The combined impact of mechanical factors on the wall stress of the human ascending aorta – a finite elements study

Tomasz Plonek 1*, Malgorzata Zak 2, Karolina Burzynska 2, Bartosz Ryłski 3, Anna Gozdzik 1, Wojciech Kustrzycki 1, Friedhelm Beyersdorf 3, Marek Jasinski 1 and Jaroslaw Filipiak 2

Abstract

Background: Biomechanical factors influence stress in the aortic wall. The aim of this study was to assess how the diameter and shape of the vessel, blood pressure and longitudinal systolic aortic stretching (SAS) caused by the contraction of the myocardium influence stress in the aortic wall.

Methods: Three computational models of the non-dilated aorta and aneurysms of the ascending aorta and aortic root were created. Then, finite elements analyses were carried out. The models were subjected to blood pressure (120 mmHg and 160 mmHg) and longitudinal systolic aortic stretching (0 mm, 5 mm, 10 mm and 15 mm). The influence of wall elasticity was examined too.

Results: Blood pressure had a smaller impact on the stress than the SAS. An increase in blood pressure from 120 mmHg to 160 mmHg increased the peak wall stress (PWS) on average by 0.1 MPa in all models. A 5 mm SAS caused a 0.1–0.2 MPa increase in PWS in all the models. The increase in PWS caused by a 10 mm and 15 mm SAS was 0.2 MPa and 0.4 MPa in the non-dilated aorta, 0.2–0.3 MPa and 0.3–0.5 MPa in the aneurysm of the ascending aorta, and 0.1–0.2 MPa and 0.2–0.3 MPa in the aortic root aneurysm model, respectively. The loss of elasticity of the aneurysmal wall resulted in an increase of PWS by 0.1–0.2 MPa.

Conclusions: Aortic geometry, wall stiffness, blood pressure and SAS have an impact on PWS. However, SAS had the biggest impact on wall stress. The results of this study may be useful in future patient-specific computational models used to assess the risk of aortic complications.

Keywords: Aorta, Dissection, Aneurysm, Biomechanics, Finite elements analysis

Background

Aortic complications occur in an unpredictable way. The aortic diameter and its relation to the patient’s height and body surface area (BSA) is the main parameter taken into account when qualifying a patient for surgery. There are other factors that may increase the risk of developing acute aortic syndromes, such as genetic disorders (i.e. Marfan syndrome, Loeys-Dietz syndrome, Ehlers-Danlos syndrome), arterial hypertension, a family history of acute aortic dissection or rapid increase in the aortic diameter [9, 12].

It is believed that aortic dissection occurs when the stress in the aorta is high enough to damage the intima and allows blood flow to separate the layers of the aortic wall [20]. There are several biomechanical factors that influence the stress in the wall of the ascending aorta: the aortic wall elasticity and its tensile strength, the geometry of the vessel, the arterial blood pressure, the characteristics of blood flow and longitudinal up-and-down stretching of the aorta caused by systolic-diastolic motion of the heart [4, 10, 16, 20, 24]. However, there are no diagnostic tools that allow an objective assessment of the stress in the aortic wall and the potential risk of aortic dissection.

* Correspondence: tomaszplonek@gmail.com

Meeting presentation: The manuscript was presented during the 65th International Congress of the European Society for Cardiovascular and Endovascular Surgery (Belgrade, Serbia, 21–23 April 2016)

© The Author(s). 2017 Open Access This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.
The aim of this study was to assess the stress distribution in the aortic root, ascending aorta and aortic arch using the finite elements method. Comparative analyses of the influence of elasticity and two dynamic factors (arterial blood pressure and longitudinal up-and-down movement of the ascending - systolic aortic stretching /SAS/) on stress in the wall of the non-dilated aorta, an aneurysm of the ascending aorta and an aneurysm of the aortic root, were also performed.

Methods
Model of the aorta
Three simplified computational 3D models of a non-dilated aorta, an aneurysm of the ascending aorta and an aneurysm of the aortic root were created (Fig. 1). To make the comparison between the models more feasible, their geometries differed only in terms of the diameters of the aortic root and ascending aorta. The aorta was simulated from the level of the aortic annulus (virtual basal ring) to the descending aorta. The dimensions of the models are presented in Table 1.

Finite elements analysis
The finite elements analyses were performed using the Ansys software (Ansys, Inc.) The 3D models of the non-dilated aorta and aortic aneurysms were divided into 244,000–293,000 3D tetrahedron SOLID187 elements with 430,000–519,000 nodes (discretization process). The elastic properties were chosen according to previously published studies [14, 16, 18]. Each aneurysm model had two versions: one, where the aortic wall elasticity was identical to the model of the non-dilated aorta (Young’s modulus: 6 MPa) and the other, where the Young’s modulus was increased up to 9 MPa. All mechanical properties of the aortic wall used in the computational models are presented in Table 2.

The models were subjected to pressures of 120 mmHg and 160 mmHg. Moreover, a longitudinal up-and down movement of the aortic root and ascending aorta (systolic aortic stretching – SAS), caused by contraction of the heart during systole of 0 mm, 5 mm, 10 mm and 15 mm, was simulated. The distal descending aorta and distal parts of the aortic arch branches were immobilized in all directions. The distribution and maximal values of the stress (von-Mises stress) were assessed in each simulation for every model.

Results
Stress distribution
The areas of the maximal stress were identified in all models. In the simulations where the systolic aortic stretching (SAS) was not applied, the maximal stress was observed in the sinotubular junction (STJ) (Figs. 2, 3 and 4). When the SAS was applied, the peak wall stress (PWS) was identified at the junction of the ascending aorta and aortic arch and in the lesser curvature between arch and descending aorta. In the ascending aorta, the stress was higher in the concavity than the convexity. The areas of the highest wall stress were not observed in the most dilated segments but at sites of abrupt change of diameter and geometry. In the aortic root model, an additional site of high stress was observed in the area of the aortic annulus. Overall, the highest PWS was observed in the area of the aortic annulus. The highest PWS was observed in the model of the aneurysm of the ascending aorta with a stiff wall (Young’s modulus 9 MPa), subjected to SAS of 15 mm and blood pressure of 160 mmHg. It amounted to 0.9 MPa and was localized in the area between the ascending aorta and the aortic arch (Fig. 3).

Arterial blood pressure
An increase in blood pressure from 120 mmHg to 160 mmHg caused an increase in the peak wall stress across the whole surface in all models. However, there was a larger increase in stress at sites of high peak stress, i.e. the sino-tubular junction or the area between the ascending aorta and the arch. The peak wall stress
increased on average by 0.1 MPa regardless of the geometry, the systolic aortic stretching or the aortic wall elasticity.

**Systolic aortic stretching**
Systolic aortic stretching caused a significant increase in the peak wall stress in all the models. In the non-dilated aorta, the maximal stress in the aortic wall increased with the distance to which the aorta was pulled during the systolic motion of the heart. A 5 mm increase in the SAS caused a 0.1–0.2 MPa increase in PWS in all the models. The difference in stress between models with no SAS and those with 10 mm SAS was 0.2 MPa in the non-dilated aorta, 0.2–0.3 MPa in the aneurysm of the ascending aorta, and 0.1–0.2 MPa in the aortic root aneurysm model. When the SAS was changed from 0 mm to 15 mm the PWS increased by 0.4 MPa in the non-dilated aorta, 0.3–0.5 MPa in the aneurysm of ascending aorta and 0.2–0.3 MPa in aortic root aneurysm.

**Aortic wall elasticity**
The models of the aneurysms with lower aortic wall elasticity (Young’s modulus 9 MPa) had higher PWS values than those with elastic walls (Young’s modulus 6 MPa). The stress was higher on average by 0.1 MPa in models with stiffer walls. A 0.2 MPa increase in wall stress was observed in the model of the aneurysm of the ascending aorta when 15 mm systolic aortic stretching was applied.

**Geometry of the aorta**
The highest wall stress was observed in the model of an aneurysm of the ascending aorta (0.9 MPa; Young’s modulus 9 MPa, SAS 15 mm, arterial blood pressure of 160 mmHg). The maximal wall stress in the models of non-dilated aorta and aortic root aneurysm were 0.5 MPa (Young’s modulus 6 MPa, SAS 15 mm, arterial blood pressure of 160 mmHg) and 0.5 MPa (Young’s modulus 9 MPa, SAS 15 mm, arterial blood pressure of 160 mmHg). The stress distribution differed between models, although some areas of PWS were similar, i.e. the sino-tubular junction and the area between the aortic arch and the ascending aorta. Moreover, the aortic root model had higher stress in the area of the ventriculo-aortic junction (0.5 MPa) compared to the non-dilated aorta (0.3 MPa) and the aneurysm of the ascending aorta (0.3 MPa).

**Discussion**
There is no objective method to assess the risk of development of an aortic dissection. According to a recent study, type A aortic dissection usually occurs when the diameter of the vessel is approximately 40 mm [26]. This is much lower than the threshold for qualifying the patient for surgery [9, 12]. Therefore, a new method to estimate the risk of dissection, which takes into account all currently known risk factors, is necessary. We believe that a finite elements analysis could be used to define this risk for an individual patient [16, 20].

Biomechanical factors play a crucial role in the process of aortic dissection [21]. It is believed that blood pressure, the diameter and elasticity of the aorta have the most significant impact on stress in the aortic wall [4, 6, 7, 9, 21, 27]. However, it is still unknown which factor has the biggest influence on mechanical stress. The geometry of the proximal ascending aorta varies significantly between individuals [8] and therefore a multifactorial assessment is necessary to estimate the stress in the aortic wall. Several studies assessed the influence of biomechanical parameters on the stress of the wall of the ascending aorta, including wall stiffness [4, 16, 20], aortic root motion [4], blood pressure [4, 10, 20, 24], flow shear stress [24], the geometry of the aorta and aortic valve type [1, 5, 11, 17, 22, 23, 25]. However, there are no studies assessing the wall stress in an aneurysm model subjected to aortic root motion. Moreover, no

**Table 1** The parameters of the computational models

|                      | Non-dilated aorta | Aneurysm of the ascending aorta | Aneurysm of the aortic root |
|----------------------|-------------------|--------------------------------|----------------------------|
| Aortic root          | 35 mm             | 45 mm                          | 55 mm                      |
| Sinotubular junction | 30 mm             | 50 mm                          | 40 mm                      |
| Ascending aorta      | 30 mm             | 55 mm                          | 30 mm                      |
| Aortic arch          | 30 mm             | 30 mm                          | 30 mm                      |
| Descending aorta     | 30 mm             | 30 mm                          | 30 mm                      |
| Aortic arch branches | 10 mm             | 10 mm                          | 10 mm                      |
| Aortic wall thickness [20] | 2 mm              | 2 mm                           | 2 mm                      |

**Table 2** Mechanical parameters of the aortic wall

| Model                               | Young’s modulus | Poisson’s ratio |
|-------------------------------------|-----------------|-----------------|
| Non-dilated aorta                   | 6 MPa           | 0.49            |
| Aneurysm (ascending aorta and aortic root) | 6 MPa, 9 MPa    | 0.45            |
Fig. 2 The wall stress in the model of the non-dilated aorta subjected to systolic aortic stretching of 0 mm, 5 mm, 10 mm and 15 mm. In the upper row, the models were subjected to arterial blood pressure of 120 mmHg, whereas in the lower row they were subjected to 160 mmHg. The colors represent peak wall stress in MPa.

Fig. 3 The wall stress in the model of an aneurysm of the ascending aorta subjected to systolic aortic stretching of 0 mm, 5 mm, 10 mm and 15 mm. In the upper row, the models were subjected to arterial blood pressure of 120 mmHg, whereas in the lower row they were subjected to 160 mmHg. On the left, the models have an elastic wall, whereas the models on the right have a stiff wall. The colors represent peak wall stress in MPa.
comparative analysis of stress distribution between a non-dilated aorta, aortic root aneurysm and an aneurysm of the ascending aorta has been performed. To date, this is the first study to perform complex biomechanical analyses assessing the impact of all currently known biomechanical factors responsible for aortic stress and comparing various types of aortic geometries.

The factor that significantly influences stress in the ascending aorta is longitudinal stretching of the aorta caused by systolic movement of the heart (systolic aortic stretching, SAS) [4, 25]. Based on a study that assessed magnetic resonance images (MRI) of 11 healthy patients, the SAS is on average 8.9 mm [15]. In the Reykyavik study, aortic annulus motion assessed by MRI in 347 patients over 70 years of age was 6.8 mm in men and 7.8 mm in women [3]. According to Beller and colleagues, an 8.9 mm displacement of the aortic annulus resulted in a stress increase comparable to the rise in blood pressure by 60 mmHg (from 120 mmHg to 180 mmHg). One of the most common sites for the entry of type A aortic dissection is the transverse tear near the sinotubular junction [13]. A transverse tear can occur due to the longitudinal stretch of the aortic wall. Such a longitudinal stretch is caused by SAS [4, 25]. Based on our results, SAS has a bigger impact on aortic wall stress than blood pressure. A 10 mm SAS caused a rise in wall stress two times higher than a 40 mmHg increase in blood pressure. This finding supports the thesis that a positive inotropic effect of some drugs may increase the risk of dissection [2] and certain negative inotropic drugs, i.e. beta blockers can reduce this risk [28].

The MRI studies in healthy subjects revealed a slight axial twist of 6–14 degrees of the ascending aorta during systole [29]. However, this did not impact the wall stress [4] and thus, was not simulated in our model. Blood flow shear stress in the ascending aorta was estimated at the level of around 0.0023 kPa. This is several orders of magnitude lower than the stress caused by blood pressure and may influence the growth of the vessel over years, but is not likely to cause an intimal tear and be a direct cause of a dissection. Therefore, flow shear stress was not assessed in our analyses [24].

The maximal wall stress was observed in the sinotubular junction (STJ) area, at the junction of the ascending aorta and aortic arch and in the lesser curvature between the arch and the descending aorta. These areas correlate with the most common sites of entry in type A aortic dissection [13]. In the aortic root model, an additional zone of high stress was observed in the aortic annulus. This finding may explain why aortic root aneurysms are often accompanied by annular dilatation.

The PWS was not observed in the areas where the diameter was large, but at sites of an abrupt change in the vessel geometry, i.e. the sinotubular junction. This finding is consistent with other studies, which report PWS to be located in areas of sudden morphological change, i.e. aneurysm necks [19, 22, 27].

The aortic wall stiffness had a significant influence on the wall stress. The aneurysm models with stiffer walls had higher peak stress by 0.1–0.2 MPa compared to those of normal elasticity. This was comparable to the
stress caused by a 40 mmHg increase in systolic blood pressure.

The geometry of the aortic model had a significant impact on the peak wall stress and stress distribution. The highest PWS was observed in the model of an aneurysm of the ascending aorta. However, to estimate wall stress correctly, a patient-specific model is necessary. Such simulations on patient-specific models with the aortic wall composed of three layers have already been performed by our group. The stress values and stress distribution was similar in both - patient-specific and the idealized models (Fig. 5). Nevertheless, some differences between the models were observed. Therefore, further studies are necessary to estimate what an acceptable simplification of the aortic model is. Current diagnostic tools are not able to define local aortic wall mechanical properties. In the near future, high resolution MRI scanners may be able to assess the local thickness and elasticity of the aorta with a resolution which will allow for reliable computational numerical reconstructions.

Study limitations
The aortic wall is an inhomogeneous layered structure with nonlinear anisotropic mechanical properties [10, 30]. To date, there are no examinations allowing for an exact in vivo assessment of the biomechanical properties of the wall of the ascending aorta and aortic aneurysm. Therefore, a simplified model with linear isotropic properties of the aortic wall was used. Nevertheless, in future studies, the patient specific material properties and aortic geometry must be taken into account to evaluate stress correctly [16, 20]. The fluid-structure interaction was not used because this type of simulation made it impossible to implement the movement of the aortic annulus to mimic the stretching of the aorta. This study is a theoretical view, which is more an indication that longitudinal stretching of the aorta should be implemented in the simulations of the thoracic aorta than a reliable estimation of the real values of stress in the thoracic aorta.

Conclusions
The results of this study may be useful in future patient-specific computational models used to assess the risk of aortic complications. Our results help to differentiate how individual factors influence wall stress. Systolic aortic stretching in the ascending aorta is a phenomenon that cannot be omitted when performing stress analyses. SAS had a larger impact on the wall stress than blood pressure, wall stiffness and the geometry of the vessel. Moreover, the sites of peak wall stress correlate with typical areas of dissection entry.

Abbreviations
3D: Three-dimensional; BSA: Body surface area; MRI: Magnetic resonance imaging; PWS: Peak wall stress; SAS: Systolic aortic stretching; STJ: Sino-tubular junction

Acknowledgements
Not applicable.

Funding
This study was supported by a statutory grant from Wroclaw Medical University (ST-829).

Availability of data and materials
The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors’ contributions
TP: study design, data collection, data analysis, manuscript preparation; MZ: data collection, data analysis, manuscript preparation and critical review; KB: data collection, data analysis, manuscript revision; BR: data collection; manuscript preparation and critical review; AG: data collection, manuscript preparation; WK: study design, data collection; FB: data collection, critical review, manuscript preparation; MJ: data collection and analysis, manuscript revision; JF: supervision, data analysis, critical review. All authors read and approved the final manuscript.

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Author details
1Department of Cardiac and Thoracic Surgery, Wroclaw Medical University, Borowska 213, 50-556 Wroclaw, Poland. 2Department of Biomedical Engineering, Mechatronics and Theory of Mechanisms, Wroclaw University of Science and Technology, Wroclaw, Poland. 3Department of Cardio-vascular Surgery, Heart Centre Freiburg University, Faculty of Medicine, University of Freiburg, Freiburg, Germany.
References

1. Back M, Gasser TC, Michel JB, Caligiuri G. Biomechanical factors in the biology of aortic wall and aortic valve diseases. Cardiovasc Res. 2013;99:232–41.

2. Beaven DW, Murphy EA. Dissecting aneurysm during methionine therapy; a report on nine cases treated for hypertension. Br Med J. 1956;1:77–80.

3. Bell V, Sigurdsson S, Westenberg JJ, Gotal JD, Torjesen AA, Aspelund T, Launer LJ, Harris TB, Gudnason V, de Roos A, Mitchell GF. Relations between aortic stiffness and left ventricular structure and function in older participants in the age, gene/environment susceptibility–Reykjavik study. Circ Cardiovasc Imaging. 2015;8:e003039.

4. Beller CJ, Labrosse MR, Thubrikar MJ, Robicsek F. Role of aortic root motion in the pathogenesis of aortic dissection. Circulation. 2004;109:763–9.

5. Beller CJ, Labrosse MR, Thubrikar MJ, Szabo G, Robicsek F, Hagi S. Increased aortic wall stress in aortic insufficiency: clinical data and computer model. Eur J Cardiothorac Surg. 2005;27:270–5.

6. Bodermann D, Gherebaghi-Schnell E, Wollenek G, Maurer G, Baumgardner H, Lang IM. Mechanisms underlying aortic dissection in congenital aortic valve malformation. Circulation. 1999;99:138–43.

7. Chandran KB, Gao D, Han G, Baraniewski H, Corson JD. Finite-element analysis of arterial anastomoses with vein, Dacron and PTFE grafts. Med Biol Eng Comput. 1992;30:413–8.

8. Contino M, Mangini A, Lemma MG, Romagnozzi C, Zorbi P, Gelpi G, Antonia C. A geometric approach to aortic root surgical anatomy. Eur J Cardiothorac Surg. 2016;49:93–100.

9. Erbel R, Aboyans V, Bax JJ, Bossone E, Budts L, Ceriello A, et al. ESC guidelines for the diagnosis and management of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. Eur Heart J. 2014;35:2873–926.

10. Gao F, Watanabe M, Matsuzawa T. Stress analysis in a layered aortic arch model under pulsatile blood flow. Biomed Eng Online. 2006;5:25.

11. Girdauskas E, Rouman M, Disha K, Espinoza A, Dubslaff G, Fey B, Theis B, Petersen J, Borger MA, Kuntze T. Aortopathy in patients with bicuspid aortic valve stenosis: role of aortic root functional parameters. Eur J Cardiothorac Surg. 2016;49:653–64; discussion 643–634.

12. Hiratka LF, Bakris GL, Beckman JA, Blackshear JL, Blumenthal RS, et al. ACC/AHA/ACS/SCAI/SCAI/SIR/STS/VSCV guidelines for the diagnosis and management of patients with thoracic aortic disease: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines, American association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society for Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Intervventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121:e266–369.

13. Hirst AE Jr, Johns VJ Jr, Kime SW Jr. Dissecting aneurysm of the aorta: a review of 505 cases. Medicine (Baltimore). 1958;37:217–79.

14. Khaanafer K, Duprey A, Zainal M, Schlicht M, Williams D, Berguer R. Determination of the elastic modulus of ascending thoracic aortic aneurysm – computational fluid dynamics. Eur J Cardiothorac Surg. 2010;38:643–7.

15. Kozerke S, Schmidt KB. Ascending aortic elongation and the risk of dissection. Eur J Cardiothorac Surg. 2015;47:61–7.

16. Kruger T, Forkavets O, Veseli K, Lausek H, Vohringer L, Schneider W, Bamberg F, Schlenk M, Cutting DJ, Thomas B, Becket G, Hill P, Vazquez J. Aortic valve malformation. Circulation. 2004;109:926.

17. Li WC, Yu MH, Zhang HM, Wang HQ, Xi GM, Yao BC, Deng ZH, Zeng YJ. Biomechanical properties of ascending aorta and pulmonary trunk in pigs and humans. Xenotransplantation. 2008;15:384–9.

18. Li ZY, Sadat U, UK-I J, Tang YV, Bowden DJ, Hayes PD, Gillard JH. Association between aneurysm shoulder stress and abdominal aortic aneurysm expansion: a longitudinal follow-up study. Circulation. 2010;122:1815–22.

19. Martin C, Sun W, Elefteriades J. Patient-specific finite element analysis of ascending aortic aneurysms. Ann J Phys Heart Circ Phys. 2015;30:1306–16.

20. Martelli F, Forneris A, Appoo JJ, Di Martino ES. Is there a role for biomechanical engineering in helping to elucidate the risk profile of the thoracic aorta? Ann Thorac Surg. 2016;101:90–8.

21. Nathan DP, Xu C, Rappert T, Desjardins B, Gorman JH 3rd, Bavaria JE, Gorman RC, Chandran KB, Jackson BM. Increased ascending aortic wall stress in patients with bicuspid aortic valves. Ann Thorac Surg. 2011;92:1384–9.

22. Numata S, Iitani K, Kanda K, Doi K, Yamazaki S, Morimoto K, Manabe K, Ikemoto K, Yaku H. Blood flow analysis of the aortic arch using computational fluid dynamics. Eur J Cardiothorac Surg. 2016;49:1578–85.

23. Pasta S, Rinaudo A, Luca A, Pilato M, Scandulla C, Gleason TG, Yorp DA. Difference in hemodynamic and wall stress of ascending thoracic aortic aneurysms with bicuspid and tricuspid aortic valve. J Biomech. 2013;46:1729–38.

24. Plonek T, Ryński B, Dumanski A, Siedlaczek P, Kustrzycki W. Biomechanical analysis of wrapping of the moderately dilated ascending aorta. J Cardiothorac Vasc Surg. 2015;10:106.

25. Ryński B, Blanke P, Beyersdorf F, Desai ND, Milewski RK, Siepe M, Kari FA, Czerny M, Carrel T, Schlenck C, Kruger T, Mack MJ, Brintman WK, Mohr FW, Etz CD, Luehr M, Bavaria JE. How does the ascending aorta geometry change when it dissects? J Am Coll Cardiol. 2014;63:1311–9.

26. Shang EK, Nathan DP, Sprinkle SR, Fairman RM, Bavaria JE, Gorman RC, Gorman JH 3rd, Jackson BM. Impact of wall thickness and saccular geometry on the computational wall stress of descending thoracic aortic aneurysms. Circulation. 2013;128:5157–62.

27. Shores J, Berger KR, Murphy EA, Piyeritz RE. Progression of aortic dilatation and the benefit of long-term beta-adrenergic blockade in Marfan’s syndrome. N Engl J Med. 1994;330:3135–41.

28. Stuber M, Scheidegger MB, Fischer SE, Nagel E, Steinemann F, Hess OM, Boesiger P. Alterations in the local myocardial motion pattern in patients suffering from pressure overload due to aortic stenosis. Circulation. 1999;100:361–8.

29. Xie J, Zhou J, Fung YC. Bending of blood vessel wall: stress-strain laws of the intima-media and adventitial layers. J Biomech Eng. 1995;117:336–45.