*This article can be cited as:* Moradi M, Habibi Jirdehi H. Review: Biomechanical impacts of vascular stents. Cardiovasc Biomed J. 2025; 5(1): 31-44.

**Review**

# Biomechanical impacts of vascular stents

***Mohammad Moradi* [1,*], *Hanieh Habibi Jirdehi* [1]**

[1] Biomedical Engineering, Department of Biomedical Engineering, Faculty of Khajeh Nasir al-Din Tusi Faculty, Tabriz University of Technology (Sahand), Sahand, Tabriz, Iran

*** Corresponding Author:**
**Address:** Tabriz University of Technology (Sahand), Sahand, Tabriz, Iran. **Postal code:** 5331817634; **Tel:** +98 9185056062; **Email:** Moradim506@ yahoo.com

**Article Information:**
Received: 22 Jun 2025; Revised: 25 Aug 2025; Accepted: 27 Aug 2025 DOI: 10.18502/cbj.v5i1.19450

## Abstract

**Objectives:** Coronary stents are widely used as an effective intervention for coronary artery disease (CAD). This study investigates the biomechanical and hemodynamic consequences of stent implantation on arterial blood flow. While stenting improves myocardial perfusion and decreases vascular resistance, it can also lead to adverse effects, such as altered flow patterns, endothelial injury, and increased arterial stiffness. These factors may contribute to restenosis and other complications. This review summarizes biomechanical alterations caused by stenting and highlights recent strategies aimed at enhancing stent performance. To investigate the biomechanical and hemodynamic consequences of coronary stent implantation on arterial blood flow and to assess strategies aimed at mitigating related complications.

**Keywords:** Coronary stent, biomechanics, endothelial injury, blood flow dynamics, myocardial perfusion

## Introduction

Cardiovascular diseases, particularly coronary artery disease (CAD), remain among the leading causes of morbidity and mortality worldwide. Cardiovascular diseases typically arise when cholesterol and other lipids accumulate within the arterial walls, forming atherosclerotic plaques that narrow the vessel lumen and restrict oxygen-rich blood flow to the Myocardium. Among current therapeutic options, percutaneous coronary intervention (PCI)—especially balloon angioplasty and stenting—has become the preferred method due to its minimally invasive nature, shorter recovery time, and high success rates. Stents, first conceptualized in the 1960s and clinically adopted in the 1980s, are small mesh-like tubular devices that restore vessel patency by scaffolding narrowed or occluded arteries (Pericevic et al., 2011). Each year, over two million stent procedures are performed worldwide (Peters et al., 2009). The mechanical design of these devices must ensure durability under complex multi-axial loading conditions while minimizing damage to the vessel wall and endothelial lining (Rikhtegar et al., 2014). Therefore, understanding the biomechanical interactions between stents and arterial walls is critical for improving clinical outcomes. Drug-eluting stents have significantly reduced the incidence of restenosis, particularly in patients with advanced atherosclerosis. However, complications such as in-stent restenosis (ISR) and thrombosis remain persistent concerns (Wentzel et al. 2001). ISR is largely attributed to neointimal hyperplasia, a process driven by endothelial damage and altered hemodynamic forces. Specifically, regions exposed to low wall shear stress (WSS) or disturbed flow patterns are more susceptible to pathological

remodeling and plaque development (Soulis et al., 2014; Chistiakov, 2017). In this context, stent geometry plays a pivotal role in determining post-implantation flow patterns and mechanical stress distributions. Closed-cell designs, for instance, have been associated with lower complication rates compared to open-cell configurations (Hart et al., 2006). Furthermore, the connection methods, strut thickness, and overall architecture of a stent can significantly influence blood flow dynamics and endothelial response (Gundert et al., 2012; Beier et al., 2016). To study these complex interactions, computational tools such as finite element analysis (FEA) and computational fluid dynamics (CFD) are widely employed (Balossino et al., 2008; Morlacchi et al., 2011). These methods allow for a detailed evaluation of stress distribution, flow disturbances, and wall compliance in both healthy and stented arteries. This study aims to investigate the biomechanical effects of stent implantation on arterial blood flow and vascular mechanics. By focusing on both coronary and peripheral vascular stents, particularly those deployed in femoropopliteal arteries, we offer a comprehensive assessment of stent behavior under varying anatomical and physiological conditions. The insights from this research can contribute to the design of more efficient stent systems that reduce complications such as restenosis and enhance long-term treatment efficacy. This review synthesizes findings from recent studies on the biomechanical implications of stent implantation.

**Anatomy of the Human Cardiovascular System**

The human cardiovascular system, also referred to as the circulatory system, is a complex and dynamic network that ensures the continuous transport of blood, oxygen, nutrients, hormones, and metabolic waste products throughout the body. It plays a fundamental role in maintaining homeostasis by supporting essential physiological processes such as cellular respiration, nutrient delivery, thermoregulation, and waste removal. The cardiovascular system consists of three primary components: the heart, blood vessels, and blood. The heart acts as a muscular pump that maintains blood circulation through rhythmic contractions, while blood vessels—including arteries, veins, and capillaries—serve as conduits for blood flow. Arteries in particular are of central interest in vascular biomechanics, as they undergo pulsatile pressure and complex flow patterns, especially in the presence of pathological conditions such as atherosclerosis. Understanding the anatomical and physiological principles of the cardiovascular system is effective for evaluating the biomechanical impact of stent implantation, particularly regarding blood flow dynamics, arterial wall stresses, and endothelial responses see Figure 1.

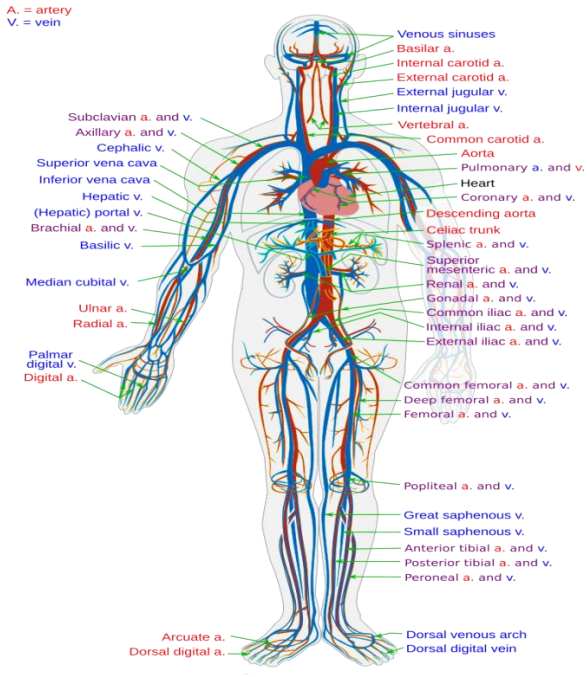


**Figure1.** The human body's vascular system

 CC BY NC 4.0

**The Heart**

The heart is a muscular organ located in the thoracic cavity, slightly left of the midline, whose primary role is to maintain continuous blood circulation throughout the body. It functions as a dual pump, facilitating both systemic and pulmonary circulations. Anatomically, the heart consists of four chambers: two atria (upper chambers) and two ventricles (lower chambers). The right atrium receives deoxygenated blood from the systemic circulation via the superior and inferior vena cava, transfers it to the right ventricle, which pumps it into the pulmonary artery for oxygenation in the lungs. The oxygenated blood then returns to the left atrium via the pulmonary veins and enters the left ventricle. From the left ventricle, blood travels to the systemic circulation through the aorta (Tortora & Derrickson, 2017). The cardiac cycle comprises two main phases: systole (contraction) and diastole (relaxation), regulated by the heart's intrinsic conduction system—including the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, and Purkinje fibers (Hall, 2020). Electrical impulses originating from the SA node ensure synchronized contraction of the atria and ventricles, which is vital for effective cardiac output and preserving physiological stability (Silverthorn, 2020). Disruptions in this conduction pathway may lead to arrhythmias and compromise hemodynamic efficiency. From a biomechanical perspective, the heart not only acts as a pressure-generating pump but also modulates flow characteristics such as pulsatility, shear stress, and wall tension throughout the vascular network. These parameters influence the performance and long-term behavior of vascular stents, specifically, in regions exposed to high mechanical stress or flow disturbances. Therefore, a detailed understanding of cardiac anatomy and function is essential for evaluating the physiological context in which stents operate.

**Blood Vessels**

The three main types of blood vessels, namely, arteries, veins, and capillaries, form the human vascular system. Each serves unique structural and functional roles in regulating blood flow and tissue perfusion.

**1- Arteries**

Arteries are thick-walled, elastic vessels that transport oxygenated blood from the heart to the peripheral tissues; however, the pulmonary artery is excluded. Their high elasticity, particularly in large elastic arteries such as the aorta, enables them to accommodate the pulsatile nature of cardiac output and maintain consistent blood flow through elastic recoil (Silverthorn, 2020). From a biomechanical perspective, arteries undergo high-pressure loading and shear stress, which makes them a critical focus in the design and evaluation of vascular stents. Atherosclerotic lesions, which commonly occur in elastic arteries, significantly alter local biomechanics and often require stent intervention.

**2- Veins**

In contrast, veins are low-pressure vessels with thinner walls and larger lumens. They transport deoxygenated blood back to the heart and rely on the presence of one-way valves and surrounding skeletal muscle contractions to maintain venous return (Marieb & Hoehn, 2018). Although less frequently treated with stents, venous stenting is becoming more relevant in cases such as chronic venous obstruction or deep vein thrombosis.

**3- Capillaries**

Capillaries are microvessels specialized for the exchange of gases, nutrients, and metabolic wastes at the tissue level (Tortora & Derrickson, 2017). While not a direct site for stent placement, the capillary network is indirectly affected by arterial pathologies and altered hemodynamics resulting from stent implantation. The structural and functional diversity among vascular segments profoundly influences the biomechanical environment within which stents operate. Understanding these differences is essential for optimizing stent design, placement, and long-term performance—particularly in addressing issues such as restenosis, altered wall shear stress, and endothelial dysfunction.

**Blood**

Blood is a specialized connective tissue that plays a central role in maintaining homeostasis by enabling the transport of gases, nutrients, hormones, and metabolic waste throughout the body. It consists of two main components—plasma and formed elements—each contributing uniquely to physiological and biomechanical functions.

**1- Plasma (~55%)**

Plasma is the liquid matrix of blood, primarily composed of water, electrolytes, plasma proteins (such as albumin, fibrinogen, and globulins), and dissolved solutes (Hall, 2020). It serves as a transport medium for hormones, nutrients, respiratory gases,

and waste products. From a hemodynamic perspective, the viscosity of plasma, influenced by protein concentration and temperature, directly affects flow resistance and shear stress—factors critical in vascular stenting.

**2- Formed Elements (~45%)**

**These include:**
**- Erythrocytes (Red Blood Cells):** Responsible for oxygen and carbon dioxide transport via hemoglobin, erythrocytes significantly influence blood viscosity and flow dynamics. Their deformability and aggregation behaviors impact microvascular flow and are essential considerations in the post-stenting environment.

**- Leukocytes (White Blood Cells):** As immune cells, leukocytes play a key role in inflammatory responses. Their recruitment and activation are especially relevant in the context of stent-induced endothelial injury, which can trigger restenosis or thrombosis.

**- Platelets:** Platelets are vital for hemostasis and are the primary mediators of thrombus formation at sites of vascular injury. Stent deployment, particularly in bare-metal or poorly coated devices, can activate platelet aggregation, increasing the risk of acute or late stent thrombosis. Understanding the cellular and fluid components of blood is critical when evaluating the performance and complications of coronary and peripheral vascular stents. Hemodynamic parameters such as blood viscosity, shear rate, and cell-surface interactions are all influenced by the composition and behavior of blood, and they directly affect stent design, placement strategy, and clinical outcomes.

**Blood Circulation Pathways**
The human cardiovascular system operates through two interconnected circuits—pulmonary and systemic circulation—which work in synchrony to ensure efficient oxygen delivery, nutrient transport, and metabolic waste removal. These circuits create distinct hemodynamic environments that directly impact the biomechanical behavior of blood flow and the design and functionality of vascular interventions such as stents.

**1- Pulmonary Circulation**
This low-pressure circuit transports deoxygenated blood from the right ventricle to the lungs via the pulmonary artery, where gas exchange occurs. After oxygenation, blood returns to the left atrium through the pulmonary veins. Although stenting in pulmonary arteries is relatively rare, the unique low-pressure, low-resistance conditions of pulmonary circulation are critical when evaluating cardiovascular load and designing right-heart support devices (Tortora & Derrickson, 2017). Additionally, alterations in pulmonary circulation dynamics may indirectly affect systemic perfusion and left ventricular afterload.

**2- Systemic Circulation**
This circuit is characterized by higher pressures and greater biomechanical loads, noticeable in stent design. Factors such as wall shear stress (WSS), pulsatile pressure, and arterial compliance vary throughout this circuit and significantly impact post-stent outcomes, such as restenosis and thrombosis. The dynamic interaction between these circulatory circuits influences global hemodynamics, including pressure gradients, flow patterns, and ventricular workload. These variables are key to understanding how vascular stents change local flow conditions and mechanical stresses. Disruption in these pathways, whether due to vascular obstruction or suboptimal stent deployment, can compromise tissue perfusion and lead to adverse clinical outcomes. Therefore, a comprehensive understanding of both pulmonary and systemic circulatory mechanics is essential for advancing stent technologies and optimizing patient-specific treatment strategies**.**

**Regulation of Cardiovascular Function and Its Relevance to Hemodynamic Homeostasis**
The cardiovascular system maintains homeostasis through complex regulatory mechanisms that ensure adequate tissue perfusion, stable blood pressure, and efficient thermal balance. These mechanisms are dynamic and tightly controlled, particularly in the presence of vascular interventions such as stents, which can significantly alter local flow dynamics and vascular responses.

**1. Blood Pressure Regulation**
Blood pressure is modulated through a combination of neural, hormonal, and local mechanisms to meet physiological demands. These include:

**- Autonomic Nervous System (ANS):** Sympathetic activation increases cardiac output and induces vasoconstriction, elevating blood pressure—particularly during stress or exercise. Conversely, parasympathetic stimulation reduces heart rate and introduces vasodilation. The ANS also modulates

 CC BY NC 4.0

vascular tone across stented regions, potentially influencing blood flow distribution and wall shear stress.

**- Renin-Angiotensin-Aldosterone System (RAAS)**
RAAS plays a critical role in long-term blood pressure regulation. Angiotensin II induces vasoconstriction and promotes endothelial dysfunction, which is particularly relevant in stented arteries where altered hemodynamics can exacerbate neointimal hyperplasia or thrombosis.

**- Local Paracrine Regulation**
Tissues experiencing hypoxia release vasodilators such as nitric oxide (NO) and prostaglandins to enhance local blood flow. However, stents may hinder endothelial responsiveness, which can decrease the release of these factors and compromise regional autoregulation.

**2. Cardiac Output and Systemic Perfusion**
Cardiac output (CO), the product of heart rate (HR) and stroke volume (SV), determines the overall perfusion capacity. Fluctuations in CO influence systemic blood flow patterns and pressure gradients. In patients with stent placements, particularly in coronary arteries, changes in CO can alter local flow rates, influencing mechanical stresses exerted on the stented region.

**3. Thermoregulation**
The cardiovascular system, through blood redistribution, also regulates core body temperature to the skin. Vasodilation enhances heat dissipation in warm conditions, while vasoconstriction conserves heat in cold environments. Since vascular tone affects blood flow resistance and shear stress, these thermoregulatory responses can indirectly impact the hemodynamic environment around stents. Overall, these regulatory mechanisms are crucial for maintaining systemic and local homeostasis. In the context of vascular stenting, any disruption in these processes—due to mechanical obstruction, impaired endothelial function, or abnormal shear stress—can compromise vascular health and therapeutic outcomes. Therefore, understanding how the cardiovascular system regulates these processes is crucial for evaluating the biomechanical implications of stents and for designing more adaptive and responsive vascular devices.

**Clinical Significance of Cardiovascular Disorders in the Context of Vascular Interventions**
Cardiovascular diseases such as atherosclerosis, hypertension, and heart failure represent significant clinical challenges due to their widespread prevalence and potential for life-threatening complications. Atherosclerosis, characterized by the progressive thickening and stiffening of arterial walls, leads to luminal narrowing that restricts blood flow and increases cardiac workload. This pathological remodeling not only compromises tissue perfusion but also elevates the risk of acute cardiovascular events, including myocardial infarction and ischemic stroke. Hypertension exacerbates vascular injury by chronically elevating arterial wall stress, promoting endothelial dysfunction, and accelerating the progression of vascular diseases. The sustained increase in afterload imposes an additional burden on myocardial function, often culminating in heart failure — a syndrome defined by the heart's inability to maintain adequate systemic perfusion to meet metabolic demands. Effective diagnosis and management of these conditions depend on a comprehensive understanding of cardiovascular anatomy and physiology, particularly hemodynamic principles and vascular biomechanics. This knowledge is critical in tailoring therapeutic strategies, including the use of stents, which aim not only to restore vessel patency but also to mitigate the biomechanical stresses contributing to disease progression and post-intervention complications. Integrating clinical insights with biomechanical analyses facilitates improved stent design and deployment strategies, ultimately enhancing patient outcomes and reducing adverse events such as restenosis and thrombosis.

**Atherosclerosis**
Atherosclerosis is a chronic, progressive vascular disease characterized by the formation of plaques within the arterial walls, leading to lumen narrowing and impaired blood flow. The disease encompasses three main types: atherosclerosis (the most prevalent form involving lipid accumulation primarily in the tunica intima), arteriolosclerosis (affecting smaller arteries and arterioles), and Monckeberg's medial calcific sclerosis (characterized by calcium deposits in the arterial media, commonly associated with aging) (Martini et al., 2015). The pathogenesis of atherosclerosis begins with endothelial injury, which increases vascular permeability and facilitates the infiltration of lipids, particularly low-density lipoproteins (LDL).These lipids become oxidized, triggering an immune response involving monocyte

recruitment and differentiation into foam cells. Foam cells release cytokines that stimulate smooth muscle cell proliferation and migration, contributing to plaque growth and arterial wall thickening (Chandran et al., 2007; Libby et al., 2009). Plaque progression leads to the development of a fibrous cap composed of collagen, which stabilizes the lesion but can also provoke thrombosis if ruptured due to platelet adhesion to exposed collagen fibers. Atherosclerotic lesions preferentially develop at sites of disturbed flow, such as arterial branch points and curvatures, notably in coronary, carotid, and renal arteries(En.wikipedia.org, 2022). Atherosclerosis is an inflammatory disease involving both innate and adaptive immunity. Clinical studies, such as the JUPITER trial, have revealed that anti-inflammatory treatments such as statins reduce C-reactive protein levels and cardiovascular events (Ridker et al., 2008). Risk factors for atherosclerosis include modifiable factors such as smoking, high-fat diets, and physical inactivity, as well as non-modifiable factors like age, gender, genetics, diabetes mellitus, and hypertension. Aging remains a major contributor to disease prevalence, with cardiovascular disease incidence increasing significantly in elderly populations (Massa et al., 2019; Ribeiro et al., 2016). Histopathologically, atherosclerotic lesions are classified into several types based on plaque morphology and cellular composition, ranging from early, asymptomatic lesions (types I-III) characterized by foam cell accumulation to advanced fibroatheromas with necrotic cores and fibrous caps, which carry higher risks for clinical events (Virmani et al., 2000).

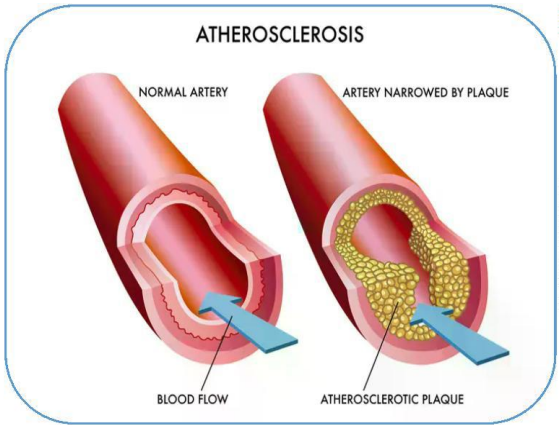


**Figure 2.** Complications of atherosclerosis (En.wikipedia.org)

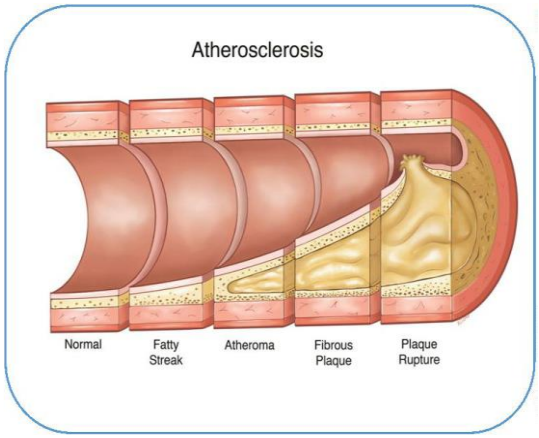


**Figure 3.** Progression of atherosclerosis (En.wikipedia.org, 2022)

 CC BY NC 4.0

## Treatment of Atherosclerosis: A Focus on Endothelial Dysfunction and the Biomechanical Impact of Stents

Extensive evidence confirms that the vascular endothelium is a dynamic organ with endocrine, paracrine, and autocrine functions essential for vascular homeostasis. Disruption of this homeostasis by various factors induces localized endothelial dysfunction, characterized by diminished anticoagulant properties, altered vascular tone, advanced leukocyte adhesion, and elevated cytokine and growth factor production. This dysfunctional endothelium exhibits increased permeability to low-density lipoprotein cholesterol (LDL-C), promotes vascular inflammation, both of which contribute to the initiation and progression of lipid-rich and inflamed atherosclerotic plaques (Rong et al., 1980). The main biological and physical risk factors, such as hypercholesterolemia and shear stress alterations, modulate endothelial gene expression, activating oxidative stress pathways including NADPH oxidase and the endogenous endothelin system, while reducing nitric oxide (NO) bioavailability. NO, synthesized from L-arginine to L-citrulline by nitric oxide synthase (NOS) enzymes, is essential for vascular health. However, oxidative stress promotes eNOS uncoupling, where endothelial NOS produces superoxide anions instead of NO, further exacerbating endothelial dysfunction (Marcus et al., 2005). Experimental models using NO synthase inhibitors demonstrate accelerated atherosclerotic plaque development, while therapies targeting NO pathways can mitigate disease progression in vulnerable vessels. Coronary endothelial dysfunction serves as an independent predictor of major cardiovascular events, linked to circadian variations in vascular function, with endothelial-dependent vasodilation impaired in patients with acute coronary syndrome (Halcox et al., 2002). Dysfunctional endothelium reduces the production of vasoprotective agents such as NO and prostacyclin (PGI2), facilitating platelet activation and aggregation. The release of vasoconstrictive peptides (serotonin, thrombin) from activated platelets worsens vascular tone, and decreased production of tissue plasminogen activator and thrombomodulin, alongside increased tissue factor expression, exacerbates thrombotic risk (Hoffmeister et al., 1980; Gertz et al., 1990). Shear stress fluctuations within plaques induce endothelial cell apoptosis and detachment, processes regulated by matrix metalloproteinases (MMP-2, MMP-9) and integrin-mediated pathways, which contribute to thrombosis (Sumi et al., 2010). Following diagnosis, treatment begins with lifestyle interventions aimed at slowing progression. Pharmacologic strategies to control blood pressure, glucose, inflammation, and coagulation are standard; however, severe cases necessitate surgical interventions. Techniques such as angioplasty with stent placement restore vessel patency, alleviating flow restriction. Balloon angioplasty involves catheter-based delivery of a balloon-mounted stent to the lesion site, where inflation expands the stent, supporting the artery walls and restoring luminal diameter (Medtronic, 2022; Hopkins Medicine, 2022). From a biomechanical perspective, stent implantation alters local hemodynamics, potentially inducing disturbed flow and oscillatory shear stress, which can influence endothelial response and neointimal hyperplasia. Thus, stent design and deployment strategies must consider biomechanical factors to optimize flow patterns and lower restenosis risk. Drug-eluting stents exemplify integration of pharmacologic and mechanical approaches to modulate vascular healing (NHLBI, NIH, 2022). Although stents have broader clinical applications (e.g., aneurysms, bile duct obstruction), this review emphasizes their role in atherosclerosis treatment, focusing on how biomechanical alterations induced by stents interact with endothelial function and vascular biology, underlining the importance of interdisciplinary research in biomedical engineering and clinical practice.

## Coronary Stents and Their Biomechanical Implications

Coronary stents have become crucial vascular implants in treating coronary artery disease, offering mechanical support to restore and maintain arterial openness. The development of stent technology has been well documented in the literature (Strauss et al., 2021), resulting in three main types: bare-metal stents (BMS), drug-eluting stents (DES), and bioresorbable scaffolds (BRS)—the latter called "scaffold" because it is biodegradable and provides temporary mechanical support. The introduction of BMS in 1987 (Sigwart et al., 1987) represented a major step forward in interventional cardiology. These early devices, made from stainless steel with thick struts, were mechanically effective but caused biological reactions, with in-stent restenosis (ISR) rates reaching up to 20% (Serruys et al., 1994). ISR mainly results from neointimal hyperplasia and is linked to disturbed flow and non-physiological wall shear stress (WSS) patterns next to the thick

struts—issues now understood as biomechanically driven. To overcome these problems, drug-eluting stents (DES) were introduced in the early 2000s. Coated with antiproliferative drugs like sirolimus or paclitaxel, first-generation DES lowered ISR rates from 17% to around 4% (Moses et al., 2003). However, concerns arose about delayed healing of the endothelium and late stent thrombosis (ST), which can happen 1–2 years after implantation (Camenzind et al., 2007). These adverse effects were connected not only to the drugs used but also to local biomechanical issues caused by the rigid metal frameworks disrupting flow and endothelial shear stress. The second generation of DES, introduced around 2007, featured improvements in material science and design. Alloys like cobalt-chromium (CoCr), platinum-chromium (PtCr), and platinum-iridium (PtIr) allowed for thinner struts (from 132–140 μm down to 80–90 μm), greatly reducing flow separation zones and supporting more natural WSS distributions (Bangalore et al., 2018). These changes also improved biocompatibility and decreased polymer-related toxicity (Navarese et al., 2014), resulting in better vascular healing and lower rates of late ST. Recent advances in DES technology have further advanced the field by introducing ultra-thin struts (<70 μm) with biodegradable polymer coatings. These modifications not only promote faster re-endothelialization but also reduce ISR rates to below 3% and the need for target lesion revascularization to under 1%, especially in non-complex lesions (Kandzari et al., 2017). The incidence of late ST has similarly decreased from 2.5% in earlier DES to 0.6% (Räber et al., 2012), indicating improved mechanical compatibility with the native vessel environment. However, despite these improvements, the risk of early ST still requires continued use of dual antiplatelet therapy (DAPT) or anticoagulants. Current clinical practice involves customizing the duration of DAPT based on individual patient risk profiles and emerging evidence about bleeding versus thrombosis risks (Kinlay et al., 2023). From a biomechanical perspective, modern stent platforms aim to closely mimic the natural flow environment. The reduction in strut thickness, optimization of strut geometry, and enhanced material compliance collectively help decrease flow disturbance, promote more uniform shear stress distribution, and reduce neointimal proliferation. These factors are essential in biomedical engineering, where the interface between device mechanics and vascular biology determines long-term therapeutic success.

## Structural Design of Modern Drug-Eluting Stents (DES) and Biomechanical Considerations

Modern drug-eluting stents (DES) have seen significant advancements in structural design aimed at improving mechanical strength, deliverability, and compatibility with blood vessels. A typical DES consists of three main geometric parts: struts (pillars), crowns, and connectors. The struts and crowns from repeating ring-like segments that provide radial support, while connectors link these rings to maintain both stability and flexibility along the length of the stent. One of the most notable recent innovations in DES design is the adoption of open-cell architecture, now common in clinical use. This design allows for better conformity to vessel curves and easier access to side branches, particularly in bifurcation lesions. Biomechanically, the open-cell structure also promotes smoother blood flow downstream of the stent, reducing recirculation zones and abnormal wall shear stress (WSS), which are known to contribute to neointimal growth and restenosis. Strut thickness is a key factor affecting both the mechanical performance and blood flow behavior. Most modern DES features struts ranging from 74 to 90 μm, balancing radial strength and flow compatibility. Two devices stand out for deviating from this range: the Resolute Onyx (Medtronic Vascular), which has a circular cross-section to improve deliverability and reduce flow disturbance, and the Orsiro stent (Biotronik Inc.), notable for its ultra-thin struts of 60 μm—the thinnest among commercial options (Blum et al., 2020). Thinner struts tend to produce better hemodynamic profiles, resulting in less flow separation and more physiological WSS patterns. This improves endothelial healing and lowers the risk of thrombosis. Nevertheless, extremely thin struts may weaken mechanical stability, especially in calcified or tortuous vessels, requiring careful selection of materials and design optimization. From an engineering perspective, the interaction between strut geometry, material elasticity, polymer coating thickness, and drug elution kinetics shapes the overall biomechanical profile of a DES. Therefore, the newest generation of DES is designed not only for drug effectiveness and biocompatibility but also to restore and maintain vascular flow dynamics after implantation—an important factor given the changes in local hemodynamics caused by the stent and their long-term clinical effects.

 CC BY NC 4.0

## Stent Materials and Coatings

### Mechanical and Hemodynamic Implications

The performance of drug-eluting stents (DES) depends on the mechanical properties and biocompatibility of their materials and coatings. Most modern DES are made from cobalt-chromium (CoCr) alloys because of their good strength-to-weight ratio and radiopacity. Notable exceptions include the Synergy stent (Boston Scientific), which uses a platinum-chromium (PtCr) alloy to improve visibility and flexibility, and the Resolute Onyx stent, which features a platinum-iridium (PtIr) core surrounded by CoCr for better radial strength and biocompatibility (Blum et al., 2020).

### Drug Coatings and Clinical Evolution

Early-generation DES used coatings of cytostatic drugs, primarily paclitaxel and sirolimus, to prevent smooth muscle cell growth and reduce in-stent restenosis (ISR). Clinical trials showed that sirolimus-eluting stents were more effective, with notably lower ISR and stent thrombosis (ST) rates (Moses et al., 2003; Stone et al., 2004; Schömig et al., 2007). However, despite their success, permanent metallic DES implants carry long-term risks such as vascular stiffening, chronic inflammation, late stent malapposition, and reduced vasomotor function (Tenekecioglu et al., 2016).

### Emergence and Challenges of Bioresorbable Scaffolds (BRS)

To overcome the limitations of permanent implants, bioresorbable scaffolds (BRS) were introduced as a next-generation solution in 2012. These scaffolds, usually made from biodegradable polymers or magnesium-based metals, are designed to provide temporary mechanical support during vessel remodeling, then degrade over two to three years. However, early BRS designs required thicker struts (150–170 μm) to compensate for material fragility, which unfortunately caused adverse hemodynamic effects such as increased flow disturbance and delayed endothelialization (Ang et al., 2017). Initial clinical enthusiasm decreased after the 2017 long-term study results showed higher late ST rates (~2.5% at 3 years) compared to newer DES (~0.6%) (Ribeiro et al., 2018). These findings led to the market withdrawal of BRS devices like Abbott's Absorb, although ongoing research continues to refine material strength and degradation kinetics to unlock the full potential of BRS technology.

## Biomechanical Criteria and Optimization of Stent Performance

From a biomechanical perspective, several parameters need to be optimized to balance radial strength, flexibility, and vascular compatibility.

**- Radial Recoil vs. Vessel Injury**

High recoil requires oversizing the stent relative to the artery, which increases risks of endothelial trauma and ISR (Chaabane et al., 2013). Conversely, reducing recoil by using thicker struts raises arterial wall stress, creating a need for a trade-off (Ribeiro et al., 2021).

**- Deliverability and Flexibility**

Stents must demonstrate sufficient bending flexibility to navigate complex coronary anatomy. While ASTM standards require system-level bending tests, most optimization studies assess stents in isolation (Ribeiro et al., 2018).

-

**Structural Integrity and Longitudinal Strength**

Resistance to axial compressive forces—especially during catheter manipulation—is essential for maintaining longitudinal integrity, which is increasingly part of design evaluations (Torki et al., 2020).

**- Expansion Quality**

Key metrics include foreshortening, malapposition, and overexpansion capacity. Foreshortening impacts lesion coverage, while malapposition-caused by improper stent-vessel wall contact—can result in thrombotic events. Overexpansion is crucial for treating calcified or large-diameter lesions and is evaluated in both bench tests and clinical trials (Ng et al., 2016). Ultimately, the material and geometric properties of a stent directly affect local hemodynamics, wall shear stress distribution, endothelial response, and long-term clinical outcomes. Therefore, a multi-criteria optimization framework that combines mechanical simulation, fluid dynamics modeling, and clinical performance data is vital for developing next-generation stents.

## Biomechanical Analysis of Vascular Stent Performance

A thorough understanding of biofluid mechanics related to atherosclerosis is essential for accurate diagnosis, treatment, and prevention. Atherosclerosis causes arteries to narrow and restrict blood flow, which can be modeled as constrained fluid flow. In the simplified flow model shown in Figure 4 (Chandran et al., 2007), blood flows from the left at

point one and slows down at point two. Importantly, point one is located upstream in the healthy artery before the narrowed region. As with all closed systems, the principle of mass conservation applies, allowing the calculation of blood flow velocity through the narrowed section. This principle states that the mass flow rate stays constant throughout the vessel and is described by Equation (1):

**Equation (1)** $$A_1V_1=A_2V_2$$

Although atherosclerosis is often modeled as fluid flow through a nozzle, stenotic arteries frequently display focal and asymmetric geometries, characterized by short lesion lengths and nonuniform cross-sectional shapes (Chandran et al., 2007). Additionally, the vessel's geometric features—such as curvature, bifurcation, or tapering—significantly influence flow dynamics. An emerging and powerful tool in biofluid mechanics is Computational Fluid Dynamics (CFD), which enables engineers to simulate patient-specific geometries with high accuracy. CFD provides detailed visualization and quantification of flow fields within diseased arteries, offering insights that surpass traditional analytical methods. However, despite significant progress, CFD still faces limitations, especially in accounting for the dynamic and elastic properties of blood vessels and cardiac motion. Current research aims to improve CFD models by incorporating arterial compliance, curvature, and pulsatile contractions (Thondapu et al., 2016). CFD modeling generally assumes that blood behaves as a Newtonian fluid—one with constant viscosity regardless of shear rate. While this assumption is valid in healthy vessels, it can break down in pathological conditions due to the complex flow patterns caused by atherosclerotic plaques (Thondapu et al., 2016). Figure 5 demonstrates a CFD-generated example of blood velocity and pressure distribution as it moves through a stented arterial segment (Grujicic et al., 2012). Section "A" shows the velocity profile during peak systole, where inflow velocity exceeds 200 mm/s and outflow velocity reduces to about 50 mm/s. Section "B" displays the corresponding pressure distribution, with inlet pressures slightly below 14 kPa rising to over 15 kPa at the outlet. Sections "C" and "D" show the velocity and pressure profiles, respectively, during end-diastole.

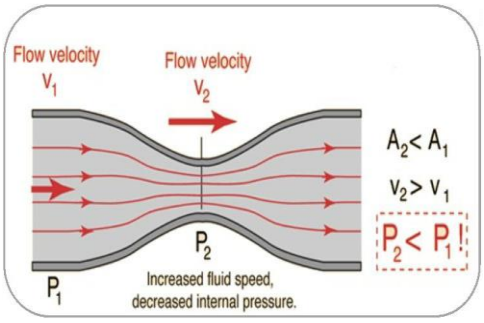


**Figure 4.** Atherosclerotic artery flow model (Chandran et al., 2007)

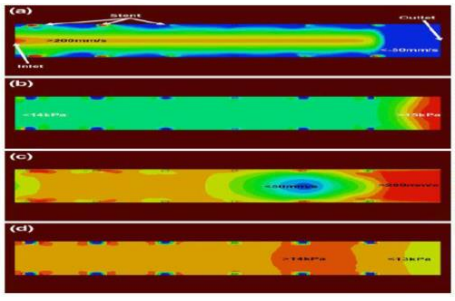

**Figure 5.** Velocity and blood pressure profile (Grujicic et al., 2012)

 CC BY NC 4.0

**Conditions for Applying Load to Vascular Stents**

Vascular stents and their surrounding arteries are exposed to pulsatile and rhythmic blood flow generated by the cardiac systolic and diastolic cycles. These cycles impose varying pressures on the arterial walls, leading to radial stresses that influence both the stent and the vessel. The magnitude of these pressures depends on the artery's distance from the heart and its specific biomechanical properties. As shown in Figure 6 (Robertson et al., 2012), stents experience a combination of compressive and expansive forces throughout the cardiac cycle, along with associated mechanical stresses. During stent deployment via balloon angioplasty, the stent diameter is initially reduced to facilitate delivery and to match the vessel size, a process known as "crimping." This crimping induces strains often exceeding 10%, as illustrated in Figure 6. After deployment, the stent gradually expands until the expansion force balances with the elasticity of the arterial wall. Subsequently, the cyclic pumping of blood during systole and diastole creates a pulsatile mechanical load pattern, as described earlier. Besides resisting pulsatile radial forces, stents must also withstand longitudinal extension, torsion, and bending moments caused by body movements to prevent fracture. The femoropopliteal artery is one of the most challenging regions anatomically due to its high mobility and complex motion patterns (Maleckis et al., 2018). As shown in Figure 7, this artery passes through the pelvic girdle, femur, and patella regions, which permit extensive limb movement. In contrast, upper-body arteries such as the subclavian and brachial arteries, depicted in Figure 8, experience less mechanical deformation. Studies measuring arterial deformation during limb flexion report a wide range of magnitudes, reflecting anatomical differences along the femoropopliteal artery. The most significant deformations occur near the adductor hiatus and below the patella, areas also more vulnerable to atherosclerotic disease (Maleckis et al., 2018). Three independent research groups have measured the range of motion in this segment. The first found that shifting from a supine to a fetal position caused torsion of 60° ± 34° and axial compression of 13% ± 11% within the stent.The second reported rotational twists of 2 to 4 degrees per centimeter, axial compression between 4% and 13%, and bending radii of 22 to 72 mm during limb flexion. The third, using custom markers implanted into cadaveric arteries to measure deformation without impacting stent performance, found higher values: axial compression of 9% to 25%, bending radii of 8 to 27 mm, and torsion from 8° to 26° per centimeter (Maleckis et al., 2018). An important engineering challenge in stent design is ensuring durability under repeated mechanical loading, as these devices endure millions to billions of stress cycles annually and cannot be repaired after placement. Fatigue resistance is therefore crucial for long-term performance and patient safety. Among materials used for stent fabrication, Nitinol is especially notable for its superelasticity, allowing the stent to be compressed for delivery, expanded in place, and recover its shape after deformation caused by limb movement. This property has resulted in widespread use of Nitinol stents, which show significantly lower fatigue failure rates and associated patient readmissions (around 18% or less), compared to conventional stents, where readmission rates have exceeded 50% (He et al., 2021). The mechanical loads come not only from cardiac cycles but also from repeated limb, muscle, and vascular motions, cumulatively subjecting stents to an extremely high number of stress cycles over their lifetime.

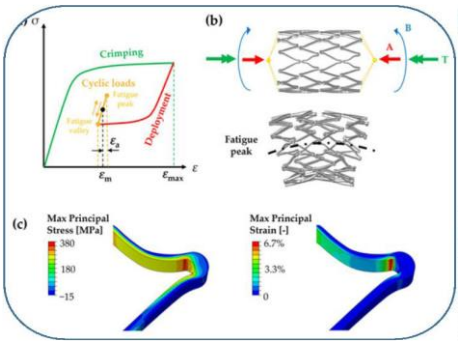


**Figure 6.** Stress-strain of a nitinol stent (Robertson et al., 2012)

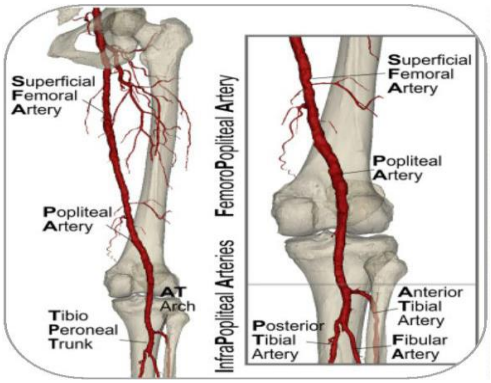


**Figure7.** Femoropiletal artery (Maleckis et al., 2018)

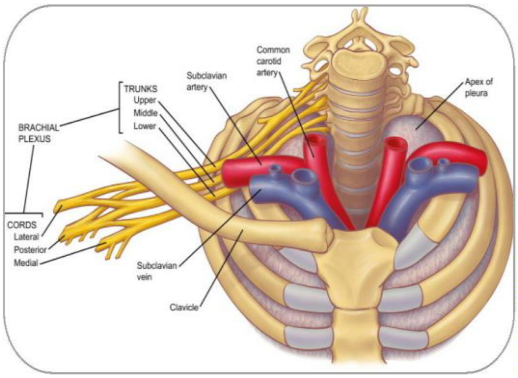


**Figure 8.** Subclavian and brachial arteries

## Conclusion

Post-deployment the biomechanical influence of stents on blood flow dynamics is significant, especially in coronary and other arterial vessels. Stents are vital for improving circulatory function by altering arterial geometry, reducing stenosis, and preventing restenosis. However, their biomechanical performance depends heavily on material properties, mechanical features, and deployment methods. Metallic materials like Nitinol, with their superelastic behavior, offer notable advantages by allowing the arteries' natural deformation and movement. This property helps stents return to their original shape after deployment, which reduces negative effects on blood flow. Despite these advantages, stents face complex loading conditions that can cause fatigue and corrosion, potentially affecting long-term durability. Additionally, the presence of stents can change arterial geometry, leading to alterations in blood flow such as lower flow velocity, increased flow stratification, and localized pressure changes along the vessel wall—all of which may contribute to adverse clinical outcomes. Bioresorbable stents offer a promising solution to the long-term issues linked with permanent metallic implants. However, challenges related to their absorption rates and biomechanical interactions with blood flow still need thorough research. In summary, while stents generally help restore proper blood flow, ongoing advances in material science and design are essential to reduce their biomechanical limitations and ensure the long-term health and function of arterial systems.

## Ethical Statements

This article provides a comprehensive review of existing literature and does not involve collecting new data from human participants or animals. Therefore, no ethical approval or consent to participate was required for preparing this manuscript. All sources cited in this review were properly acknowledged, and the study followed ethical standards regarding the use of previously published materials.

 CC BY NC 4.0

## Authors Contributions

Mohammad Moradi wrote and revised the manuscript. Hanieh Habibi Jirdehi assisted with the literature review and supported the study throughout. All authors have read and approved the final version of the manuscript.

## Conflicts of Interest

The authors declare that they have no conflicts of interest related to this manuscript.

## Funding

This research received no external funding.

## Reference

1.Ang HY, Bulluck H, Wong P, et al. Bioresorbable stents: Current and upcoming bioresorbable technologies. *Int J Cardiol*. 2017;228: 931-939.
2. Bangalore S, Toklu B, Patel N, et al. Newer-Generation Ultrathin Strut Drug-Eluting Stents Versus Older Second-Generation Thicker Strut Drug-Eluting Stents for Coronary Artery Disease. *Circulation*. 2018; 138(20): 2216-2226.
3. Blum M, Cao D, Mehran R. Device profile of the Resolute Onyx Zotarolimus eluting coronary stent system for the treatment of coronary artery disease: overview of its safety and efficacy. *Expert Rev Med Device*. 2020;17(4): 257-265.
4. Camenzind E, Steg PG, Wijns W. Stent thrombosis late after implantation of first-generation drug-eluting stents: a cause for concern. *Circulation*. 2007;115(11):1440-1455.
5. Chandran K, Rittgers S, Yoganathan A. Biofluid Mechanics. *Taylor & Francis Group*. 2006.
6. Chistiakov DA, Orekhov AN, Bobryshev YV. Effects of shear stress on endothelial cells: go with the flow. *Acta physiol (Oxf)*. 2017;219(2):382-408.
7.En.wikipedia.org. Atherosclerosis. *Wikipedia*. 2022.
8. Gertz SD, Roberts WC. Hemodynamic shear force in rupture of coronary arterial atherosclerotic plaques. *Am J Cardiol*. 1990;66(19):1368–1372.
9.Grujicic M, Pandurangan B, Arakere A, et al. Fatigue-Life Computational Analysis for the Self-Expanding Endovascular Nitinol Stents. *JMEP*. 2012;21(11):2218-2230.
10. Halcox JP, Schenke WH, Zalos G, et al. Prognostic value of coronary vascular endothelial dysfunction. *Circulation*. 2002;106(6):653–658.
11. Hall JE, Hall ME. Guyton and Hall Textbook of Medical Physiology. *Elsevier Health Sciences*. 2020.
12. Hoffmeister HM, Jur M, Ruf-Lehmann M, et al. Endothelial tissue-type plasminogen activator release in coronary heart disease: Transient reduction in endothelial fibrinolytic reserve in patients with unstable angina pectoris or acute myocardial infarction. *J Am Coll Cardiol*. 1998;31(3):547–551.
13. Kandzari DE, Mauri L, Koolen JJ, et al. Ultrathin, bioresorbable polymer sirolimus-eluting stents versus thin, durable polymer everolimus-eluting stents in patients undergoing coronary revascularisation (BIOFLOW V): a randomised trial. *Lancet*. 2017; 390(10105):1843-1852.
14. Kinlay S, Young MM, Sherrod R, et al. Long-Term Outcomes and Duration of Dual Antiplatelet Therapy After Coronary Intervention With Second-Generation Drug-Eluting Stents: The Veterans Affairs Extended DAPT Study. *J Am Heart Assoc*. 2023;12(2):e027055.
15. Libby P, Ridker PM, Hansson GK, and Leducq Transatlantic Network on Atherothrombosis. Inflammation in Atherosclerosis: from pathophysiology to practice. *J Am Coll Cardiol*. 2009;54(23):2129-38.
16. Marcus AJ, Broekman MJ, Drosopoulos JH, et al. Role of CD39 (NTPDase-1) in thromboregulation, cerebroprotection, and cardioprotection. *Semin Thromb Hemost*. 2005;31(2):234–246.
17. Marieb EN, Hoehn K. Human Anatomy & Physiology. *Pearson Education*. 2018.
18. Martini F, Nath J, Bartholomew E. Fundamentals of Anatomy and Physiology. 10th ed. *Pearson Education Inc*. 2015.
19. Massa KHC, Duarte YAO, Chiavegatto Filho ADP. Analysis of the prevalence of cardiovascular diseases and associated factors among the elderly, 2000-2010. *Cien Saude Colet*. 2019;24(1):105-14.
20.Moses JW, Leon MB, Popma JJ, et al. Sirolimus-eluting stents versus standard stents in patients with stenosis in a native coronary artery. *N Engl J Med*. 2003;349(14):1315-23.
21.Navarese EP, Kowalewski M , Kandzari D, et al. First-generation versus second-generation drug-eluting stents in current clinical practice: updated evidence from a comprehensive meta-analysis of randomised clinical trials comprising 31,379 patients. *Open Heart*. 2014;1(1): e000064.
22.Ng J, Foin N, Ang HY, et al. Over-expansion capacity and stent design model: An update with contemporary DES platforms. *Int J Cardiol*. 2016;221:171-179.
23. Pericevic I, Lally C, Toner D, et al. The influence of plaque composition on underlying arterial wall stress during stent expansion: the case for lesion-specific stents. *Med Eng Phys*. 2009;31(4):428-33.
24.Peters B, Ewert P, Berger F. The role of stents in the treatment of congenital heart disease: Current status and future perspectives. *Ann Pediatr Cardiol*. 2009;2(1):3-23.
25. Räber L, Magro M, Stefanini GG, et al. Very Late Coronary Stent Thrombosis of a Newer-Generation Everolimus-Eluting Stent Compared With Early-Generation Drug-Eluting Stents: a Prospective Cohort

Study. *Circulation*. 2012;125(9): 1110-1121.
26. Ribeiro ALP, Duncan BB, Brant LCC, et al. Cardiovascular health in Brazil: Trends and Perspectives. *Circulation*. 2016;133(4):422-33.
27. Ribeiro NS, Folgado J, Rodrigues HC. Surrogate-based multi-objective design optimization of a coronary stent: Altering geometry toward improved biomechanical performance. *Int J Numer Method Biomed Eng*. 2021;37(6): e3453.
28. Ribeiro NS, Folgado J, Rodrigues HC. Surrogate-based visualization and sensitivity analysis of coronary stent performance: A study on the influence of geometric design. *Int J Numer Method Biomed Eng*. 2018; 34(10): e3125.
29. Ridker PM, Danielson E, Fonseca FAH, et al. Rosuvastatin to prevent vascular events in men and women with Elevated C-Reactive Protein. *N Engl J Med*. 2008;359(21):2195-207.
30. Rikhtegar F, Wyss C, Stok KS, et al. Hemodynamics in coronary arteries with overlapping stents. *J Biomech*. 2014; 47(2):505-11.
31. Rong JX, Rangaswamy S, Shen L, et al. Arterial injury caused by cholesterol oxidation products leads to endothelial dysfunction and arterial wall cholesterol accumulation. *Arterioscler Thromb Vasc Biol.* 1998; 18(12):1885–1894.
32. Serruys PW, de Jaegere P, Kiemeneij F, et al. A Comparison of Balloon-Expandable-Stent Implantation with Balloon Angioplasty in Patients with Coronary Artery Disease. *N Engl J Med*. 1994; 331(8):489-495.
33. Sigwart U, Puel J, Mirkovitch V, et al. Intravascular Stents to Prevent Occlusion and Re-Stenosis after Transluminal Angioplasty. *N Engl J Med*. 1987;316(12) :701-706.
34. Silverthorn DU. Human Physiology: An Integrated Approach. *Pearson Education*. 2020.
35. Soulis JV, Fytanidis DK, Seralidou KV, et al. Wall shear stress oscillation and its gradient in the normal left coronary artery tree bifurcations. *Hippokratia*. 2014;18(1):12-6.
36. Stary HC, Chandler AB, Glagov S, et al. A definition of initial, fatty streak, and intermediate lesions of atherosclerosis. A report from the Committee on Vascular Lesions of the Council on Arteriosclerosis, American Heart Association. *Circulation.* 1994;89(5):24, 62-78.
37. Stone GW, Ellis SG, Cox DA, et al. A polymer-based, paclitaxel-eluting stent in patients with coronary artery disease. *N Engl J Med*. 2004;350(3):221-31.
38. Strauss BH, Tanguay JF, Picard F, et al. Coronary Stenting: Reflections on a 35-Year Journey. *Can J Cardiol*. 2022;38(10 Suppl1): S17-S29.
39. Sumi T, Yamashita A, Matsuda S, et al. Disturbed blood flow induces erosive injury to smooth muscle cell-rich neointima and promotes thrombus formation in rabbit femoral arteries. *J Thromb Haemost.* 2010;8(6):13 94–1402.
40. Tenekecioglu E, Farooq V, Bourantas CV, et al. Bioresorbable scaffolds: a new paradigm in percutaneous coronary intervention. *BMC Cardiovasc Disord*. 2016; 16:38.
41. Thondapu V, Bourantas CV, Foin N, et al. Biomechanical stress in the coronary atherosclerosis: emerging insights from computational modelling. *Eur Heart J*. 2017; 38(2): 81-92.
42. Torki MM, Hassanajili S, Jalisi MM, Design optimizations of PLA stent structure by FEM and investigating its function in a simulated plaque artery. *Mathematics and Computers in Simulation*. 2020;169 :103-116.
43.Tortora GJ, Derrickson BH. Principles of Anatomy and Physiology. *John Wiley & Sons*. 2018.
44. Virmani R, Kolodgie FD, Burke AP, et al. Lessons from sudden coronary death: a comprehensive morphological classification scheme for atherosclerotic lesions. *Arterioscler Thromb Vasc Biol*. 2000;20(5):1262 75.
45.Wentzel JJ, Krams R, Schuurbiers JC, et al. Relationship between neointimal thickness and shear stress after Wallstent implantation in human coronary arteries. *Circulation*. 2001; 103(13):1740-5.
46. Widmaier EP, Raff H, Strang KT. Vander's Human Physiology: The Mechanisms of Body Function. *McGraw-Hill Education*. 2018.

 *CC BY NC 4.0*