Angiographic Follow-Up After Balloon Angioplasty For Coarctation of the Aorta

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Balloon angioplasty for coarctation of the aorta was performed in 45 patients; 17 (Group 1) were infants (mean age 34 ± 43 days). This study focuses on the remaining 28 patients (Group 2), children and adults ranging in age from 2 to 31 years (mean 13 ± 8). Two patients had had previous surgery for coarctation; in the remaining 26 the coarctation had not been surgically treated. Twenty patients (71%) had a discrete type of coarctation; the remaining eight had a diffuse or eccentric stenosis. Angiographic studies of the aorta were performed before and immediately after angioplasty in all 28 patients; 16 (57%) of them also underwent angiographic reevaluation 10 ± 2 months later.

The pressure gradient decreased immediately after angioplasty and was unchanged at follow-up. Residual gradient at follow-up ranged from 0 to 40 mm Hg (mean 11 ± 13). Patients with a discrete type of coarctation had a lesser residual gradient than did patients with other anatomic types of coarctation. Angiographically, the stenosis also decreased after angioplasty (68 ± 10% versus 24 ±

17%; p < 0.01); a new small, but significant decrease was observed at follow-up (9 ± 18%; p < 0.05).

A quantitative analysis of the aorta at different levels revealed no significant changes proximal and distal to the coarctation segment; however, at the level of coarctation, a significant (p < 0.01) and homogeneous increment was observed after dilation that was unchanged at follow-up. One (6%) of 16 reevaluated patients had a clear aneurysm at the site of dilation. In addition, changes in configuration of the aorta showing a tendency to realignment were observed and quantified by the change in configuration angle determined from the proximal and distal aortic segments (169 ± 17°). This angle did not change immediately after angioplasty, but increased significantly at follow-up (186 ± 17°, p < 0.05). These findings suggest that flow-dependent configuration changes can develop in the aorta after angioplasty.

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Coarctation of the aorta occurs clinically in two patterns. In one pattern, severe isthmic coarctation occurs in neonates and infants, usually as part of a constellation of lesions. Balloon angioplasty in these severely ill infants is performed as a palliative procedure that permits, in most patients, delay of surgery until better conditions for repair exist (1–3). In the other clinical pattern, the coarctation is usually an isolated lesion and may be seen at any age. Because balloon angioplasty in children and adults can provide more prolonged relief than is afforded to infants, it has been considered as an alternative to surgery in these patients (2,4). However, information is still needed regarding 1) those patients in

whom balloon angioplasty may be more beneficial than surgery, and 2) the long-term anatomic and physiologic effects of the procedure.

This report presents our findings from serial hemodynamic studies in patients undergoing balloon dilation for coarctation of the aorta at our institution.

Methods

Study patients. We studied the hemodynamic and angiographic effects of balloon angioplasty in 45 patients treated for coarctation of the aorta from May 1983 to May 1988. We classified the patients into two groups according to clinical presentation and course. Group 1 comprised 17 neonates or infants with congestive heart failure. Initial and late results in this group have been reported (3,5,6). Group 2 comprised 28 patients (19 children [mean age 8 ± 4 years] and 9 young adults [mean age 22 ± 5 years]) ranging in age from 2 to 31 years (mean 13 ± 8). This study focuses on Group 2. Five

Table 1. Technical and Angiographic Measurements in 28 Patients

	Mean ± SD	Range
Age (yr)	13 ± 8	2 to 31
BSA (m²)	1.2 ± 0.4	0.48 to 1.94
Technical aspects of angioplasty		
Balloon size (mm)	15.2 ± 3.4	8 to 20
Inflation pressure (atm)	5.3 ± 2	3 to 8
Angiographic measurements/BSA		
Isthmus (mm/m²)	12.9 ± 4.7	6 to 24
Coarctation (mm/m²)	4.9 ± 2.3	1.5 to 11
"Ideal aorta" (mm/m²)	14.9 ± 5	7 to 27
Poststenotic dilation (mm/m²)	17 ± 5	9 to 28
Distal aorta (mm/m²)	15.4 ± 4.9	7.5 to 26

BSA = body surface area.

(18%) of these 28 patients had associated lesions (congenital aortic stenosis in 2, mitral stenosis in 1, mitral valve prolapse in 1 and cerebral aneurysm in 1). All 28 presented with systemic hypertension, 1 (4%) with dyspnea and 1 31 year old patient (4%) with rupture of a cerebral aneurysm. Two patients had had previous surgery for coarctation; in the remaining 26 the coarctation had not been surgically treated.

Diagnostic and therapeutic procedures. With patients mildly sedated we performed percutaneous right and left cardiac catheterization. Table 1 shows data on patient age and size, technical aspects and angiographic measurements. The coarctation was passed retrogradely. Diagnostic procedures always included hemodynamic and cardiac output measurements and left ventricular and aortic angiograms. After this, an 8 to 9F single balloon catheter was retrogradely advanced through a guide wire previously positioned in the ascending aorta. To avoid pain during angioplasty, mild anesthesia was applied at that point. To avoid hemorrhagic damage in the dilated aortic wall, we never administered heparin. The balloon was placed within the stenosis and one or two full balloon inflations were performed. During dilation there was a notch in the balloon that, in most instances, disappeared at full inflation. Once the notch disappeared, the balloon catheter was rapidly interchanged through the guide wire for a diagnostic catheter. A new hemodynamic and angiographic evaluation was then performed.

Selection of balloon diameter was always based on immediate angiographic measurements of the mean value between isthmic and distal aortic diameter (descending aorta distal to the poststenotic dilation). Four angiographic types of coarctation were dilated: 20 patients (71%) had a discrete type; 5 (18%) had a more diffuse but concentric stenosis; 1 (4%) of 2 postoperative patients showed an irregular type of coarctation and 2 other (7%) an eccentric stenosis. In one patient we performed sequential dilations of the coarctation and of a discrete subaortic membrane (5).

Quantitative aortography. Qualitative and quantitative angiographic studies of the aorta were performed before and after angioplasty in all 28 patients; 16 (57%) of them with native coarctation have been angiographically reevaluated a mean of 10 ± 2 months later. The quantitative studies were performed over the drawn silhouettes obtained from the projected diastolic image of the 60° left anterior oblique aortogram. The same projection and conditions were observed in all three studied situations (basal, postangioplasty and follow-up). Figure 1A shows the method used for segmental and radial analyses at different levels of the aortograms. The "ideal aorta" was traced in all three situations by prolonging the theoretical aortic walls between the proximal and distal aorta. The coarctation segment stenosis was expressed as a percent of the "ideal aorta" (ideal aorta − coarctation diameter/ideal aorta × 100). The central line of this reconstructed aorta was traced at the mid distance from theoretic aortic walls, irrespective of the coarctation center point position. Taking the midpoint of the left subclavian artery (A) and the coarctation segment (C) as reference points, five transverse segments (A to E) were traced. Segment B was at the mid distance between A and C; segments D and E were traced 1.5 and 3 cm, respectively, below the coarctation level. The central aortic line divided segments in radii that were analyzed separately. All measured values were corrected for X-ray magnification and expressed in millimeters per square meter of body surface area. We also studied the possible change in aortic configuration after angioplasty by measuring the angle determined by the proximal and distal aorta at the level of the coarctation (Fig. 1A). Both angle axes were determined by the lines between two midsegment points proximal (0.5 and 1.5 cm) and distal (0.5 and 1.5 cm) to the coarctation segment.

Statistical study. Data are presented as mean values ± SD. The nonpaired Student's *t* test was used to compare two groups with approximately normal distributions. Differences between the conditions (basal, post and follow-up) were tested by a one-way analysis of variance, and significant differences were evaluated by Schéffe's test.

Results

Outcome. One patient (4%) had a brain embolism after angioplasty; three others (11%) had an absent femoral pulse (in one of these the pulse was restored after intravenous infusion of streptokinase [7]). Paradoxic hypertension appeared in six patients (21%) within the first 24 h after angioplasty. This was treated to reduce the stretch on the dilated aortic wall. Systemic blood pressure decreased and stabilized in all six. All patients were followed up clinically for a mean of 24 ± 11 months. No symptoms developed after the procedure and no patient showed basal hypertension
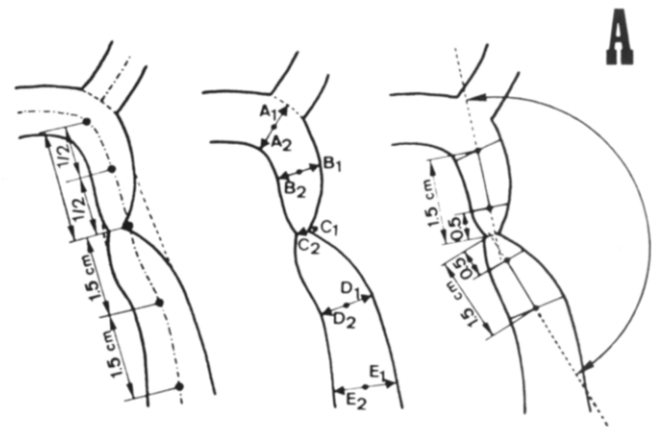
Figure 1. A, Methodology used for quantitative aortography. A segmental (A to E) and radial (A_1, A_2 . . .) analysis was performed. Configuration angle was determined between the proximal and distal aorta; both angle sides were determined by the lines between two midsegments points at 0.5 and 1.5 cm from the coarctation segment. **B,** Graphic results of quantitative aortography. BSA = body surface area; Post = postangioplasty.
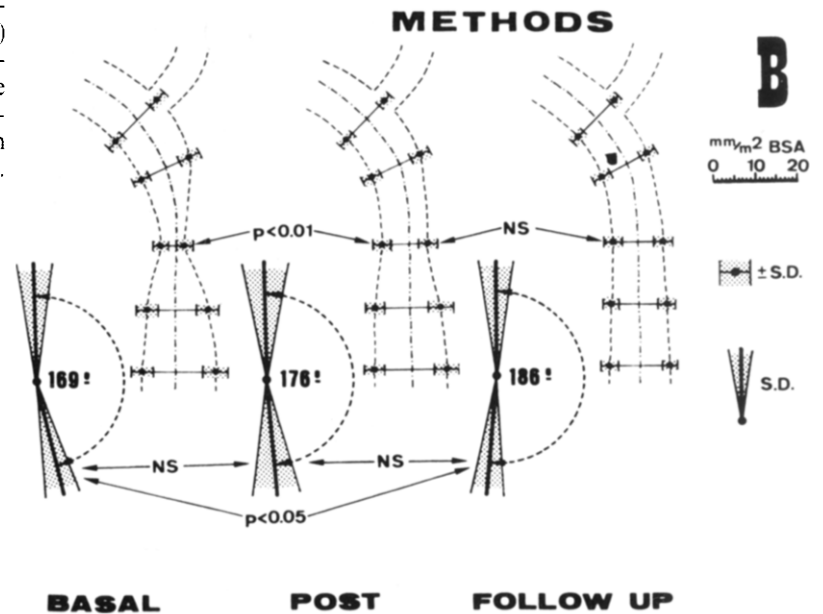
after discharge. Sixteen patients were angiographically reevaluated 6 to 12 months later (mean 10 ± 2).

Hemodynamic and angiographic findings (Table 2). There

Table 2. Hemodynamic and Angiographic Findings in 28 Patients

	Basal	Postdilation	Follow-Up
PSAP (mm Hg)	159 ± 31	147 ± 21	127 ± 17†‡
PSG (mm Hg)	49 ± 16	8 ± 8*	11 ± 13
LVEDP (mm Hg)	11 ± 4	7 ± 2	6 ± 2
C of A/BSA (mm/m²)	5 ± 2	10 ± 3*	12 ± 3
Stenosis (%)	68 ± 10	24 ± 17*	9 ± 18†‡
Increment of C of A (mm/m²)		5.4 ± 3	7.2 ± 3.6

*p < 0.01 comparing postdilation versus basal values; †p < 0.01 comparing basal values versus follow up values; ‡p < 0.05 comparing values at follow-up with postdilation values. BSA = body surface area; C of A = coarctation of aorta; LVEDP = left ventricular end-diastolic pressure; PSAP = peak systolic aortic pressure; PSG = peak systolic gradient.

was a significant decrease in the pressure gradient immediately after angioplasty and it remained low at follow-up. The residual gradient at that time ranged from 0 to 40 mm Hg and it correlated significantly (p < 0.05) with the peak systolic pressure in the ascending aorta (r = 0.56). The percent angiographic stenosis also decreased after angioplasty; a new small but significant reduction of stenosis was observed at follow-up.

Inspection of the aortograms in two projections (posteroanterior and 60° left anterior oblique) showed no aneurysm at the dilation site immediately after balloon angioplasty, although in one patient (Fig. 2) there was an image suggesting a damaged aortic wall that changed to an aneurysm at follow-up (1 of 16 patients; 6%). In eight (29%) of the patients, we observed angiographic intimal tears after angioplasty; all eight patients had a discrete type of coarctation. Some abnormalities resembling an aneurysm were present in three patients before the
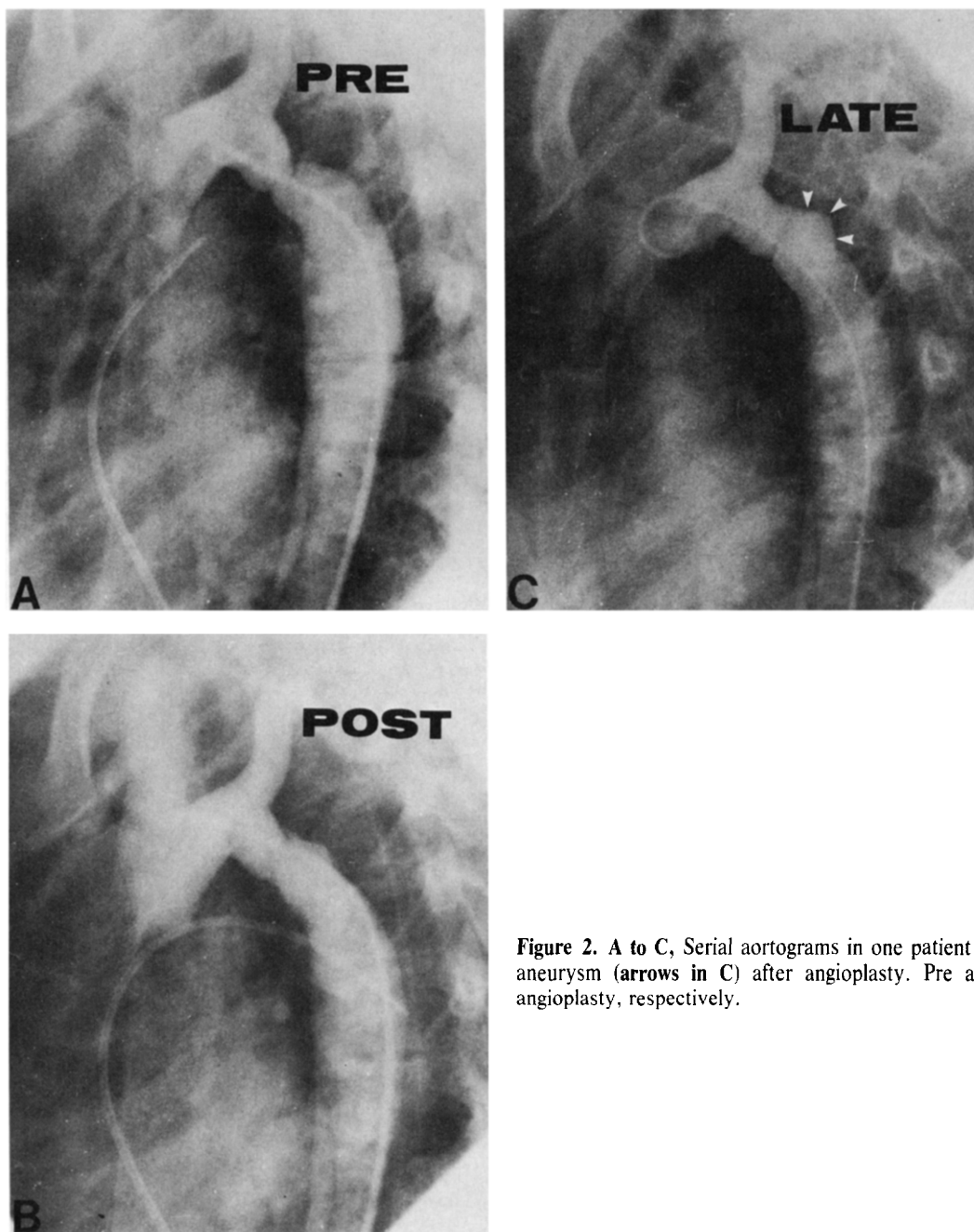
Figure 2. **A to C,** Serial aortograms in one patient (6%) with development of an aneurysm (**arrows in C**) after angioplasty. Pre and Post = before and after angioplasty, respectively.

procedure and persisted afterward (Fig. 3). Changes in configuration of the aortic arch were clearly noted in seven patients (44%) (Fig. 4).

Quantitative aortography. Figure 1B shows graphically the results of the analyses at different levels of the aorta, as well as the evolution of the configuration angle. No significant changes were observed proximal or distal to the coarctation segment. However, at that level, a significant ($p < 0.01$) increment of the coarctation segment was observed after dilation and was unchanged at follow-up. Radial incre-

ments of internal and external walls were homogeneous (that is, not significantly different). In contrast, the configuration angle, determined between the proximal and distal aorta, did not change immediately after angioplasty, but increased significantly ($p < 0.05$) at follow-up (Fig. 1B).

Determinants of residual gradient (Table 3). Patients with a discrete type of coarctation had a lesser residual gradient at follow-up as compared with patients with other types of coarctation. In addition, the presence of an angiographic intimal tear after angioplasty was associated with a signifi-
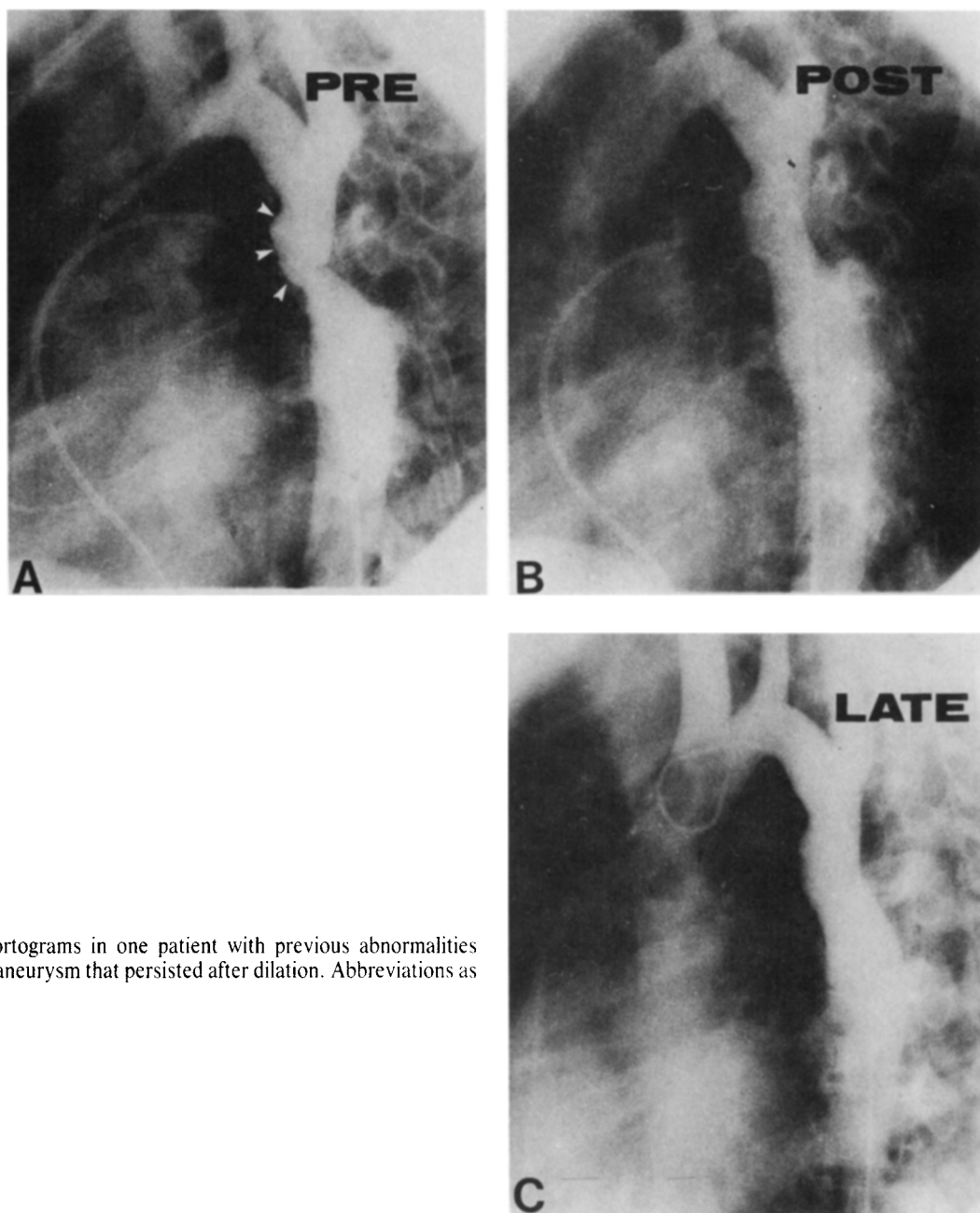
Figure 3. A to C, Serial aortograms in one patient with previous abnormalities (**arrows in A**) resembling an aneurysm that persisted after dilation. Abbreviations as in Figure 2.

cantly smaller gradient at follow-up. However, these findings were not present immediately after angioplasty, indicating that the definitive residual gradient cannot be evaluated early after dilation (2,8).

Discussion

Hemodynamic changes after angioplasty. Although the relief of pressure after angioplasty persists in most patients older than 1 year with surgically untreated or postoperative coarctation (2,6,8–14), restenosis or a persistently high residual gradient at follow-up has also been described (2,6,10,11,15). Several factors can influence the residual gradient after angioplasty. Our study of such factors suggests that the discrete type of coarctation is associated with a lower residual gradient at follow-up, as compared with other anatomic types. Angiographic intimal tears at the site of dilation are also associated with a lower residual gradient at follow-up.

Anatomic changes after angioplasty. On the other hand, there is generalized concern about the long-term anatomic changes in the aortic wall. Angioplasty in aortic coarctation
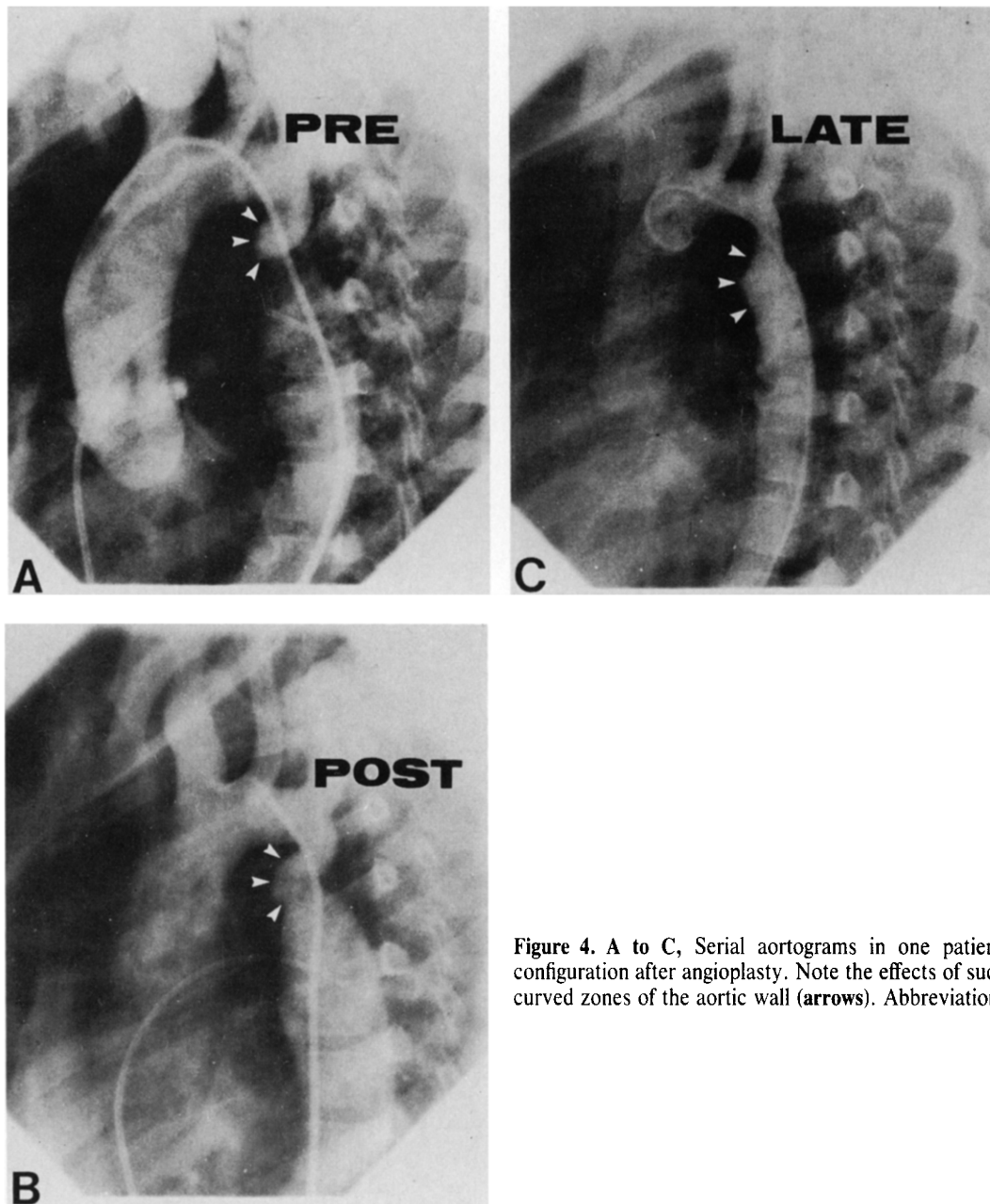
Figure 4. A to C, Serial aortograms in one patient with marked changes in configuration after angioplasty. Note the effects of such changes on the previously curved zones of the aortic wall (arrows). Abbreviations as in Figure 2.

produces a controlled injury of the intima and part of the media, increasing the vessel diameter, and healing over a period of months by way of a fibrous scar (16). However, uncontrolled damage of the aortic wall at the time of dilation can also occur. Observations of such patients at operation (9,17) have shown complete rupture of the media progressing into a saccular aneurysm. Factors determining such damage are not well known. Balloon size could be one factor and there are no general criteria for selecting balloon size. Different degrees of cystic necrosis of the media were recently described (18) as a common pathologic finding in aortic coarctation. Thus, a previously diseased or weak aortic wall could also determine the appearance of an aneurysm after angioplasty in certain groups of patients.

Angiographic follow-up. The reported total number of reevaluated patients is still small and the period of time before such reevaluation is still short. Follow-up angiographic studies have shown persistent gradient relief in most patients and a wide difference (0 to 55%) in frequency of aneurysm formation over the dilated area (2,8– 12,14,15,19). The frequency of this complication has been 6% in our group; 1 of the 16 reevaluated patients had a clear-cut

Table 3. Factors Influencing Residual Gradient After Angioplasty

	Residual Gradient (mm Hg)	
	Postdilation	Follow-Up
Age (yr)		
Adults	11 ± 9	13 ± 10
≤7	7 ± 8	9 ± 10
Isthmus diam/BSA (mm/m²)		
≥12	6 ± 7	10 ± 12
<12	12 ± 9	11 ± 13
Anatomic type		
Discrete	9 ± 8	6 ± 8*
Other	10 ± 9	21 ± 14*
Balloon diam/ideal Ao		
≥1	7 ± 7	11 ± 14
<1	10 ± 9	10 ± 11
Intimal tear (postdilation)		
Yes	7 ± 8	2 ± 3†
No	9 ± 9	15 ± 13†

Ao = aorta; BSA = body surface area; C of A = coarctation of aorta; diam = diameter. *p < 0.05 comparing discrete and other anatomic types at follow-up; †p < 0.01 comparing presence and absence of postdilation intimal tear at follow-up.

aneurysm at the site of dilation. This wide difference in the frequency of aneurysm formation probably reflects the lack of unified criteria in the difficult analysis of anatomic changes after angioplasty. Because morphology of the stenotic area in coarctation of the aorta can vary from patient to patient, angiographic evaluation after dilation should consider the previous anatomy, the type of coarctation and the possible influence of newly reestablished flow through the dilated aortic segment.

The persistence after angioplasty of previously existing abnormalities resembling an aneurysm (Fig. 3) cannot be considered dilation-related damage to the aortic wall (8,11). On the other hand, the aortic arch is an elastic and modifiable artery and anatomic changes in its configuration can be expected to occur after reflow (16). Our observations on serial aortograms after angioplasty suggest that there are some changes in configuration of the aorta that could generate bulges or dilated zones of the arterial wall that are not injury induced. In certain patients they could be the result of changes in configuration of previously distorted and curved stenoses (Fig. 4). A larger series over a longer period of time (16) is needed to assess the real incidence of aneurysm formation at the site of dilation, as a consequence of uncontrolled damage to the aortic wall.

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