Assessment of Ascending Aorta Distensibility After Successful Coarctation Repair by Strain Doppler Echocardiography

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Background: Increased arterial stiffness may participate in the genesis of hypertension and increase of left ventricular (LV) mass after surgical correction of coarctation of the aorta. The purpose of the current study was to assess the aortic elastic properties using Doppler tissue imaging and strain rate imaging in patients after coarctoplasty.

Methods: Echocardiography with Doppler tissue/strain rate imaging capabilities was performed in 26 adult normotensive patients who had successful repair of coarctation of the aorta in infancy and in 24 control subjects. Transesophageal aortic transverse sections were imaged at the level of the proximal and distal segments to the repair site. Doppler tissue imaging wall velocities during systole (S_w), early relaxation (E_w), and atrial systole (A_w) and peak systolic strain (ps ε) were measured in both groups. Transthoracic ascending aorta (AAo) measurements were also obtained.

Results: In the patients with coarctoplasty, S_w velocities and ps ε were significantly decreased in the proximal segments compared with control subjects. Both peak systolic blood pressure after exercise ($P < .001$) and pulse pressure after exercise ($P < .001$) were directly related to AAo wall strain. LV annular early diastolic velocity was significantly reduced compared with control subjects in patients with decreased AAo wall strain and exercise-induced hypertension ($P < .001$) and related to AAo wall velocity ($P < .005$) and strain ($P < .001$). In multiple linear regression analysis, only weight, study group, and AAo wall strain were correlated to LV mass index.

Conclusions: Patients with coarctation of the aorta have reduced proximal aortic wall velocities and strain and increased stiffness even after successful repair. This amplifies stress-induced hypertension and increases LV burden.

Keywords: Echocardiography, Transesophageal echocardiography, Tissue Doppler imaging, Aortic function, Aortic coarctation

Although aortic coarctation (CoA) has been considered a curable disease since the first surgical repair, life expectancy remains reduced even after surgery.[1] Coronary artery disease, stroke, sudden cardiac death, and heart failure account for the majority of premature deaths. Several studies have shown that abnormalities of the precoarctation arterial bed may participate in the genesis of persistent hypertension and increases in left ventricular (LV) mass after successful surgical correction of CoA.[1-4] Coarctation does not appear only as a localized mechanical problem of the aortic isthmus[4] but also as a systemic vascular disease of the precoarctational arteries. Impaired aortic distensibility, primarily present in these patients, contributes to increased cardiovascular morbidity. Color Doppler myocardial imaging has emerged as a new technique for the assessment of ventricular myocardial velocities and regional ventricular contractility[5,6] and recent reports[7-9] suggest it suitable for the evaluation of aortic wall velocities and superior to standard measurements of arterial function as predictor of cardiovascular events. The purpose of the current study was to assess the aortic elastic properties using Doppler tissue imaging (DTI) and strain rate imaging (SRI) echocardiography in adult patients with apparently good results from surgical repair of CoA. In addition, we examined the relationship between the aortic wall properties as determined by strain Doppler echocardiography and the abnormal responses to elevation of blood pressure (BP) and/or increase in LV mass.

METHODS

Population

We studied 26 adult patients aged 19 to 58 years who had successful CoA repair in infancy and early childhood and who were not hypertensive at rest. They underwent the arch repair at 16.3 ± 29.1 months (Table 1) by end-to-end anastomosis in 21 patients, and by subclavian flap in 5. In all of them the aortic arches were recon-

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Table 1 Clinical features of study population

	Normotensive patients postcoartectomy (n = 26)	Control subjects (n = 24)	*P*
Age (y) mean ± SD (range)	29 ± 17 (19-58)	26 ± 18 (17-53)	.8
Male/female (n)	15/11	14/10	.5
BP at rest (right arm)			
Systolic BP (mm Hg)	110 ± 15 (100-135)	105 ± 15 (95-130)	.4
Diastolic BP (mm Hg)	70 ± 15 (65-85)	65 ± 10 (60-85)	.2
Pulse pressure (mm Hg)	45 ± 5 (25-50)	40 ± 7 (25-45)	.5
Blood pressure after exercise (right arm)			
Systolic BP (mm Hg)	180 ± 20 (175-230)	145 ± 15 (140-190)	.001
Diastolic BP (mm Hg)	85 ± 15 (80-115)	75 ± 10 (80-95)	.01
Pulse pressure (mm Hg)	65 ± 5 (35-85)	55 ± 5 (25-55)	.01
Biochemistry			
Glucose (mmol/L)	4.4 ± 0.5	4.6 ± 0.4	.7
Cholesterol (mmol/L)	4.03 ± 0.9	4.01 ± 0.7	.5
Triglycerides (mmol/L)	0.97 ± 0.3	0.86 ± 0.4	.2
HDL (mmol/L)	1.39 ± 0.6	1.46 ± 0.4	.6
LDL (mmol/L)	2.1 ± 1.3	2.2 ± 1.2	.5
Surgery			
Age at operation (mo)	16.3 ± 29.1 (2-33)		
Time since surgery on first examination (y)	21.4 ± 6.2 (15-39)		
Type of surgery			
End-to-end anastomosis (n)	21		
Subclavian flap angioplasty (n)	5		

BP, Blood pressure; *HDL*, high-density lipoprotein; *LDL*, low-density lipoprotein.

structed using their native tissues. Clinical and echocardiographic evidence of successful repair was defined as normal femoral pulses, right arm-leg systolic BP gradient at rest less than 15 mm Hg, and systolic peak flow velocity less than 3 m/s without diastolic flow in the descending aorta. Cardiovascular magnetic resonance (CMR) imaging was performed in 19 patients. All patients were in New York Heart Association class I and were not taking any antihypertensive medication. None of them were diagnosed as having Turner syndrome. Patients who had associated heart defects were excluded, as were patients with an aberrant right subclavian artery. Four patients with a bicuspid aortic valve unassociated with significant valvar steno-insufficiency or pathology of the ascending aorta (AAo) were included. A total of 24 age- and sex-matched individuals with no signs of heart disease were selected as healthy control subjects. Ethical approval was obtained.

Echocardiography

All patients underwent a complete clinical examination and transthoracic echocardiography (TTE) and (after given written informed consent) transesophageal echocardiography (TEE) for the assessment of LV mass and for confirmation of the absence of recoarctation, aortic aneurysm, and functional abnormalities of the mitral and aortic valves. All echocardiographic findings were analyzed by one of the authors who was blinded to patient histories.

The AAo and descending aorta was imaged by TEE in an optimized circular plane. Diameters were measured in the AAo at the level of pulmonary artery bifurcation (2-3 cm above the aortic valve) and in the descending thoracic aorta just distal to the branching site of the left subclavian artery. M-mode diameter measurements (Figure 1, *A*) were made in systole (point of maximal anterior motion of aorta) and at end diastole (q wave on electrocardiogram). The means of 5 diameter measurements in sequential cardiac cycles were used for data analysis. Systemic arterial BP was measured at the right brachial artery by sphygmomanometry with the patient supine using an adequately sized cuff. BP was measured 5 times on each occasion at 2-minute intervals and averaged. Pulse pressure was obtained by subtracting the diastolic BP from the systolic BP.

The elastic properties of the aorta were indexed by calculation[3,10] of aortic distensibility (D), stiffness index (β), and pressure-strain elastic modulus (E_p) as: $D = 2(A_s - A_d)/[A_d (P_s - P_d)]$, $\beta = \ln(P_s/P_d)/(A_s - A_d)/A_d$, and $E_p = (P_s - P_d)/[(A_s - A_d)/A_d]$, respectively, where A_s = aortic diameter at end systole, A_d = aortic diameter at end diastole, P_s = systolic BP, P_d = diastolic BP, and ln = natural logarithm.

Measurements of cardiac chambers were made by TTE according to established criteria.[11] Fractional shortening, ejection fraction by modified Simpson method, and LV mass index (LVMI) were estimated.[11-13] Peak early and late diastolic velocities, deceleration time, LV isovolumic relaxation time, and pulmonary venous Doppler recording were obtained from apical 4-chamber view using standard Doppler practices.[14,15]

Tissue Doppler Echocardiography

The general principles that underlie the DTI modalities have been described previously.[9] By using transesophageal cross-sectional views, the thoracic aortic wall-motion velocities were assessed during the cardiac cycle. Two-dimensional tissue velocity images of the aortic wall were obtained at 130 ± 15 frames/s, which implies a temporal resolution of approximately 16 milliseconds. The velocity scale was modified to avoid aliasing. To minimize noise, the pulse repetition frequency was set to 0.5 to 1.0 kHz. A sample volume was placed within the region of interest so that there was no migration beyond the limits of the selected wall. Samples varied from 2 to 4 mm, depending on the size of the region of interest and looking for a compromise between maximizing signal-to-noise ratio (large samples) or spatial resolution (small samples). By marking a region of interest on the 2-dimensional image in the anterior aspect of the aorta (Figure 1, *B*) at the same point as in M-mode measurements, velocities

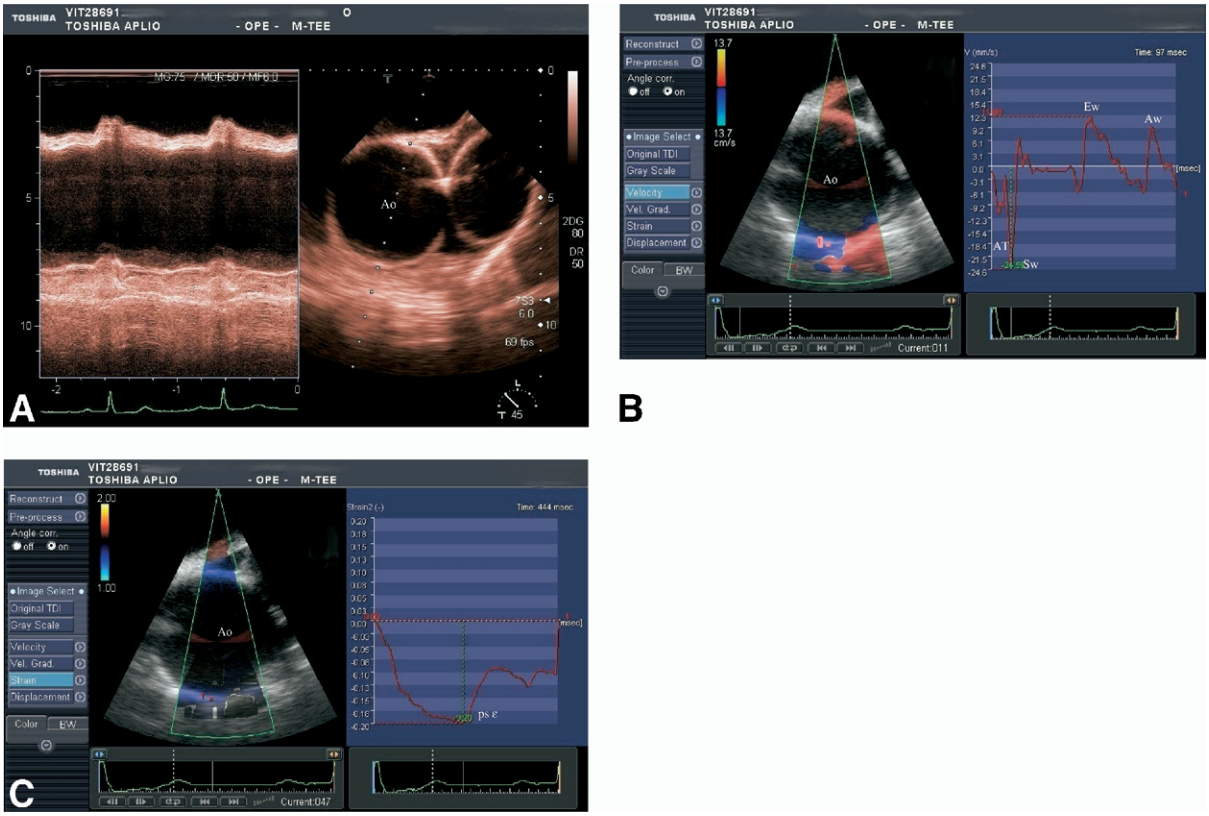

Figure 1 Two-dimensional (2D), M-mode, and Doppler tissue images from transesophageal aortic (*Ao*) short-axis views. (**A**), M-mode 2D-guided Ao tracing. (**B**), Velocity profile from anterior Ao wall site. Systolic and early diastolic induced velocities are shown. (**C**), Strain profile from same sample site. *AT*, Acceleration time; A_w, wall late diastolic velocity; E_w, wall early diastolic velocity; *ps* ε, wall peak systolic strain; S_w, wall systolic velocity. Color figure online.

throughout the cardiac cycle for this area can be determined. Offline analysis of the velocity data sets was performed using dedicated software (Toshiba Corp, Tokyo, Japan). DTI tracing displayed accelerated expansion of the aortic wall followed by a slow deceleration, a plateau, and then a rapid deceleration into diastole. This trace represents the mean of the instantaneous velocity spectrum. Acceleration time (milliseconds), systolic maximum wall expansion velocity (S_w) (cm/s), wall contraction E_w and A_w diastolic velocities (cm/s), and wall peak systolic strain (ps ε) (%) were derived.

Velocity and strain traces were processed from the same wall site in transverse aortic views (Figure 1, *B* and *C*). Circumferential strain (change in length per unit length) in each segment was defined[6] and represented as peak systolic strain. From tissue Doppler data, incremental strain rate can be estimated by calculating the velocity gradient. The time integral of incremental strain rate yields logarithmic strain: e = log(L/L0). In this study, the logarithmic strain was converted to Lagrangian strain: $\varepsilon = (L - L0)/L0$. The velocity gradient was estimated between two points with an offset distance (strain length) of 3 mm. This spatial offset was lower than the optimal intersite distance required for ventricular strain measurements and selected as a compromise between acceptable signal-to-noise ratio and longitudinal spatial resolution.

The motion of the aortic wall has been automatically tracked throughout the cardiac cycle to be sure we were measuring changes within the vessel wall and avoiding the partial volume effect of endoluminal blood pool.

The same DTI modalities were used to assess aortic wall properties in the AAo by TTE. Velocity and strain traces were processed from the same wall site in longitudinal parasternal aortic views at a level 3 cm above the aortic valve and the above-described measurements were obtained.

Lateral mitral annulus velocities (S_a, E_a, A_a) were also measured on the transthoracic 4-chamber views.

Exercise Testing

Patients underwent a bicycle exercise test the following day to TTE/TEE examination, and BP was again acquired in a supine position before and immediately after the test. All patients underwent a maximal, symptom-limited exercise test, after a standardized supine bicycle ergometry protocol. BP was measured by conventional sphygmomanometry. Patients with an exercise maximal systolic BP greater than or equal to 200 mm Hg were considered to have exercise-induced hypertension.[16]

Statistics

Data are presented as mean value ± SD. Linear correlations, and univariate and multivariate analysis were used for comparisons. Comparisons between different regions were analyzed with a paired Student *t* test and differences were considered statistically significant when the *P* value was less than .05. To test intraobserver variability, measurements of systolic and diastolic DTI were made at 50 sites in different patients on two different occasions. For interobserver variability, a second investiga-

Table 2 Left ventricular and aortic findings in normotensive patients postcoartectomy and control subjects

Parameters	Patients (n = 26), mean ± SD	Control subjects (n = 24), mean ± SD	*P* values
Left ventricle			
LVFS	0.34 ± 0.1	0.35 ± 0.2	NS*
LVEF	0.61 ± 0.8	0.59 ± 0.9	NS*
LVMI (g/m^2)	128 ± 7	106 ± 5	<.05*
LVIRT (ms)	44.1 ± 4.2	41.8 ± 2.3	NS*
LVDT (ms)	143 ± 22	136 ± 17	NS*
PVSFF	0.43 ± 0.11	0.51 ± 0.19	NS*
E/A	1.9 ± 0.7	2.2 ± 0.9	NS*
E_a (cm/s)	7.3 ± 2.2	12.7 ± 2.1	<.001*
E_a/A_a	0.8 ± 0.3	1.9 ± 0.4	<.001*
Aorta			
AAo β stiffness index	4.11 ± 2.51	2.97 ± 0.65	<.05*
AAo elastic modulus (kPa)	47.6 ± 6.5	25.8 ± 2.9	<.05*
AAo distensibility (m^2/N)	48 ± 14	71 ± 16	<.05*
AAo S_w velocity (cm/s)	4.1 ± 0.5	6.9 ± 0.9	<.001*
AAo ps ε (%)	7 ± 1.4	23 ± 2.5	<.0001*
AAo AT (ms)	36.9 ± 6.8	24.2 ± 5.6	<.05*

A, Mitral inflow late diastolic velocity; A_a, annular late diastolic velocity; *AAo*, ascending aorta; *AT*, acceleration time; *E*, mitral inflow early diastolic velocity; E_a, annular early diastolic velocity; *LVDT*, left ventricular deceleration time; *LVEF*, left ventricular ejection fraction; *LVFS*, left ventricular fractional shortening; *LVIRT*, left ventricular isovolumic relaxation time; *LVMI*, left ventricular mass index; *NS*, not significant; *ps* ε, wall peak systolic strain; *PVSFF*, pulmonary vein systolic filling fraction; S_w, wall systolic.
*Similar significance levels were obtained after excluding from analysis 4 patients with bicuspid aortic valves, 5 patients with subclavian flap repair, or 7 patients with no cardiovascular magnetic resonance confirmation.

tor randomly made measurements at the above different sites without knowledge of other echocardiographic parameters. The intraobserver and interobserver variabilities were determined (as the difference between the two sets of observations divided by the mean of the observations and expressed as a percentage).

RESULTS

Of 27 patients with CoA initially evaluated, 26 were included in the study. Although great care was taken to ensure the quality of the data collected, 3.7% of all aortic segments had to be excluded from analysis as the velocity/strain traces were defined to be noninterpretable. Intraobserver variation ranged from 3% to 7% and interobserver variation ranged from 4% to 9%. The patient demographic data are given in Table 1. The main echocardiographic features in the control and CoA groups are compared in Table 2.

None of the patients had a difference in BP between upper and lower extremities greater than or equal to 15 mm Hg or a diastolic runoff on spectral Doppler examination. The 19 patients who underwent magnetic resonance imaging had a site of repair/diaphragmatic ratio greater than 0.7, showing no significant residual coarctation in agreement with previous reports.[17,18]

Correlation Between DTI-derived and Standard M-Mode–derived Indexes of Aortic Function

The relationship between DTI measurements and aortic stiffness and distensibility was tested. Univariate correlations in the coarctoplasty group showed significant inverse relationships of β and S_w velocities ($r = -0.66$, $P < .01$) and β and ps ε ($r = -0.71$, $P < .001$). Aortic distensibility was positively related to S_w velocities ($r = 0.57$, $P < .01$) and ps ε ($r = 0.59$, $P < .01$). Elastic modulus was negatively related to S_w velocities ($r = -0.51$, $P < .05$) and ps ε ($r = -0.57$, $P < .01$).

Compared with control subjects, in the coarctoplasty patients there was a significant increase of Ep (47.6 ± 6.5 vs 25.8 ± 2.9 kPa, $P < .05$) and β (4.11 ± 2.51 vs 2.97 ± 0.65, $P < .05$) and significant decrease of D (48 ± 14 vs 71 ± 16 m^2/N, $P < .05$) at the level of the segments proximal to the repair site (Figure 2, *A*).

At the same level S_w velocities and ps ε were decreased with higher significance (Figure 2, *B*, and Table 2) compared with control subjects (S_w = 4.1 ± 0.5 vs 6.9 ± 0.9 cm/s, $P < .001$; ps ε = 7 ± 1.4% vs 23 ± 2.5%, $P < .0001$). A significant increase of acceleration time (36.9 ± 6.8 vs 24.2 ± 5.6 milliseconds, $P < .05$) was also shown.

No significant difference was shown between M-mode–derived and DTI-derived indexes of aortic function of patients and control subjects at the level of the segments distal to the repair site (Figure 2, *B*).

The exclusion of the 4 patients who had bicuspid aortic valves did not significantly affect the results (Table 2) nor did the exclusion of the 7 patients who did not have CMR confirmation. The exclusion of the 5 patients who had a different repair from the whole group (subclavian flap angioplasty) did not significantly affect the results (Table 2).

A significant correlation was shown between DTI AAo values determined by TEE and those determined by TTE (S_w: $r = 0.81$, $P < .005$; ps ε: $r = 0.84$, $P < .001$; acceleration time: $r = 0.61$, $P < .01$).

Correlation Between DTI-derived Indexes of Aortic Function and BP After Exercise

There was no significant difference between resting BP values acquired before TTE/TEE examination and those acquired the next day before the exercise test. Both peak systolic BP after exercise and pulse pressure after exercise were directly related to AAo wall strain (Figure 3).

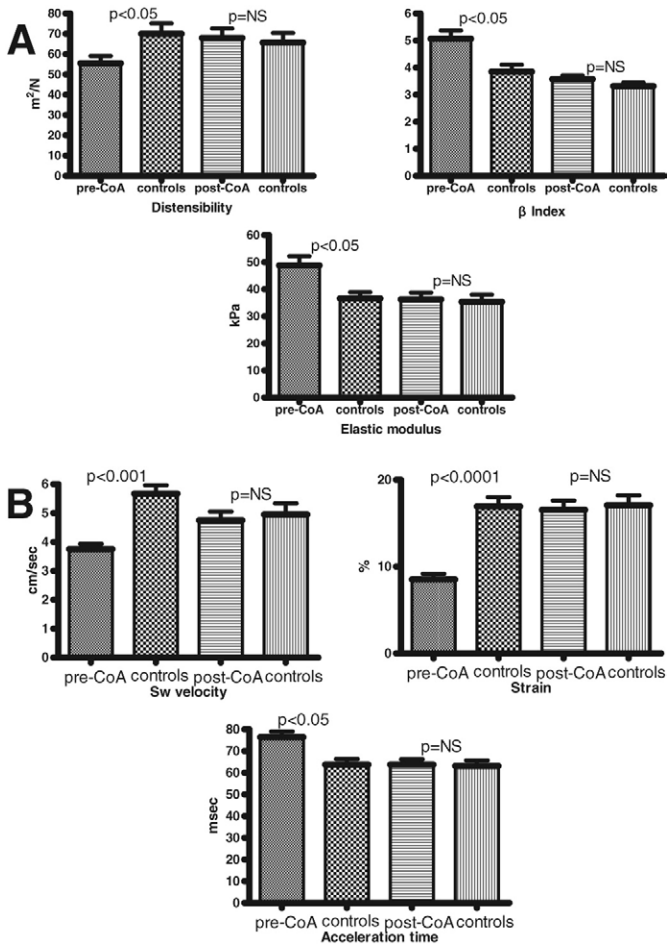

Figure 2 M-mode–derived (**A**), and Doppler tissue imaging–derived (**B**), values in patients after coarctoplasty and in control subjects. *NS*, Not significant; *pre-CoA*, aorta proximal to repair site; *post-CoA*, aorta distal to repair site; S_w, wall systolic.

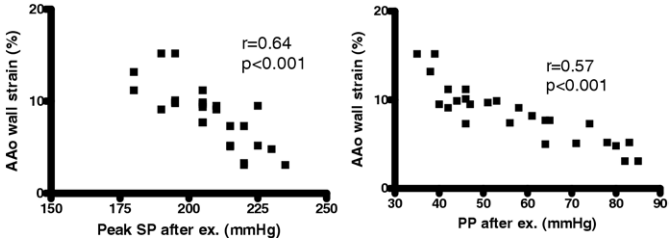

Figure 3 Regression plot showing correlation between ascending aorta (*AAo*) wall strain and peak systolic blood pressure (*SP*) and pulse pressure (*PP*) after exercise (*ex*).

There was a direct relationship between age and AAo wall strain in patients after aortic arch repair but no relationship was shown in the descending aorta data (Figure 4).

Correlation Among DTI-derived Indexes of Aortic Function, LV Mass, and LV Diastolic Function

LVMI was related to peak systolic BP ($r = 0.38$, $P < .05$), pulse pressure after exercise ($r = 0.41$, $P < .05$), and AAo stiffness index ($r = 0.42$, $P < .05$) and inversely related to AAo S_w velocity ($r = -0.44$, $P < .01$) and wall strain ($r = -0.51$, $P < .01$). A significant negative

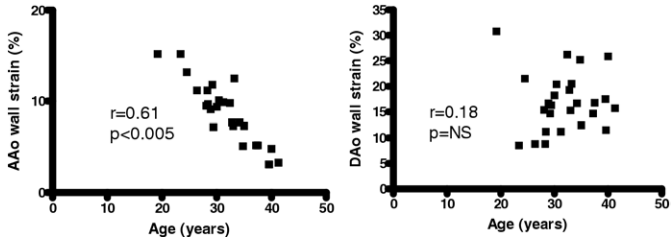

Figure 4 Relationship between ascending aorta (*AAo*) and descending aorta (*DAo*) wall strain and age. Direct relationship is shown between age and AAo wall strain but no relationship in DAo data.*NS*, Not significant.

correlation existed between LVMI and the early diastolic myocardial velocity from annular site ($r = -0.72$, $P < .001$). LV annular early diastolic velocity was significantly reduced compared with control subjects (Table 2) and related to AAo S_w velocity ($r = 0.53$; $P < .005$) and strain ($r = 0.59$; $P < .001$). In univariate regression analysis, there was a positive correlation between LVMI and age at surgery, age at time of the study, height, weight, and exercise-induced hypertension. In multiple linear regression analysis (Table 3), only weight, study group, and AAo wall strain were correlated to LVMI.

DISCUSSION

Our study shows that adult patients who had successful repair of CoA in infancy still have increased proximal aortic stiffness and reduced wall velocities and wall strain.

Strain represents the fractional or percentages change from the original or unstressed dimension and equals the relative change of segmental length occurring between the reference state and the state of deformation expressed in percentage of end-systolic aortic wall length. Radial, circumferential, and axial components of arterial strain have been reported.[10,19,20] A pressure-strain elastic modulus for the arterial wall has been described as has a relationship between elastic modulus and pulse wave velocity.[10]

Tissue Doppler echocardiography has made possible to measure ventricular and arterial strain on the basis of velocity wall gradients. It has been shown[6] that the strain Doppler technique is a more sensitive method for detecting regional ischemic myocardial wall-motion abnormalities because it excludes the tethering effect of adjacent myocardial tissue. Our results indicate that the decreased vessel wall circumferential strain measured by Doppler may be an important supplement to the assessment of the elastic properties of thoracic aorta and even more accurate compared with wall velocities presumably because the systolic deformation is more uniformly distributed in all aortic segments.

We studied aortic stiffness and distensibility with a method accepted in the literature and provided their relationship with DTI-SRI measurements. The aortic wall systolic velocity and strain showed a negative correlation with the increase in stiffness index and a positive correlation with distensibility. Compared with stiffness index and distensibility, DTI-SRI values in the AAo had a higher discriminating power in differentiating patients from control subjects. Plus, we have shown in a previous report[9] that the assessment of these parameters is particularly relevant in the follow-up of patients with Marfan disease because aortic dissection may occur in the absence of marked aortic root dilation and that aortic diameter was less predictive of dissection compared with DTI-SRI measurements. Positive results validating DTI assessment of arterial wall properties have also been

Table 3 Associations of left ventricular mass analyzed by multiple regression analysis in normotensive patients postcoartectomy

	Regression coefficient	SE	95% CI	*P*
Age at operation	0.47	0.32	0.12-1.23	.4
Age	−0.08	0.11	−0.22-0.10	.6
Weight	0.32	0.21	0.28-0.69	.005
Patients (26) vs control subjects (24)	−21.4	0.34	−33.6-12.6	.01
AAo wall strain	−0.92	0.23	−1.72-0.21	.03

AAo, Ascending aorta; *CI*, confidence interval.
Units for regression coefficient are g/U change in explanatory variable. $R^2 = 0.55$.

obtained in the evaluation of abdominal aorta in children with Marfan disease[8] and characterization of common carotid artery stiffness in healthy adults.[21,22]

The novel findings in the current study include the demonstration of the value of strain/DTI echocardiography in assessing aortic wall mechanics after surgical repair of coarctation and demonstration of the relation of abnormal aortic wall properties as determined by strain/DTI to abnormal responses for BP at peak exercise, which results in LV pressure overload.

The pathophysiology of late hypertension in postrepair coarctation is still controversial and probably multifactorial.[16] Residual narrowing at the anastomosis site, structural abnormalities of the arterial wall, abnormal shape of the aortic arch,[23] and resetting of the sympathetic nervous and/or renin-angiotensin system are potential mechanisms that might influence BP at rest and after exercise. In a recent report[18] it has been pointed out that the definition of recoarctation is variable and a threshold for reintervention of residual aortic narrowing may be lower than stated in recently described guidelines (resting arm/leg BP gradient ≥ 30 mm Hg). On the basis of the criteria we used to define significant recoarctation, none of our patients had right arm-leg systolic BP gradient at rest greater than or equal to 15 mm Hg. We obtained no association between Doppler velocities in the aortic arch, anatomic abnormalities, or type of repair and any measurement of arterial function and this suggests that our findings are not related to minor degrees of residual coarctation or recoarctation.

The arterial system appears divided in two parts with respect to wall velocities and strain. Wall strain in AAo before the reconstructed site of the aortic arch is lower than wall strain in descending aorta. A dynamic narrowing, caused by differences in the elastic properties proximal and distal to the repair site, could cause exercise-induced arm-leg pressure gradients, high exercise pulse pressures, and increased stiffness in the pre-CoA even in normotensive patients at rest. This may be one of the causes of postrepair arterial hypertension and subsequent cardiovascular complications. BP could function both as cause and effect: increased arterial stiffness and decreased distensibility increase systolic BP, whereas increased BP contributes to decreased arterial elastic properties. In other words, reduced arterial strain and elevated BP may be mutually causally related. However, the potential of aortic strain as reliable predictor of the risk of progression to hypertension in these patients still needs further investigation.

LV mass is another important predictor of cardiovascular events in the general population, independent of BP. There are several possible explanations for the increased LV mass in postrepair CoA.[24] Increase might be an adaptation of hemodynamic changes in arterial pressure wave and/or decrease in aortic wall strain. It might also be determined by hormonal differences that persist even after successful repair such as resetting of the sympathetic nervous and/or renin-angiotensin system. It has also been hypothesized as a genetically determined hypertrophic response in CoA.[24] In our postrepair normotensive patients LVMI was found to be significantly and positively correlated to peak systolic BP after exercise and inversely related to aortic wall strain. This suggests that reduced wall strain could act either directly or indirectly through higher daytime systolic BP as manifest by higher exercise BP. None of our patients had long-term preoperative hypertension/increased afterload justifying increased LV mass because all of them had surgery in infancy and early childhood.

Increased LV mass and parallel decrease in LV annular early diastolic velocity suggest LV diastolic dysfunction. This finding is consistent with previous studies[25] showing that elevation of LV filling pressures is an early abnormality in the progression of hypertensive heart disease. Measurement of DTI-derived E_a velocities appeared[25] as a robust technique for the assessment of LV relaxation and relatively load-independent compared with pulsed Doppler–derived E/A ratio and deceleration time intervals. Alterations of diastolic function may precede a significant increase in LV mass in patients at risk of essential hypertension.[26] Other studies have shown that in hypertensive heart disease arterial compliance is an independent predictor of LV diastolic dysfunction as assessed by DTI,[27] suggesting a possible interaction between arterial compliance and diastolic heart failure. Although a causal relation was not investigated in our study, the association of decreased arterial strain and DTI parameters of diastolic dysfunction in patients postrepair CoA supports a mechanistic link between these two pathophysiologic entities.

The increase in LV mass is likely to evolve with age but its potential to progress to high-risk levels requires a serial longitudinal assessment. No attempt was done in our study to connect these pathophysiologic changes to clinical outcome. However, the adverse impact of BP elevation and LV mass increase in the general population are likely to be amplified in patients with CoA because of the summation of the effects of structural wall abnormalities to age-related changes in arterial stiffness reported in prehypertensive individuals and linked to progression to hypertension.[28] Thus, patients postrepair CoA need close long-term surveillance and tailored therapy, especially in the presence of low values of aortic wall strain and high levels of exercise hypertension.

In our series, we used both DTI TTE and TEE, together with noninvasive BP measurement, to evaluate aortic wall properties. We obtained comparable results in the assessment of AAo wall mechanics. The advantage of TTE method is its noninvasive nature suitable for serial measurements. However, TTE visualization of the aorta is limited to the AAo and parts of the aortic arch and a reproducible short-axis plane of the AAo and an adequate visualization of the descending thoracic aorta cannot usually be achieved. The advantage of TEE method, although semi-invasive, is that it can be used both for the assessment of morphologic complications of CoA (re-coarctation, aneurysm, dissection) and individual long-term follow-up of aortic

wall function as it has previously been reported in patients with Marfan syndrome.[9] Furthermore, in the adult patient, such studies may be obtained without the use of general anesthesia providing more appeal than in the child or adolescent for whom anesthesia is almost always necessary. However, the availability of magnetic resonance imaging to follow morphologic changes should be considered and a cost/benefit analysis will be required of these two techniques in future studies.

Limitations of the Study

This study has some limitations. First, technical limitations of strain Doppler echocardiography should be considered. Strain measurements are angle-dependent deformation, therefore, interpretations of strains should be performed with caution if tissue direction deviates more than 30 degrees from the beam direction. Second, we included 4 patients with bicuspid aortic valve who might have influenced our analysis. However, our patients with bicuspid valves had no functional abnormalities and some studies[3] have shown that in patients with CoA the presence of a bicuspid aortic valve had no independent effect on BP, LV mass, or artery function. Moreover, in our study the exclusion of the 4 patients who had bicuspid aortic valves did not significantly affect the results. Third, the act of surgery itself may have altered the mechanical properties of the aorta. However, in all our patients the aortic arches were reconstructed using their native tissues and other studies[4] analyzing aortic wall properties with alternative techniques have described coarctation as a systemic vascular disease of the precoarctational arteries and shown that elastic properties of descending aorta did not differ preoperatively or postoperatively compared with those in healthy subjects. Furthermore, the small group of 5 patients with subclavian flap repair and a different type of scar has been analyzed separately and their exclusion did not significantly impact the results. Fourth, we did not perform CMR and/or aortogram in all patients and could have missed some minor structural stenosis. However, in the 19 patients who underwent CMR we did not find any anatomic abnormalities, thus corroborating our spectral Doppler data, and the exclusion from the analysis of the patients with no CMR confirmation did not modify our results. Furthermore, we adhered to strict criteria of successful repair. Even if pressure gradient alone as an index of aortic narrowing is often inadequate, in previous reports[17,29] Doppler diastolic velocities and pressure decays had excellent sensitivity and specificity in assessing coarctation severity as defined by CMR imaging and the presence and degree of antegrade diastolic flow (diastolic runoff) in the descending thoracic aorta has been shown highly specific for a haemodynamically significant recoarctation. Finally, the small size of the study could be problematic for the regression analysis among patients and tracking data would be helpful over a prolonged period in a large group of patients to confirm our preliminary observations.

Conclusions

Adult patients with CoA have reduced AAo wall strain and increased stiffness even after successful repair in infancy. This amplifies stress-induced hypertension and increases LV burden suggesting a careful clinical postoperative surveillance. However, a long-term tracking study is needed to clarify the relationship between the reduced aortic wall strain and the occurrence of cardiovascular diseases in these patients.

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