Mechanical assessment of arterial dissection in health and disease: Advancements and challenges

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Arterial dissection involves a complex series of coupled biomechanical events. The past two decades have witnessed great advances in the understanding of the intrinsic mechanism for dissection initiation, and hence in the development of novel therapeutic strategies for surgical repair. This is due in part to the profound advancements in characterizing emerging behaviors of dissection using state-of-the-art tools in experimental and computational biomechanics. In addition, researchers have identified the important role of the microstructure in determining the tissue’s fracture modality during dissection propagation. In this review article, we highlight a variety of approaches in terms of biomechanical measurements, computational modeling and histological/microstructural analysis used to characterize a dissection that propagates in healthy and diseased arteries. Notable findings with quantitative mechanical data are reviewed. We conclude by discussing some unsolved problems that are of interest for future research.

1. Introduction

Arterial dissection is a complex and severe vascular pathology which may occur spontaneously or non-spontaneously as a result of trauma (Lee et al., 2006; Sommer et al., 2008). As a diseased condition, tissue dissection has been observed in several arterial branches including the thoracic and abdominal aortas, carotid arteries and coronary arteries. According to clinical analysis (Davies et al., 2002; Knipp et al., 2007), the annual occurrence of aortic dissection is approximately 5–30 cases per million of the population; however, the mortality rate during the first 24–48 h are high if patients are not treated appropriately. Thus, a careful radiologic follow-up and preemptive surgical intervention is often required. It is well-known that arterial dissection frequently initiates from an intimal tear (Thubrikar et al., 1999) or from a perforation of the intima caused by intramural hemorrhage and hematoma formation (Khan and Nair, 2002). This kind of intimal defect may continue propagating in the radial direction, then through the media, and may cause delamination of arterial layers. One of the most significant clinical consequences due to dissection propagation is the creation of a false lumen, which may affect the hemodynamics and may change the distribution of mechanical stresses on the underlying arterial wall. More importantly, the false lumen narrows the true lumen and decreases oxygen supply from the blood flow to the tissues. Thus, both the mechanical properties and the structural stability of the local arterial wall might alter.

In the human vasculature, tissue dissection is fatal when it propagates, in particular, by running to the adventitial side to cause an acute rupture of the arterial wall (Mokri, 1990; Kraus et al., 1999; Schievink, 2001). This severe case is relatively rare (Criado, 2011), but it is difficult to be diagnosed in clinical practice. It should be noted that most factors which contribute to the initiation of arterial dissection are trauma-related. Other predisposing factors can be pathological changes due in large part to vascular hypertension and atherosclerosis (Pratt and Curci, 2010). Recently, pooling of glycosaminoglycans in the aortic media is suggested to cause stress concentrations and to increase intramural swelling pressures, both of which could result in local tissue delamination (Humphrey, 2013; Roccabianca et al., 2014). Moreover, catheter-based interventional treatments of arteries have been identified as an alternative trigger to initiate dissection (Humphrey, 2002). For example, balloon angioplasty leads to denudation of the endothelium, disruption of the intima, fracture of the atherosclerotic plaque, and overstretch of the non-diseased portions of the wall. Therefore, severe tissue injuries may be a consequence followed by this interventional procedure.

Although the underlying mechanism is still elusive in many aspects, dissection in the cardiovascular system involves a complex series of coupled biomechanical events (Sommer et al., 2008; Tong et al., 2011b, 2014; Wang et al., 2014). The mechanical properties of arterial walls, which are predominated
by the structure of the media and the adventitia (Humphrey, 2002; Schrieffl et al., 2012; Holzapfel et al., 2015; Niestrawska et al., 2016), vary with age, disease and location. These factors certainly affect the load-bearing capacity of the arterial wall, leading to changes in the delamination strength within or between different arterial layers. From a biomechanical point of view, dissections are primarily attributed to intralamellar failure of main load-bearing microstructural components within the arterial wall, i.e. elastin and collagen fibers (Pal et al., 2014). To understand the intrinsic mechanism and patterns in propagating a dissection within a tissue, using state-of-the-art tools in experimental and computational biomechanics in order to investigate dissection properties is of fundamental importance.

In the past decade, quantitative measurements have been carried out to determine dissection properties of diverse arterial tissues (Sommer et al., 2008; Tong et al., 2011b; Wang et al., 2011; Pasta et al., 2012; Wang et al., 2013, 2014; Tong et al., 2014; Sommer et al., 2016). Various biomechanical data have advanced our understanding of pathogenesis and have provided helpful insights into prevention and surgical treatment. The main goal of this paper is to review and summarize key advances in the mechanical characterization of tissue dissection associated with the microstructure and morphology of arteries in health and disease. In particular, we focus on (i) the experimental investigation and modeling of dissection in healthy arteries, (ii) the dissection properties of arteries in disease with particular emphasis on aortic aneurysms, (iii) the microstructure and morphology of dissected arterial tissues. A variety of aspects including experimental, computational and histological/microstructural contributions spanning the last 20 years are considered. Based on the provided review, we finally point to some challenges which may be relevant in future research.

2. Dissection in healthy arteries

2.1. Experimental investigations, related interpretations

Dissection is defined as a characteristic form of trauma involving laceration and/or cleavage of the arterial wall. Despite primarily along the medial plane, in reality a dissection is the consequence of a three-dimensional crack propagation in a tissue. Thus, the created fissures may run either to the luminal side or to the adventitial side to form a false lumen or to cause a rupture through the adventitia, both of which are potentially lethal (Khan and Nair, 2002).

The mechanics underlying arterial dissection remains largely unclear and the quantified biomechanical data are rare. Previous experimental studies focussed on the aortic (medial) dissection using both human and porcine samples. An early study by Carson and Roach (1990) measured the strength of the porcine aortic media and evaluated its role in the propagation of the aortic dissection. According to their pressure-volume measurements, the mean peak pressure to tear the thoracic aortic media was quantified to be 772 ± 1.5 kPa and the work per unit area of tissue required to propagate a tear in the aorta was 15.9 ± 0.9 mJ/cm². Later, Tiessen and Roach (1993) focused on the evaluations of physical factors in the initiation and propagation of dissections in human aortas. These authors found that dissections occurred at a very high non-physiological mean pressure of 79 ± 29 kPa. Both age and tear depth had no significant effect on the medial strength of the human aortas. However, gender, location and atherosclerotic plaque formation significantly influenced the pressure values to initiate aortic medial dissection. Using a similar experimental approach, Roach and Song (1994) investigated variations of the dissection work as a function of location based on 17 porcine aortas. Notably, the work/area ranged from 1.88 ± 0.98 mJ/cm² for the upper abdominal aorta to 11.34 ± 4.05 mJ/cm² for the lower abdominal aorta. Such a variation can be partially attributed to structural differences in the elastin pattern and collagen structure of abdominal aortas.

Another important work regarding aortic dissection was conducted by Tam et al. (1998), who investigated the effects of tear depth on the propagation pressure of aortic dissections. The depth of tear was determined as the ratio of elastin layers in the intact wall. The propagation pressure was strongly positively correlated with the number of elastin layers in the dissected wall (Fig. 1(a)) but significantly inversely correlated with the tear depth (i.e. with tear depths near 1 requiring the lowest pressures) (Fig. 1(b)). In contrast to fluid infusion, each aorta in this study was pressurized under static (no-flow) conditions until propagation occurred.

In addition to these studies, Rajagopal et al. (2007) linked abnormality of aortic mechanical anisotropy and geometry to the occurrence of dissection. In their opinion, the mechanical issue can be answered by detailed imaging of the aorta. Therefore, in combination with a hemodynamic risk assessment, image analysis may have the potential to prospectively identify patients at high risk for future aortic dissection and, thereby, facilitate prophylactic intervention. Moreover, Sommer et al. (2008) provided a series of new experimental methods, i.e. direct tension (DT) and peeling tests, to quantitatively determine the dissection properties of the aortic media. A DT test measures the mechanical strength of the tissue components in the radial direction, while a peeling test quantifies the fracture energy required to propagate a dissection in the tissue (see Fig. 2). The peeling protocol is predominantly controlled by pulling two ‘tongues’ of a tissue strip with an initial incision (see Fig. 1). The open circles represent the ruptured aortas. Bars represent standard deviations. Reproduced from Tam et al. (1998).

![Fig. 1](image-url)
the 3D sketch in Fig. 2(b)). DT tests of 8 coin-shaped medial specimens resulted in a radial failure stress of 140.1 ± 15.9 kPa. The dissection energies per reference area during the circumferential and axial peeling were 5.1 ± 0.6 mJ/cm² and 7.6 ± 2.7 mJ/cm², respectively (according to Sommer et al., 2008, the dissection energy is determined by subtracting the elastic stored energy just before complete separation of the specimen – from the external work). The anisotropic dissection properties for the aortic media are mainly attributed to the alignment of structural components such as collagen fibers and smooth muscle cells. Note also that the exact location of the dissected surface during a peeling test is much dependent on where the initial cut is induced to the tissue.

Besides aortas, the dissection properties of some other arterial branches were also investigated. Tong et al. (2011b) reported a set of dissection data on tissues obtained from human carotid bifurcations which are at a prominent site in the cardiovascular tree. Specifically, the data encompass dissection propagating not only within the media but also at/around the interfaces between two arterial layers. Moreover, a significant regional variation in the dissection energy is observed for the media of common carotid arteries with respect to internal carotid arteries. The dissection energy is observed for the media of common carotid arteries for different protocols are summarized in Table 1.

2.2. Mechanical modeling

Compared to experimental investigations, mechanical modeling of arterial dissection is relatively rare in the literature. To date only a few (mainly) computational models are available which allow the study of arterial dissection from a mechanical point of view. Gasser and Holzapfel (2006) developed a non-linear continuum framework to model dissection propagation and tissue failure of aortic media strips during peeling. Notably, the aortic media is modeled as a fiber-reinforced composite in which collagenous fibers are assumed to be embedded in a non-collagenous isotropic ground matrix. The constitutive descriptions for the continuous and the cohesive materials are considered to be independent from each other. The continuum framework and the related numerical implementation serve the basis to better analyze 3D propagation of arterial dissection at finite strains and to predict the damage state in the cohesive zone. Based on cohesive theories of fracture, Ferrara and Pandolfi (2010) have proposed and calibrated a numerical model of arterial dissection which was used to characterize the critical mechanical conditions for the interlamellar propagation of a tear in the media and to study the influence of the collagen fibers on the separation process. A more recent computational model by Wang et al. (2015) was developed to propagate a tear in fiber-reinforced soft tissue undergoing finite deformations. The calculation of the energy release rate for a two-dimensional tissue strip with a pre-existing tear under internal pressure was presented, and the effect of the fiber orientation shown.

In fact, the microstructure such as collagen fiber arrangement is of utmost importance to control dissection propagation within arterial tissues. Since the disruption of elastin and collagen fibers are present along the dissection planes, Pal et al. (2014) assumed that the radically running collagen fibers embedded within the matrix may act as ‘bridges’ between the dissected layers. A schematic representation of fiber bridge and its failure during an artificial dissection is shown in Fig. 4. To link the fiber microstructure to the delamination strength, Pal et al. (2014) further proposed a predictive mechanistic model to investigate the role of
energy of the radially running collagen walls. The results indicate that both the density and the failure the fiber microstructure on the delamination strength of aortic walls. The results indicate that both the density and the failure energy of the radially running collagen fibers determine the delamination strength. To our knowledge, this is the first modeling paradigm which relates collagen fiber arrangements to the dissection properties. Although the model is only validated based on data obtained from protocol-controlled peeling tests, the study advances our understanding of contributing factors that predispose to dissection development and propagation. Together with improved imaging techniques, this mechanistic framework can further develop a promising predictive model to characterize emergent behaviors of dissection for clinical utility.

Moreover, computational fluid dynamic (CFD) analysis is of particular interest to explore the underlying hemodynamic mechanism involved in the dissection propagation. By assuming aortas as simplified 2D pipes, Chitsaz et al. (2012) performed CFD modeling to determine the effects of the pressure distribution on the tearing force, which is the force required to separate the dissected flap from the rest of the wall. These authors found that the tearing force is proportional to both the rate of luminal pressure increase and the dissected length. More importantly, flow patterns do affect the magnitude of the tearing force; for example, the tearing force under steady flow is much lower than that under pulsatile flow. Based on three-dimensional patient-specific aortic geometries, Zhang et al. (2014) suggested that the pressure gradients in the true and the false lumina cause imbalance of pressure between two lumens, contributing to cleavage force within the aortic structure and the longitudinal dissection propagation. Findings from the above hemodynamic simulations are essential in supporting hypothesis that sudden changes in intraluminal pressure are a key trigger to initiate and propagate a dissection. From a clinical perspective, there is a need to examine whether blood pressure abnormality of patients is significantly correlated with propensity for dissection occurrence.

3. Dissection properties of arteries in disease

Due to vulnerability, tissue in vascular pathologies may have a higher propensity to initiate a dissection which frequently results in life-threatening clinical events. In particular with a disorganized microstructure, patterns in propagating a dissection within...
diseased tissues may vary apparently when compared to those in health. Hence, the investigation of the delamination strength of diseased arteries offers the potential to refine our understanding of dissection-induced tissue failure, and to predict more reliably the rupture risks. In this section we focus on quantitative dissection measurements with respect to dissected thoracic aortas, aortic aneurysmal walls and atherosclerotic plaques.

A devastating separation of aortic layers and a dissection within aortic aneurysms occur when the hemodynamic loads exerted on the diseased wall exceed the adhesive strength between the layers. Compared to healthy aortic tissues, aortic aneurysmal dissections are rarely studied from the biomechanical perspective. To the best of our knowledge, the first study in this field was conducted by Pasta et al. (2012) who used the same experimental protocol as Sommer et al. (2008) (peeling tests) to quantify and compare the dissection properties of non-aneurysmal with aneurysmal human ascending thoracic aortas with bicuspid aortic valve (BAV) morphology or with tricuspid aortic valve (TAV) morphology. Their results indicate that aneurysmal tissues with BAV or TAV have significantly lower delamination strength than tissues from non-aneurysmal thoracic aortas, suggesting a greater propensity and risk of aortic dissection. Note, however, that these data are not representative of any spontaneous initiation of aortic dissection in vivo. In addition, rupture of elastin and collagen fibers that occurs during a peeling test is mainly caused by tensile stresses between the two dissected halves. Hence, a peeling protocol provides little information about the role of shear stresses in the formation of arterial dissection. In fact, shear stresses are considered as an important contributor to propagate a dissection in vivo and may lead to a different failure mode. A recent study by Sommer et al. (2016) developed novel biomechanically based strategies to characterize dissection pattern of tissues and failure modes in response to shear deformation in different orientations regarding the aorta’s orthotropic microstructure. Fig. 5 shows shear responses in the $\theta$z-plane of the tissue (‘in-plane’) where $\theta$ characterizes the circumferential direction of the aorta and $z$ the longitudinal direction. In particular, shear responses in the circumferential (r0-mode) and longitudinal (rz-mode) directions of medias taken from dissected ascending thoracic aortas are depicted. As can be seen, the ultimate (Cauchy) shear stresses and the related amounts of shear in the longitudinal direction are higher than in the circumferential direction under ‘in-plane’ shear loading which reveals anisotropic shear failure properties of the media (detailed numbers of the ultimate shear stresses and the corresponding amount of shear values are documented in Sommer et al., 2016). Recently Haslach et al. (2015) performed shear tests on rectangular aortic wall blocks observing that interstitial fluid is redistributed with shear deformation which suggests that no simple modification of classical rupture theories suffices to predict the rupture of hydrated soft biological tissue.

In contrast to ascending thoracic aortic aneurysms (ATAAs), abdominal aortic aneurysms (AAAs) have pronounced differences in histopathological characteristics such as inflammation (Davies, 1998; Kazi et al., 2003). It is well-known that the AAA wall is a type of degenerated abdominal aortic wall which may also be associated with atherosclerosis. Although dissection involved in the AAA is clinically rare compared with the dissection of the ATAA, relevant data can be used as a surrogate measure for the propensity to aneurysmal rupture. Tong et al. (2014) accomplished a quantitative analysis in regard to dissection properties of AAA walls, which were anatomically separated into two parts, i.e. adventitia and intima-media composite. The adventitia, in general, shows the higher mean dissection energy values in both the circumferential and the longitudinal directions, when compared to the associated intima-media composite. Unlike the healthy aortic media, anisotropy is not shown in the dissection properties of the atherosclerotic intima-media composites. More importantly, the dissection properties of the diseased aortic walls are found to be rate dependent. For example, the force/width values with the extension rate of 1 mm/s were on average 28% (SD 9%) higher than those quantified by using 1 mm/min. However, whether a similar result can be obtained based on healthy aortic tissues remains unknown.

In addition to aortic aneurysms, measurements of the adhesive strength between the fibrous cap and the underlying arterial wall offers a helpful metric to assess plaque stability. In this regard, Wang et al. (2011) quantified a local energy release rate that is required to peel off plaques from the internal elastic lamina, i.e. at the plaque-wall interface. The entire experiment is conducted based on apoE-KO mouse aortas and, in general, the quantified dissection energies are comparatively lower than those of healthy or aneurysmal human aortic tissues (Roach and Song, 1994; Sommer et al., 2008; Tong et al., 2014). One typical feature for atherosclerotic plaques is the presence of lipids which are supposed to affect plaque-wall bond strength. Therefore, an uneven distribution of lipids at a specific region will result in considerable variation in the measured local energy release rate. To correlate dissection data to lesion compositions, a later study by Wang et al. (2013) found that the plaque-wall adhesive strength varies with local collagen content of plaques and elastin fragmentation of the underlying arterial wall. Specifically, these authors linked the mechanical data points to the related histological sections along the plaque length for a thorough investigation of structure–mechanics relationship. It should also be emphasized that the collagen content in this work is expressed as a percentage of collagen area to total plaque area via histological sectioning and staining. In the authors’ opinion, however, this quantification

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**Fig. 5.** Cauchy shear stress vs. amount of shear during ‘in-plane’ shear tests of medias taken from dissected ascending thoracic aortas: (a), (b) ‘in-plane’ shear behavior in the circumferential (rz-mode) and longitudinal (rz-mode) directions, respectively. Reproduced from Sommer et al. (2016).
method needs to be improved as examination within a 2D plane may not be accurately representative for the 3D collagen content. Thus, we suggest that mass fraction analysis based on an entire sample is more appropriate (Tong et al., 2013). The quantified dissection energies of diseased arteries within the reviewed studies are summarized in Table 1.

Besides peeling experiments, the previously mentioned DT tests are also utilized to measure mechanical adhesive strength of the arterial tissues associated with atherosclerotic plaques, see, for example, Tong et al. (2011b). One limitation for the DT test in comparison with peeling is that it cannot characterize tissue delamination during a continuing propagation but it rather represents a fast de-adhesion of the tissue within a small area. Specifically for diseased arterial tissues where significantly regional variations exist, it is always required to perform multiple DT tests in various regions to comprehensively evaluate adhesive strength for the whole tissue piece.

4. Microstructure and dissected morphology

It has been long suggested that the microstructure plays a pivotal role in governing the mechanical behavior of arterial walls in response to physiological loads (Holzapfel et al., 2000; Humphrey, 2002; Holzapfel and Ogden, 2010). According to our knowledge dissection mainly occurs and propagates within the media of the artery and needs a higher fracture energy value for a continuation in the adventitia (Tong et al., 2014). Therefore, the microstructural organization of the media directly affects the dissection properties of the entire arterial wall. From a histological point of view, the media is a complex three-dimensional network which consists of smooth muscle cells (SMCs) embedded in an extracellular matrix with elastin and multiple types of collagen (types I and III) (Holzapfel et al., 2000; Humphrey, 2002). Within the media of a healthy artery the rubber-like protein elastin, the stiff fibrous protein collagen and the SMCs are found to be organized into lamellar units around 10 μm thick (Wolinsky and Glagov, 1967; Rhodin, 1980). These laminated structures may be prone to separation by creating a cleavage plane parallel to the elastic lamellae (Tam et al., 1998). Hence, the healthy (aortic) media particularly favors the peeling mechanism along its lamellar layers.

Compared to multi-axial extension, compression and shear tests, peeling of arterial tissue primarily occurs within the plane perpendicular to the radial direction, that is, in parallel to the lumen. As two main components, alignments of collagen fibers and SMCs essentially contribute to the resistance of a dissection propagation. In particular, image analysis has shown that the collagen fibrils in the healthy aortic media are generally aligned closer to the circumferential orientation (Schriefl et al., 2012). This particular microstructural feature for healthy arteries is thought to be the main cause for the anisotropic dissection properties of the tissue. Moreover, the directional alignment of SMCs and cellular heterogeneity of the vascular media are in part responsible for anisotropy (Rhodin, 1980; Seidel, 1997). Consequently, a more pronounced resistance will be encountered if we perform an axial peeling to delaminate arterial tissues. In histology, a remarkably ‘rougner’ dissection surface is thus often generated during the axial peeling of the aortic media, as shown in Fig. 6.

Although most quantified dissection data are controlled by a well-defined protocol in terms of the initial cut location, extension rate, etc., a dissection within one lamella throughout the whole dissection path never occurs. There are always inward and outward deviations from the lamellar layers (Sommer et al., 2008; Tong et al., 2011b; Wang et al., 2014). It indicates that propagating a dissection within arterial tissues involves an oscillatory process even though the extension rate is kept very low (1 mm/min). The relevant damage zone due to a dissection spreads over approximately 6–7 elastic lamellae for aortic media (Sommer et al., 2008), and 3–4 elastic lamellae for the carotid media (Tong et al., 2011b). The circular-shaped nuclei of the SMCs are also observed within the damage zone, as shown by Tam et al. (1998). Moreover, and that is interesting, Sommer et al. (2008) reported that the dissection during the axial peeling sometimes runs towards either the internal or the external elastic laminae, which are defined as the two interfaces (boundaries) among the three arterial layers. This observation pretty much matches the in vivo scenario case, as reported in clinical diagnosis, i.e., running either back into the lumen or outward into the adventitia.

For diseased arteries, a more disorganized microstructure is frequently identified. It includes significant changes in the content of elastin and collagen, realignment of collagen fibers, accumulation of collagen cross-links, and combinations thereof (Carmo et al., 2002; Kazi et al., 2003; Tong et al., 2014). Microstructural analysis (Pasta et al., 2012) particularly emphasized that local breakdown of elastin fibers also exists in the aneurysmal aortas such as the ATAA. These ruptured elastin fibers might lead to cavities on the micro-scale level. The disorganization of elastin and collagen fibers due to vascular pathology directly affects the dissection properties of arteries. For example, the peeling curve to characterize an aneurysmal dissection is highly oscillatory and sometimes is associated with a discontinuous dissection path owing to the resistance of embedded calcified plaques (Pasta et al., 2012; Tong et al., 2011a, 2014); however, this phenomenon seldom occurs during the peeling of healthy aortic tissue. Moreover, atherosclerosis involved in the aneurysmal aortas potentially contributes to the local structural instability and intralamellar tissue de-adhesion (Shah, 2003; Holzapfel et al., 2004, 2014;
Walsh et al., 2014; Tong et al., 2015). Hence, a lower mean delamination strength is quantified to propagate a dissection in the aneurysmal aortas when compared with the non-aneurysmal aortas (Pasta et al., 2012). Regarding dissected morphologies, peeling of aneurysmal aortas generates rougher fracture surfaces than those of the normal aortas (Pasta et al., 2012). In contrast to healthy aortic tissue (Sommer et al., 2008; Tong et al., 2011a; Wang et al., 2014), the roughnesses of these dissected surfaces have no significant distinction between the circumferential and axial directions evidently shown by histology. The similar roughness is probably indicative of isotropic dissection properties for the diseased arteries, which are consistent with the conclusions drawn from peeling experiments (Pasta et al., 2012; Tong et al., 2014).

It is important to note that fiber bridging is a representative failure mode for fatigue delamination in composite materials (Gregory and Spearing, 2004); this failure mode is also applicable to fiber reinforced biological soft tissue. As mentioned previously, fibers embedded in human arteries may act as bridges between the dissected layers. Therefore, the un-ruptured fibers bridges the delamination when the dissection propagates from one fiber-matrix interface to another. As can be seen from scanning electron microscope images of delaminated planes, bundles of ruptured elastin fibers exist in between elastic sheets, essentially supporting such a failure mode (Fig. 7). The elastin fibers across dissected halves are supposed to endure high stretches to resist tissue delamination. However, the eventual failure of the elastin fibers leads to a significant and rapid decrease in the quantified force/width values. Remarkably, this manner repeats throughout the whole process as the dissection path moves forward.

5. Conclusion

In the present review we discussed the basic mechanisms and factors that possibly contribute to the initiation of arterial dissections, and then we summarized a variety of experimental and computational approaches used to characterize the mechanical behavior of tissues during dissection propagation. These approaches included quantitative experimental measurements in terms of the dissection energy using peeling tests and other techniques, mechanical modeling, finite element simulation, and histological/structural investigations of tissues.

As a critical indicator in assessing the propensity of dissection occurrence, the identification of the dissection properties of arteries are of particular importance in the area of vascular biomechanics. Hence, developing improved biomechanical protocols to quantitatively determine the dissection properties is of pressing need. In general, the peeling test has been extensively utilized in previous studies and is regarded as the most effective method which can be easily applied to different types of arterial tissues. With notable technical advances in imaging and multi-scale computational modeling, we are gaining more knowledge of how a dissection initiates due to tissue defects on the micro-scale level, and how dissection properties alter in response to pathological changes. This is helpful to better interpret the intrinsic dissection mechanism for arteries in health and disease.

We conclude by discussing some challenges for possible future work that we believe are exciting to explore. Although previous studies have shown pronounced differences in the changes of the dissection energies with respect to the strain rate, see, for example, Tong et al. (2014), effects of the extension rate on the quantified dissection properties remain largely unknown. In this regard, a systematic investigation based on multiple extension rate values is required. It is of particular interest to examine whether the dissection properties have to be considered as a function of the extension rate applied to peeling. Next, it appears that the dissection properties vary with changes in the microstructure and the mass fractions of elastin and collagen within arterial walls, and these variations correlate with the biomechanical tissue properties. Therefore, investigating how dissection properties alter with changes in the (elastin and collagen) mass fractions and the collagen orientation and dispersion due to pathological conditions is of fundamental importance to better identify potential triggers for arterial dissection in clinical practice. The 3D collagen architecture may be identified by means of, for example, second-harmonic generation imaging, and in this regard dissected aortas can be compared with healthy aortas. Finally, from the modeling point of view, the development of 3D failure criteria for the (diseased) thoracic aorta are of pressing need to be used to perform patient-specific simulations that ultimately aid the clinical decision making.

Conflict of interest statement

None declared.

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