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Review of biomechanical studies of arteries and their effect on stent performance

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ABSTRACT

Factors such as aging, atherosclerosis, hypertension, genetic defects and diabetes mellitus have been known to cause arteries to develop various shapes and characteristics in patients such as tortuosity, kinking, twisting, elongation, contraction, and curving. The change in artery mechanics can cause a variety of cardiovascular diseases among men and women. The improvement in technology and techniques has allowed access to different therapies such as balloon angioplasty or stenting. Stents are permanent implants that undergo repetitive deformations as a result of patient daily activities such as walking, flexing, sitting, climbing stairs, and getting into a car. Often, these deformations imposed on the stents result in stent failures. It is imperative that the biomechanics environment of the arteries causing stent failure is well understood and the stents be evaluated under multiple loading modes for increased life-cycle. As a result, this paper aims to summarize part of the available literature that reports studies on biomechanical environment in healthy and diseased arteries using various analytical methods.

1. Introduction

Heart diseases are playing a major cause of death with about 600,000 deaths per year. Among heart diseases, coronary heart disease alone kills more than 350,000/year and it costs $108.9 billion for the United States each year which includes the cost of health care services, medications and lost productivity [1]. In spite of significant advancements in clinical care and education for public, cardiovascular diseases (CVDs) are a leading cause of death and disability to the nation. Cardiovascular diseases (CVDs) include peripheral artery disease (PAD), high blood pressure (HBP), coronary heart disease (CHD), heart failure (HF) and stroke. According to the latest statistical data, CVDs are the number one cause of morbidity and mortality in countries of the western world [1]. Nearly 2400 Americans die of CVDs each day (an average of 1 death every 37 s) [2]. The causes of CVD include mainly atherosclerosis, syphilis, atheroma, heart attacks, congenital defects, obesity, smoking, hypertension, trauma, and hereditary conditions as well as hemodynamic and biomechanical factors. Atherosclerosis is a common disorder of the arteries, characterized by the accumulation of cells, lipids, connective tissue, calcium, and other substances inside the inner lining of the arterial wall. This fatty tissue – known as atheroma – can cause hardening of the arteries, rupture or erosion of the arterial wall, and eventually reduction or complete blockage of the blood flow. Atherosclerosis appears preferentially at sites of complex geometry (e.g., along the outer portions of the bifurcation), most often in the abdominal aorta, iliacs, coronaries, femorals, popliteals, carotids, and cerebrals. The atherosclerotic process starts early in life and advances throughout adulthood. In CVD the first line of defense is prevention by taking some strategies whereas sometimes it cannot be effective due to its advanced stages which are cured only by treatment. The treatment is being done by angioplasty, an invasive procedure where a balloon-tipped catheter is inserted into the narrowing and expanded which is also called stenosis. It helps in the widening of the vessel lumen and makes the flow of blood effective. By using this technique there are some complications like closing of the vessel after few days or weeks. In order to overcome issues during treatment a metal mesh tube called stent (Fig. 1) is used which is expandable and remains inside the vessel even after expanding and prevents it from closure [2–6]. There were issues associated with the stent treatment as well like tissue growing inside the stent after few days or

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few weeks which make the tube narrow inside and thereby restenosis occurs. Now the stent models are improved in certain aspects like fabrication techniques, strut shapes and the placement of stent is also considered depending upon the flexibility of the target vessel. Recently the stents are being improved with the drug eluting stents (DES) where they release the drug which regulates the metabolic activity of the vascular tissue and prevents the tissues from growing inside the stents [7].

Even though stents are good alternative to treat artery disease they undergo significant deformations during daily activities that patients perform especially in larger arteries like those in the limbs. During everyday body function, parts of the legs are exposed to multiaxial deformations with up to 60% rotation and 20% contraction as the leg is bent from an extended position [6]. As a result stent deployed in the intersection of the femoral and popliteal arteries is exposed to significant multiaxial displacements due to the motion as well as bending, torsion, tension, and compression during walking cycle [7]. This can lead to fracture of the metal stent in this region, which aligns with the fact that stents fracture at a measurable rate of more than 50%. Even the treatment procedures alone affect the vessels mechanically. As a result, understanding in details the mechanical environment of the arteries (with special interest on the large arteries in the limbs such as superficial femoral artery (SFA) and popliteal artery (PA)) imposing deformations in stents and the stent failures under cycle-mode is imperative for the improvement of the outcome of these treatments. In an effort to do so, this paper aims to summarize part of the available literature that reports studies on biomechanical environment in healthy and diseased arteries using various analytical methods.

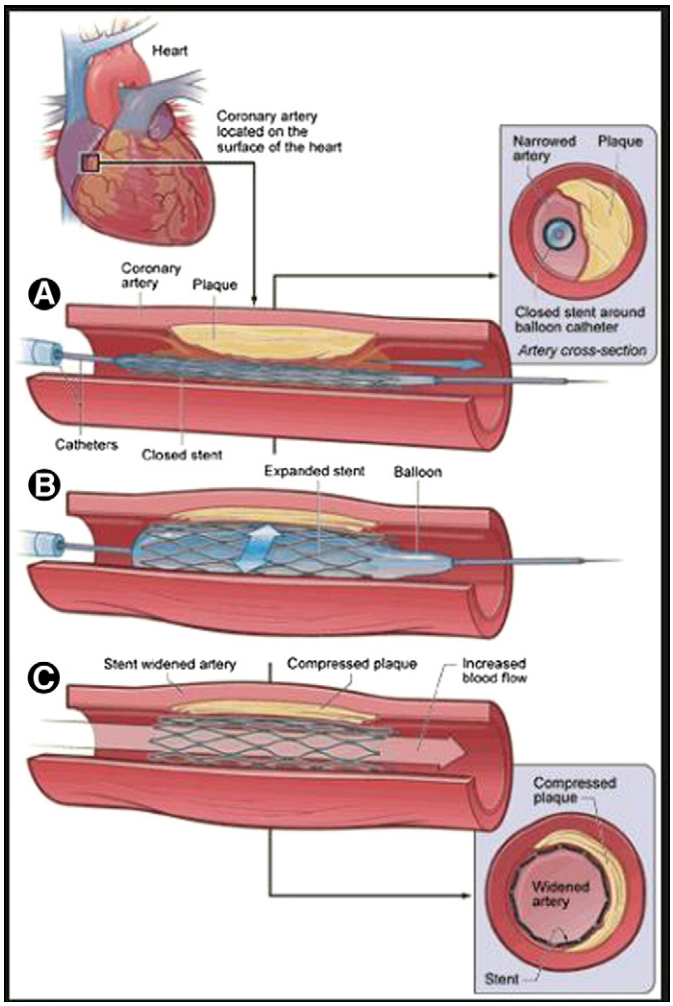

Fig. 1. Schematic showing stent implant into artery obstructed by plaque [8].

2. Characteristics of healthy arterial wall

All blood vessels consist of three distinct layers or tunicae: the tunica intima, tunica media and tunica adventitia (Fig. 2).

The intima is a thin endothelial layer that lines the inside walls, and sits on a very thin (~80 nm) basal lamina of a net-like type IV collagen in young human. Endothelial cells, typically elongated in the direction of the blood flow, act as a semipermeable membrane, through which nutrients and chemical signals can reach the cells in the vessel wall from the bloodstream. The intima has also a key role in regulating the active response of the vessel through which pressure regulating agents reach the media. Additionally, in order to help control the vascular tone the intima produces NO (nitric oxide), which relaxes smooth muscle cells in the media. Despite its great functional importance, due to its small thickness in young arteries the intima is usually neglected when considering the different layer contributions to the global mechanical resistance of the vessel wall. A fenestrated sheet of elastin called internal elastic lamina separates the intima from media [10].

The media is formed primarily by smooth muscle cells (SMC) that are embedded in an extracellular plexus of elastin and collagen (mainly types I and III) and an aqueous ground substance that also contains proteoglycans. Depending on the internal arrangement of the smooth muscle cells in the media, it is distinguished between elastic arteries and muscular arteries. The former tend to be large-diameter vessels close to the heart, and include the aorta, the main pulmonary artery, the common carotid and common iliac arteries. Their most characteristic histological feature is the so-called lamellar unit, a sandwich-like 'sublayer' of smooth muscle cells and thin elastic laminae. Elastic arteries have concentric ring-like structures that are tied together by radially oriented collagen. In muscular arteries, the media appears as a single thick ring of smooth muscle cells. The SMC are embedded in a loose connective tissue matrix and arranged as a sequence of concentric layers of cells, which can reach numbers of 25–40 in larger vessels like in the femoral artery [10].

The adventitia is the outermost layer of the vessel wall. It consists of a dense network of type I collagen fibers with scattered fibroblasts, elastin and nerves. In medium and large arteries there is also the vasa vasorum, an intramural network of arterioles, capillaries and venules that supply large vessels where the distance from the main bloodstream to the outer sections of the wall does not allow for proper interchange of O_2, CO_2, nutrients and metabolites. The presence of nerves in the adventitia allows innervation of smooth muscle in the outer media, via the diffusion of neurotransmitters. As for the fibroblasts, they are responsible for collagen production, particularly type I, and thus regulate the connective tissue. At higher pressures, the fibers gradually straighten, confirming the hypothesis that the adventitia serves as a protective sheath, preventing rupture of the vessel due to an acute increase in pressure [10].

3. Mechanics of arteries

The mechanical behavior of blood vessels has been a subject of research for many years with first few reports dating back to the end of the 19th century [11,12]. The first characteristic of arterial mechanics is the presence of residual stresses where it has been reported that if arterial ring is cut radially it springs open and an axial strip excised from the artery bends away from its vessel axis which signals the presence of residual stresses both in axial and circumferential directions [10,13–17]. Further studies report that the luminal part is under compression and the external part is under tension where the internal pressure equilibrates these stresses and they have been reported as a positive feature as part of the compatible growth and remodeling and not of physical