

Are There Surgical Implications to Aortic Root Motion?

Carsten J. Beller¹, Michel R. Labrosse², Mano J. Thubrikar²,
Gabor Szabo¹, Francis Robicsek², Siegfried Hagl¹

¹Department of Cardiac Surgery, University Hospital Heidelberg, Heidelberg, Germany, ²Heineman Medical Research Laboratory, Carolinas Medical Center, Charlotte, NC, USA

Background and aim of the study: By increasing the longitudinal stress in the ascending aorta, downward movement of the aortic root might promote the proximal transverse tears seen in aortic dissections. The study aim was to evaluate the influence of five common cardiac conditions on the magnitude of aortic root displacement in cardiac patients.

Methods: Aortic root contrast injections were analyzed in 90 patients (mean age 68 years) to measure downward motion of the root perpendicular to the plane of the sinotubular junction (STJ).

Results: Displacement of the aortic root ranged from 0 to 14 mm (mean 4.8 mm). Patients with aortic insufficiency (AI) showed increased aortic root movement (7.3 versus 4.3 mm, $p = 0.003$), whereas those with left ventricular hypokinesia (3.7 versus 5.5 mm, $p = 0.014$) or with myocardial hypertrophy (3.8 versus 5.1 mm, p

$= 0.073$) exhibited reduced downward movement. These variables were independent, and correlated with the magnitude of aortic root motion. A stress analysis of the aortic root, arch and branches of the arch determined that the longitudinal stress approximately 2 cm above the STJ, in the outer curve of the aorta, was increased by 32% in patients with AI compared to patients without AI.

Conclusion: Patients with cardiac conditions associated with increased aortic root motion such as AI may be at greater risk of aortic dissection because of increased longitudinal stress in the ascending aorta. Therefore, AI should be used as an indicator and aortic root displacement monitored to prevent the risk of aortic dissection.

The Journal of Heart Valve Disease 2005;14:610-615

A majority of aortic dissections occur a few centimeters above the aortic valve, with a transverse intimal tear (1). Abnormalities in the aortic wall probably promote dissections, but similar changes have been reported with normal aging and may not alone cause dissections (2,3). Hypertension and aortic dilatation are other well-recognized risk factors for dissection, because the mechanical stress in the aortic wall is proportional to the blood pressure and the vessel diameter. However, all of these reasons fail to explain the common transverse orientation and proximal location of the tear observed in aortic dissections.

Cinematographic and contrast injection studies, on the other hand, have documented that the aortic root is displaced downward during systole and returns to its previous position in diastole (4). More specifically,

cardio-magnetic resonance imaging (MRI) studies reported an average 8.9 mm axial downward motion and 6° clockwise axial twist during systole in healthy subjects (5,6). In a recent finite element study of the aortic root, aortic arch and supra-aortic vessels, it was found that the aortic root motion could significantly increase the mechanical stress present in the aortic wall, possibly determining both the tear location and orientation observed in aortic dissections (7). Based on the laws of physics, such an explanation clearly identified aortic root motion as an additional risk factor for aortic dissection.

Despite these findings, those clinicians whose aim it is to prevent aortic dissection from occurring require more concrete information. To help in this respect, as a first step, the goal of the present study was to identify those patients who might be at a higher risk of aortic dissection due to the magnitude of their aortic root motion. Therefore, aortic root motion was measured in a large series of patients and analyzed to determine which common cardiac conditions (namely aortic insufficiency, aortic stenosis, left ventricular hypokinesia

Address for correspondence:
Dr. Michel Labrosse, Department of Mechanical Engineering,
University of Ottawa, Ottawa, Ontario, Canada K1N 6N5
e-mail: labrosse@eng.uottawa.ca

sis, myocardial hypertrophy and previous cardiac operations) might be independent factors predicting the magnitude of aortic root motion. The influence of these predictors on the mechanical stress in the aortic wall was then established by finite element analysis.

Materials and methods

Measurement of aortic root motion

Aortic root contrast injections recorded on 35-mm cine films and compact discs were analyzed in 90 cardiac patients (mean age 68 years; range: 43 to 88 years). Cardiac conditions identified among patients are listed in Table I. None of the patients had aortic dissection, and 26 were examined for elective coronary artery bypass grafting (CABG) but showed none of the listed conditions.

Most aortograms were recorded with the left anterior oblique projection, though some were in the right anterior oblique view. The aortograms were analyzed frame by frame, and the aortic root outlines in the most upward and downward positions were traced on a transparency. The base of two sinuses and the sinotubular junction (STJ) were marked. The outline of the 2-mm diameter angiocatheter present in the field was also traced for distance calibration. The distances between the marked points were determined using image analysis software (Image-Pro; Media Cybernetics). The actual-size downward motion (axial displacement) of the aortic root perpendicular to the plane of the sinotubular junction was measured in millimeters.

Statistical analysis

Correlations between the patients' parameters and the magnitude of aortic root motion were investigated using univariate and multivariate techniques. The Wilcoxon rank sum test was used to determine the presence of which cardiac conditions influenced aortic root motion, at the 0.05 significance level. A linear multivariate model was then established based on the conditions associated with a trend or a significant difference in the aortic root motion. The graded values of aortic stenosis (AS) and aortic insufficiency (AI) were examined using Spearman's correlation coefficient.

Stress analysis

To illustrate the mechanical stress associated with different levels of aortic root displacements, a finite element model of the human aortic root, aortic arch and supra-aortic vessels was used. The model has been described extensively and validated elsewhere (7). The distal ends of the supra-aortic vessels and the aorta were fixed in all directions to allow physiological

deformation of the model.

The twist of the aortic root base was not included because it was shown to have a minor influence on the stress experienced by the aortic wall (7). A luminal pressure of 120 mmHg was applied in combination with axial displacement of the aortic root. In contrast to the comparative stress analysis detailed previously (7), the values of displacement were taken as the average values obtained from the statistical analysis of the measurements in the patients with AI and without AI, respectively.

Results

Magnitude of aortic root motion in patients

The mean downward axial displacement of the aortic root during the cardiac cycle was 4.8 mm (range: 0 to 14 mm). The aortic root motion was between 0 and 7 mm in 73 of the patients (81%) (Table I).

Effect of cardiac pathology on aortic root movement

Root movement in patients with AI was significantly greater than in those without AI (7.3 mm versus 4.3 mm, $p = 0.003$) (Table II).

Root movement in patients with left ventricular hypokinesia (HKI) was significantly reduced to 3.7 mm, compared to 5.5 mm in patients without hypokinesia ($p = 0.014$) (Table II).

The effect of myocardial hypertrophy was unclear, but presence of the condition tended to reduce aortic root movement compared to patients without hypertrophy (3.8 mm versus 5.1 mm, $p = 0.073$) (Table II).

Although previous cardiac operations may have been thought to reduce aortic root motion due to fibrotic adhesions, they were found not to play any significant role on the magnitude of aortic root motion, and neither did aortic stenosis (Table II).

A linear multivariate analysis established that grade of AI, HKI and myocardial hypertrophy (HTR) were independent variables that correlated with the magnitude of the aortic root axial displacement (DISP), such that:

$$\text{DISP (mm)} = 5.379 (p < 0.0001) + 1.186 \times \text{AI grade} (p = 0.0016) - 1.611 \times \text{HKI} (p = 0.0078) - 1.399 \times \text{HTR} (p = 0.0355), \text{ with } R^2 = 0.23.$$

This equation was based on the assumption that the distribution of DISP was normal. The results were confirmed when \log_{10} (DISP) was studied, making the assumption unnecessary.

Stress analysis

The magnitude of the deformation due to aortic root motion decreases distally as it affects the ascending

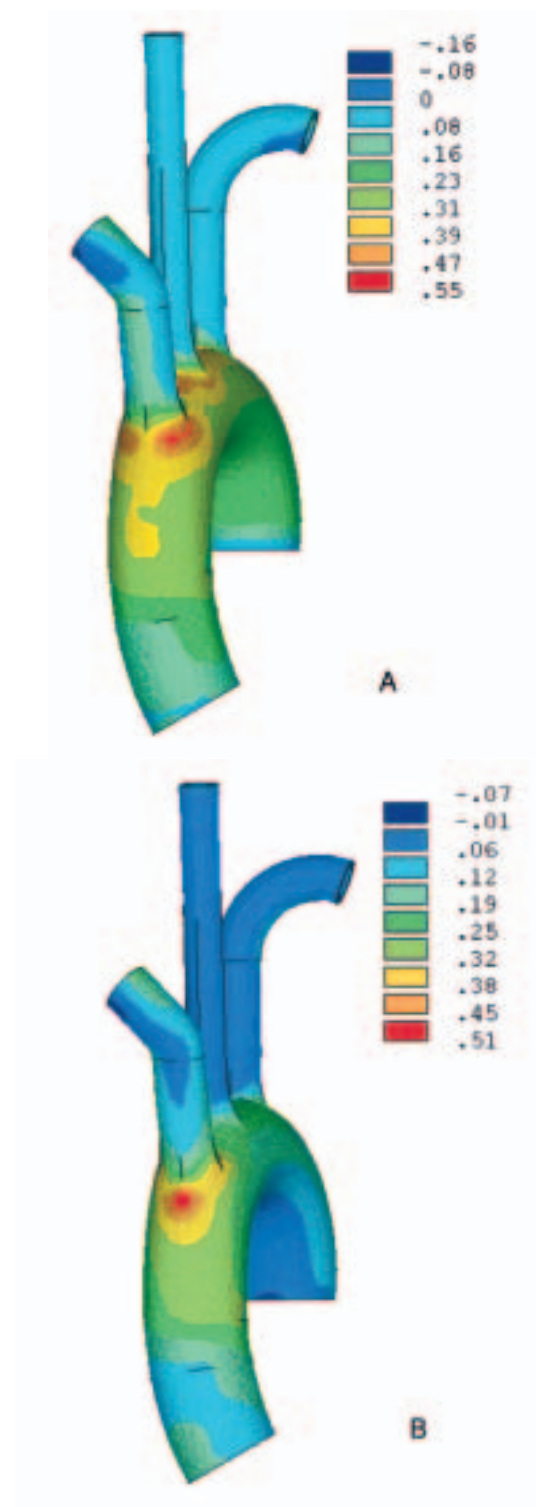
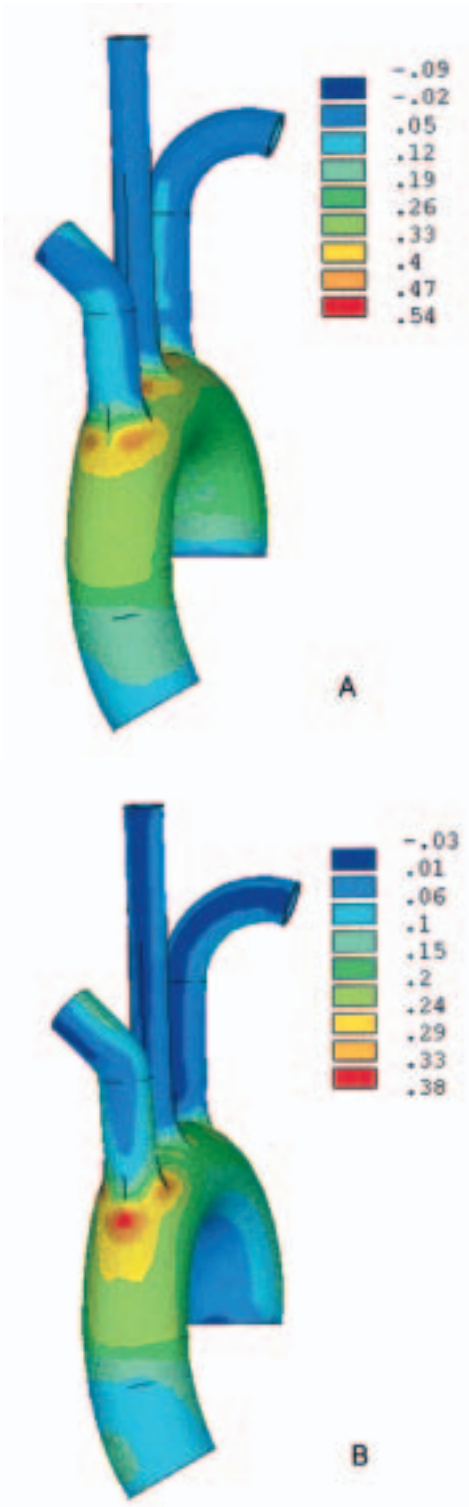


Figure 1: Distribution of circumferential (A) and longitudinal (B) stresses (MPa) in the aortic arch under 120 mmHg luminal pressure. In this control model, the measured average 4.3 mm axial displacement of the aortic root was applied to represent patients without aortic insufficiency. Expected stress concentrations around the ostia of the supra-aortic vessels were observed.

Figure 2: Same as Fig. 1, but with 7.3 mm axial displacement applied to the aortic root to represent the average measurements in patients with aortic insufficiency. The longitudinal stress in the outer curve of the ascending aorta was increased by 32% compared to the control model.

Table I: Measured aortic root axial displacement (DISP) in patients, and listed cardiac conditions.

DISP (mm)	No. of patients	AI count*	HKI count	HTR count	CABG count	AS count*
0-1	9	1 (1/0)	6	3	2	3 (0/3)
2-3	28	2 (2/0)	11	10	6	9 (1/8)
4-5	23	2 (0/2)	13	6	2	7 (2/5)
6-7	13	2 (1/1)	3	2	1	1 (0/1)
8-9	8	2 (2/0)	2	0	1	2 (0/2)
10-11	7	5 (4/1)	1	2	0	2 (1/1)
12-13	1	0 (0/0)	0	0	0	0 (0/0)
14-15	1	1 (1/0)	0	0	1	1 (0/1)
Total	90	15 (11/4)	36	23	13	25 (4/21)

*Partial counts of AI and AS are indicated in parentheses per severity range (I-II/III-IV).

AI: Aortic insufficiency; AS: Aortic stenosis; CABG: Previous coronary artery bypass grafting; HKI: Left-ventricular hypokinesis; HTR: Myocardial hypertrophy.

aorta, the transverse aortic arch, and the supra-aortic vessels (7). The average stress across the vessel wall is illustrated diagrammatically in Figures 1 and 2. The specification of orientation is restricted to the region of interest, including the aortic arch and the ascending aorta.

Stresses with aortic root motion in patients without AI

The results for mechanical stress in the control model subjected to 120 mmHg pressure and 4.3 mm axial displacement of the aortic root are shown in Figure 1. Stress concentrations were present at the ostia of the supra-aortic vessels, as expected. Between the brachiocephalic trunk and the left common carotid artery (LCCA), the circumferential stress was approximately 0.44 MPa and the longitudinal stress approximately 0.27 MPa. Above the STJ, the circumferential and lon-

gitudinal stresses in the aortic wall were 0.30 and 0.22 MPa, respectively.

Stresses with aortic root motion in patients with AI

At 120 mmHg luminal pressure, the circumferential and longitudinal stresses did not change markedly between the brachiocephalic trunk and LCCA when 7.3 mm axial displacement was applied to the aortic root. The area where the most significant changes occurred was in the outer curve of the aorta, about 2 cm above the STJ. In this region, the longitudinal stress increased by 32% up to 0.29 MPa, while the circumferential stress was unchanged (Fig. 2).

Discussion

Magnitude of aortic root motion

The finding that measurements of axial displacement of the aortic annulus (0-14 mm) in the present study were in the same range as those reported by Kozerke et al. (5) from three-dimensional (3D) MRI (6.4-11.3 mm in healthy subjects) implies that the plane of the aortogram is where most of the 3D displacement of the aortic root occurs. The average displacement of 8.9 mm reported by Kozerke et al. in healthy subjects may have been related to their relatively young age (mean 32 years; range: 26 to 56 years) and a greater elasticity of their aortae. By contrast, the present patients were older (mean age 68 years; range: 43 to 88 years) with an average aortic root displacement of 4.8 mm. Interestingly, Kozerke et al. reported values of aortic root displacement in four patients with aortic regurgitation (mean age 60 years; range: 39 to 73 years), and found an average value of 6.5 mm (range: 3.4 to 10.2 mm), which agreed well with the present findings. Most importantly, even though only a few of

Table II: Univariate statistics (Wilcoxon rank sum test; two-sided comparison).

Condition	No. of patients (n = 90)	DISP* (mm)	p-value
AI/No AI	15/75	7.3 ± 3.7/4.3 ± 2.6	0.003
HKI/No HKI	36/54	3.7 ± 2.3/5.5 ± 3.2	0.014
HTR/No HTR	23/67	3.8 ± 2.7/5.1 ± 3.1	0.073
CABG/No CABG	13/77	4.0 ± 3.7/4.9 ± 2.9	0.128
AS/No AS	25/65	4.6 ± 3.4/4.8 ± 2.9	0.429
Average	-	4.8 ± 3.0	
LC/No LC	64/26	4.5 ± 3.0/5.5 ± 3.1	0.076

*Values are mean ± SD.

DISP: Aortic root axial displacement; LC: Listed conditions (for abbreviations, see Table I).

the present patients had a large aortic root displacement (8-14 mm; see Table I), they may have been at considerable risk of mechanical damage to their aortae through acute (rupture) or chronic (fatigue-related) events.

Cardiac pathology and aortic root movement

Aortic insufficiency admittedly leads to increased stroke volume as a compensation mechanism. The present results suggest that aortic insufficiency also increases aortic root displacement in its axial direction. Whether increased stroke volume is related to increased or unchanged aortic root motion has been debated, based on the results of studies focusing on aortic displacement in the frontal-dorsal direction using two-dimensional (2D) echocardiography (8-10). Some values reported in the literature are very large and do not agree with either the present findings or those of Kozerke et al. (5), but pulsations of the aorta are well known in severe cases of AI, and can even be transmitted to the patient's head (nodding or de Musset's sign). The augmenting influence of AI on aortic root displacement was noted previously in a group of 40 cardiac patients (7), and is confirmed in this larger series by multivariate analysis.

Interestingly, in most clinical series, one-half to two-thirds of the patients with proximal aortic dissection exhibit aortic insufficiency. In contrast, less than 10% of those patients with distal aortic dissection show aortic insufficiency and, in the majority of cases, the murmur derives from a condition which preceded the dissection, namely severe hypertension, valvular heart disease or annuloaortic ectasia (11,12). In view of the present findings, it might be hypothesized that undiagnosed, pre-existing AI could have triggered aortic dissection by increasing stress in the aortic wall.

The magnitude of aortic root displacement was decreased in cases of left ventricular hypokinesia, most probably due to reduced ventricular traction. Myocardial hypertrophy showed a trend to lessen the aortic root movement, but the reasons for this were not clear. In contrast, Caruso et al. (13) recently showed evidence that left ventricular hypertrophy may be associated with aortic enlargement and subsequent thoracic aortic dissection. The interplay between different conditions is complex however, and would require further investigation.

Aortic wall stress

The significance of the aortic wall stress, in a comparative sense, and the validity of the finite element model used in the present study, has been discussed elsewhere (7). The 32% increase in longitudinal stress in the ascending aorta between patients with and with-

out AI shows the significant change when the average aortic root motion is increased from 4.3 to 7.3 mm. Even more detrimental effects are expected with larger values of displacement found in some patients (7). It is interesting that the model shows the location at 2 cm above the STJ, in the outer curve of the ascending aorta, to be where the largest increase in longitudinal stress occurs due to aortic root motion. This is also the exact location where most tears and dissecting channels are reported in patients with aortic dissection (1). Given the threat posed by higher values of longitudinal stress in the ascending aortic wall, it would be important to measure aortic root motion by non-invasive techniques such as 3D echocardiography in order to assess an individual's risk for aortic dissection. This information would be valuable in the rationale to plan surgical corrections in susceptible patients

Clinical extension of the study

As aortic root motion varies notably between patients, and significantly increases the risk of aortic rupture in cases combining a large displacement with stiffer aortic tissue, one natural extension of the present study would be to seek pharmacological agents that would specifically target the magnitude of aortic root displacement. This approach, which is aimed at controlling blood pressure and cardiac contractility (dP/dt), has already led to the implementation of successful drug therapy in patients with acute aortic dissection (1). At the other end of the spectrum, crack cocaine has recently been reported to increase the risk of aortic dissection in humans. Although these mechanisms may be multifactorial (14-17), the increase in myocardial contractility translating into increased aortic root motion may be a key factor. It would be interesting to investigate these and other drugs with respect to their roles on the aortic root motion, and not just cardiac contractility.

In conclusion, the magnitude of aortic root axial displacement appears to be influenced by cardiac pathologies. While left ventricular hypokinesia and myocardial hypertrophy were found to reduce aortic root motion, AI significantly enhanced it. Due to the direct impact of the magnitude of aortic root motion on longitudinal stress in the ascending aorta (and therefore on the risk of aortic dissection and rupture), AI appears to be a significant cardiac pathology with regard to aortic dissection. Thus, it should be used as an indicator and as part of the rationale to plan surgical corrections in susceptible patients. Alternatively, drugs which would specifically target aortic root motion and maintain it within a lower range would offer potential protection against aortic dissection.

References

1. Doroghazi RM, Slater EE. Aortic dissection. McGraw-Hill Book Co., New York, 1983:33
2. Leonard JC, Hasleton PS. Dissecting aortic aneurysms: A clinicopathological study. *Q J Med* 1979;XLVIII:55-76
3. Schlatmann TJM, Becker AE. Histologic changes in the normal aging aorta: Implications for dissecting aortic aneurysm. *Am J Cardiol* 1977;39:13-20
4. Mercer JL. Movement of the aortic annulus. *Br J Radiol* 1969;42:623-626
5. Kozerke S, Scheidegger MB, Pedersen EM, Boesiger P. Heart motion adapted cine phase-contrast flow measurements through the aortic valve. *Magnet Reson Med* 1999;42:970-978
6. Stuber M, Scheidegger MB, Fischer SE, et al. Alterations in the local myocardial motion pattern in patients suffering from pressure overload due to aortic stenosis. *Circulation* 1999;100:361-368
7. Beller CJ, Labrosse MR, Thubrikar MJ, Robicsek F. Role of aortic root motion in the pathogenesis of aortic dissection. *Circulation* 2004;109:763-769
8. Pratt RC, Parisi AF, Harrington JJ, Sasahara AA. The influence of left ventricular stroke volume on aortic root motion. *Circulation* 1976;53:947-953
9. Keltai M, Lengyel M, Tonelli M, Babor G. Aortic root motion for the assessment of left ventricular function in acute myocardial infarction. *Acta Cardiol* 1979;XXXIX:51-56
10. Biamino G, Wessel HJ, Schlag W, Schroeder R. Echocardiographic pattern of motion of the aortic root as a correlate of left atrial volume changes. *Am Heart J* 1980;100:191-200
11. Slater EE, DeSanctis RW. Clinical recognition of dissecting aortic aneurysm. *Am J Med* 1976;60:625
12. Lindsay J, Jr., Hurst JW. Clinical features and prognosis in dissecting aneurysm of the aorta: A reappraisal. *Circulation* 1967;35:880
13. Caruso ID, Galderisi M, Covino FE, et al. Association of left ventricular hypertrophy and aortic dilation in patients with acute thoracic aortic dissection. *Angiology* 2001;52:447-455
14. Hsue PY, Salinas CL, Bolger AF, Benowitz NL, Waters DD. Acute aortic dissection related to crack cocaine. *Circulation* 2002;105:1592-1595
15. Wilson LD, Jeromin J, Garvey L, Dorbandt A. Cocaine, ethanol and cocaethylene cardiotoxicity in an animal model of cocaine and ethanol abuse. *Acad Emerg Med* 2001;8:211-222
16. Kloner RA, Hale S, Alker K, Rezkalla S. The effects of acute and chronic cocaine use on the heart. *Circulation* 1992;85:407-419
17. Mehta PM, Grainger TA, Lust RM, et al. Effect of cocaine on left ventricular function: Relation to increased wall stress and persistence after treatment. *Circulation* 1995;91:3002-3009