10 Mechanics of the Thoracic Aortic Wall

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10.1 Introduction

The aorta is a vital organ of the human body. This tubular structure delivers oxygen-enriched blood to all organs and tissues of the human body. However, unlike one would imagine, the aorta is much more than a hollow, passive conduit for transportation of a fluid. The aorta is a living organ with a unique anatomy, complex intrinsic biology, and sophisticated mechanical properties [1] that permit its active "playing a game of catch" with the stroke volume.

Aneurysms of the aorta are currently the 19th most common cause of death in the United States, causing approximately 13,000 deaths annually [2], a number that in all likelihood markedly underestimates the true prevalence of aorta-related deaths, which are often misdiagnosed as heart attacks. The nature of aneurysm disease makes it "silent" in the absolute majority of affected individuals. Most affected patients do not know that they are harboring a growing aortic aneurysm in their chest or abdomen [1]. The first presentation of this disease may be devastating, either producing an instant death or resulting in a complication that is likely to produce death—such as rupture or dissection—unless treated emergently in a specialized center [3]. Therefore, timely identification of individuals with an aortic aneurysm and preemptive extirpation are key to preventing deaths related to this condition.

Current guidelines for surgical treatment of thoracic aortic aneurysm recommend elective surgical resection of ascending aortic aneurysms before they reach 5.5 cm (for descending aortic aneurysms before they reach 6.0 cm) [4]. These intervention criteria have been developed largely based on detailed examination of the natural history of the disease and the risks of dissection, rupture, and death of aortas reaching a certain size [5–7]. These intervention criteria were recently further refined to account for differences in a person's height and weight. The risks of adverse events were calculated in relation to body surface area, providing a more accurate estimate of the actual risk in a specific patient [8]. Similarly, guidelines on abdominal aortic aneurysm recommend preventive surgical/endovascular treatment at 5.0 cm for females and 5.5 cm for males [9].

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However, it is becoming increasingly apparent that estimation of risk of aortic dissection or rupture purely based on a size criterion is, although good, far from perfect [10]. Currently, establishment of the genetic nature of thoracic and abdominal aortic aneurysm is burgeoning [11–13]. The aorta in patients with certain specific genetic mutations can behave more malignantly and unpredictably, so that the general size criteria may not be applicable. Likewise, bioengineering understanding of aortic disease is also advancing rapidly, permitting understanding of the mechanical processes that occur within the aorta, both healthy and diseased. Understanding the mechanics of the aortic wall, which may vary from individual to individual, is critical for further refinement of the surgical intervention criteria.

The mechanics and mechanobiology of abdominal aortic aneurysm have been studied in detail and reported in an excellent review article by Humphrey and Holzapfel [14]. In this chapter we describe the current understanding of the mechanics of the thoracic aorta and thoracic aortic aneurysm (TAA). We explore the potential clinical application of engineering parameters to surgical decision-making.

10.2 Normal Anatomy, Structure, and Histology of the Aortic Wall

The histologic structure of the thoracic aorta is similar to other types of blood vessels and is described in detail in earlier chapters of this textbook. We focus on the main histologic components that are critical for understanding the mechanics of the aortic wall.

The aortic wall consists of three main layers: the internal layer, *tunica intima*; the medial layer, *tunica media*; and the outer layer, *tunica adventitia* [1]:

- *Tunica Intima*—accounts for approximately 5% of the wall thickness [15] and consists of a single layer of flattened endothelial cells, lying on a basal lamina (internal elastic lamina). The endothelial cells play multiple important functions, including barrier, blood clotting, inflammatory, tone regulatory, reparatory, and other functions. The subendothelial layer contains elastic fibers and type I collagen fibrils, fibroblasts, and myointimal cells (cells that structurally resemble smooth muscle cells) [16, 17]. The intimal layer is supported by an internal elastic lamina that lies between the intimal and medial layers.
- *Tunica Media*—is the largest layer of the aortic wall and accounts for approximately 77–80% of the wall thickness [15]. It consists of concentric layers of smooth muscle cells (43%), collagen (30%), elastin (23%), and glycosaminoglycans and proteoglycans (4%) [15]. According to the general classification of blood vessels, the aorta is considered to be an *elastic artery* [16, 17] (together with the brachiocephalic trunk, the common carotid, subclavian and common iliac arteries, etc.) since elastic fibers predominate in this layer. Individual elastic fibers (0.1–1.0 μm in diameter) fuse to produce lamellae of elastic material. The media has a layered structure, in which layers of elastic lamellae alternate with interlamellar smooth muscle cells and collagen [16]. Such regular repetition of these medial elements allows us to define a "lamellar unit," first described by Wolinsky and Glagov [18], which consists of two layers of elastic lamellae (surrounded by proteins of the extracellular matrix, including collagen, adhesion molecules, and glycosaminoglycans/proteoglycans) and one smooth muscle cell in between them (Fig. 10.1) [19, 20]. It is estimated that the ascending aorta has anywhere from 53 to 78 lamellar units [19, 21], while the abdominal aorta has approximately 28 medial lamellar units [21]. Blood flow within the aorta is highly pulsatile, which is why the medial layer, with its layers of elastic lamellae, is designed to sustain and smooth out the blood flow despite the pulsatile cardiac output [16].
- *Tunica Adventitia*—accounts for approximately 15–18% of the wall thickness [15] and is formed from general connective tissue

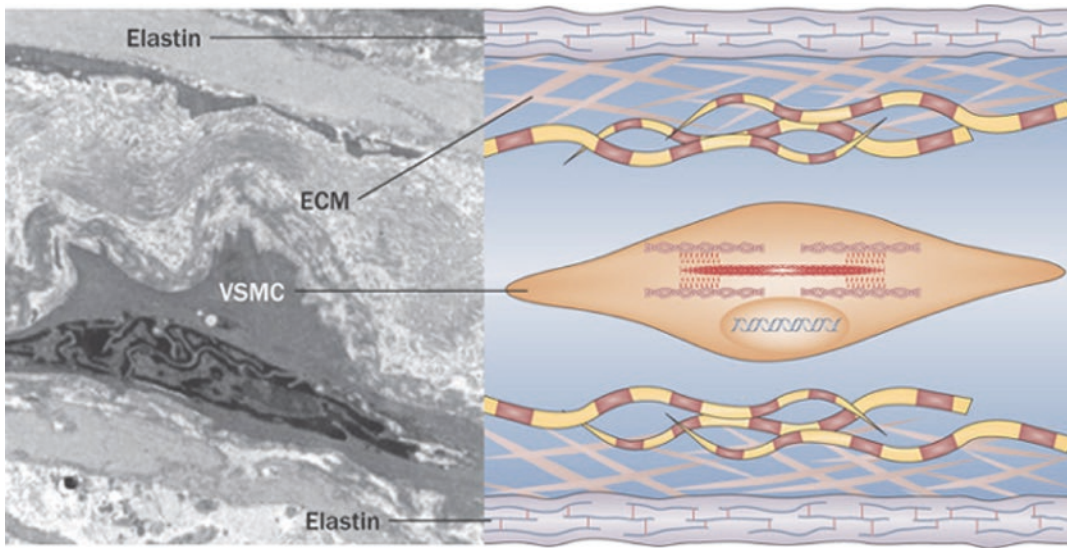

Fig. 10.1 Electron micrograph of a single lamellar unit in the medial layer of the aortic wall showing a VSMC lying between two layers of elastin fibers and surrounded by ECM proteins containing microfibrils and proteoglycans. Lamellar units are intercalated by collagen bundles. The lamellar unit represents the basic structural and functional unit of the aortic wall. Abbreviations: *ECM* extracellular matrix, *VSMC* vascular smooth muscle cell (Reprinted with permission from [20])

and includes blood vessels that supply the aorta (*vasa vasorum*) and nerves that innervate the aorta (*nervi vasorum*). Interestingly, studies have shown that in the thoracic aorta, the outer parts of the medial layer also contain the *vasa vasorum*, which contrasts with the medial layer of the abdominal aorta, which is completely avascular [21]. The adventitial layer can vary in thickness but is primarily quite thin. Despite the adventitia being so thin, it is highly respected by surgeons, who feel it is the "strength layer" of the aorta, essential for secure suturing of aortic tissues [1, 3].

10.3 Engineering Definitions and Characteristics

In mechanics, "strain" is defined as a change in size (e.g., length) produced by a given force.

"Stress" is defined as the force applied per cross-sectional area of the aortic wall (force/area unit). There are two types of stress that the aorta experiences:

- *Tensile stress* (*wall stress*)—an applied force, for arteries primarily dependent on the arterial geometry and the hemodynamic load (blood pressure). Tensile stress can be circumferential, radial, and longitudinal.
- *Shear stress*—is the dragging frictional force created by blood flowing against the inner surface of the aortic wall. Shear stress is dependent on the arterial geometry, the characteristics of blood flow, and the viscosity of the blood.

The result of stress that is applied to the aortic wall is strain—a change in aortic size. Stated briefly, stress is applied and strains results.

Other important engineering characteristics that are used to describe biomechanical properties of the aorta include:

- *Distensibility*—the capacity of the aortic wall to dilate during changes in intraluminal pressure. Vessel wall distensibility is directly related to elastin and collagen content and their structural characteristics.
- *Elastic modulus* (*stress/strain relationship*)—quantification of how much force is needed to

produce a specific change in size. The stress/strain relationship describes the amount of strain (deformation) that elastin and collagen can absorb in response to a specific stress (pressure).

10.4 Assessing the Mechanics of the Aortic Wall

Since the geometry of the aorta plays an important role in determining the mechanical properties of the aorta, accurate imaging modalities are vital to enable bioengineering calculations and models. Recent advances in computed tomography (CT) and magnetic resonance imaging (MRI) have substantially enhanced the accuracy and resolution of aortic imaging, thus making biomechanical analysis and modeling considerably more accurate. In addition, new MR angiography methods have expanded the use of MR angiography beyond purely anatomical information toward quantitative hemodynamic analyses [22]. On the other hand, ex vivo studies using uniaxial [23] and biaxial [24, 25] stretch models of explanted aortic tissue (both healthy and aneurysmal) have contributed to understanding the tensile limits of the aorta. Computational fluid dynamics [26], fluid-solid interactions [27], and finite element analysis [28] are all computational models that are becoming increasingly more sophisticated due to advancements in imaging techniques.

10.5 In Vivo Evaluation of the Aortic Wall Mechanics Via Epiaortic Echocardiography

At our institution, we have utilized epiaortic echocardiography to assess the mechanical properties of the ascending aorta in patients undergoing open-heart surgery [29, 30]. The measurements are conducted after median sternotomy, and pericardiotomy is performed (before arterial or venous cannulation) by using an echocardiographic probe in a sterile sheath together with a cushion constructed from a surgical glove finger filled with normal saline to act as an interface between the probe and the aorta (Fig. 10.2).

Epiaortic echocardiography makes it possible to measure six specific parameters that are needed to determine the important mechanical properties of the aorta (Fig. 10.3):

- Aortic diameter (systolic and diastolic)
- Aortic wall thickness (systolic and diastolic)
- Aortic blood pressure (systolic and diastolic)

Based on these six measurements, a complete mechanical profile of the aorta can be constructed, including the following engineering characteristics: aortic wall distensibility, aortic wall stress, and the incremental elastic modulus (stress/strain relationship).

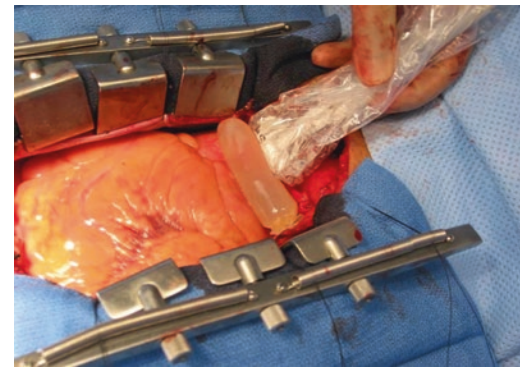

Fig. 10.2 Epiaortic echocardiography. The echo probe is placed directly on the aorta at the time of surgery, with an interposed fluid-filled interface (the tip of a sterile glove) (Reprinted with permission from [1])

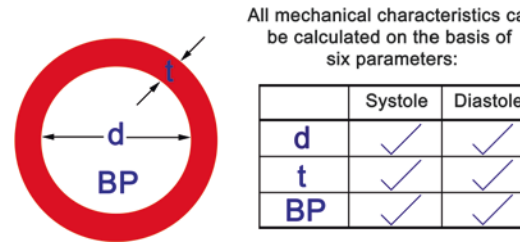

Fig. 10.3 The six parameters needed to calculate the important engineering characteristics of the aorta: aortic diameter (*d*), in systole and diastole; aortic wall thickness (*t*), in systole and diastole; and blood pressure (*BP*), in systole and diastole (Reproduced with permission from [1])

10.5.1 Distensibility

Intraoperative measurements done via epiaortic echocardiography have shown that as the aorta enlarges in diameter, the distensibility of the aortic wall significantly decreases (Fig. 10.4). For example, distensibility values for the ascending aortas greater than 5.0 cm in diameter are 1.45 ± 0.38 mm Hg^{-1}, while the distensibility of the normal ascending aortas and ascending aortic aneurysms smaller than 4.0 cm was significantly greater (2.499 ± 0.49 and 3.02 ± 0.60 mm Hg^{-1}, respectively). By 6 cm aortic diameter, the distensibility values are extremely low (0.81 ± 0.32 mm Hg^{-1}), suggesting that the severely enlarged aorta becomes essentially a rigid, inelastic, and nondistensible tube [29]. As a consequence of such loss of elasticity, the force generated by the left ventricle in systole can no longer be dissipated in expanding the aorta.

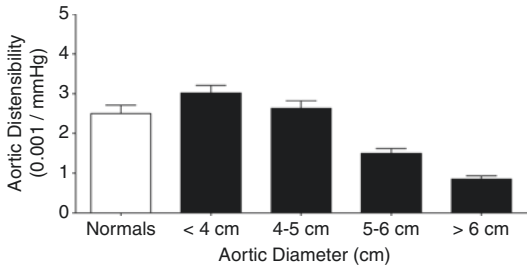

Fig. 10.4 Distensibility values in normal aortas and aortic aneurysms of different diameters. Distensibility of ascending aortic aneurysms decreases rapidly as diameter increases to very low values at dimensions greater than 6 cm (Reproduced with permission from [29])

10.5.2 Wall Stress

Since the aneurysmal aortic wall does not possess normal elasticity properties, the full force of cardiac contraction is translated into wall stress. Therefore, the enlarged aorta demonstrates very high wall tension. The relationship between wall stress and size of the ascending aorta is shown in Fig. 10.5. The mean wall stress for the normal-sized aortas is approximated at 92.51 ± 6.35 kPa. However, as the size of the aorta increases, the wall stress also increases in a step-wise exponential manner. Ascending aortas with a diameter greater than 6.0 cm have a mean wall stress of 376 ± 146.6 kPa. This figure was recorded at a systolic blood pressure of 100 mm Hg. Furthermore, at a mathematically extrapolated systolic blood pressure of 200 mm Hg, the wall stress value for a 6 cm aorta is 857 ± 291 kPa [29]. Such blood pressure, although uncommon in a controlled environment of an operating room, is quite common in real life and corresponds to

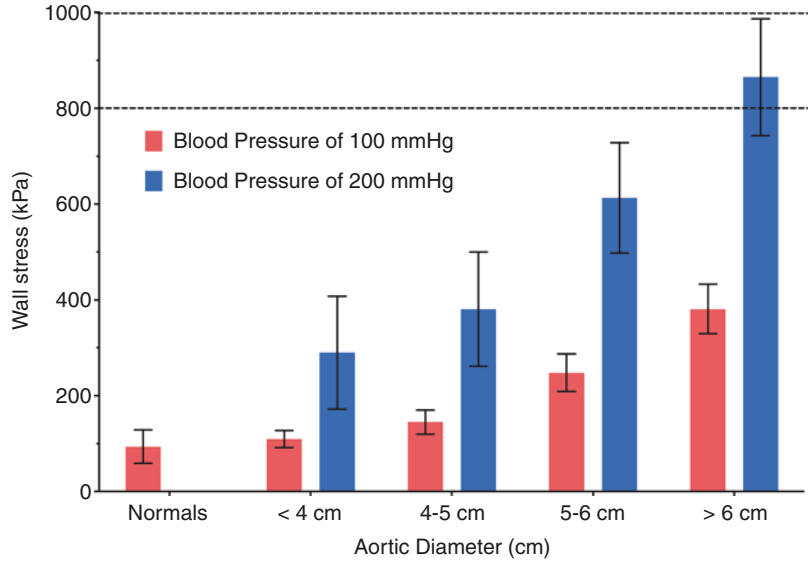

Fig. 10.5 In vivo mechanical properties of human ascending aorta. Exponential relationship between wall stress and aneurysm size in ascending aortic aneurysms. The red columns represent a blood pressure of 100 mm Hg, and the blue columns represent a blood pressure of 200 mm Hg. The lines at 800–1000 kPa represent the range of maximum tensile strength of the human aorta (Reproduced with permission from [10])

episodes of strenuous physical activity [31] and emotional stress [32] or a hypertensive crisis of other etiology. From previous studies we know that the ultimate tensile strength of the aorta is about 800 to 1000 kPa [23, 33]. It is interesting to note that a 6 cm aorta at a blood pressure of 200 mm Hg is exceeding (or "flirting" with) the ultimate tensile limits of the aorta, which can lead to rupture of the aortic wall [1, 10, 29].

10.5.3 Elastic Modulus

Similarly, the elastic modulus values for aortic aneurysms (1.93 ± 0.88 MPa) were significantly greater than those in the normal aorta (1.18 ± 0.21 MPa), while large aneurysms (greater than 5 cm in diameter) had even greater values (3.56 ± 0.88 MPa), suggesting that large ascending aortic aneurysms have already been stretched to their limits [29].

10.5.4 Confluence of Clinical and Bioengineering Findings

Remarkably, these intraoperative bioengineering measurements showed that the mechanical properties of the human aorta deteriorate dramatically at precisely the same diameter—6 cm—that studies on the natural history of thoracic aortic aneurysms [5–8, 34] had identified as critical. Studies of the mechanical properties of the thoracic aorta provide independent engineering justification for our clinical intervention criteria. At 6 cm, the aorta is no longer distensible and thus becomes essentially a non-distensible rigid tube manifesting malignant behavior such as rupture or dissection [1, 10, 29, 35].

10.6 Ex Vivo Biaxial Stretch Mechanics of the Thoracic Aorta

There are limited data on the mechanical properties of the aneurysmal ascending aortic wall subjected to planar biaxial testing, as most studies of this type have relied on uniaxial deformation of aortic wall specimen. However, in real life the aortic wall deforms in both longitudinal and circumferential directions, which is why testing the aortic samples in biaxial deformation is important for complete understanding of the mechanical behavior of the aortic wall. Dr. Wei Sun and colleagues have developed a technique for biaxial testing of samples of aneurysmal aortas that are obtained intraoperatively during aneurysm resection [24, 25], illustrated in Fig. 10.6 and Video 10.1 (see online supplementary material).

In collaborative experiments with Dr. Sun, we aimed to perform a predictive biomechanical analysis of ascending aortic tissue samples to assess the risk of rupture. Once again we were

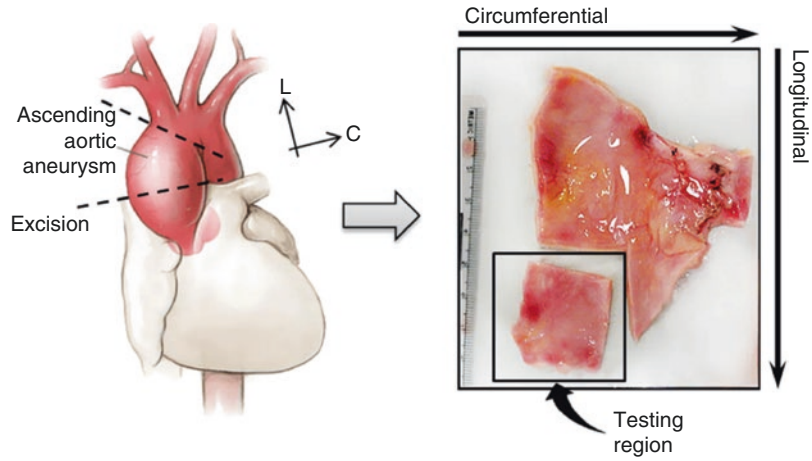

Fig. 10.6 Illustration of a representative testing sample and its orientation with respect to the excised region of ascending aortic aneurysm. *C* circumferential, *L* longitudinal (Reprinted with permission from [25])

able to demonstrate a close correlation of engineering findings with the natural behavior of the aortas in our patients. By combining the uniaxial and biaxial testing techniques, we were able to plot the ultimate tensile strength values (found in uniaxial experiments) into equations modeled from biaxial stretch experiments [24, 25, 36]. This analysis gave us an exact prediction of when aortic dissection or rupture would occur in a particular patient (Fig. 10.7). Please note that a patient would experience an ascending aortic dissection at a blood pressure of 168 mm Hg when the aorta is 5.29 cm in diameter. Rupture would have occurred at a diameter of 5.47 cm and a blood pressure of 263 mm Hg [24, 36]. Similarly to the biomechanical studies performed in vivo, these experiments correlate well with our clinical recommendation to perform prophylactic surgery on patients with ascending aortic aneurysm before they reach the 5.5 cm size.

Bicuspid aortic valve [37, 38] and a bovine aortic arch anomaly [39, 40] have been associated with increased risk of aortic dilation and aortic dissection. Studies of the mechanical properties of human ascending aortic specimens conducted by our group via planar biaxial testing did not reveal a significant difference of the mechanical properties of the aortas with or without these conditions [24, 25]. On the other hand, a recent study by Forsell et al. [41] that compared the mechanical properties of the aneurysmal ascending aorta from patients with trileaflet (TAV) and bicuspid aortic (BAV) valves found that the BAV ascending aorta was significantly stiffer than the TAV aorta. The authors attributed this to increased collagen-related stiffness [41]. This study used uniaxial tensile strength measurements, which were input into a finite element model, although the sample size was relatively small (13 BAV and 11 TAV patients). The authors suggest that the mechanism of aneurysm formation is clearly distinct between the BAV and TAV patients and involves differences in collagen turnover and composition [41–43].

10.7 Measuring Aortic Wall Shear Stress Using 4D PC-MRI

Most recently, high-resolution, time-resolved, three-dimensional (3D), three-directional velocity encoded, radially undersampled phase-contrast MR sequence (4D PC-MRI) (Video 10.2 in online supplementary materials) has been used to estimate aortic wall shear stress in non-aneurysmal ascending aortas [44, 45] (with and without BAV) [46] as well as in patients with ascending aortic dilatation [45, 47] and aortic dissection [48].

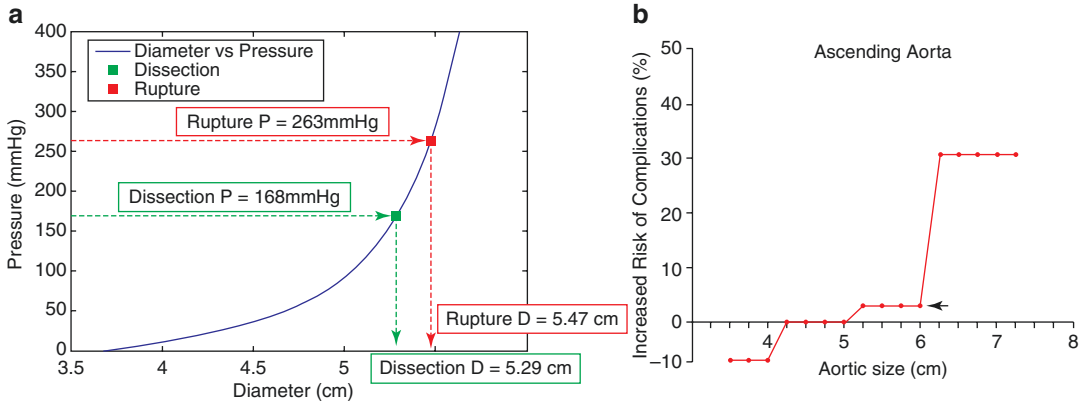

Fig. 10.7 Panel **a**: engineering analysis combining ultimate tensile strength from uniaxial testing with mathematic modeling from biaxial testing permits "prediction" of exact sizes at which rupture and dissection would have occurred (Video 10.1) (Reprinted with permission from [36]). Panel **b**: note that the predicted dissection and rupture points for the patient illustrated in panel **a** fall exactly in the size range for adverse events (arrow) found in our previous clinical studies (Reproduced with permission from [5])

In normal, non-aneurysmal aortas, the time-averaged wall shear stress was found to be greatest on the inner and outer curvature of the ascending aorta and lowest on the left and right walls [44]. At the same time, in patients with a dilated ascending aorta, the peak systolic wall shear stress in the ascending aorta and aortic arch was significantly lower than in healthy controls [45]. The same study also found that the incidence of alterations in blood flow patterns, such as the presence of vortex and supra-physiologic helical flow, was significantly increased in the setting of a dilated ascending aorta. The strength of the ascending aortic supra-physiologic-helix and vortex formation increased directly with an increase in ascending aortic diameters [45]. In contrast, during diastole, the wall shear stress in the setting of ascending aortic dilatation is increased (compared to non-dilated aortas) [47]. Interestingly, a study that compared quantitative flow metrics in patients with normal-size ascending aorta with or without BAV (mean aortic size in these groups was 2.8 ± 0.8 cm and 2.2 ± 0.6 cm, respectively) found reduced wall shear stress in the BAV group [46]. Thus, the authors raise the question of whether a dilated ascending aorta causes deranged flow, or, alternatively, whether deranged flow (through a BAV) causes the aorta to dilate [46]. Clearly further understanding of the cumulative changes in aortic wall strain and wall shear stress will enhance our understanding of the mechanics and pathophysiology of aortic dilatation.

10.8 Biomechanics of Aortic Dissection

Aortic dissection is the most common lethal complication of an ascending aortic aneurysm. A dissection is the splitting of the layers of the aorta that, by conventional understanding, usually starts with a tear in the intimal layer, which then propagates and dissects (splits) the medial layer of the aortic wall. As a result, a pressurized false lumen is created that is separated from the true lumen of the aorta by the "dissection flap" [1, 3]. From a biomechanical perspective, the aortic dissection is a separation or delamination of the elastic layers of the degenerated aortic wall that occurs when the hemodynamic loads exerted on the aneurysmal wall exceed the bonding forces that normally hold the layers together [49]. To date, the exact mechanics involved in the development of an aortic dissection are not completely clear, although several studies have shed light on the possible underlying mechanisms [50–55].

One recent study evaluated the delamination strength of the aneurysmal ascending aorta, in patients with BAV or TAV, in comparison with a normal-size (non-aneurysmal) aorta. The authors showed that aneurysmal aortas had significantly lower delamination strength than non-aneurysmal aortas. The same study also showed that the intimal half of the delaminated/dissected aorta is significantly weaker than the outer half that is attached to the adventitia [49]. Humphrey et al. [56] proposed a new hypothesis of initiation and propagation of aortic dissection (Fig. 10.8). Their hypothesis suggests that the pooling of glycosaminoglycans and proteoglycans may contribute to an initial delamination within the media due to a combined localized loss of tensile strength, genesis of stress concentrations, and increased swelling pressures, which in turn could promote dissections in the thoracic aorta [56, 57]. This hypothesis also challenges the generally accepted concept that the dissection starts with a tear in the intima and the false lumen is then generated under the action of blood pressure. This novel hypothesis suggests that the dissection might start with an initial delamination process of the media, which in turn propagates proximally or distally, subsequently creating an intimal tear. Such pooling of glycosaminoglycans and proteoglycans could be related to the dysregulation of transforming growth factor beta (TGF-β) [57–59]. The hypothesis is that aortic smooth muscle cells increase their production of glycosaminoglycans and proteoglycans in response to increased TGF-β activity [56, 57].

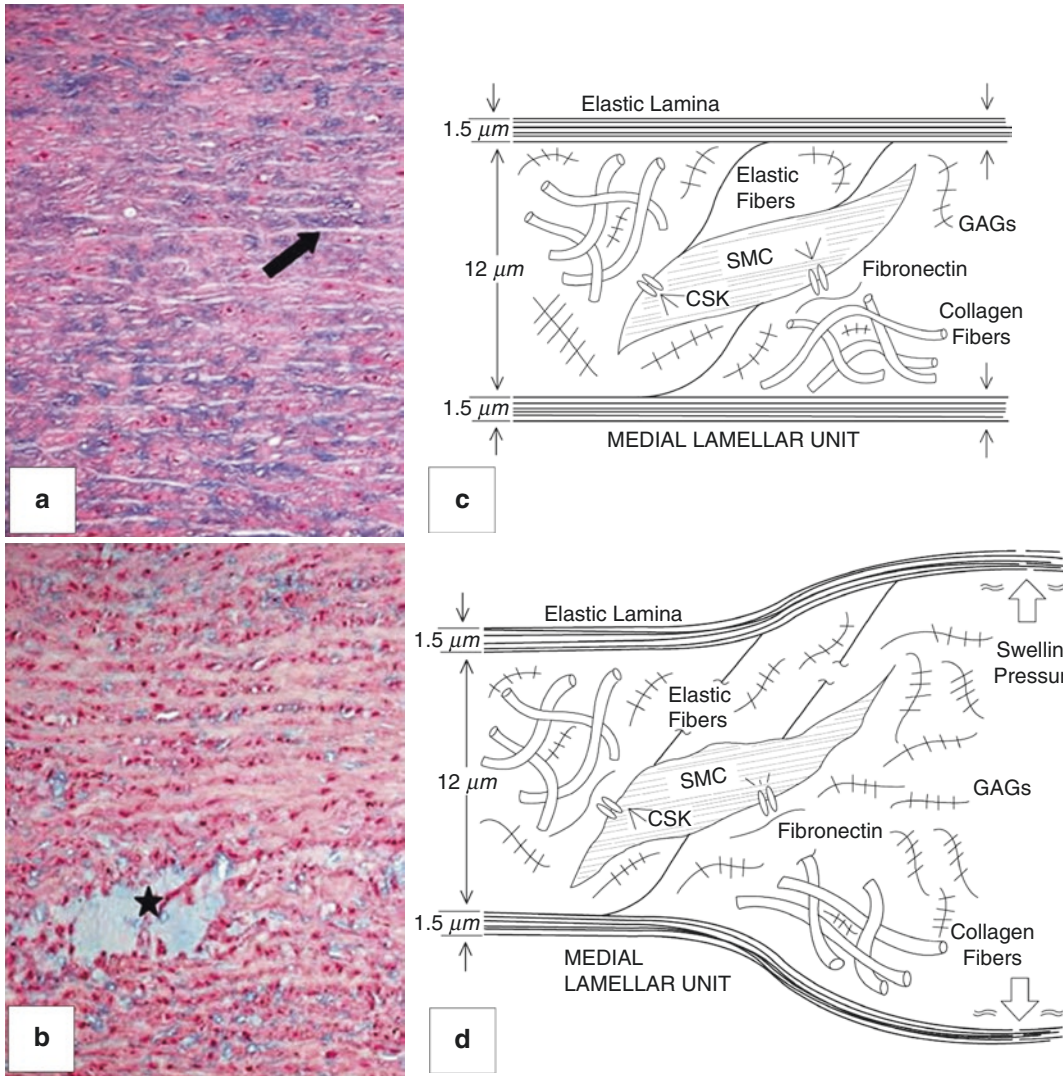

Fig. 10.8 This comparative illustration summarizes the hypothesis that pools of glycosaminoglycans/proteoglycans initiate the dissection from within. Panels **a** and **b**: sections of the medial layer of the human ascending aorta stained with Alcian blue (which stains glycosaminoglycans blue) for both a normal (panel **a**) and an aneurysmal (panel **b**) aortic wall. (Reproduced with permission from [75]). Panel **c**: Schematic drawing of a normal aortic medial lamellar unit consisting of paired elastic laminae (at the top and bottom) with an embedded smooth muscle cell (SMC) as well as collagen fibers, adhesion molecules (e.g., fibronectin), and glycosaminoglycans (GAGs) (not to scale). Shown, too, are thin "radially oriented" elastic fibers (i.e., elastin and associated microfibrils, predominately fibrillin-1) that may provide direct mechanical connections between the elastic laminae and smooth muscle cell and thus a mechanosensory capability beyond the typical cytoskeletal (CSK)—integrin—extracellular matrix (fibronectin/collagen) axis. It is possible that negatively charged GAGs sequester water and may thereby contribute a normal intralamellar pressure that could help maintain the thin elastic fibers in tension. Panel **d**: This schematic drawing depicts a localized accumulation of GAGs, on the right side of the medial lamellar unit, which results in an increased swelling pressure, which in turn helps to separate the elastic laminae and possibly disrupt connections between the SMCs and either thin elastic fibers or the collagenous matrix. Such effects could initiate a local delamination and/or altered mechanosensitive cellular response leading to dysregulated wall homeostasis (Panels **c** and **d** are reprinted with permission from [57])

10.9 The Aging Aorta

It is well known that as the aorta ages, it becomes stiffer, thus losing its elastic properties and, subsequently, dilates [60]. Both of these phenomena are most pronounced in the proximal aorta [61]. The histologic aging changes that occur in the aorta are listed in Table 10.1 [62]. Such age-related remodeling affects the intimal layer, which becomes thicker due to accumulation of disorganized collagen [28]. However, there is evidence to suggest that aging also affects the medial layer of the aorta—the elastic lamellae fracture [63] and thin [60]. As a result the lamellae become separated by increasing amounts of material that is not load-bearing. All these factors contribute to stiffening of the aorta and its dilation [60]. Interestingly, some studies have shown that young females have more compliant arteries than men, while older females tend to have much stiffer arteries than age-matched male individuals [64, 65].

Previous studies have shown that in youth the abdominal aorta dilates approximately 10% with every heartbeat [66], which is primarily accomplished via stretching of the elastin. Our own studies have shown that the ascending aorta expands by 9.2% between diastole and systole in young male patients, and this value decreases with age to about 5.6% in male patients of advanced age [67]. This implies that the elastin, the half-life of which is several decades, might be subject to the so-called material fatigue, a term used in engineering science that explains why physical materials exposed to repetitive cycles of bending or stretch can fracture or break. As O'Rourke states in his review of aging changes to the aorta [60, 61], the phenomenon of material fatigue is often overlooked by many biologists who concentrate on living cells, whose components are turned over on a regular basis. Thus, it is possible that principles of fatigue and fracture might very well apply to elastin, as well as to all nonliving material structures in the human body [68, 69].

Multiple studies have investigated the effects of aging on the properties of the ascending aorta based on *in vivo* imaging diagnostic studies (echocardiography, CT, MRI), which provide additional evidence to support the phenomena of stiffening, dilatation, and elongation that occur in the aging aorta [70–74]. Our group conducted a study [67] to investigate the age-dependent in vivo mechanical properties of the ascending aorta in males (all individuals had no known aortopathies) by analyzing images of multiphase CT scans taken at different time points with 3D reconstructions. We assessed the following mechanical characteristics under physiological loading conditions: circumferential strain, pressure-strain modulus, and wall tension. Our measurements showed a significant negative linear correlation between the peak circumferential strain and patient age (Fig. 10.9a), as well as significant positive linear correlations between the mean diameter of the ascending aorta, systolic wall tension, and pressure-strain modulus and the patient age (Fig. 10.9b–d) [67]. The mean pressure-strain modulus was found to be significantly less for young individuals (30–49 years), compared with those of middle (50–59 years) and advanced age (60–79 years). Pressure-strain modulus was significantly less in normotensive individuals (systolic blood pressure < 140 mm Hg) than in hypertensive individuals (systolic blood pressure ≥ 140 mm Hg) (Fig. 10.10) [67].

Table 10.1 Aging changes in the aorta (Reproduced with permission from [62])

Major macroscopic and histologic features
• Progressive and linear increase in diameter with age
• Scattered or diffuse fibrous intimal thickening
• Fragmentation of elastic lamellae with widening of interlamellar spaces
• Focal amyloid deposits
• Thickening of walls of vasa vasorum

10.10 Conclusions

The biomechanics of the aortic wall are complex due to multiple structural and functional factors, which are being investigated through *ex vivo* mechanical testing and *in vivo* modeling using data from high-quality imaging studies. At the

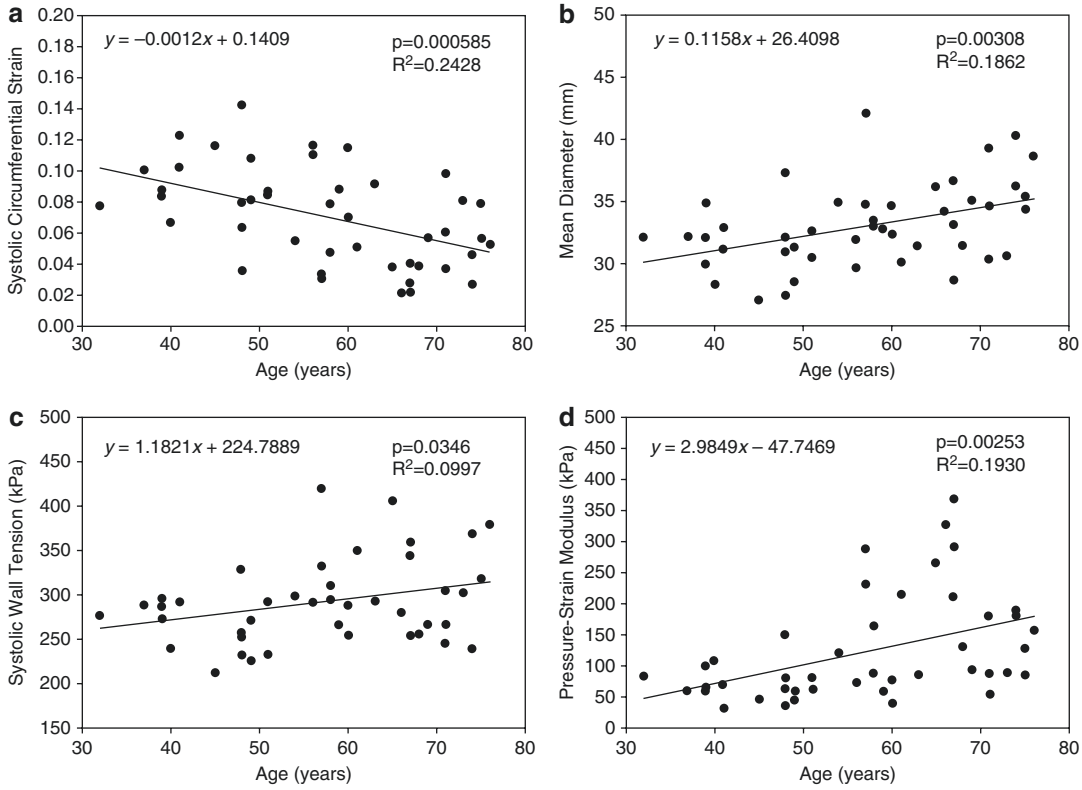

Fig. 10.9 Raw (**a**) peak circumferential ascending aortic strain, (**b**) mean diameter, (**c**) systolic wall tension, and (**d**) pressure-strain modulus data plotted versus age with linear regression lines (Reprinted with permission from [67])

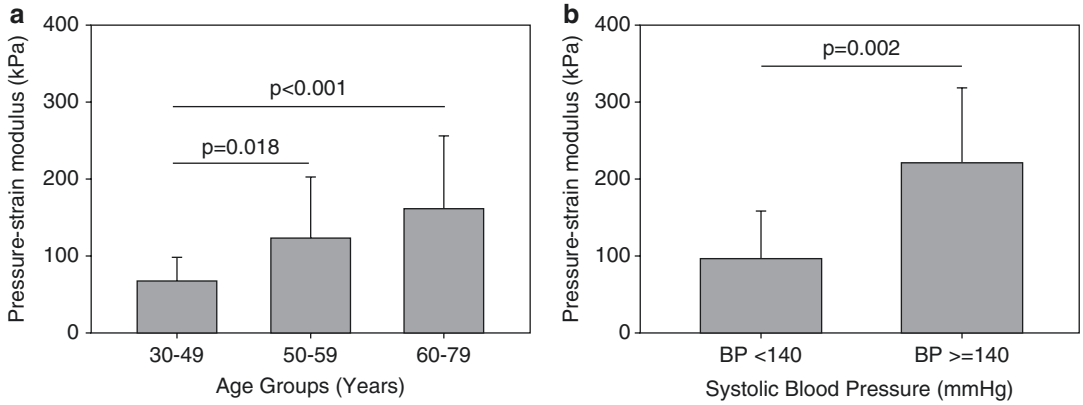

Fig. 10.10 Comparison of the mean pressure-strain modulus value between (**a**) patients in each of the three age groups and (**b**) normotensive and hypertensive patients (Reprinted with permission from [67])

same time, it is fascinating to see how well the engineering findings correlate with the empirically observed clinical data on the natural behavior of aortic aneurysm. Such confluence of data increases the confidence of the findings and provides stronger evidence for the accepted data-driven guidelines for management of thoracic aortic disease.

Looking forward, we feel that the potential to analyze accurately the hemodynamic forces and biomechanical properties of the thoracic aortic wall that potentially contribute to aneurysm, growth, dissection, and rupture will be useful in optimizing management strategy for each individual patient. The appropriate time for surgical intervention will be more precisely determined. Together with rapidly advancing knowledge of the genetic causes of thoracic aortic aneurysm and dissection, detailed and accurate determination of the biomechanical properties of the aortic wall will enable the physician to establish and execute personalized care for each individual patient.

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