102-105, 1987

77. Choy M, Rocchini AP, Beekman RH, et al: Paradoxical hypertension after repair of coarctation of the aorta in children: balloon angioplasty versus surgical repair. Circulation 75:1186-1191, 1987

78. Lock JE, Keane JF, Fellows KE: The use of catheter intervention procedures for congenital heart disease. J Am Coll Cardiol 7:1420-1423, 1986

79. Inoue K, Owaki T, Nakamura T, et al: Clinical application of transvenous mitral commissurotomy by a new balloon catheter. J Thorac Cardiovasc Surg 87:394-402, 1984

80. Kveselis DA, Rocchini AP, Beekman R, et al: Balloon angioplasty for congenital and rheumatic mitral stenosis. Am J Cardiol 57:348-350, 1986

81. Lock JE, Khalilullah M, Shrivastava S, et al: Percutaneous catheter commissurotomy in rheumatic mitral stenosis. N Engl J Med 313:1515-1518, 1985

82. Palacios I, Block PC, Brandi S, et al: Percutaneous balloon valvotomy for patients with severe mitral stenosis. Circulation 75:778-784, 1987

83. Kirklin JW, Barratt-Boyes BG: Atrial septal defect and partial anomalous pulmonary venous connection, in: Cardiac Surgery, Morphology, Diagnostic Criteria, Natural History, Techniques, Results and Indications. New York, Wiley, 1986, pp 463-497

84. Hawe A, Tastelli GC, Brandenburg RO, et al: Embolic complications following repair of atrial septal defects. Circulation 39,405 I-185, 1969 (Suppl I)

85. Morriss JH, McNamara DG: Residua, sequelae, and complications of surgery for congenital heart disease, in Rosenthal A, et al (eds): Postoperative Congenital Heart Disease. Philadelphia, Saunders, 1975, pp 3-27

86. Sealy WC, Farmer JC, Young WG, et al: Atrial dysrhythmia and atrial secundum defects. J Thorac Cardiovasc Surg 57:245-250, 1969

87. Sasaki R, Theilen EO, January LE, et al: Cardiac arrhythmias as associated with the repair of atrial and ventricular septal defects. Circulation 18:909-914, 1958

88. Mills NL, King TD: Non-operative closure of left-to-right shunts. J Thorac Cardiovasc Surg 72:371-378, 1976

89. Rashkind WJ: Interventional cardiac catheterization in congenital heart disease. Int J Cardiol 7:1-11, 1985

90. Rashkind WJ: Transcatheter treatment of congenital heart disease. Circulation 67:711-716, 1983

91. Kirklin JW, Barratt-Boyes BG: Patent ductus arteriosus, in Cardiac Surgery, Morphology, Diagnostic Criteria, Natural History, Techniques, Results and Indications. New York, Wiley, 1986, pp 679-697

92. Panagopoulos PH, Tatooles CJ, Aberdeen E, et al:

Patent ductus arteriosus in infants and children: A review of 936 operations. Thorax 26:137-144, 1971

93. Trippestad A, Efskind L: Patent ductus arteriosus. Surgical treatment of 686 patients. Scand J Thorac Cardiovasc Surg 6:38, 1972

94. Porstmann W, Wierny L, Warnke H: Closure of persistent ductus arteriosus without thoracotomy. German Med Monthly 12:259-261, 1967

95. Porstmann W, Wierny L, Warnke H, et al: Catheter closure of patent ductus arteriosus. 62 cases treated without thoracotomy. Radiol Clin North Am 9:203-218, 1971

96. Rashkind WJ, Cuaso CC: Transcatheter closure of a patent ductus arteriosus: Successful use in a 3.5 kg infant. Pediatr Cardiol 1:63, 1979

97. Rashkind WJ, Mullins CE, Hellenbrand WE, et al: Nonsurgical closure of patent ductus arteriosus: Clinical application of the Rashkind PDA Occluder System. Circulation 75:583-592, 1987

98. Bash SE, Mullins CE: Insertion of patent ductus occluder by transvenous approach: A new technique. Circulation 70:II-285, 1984 (Suppl II)

99. Zuberbuhler JR, Ankner E, Zoltun R, et al: Tissue adhesive closure of aortic-pulmonary communications. Am Heart J 88:41-46, 1974

100. Fellows KE, Khaw KT, Schuster S, et al: Bronchial artery embolization is cystic fibrosis: Technique and long term results. J Pediatr 95:959, 1979

101. Kaufman SL, Kan JS, Mitchell SE, et al: Embolization of systemic to pulmonary artery collaterals in the management of hemoptysis in pulmonary atresia. Am J Cardiol 58:1130-1132, 1986

102. Terry PB, White RI Jr, Barth KH, et al: Pulmonary arteriovenous malformations: Physiologic observations and results of therapeutic balloon embolization. N Engl J Med 308:1197-1200, 1983

103. Barth KH, White RI, Kaufman SL, et al: Embolization of pulmonary arteriovenous malformations with detachable balloons. Radiology 142:599, 1982

104. Grinnell VS, Mehringer CM, Hieshima GB, et al: Transaortic occlusion of collateral arteries to the lung by detachable valved balloons in a patient with tetralogy of Fallot. Circulation 65:1276-1278, 1982

105. Florentine M, Wolfe RR, White RI Jr: Balloon embolization to occlude a Blalock-Taussig shunt. J Am Coll Cardiol 3:200-202, 1984

106. Culham JAG, Izukawa T, Burns JE, et al: Embolization of a Blalock-Taussig shunt in a child AM J Roentgenol 137:413-415, 1981

107. Castaneda-Zuniga W, Epstein M, Zollikofer C, et al: Embolization of multiple pulmonary artery fistulas. Radiology 134:309-310, 1980

108. Fuhrman BP, Bass JL, Castaneda-Zungia W, et al: Coil embolization of congenital thoracic vascular anomalies on infants and children. Circulation 70:285-289, 1984

109. Lock JE, Cockerham JT, Keane JF, et al: Transcatheter umbrella closure of congenital heart defects. Circulation 75:593, 1987

110. Radtke W, Perry SB, Fellows KE, et al: Pre- and postoperative coil embolization of aortopulmonary and arteriovenous collaterals and shunts. J Am Coll Cardiol 11:28A (abstr)