

Hemodynamic responses to physical activity: Numerical analysis of dynamic behavior in microvascular structures under exercise-induced forces

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Abstract. This study investigates the complex relationship between physical activity and hemodynamic changes in the circulatory system using advanced mechanical and mathematical modeling. Under dynamic load, blood vessels are portrayed as microtubular structures, allowing for precise characterization of their biomechanical responses to exercise-induced forces. The microscale effects of pulsatile blood flow caused by physical exertion are accurately captured by the proposed model, which combines classical beam and tube theories with the size-dependent modified couple stress theory. The governing equations are solved using a rigorous numerical framework, allowing for detailed analysis of stress-strain distributions, wall shear stress, and vascular deformation across a wide range of hemodynamic conditions. The results show that exercise-induced shear stresses and pressure variations help to strengthen vascular walls, emphasizing sports' critical role in improving vascular resilience. This study combines sports physiology and biomechanical engineering to provide a predictive framework for assessing athletic training-induced vascular adaptations. By emphasizing the importance of exercise in cardiovascular health, the study provides valuable insights for optimizing training regimens and developing targeted rehabilitation strategies. This interdisciplinary approach improves our understanding of hemodynamic behavior in physically active people, paving the way for novel applications in sports medicine and vascular health management.

Keywords: exercise-induced shear stress; hemodynamic adaptations; microtube modeling; modified couple stress theory; sport activities; vascular biomechanics

1. Introduction

Hemodynamics examines how blood moves and flows through vessels, as well as the physical elements that influence blood circulation in the human body (de Keijzer and Scheeren 2021). Researchers have conducted several research on hemodynamics since it is an important aspect of cardiovascular physiology, with the heart serving as the primary force that drives all blood flow (Pollock *et al.* 2023). The circulatory system is regulated by homeostatic mechanisms such as autoregulation, which ensure that oxygen and nutrients reach the tissues while keeping the body steady. Previous studies have identified hemodynamic variables such as arterial pressure, tissue perfusion, and cardiac output as critical indicators of cardiovascular health (Chen *et al.* 2024, Fang *et al.* 2024, Gao *et al.* 2024). The qualities were critical for understanding normal physiological processes, as well as diagnosing and treating disorders. Changes in venous saturation and lactic acid levels were often connected to inadequate tissue perfusion and

metabolic problems. Furthermore, the link between pressure, flow, and resistance was identified as a basic feature of hemodynamics that determines how blood travels as a fluid through the vascular system (Tamimi 2024). The principles of hemodynamics, namely blood volume and deoxyhemoglobin concentration, were studied in the context of cerebral activity and cardiovascular disease. The researchers emphasized the importance of understanding these dynamics in establishing the etiology of hemodynamic changes in sickness development (Wong *et al.* 2017). Such discoveries paved the way for advancements in medical imaging and computer modeling, enabling for the creation of relevant hemodynamic indicators to help clinical decision-making. Scientists try to comprehend the complexities of hemodynamic behavior under diverse physiological conditions by integrating theoretical frameworks with empirical facts. This foundational knowledge laid the door for further study into how external stimuli, such as physical effort, affect hemodynamic responses (Hu and Feng 2017, Li and Xu 2024, Liu *et al.* 2025b).

Physical exercise has a substantial impact on hemodynamic responses, aerobic and resistance training cause a variety of physiological changes. Research was

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conducted to assess the effect of various exercise regimes on key hemodynamic variables such as blood pressure, cardiac output, and vascular resistance (Liu *et al.* 2024a, Mao *et al.* 2024, Shen *et al.* 2024). Diastolic blood pressure rose considerably after 15 and 30 minutes of aerobic exercise, but not after strength training, demonstrating that various exercise modalities have distinct effects on cardiovascular health. Heat stress and other environmental factors had a considerable impact on limb blood flow and cardiac output during physical exercise. Heat stress raises thermoregulatory demands, resulting in increased skin perfusion and altered blood flow distribution during rest and exercise (Petrov Fieril *et al.* 2016). The findings show that the complex combination of metabolic and thermoregulatory demands impacts hemodynamic behavior under a variety of environmental situations. Physical exercise has a substantial impact on hemodynamic responses, aerobic and resistance training cause a variety of physiological changes. A study was done to look at the impact of different exercise regimens on crucial hemodynamic variables as blood pressure, cardiac output, and vascular resistance (Wang *et al.* 2023a, b, Tang *et al.* 2024). Diastolic blood pressure rose considerably after 15 and 30 minutes of aerobic exercise, but not after strength training, demonstrating that various exercise modalities have distinct effects on cardiovascular health. Heat stress and other environmental variables significantly influenced limb blood flow and cardiac output during physical activity. Heat stress increases thermoregulatory needs, resulting in improved skin perfusion and altered blood flow distribution during rest and activity. The results show that the complicated interaction of metabolic and thermoregulatory needs influences hemodynamic behavior in a variety of environmental conditions (Pearson *et al.* 2010). Low-load blood flow restriction (BFR) exercise has piqued interest, with studies contrasting its immediate hemodynamic effects with unrestricted low- and high-load exercises. Blood flow restriction exercise for small muscle groups resulted in higher systolic blood pressure increases than standard strength training without BFR (Bonorino *et al.* 2019). The study found that mechanical and metabolic stressors associated with blood flow restriction had a significant impact on hemodynamic responses. Peak activity research has shown that people with equal peak oxygen consumption have identical hemodynamic responses, regardless of metabolic status. The observed consistency indicates that exercise intensity and duration have a greater impact on hemodynamic changes than pre-existing medical conditions (Hibner *et al.* 2024). Patients with chronic thromboembolic illness demonstrated abnormal pulmonary vascular responses during exercise, emphasizing the importance of detecting pathological deviations from routine hemodynamic profiles (Tamimi 2024). The research found that physical activity induces complex and varied hemodynamic changes, influenced by the type, intensity, and context of the exercise. This research enhances comprehension of cardiovascular physiology and establishes a foundation for creating exercise programs aimed at improving vascular health and resilience (Karlsdottir *et al.* 2002).

Microvascular structures have been found as critical

components of the circulatory system, regulating blood flow and maintaining tissue homeostasis. The coronary microvasculature, in particular, has gained a lot of interest because it plays an important role in maintaining a tight relationship between myocardial perfusion and oxygen demand under a range of physiological situations (Brandt *et al.* 2021). Researchers proved that these intricate networks not only supplied nutrients but also facilitated waste elimination, underscoring their dual function in cellular health (Qi *et al.* 2024). A notable focus was on the structural adaptation of microvascular networks, with research demonstrating how hemodynamic and metabolic stressors affected vessel diameter and density (Pries *et al.* 2001). The alterations were prompted by mechanical forces applied to the endothelium, including wall shear stress, which began vascular remodeling (Brandt *et al.* 2021). Alterations in microvascular diameter have been shown to modify blood flow resistance, hence influencing total cardiovascular function (Pries *et al.* 2001). Likewise, changes in capillary density have been associated with enhanced oxygen delivery and tissue perfusion, especially in reaction to exercise or pathological states (Nielsen *et al.* 2020). The many functions of microcirculation were investigated in both pulmonary and systemic settings. The researchers emphasized the importance of pulmonary microcirculation in promoting successful gas exchange and maintaining oxygen levels. Systemic micro-vessels are structurally adaptable to biomechanical parameters such as blood pressure, velocity, and viscosity, which vary between healthy and diseased situations (Hoefer *et al.* 2013). The findings emphasize microvascular systems' adaptive properties and ability to respond to external and internal stimuli. Alterations in the architecture of small arteries and arterioles are linked to an increased risk of cardiovascular disease, particularly in those with hypertension and diabetes. Increased arterial wall thickness and decreased lumen widths were noted, indicating compromised blood flow regulation and intensified tissue hypoxia (Agabiti-Rosei and Rizzoni 2017). This study emphasizes the significance of comprehending microvascular function in both health and disease progression. The microvasculature is crucial for hemodynamic stability and tissue survival, with its structural and functional properties profoundly influenced by physiological demands and pathological stressors (Dong *et al.* 2024). These findings provide a basis for further research on the biomechanical and hemodynamic interactions within microvascular networks, especially under physical effort or stress (Pries *et al.* 2001).

Numerical analysis and computational modeling approaches have substantially enhanced the understanding of hemodynamic dynamics in complicated vascular systems (Zhao *et al.* 2024). Researchers widely used computational fluid dynamics (CFD) to model pulsatile blood flow and arterial wall mechanics, especially in situations involving pathological states like stenosis (Zisong and Habibi 2024). The computer models enabled a comprehensive analysis of flow patterns and hemodynamic features that were previously difficult to examine experimentally (Li *et al.* 2024b). The creation of idealized and patient-specific models to assess the impact of vascular geometry on

hemodynamics is a significant accomplishment in this domain. For example, investigations indicated that the degree and form of stenosis had a significant impact on flow patterns, wall shear stress distributions, and pressure gradients inside affected channels (Zhiqiang *et al.* 2024). Such findings were crucial for understanding the processes that underpin plaque development and progression, as well as the consequences for cardiovascular health (Zhu *et al.* 2024b). In addition to stenosis, researchers investigated the hemodynamic consequences of asymmetric and curved vascular geometries, which are typical in real-world settings (Song *et al.* 2024). These studies found that uneven degrees of stenosis and curved artery segments resulted in complicated flow phenomena such as recirculation zones and secondary flows, exacerbating vascular dysfunction (Liang *et al.* 2024). The combination of accurate analytical answers and numerical methodologies offered a solid foundation for deciphering these complex flow dynamics (Huo *et al.* 2021). Additionally, numerical modeling was used to evaluate cerebrovascular hemodynamics, resulting in the development of multi-EMG models that replicate cerebral blood flow under a variety of physiological situations (Zhang *et al.* 2024c). The models emphasized the asymmetrical characteristics of cerebral hemodynamics and provided prediction capacities for determining the probability of intracranial aneurysms (Huang *et al.* 2024). The validation of Computational fluid dynamics (CFD) models demonstrates the accuracy and dependability of these computational approaches, emphasizing their importance in clinical applications (Man *et al.* 2024). The ability to predict systemic hemodynamics under orthostatic stress and other challenging conditions has been enhanced through the use of advanced computational methods, including one-dimensional modeling of arterial blood flow (Xiao *et al.* 2024). These approaches allowed researchers to examine the transmission of pressure and flow waves across the arterial network, yielding significant insights into the biomechanical reactions of the circulatory system (Xia *et al.* 2025). Artificial neural networks combined with computational methods were used to forecast the stability of non-uniform functionally graded microscale cylindrical constructions pertinent to sports equipment design (Liu *et al.* 2024b). This multidisciplinary approach combines sports physiology and biomechanical engineering to create novel solutions for enhancing athletic performance and safety (Yin *et al.* 2024). Furthermore, hybrid nonlocal strain gradient theory was used to assess the dynamic stability of inclined curved nano-pipes in supersonic airflow, demonstrating the usefulness of numerical methods in a wide range of engineering applications. This study highlighted the importance of combining machine learning and optimization techniques, such as PSO-ANN and firefly algorithms, to improve the accuracy and efficacy of numerical simulations (Zhang *et al.* 2024a). The use of numerical analysis and sophisticated modeling techniques has resulted in a thorough understanding of hemodynamic behavior in both healthy and diseased vascular systems. The methodologies lay a solid foundation for advances in cardiovascular research and clinical practice by successfully connecting theoretical principles with practical applications.

Simulation and analysis of complex blood flow dynamics have allowed for the development of more effective diagnostic tools, treatment strategies, and therapeutic interventions.

The integration of sports physiology and biomechanical engineering signifies a significant advancement in comprehending human movement and enhancing athletic performance (Potop *et al.* 2024). Scholars meticulously examined the application of biomechanical principles in assessing and enhancing physiological responses throughout physical exercise. Biomechanical analysis has been employed extensively to discern the most suitable methodologies for assessing movement patterns, thereby facilitating the development of tailored rehabilitation treatments and training programs (Rebelo *et al.* 2023). Biomechanics has made significant contributions to sports medicine by providing a thorough understanding of human movement, resulting in improved athletic performance and injury prevention. Advanced motion analysis methodologies, wearable technologies, and biomechanical modeling are considered critical tools for better understanding athlete performance and recovery processes. The methodologies used allowed for precise measurement of a number of variables, including joint kinematics, muscle activation patterns, and ground reaction forces, resulting in a successful correlation between physiological demands and mechanical stress (Vancini *et al.* 2023). The multi-disciplinary cooperation of sports physiologists and biomedical engineers enhanced the profession by tackling complicated issues in sports rehabilitation. For example, biomechanical therapy has been demonstrated to dramatically improve motor abilities in patients, with more than 60% displaying improved movement quality post-intervention (Zhang 2024). The research results showed that effective treatment strategies require the incorporation of biomechanical understanding and physiological assessments (Tai *et al.* 2023). Furthermore, innovative research has demonstrated how bionic engineering and biomechanical advancements can improve sports equipment and training environments (Kanjilal and Mondol 2017). Advances in biomechanics have benefited both elite athletes and the general public, promoting physical activity and health across all demographics (Kanjilal and Mondol 2017). Researchers were able to create prediction models that incorporated physiological and biomechanical data, paving the way for personalized therapies in sports medicine (Vancini *et al.* 2023). To summarize, the relationship between sports physiology and biomechanical engineering has been recognized as a cornerstone of modern sports science, providing novel approaches to improving performance, reducing injuries, and accelerating recovery. This interdisciplinary collaboration not only improved theoretical knowledge, but also produced practical applications that benefited athletes and the general public (Zatsiorsky 2008).

Shear stress caused by exercise is an important physiological stimulus for controlling vascular function and improving cardiovascular health (Cook 2020). Researchers investigated the relationship between increased blood flow during exercise and the resulting frictional forces on the

endothelium, which trigger adaptive responses at both the molecular and structural levels (Tinken *et al.* 2010). These adaptations were critical for increasing vascular resilience and lowering the risk of cardiovascular disease (Niebauer and Cooke 1996). Shear stress's ability to modulate key molecular pathways best describes the relationship between exercise and endothelial function. For example, exercise-induced increases in shear stress were shown to promote the upregulation of endothelial nitric oxide synthase (eNOS), a key enzyme involved in vasodilation and vascular protection (Garcia *et al.* 2022). Moderate-intensity exercise, in particular, was discovered to produce the highest levels of eNOS expression, indicating the optimal intensity for maximizing endothelial benefits (Gurovich *et al.* 2022). These findings demonstrated the significance of shear stress as a powerful physiological stimulus for improving vascular health (Conde *et al.* 2024). Exercise-induced shear stress has been shown to promote mRNA translation in vitro, emphasizing its importance in activating endothelial protective mechanisms. Messenger ribonucleic acid (mRNA) is a single-stranded RNA required for protein production. Transcription converts a DNA template into RNA, which serves as an intermediate between DNA and ribosomes, where protein building occurs. The major purpose of mRNA is to aid in the translation of genetic instructions into functional proteins, which are required for cellular functions and general biological function (Wang *et al.* 2024h, Wang *et al.* 2024i, Wu *et al.* 2025). This process increased both the availability of eNOS and the activity of SIRT1, a protein involved in anti-aging and vascular repair. Shear stress reduces the expression of endothelin-1 (ET1), a vasoconstrictor linked to vascular injury, resulting in an anti-atherogenic phenotype. Molecular adaptations were crucial for the restoration of vascular damage and the maintenance of endothelial integrity (Rodríguez-Núñez *et al.* 2016). Exercise-induced shear stress influences clinical outcomes by affecting wall shear stress (WSS) patterns in individuals with cardiovascular disorders. Exercise was found to cause significant changes in WSS, which were associated with improvements in vascular structure and function (Xu and Xu 2023). These changes were particularly important for non-invasive cardiovascular disease prevention because they helped to restore endothelial homeostasis and reduce atherosclerotic risk (Königstein *et al.* 2023). Finally, exercise-induced shear stress was identified as a critical component of vascular adaptation, resulting in both molecular and clinical improvements in endothelial function (Rodríguez-Núñez *et al.* 2016). Researchers clarified the physiological basis and clinical significance of shear stress, paving the way for targeted interventions aimed at improving vascular health through physical activity. These findings not only improved our understanding of cardiovascular physiology, but also showed that exercise has therapeutic potential in the prevention and treatment of vascular diseases. The discovery of a direct link between shear stress and vascular function paved the way for evidence-based strategies to improve vascular health, emphasizing the value of exercise as a powerful tool in both clinical and preventive care (Fiuza-Luces *et al.* 2018).

Despite substantial advances in our knowledge of hemodynamic responses to physical exercise, numerous crucial gaps remained unfilled in the extant research. One major shortcoming was the scarcity of thorough research examining the microscale effects of exercise-induced pressures on vascular systems (Green *et al.* 2017). Despite the extensive research conducted on macroscopic hemodynamic parameters, such as blood pressure and cardiac output, there is still a substantial gap in the comprehension of the intricate biomechanical interactions that take place within microvascular networks during physical activity (Secomb 2016). The current ambiguity has hampered a thorough understanding of how localized shear stresses and pressure fluctuations affect vascular adaptation at the microscale level. Furthermore, future research has been identified as a promising avenue for applying sophisticated numerical modeling methodologies to forecast hemodynamic dynamics in changing contexts. Addressing these gaps may result in a more nuanced and comprehensive understanding of microvascular responses to physical exercise and other dynamic conditions, hence enhancing vascular health management strategies. While computational techniques can predict blood flow and vascular mechanics, their use in exercise-specific contexts is still restricted (Zhao *et al.* 2021, 2023). This restriction emphasizes the need for more focused and specialized research in this sector, which may provide unique insights into the complicated dynamics within microvascular networks and improve the development of tailored methods for vascular well-being (Cioccarri *et al.* 2018). Moreover, there was an absence of research that combined sports physiology with biomechanical engineering to create prediction models for vascular health management. Current research often emphasizes either physiological responses or biomechanical adaptations, neglecting their interrelationship. This disjointed methodology restricted the capacity to create comprehensive medications that enhanced both athletic performance and cardiovascular endurance (Zhang *et al.* 2021a, Zhu *et al.* 2024a, Zhan *et al.* 2025). Furthermore, the diagnostic potential of noninvasive methods, such as cardiopulmonary exercise testing (CPET), has not been well used to distinguish between healthy adaptations and pathological conditions in response to exercise. This research aims to examine the intricate link between physical activity and hemodynamic alterations via sophisticated mechanical and mathematical models. The research aimed to describe the dynamic behavior of microvascular structures under exercise-induced stresses, using a comprehensive computational framework to assess stress-strain distributions, wall shear stress, and vascular deformation. The research sought to provide a prediction paradigm for evaluating vascular responses to athletic training by integrating ideas from sports physiology and biomechanical engineering. These goals helped to close current gaps in the literature and support creative uses in sports medicine and vascular health management.

The findings of this study highlighted the crucial role that exercise-induced hemodynamic changes play in improving cardiovascular resilience, which has significant ramifications for the development of sports medicine and

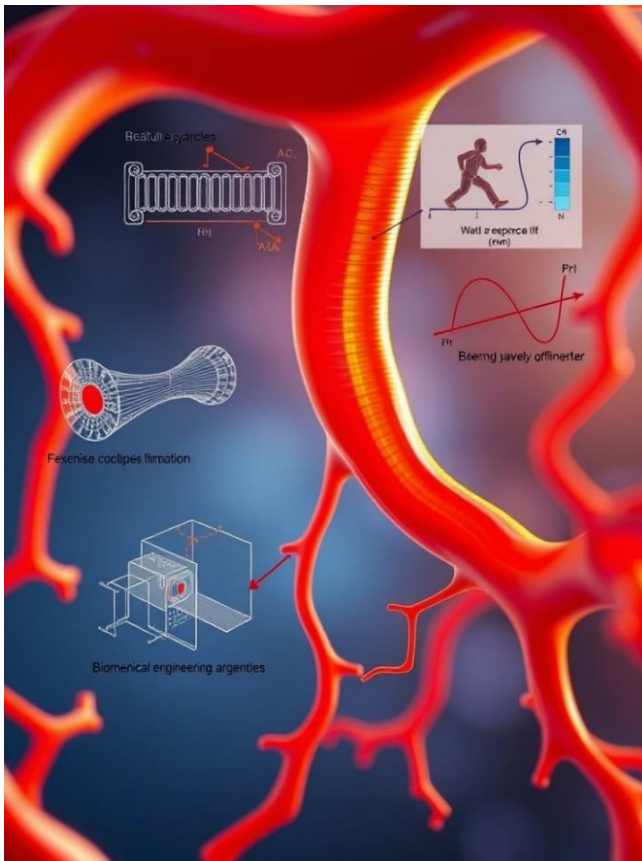


Fig. 1 Schematic representation of the integration of mechanical engineering principles with physical activity to model hemodynamic responses in microvascular structures

vascular health management. The potential of physical exercise to enhance vascular function by increasing blood flow and shear stress was widely acknowledged as an essential element of preventive healthcare. In addition to having positive impact on athletic performance, these physiological adaptations have a preventive mechanism against cardiovascular disorders, making them very important to clinical practice (Green and Smith 2018). Insights into enhancing training regimens for athletes were obtained by understanding the molecular and clinical impacts of exercise on vascular structures within the realm of sports medicine. Research has shown that exercise-induced increases in luminal shear stress result in enhanced production of endothelial nitric oxide (NO), which is crucial for vascular repair and vasodilation. This mechanistic understanding could support the creation of targeted interventions designed to improve vascular health and reduce the risk of injury during intense physical activity (Newcomer *et al.* 2011). Moreover, earlier research indicates that physical activity can alleviate the adverse effects of sedentary behavior on vascular parameters, underscoring its significance in both prevention and rehabilitation (Soto-Rodríguez *et al.* 2024). The work focuses on microscale hemodynamic reactions, which provide novel paths for diagnosing and treating cardiovascular problems, especially vascular health. Noninvasive monitoring methods, such as cardiopulmonary

exercise testing (CPET), have shown promise in assessing vascular function and detecting early symptoms of disease (Björdalsbakke *et al.* 2024). By combining these methods with computer modeling methodologies, doctors may get a more detailed understanding of how exercise-induced stresses have affected vascular adaptation over time (Sun *et al.* 2024). These prediction frameworks are critical in the treatment of chronic illnesses such as hypertension and atherosclerosis, where vascular remodeling is a key determinant. Furthermore, the multidisciplinary nature of this study linked the fields of sports physiology and biomechanical engineering, allowing for unique applications in customized medicine. For example, the ability to simulate hemodynamic responses under various exercise situations may help greatly in the creation of personalized rehabilitation regimens for those recovering from cardiovascular events. Recent advances have considerably increased the accuracy of treatment interventions, while also emphasizing the importance of exercise as an inexpensive and generally accessible way to improving vascular health in many groups.

2. Mathematical simulation

The investigation of hemodynamic responses to physical activity, as well as numerical analysis of dynamic behavior in microvascular systems, is closely tied to mechanical engineering ideas, particularly the use of mathematical modeling and simulation. In mechanical engineering, mathematical modeling is critical for understanding complex systems and forecasting system behavior in a variety of scenarios. Hemodynamics employs analogous concepts to model blood flow dynamics and vascular responses to exercise-induced stress. This study employs a modeling paradigm for translational and rotational systems within mechanical frameworks, conceptualizing blood vessels as microtubular structures. It utilizes classical beam and tube mechanics alongside advanced methodologies, including modified couple stress theory. These models not only characterize biomechanical responses, but also reveal stress-strain distributions, wall shear stress, and deformation patterns, similar to how mechanical engineers assess structural integrity and performance in designed systems. Thus, the approaches used here are based directly on the rich legacy of mathematical modeling and simulation that supports current mechanical engineering procedures.

The integration of mechanical engineering principles with physical activity presents a unique method for examining the dynamic responses of microvascular structures to exercise-induced pressures. Fig. 1 illustrates the interconnections among these domains, emphasizing how mathematical modeling and biomechanical simulations elucidate the intricate interactions between physical exertion and hemodynamic responses. This research investigates the vascular changes that transpire during physical exercise, envisioning blood vessels as microtubular structures that respond to stress, strain, and shear pressures. The methodology utilizes conventional beam and tube theories in conjunction with a modified couple stress theory.

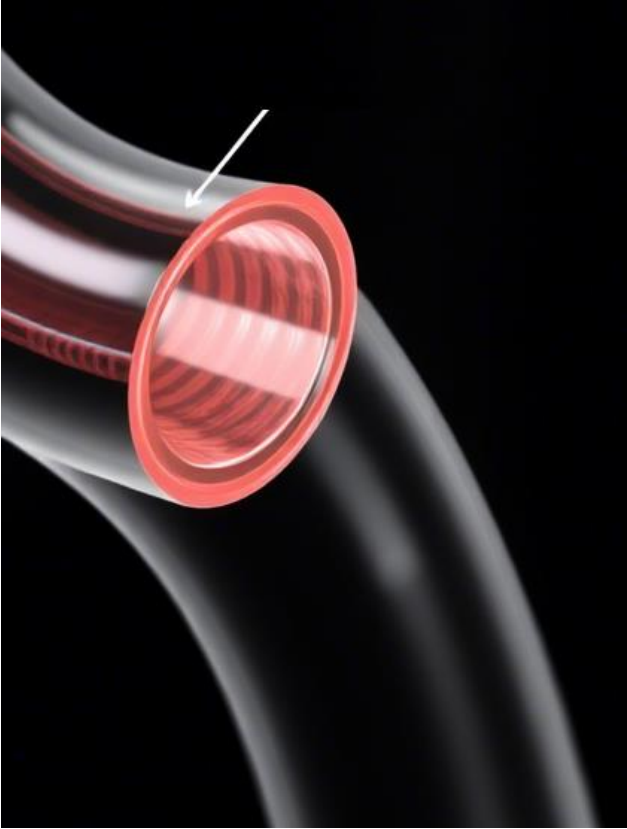


Fig. 2 Geometry detail for a schematic of a blood vessel

The graphic depicts essential aspects of vascular deformation, wall shear stress distribution, and pressure fluctuations, highlighting the significance of exercise in improving vascular resilience via biomechanical processes. This thorough approach underscores the importance of physical activity in cardiovascular health and lays the groundwork for creating prediction models in sports medicine and vascular health management.

2.1 Governing equations and theoretical framework

This study is based on the mathematical modeling of microvascular structures as microtubes under internal pressure, emphasizing their dynamic behavior in response to exercise-induced forces. This framework combines beam and tube theories with the modified couple stress theory to address size-dependent effects at the microscale. The model depicts blood arteries as microscopic tube-like structures, which aids in understanding how their walls adapt to the pressures of physical activity. Blood arteries are shown as thin-walled cylindrical tubes, with internal pressure from blood flow during exercise acting as the principal mechanical stress. Traditional beam and tube theory describes the deformation of microtubular structures and defines the relationship between internal pressure, wall thickness, and material characteristics.

In order to generate the governing equation, the Hamilton principle (H) was employed, which is a classic technique in mechanics for generating equations of motion by minimizing the action integral over time. This concept

assures that the system's behavior aligns with energy conservation and dynamic equilibrium (Dai *et al.* 2021, Guo *et al.* 2024, Wang *et al.* 2024c, d, f, Jining *et al.* 2025, Li *et al.* 2025, Liu *et al.* 2025a).

$$\int_{t_1}^{t_2} \delta H dt = \int_{t_1}^{t_2} \delta S + \delta W - \delta K dt = 0 \quad (1)$$

where ' S ' refer to the strain energy, ' W ' is the energy of the external load, and ' K ' is the Kinetic energy. The strain energy based on the high-order sinusoidal shear deformation beam theory coupled with the modified couple stress theory is defined as follows:

$$S = \frac{1}{2} \iiint \sigma_{ij} \varepsilon_{ij} + m_{ij} \chi_{ij} dV \quad (2)$$

where ' σ_{ij} ' and ' ε_{ij} ' represent the stress and strain tensors, respectively, while ' m_{ij} ' and ' χ_{ij} ' denote the deviatoric part of the couple stress tensor and the curvature tensor, respectively. According to the high-order sinusoidal shear deformation beam theory the displacement fields (U) and defined as follows:

$$U_x = u - z \left(\frac{\partial w}{\partial x} \right) + \varpi(x, z) \left(\phi + \frac{\partial w}{\partial x} \right) \quad (3a)$$

$$U_y = 0 \quad (3b)$$

$$U_z = w \quad (3c)$$

where ' u ', ' w ' and ' ϕ ' are the axial, lateral and rotational parameter, respectively. Also, ' ϖ ' is a parameter regarding the high-order sinusoidal shear deformation beam theory and define as follows:

$$\varpi = \frac{(R_{ex} - R_i)}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \quad (3d)$$

Here ' R_{ex} ' and ' R_i ' are the internal and external vessel radius (Fig. 2).

Then the strain energy based on the mentioned beam theory linked with the size-dependent theory are calculated as follows:

$$\begin{aligned} S = & -Ez \frac{(R_{ex} - R_i)}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x^2} \right) \left(\frac{\partial^2 w}{\partial x^2} \right) \\ & + \frac{E}{2} \left(\frac{\partial u}{\partial x} \right)^2 + \frac{E}{2} \left(\frac{(R_{ex} - R_i)}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x^2} \right) \right)^2 \\ & - Ez \left(\frac{\partial u}{\partial x} \frac{\partial^2 w}{\partial x^2} \right) + \frac{1}{4} GK_S \cos \left(\frac{\pi z}{R_{ex} - R_i} \right)^2 \left(\frac{\partial w}{\partial x} + \phi \right)^2 \\ & + \frac{1}{8} \left(l^2 G \cos^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x^2} \right)^2 \right) + \frac{1}{2} Ez^2 \left(\frac{\partial^2 w}{\partial x^2} \right)^2 \\ & - l^2 GK_S \cos \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x^2} \right)^2 - \frac{1}{4} l^2 GK_S \left(\frac{\partial^2 w}{\partial x^2} \right)^2 \\ & + \frac{1}{4} l^2 GK_S \cos \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x^2} \right)^2 \\ & + \frac{1}{8} l^2 GK_S \left(\frac{\pi}{R_{ex} - R_i} \right)^2 \sin^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial w}{\partial x} + \phi \right)^2 \end{aligned} \quad (4)$$

Table 1 Mechanical properties of blood vessels

Property	Value
Poisson's Ratio (ν)	~0.45–0.5 (arteries) (Camasão and Mantovani 2021)
Density (ρ)	~1,000 kg/m ³ (approximation) (An <i>et al.</i> 2023)
Elastic Modulus (E)	4.70 ± 0.5 MPa (arteries) (Silver <i>et al.</i> 2021)

where ‘ E ’ is elastic modulus, along with the other mechanical properties of blood vessels, are listed in Table 1. Also, ‘ $G = \frac{1}{2}(E/1 + \nu)$ ’ is the shear modulus, and ‘ l ’ is the size-dependent parameter based on the modified couple stress theory. Furthermore, ‘ K_S ’ is the shear correction factor (Zhang *et al.* 2021b, Chen *et al.* 2022, Li *et al.* 2022, Li *et al.* 2024a, Yu *et al.* 2024, Zhu *et al.* 2024c, Wang *et al.* 2025, Zhou *et al.* 2025).

This discussion aims to examine the influence of internal pressure on natural frequency. The literature indicates that the natural frequency is influenced by the effective axial force, which diminishes with increasing internal pressure. The reduction arises from the pipeline undergoing compression as a result of the effective axial force concept (Fyrileiv and Collberg 2005). Assuming a constant temperature and ignoring bottom tension effects, the internal pressure exerts a compressive force on the pipe. The resultant force over a whole cross-section is calculated by examining the forces operating on that cross-section, as shown in the following equations (Massa *et al.* 2009):

$$F = \pi(2\nu - 1)pR_{ex}^2 \quad (5)$$

where ‘ p ’ is the internal pressure that change due to the blood flow of physical exercise. Then the virtual external energy (δW) due to the internal pressure calculated as follows:

$$\delta W = \iint \frac{\partial w}{\partial x} F \delta \left(\frac{\partial w}{\partial x} \right) dv \quad (6)$$

Then, the virtual Kinetic energy (δK) are assumed as follows:

$$\begin{aligned} \delta K = & \frac{1}{2} \iiint \rho \delta(\bar{K}) dV \\ \bar{K} = & \frac{(R_{ex} - R_i)^2}{\pi^2} \sin^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x \partial t} \right)^2 + z^2 \left(\frac{\partial^2 w}{\partial x \partial t} \right)^2 \\ & + \frac{(R_{ex} - R_i)^2}{\pi^2} \sin^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial \phi}{\partial t} \right)^2 - 2z \left(\frac{\partial u}{\partial t} \right) \left(\frac{\partial^2 w}{\partial x \partial t} \right) \\ & - \frac{2z(R_{ex} - R_i)}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x \partial t} \right) \left(\frac{\partial^2 w}{\partial x \partial t} + \frac{\partial \phi}{\partial t} \right) + \\ & \frac{2}{\pi} (R_{ex} - R_i) \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial u}{\partial t} \right) \left(\frac{\partial^2 w}{\partial x \partial t} + \frac{\partial \phi}{\partial t} \right) + \left(\frac{\partial w}{\partial t} \right)^2 \\ & + \frac{(R_{ex} - R_i)^2}{\pi^2} \sin^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial^2 w}{\partial x \partial t} \right) \left(\frac{\partial^2 w}{\partial x \partial t} + \frac{\partial \phi}{\partial t} \right) + \\ & \left(\frac{\partial u}{\partial t} \right)^2 + \frac{(R_{ex} - R_i)^2}{\pi^2} \sin^2 \left(\frac{\pi z}{R_{ex} - R_i} \right) \left(\frac{\partial \phi}{\partial t} \right) \left(\frac{\partial^2 w}{\partial x \partial t} + \frac{\partial \phi}{\partial t} \right) \end{aligned} \quad (7)$$

The virtual strain energy, virtual kinetic energy, and external work energy were identified as essential elements in the derivation process. Utilizing Hamilton's principle,

which necessitates the systematic integration of expressions for kinetic energy, elastic strain energy, and work done, the total potential energy of the system was determined. By employing the Euler-Lagrange principle to minimize the total potential energy, the final size-dependent governing equations were effectively derived. This methodology guarantees precise representation of the system's dynamic behavior while incorporating size-dependent effects characteristic of microscale structures.

$$\begin{aligned} & \delta(u), \\ & -\frac{\partial}{\partial x} \left(\theta_3 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) \right) - \theta_2 \frac{\partial^2 u}{\partial x^2} \\ & -\theta_4 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_1 \frac{\partial^3 w}{\partial x^3} = \Omega_{10} \frac{\partial^2 u}{\partial t^2} \end{aligned} \quad (8a)$$

$$\begin{aligned} & \delta(w), \\ & \theta_5 \frac{\partial^3 w}{\partial x^3} - \theta_{10} \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) - \frac{\partial}{\partial x} \left(F \frac{\partial w}{\partial x} \right) - \theta_{14} \frac{\partial^4 w}{\partial x^4} \\ & + \theta_3 \frac{\partial^3 u}{\partial x^3} + \theta_{12} \left(\frac{\partial^3 \phi}{\partial x^3} + \frac{\partial^4 w}{\partial x^4} \right) + \frac{3}{8} l^2 \theta_8 \left(\frac{\partial^3 \phi}{\partial x^3} + \frac{\partial^4 w}{\partial x^4} \right) \\ & - \theta_{14} \left(\frac{\partial^3 \phi}{\partial x^3} + \frac{\partial^4 w}{\partial x^4} \right) - \frac{1}{2} \theta_8 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \frac{1}{2} l^2 \theta_{16} \frac{\partial^4 w}{\partial x^4} \\ & - \frac{1}{4} l^2 \theta_8 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \frac{1}{2} l^2 \theta_9 \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) - \theta_4 \frac{\partial^2 u}{\partial x^2} \\ & + l^2 \theta_{15} \frac{\partial^4 w}{\partial x^4} + \theta_{10} \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) + \frac{1}{4} l^2 \theta_{11} \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) \\ & - \frac{1}{4} l^2 \theta_7 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \frac{1}{2} l^2 \theta_{16} \left(\frac{\partial^3 \phi}{\partial x^3} + \frac{\partial^4 w}{\partial x^4} \right) - \theta_1 \frac{\partial^3 u}{\partial x^3} \\ & - \frac{1}{4} l^2 \theta_{11} \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) - \theta_6 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_{13} \frac{\partial^4 w}{\partial x^4} \\ & + \frac{1}{2} l^2 \theta_9 \frac{\partial^3 w}{\partial x^3} - \theta_5 \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) \\ & = \Omega_{10} \frac{\partial^2 w}{\partial t^2} + \Omega_{20} \frac{\partial^4 w}{\partial x^2 \partial t^2} + \Omega_{30} \frac{\partial^3 \phi}{\partial x \partial t^2} \end{aligned} \quad (8b)$$

$$\begin{aligned} & \delta(\phi), \\ & \frac{1}{2} l^2 \theta_{14} \frac{\partial^3 w}{\partial x^3} + \frac{1}{4} l^2 \theta_{11} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_6 \left(\phi + \frac{\partial w}{\partial x} \right) \\ & + \frac{1}{2} \theta_8 \frac{\partial w}{\partial x} + \theta_{10} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \frac{1}{4} l^2 \theta_8 \left(\phi + \frac{\partial w}{\partial x} \right) \\ & - \theta_3 \frac{\partial^2 u}{\partial x^2} + \theta_4 \frac{\partial u}{\partial x} + \theta_{14} \frac{\partial^3 w}{\partial x^3} + \frac{1}{4} l^2 \theta_7 \left(\phi + \frac{\partial w}{\partial x} \right) \\ & - \theta_{12} \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) - \theta_{10} \frac{\partial^2 w}{\partial x^2} - \frac{1}{4} l^2 \theta_8 \left(\frac{\partial^2 \phi}{\partial x^2} + \frac{\partial^3 w}{\partial x^3} \right) \\ & - \frac{1}{2} l^2 \theta_9 \frac{\partial^2 w}{\partial x^2} - \frac{1}{4} l^2 \theta_{11} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \theta_5 \frac{\partial^2 w}{\partial x^2} \\ & = \Omega_{30} \frac{\partial^3 w}{\partial x \partial t^2} + \Omega_{40} \frac{\partial^2 \phi}{\partial t^2} \end{aligned} \quad (8c)$$

The boundary conditions are generated as follows:

$$\delta(u), \\ \theta_4 \left(\phi + \frac{\partial w}{\partial x} \right) - \theta_1 \left(\frac{\partial^2 w}{\partial x^2} \right) + \theta_3 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_2 \left(\frac{\partial u}{\partial x} \right) = 0 \quad (9a)$$

$$\delta(w), \\ \frac{1}{4} l^2 \theta_{11} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_{10} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + F \frac{\partial w}{\partial x} \\ + l^2 \theta_{16} \left(\frac{1}{2} \frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \frac{1}{4} l^2 \theta_8 \left(\phi + \frac{\partial w}{\partial x} \right) - \theta_3 \frac{\partial u}{\partial x} \quad (9b)$$

$$\begin{aligned}
& \theta_6 \left(\phi + \frac{\partial w}{\partial x} \right) + \theta_4 \frac{\partial u}{\partial x} + \frac{1}{4} l^2 \theta_7 \left(\phi + \frac{\partial w}{\partial x} \right) + \theta_1 \frac{\partial u}{\partial x} \\
& + \theta_{14} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \frac{1}{2} \theta_8 \left(\phi + \frac{\partial w}{\partial x} \right) - l^2 \theta_{15} \left(\frac{\partial^2 w}{\partial x^2} \right) \\
& - \frac{3}{8} l^2 \theta_8 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \theta_{10} \left(\phi + \frac{\partial w}{\partial x} \right) - \theta_5 \left(\frac{\partial^2 w}{\partial x^2} \right) \\
& - \theta_{12} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \frac{1}{4} l^2 \theta_{11} \left(\phi + \frac{\partial w}{\partial x} \right) - \theta_{13} \frac{\partial^2 w}{\partial x^2} \\
& + \theta_5 \left(\phi + \frac{\partial w}{\partial x} \right) + \frac{1}{2} l^2 \theta_9 \left(\phi + \frac{\partial w}{\partial x} \right) + \theta_{14} \frac{\partial^2 w}{\partial x^2} \\
& - \frac{1}{2} l^2 \theta_9 \left(\frac{\partial^2 w}{\partial x^2} \right) = 0
\end{aligned}$$

$$\begin{aligned}
& \delta(w_{,x}) \\
& \theta_{12} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_3 \frac{\partial u}{\partial x} + \frac{3}{8} l^2 \theta_8 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) \\
& + \frac{1}{4} l^2 \theta_{11} \left(\phi + \frac{\partial w}{\partial x} \right) + \theta_{10} \left(\phi + \frac{\partial w}{\partial x} \right) + l^2 \theta_{15} \frac{\partial^2 w}{\partial x^2} \\
& - \frac{1}{2} l^2 \theta_{16} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \theta_{14} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) - \theta_1 \frac{\partial u}{\partial x} \quad (9c) \\
& - \frac{1}{2} l^2 \theta_9 \left(\phi + \frac{\partial w}{\partial x} \right) - \frac{1}{2} l^2 \theta_{16} \frac{\partial^2 w}{\partial x^2} - \theta_{14} \left(\frac{\partial^2 w}{\partial x^2} \right) \\
& + \theta_{13} \left(\frac{\partial^2 w}{\partial x^2} \right) - \theta_5 \left(\phi + \frac{\partial w}{\partial x} \right) = 0
\end{aligned}$$

$$\begin{aligned}
& \delta(\phi) \\
& + \frac{1}{4} l^2 \theta_{11} \left(\phi + \frac{\partial w}{\partial x} \right) - \theta_{14} \frac{\partial^2 w}{\partial x^2} + \frac{1}{4} l^2 \theta_8 \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \quad (9d) \\
& \theta_{12} \left(\frac{\partial \phi}{\partial x} + \frac{\partial^2 w}{\partial x^2} \right) + \theta_3 \frac{\partial u}{\partial x} - \frac{1}{2} l^2 \theta_{16} \frac{\partial^2 w}{\partial x^2} + \theta_{10} \frac{\partial w}{\partial x} = 0
\end{aligned}$$

where

$$\begin{aligned}
\begin{Bmatrix} \theta_2 \\ \theta_3 \\ \theta_8 \\ \theta_{12} \\ \theta_{13} \\ \theta_{14} \\ \theta_{15} \\ \theta_{16} \end{Bmatrix} &= \iint \left\{ \begin{array}{l} E \\ E \frac{(R_{ex} - R_i)}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \\ G \left(\frac{\partial}{\partial z} \left(\frac{R_{ex} - R_i}{\pi} \sin \frac{\pi z}{R_{ex} - R_i} \right) \right)^2 \\ E \left(\frac{R_{ex} - R_i}{\pi} \sin \frac{\pi z}{R_{ex} - R_i} \right)^2 \\ E z^2 \\ E z \left(\frac{R_{ex} - R_i}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \right) \\ G \\ G \frac{\partial}{\partial z} \left(\frac{R_{ex} - R_i}{\pi} \sin \left(\frac{\pi z}{R_{ex} - R_i} \right) \right) \end{array} \right\} dA \quad (10a)
\end{aligned}$$

$$\begin{aligned}
\begin{Bmatrix} \theta_1 \\ \theta_4 \\ \theta_5 \\ \theta_6 \\ \theta_7 \\ \theta_9 \\ \theta_{10} \\ \theta_{11} \end{Bmatrix} &= \iint \left\{ \begin{array}{l} E z \frac{\partial \varpi}{\partial x} \\ E \frac{\partial \varpi}{\partial x} \\ E z \frac{\partial \varpi}{\partial x} \\ E \left(\frac{\partial \varpi}{\partial x} \right)^2 \\ G \left(\frac{\partial^2 \varpi}{\partial x \partial z} \right)^2 \\ G \frac{\partial^2 \varpi}{\partial x \partial z} \\ E \varpi \frac{\partial \varpi}{\partial x} \\ G \frac{\partial \varpi}{\partial x} \frac{\partial^2 \varpi}{\partial x \partial z} \end{array} \right\} dA = 0 \quad (10b)
\end{aligned}$$

$$\begin{Bmatrix} \Omega_{10} \\ \Omega_{20} \\ \Omega_{30} \\ \Omega_{40} \end{Bmatrix} = \iint \left\{ \begin{array}{l} \rho \\ \rho(\varpi - z)^2 \\ \rho \varpi(\varpi - z) \\ \rho \varpi^2 \end{array} \right\} dA \quad (10c)$$

3. Numerical analysis and advanced modeling techniques

The Finite Element approach (FEM), a powerful computational approach often used in mechanical engineering and biomechanics to solve complex systems involving partial differential equations, is used to numerically solve the governing equations created in the preceding section. By discretizing the continuous domain of microvascular structures into manageable finite elements, FEM provides a powerful foundation for simulating these structures' dynamic responses to internal pressures caused by exercise. This approach best serves the fundamental aims of this research, which are to understand the hemodynamic behavior of blood vessels during physical activity and to link these findings to sports physiology and vascular health management (Jin *et al.* 2024, Ma *et al.* 2024, Wang *et al.* 2024a, b, e, g, Xue *et al.* 2024, Zhang *et al.* 2024b).

The Finite Element Method is selected as the primary numerical technique because it effectively manages nonlinearities, complex geometries, and boundary conditions present in biological systems like blood vessels. This study models microvascular structures as microtubular systems influenced by internal pressure variations due to physical exertion. The governing equations, which combine traditional beam and tube theories with the modified couple stress theory, are solved using FEM to capture macroscopic deformations as well as size-dependent effects at the microscale. The blood vessel domain has been discretized into a finite element mesh, with each element representing a short microtube segment. The displacement field in each element is determined using shape functions that interpolate nodal displacements across the domain. The weak form of the governing equations, obtained from Hamilton's principle and the Euler-Lagrange equations, is utilized to ascertain stress and strain distributions under different internal pressures. The governing equations are integrated into the FEM framework to model the biomechanical reactions of blood vessels during physical exercise.

The finite element method (FEM) is used to generate the numerical solution of the governing equations formulated from high-order tube theory along with modified couple stress theory. The solution process starts with the discretization of the computational domain into finite elements, whereby the displacement field is approximated using appropriate shape functions that adhere to the continuity conditions of the issue. Utilizing the Galerkin weighted residual approach on the governing equations, a weak formulation is obtained by multiplying each equation by test functions and integrating throughout the domain. Integration by parts reduces the order of differentiation, hence assuring the continuation of the necessary field variables. The resultant system of equations, expressed in

Table 2 Comparison of Natural Frequencies with Published Results for Validation

Boundary Conditions	(Xu <i>et al.</i> 2021)	(Wang <i>et al.</i> 2007)	Present
Fundamental Frequency			
Clamped-Simply Supported	3.926599	3.9266	3.88733279
Fully Simply Supported	3.14159	3.1416	3.18871408
Fully Clamped	4.730036	4.73	4.77733656
Second frequency			
Clamped-Simply Supported	7.068559	7.0686	7.139286
Fully Simply Supported	6.283166	6.2832	6.220368
Fully Clamped	7.853176	7.8532	7.970998

terms of nodal displacements, is then converted into matrix form, producing the finite element equations in standard format:

$$[K] \begin{Bmatrix} u \\ w \\ \phi \end{Bmatrix} - [M] \frac{\partial^2}{\partial t^2} \begin{Bmatrix} u \\ w \\ \phi \end{Bmatrix} = 0 \quad (11)$$

' M ' is the global mass matrix, ' K ' is the global stiffness matrix with size-dependent effects from the modified couple stress theory, ' $\{u, \phi, w\}$ ' are the nodal displacement vector. The global stiffness and mass matrices are derived by aggregating the contributions of each component throughout the whole domain. The resultant system of equations is then resolved using a suitable numerical method. In free vibration analysis, the eigenvalue issue is expressed as

$$\det[[K] - \omega^2[M]] = 0 \quad (12)$$

where ' ω ' is the natural frequency. The transient analysis is conducted by integrating the system of equations over time with the Newmark-beta method, an implicit time-stepping technique that guarantees numerical stability. The displacement and velocity fields are consistently revised at every time step according to the used integration parameters. Essential and natural boundary conditions are implemented by either altering the system matrix or using Lagrange multipliers. This numerical method accurately accounts for the effects of coupling stress and internal pressure on the vessel's vibrational response, allowing a precise and computationally efficient investigation of microstructural size-dependent phenomena.

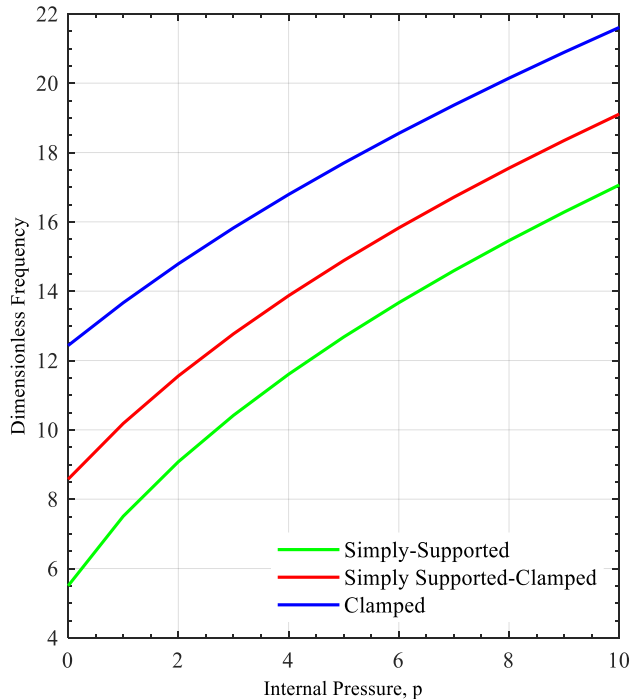
This work use modal analysis to combine sports physiology and biomechanical engineering, yielding new insights into the dynamic reactions of microvascular systems during physical effort. The model investigates the intrinsic frequencies and mode shapes of blood arteries under exercise-induced internal pressures, demonstrating how they respond to mechanical stress during various types of physical activity, including aerobic and resistance training. The numerical simulations demonstrate how variations in internal pressure affect the vibrational modes of blood vessels, offering a mechanistic insight into

vascular remodeling during exercise. These findings are essential for enhancing training protocols and formulating specific rehabilitation techniques for athletes and people recuperating from cardiovascular incidents. Determining resonant frequencies enables doctors to evaluate the risk of vascular injury under intense loading circumstances, hence providing safer and more effective workout regimes.

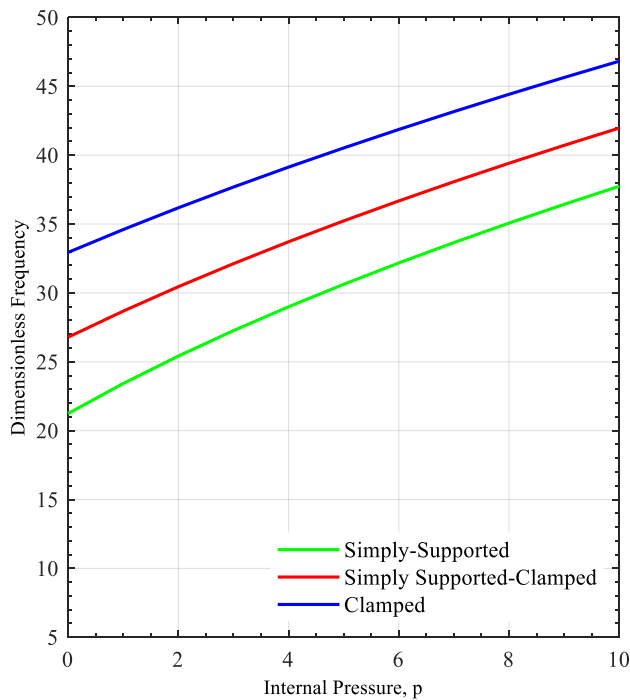
4. Numerical results and discussion

The combination of sports physiology and bio-mechanical engineering offers a unique perspective on how microvascular systems respond to exercise-induced stress. This section presents the numerical results obtained from modal analysis and finite element modeling, which clarify how physical activity influences the vibrational properties, stress distributions, and deformation patterns of blood vessels. These findings connect theoretical principles with practical applications and provide valuable insights for optimizing athletic performance and enhancing vascular health. To verify the precision and dependability of the governing equations and the solution methodology employed in this study, Table 2 has been listed. Comparative investigation of the results of Xu *et al.* (2021) and Wang *et al.* (2007) helps one ascertain the correctness and dependability of the governing equations and the technique of solution used in this work. Both works (current research and research published by Xu *et al.* (2021)) used modified couple stress theory with beam theory to analyze the vibrational response of microtubes. The natural frequencies of the structures under various boundary conditions and material properties were analyzed. The comparison demonstrates excellent agreement, with deviations in natural frequencies confined to 1–2%. This validation verifies the precision of the governing equations and the efficacy of the finite element framework in modeling the dynamic behavior of microvascular structures. The results provide a robust basis for the forthcoming presentation and interpretation of numerical data about hemodynamic responses triggered by exercise.

Physical exercise significantly influences internal blood vessel pressure, primarily via lowering peripheral vascular resistance and modifying neurohormonal activity (Hegde and Solomon 2015). Regular physical activity has long-term effects, including improved endothelial function and decreased systemic vascular resistance, eventually lowering resting blood pressure. The heart increases blood flow to meet the increased oxygen demand during exercise, so producing a temporary rise in internal pressure (Song *et al.* 2022). The drop in internal pressure implies improved vascular resilience and efficiency since physical exercise promotes structural remodeling and capillary proliferation, especially in those with hypertension (De Ciuceis *et al.* 2023). Furthermore, physical exercise improves blood circulation by increasing cardiac output and enhancing arterial flexibility, which leads to better tissue perfusion (Saladini 2024). These modifications support vascular health, resulting in sustained reductions in systolic blood pressure and improved microcirculatory function (Green



A: Fundamental Frequency



B: Second Frequency

Fig. 3 Frequency response of microtubes under varying internal pressure for different boundary conditions

and Smith 2018). The variations in internal pressure observed during or after exercise highlight the complex relationship between immediate hemodynamic responses and long-term vascular adaptations, emphasizing the importance of physical activity for maintaining cardiovascular health.

The numerical findings are shown by three main figures, which depict the dynamic behavior of microvascular

structures under various internal pressures and boundary conditions. This research provides critical insights into the vibrational properties, size-dependent effects, and geometric implications on the frequency response of blood arteries modelled as microtubes.

Fig. 3 depicts the dimensionless frequency ($\omega \frac{L^2}{\mu R_{ex}^2} \sqrt{\rho A/EI}$) of blood vessels (microtubes) in proportion to internal pressure under several boundary conditions, including simply-supported, clamped, and clamped-simply supported configurations. The data show a similar trend for both fundamental and second frequencies: an increase in internal pressure corresponds to a rise in frequency. The observed phenomenon is the consequence of internal pressure stiffening vessel walls, improving their resistance to deformation.

The size of the frequency response is strongly affected by the sort of boundary condition used. Among the configurations investigated, the clamped border condition predicts the highest frequency values, while the simply-supported condition produces the lowest. This difference arises when clamped borders place additional limitations on the system, restricting its degrees of freedom and hence raising its natural frequencies. Simply supported circumstances, on the other hand, allow for more flexibility, resulting in lower frequencies. The results emphasize the importance of boundary constraints in modeling the dynamic behavior of microvascular structures, which influence the vibrational features of blood vessels during physical activity.

Fig. 4 investigates the effect of the size-dependent parameter (l), as defined by the modified couple stress theory, on the frequency characteristics of microtubes under various internal pressures. The results show that both internal pressure and the size-dependent parameter play an important role in increasing the frequency of the first and second modes of vibration. This finding emphasizes the need to include small-scale effects in the study of microvascular systems, since these effects become more evident at the microscale. The size-dependent parameter explains the effect of material length scales on mechanical behavior, including phenomena such as increased stiffness and resistance to deformation. As internal pressure increases, the interaction of mechanical stress and size-dependent stiffening causes a considerable increase in frequency. This finding is consistent with prior studies emphasizing the need of taking into account size-dependent effects in microscale systems, particularly when predicting the biomechanical responses of blood vessels to exercise-induced stresses. By including the modified pair stress theory into the model, this work provides a more detailed portrayal of the dynamic behavior of microvascular structures.

Fig. 5 analyzes how the radius ratio (R_{ex}/R_i) affects the frequency characteristics of microtubes under various internal pressures and boundary conditions. The data show that raising the radius ratio significantly reduces the natural frequencies of the microtube, with this behavior becoming more noticeable at higher internal pressures. This inverse relationship between ' R_{ex}/R_i ' and frequency suggests that thicker vessel walls (larger R_{ex}/R_i) diminish the vibrational

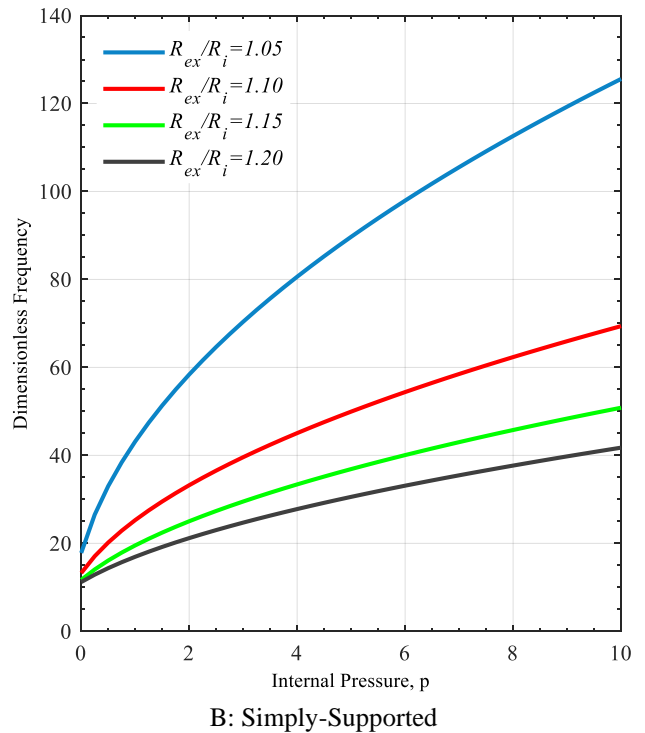
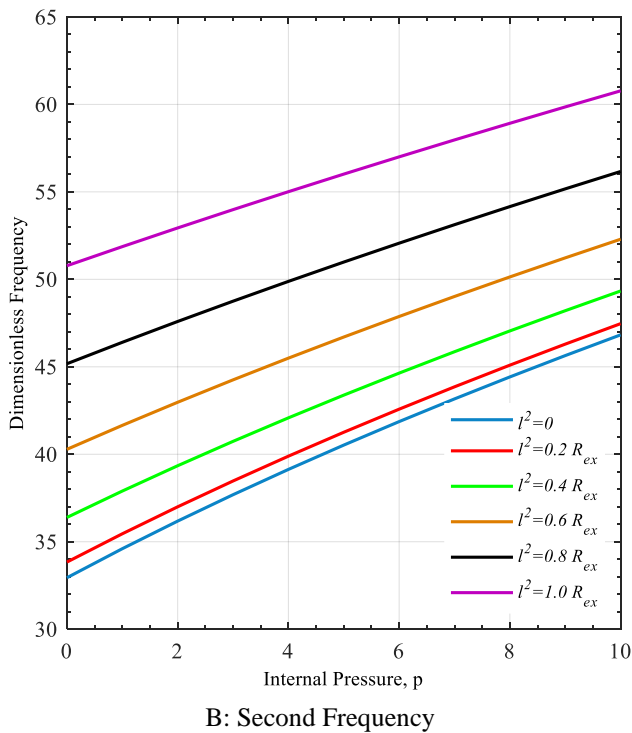
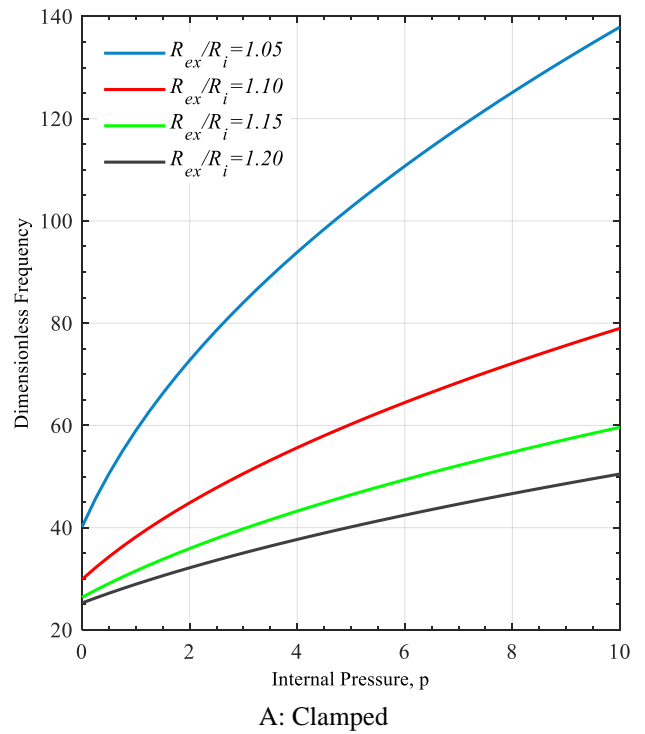
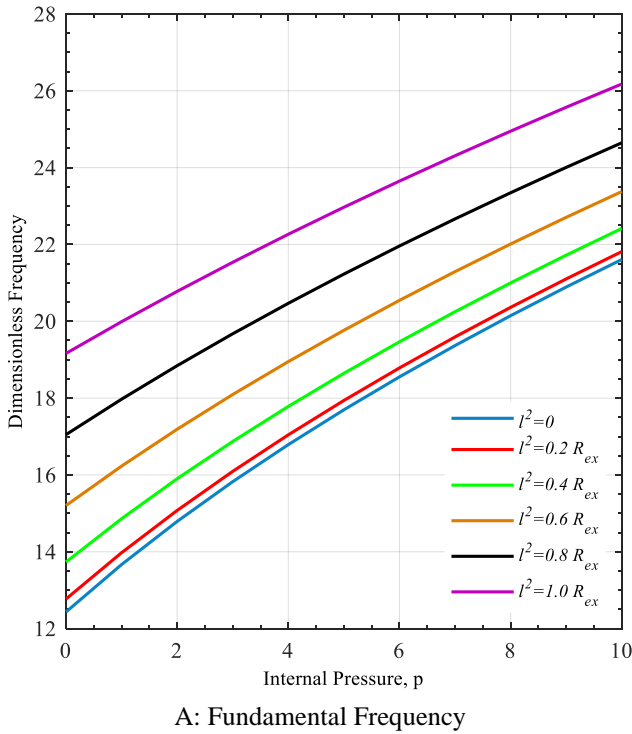


Fig. 4 Impact of the size-dependent parameter on the frequency response of a clamped microtube under varying internal pressures for the first two natural frequencies

Fig. 5 Influence of radius ratio on frequency response under varying boundary conditions and pressures, $l^2=0.5R_{ex}$

stiffness of the structure, thereby lowering its dynamic response. Consistent with prior observations, boundary conditions continue to play a dominant role in determining frequency magnitudes. Clamped configurations predict higher frequencies compared to simply-supported conditions, reflecting the restrictive influence of boundary constraints on vibrational freedom. These findings highlight the combined importance of geometric parameters (e.g., wall

thickness) and boundary conditions in governing the dynamic behavior of microvascular structures under exercise-induced mechanical loading.

During physical exercise, the body's metabolic requirement rises, causing the heart to pump more blood. This is accomplished by increased heart rate and greater contractions. Simultaneously, vasodilation (widening of blood vessels) in active muscles lowers vascular resistance, enabling more blood to flow to working tissues. Conversely,

vasoconstriction occurs in less active areas, enabling more effective resource distribution. Physical activity increases blood flow by enhancing cardiac output and strategically adjusting vascular tone. The relationship between cardiac output and peripheral vascular resistance determines blood pressure.

Increased blood flow during exercise raises systolic blood pressure owing to increased cardiac output despite lower resistance from vasodilation. The pressure wave produced by heart contractions (pulse pressure) travels through the arteries, with the stiffness and elasticity of artery walls determining how pressure is transferred. Higher flow rates may also cause turbulent flow, particularly in constricted or irregular pipes, which affects pressure dynamics.

The vibrational frequency of blood arteries is intimately related to their mechanical properties and the forces acting on them. Important factors to consider are:

- **Elasticity and Stiffness:** Vessels with greater stiffness, such as those caused by hypertension or aging, have higher resonance frequencies. The increased pressure causes the vessel walls to flex, influencing their stiffness and, hence, vibrational properties.

- **Pulsatile Flow:** Constant contraction of the heart generates pressure waves that vibrate blood vessels. Higher pressure might intensify these pulsations, influencing their vibrational frequencies.

- **Turbulence and Oscillations:** Turbulent blood flow, caused by elevated velocities or vascular constriction, may result in vortices and mechanical vibrations. These effects materialize as frequency variations, as shown by noises discernible with a stethoscope.

- **Acoustic Resonance:** Blood vessels may be seen as fluid-filled channels whose pressure changes produce resonant frequencies similar to those of acoustic pipes. Elevated pressure may cause these frequencies to shift owing to changes in vessel tension and shape.

Physical exercise increases blood flow via cardiac output and vasodilation, which raises blood pressure. This pressure affects vessel wall mechanics, with greater pressures possibly increasing vibrational frequencies due to elasticity changes, pulsatile forces, and turbulent flow. While the precise link between pressure and frequency needs additional investigation, clinical observations (for example, arterial stiffness assessments) show their dependency.

This section presents numerical results illustrating the complex relationship between sports physiology and biomechanical engineering, concentrating on how microvascular structures respond to exercise-generated forces. By utilizing modal analysis and finite element modeling, the research thoroughly characterizes natural frequencies, stress distributions, and deformation patterns across varying internal pressures. These insights enhance our comprehension of hemodynamic behavior during exercise and lay the groundwork for novel strategies in sports medicine and vascular health management. The upcoming sections will explore particular case studies and discuss the wider implications of these findings for clinical practices and improving athletic performance.

5. Conclusions

This work employed computational analysis to assess the response of microvascular structures to exercise-induced stressors, focusing on their hemodynamic reactions during physical activity. A comprehensive mathematical framework was created to outline the vibrational characteristics, stress distributions, and deformation patterns of blood vessels, modeled as microtubes, by integrating modified couple stress theory with advanced beam and tube theories.

The results indicated that the intrinsic frequencies of microvascular structures were significantly influenced by internal pressure, with increased pressures leading to elevated frequencies. The significant role of boundary constraints in vascular dynamics was underscored by the observation that simply-supported topologies yielded the lowest frequencies, whereas clamped boundary conditions consistently forecasted the highest frequencies. The importance of microscale events in accurately modeling hemodynamic responses was highlighted by including size-dependent effects using the modified pair stress theory. Moreover, it was shown that the radius ratio decreases frequency response, particularly at elevated internal pressures, highlighting the interplay between geometry and mechanical stress.

The findings elucidate the mechanisms by which physical activity influences vascular resilience via exercise-induced shear stress and pressure fluctuations. This study integrates biomechanical engineering principles with sports physiology to create a predictive framework for understanding vascular adaptations in response to athletic training and pathological conditions. The research emphasizes the benefits of exercise for endothelial function, systemic vascular resistance, and cardiovascular health.

This multidisciplinary approach combines theoretical and clinical components, providing critical resources for improving training programs, developing personalized rehabilitation plans, and improving noninvasive diagnostic procedures in vascular healthcare. Future study might apply these results to patient-specific models, looking at the role of microvascular dynamics in disease prevention and individualized therapy.

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