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Mechanical behavior and wall remodeling of blood vessels under axial twist

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Abstract

Blood vessels are often subjected to axial torsion (or twist) due to body movement or surgery. However, there are few studies on blood vessel under twist. This review first summarizes the clinical observation on the twist of blood vessels and then presents what we know about the mechanical behaviors of blood vessel under twist, including the constitutive models. The state of art researches on the remodeling of blood vessels under twist via *ex vivo* organ culture, *in vivo* animal experiments, and mathematical model simulations are further discussed. It is our hope that this review will draw attention for further in-depth studies on the behavior and remodeling of blood vessels under twist.

Keywords

Blood vessel; Axial twist; Mechanical behavior; Remodeling; Model

Arteries and veins *in vivo* are subjected to complex mechanical loads including the tensile stress due to lumen pressure, shear stress generated by blood flow, and the axial stretch due to tissue tethering. These blood vessels often subject to axial torsion, i. e. the twist along its central axis. While extensive studies have been conducted on artery and vein behaviors under lumen blood flow, pressure, and axial tension, little is known about the behavior of arteries and veins under twist.

Accordingly, this review summarizes clinical observation and recent biomechanical studies on the mechanical behaviors and possible wall remodeling of blood vessels under axial twist.

1 Clinical prevalence of artery twisting

Arteries are subjected to twist along their longitudinal axes due to body movement and surgical procedures[1–4], and twisting will increase blood flow resistance and may cause ischemia in organs[5]. The vertebral artery is vulnerable to torsion injury, sudden movement

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of the neck may induce thrombosis and stroke[6]. Rotations of the head also induce considerable torsion in the common carotid and internal carotid arteries[6], and may partially occlude the vertebral artery at the atlantoaxial articulation and lead to vertebrobasilar symptoms[8–9]. The internal jugular vein may also be obstructed by head rotation, leading to the risk of hydrocephalus[10]. Coronary arteries experience bending and twisting due to shape and volume changes of the heart during cardiac cycle[11–14]. In studies for stent design and fracture, Choi *et al*. reported shortening and twisting of abdominal aorta and common iliac artery due to hip flexion[15–16]. Significant shortening and twisting of the superficial femoral artery was observed in old people during hip and knee flexion, due to loss of arterial elasticity[17]. Under maximum knee and hip flexion, the superficial femoral artery might shorten and twist significantly when patient changed from supine position to fetal position[2]. Similarly, position change from straight-leg to crossed-leg would lead to significant changes of length, curvature, and twist in femoropopliteal artery[1].

Blood vessels may also be twisted during surgery. Implantation of vascular grafts and microvascular anastomoses may generate inadvertent twisting of the grafts or native arteries at the anastomosis site[5, 18–24]. Small twisting may not be detectable or lead to severe consequences, but a big amount of twisting may result in kink[22] or a wave shape[18] in thin-walled vessel such as vein and thin-wall grafts. Twist at 180° and above significantly impaired the patency rate for venous microanastomoses, which greatly increases the risk of graft thrombosis[5, 9–20]. Propeller-type flap design has been used in reconstructive surgery[25–29], in which blood vessels are often twisted. Wong *et al*[4]. performed simulation to determine factors that affect the perforator patency of artery and vein when subjected to twist in propeller flaps. They demonstrated that twist angle is an important factor affecting the patency, and suggested the angel to be < 180° to maintain perforator patency.

Twist of blood vessels also occurs in umbilical cord[30–32].

It is of clinical interest and need to determine how arteries behave and remodel in response to axial twist.

2 Mechanical behavior of arteries and veins under axial twist

2. 1 Experimental test and constitutive equation under torsion

In a pioneering work, Deng and colleagues designed a delicate torsion device to measure the shear modulus of rat thoracic aorta under twist[33]. The artery was fixed at one end and the other end was twisted an angle θ. The twisting angle and corresponding torque were recorded and their relationship was examined. This study was the first to find that torque (T) is linearly proportional to twist angle per unit length (θ/L) in a certain range of shear strain in the artery, i. e.

$$T=GJ\frac{\mathrm{d}\theta}{\mathrm{d}L} \quad (1)$$

where G is the shear modulus, and J is the polar moment of inertia of the artery. The shear modulus G was found a function of the average longitudinal and circumferential stresses and was independent of shear stain; it also increased significantly with increasing longitudinal and circumferential stretch ratios, but it did not vary significantly with temperature. The effect of longitudinal and circumferential stress and strain on shear modulus was incorporated in a strain energy function density as[33]

$$\rho^W = q + Ce^Q \quad (2)$$

They modeled the vessel wall as a membrane and given q and Q as

$$Q = a_{11}E_\theta^2 + a_{22}E_z^2 + 2a_{12}E_\theta E_z + a_5 E_{z\theta}^2$$

$$q = b_{11}E_\theta^2 + b_{22}E_z^2 + 2b_{12}E_\theta E_z + b_5 E_{z\theta}^2$$

Accordingly, the shear modulus was given as

$$G = b_5 + Ca_5 e^Q \quad (3)$$

where ρ is the density of arterial wall; C, a' s, and b' s are material constants to be determined from the stress-strain relationship.

Later, Lu and colleagues[13] modeled the porcine coronary artery as a two-layer vessel consisting of the intimamedial and adventitial, and determined the torsional properties of each layer as well as the intact vessel. In addition to observations by Deng *et al*[33], Lu and colleagues demonstrated that shear modulus changes linearly with longitudinal and circumferential stresses but nonlinearly with corresponding strains. Shear modulus of the adventitia layer is bigger than that of the media layer, and their difference increases with pressure and stretch ratio. Van Epps and Vorp[34] adapted Fung strain energy function for modeling coronary arterial wall under twist, which is given as[35–36]

$$W = \frac{1}{2}(Ce^Q - 1) \quad (4)$$

and

$$Q = b_1E_\theta^2 + b_2E_z^2 + b_3E_r^2 + 2b_4E_\theta E_z + 2b_5E_z E_r + 2b_6E_r E_\theta + 2b_7E_{\theta z}^2$$

where C and $b_1 - b_7$ are material constants. Using the relationship between shear modulus and circumferential stress and longitudinal stretch ratio obtained from previous studies, they determined the torsional shear strain parameter b_7 in Fung model. Their in silico triaxial

tests also showed the proportional relationship between shear modulus and longitudinal stretch ratio and pressure. Using Fung model, only one parameter is needed to describe the twist behavior of coronary artery. Our lab also determined the shear constant b_7 of porcine carotid artery by in vitro experiments, and further studied the behavior of porcine carotid artery under twist[37]. We found that the torque could increase linearly with the twisting angle until a sudden drop when the artery lost stability and buckled.

Micro-structurally motivated constitutive equations have been established to model arterial wall behavior under various conditions[38–40]. Recently, Hollander and colleagues[41] developed a purely micro-structural model of porcine coronary left anterior descending (LAD) artery media using the mixture theory, in which both the collagen fiber and non-collagenous matrix are modeled structurally, and the total strain energy function of the artery is the sum of strain energies of fibrous constituents, elastin and collagen, which is given as

$$W=\sum \phi_i^0 \int\int R_i(\Psi,\theta) w_i(e)\mathrm{d}\Psi\mathrm{d}\theta \quad (5)$$

where φ_i^0 is the volume fraction of fiber family i in the reference configuration, $R_i(\psi, \theta)$ is the fiber orientation density distribution function for fiber family i, ψ and θ are azimuthal and polar angles respectively, and $w_i(e)$ is the strain energy function of each fiber family which depends on the uniaxial strain e. Using this model, they determined the torsional stiffness (ratio of torque to twisting angle) of the artery, which is a function of pressure and axial stretch. Their model can also provide good predictions of twist in the LAD media from biaxial inflation and extension tests only. In another study[42] Hollander and colleagues compared models of LAD under twist using the micro-structural model (Eq. 5) with Fung model (Eq. 4) and Holzapfel fiber model, where Holzapfel model is given by[38, 43–44]

$$W=c_1(I_1-3)+\frac{b_1}{2b_2}\sum\nolimits_{\alpha=4,6}\{\exp[b_2(I_\alpha-3)^2]-1\} \quad (6)$$

where $I_1 = \mathrm{tr}C$, C is the right Cauchy-Green tensor; $I_4 = C{:}\,MM$, $I_6 = C{:}\,M'M'$, where M and M' are unit direction vectors of two helical collagen fibers in the reference configuration, and c_1, b_1, b_2 are material constants. They determined the torsional stiffness using each model and compared the models in predicting tissue mechanical response, and showed that their structural model and Holzapfel model provide better description of test results and prediction of tissue response.

In studying artery buckling, we have recently obtained that material constants of porcine carotid arteries with a four-fiber model to illustrate the effect of collagen fiber alignment[45]. Very recently, we measured the collagen alignment distribution in these arteries incorporated the distribution into an improved a hyperelastic strain energy function model proposed by Gasser *et al*[40] which based on generalized structured tensor to characterizes the dispersed collagen distribution in the arteries[46]. The strain energy density functions are given by

$$W_j=\frac{C_j}{2}(I_1-3)+\frac{b_{1j}}{b_{2j}}[e^{b_{2j}[\kappa_j I_1+(1-3\kappa_j)I_4-1]^2}-1]$$

$$j=\text{intima or adventitia} \quad (7)$$

where b_{1j}, b_{2j} and c_j are positive material constants, κ_j is the dispersion parameter ranges between $[0, \frac{1}{3}]$, with $\kappa_j = 0$ for fully aligned fibers and $\kappa_j = 1/3$ for evenly distributed fibers[40].

We have further applied the model in the analysis of vein twist buckling and demonstrated that the model can capture the twist behavior and kinking in veins under torsion[47]. It is also of interest to determine whether microstructure-based constitutive models can describe the twist behavior of arteries.

2. 2 Twist stability and buckling of blood vessels

Severe twist in arteries and veins can lead to loss of stability and vessel buckling. It hinders blood flow, reduces the patency of microanastomoses of vein grafts, and may even lead to kinking of the vessel that blocks blood flow[22, 37]. Early experimental work by Dobrin and colleagues demonstrated the effects of vein twist buckling on the patency of the vein grafts[5, 22]. Twist buckling was also studied in microvessel anastomosis[20, 48]. These twist buckling behaviors have also been simulated using finite element models[4, 49]. Recently, our lab has performed both theoretical and experimental studies on blood vessel buckling due to twisting[3, 37, 47, 50–52]. We found that the torque increases linearly with twisting angle until it reaches a critical point followed by a sudden drop of torque, which indicates the occurrence of buckling. The critical buckling torque is found significantly affected by axial stretch ratio and lumen pressure[37].

We have recently demonstrated that collagen alignment affects artery critical buckling pressure using a four-fiber model[45]. An improved model could be employed for representing anisotropic behavior of the arterial wall. To this end, Gasser *et al*[40] proposed a hyperelastic strain energy function based on generalized structured tensor to characterize the dispersed collagen distribution.

3 Arterial wall remodeling under axial twist

It has been well documented that changes in mechanical stress will stimulate arterial wall remodeling. Numerous studies have documented arterial adaptive responses to changes in pressure, flow rate, and axial stretch ratio both *in vivo* and *ex vivo*[53–54], however, little is known about artery's adaptive response to axial twist. We expect that axial twist would trigger arterial wall remodeling since that similar to the stress changes due to lumen pressure and blood flow that will also lead to wall remodeling, axial twist alters both the magnitude and distribution of wall stress. Recently, our group has conducted *ex vivo* and *in vivo*

experiments to study early and long-term remodeling of artery under twist, which are discussed below.

3. 1 Early stage remodeling from organ culture model

The early stage of arterial wall remodeling under twist was examined by *ex vivo* organ culture experiment[51]. Briefly, porcine carotid arteries were twisted axially and maintained for three days in *ex vivo* organ culture system, while lumen pressure and flow remained the same as untwisted controls. Our results demonstrated that 1) cell proliferation increased significantly in the intima and media of twisted arteries (5. 7-fold and 4. 3-fold respectively) compared with controls; 2) the total area of internal elastic laminar (IEL) fenestrae decreased 43% and average size decreased 53%, but the aspect ratio increased 1. 2-fold in twisted arteries; 3) matrix metalloproteinases (MMP)-2 expression significantly increased in twisted arteries while there were no significant changes in MMP-9 and TIMP-2; and 4) the endothelial cells (ECs) in the twisted arteries were significantly elongated, as measured by the shape index and aspect ratio, and were aligned towards blood flow direction after 3 days organ culture. These results demonstrate obvious signs of artery remodeling when subjected to twisting.

3. 2 Long-term remodeling of arteries under twist

One advantage of the *ex vivo* organ culture model is that it allows us to apply an axial twist to the arteries without altering the mean pressure, flow rate, or axial stretch ratio in a well-controlled environment. The limitation of the organ culture model is the limited duration of observation, therefore, an *in vivo* animal model is needed for long-term study.

To this end, we have developed an innovative *in vivo* rat model to investigate arterial wall remodeling under twist for short to long term studies (to be published)[55]. Rat carotid arteries were cut apart and then twisted for a given angle by surgery and maintained for various durations up to 4 weeks, and the arterial wall remodeling in twisted arteries were analyzed using histology, immunohistochemistry and fluorescent microscopy. Artery remodeling demonstrated a significant time-dependent manner in this 4-week duration: cell proliferation, expressions of MMP-2 and MMP-9 increased significantly; IEL fenestrae shape was elongated with area and its number increased significantly; ECs were gradually elongated and aligned towards the blood flow direction; wall thickness to lumen ratio increased significantly; and collagen to elastin ratio decreased significantly. These results demonstrated that sustained axial twist can stimulate *in vivo* artery remodeling. The twisting rat carotid model is an effective and reliable *in vivo* approach for studying long term arterial wall remodeling under torsion. Further longer term studies up to several months are needed in the future.

3. 3 Possible mechanisms

While the effects and mechanisms of flow-induced shear stress on vascular cells have been studied very extensively, both *in vivo* and in vitro cell culture, there has been no report of the effects or mechanism of twist-induced shear stress on vascular cells. The mechanisms of the observed remodeling in arteries under twist may be extrapolated from our knowledge on the

effects and mechanisms of the normal and shear stress induced from pressure and flow in arteries.

It is well accepted that the extracellular matrix (ECM) of arterial wall remodels in response to mechanical stress and that the process is modulated by matrix metalloproteinases MMP-2 and MMP-9. Our studies showed that the expression of MMP-2 significantly increased in the twist group while there was no significant change of TIMP-2, indicating that elevated ECM remodeling in twisted arteries is associated with pathological changes in the development of cardiovascular diseases such as atherosclerosis and plaque instability[56].

ECs in arteries are exposed to both fluid shear stress due to blood flow and periodic stretching due to by pulsatile pressure and axial tension. Their functions are affected by the morphology and cytoskeleton microstructure. It is well known that EC orientation and shape are influenced by shear flow, cyclic circumferential stretch, and axial stretch[54, 57]. EC morphology is associated to local shear stress distribution pattern in the vasculature. They tend to elongate and align in the direction of flow but perpendicular to cyclic stretch[58]; the alignment is only determined by the magnitude and direction of the shear and is independent of acute exposure time[59]. We have shown that ECs significantly elongate in response to twist deformation. Since the vessel deformation can result in cell deformation, we compared EC morphology in fresh arteries under twist and control. EC realign partially back towards the (axial) flow direction after 3-day organ culture, indicating EC's adaptation to the flow.

The increases in cell proliferation and MMP-2 expression as well as the changes in EC and IEL fenstrae morphology due to axial twist are similar to the features of the remodeling process that occurs in response to elevated axial stretch and pulse pressure[53, 60]. These results suggest that the shear stress generated by axial twist has similar biological effects compared to increased normal stress generated by elevated pressure or axial stretch.

3. 4 Mathematical modeling

In addition to long-term *in vivo* animal experiments, mathematical models of wall remodeling under axial twist are useful for better understanding the mechanisms and predicting the growth pattern.

Various models have been proposed for studying artery growth and remodeling, which is modulated by wall stress and fluid shear stress, and extensive studies have been carried out on artery adaptation to changes in pressure, axial stretch, and flow[61–69]. However, there are very few studies on the effect of twist on blood vessel growth and remodeling, even though such study could be a natural extension of the current well-formed computational models[61, 64–66, 68].

Demirkoparan and colleagues have developed a constitutive theory for fiber reinforced hyperelastic materials that fibers can undergo microstructural changes[70–71]. Using this model, they studied the nonhomogeneous deformation of a circular solid cylinder, which composes of isotropic matrix and helical fiber families, under axial extension and torsion[72]. They showed that complex morphological changes occur during fiber dissolution and reassembly. With increasing axial stretch and twisting, fibers reassemble in the direction of

maximum principal stretch, which is difference from the fiber direction. Reassembly also changes fibers' contributions to the torque and axial force. In another study, Demirkoparan and colleagues[73] studied the unloading behavior following fiber remodeling of the cylinder under torsion. The cylinder is given an initial twist and remodel until has an inner core of original fiber/matrix and an outer sheath of remodeled fiber/matrix, the twist is then decreased back to zero. Unloading can occur with no additional fiber dissolution and reassembly, and a residual axial stretch and twist exist if the axial force and torque become zero. Their studies provide a theoretical foundation that can be adapted for investigating artery remodeling under twist, nevertheless such studies are still few.

Recently, our group has conducted mathematical modeling of artery growth and remodeling under twist. The artery is modeled using Holzapfel's fiber model that composing of elastin, smooth muscle, and 4 families of collagen fibers in the axial, circumferential, and helical directions. Our preliminary results (to be published[74]) showed that when the artery is subjected to a step increase in twisting angle from 0° to 90°, arterial radius decreases as an immediate response, and the remodeling process starts due to stress changes in each constituent. Stresses increase in the axial and circumferential fiber families, and in the helical fibers in the twisting direction. Stress increase triggers the growth of new fibers align close to these directions. For helical fibers in the direction opposite to twisting, the stress decreases and hence no new fibers are produced in this direction. With the growth, stress in each direction gradually restores to its initial level, mass production rate gradually decreases and the mass eventually reaches to a steady level, and remodeling process stops. This model predicted the long term artery remodeling under twist, and further validation with data from long-term *in vivo* experiment is needed.

4 Conclusions

Twist of blood vessels are frequent clinical observations though the mechanism and outcome vary. While we have gained some understanding of the twists mechanical behavior of blood vessels, further investigation of the mechanical properties and wall remodeling is needed. Axial twist of arteries affects EC morphology and stimulates cell proliferation and ECM remodeling in the arterial wall. Further investigation of the behavior and remodeling of blood vessels under twist will enrich our understanding of the artery mechanics and wall remodeling in response to alterations in the mechanical environment.

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