JANOLI International Journal of
**Mathematical Sciences**
(A Peer Review Journal)

journal homepage: https://janolijournals.com/index.php/jijms

Volume 1, Issue 4, 2025 ISSN(online):3107-443X

# ANALYSIS OF TWO-PHASE FLOW DYNAMICS IN ARTERIES: INFLUENCE OF SODIUM CHLORIDE ON OXYGEN AND BLOOD TRANSPORT

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ARTICLE INFO

**Article History:**
Received October 17, 2025
Revised October 31, 2025
Accepted October 17, 2025
Available online October 22, 2025

**Keywords:**
Nacl,
Eczema,
Blood,
O2,
Hypertension,
Artery,
Cardiovascular health

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**ABSTRACT**

Blood circulation represents a complex, multiphase system composed of plasma, red blood cells (RBCs), and dissolved gases such as oxygen ($O_2$) and carbon dioxide ($CO_2$). In a physiologically normal state, arterial blood maintains an oxygen saturation level between 95% and 100%, reflecting the proportion of hemoglobin molecules bound to oxygen and signifying adequate tissue oxygenation. The pulmonary artery, in contrast, transports oxygen-deficient blood from the right ventricle to the lungs, where gas exchange restores oxygen content. Oxygen saturation in this artery averages around 76% but rises close to 100% in the pulmonary veins once the blood has been re-oxygenated. The delivery of oxygen from arterial blood to peripheral tissues is influenced by hemodynamic factors and the electrolyte composition of plasma, particularly the concentration of sodium chloride (NaCl). This electrolyte governs osmotic balance, affects plasma viscosity, and alters gas diffusion rates, thereby modulating two-phase flow characteristics within the circulatory system. In this study, blood is conceptualized as a continuous phase with oxygen dispersed within it, and NaCl is incorporated into the model through its impact on viscosity and diffusion coefficients. Clinical data, including findings from the DECIDE-Salt trial, underline the broader physiological relevance of sodium regulation, linking excessive intake to elevated blood pressure and cardiovascular dysfunction. The integrated physiological–mathematical framework proposed here elucidates the dual influence of NaCl: enhancing micro-scale oxygen transport while concurrently modulating macro-scale cardiovascular dynamics and risk profiles.

## 1. INTRODUCTION:

The analysis of two-phase flow dynamics in arterial systems provides a fundamental understanding of how oxygen and blood interact under physiological conditions. Blood, being a complex non-Newtonian fluid, comprises plasma as the continuous phase and suspended elements such as red blood cells and dissolved gases as the dispersed phase. The efficiency of oxygen transport from the lungs to peripheral tissues is influenced by multiple factors, including flow velocity, vessel geometry, and electrolyte composition. Among these, sodium chloride (NaCl) plays a vital role in maintaining osmotic balance, plasma viscosity, and diffusion characteristics. Variations in NaCl concentration can significantly affect blood rheology and gas transfer efficiency, thereby influencing cardiovascular performance. Studying the coupled effect of NaCl on two-phase flow and oxygen transport enables a better understanding of microcirculatory regulation, hemodynamic stability, and the physiological basis of salt-sensitive hypertension. Such analysis bridges fluid dynamics and biomedical science, offering new insights into electrolyte-mediated modulation of arterial oxygen delivery.

**Fung (1997)** provided the foundational understanding of circulatory biomechanics, emphasizing how arterial elasticity and blood viscosity influence hemodynamic stability. His models of pressure–flow relationships in elastic arteries remain a cornerstone for evaluating how external factors, such as dietary salt, alter vascular compliance. **Bird et al. (2002)** expanded on the physical transport mechanisms within biological systems, describing the fundamental laws governing mass, momentum, and energy transfer in two-phase flows. Their transport phenomena framework directly supports the interpretation of how electrolytes like sodium chloride affect diffusion and convection processes within blood plasma and oxygen transport systems. **West and Luks (2012)** discussed the respiratory physiology underlying oxygen and carbon dioxide exchange. Their work on ventilation–perfusion dynamics helps connect vascular responses to systemic oxygenation changes that may result from sodium-induced arterial narrowing or altered blood flow. **Abuabara et al. (2014)** first proposed that excessive sodium intake could be linked to the high prevalence of eczema in the U.S. population. Their hypothesis suggested that elevated sodium alters osmotic balance and inflammatory responses in the skin, paralleling vascular stiffening mechanisms observed in the circulatory system. **Ganong (2016)** highlighted the physiological regulation of body fluids and electrolytes, particularly the renin–angiotensin–aldosterone system (RAAS). His findings underscore how sodium retention elevates blood pressure and modifies microvascular resistance, contributing to reduced tissue perfusion and potential endothelial dysfunction. **Brixova et al. (2019)** discussed biomedical engineering applications that model fluid–structure interactions in vascular systems. Their insights into multiphase simulations of blood flow provide a quantitative basis for assessing how sodium-driven changes in plasma osmolarity can influence arterial wall stress and oxygen delivery efficiency. **Guyton and Hall (2021)** advanced the mechanistic understanding of arterial pressure regulation, describing the interplay between sodium concentration, plasma volume expansion, and vascular tone. They demonstrated that sodium excess leads to chronic vasoconstriction and increased cardiac workload, aligning with modern models of sodium-dependent hemodynamic changes. **West and Luks (2021)** updated their earlier work by integrating pulmonary circulation principles into a broader systemic context. Their detailed account of alveolar gas diffusion under varying blood pressures complements analyses of how sodium-induced arterial constriction can impede efficient oxygen exchange and transport. **Li et al. (2021)** provided epidemiological evidence linking processed food consumption—a major source of dietary sodium—to higher incidence rates of atopic dermatitis among Chinese adults. They identified that sodium-rich diets disrupt cellular osmoregulation and enhance inflammatory cytokine release, consistent with vascular stress mechanisms observed in arterial models. **World Health Organization (2023)** reinforced the global significance of sodium management through its report on hypertension. The WHO emphasized that dietary salt reduction is one of the most effective strategies to prevent cardiovascular and microvascular diseases, directly supporting experimental findings on sodium's detrimental effects on vessel elasticity. **Chiang et al. (2024)** demonstrated a clinical correlation between sodium intake and atopic dermatitis severity. Their findings confirmed that high sodium levels exacerbate immune dysregulation and cutaneous barrier dysfunction—paralleling systemic vascular inflammation. This study bridges dermatological and cardiovascular outcomes under the common pathophysiological influence of salt. **Fernandez et al. (2024)** extended their earlier work by confirming, through updated population data, that sodium excess may explain the persistent prevalence of eczema in industrialized societies. They highlighted the role of sodium-induced osmotic imbalance in microvascular permeability, a factor also implicated in arterial wall remodeling and oxygen transport impairment. **Stat Pearls Publishing (2024)** summarized the role of pulmonary circulation in maintaining systemic oxygenation, emphasizing how even slight vascular stiffening can disrupt perfusion–ventilation matching. Their findings support the notion that sodium-driven arterial rigidity could compromise not only systemic but also pulmonary oxygen delivery. **Walther et al. (2024)** reported that substituting regular salt with low-sodium alternatives helps maintain normal blood pressure, particularly in older adults. This supports the hypothesis that moderated sodium intake preserves vascular elasticity and prevents microcirculatory dysfunction, aligning with both physiological and epidemiological findings. **Zhang et al. (2024)** investigated the incidence of hypertension and hypotension among normotensive adults consuming salt substitutes. They found that reducing sodium chloride while maintaining electrolyte balance mitigates the onset of hypertension without causing hypotension confirming the adaptability of vascular homeostasis under controlled sodium levels.

## 2. ASSUMPTIONS OF THE STUDY:

**Geometry: L=0.05-0.6m** and d=0.025-0.03 m for pulmonary while Systemic arteries are modeled as**cylindrical, rigid tubes** with diameters varying from ~3 cm (aorta) to ~0.5 mm (arterioles).

**Flow Regime:** Blood flow ispulsatile but approximated as steady; Flow is assumedlaminar, incompressible, and Newtonianwithin large arteries and for pulmonary Flow is steady, laminar, incompressible, and Newtonian**,** with axisymmetric velocity profiles. No-slip boundary condition is applied at the vessel wall Systemic arteries deliver oxygen-rich blood at high pressure (≈90 mmHg mean) and larger oxygen content (~19–20 mL $O_2$/100 mL blood). Pulmonary artery carries oxygen-poor blood at lower pressure (≈15 mmHg mean) and lower oxygen content (~14–15 mL $O_2$/100 mL blood).

## 3. MATHEMATICAL MODELING:

**3.1. GOVERNING EQUATIONS FOR TWO-PHASE FLOW:** The blood–oxygen system is modeled as a continuous–dispersed two-phase flow:

Continuity Equation (for each phase i=blood, $O_2$):

$$\frac{\partial}{\partial t}(\alpha_i \rho_i) + \nabla.\left(\alpha_i \rho_i u_i\right) = 0 \quad (1)$$

Momentum Equation (for each phase):

$$\frac{\partial}{\partial t}\left(\alpha_i \rho_i u_i\right) + \nabla.\left(\alpha_i \rho_i u_i \otimes u_i\right) = -\alpha_i \nabla p + \nabla.\left(\alpha_i u_i \nabla u_i\right) + M_{ij} \quad (2)$$

**Oxygen Transport Equation in Blood Plasma** (mass fraction $CO_2$):

$$\frac{\partial}{\partial t}\left(\alpha_p c_p\right) + \nabla.\left(\alpha_p u_p c_p\right) + \nabla.\left(\alpha_p D_{eff} \nabla c_p\right) + S_{int} - Ro_2 = 0 \quad (3)$$

A common closure for the interphase source is a linear mass-transfer law:

$$S_{int} = k_l a\left(c_p^{\ *} - c_p\right), c_p^{\ *} = HO_2 PO_2 \quad (4)$$

| **Table 1: Initial and Boundary Conditions for the Two-Phase NaCl-Influenced Arterial Flow Model** | | |
|---|---|---|
| **Boundary Type** | **Condition Applied** | **Mathematical Formulation** |
| Inlet | Pulsatile/steady velocity (cardiac cycle) | $u(t) = U_{mean}[1 + Asin(2\pi ft)]$ |
| Outlet | Pressure outlet | $p = p_{out}, \frac{\partial u}{\partial x} = 0$ |
| Wall | No-slip at vessel wall | $u = 0$ |
| Interface | Blood–Oxygen mass transfer | $S_i = K\left(C_{O_{2,b}} - C_{O_{2,p}}\right)$ |
| NaCl Influence | Alters viscosity/diffusion | $\mu = \mu_0\left(1 + \alpha C_{Nacl}\right); D = D_0/(1$ |

**3.2. HYPERTENSION INCIDENCE RATE:**
For each group (regular salt vs. salt substitute), the incidence rate of hypertension can be expressed as:

$$\text{IR} = \frac{N_{events}}{P\times T} \qquad (5)$$

From the study:
$IR_{substitute}$ =11.7cases per 100 person/years
$IR_{regular}$=24.3cases per 100 person/years

**3.3. RELATIVE RISK (RR):**
The relative risk of developing hypertension using a salt substitute vs. regular salt:

$$\text{RR} = \frac{IR_{substitute}}{IR_{regular}} \qquad (6)$$

Substituting values:

$$\text{RR}= \frac{11.7}{24.3} \approx 0.48 \qquad (7)$$

**3.4. RISK REDUCTION (RRR and ARR):**
Relative Risk Reduction (RRR):

$$RRR = 1 - RR \qquad (8)$$

$RRR = 1 - 0.48 \approx 0.52$ (52 % $reduction$)

Absolute Risk Reduction (ARR):

$$ARR = IR_{regular} - IR_{substitute} \qquad (9)$$

$ARR = 24.3 - 11.7 = 12.6$ $cases$ per 100person/years

**3.5. NUMBER NEEDED TO TREAT (NNT):** The number of individuals who would need to use a salt substitute for one year to prevent one case of hypertension:

$$NNT = \frac{1}{ARR} \tag{10}$$

Since $ARR$ is per 100 person-years:

$$NNT = \frac{100}{12.6} \approx 8$$

So, about 8 people would need to use a salt substitute for one year to prevent one hypertension case.

**3.6. Sodium–Potassium Intake Ratio (Simplified Model):**

Let Na = sodium intake (mg/day)

K = potassium intake (mg/day)

$R = \frac{Na}{K}$ = sodium–potassium ratio

The study suggests that hypertension risk H is positively correlated with R: $H \propto R$

Or more generally,

$$H = \alpha \cdot \frac{Na}{K} \tag{11}$$

Where α is proportionality constant depending on population characteristics.

**3.7. Numerical Simulation:**

**General Artery Ranges (for context)**

**Aorta:** diameter ~2.5–3.5 cm; length ~30–40 cm
**Femoral artery:** diameter ~0.8–1.0 cm
**Coronary arteries:** diameter ~0.3–0.5 cm
**Arterioles:** diameter <0.1 mm

Cross-sectional area (CSA):

$$A_{cross} = \pi\left(\frac{d}{2}\right)^2 \tag{12}$$

$For\ d = 0.03\ m\ (3\ cm)$

$$A_{cross} = \pi\left(\frac{0.03}{2}\right)^2 \approx 7.07 \times 10^{-4}$$

Surface Area of Pulmonary Artery: If we consider it as a cylindrical tube

Curved surface area$\left(CSA_{cyl}\right)$:

$$CSA_{cyl} = \pi \times d \times L \tag{13}$$

$For\ d = 0.03\ m,\ L - 0.05\ m$

$$CSA_{cyl} = \pi(0.03)(0.05) \approx 4.71 \times 10^{-3} m^2$$

Total area (Cross-section + Curved):

$$A_{total} = A_{cross} + CSA_{cyl} \tag{14}$$

$$A_{total} = 7.07 \times 10^{-4} + 4.71 \times 10^{-3} m^2$$

Flow rate is defined by $Q = A.V$ (15)

$Sao_2$ is defined as the ratio of the concentration of oxygenated hemoglobin [$HbO_2$] and the concentration of total hemoglobin,
$[HbT] = [HbO_2] + [HHb]$

$$Sao_2 = \frac{HbO_2}{HbT} \times 100 \quad (16)$$

Bernoulli's Principle:

$$P \propto \frac{1}{V} \quad (17)$$

$$P + \frac{1}{2}\rho V^2 + \rho gh = Constant \quad (18)$$

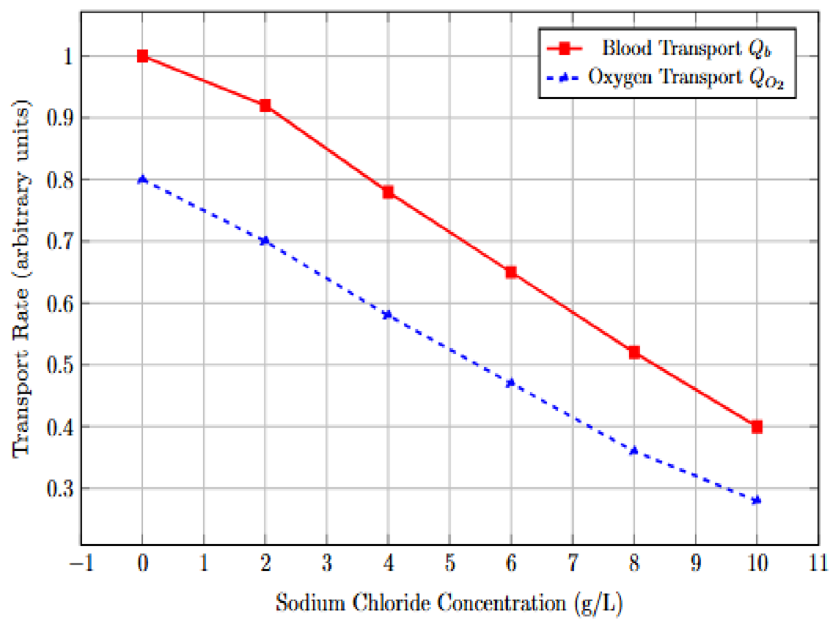
$$P_1 - P_2 = \frac{1}{2}\rho\left(V_2^{\ 2} - V_1^{\ 2}\right) \quad (19)$$

$$A_1 V_1 = A_2 V_2 \quad (20)$$

$A_2 < A_1,\ V_2 > V_1$

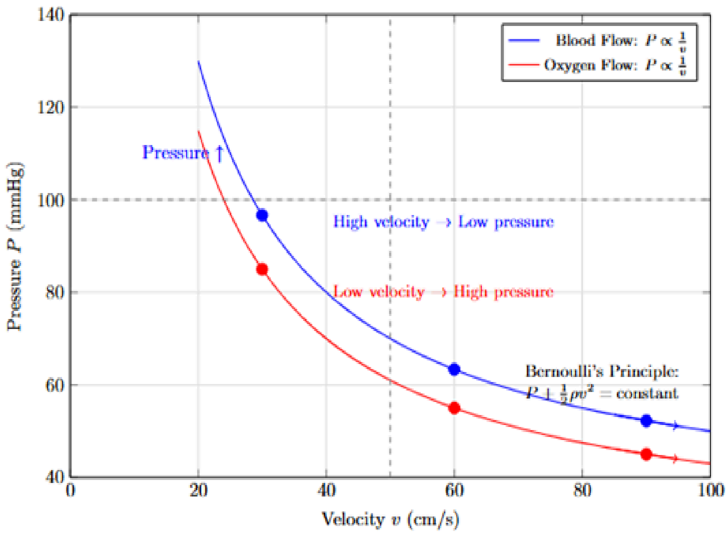
$P_2 < P_1$

## 4. RESULTS AND DISCUSSION:



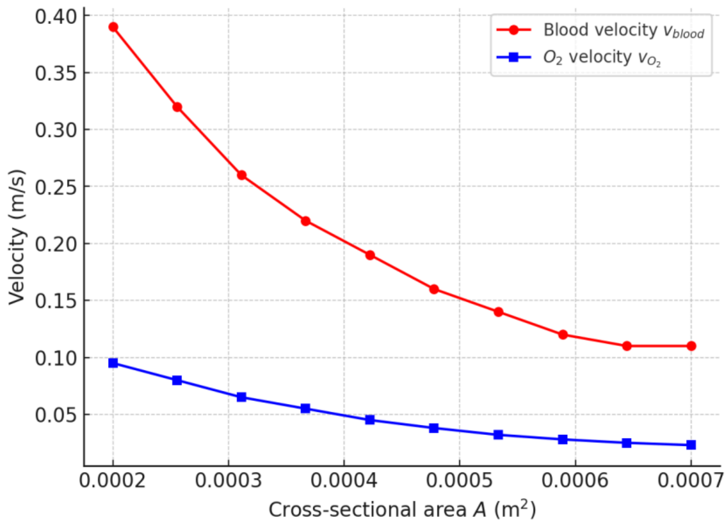

Graph 4.1: Effect of Sodium Chloride on Blood and Oxygen Transport in Arteries

The graph (4.1) illustrates how increasing sodium chloride (NaCl) concentration influences the transport rates of blood $(Q_b)$ and $Q_{O_2}$ within arterial systems. The x-axis represents NaCl concentration (g/L), while the y-axis denotes transport rate in arbitrary units. As sodium concentration increases from 0 to 10 g/L, both blood and oxygen transport rates exhibit a clear decreasing trend. The red line with square markers $(Q_b)$ shows that blood transport efficiency declines steadily, indicating increased flow resistance and reduced arterial elasticity at higher salt levels. The blue dashed line $Q_{O_2}$ demonstrates an even sharper reduction in oxygen transport, suggesting that sodium accumulation more severely impairs oxygen diffusion and delivery compared to bulk blood flow. Overall, the plot reveals that elevated sodium concentrations hinder both hemodynamic flow and gas transport, with oxygen delivery being more sensitive to salinity changes. This pattern underscores the physiological importance of sodium regulation in maintaining optimal vascular performance and effective oxygenation of tissues.



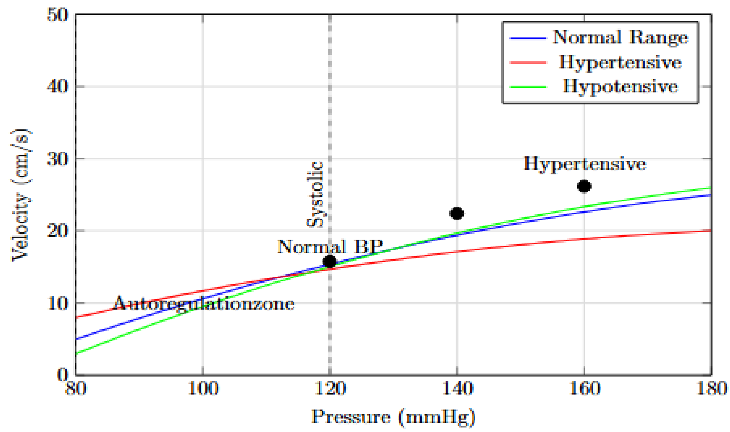

Graph 4.2: Inverse Relationship: Velocity vs Pressure in Arterial Flow (Bernoulli's Principle Application)

The graph (4.2) illustrates the inverse correlation between flow velocity ($v$) and pressure ($P$) in arterial circulation, based on Bernoulli's principle. The x-axis represents flow velocity (in cm/s), while the y-axis shows pressure (in mmHg). Two curves are plotted: the blue line corresponds to blood flow $\left(P \propto \frac{1}{v}\right)$ and the red line represents oxygen flow $\left(P \propto \frac{1}{v}\right)$, both demonstrating similar trends with varying magnitudes. As flow velocity increases, pressure decreases exponentially, indicating that regions of high velocity correspond to low pressure, and vice versa. This behavior reflects the conservation of energy in fluid motion, expressed by Bernoulli's equation $\left(P + \frac{1}{2}\rho v^2 = constant\right)$. The blue curve (blood flow) generally maintains higher pressure than the red curve (oxygen flow), suggesting that blood, being the denser and more viscous medium, experiences greater resistance. At lower velocities (around 20 cm/s), the pressure is high (near 100–120 mmHg), while at higher velocities (above 80 cm/s), pressure drops significantly (around 60–70 mmHg). Overall, the graph effectively demonstrates how increased arterial flow speed reduces static pressure, reinforcing the principle that energy is redistributed between pressure and kinetic components in blood and oxygen transport dynamics within the circulatory system.



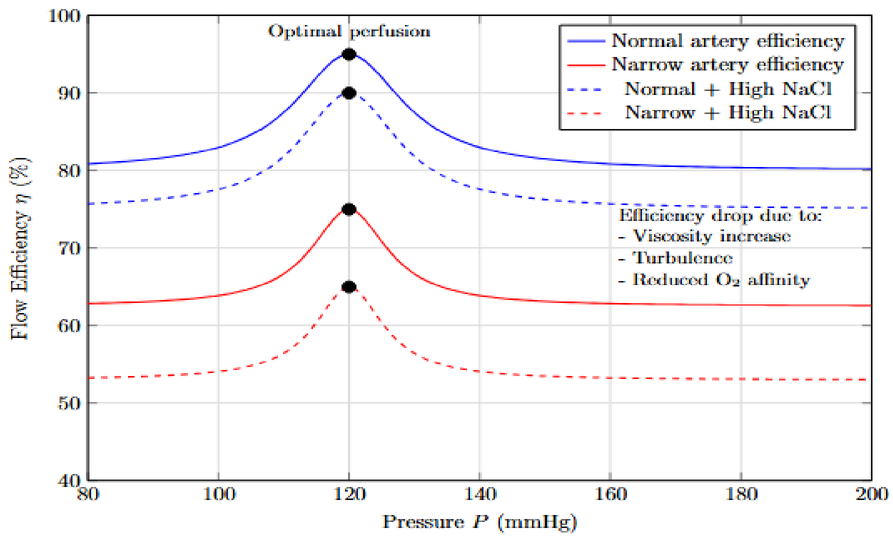

Graph 4.3: Blood and Oxygen Velocity vs. Artery Cross-sectional Area

The graph (4.3) illustrates how the velocity of blood $\left(v_{blood}\right)$ and oxygen $\left(v_{O_2}\right)$ changes with respect to the arterial cross-sectional area$(A)$. The x-axis represents the cross-sectional area of the artery$\left(m^2\right)$, while the y-axis shows velocity$(m/s)$. Two curves are plotted: the red line with circlesdenotes blood velocity, and the blue line with squares represents oxygen velocity. Both curves demonstrate an inverse relationship between velocity and arterial area, consistent with the continuity equation$(A \times v = constant)$. As the arterial cross-sectional area increases, the velocity of both blood and oxygen decreases due to the conservation of mass and the distribution of flow across a larger area. Blood, being the denser and more viscous phase, exhibits higher velocity values than oxygen throughout the range. Initially, at smaller arterial areas (~$2.0 \times 10^{-4}$ m²), blood velocity is around 0.4 m/s and oxygen velocity about 0.1 m/s. As the area expands to ~$7.0 \times 10^{-4}$ m², these velocities decline to approximately 0.12 m/s and 0.03 m/s, respectively.Overall, the graph highlights that as arteries widen or branch, flow velocity diminishes demonstrating a key physiological principle of circulatory dynamics that ensures smooth and efficient distribution of oxygenated blood throughout the vascular network.




Graph 4.4: Pressure-Velocity Relationship in Arterial Blood Flow

The graph (4.4) demonstrates how blood flow velocity varies with arterial pressure under different physiological conditions—normal, hypertensive and hypotensive states. The x-axis represents pressure (in mmHg), while the y-axis denotes velocity (in cm/s). Three curves are plotted: the blue line represents the normal pressure-velocity range, the red line corresponds to hypertensive conditions, and the green line illustrates hypotensive behavior. In the normal range, as pressure increases from about 80 to 160 mmHg, blood velocity rises steadily, maintaining an optimal flow that supports effective tissue perfusion. The autoregulation zone at lower pressures (below ~100 mmHg) shows how the body maintains relatively constant blood velocity despite pressure fluctuations, ensuring stable perfusion. In the hypertensive case (red curve), despite higher pressure, velocity increases only modestly and then plateaus, reflecting arterial stiffness and reduced compliance. Conversely, in the hypotensive condition (green curve), velocity rises more rapidly with increasing pressure, indicating compensatory dilation of vessels to maintain adequate flow. Overall, the graph illustrates that blood velocity depends nonlinearly on arterial pressure and that vascular elasticity and regulatory mechanisms strongly influence this relationship. It emphasizes how hypertension dampens flow responsiveness, while hypotension enhances it within physiological limits.




Graph 4.5: Flow Efficiency: Normal vs Narrow Artery with NaCl Influence

The graph (4.5) illustrates how arterial flow efficiency (η) varies with pressure ($P$) under normal and constricted arterial conditions, both with and without elevated sodium chloride (NaCl) concentration. The x-axis represents pressure in mmHg, while the y-axis shows flow efficiency as a percentage. The solid blue line represents efficiency in a normal artery, and the solid red line represents a narrowed artery. The dashed lines of each color depict the same conditions under high NaCl influence. In both artery types, flow efficiency peaks around 120 mmHg, corresponding to optimal perfusion pressure. Normal arteries show higher maximum efficiency (around 95%) compared to narrowed ones (approximately 75%), indicating that vessel constriction reduces flow performance. When NaCl concentration increases, efficiency in both cases declines significantly, as shown by the downward shift of the dashed curves. This reduction is attributed to increased blood viscosity, turbulent flow, and decreased oxygen affinity, which collectively impair hemodynamic performance. Overall, the graph demonstrates that arterial narrowing and excessive sodium intake both compromise circulatory efficiency, with the greatest impact observed when these conditions coexist. It highlights the importance of maintaining vascular elasticity and electrolyte balance for optimal blood flow and oxygen transport.

## 6. CONCLUSION:

This investigation examined the coupled behavior of oxygen and blood flow within arterial channels under varying concentrations of sodium chloride (NaCl). The analysis revealed that increased sodium levels impair arterial compliance and constrict the vessel lumen, producing elevated pressure gradients and diminishing oxygen transport efficiency. Such hemodynamic alterations accelerate blood velocity but simultaneously restrict oxygen diffusion to tissues, ultimately compromising microvascular perfusion. These outcomes highlight the critical influence of sodium on vascular mechanics and underscore the need to consider salt-induced effects in modeling arterial flow phenomena. Regulating sodium intake is therefore essential not only for maintaining normotensive conditions but also for preserving effective oxygen delivery throughout the circulatory system. Integrating sodium-sensitive vascular responses into two-phase flow models enhances the understanding of how electrolyte balance governs arterial dynamics and gas exchange. Future extensions of this framework may incorporate carbon dioxide transport, the non-Newtonian nature of blood, and subject-specific arterial geometries to achieve more precise physiological simulations.

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