Endovascular Treatment Strategies for Coarctation of the Aorta

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37.1 Introduction

Advances in the surgical treatment of coarctation of the aorta have increased life expectancy and reduced mortality [1]. Unfortunately, the average lifespan after repair remains only 35–50 years [2] and significant morbidity persists as a result of coronary artery disease, aneurysm formation, hypertension and stroke [3]. In addition, follow-up studies have revealed that restenosis rates of 30%, hypertension at rest and during exercise and compromised cardiac function can persist with only mild residual disease [4–6].

The invasive nature of surgical repair coupled with the shorter hospitalization, reduced pain and decreased cost associated with transcatheter therapies has led to balloon angioplasty and, most recently, stent implantation playing increasing roles in the treatment of aortic coarctation. As with the early surgical literature, transcatheter studies conducted over the last 2 decades have focused on the feasibility of treatment and on assessing the ability of these devices to reduce the gradient across a coarctation site. These studies have documented several important sequelae of transcatheter techniques, including aortic dissections and aneurysm formation [7–10]. This chapter will review the current indications, limitations and suggested guidelines associated with the use of balloon angioplasty and stent implantation for the treatment of coarctation of the aorta, and suggest a new paradigm aimed at optimizing transcatheter and surgical treatment strategies and reducing the associated long-term morbidity.

37.2 Endovascular Treatments for Coarctation of the Aorta

37.2.1 Balloon Angioplasty for Recurrent Coarctation

In a recent study of patients who underwent surgical correction for coarctation at a mean age of 10 years, the cumulative 40-year survival rate was 79%. Recoarctation was observed in 16% of these survivors [11]. Repeat surgical procedures are often more complicated than the original surgery and only moderately successful [12]. In one recent study examining the efficacy of repeat surgery for recurrent coarctation primarily in infants, 24% of the patients had residual pressure gradients of 30–48 mmHg an average of 2.5 years after the reoperation [12]. Findings such as these prompted Singer et al. [13] to attempt balloon angioplasty for the treatment of this group of patients and have made balloon angioplasty the current treatment of choice for most patients with recoarctation of the aorta [14, 15].

From a purely technical standpoint, the technique of balloon angioplasty for native or recurrent coarctation is one of the simplest of pediatric interventions. Access is obtained in the femoral artery and vein. Right heart catheterization is performed in the standard fashion, obtaining saturations and/or blood pressure measurements in the superior vena cava, right atrium, right

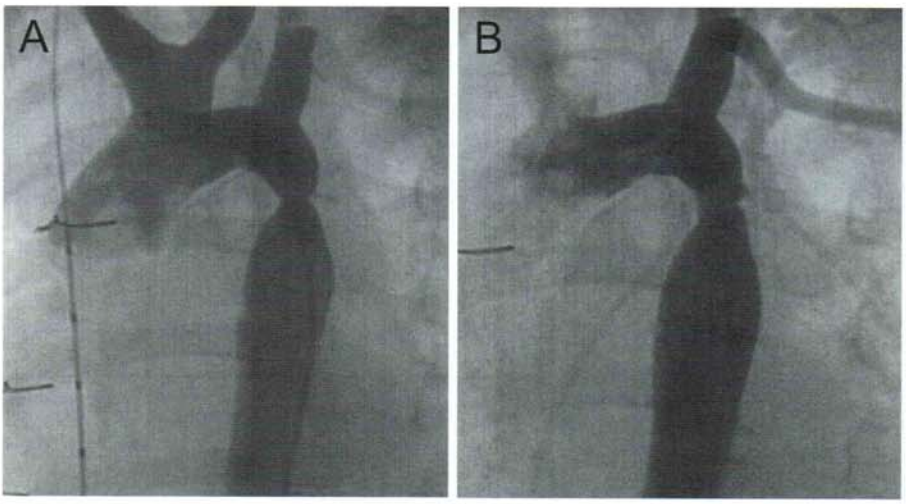

Fig. 37.1. Angiographic images of a coarctation before (**A**) and after (**B**) balloon angioplasty

ventricle and pulmonary arteries. In the absence of intracardiac shunts, the thermodilution method is used to measure the cardiac output, otherwise the Fick method, based on the mixed venous saturation, is employed.

Balloon dilation of the coarctation can be performed using antegrade or retrograde approaches, with the decision being a matter of clinician preference. In the antegrade approach, a trans-septal or Brockenbrough puncture is used to enter the left atrium, followed by catheter manipulation through the left ventricle and into the ascending aorta [10, 16]. Simultaneous ascending and descending aortic pressures or a pullback pressure measurement is made. The coarctation is then crossed and the balloon delivered from the femoral vein. This method decreases the size of the sheath required in the femoral artery and the likelihood of arterial injury.

Most clinicians prefer the retrograde technique owing to its simplicity, speed and potential complications associated with trans-septal puncture and subsequent catheter/balloon course [15, 17]. In mild coarctations, a pigtail catheter may be directed across the obstruction, while with severer stenoses, a diagnostic catheter and soft-tipped wire may be required to traverse the narrowest area. The catheter is manipulated to the right subclavian artery or the ascending aorta and a J-tipped wire is advanced through the catheter and left in place to guide the balloon. Simultaneous pressure measurements from the catheter and femoral arterial sheath provide the gradient across the coarctation. The chosen balloon is advanced over the wire to the level of the coarctation and then inflated (Fig. 37.1). The balloon is deflated and removed and angiography is performed to look for intimal tears, dissections or extravasation of contrast outside the vessel. Larger balloons or stents may be required if a significant gradient remains.

Balloon angioplasty for the treatment of recurrent coarctation has been accepted as the treatment of choice based on the assumption that scar tissue in the coarctation region is resistant to rupture or aneurysm formation [18]. Histological reports have revealed that the acute increase in lumen size and reduction in systolic pressure gradients resulting from balloon angioplasty are a result of tears in the aorta. These are generally confined to the intimal and medial layers, but in rare instances may be transmedial [19, 20]. The intimal and medial tears appear to heal partially or completely [19, 21], while the transmedial tears have been associated with aneurysms and, occasionally, aortic dissection [8, 9].

Mortality associated with balloon angioplasty for the treatment of recurrent coarctation varies with age and concurrent cardiac abnormalities [22]. In infants it is 2– 10% [16], with aneurysms occurring in 1% of cases [16]. In children and adults the mortality rates are approximately 2.5 and 1%, respectively [22, 23].

Balloon angioplasty has, in some instances, been unable to reduce the gradient in cases of recurrent coarctation [10, 16, 24]. The prevalence of residual pressure gradients greater than 20 mmHg has ranged from 11 to 26%. Severe preprocedure systolic pressure gradients and the presence of transverse arch hypoplasia were predictive of poor procedural outcomes in this study.

37.2.2 Balloon Angioplasty for Native Coarctation

Following the first documented treatment of balloon angioplasty for recurrent coarctation [13], this technique was applied to native coarctation in a critically ill neonate [25] and led to several clinical studies [26–28]. The

greatest limitation of balloon angioplasty for the treatment of native coarctation is the occurrence of aortic aneurysms [28]. Patients treated for coarctation by surgical correction or stent implantation are also susceptible to aneurysms [9, 29], but there is an increased susceptibility with balloon angioplasty of native coarctation that is particularly pronounced in infants. This susceptibility has prevented several institutions from applying this technology to this patient population [10, 30]. Not surprisingly, recoarctation after balloon angioplasty for native coarctation is also pronounced in neonates and infants (31–83%) [28, 31] as compared with children (11–36%) [14, 27] and adults (6–19%) [17, 32].

Balloon angioplasty has been unable to reduce pressure gradients in some cases of native coarctation, specifically those with isthmus hypoplasia [10, 26, 33]. In a study of patients with a mean age of approximately 5 years, half the patients with isthmus hypoplasia required additional procedures [26]. Use of this technology has also met with suboptimal results in cases of mild, long segment or tortuous coarctations [34, 35]. Symptomatic restenosis resulting from enduring ductal tissue, vascular recoil or neointimal hyperplasia in patients with smaller aortic diameters may contribute to residual narrowing and systolic pressure gradients greater than 20 mmHg following balloon angioplasty for native coarctation [23, 36].

Smaller peripheral arteries in neonates and infants are susceptible to femoral artery occlusion in 10–16% of patients [26–28]. The loss of femoral artery pulses is fairly common, but can be successfully treated by intravenous heparin or tissue plasminogen activator [27, 37]. To minimize these risks, some operators prefer to gain vascular access through the umbilical vessels [38].

Most institutions currently choose to treat neonates, infants and young children with severe aortic coarctation surgically. Others justify primary balloon angioplasty since the procedure does not preclude future surgery [14, 26, 39]. Angioplasty may also be chosen in cases of discrete coarctation when other catheter-based techniques are being used to treat concomitant cardiac defects [39].

The prevalence of aneurysm formation, aortic dissection and recoarctation from restenosis or residual obstruction varies greatly after balloon angioplasty. This disparity is likely due, in part, to the absence of universally accepted guidelines for the procedure. The selection of an appropriate balloon diameter, type of system (standard versus high pressure) and the number and duration of inflations has been inconsistent between studies [10, 16, 28, 31, 32, 37]. Numerous studies have attempted to correlate a variety of balloon-to-coarctation diameter ratios with postoperative results [10, 22, 26]. Table 37.1 provides a sampling of the criteria that have been used for balloon dilation. The Valvuloplasty and Angioplasty of Congenital Anomalies registry revealed that the average balloon-to-coarctation ratio among 92 patients was 3.1 ± 1.0. Although indications for treatment and catheter selection were not regulated in the registry [22], the results showed that patients with minimal residual pressure gradients up to 12 months after the procedure were treated using a balloon-to-coarctation ratio of approximately 3. Interestingly, those with residual gradients greater than 20 mmHg had balloon-to-coarctation ratios closer to 4. The few reported cases of aneurysm formation corresponded to a ratio of 2.7 ± 0.9, suggesting that these ratios, in isolation, are a poor predictor of procedural outcome and aneurysm formation [34].

This conclusion is further supported by seemingly "routine" procedures of transcatheter intervention that have resulted in unexpected mortality [16, 40]. These cases suggest the preoperative integrity of the aortic wall may be a more useful indicator of proper balloon diameter sizing and the feasibility of angioplasty for a given lesion. Several studies have calculated acute gain, stretch and recoil during treatment for postoperative correlation to recoarctation, but the collective results are contradictory [36, 41]. Only one study to date has performed preoperative assessment of aortic integrity prior to intervention [42]. In this study, aortic stiffness, distensibility and compliance were calculated offline using intravascular ultrasound diameter and pressure measurements obtained proximal, distal and within the

Table 37.1. Suggested balloon diameters for the treatment of coarctation of the aorta

Balloon inflation diameter	Reference(s)
2 or more times the coarctation diameter, and less than the diameter of the aorta at the diaphragm	Rao et al. [14, 28, 31]
Diameter of the aorta at the diaphragm	Anjos et al. [37], Maheshwari et al. [82]
1–2 mm less than or equal to the diameter of the aorta at the diaphragm	Fawzy et al. [15, 83]
Average of the aortic diameter at the diaphragm and proximal to the origin of the subclavian artery	Huggon et al. [84]
Balloon-to-isthmus diameter ratio approximately equal to 1	Mendelsohn et al. [27]
Less than or equal to the diameter of the aorta at the subclavian plus 2 mm	Fletcher et al. [26], Ovaert et al. [10]
Balloon-to-native aorta ratio less than or equal to 1.1:1 (excluding coarctation, hypoplasia, and pre- and post-stenotic dilatation)	de Giovanni et al. [85]
Less than or equal to the diameter of the aorta at the isthmus plus 2 mm	Park et al. [38]
150% of the diameter of the aorta proximal to the coarctation	Yetman et al. [16]
Less than or equal to the diameter of the aorta at the diaphragm plus 2 mm	Koerselman et al. [17], Walhout et al. [24, 86]
2–3 times the coarctation diameter, and less than or equal to the diameter of the aorta at the diaphragm	Patel et al. [30]

region of the coarctation. Aortic stiffness, compliance and distensibility were elevated prior to balloon angioplasty compared with for patients without coarctation. These indices were unchanged within and proximal to the coarctation region after angioplasty and were unrelated to the severity of the resulting pressure gradient [42]. These results support the finding that coarctation is associated with temporal adaptations in vascular morphology [43].

37.2.3 Stent Implantation

The use of stents has recently been applied to the treatment of coarctation for the same minimally invasive reasons that prompted the popularity of balloon angioplasty in this patient population. Stenting works by stretching and scaffolding rather than tearing the aorta and this likely accounts for the observed lower short-term instances of aneurysms and recoarctation as compared with those from balloon angioplasty alone. The popularity and enthusiasm resulting from studies conducted over the last 15 years may eventually lead to the preferential use of stents in the treatment of coarctation [44]. Before stenting can be universally applied for the treatment of coarctation however, several important considerations must be addressed by the research and clinical communities. Most notably, studies describing the long-term efficacy of stent implantation must be completed.

37.2.4 Immediate and Intermediate-Term Outcomes After Stenting

In 1991 O'Laughlin et al. [45] reported the first use of a Palmaz iliac artery stent to reduce the pressure gradient across a coarctation in the thoracic aorta (50–25 mmHg) of a 12-year-old patient previously treated using balloon angioplasty. Subsequent case reports documented successful deployment of stents for palliative treatment of patients with severe coarctation that had been treated by surgery [46], balloon angioplasty [47] or both [44]. Larger studies then emerged with six to 54 patients [9, 48–56]. To date, no cases of mortality have been officially reported, but correspondence between interventionalists indicates that they do exist [57]. The majority of these reports include a mixture of patients with native and recurrent coarctation and the results demonstrate that stents generally restore systolic pressure gradients to 20 mmHg or lower in both patient populations. However, the mean patient age in these studies ranges from infants to older adults. Follow-up times have also fluctuated and success is typically measured according to current lower limits for mortality, aneurysm formation, recoarctation and hypertension. Table 37.2 summarizes the follow-up of this group of patients reported since the onset of this procedure. These studies have revealed an overall rate of aneurysm formation of 0–12% [9]. Many patients were hypertensive at rest prior to stent implantation and this hypertension was often attenuated after treatment [9, 54].

The technique for stent implantation is similar to that for balloon angioplasty with the obvious difference that a stent is left in place at the end of the procedure. In nearly all cases, the stent is delivered in a retrograde fashion. A long sheath (typically 35–70 cm in length) is advanced across the coarctation to protect both the stent while in transit and the coarctation site. It also facilitates passage of the stent through tight or tortuous coarctations. An additional pigtail catheter placed in the ascending aorta via a trans-septal approach is more commonly employed in this instance than with balloon angioplasty and is used for angiographic assistance in exact stent placement. Angiography can be performed through the long sheath if the operator chooses not to place a second catheter in the ascending aorta.

In tight lesions, some operators choose to gently inflate a balloon at the coarctation site to "get a feel" for

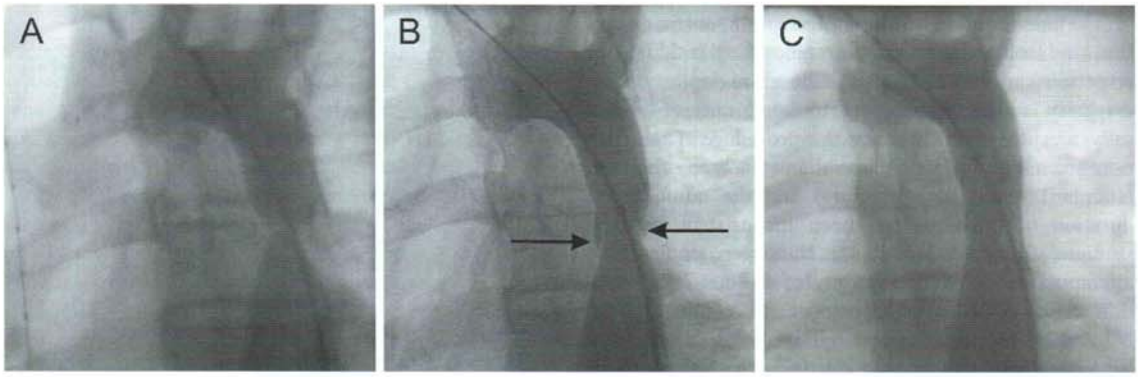

Fig. 37.2. Angiographic images of a coarctation before (**A**) and after (**B**) balloon angioplasty and subsequent stent implantation (**C**). Stent implantation can stabilize intimal flaps (*arrows*) caused by balloon angioplasty while providing relief of the coarctation

Table 37.2. Summary of characteristics and results in groups of patients treated for coarctation of the aorta by stent implantation

Reference(s)	Patients			Follow-up (months)	Stent diameter	Aneurysm	Hypertension	Recoarctation	Systolic gradient (mmHg)		
	Number	Prior surgeries	Age (years)						Preimplantation	Postimplantation	Follow-up
Bulbul et al. [49]	6	4	20±5	8	16±3	0	3/6 (50%)	1/6 (17%)	37±17	13±23	–
Ebeid et al. [51]	9	7	14–63	18	15	0	2/9 (22%)	1/9 (11%)	37±7[a]	4±1[a]	7±2[a]
Magee et al. [54]	17	17	17	7.5	11	1/17 (6%)	10/17 (59%)	2/17 (12%)	26	5	–
Suárez de Lezo et al. [55]	48	17	14	25	12±3	2/30 (7%)	0	8/30 (27%)	42±12	3±4	4±8
Thanopoulos et al. [56]	17	9	11[b]	33	14±2	0	2/17 (12%)	0	50±25	2±2	7±5
Hamdan et al. [52]	34	21	16±8	29±17	–	0	1/22 (5%)	0	64±21	3±4	–
Harrison et al. [9]	27	27	30	16	17	3/26 (12%)	7/26 (27%)	7/26 (27%)	46±20	3±5	4±8
Ledesma et al. [48]	54	–	23	25	–	3/53 (6%)	15/53 (28%)	2/53 (4%)	50±20	5±8	–
Zabal et al. [33]	22	–	26	22	–	0	1/22 (5%)	0	64±21	3±4	–
Johnston et al. [53]	32	23	15	18	16±3	0	3/6 (50%)	1/6 (17%)	37±17	13±23	–

The number of total patients listed under the aneurysm, hypertension and recoarctation columns represents the number of patients observed at follow-up and not necessarily the number of patients initially treated.
The data are presented as the mean ± standard deviation except where indicated.
[a] Mean ± standard error of the mean.
[b] Median

the resistance to dilation [58]. The stent is then mounted on a balloon of the appropriate size and advanced over the wire through the long sheath. The long sheath is retracted to allow expansion of the balloon and its associated stent. The balloon is then inflated and the stent fully apposed to the wall of the aorta. Repeat pressure measurement and angiography are performed and the stent may be further dilated if necessary. There is considerable debate around the need for flaring of the stent ends after implantation. Poor proximal and distal stent apposition was reported in 13 of 18 patients 1 year after stent implantation [9]. Although there were no ramifications of these observations, some clinicians have elected to flare the proximal and distal stent edges to achieve better aortic apposition [51–53]. This may reduce the instances of stent migration and embolization occasionally observed during treatment of coarctation [52], but may also increase the potential for aortic puncture due to the sharpness of stent struts or drastic mismatch between the compliant aorta and the rigid stent. Currently most operators feel that flaring is not necessary if the stent is well apposed to the aorta proximal to the original coarctation site.

Stents also have the ability to stabilize intimal tears resulting from balloon angioplasty (Fig. 37.2). This stability may be derived from scaffolding and supporting the intimal flaps or the development of neointimal hyperplasia that facilitates healing of the vessel and decreases the potential for aortic dissection. In coarctation patients with stenotic aortic valves or modestly calcified aortas, preoperative stent implantation prior to surgical valve replacement may be practical and may decrease the overall risk of surgery [35].

37.2.5 Complications Associated with Stent Implantation

Recoarctation following stent implantation may occur as a result of neointimal hyperplasia or patient growth. Some amount of neointimal hyperplasia occurs after non-drug-eluting stent implantation in all vascular beds [59], but is typically inconsequential in comparison to the diameter of the aorta [9]. Stent overexpansion has been correlated with the severity of neointimal hyperplasia in the aorta and other vascular beds [60, 61]. Recoarctation resulting from patient growth in cases of stent implantation during childhood has been observed in several previous studies [49, 50, 52, 55] and has been a concern since the onset of the procedure [47]. However, this problem is not unique to stent implantation, since severe growth-induced recoarctation has also been observed following correction by balloon angioplasty or surgery [12]. Previous studies using experimental models of coarctation in animals have demonstrated that stents may be reexpanded [62]. In the latter study, Palmaz stents with a length of 30 mm implanted to a diameter of 10 mm could be radially expanded by approximately 4 mm. Unfortunately 29% (two of seven) of the animals experienced aortic rupture resulting in death after stent redilation [62]. Several studies conducted in humans have described successful expansion of stents following primary implantation on an individual basis, and a recent study was conducted to evaluate the efficacy of reexpansion for the serial treatment of coarctation [50, 53, 54]. In these studies, progressive expansion was able to restore the aorta to the diameter

obtained immediately after initial implantation. None of these studies have demonstrated that this technique can be used to account for somatic growth [50].

Several studies have documented the occurrence of aneurysms after stent implantation for treatment of coarctation [9, 26, 48, 54, 55]. Some of the aneurysms may be attributed to predilation with balloon angioplasty [26, 48, 54, 55]. In such cases the rate of aneurysm formation was approximately 6% [9, 26, 51]. Conversely, in cases where stent implantation was performed in the absence of predilation with balloon angioplasty, the rate of aneurysm formation was less than 1% [49, 52–54, 56]. These collective results suggest that primary stent implantation should be advocated to avoid the increased risk of aneurysms associated with balloon angioplasty. However, the prevalence of aneurysms following stent implantation may differ from that published as a result of variations in the definition of an aneurysm and questions as to the utility of imaging techniques for their assessment [9].

Other complications have been reported after stent implantation and include loss of femoral artery pulses and iliac artery bleeding [52, 55]. Improved delivery devices, lower-profile balloon–stent combinations and a trend toward primary stent implantation have reduced the frequency of these occurrences in recent years. Neurological complications, either transient ischemic attacks or cerebrovascular accidents, have been described with attempted balloon angioplasty or stent implantation [9]. Balloon rupture and partial embolization have also been an occasional occurrence during stent implantation primarily owing to resistant lesions and the sharp edges of the Palmaz stent [52]. As a result, some clinicians have suggested using high-pressure angioplasty balloons to minimize the potential for rupture and aortic damage during stent deployment or reexpansion [50, 52]. Newer balloon-in-balloon technology has reduced the incidence of balloon rupture, but has not gained widespread favor owing to the significantly larger sheaths required for stent delivery [63].

37.2.6 Indications for Stent Implantation

In addition to adult or near-adult-sized patients in whom stent placement is now becoming the treatment of choice, stent implantation may be especially practical for diffuse lesions where longer angioplasty balloons would be more likely to induce aneurysms and surgical repair would require the resection of a large portion of the aorta [64]. Similarly, older patients with longer coarctation segments, but modestly compromised aortic elasticity, may opt for stent implantation as an alternative to the insertion of an interposition graft or synthetic patch. Stents also have the ability to realign convoluted proximal and distal portions of the aorta that may be tortuous as a result of coarctation [55]. However, Harrison et al. [9] recently reported stent patient with a geometrically induced aortic obstruction that was complicated by aneurysm formation. The authors caution that surgery should be considered for these patients, or that stents should be gradually expanded over several catheterization procedures. Stent placement is also indicated following previous surgical repair or balloon angioplasty as repeating either treatment has met with marginal results [12, 13] and investigators have also used stents for palliative treatment when the risk of surgical repair was too great [45].

Isthmus and transverse arch hypoplasia are often present before stenting [55], but have also appeared with significant residual gradients after stenting of the primary coarctation region [52]. Successful stent implantation was recently reported in 29 coarctation patients presenting with aortic isthmus or transverse arch hypoplasia [55, 65]. In this study, concomitant stenting of the hypoplastic and coarctation regions was performed without short-term or intermediate-term complications. Immediate and follow-up angiographic images demonstrated that the aortic anatomy was hemodynamically advantageous after stent implantation [55]. This may prompt others to stent hypoplastic segments in future studies.

Contraindications for the use of stents in the treatment of coarctation are more controversial. Numerous case reports, first with balloon angioplasty and now with respect to stent implantation, have proclaimed or suggested that the use of such devices is efficacious despite the lack of long-term follow-up data. For example, the diameter of stents deployed to treat coarctation reported in the literature has ranged from 11 to 18 mm [9, 49, 54, 55, 58]. The typical diameter in an adult aorta is approximately 24 mm [66]. This disparity suggests that these stents will likely result in reports of recurrent aortic stenosis once long-term data are available. To our knowledge, no study conducted to date has expanded stents to account for progressive somatic growth in humans. As a result, additional studies properly designed to assess the chronic treatment of patients will be necessary to determine the ultimate utility of stents for the treatment of aortic coarctation. Moreover, there may be ramifications to this treatment as progressive expansion and inherent stent shortening may cause serious damage to the lumen of the aorta both circumferentially and longitudinally. Currently, a general consensus exists that surgery is preferred over stent implantation in neonates and infants as large sheaths and delivery catheters have the potential to cause occlusive femoral artery damage [49, 52], and somatic growth may cause recoarctation, prompting additional procedures [52, 55].

Long-term, randomized and prospective studies have not yet been conducted owing to the relatively recent application of this technology and scarcity of these cases at a single institution. The Congenital Cardiovas-

cular Interventional Study Consortium (CCISC), a concerted, multicenter registry is currently enrolling transcatheter and surgically patients. This will provide invaluable data about long-term morbidity and how treatment options may influence the overall outcomes.

Partial obstruction of peripheral vessels by stent struts may occur during and after stent implantation [9], but no adverse results have been reported in short-term or midterm follow-up studies. However, until long-term data are available, this situation should be avoided, if possible, to limit the potential restriction of blood flow to the carotid, subclavian, spinal and brachial arteries [67]. When partial obstruction of peripheral arteries is inevitable, expansion of obtrusive struts with subsequent balloon angioplasty catheters may be a viable option after coarctation relief is obtained.

A variety of anatomical diameters throughout the aorta have been referenced during stent implantation in an attempt to minimize aortic damage [51–53, 56]. Many investigators have selected stent deployment balloons with diameters equal to that of the transverse arch, but not exceeding that at the level of the diaphragm [33, 51, 56]. Still other studies have attempted to expand stents to a final diameter based on that of the stenosis [52, 53].

Although transcatheter techniques have revolutionized the treatment of coarctation, it is of concern that the practice of balloon angioplasty and stent implantation does not ubiquitously consider the compliance, pathology or functional integrity of the stenosed region prior to treatment. Preliminary evidence indicates that surgical correction or stent implantation for the treatment of coarctation may alter properties of the vascular wall [43, 50]. Collectively, these results strongly suggest that future procedures should work toward establishing noninvasive indices to determine the severity of vascular dysfunction within the region of the stenosis before treatment. Some investigators have suggested assessing stenosis compliance using the response of the vascular lesion to balloon predilation at 3 or 4 atm [58]. However, such invasive methods may predispose the region to aortic damage or embolization during stent implantation [49]. Nevertheless, implementation of novel pathological assessment tools prior to treatment may delineate which patients are more susceptible to aortic rupture, and should be referred for surgery.

Modest residual gradients persisting after correction for coarctation are an important indicator of future complications [33] and cause mild, but persistent stress on the left ventricle. Left ventricular end diastolic pressure (LVEDP) decreased 7 months after stent implantation in a recent study of 16 patients treated for mild residual or recurrent coarctation [58]. In this study, stent implantation was indicated and performed as a secondary treatment when inadequate pressure gradient relief was observed after balloon angioplasty. These results suggest that stent implantation may be superior to balloon angioplasty for eliminating these mild residual stenoses and for reducing LVEDP [58].

Although there are currently no FDA-approved stents designed to treat aortic coarctation, new designs that specifically consider somatic growth will likely be developed in the next decade. The Palmaz stent is currently the most popular choice for treatment of coarctation [68] and has recently been made available in 40- and 50-mm lengths. Studies have suggested that stent redilation of shorter Palmaz stents may be possible using animal models of aortic coarctation, but that this expansion is associated with appreciable reductions in stent length [63]. If long-term data prove that reexpansion of these stents is both necessary and possible in response to aortic growth, longer designs may ensure that foreshortening does not displace the stent from the localized region of the coarctation. Biodegradable stents may be the ideal choice for congenital stent implantation, but several technical limitations, including inadequate radial strength and small diameter sizes, prevent their application to coarctation at the present time [68]. In theory, these devices represent an important treatment option as their gradual degradation may provide the greatest chronic benefit during the growth of a patient.

37.3 Comparing Treatment Modalities

Comparisons between groups of patients treated with the various methods of coarctation repair have only recently emerged and should be interpreted within the constraints of their inherent limitations. Procedural details associated with balloon angioplasty and surgery have evolved from empirical evidence and revised techniques that have developed since the introduction of these procedures. In comparison, early case reports and studies with modest patient populations treated by stent implantation have only been conducted since 1991 [45], and the results are therefore biased by the relatively recent application of stents to the treatment of aortic coarctation. Fortunately, the short- and intermediate-term results of stent implantation for the treatment of coarctation in certain patient populations appear to be comparable to those obtained after surgery or balloon angioplasty [48, 52, 55].

In a recent retrospective study, comparisons were attempted between two treatment modalities despite significant differences in follow-up periods [24, 33]. The median ages for patients in the surgical and balloon angioplasty groups of one study were 0.63 and 5.8 years, respectively [24]. Immediate success rates were nearly equivalent for both techniques and they were comparable in their ability to reduce pressure gradients acutely at rest. However, recoarctation was modestly higher at follow-up for patients treated with balloon angioplasty

as compared with that for those treated surgically during infancy (7 versus 5.6%, respectively) [24].

Studies have indicated that mortality after balloon angioplasty and surgery increases with patient age at coarctation repair, and preliminary results after stent implantation suggest that procedural outcome is better if stent implantation is performed shortly after identifying the coarctation [22]. These findings suggest that favorable chronic results after any treatment to alleviate coarctation depend on recovery of previously depressed cardiac function after the procedure [58]. Conversely, the potential for recoarctation following balloon angioplasty or surgical correction for coarctation is greater when patients are treated during infancy. This finding may be explained by the local vascular morphology of the aorta within the region of the coarctation during this period [27, 28, 38] as 67–91% of patients with a patent ductus arteriosus developed recoarctation following balloon angioplasty for treatment of native coarctation [26, 38]. Comparisons between surgical and transcatheter treatments for coarctation are also complicated by different definitions of procedural success and inconsistencies in the extent of patient data reported in each study. Comparison of experiences during surgical repair of native coarctation and those after unsuccessful angioplasty in the absence or presence of transcatheter-induced aneurysms revealed no differences in procedural complications or outcome [69]. These findings suggest that prior balloon angioplasty will not preclude future successful surgical intervention for recoarctation. This may not be true, however, in the case of surgery after stent implantation. If further expansion of stents becomes necessary but is not possible, the removal of large portions of the aorta or the continued presence of the stent in the aortic wall may prove less beneficial when compared with initial surgical treatment of the coarctation.

37.4 Future Considerations for Transcatheter Treatment of Aortic Coarctation

37.4.1 Criteria for Successful Treatment

A universal standard for assessing the success of treatments for coarctation has not yet emerged. A residual pressure gradient of 20 mmHg or lower at rest has been the historical goal after transcatheter or surgical interventions [52, 70], though some clinicians define success as gradients ranging from 15 to 40 mmHg [27, 52, 71, 72]. Other clinicians have suggested treatment options based on functional flow charts of immediate-term results [33], while still others reference an average blood pressure percentile based on the patient's age [14]. Unfortunately, while pressure gradients represent fairly standardized guidelines suggesting when the severity of a coarctation warrants intervention, there are few experimental or physiological data supporting any of these guidelines and they remain the only parameter of "success" monitored. The current 20-mmHg guideline for intervention has evolved from the best possible results that were previously obtained, on average, by surgical repair or balloon angioplasty without the increased risk associated with additional procedures [67]. With the introduction of stent implantation, awareness of cardiac dysfunction that can result from modest residual gradients, and advances in computational tools to assess the ramifications of a particular treatment, the hemodynamic and physiological influence of various pressure gradients, including the putative 20-mmHg guideline, should be revisited.

Not surprisingly, coarctation causes increased blood pressure and fatigue during exercise and several studies have advocated exercise testing as an indication for recoarctation after surgical repair [5]. Pathological modifications resulting from increased afterload prior to the correction of a coarctation can persist even after surgical or transcatheter repair and are important risk factors for morbidity and mortality in adulthood. This finding is underscored by a study demonstrating that patients surgically treated for coarctation rely on anaerobic metabolism to a greater extent during exercise than age- and gender-matched controls [4]. Interestingly, previous studies have demonstrated that patients who are normotensive at rest after coarctation repair are often hypertensive during exercise [5]. These studies, combined with the premature life expectancies of patients with coarctation, suggest that the current means of assessing procedural success predominantly under resting conditions should be reassessed. To our knowledge, no studies have examined exercise-induced systolic pressure gradients after stent implantation. Although a previous porcine model demonstrated that aortic compliance and hemodynamics were unchanged shortly after stent implantation at rest [73], subtle differences unique to each treatment option at rest may be amplified during exercise conditions.

37.4.2 A New Paradigm for Treatment

Many of the risk factors for morbidity associated with coarctation can be attributed to abnormal hemodynamics throughout the aorta. The coarctation causes pre- and poststenotic dilatation that, in turn, reduces capacitance and leads to elevated pulse pressure in the ascending aorta. Pressure-wave amplification from the summation of normal incident waves and those reflected from the stenosis account for this increased pulse pressure and hypertension during rest and exer-

cise [74]. Subsequent perfusion of the coronary arteries during diastole is also compromised, resulting in decreased flow and an increase in precursors of coronary artery disease [75]. Reduced coronary artery perfusion and concomitant increases in afterload may also explain the high instance of indices associated with heart failure in these patients [58, 76]. Relief of these deleterious hemodynamics was observed following the alleviation of an experimental coarctation [74], suggesting that treatment strategies that optimize vascular hemodynamics may provide the greatest chronic benefit to patients.

Hypertension is the most frequent complication associated with repair of coarctation regardless of treatment modality. In a study of patients subjected to exercise testing approximately 20 years after treatment for coarctation by surgical repair, nearly 50% of patients were found to have ambulatory and exercise-induced hypertension, a finding that is commoner when treatment is obtained after 1 year of age [77]. Residual coarctation caused by scarring at the suture site or persistent aortic arch hypoplasia after treatment may contribute to this finding [5]. Although long-term data after stent implantation for coarctation are not yet available, it seems possible that the presence of a rigid stent in the compliant aorta may also cause hypertension. Persistent pathologic arterial modifications such as increased systemic vascular resistance, aortic stiffness, elevated left ventricular contractility [43, 78] and anatomical abnormalities of the transverse arch not unique to a particular treatment strategy are also thought to contribute to hypertension [79].

Coronary artery disease, cerebral aneurysms and stroke also occur despite "successful" coarctation repair, indicating that the current perception of success may be incorrect and the ongoing severity of treatment-specific hemodynamic alterations manifested in the aorta and coronary, head and neck vessels during ambulatory or exercise conditions may contribute to long-term morbidity. For example, studies conducted on canine coronary arteries have demonstrated that the compliance mismatch between a stent and a native vessel is masked during conditions of resting blood flow, and causes deleterious alterations in local flow patterns during maximum vasodilation [80]. Similarly, the coarctation causes drastic reductions in the capacitive function of the aorta and there are likely hemodynamic ramifications of the compliance mismatch caused not only by coarctation prior to surgical or catheter-based intervention at rest, but also during ambulation.

It is clear from the clinical literature that parametric alterations within a single treatment, or relying on the gradual empirical modification of these treatments, will only modestly increase the life expectancies of patients with aortic coarctation. Alternatively, more favorable long-term results may be possible by examining the origin of coarctation symptoms that emanate from alterations in vascular hemodynamics within the ascending aorta.

Researchers in the Cardiovascular Biomechanics Research Laboratory at Stanford University, in collaboration with Departments of Pediatric Cardiology and Cardiothoracic Surgery, are currently investigating a new paradigm to improve our understanding of the hemodynamic and physiologic conditions before and after treatment for coarctation. This research is based on the hypothesis that treatment strategies that optimize vascular hemodynamics at rest and during exercise will minimize known risk factors for long-term morbidity associated with aortic coarctation. Rather than modifying the technique of a given treatment or evaluating strategies based on the current standards for mortality, recoarctation, aneurysm formation and hypertension, treatment strategies could be scrutinized according to their ability to restore optimal hemodynamics in the ascending and descending aorta and head and neck vessels. A similar approach to treatment planning has previously been described for occlusive vascular disease in adults [81].

Through this interdisciplinary collaboration, computer models can be created from time-resolved 3D phase-contrast magnetic resonance imaging data obtained at rest and during lower limb exercise using a

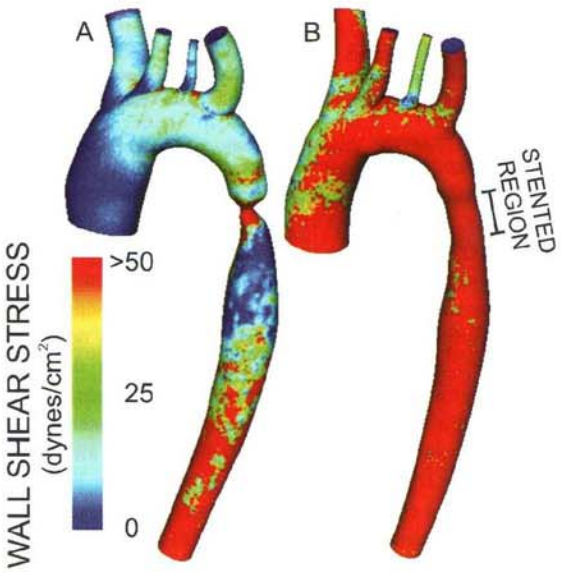

Fig. 37.3. Average wall shear stress (*WSS*) in a patient with coarctation of the aorta before (**a**) and after (**b**) stent implantation. Aortic coarctation causes ascending aortic dilation and pre- and poststenotic dilatation that is responsible for low WSS in the arch, ascending and descending aorta and branch arteries. Most of these low WSS regions are alleviated after stent implantation, but some areas of the aortic arch, branch vessels and anomalous vertebral artery remain and may be deleterious as low WSS is known to correlate with sites of atherogenesis and vascular inflammation

supine stationary ergometer. Treatment-specific alterations in vascular wall motion, indices of wall shear stress and pressure wave reflection and amplification manifested in the head and neck vessels and throughout the aorta can then be quantified and interpreted as surrogates of the potential for morbidity (Fig. 37.3). The results may reveal hemodynamic adaptations associated with the acceptable systolic pressure gradient of 20 mmHg and determine if treatment-specific guidelines may be more appropriate for minimizing morbidity. In addition, the simulations can reveal 3D spatial and temporal hemodynamic ramifications of compliance mismatch caused by the coarctation prior to intervention, and the surgical suture line or rigid stent afterward. This hemodynamic characterization process may be amenable to predicting which treatment strategies will be advantageous for a particular patient and to identifying deleterious processes that lead to morbidity decades before they are clinically apparent.

In the future, computational models may be used to determine which strategy will benefit the patient from a hemodynamic and physiologic perspective. If the development of these computational models based on patient-specific anatomy and physiology is successful, they may provide the potential to increase our scientific understanding of this problem and the various treatment options. In the long term, patient-specific modeling may provide clinicians with a resource to decrease disease- and procedure-related morbidity and mortality.

37.5 Summary

Balloon angioplasty and stent implantation are now widely accepted as treatment options for coarctation of the aorta. Both of these strategies, as well as surgical repair, have advantages and disadvantages in specific patient populations. In the future, changes in stent design and materials and better predictive models of appropriate candidates for endovascular treatment will optimize treatment outcomes. As additional long-term data regarding procedural success, morbidity and mortality become available and surgical and transcatheter techniques progress, management strategies will also continue to evolve. As always, close collaboration between surgeons and cardiologists will remain imperative.

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