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Analysis of Two-Phase Flow Dynamics in Arteries: Influence of Sodium Chloride on Oxygen and Blood Transport

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ABSTRACT

Blood circulation is a multiphase phenomenon involving plasma, red blood cells (RBCs), and dissolved gases such as oxygen and carbon dioxide. In a healthy individual, the oxygen saturation in the blood flowing through the arteries is typically between 95% and 100%. This is measured as a percentage of hemoglobin that is saturated with oxygen and indicates that the body is receiving sufficient oxygenated blood [1]. While the pulmonary artery carries oxygen-poor blood from the heart to the lungs, where gas exchange occurs and the blood picks up oxygen. In a healthy individual, the oxygen saturation in the pulmonary artery is around 76%, and this value increases to nearly 100% in the pulmonary veins after the blood has been oxygenated in the lungs [2]. The transport of oxygen from arteries to tissues depends on hemodynamic parameters and electrolyte concentration, particularly sodium chloride (NaCl). NaCl plays a crucial role in regulating osmotic pressure, plasma viscosity, and gas diffusion, thereby modifying two-phase flow dynamics. This study develops a two-phase flow framework in which blood is treated as the continuous phase and oxygen as the dispersed phase, incorporating NaCl effects into viscosity and diffusion coefficients. Clinical evidence from recent studies, including the DECIDE-Salt trial, highlights the systemic impact of sodium intake on hypertension and cardiovascular health [3]. The combined physiological and mathematical approach presented here provides insights into the dual role of NaCl in micro-scale oxygen transport and macro-scale cardiovascular risk.

1. Introduction

Blood circulation is a complex multiphase process that involves interactions between plasma, red blood cells (RBCs), and dissolved gases. The transport of oxygen from arteries to tissues depends not only on the hemodynamic parameters but also on electrolyte concentration in plasma. Sodium chloride, the predominant electrolyte, affects osmotic balance, viscosity, and diffusion rates of gases, thus influencing two-phase flow characteristics. This work models the interaction between oxygen (dispersed phase) and blood (continuous phase) as a two-phase flow in arterial channels, while incorporating the effect of NaCl concentration on viscosity and diffusion. A recent study published in the *Journal of the American College of Cardiology* suggests that replacing regular table salt with a salt substitute may help reduce the occurrence of hypertension (high blood pressure) in older adults without raising the risk of hypotension (low blood pressure). The findings showed that individuals who consumed salt substitutes had about a 40% lower chance of developing hypertension compared to those using standard salt [4]. The World Health Organization identifies hypertension as the primary risk factor for cardiovascular disease and premature death, affecting more than 1.4 billion people worldwide and accounting for 10.8 million deaths annually. Since excessive sodium intake is one of the leading contributors, reducing sodium is considered one of the most effective strategies to lower hypertension risk. Because excessive sodium intake is a major cause of hypertension, the WHO recommends lowering sodium consumption to less than 5 grams of salt (or 2 grams of sodium) per day as a cost-effective strategy to reduce blood pressure and cardiovascular risk. This research highlights that salt substitutes could be a more sustainable and effective approach to maintaining healthy blood pressure compared to simply cutting down on salt intake [5]. “Many adults unknowingly consume too much salt through inexpensive and readily available processed foods,” explained Dr. Yang Feng Wu, lead researcher and Executive Director of the Peking University Clinical Research Institute. “It is vital to raise awareness of healthier, lower-sodium alternatives and the impact dietary choices have on cardiovascular health.” The investigation, known as the DECIDE-Salt study, focused on elderly adults in Chinese care facilities. While earlier studies confirm that limiting salt reduces the risk of developing hypertension, sticking to such restrictions long-term remains difficult. To test alternatives, researchers enrolled 611 participants aged 55 or older from 48 facilities. Half of the facilities (313 participants) replaced their regular salt with a substitute, while the other half (298 participants) continued using normal salt. None of the participants had hypertension at the start of the study, and none were taking blood pressure medication. The main outcomes observed were the development of hypertension, the start of blood pressure medication, or major cardiovascular events during follow-up. After two years, the rate of hypertension was 11.7 cases per 100 person-years in the salt substitute group, compared to 24.3 cases in the regular salt group. This showed a 40% reduced risk of developing high blood pressure with the use of a substitute, without increasing the chances of hypotension—a common concern in older populations. “These results demonstrate an important step forward in managing blood pressure,” said Dr. Wu. “Salt substitutes provide a practical way to enhance health and reduce cardiovascular risk while still allowing people to enjoy flavorful meals. Given their blood pressure-lowering benefits, they can be valuable for both hypertensive and non-hypertensive individuals and represent a promising population-wide strategy to prevent hypertension and related diseases.” However, the study had limitations, including its post-hoc analysis design, the lack of pre-specified outcomes, and missing follow-up data for some participants. Despite these challenges, sensitivity analyses indicated the findings remain reliable. In an editorial commentary, Dr. Rik Olde Engberink of Amsterdam University Medical Center emphasized that salt substitutes offer a promising alternative to current global sodium-reduction strategies, which often fall short. He noted that in the

DECIDE-Salt trial, kitchen staff were directly provided with the substitute, and facilities limited outside food to once per week. This controlled approach may have amplified the impact. He suggested that for broader effectiveness, the food industry should adopt salt substitutes earlier in the supply chain to improve the sodium–potassium balance in processed foods.

Why Do 1 in 10 Americans Get Eczema? Is it Too Much Salt?



Sodium Intake and Risk of Eczema

Recent evidence suggests that excessive dietary sodium may contribute to the development and progression of eczema. Researchers at the University of California, San Francisco, reported that even a modest increase of one gram of sodium per day—comparable to the amount in a standard fast-food meal—was associated with a 22% higher probability of experiencing flare-ups [6]. Eczema, also referred to as atopic dermatitis, is a chronic inflammatory skin condition characterized by persistent dryness and itching. It is among the most common dermatological disorders, affecting more than 31 million individuals in the United States, with approximately one in ten developing the condition at some stage in life [7]. Its growing prevalence, particularly in industrialized nations, indicates that environmental and lifestyle factors, such as diet, may significantly influence disease onset and severity. Sodium, primarily consumed in the form of salt, is already well recognized for its role in hypertension and cardiovascular diseases [8]. More recent studies have shown that sodium accumulates in skin tissue, potentially driving the inflammatory mechanisms underlying eczema [9]. This finding suggests that reducing dietary sodium intake could represent a practical and accessible strategy for managing symptoms in affected patients. In a large-scale cross-sectional analysis involving over 215,000 individuals aged 30–70 years from the UK Biobank, urinary sodium excretion served as a biomarker of dietary intake, while electronic health records and prescription codes provided diagnostic and severity data for atopic dermatitis. The results revealed that each additional gram of sodium excreted over a 24-hour period was linked to an 11% increase in the likelihood of receiving an eczema diagnosis, a 16% increase in the risk of active disease, and an 11% increase in disease severity. These findings were further validated using data from approximately 13,000 U.S. adults in the National Health and Nutrition Examination Survey. Here, it was observed that an additional daily gram of sodium intake—equivalent to roughly half a teaspoon of table salt—was associated with a 22% higher likelihood of presenting with an active case of eczema [10]. Taken together, these results highlight a potential association between sodium intake and eczema activity, underscoring

the need for further investigation into dietary interventions as a means of disease prevention and management.

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 is pulsatile but approximated as steady; Flow is assumed laminar, incompressible, and Newtonian within 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 O₂/100 mL blood). Pulmonary artery carries oxygen-poor blood at lower pressure (≈ 15 mmHg mean) and lower oxygen content (~ 14 – 15 mL O₂/100 mL blood).

3. Mathematical Modeling

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, O₂):

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

Momentum Equation (for each phase):

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

Oxygen Transport Equation in Blood Plasma (mass fraction CO₂):

$$\frac{\partial}{\partial t}(\alpha_p c_p) + \nabla \cdot (\alpha_p u_p c_p) + \nabla \cdot (\alpha_p D_{eff} \nabla c_p) + S_{int-} R o_2 \quad (3)$$

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

$$S_{int} = k_L a (c_p^* - c_p), \quad c_p^* = H o_2 p o_2 \quad (4)$$

Hypertension Incidence Rate

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

$$IR = \frac{N_{events}}{P.T} \quad (5)$$

From the study:

IR_{substitute} = 11.7 cases per 100 person/years

IR_{regular} = 24.3 cases per 100 person/years

Relative Risk (RR)

The relative risk of developing hypertension using a salt substitute vs. regular salt:

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

Substituting values:

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

Risk Reduction (RRR and ARR)

Relative Risk Reduction (RRR):

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

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

Absolute Risk Reduction (ARR):

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

$$ARR = 24.3 - 11.7 = 12.6 \text{ cases per 100 person/years}$$

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:

$$NNT = \frac{1}{ARR} \quad (10)$$

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.

Sodium–Potassium Intake Ratio (Simplified Model) [11]-[17]

Let: Na = sodium intake (mg/day)

K = potassium intake (mg/day)

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

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

$$H \propto R$$

Or more generally,

$$H = \alpha \cdot \frac{Na}{K} \quad (11)$$

Where α is proportionality constant depending on population characteristics.

General Artery Ranges (for context)

Aorta: diameter $\sim 2.5\text{--}3.5$ cm; length $\sim 30\text{--}40$ cm

Femoral artery: diameter $\sim 0.8\text{--}1.0$ cm

Coronary arteries: diameter $\sim 0.3\text{--}0.5$ cm

Arterioles: diameter <0.1 mm

Cross-sectional area (CSA):

$$A = \pi \left(\frac{d}{2} \right)^2 \quad (12)$$

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

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

Surface Area of Pulmonary Artery

If you consider it as a cylindrical tube:

Curved surface area (CSA_cyl):

$$A_{cyl} = \pi dL \quad (13)$$

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

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

Total area (Cross-section + Curved):

$$A_{total} = A_{cross} + A_{cyl} \quad (14)$$

$$A_{total} \approx (7.07 \times 10^{-4}) + (4.71 \times 10^{-3}) \approx 5.42 \times 10^{-3} \text{ m}^2$$

To find flow rate

$$Q = A \cdot V \quad (15)$$

SaO_2 is defined as the ratio of the concentration of oxygenated hemoglobin $[HbO_2]$ and the concentration of total hemoglobin, $[HbT] = [HbO_2] + [HHb]$:

$$SaO_2 = \frac{HbO_2}{HbT} \times 100\% \quad (16)$$

The value of SaO_2 in health is the same throughout the whole arterial system. It is directly related to the oxygen supply to organs, and normal values lie between 95% and 100%.

Bernoulli's Principle:

Increase in fluid speed is associated with a decrease in pressure, and vice versa

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

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

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

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

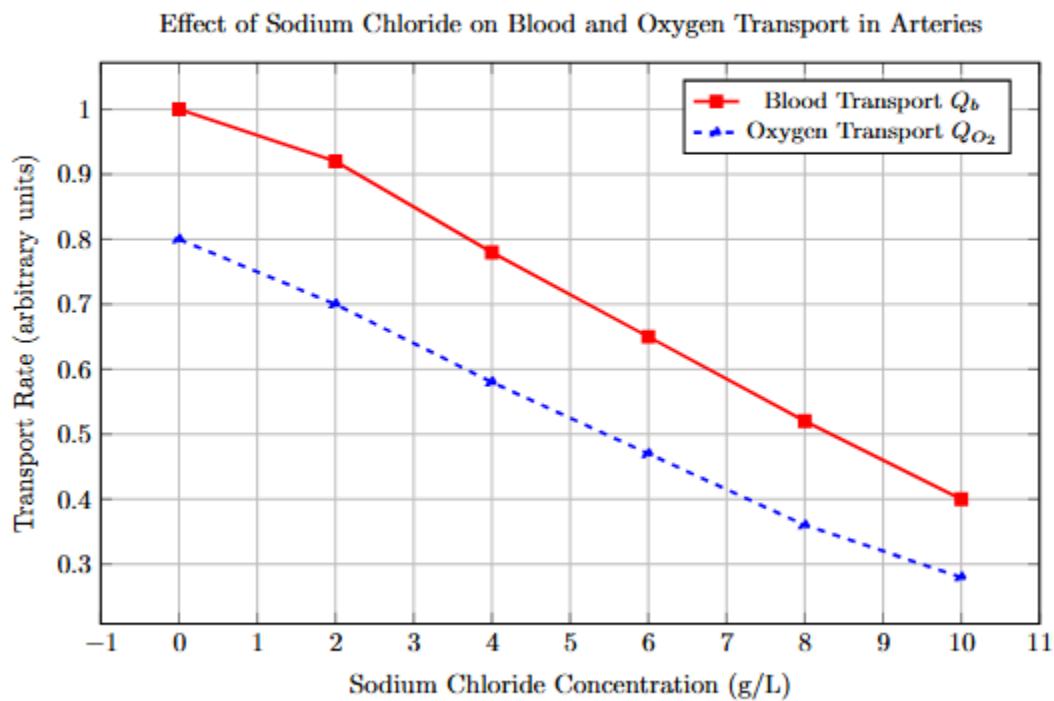
$$A_2 < A_1, V_2 > V_1$$

$$P_2 < P_1$$

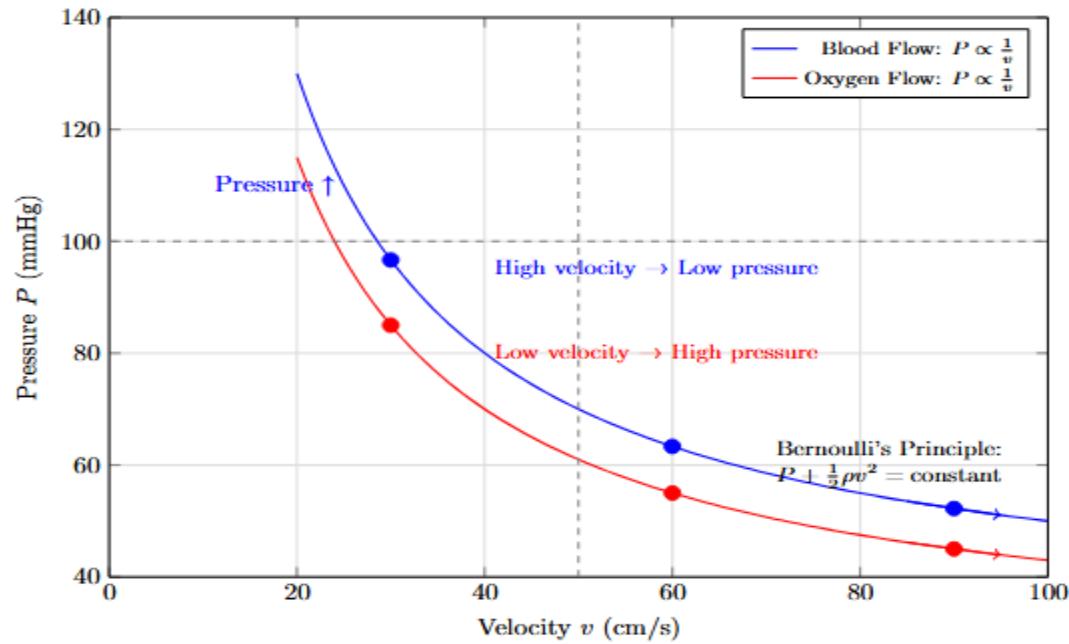
Decreasing area- Increasing velocity

Increasing velocity – decreasing pressure

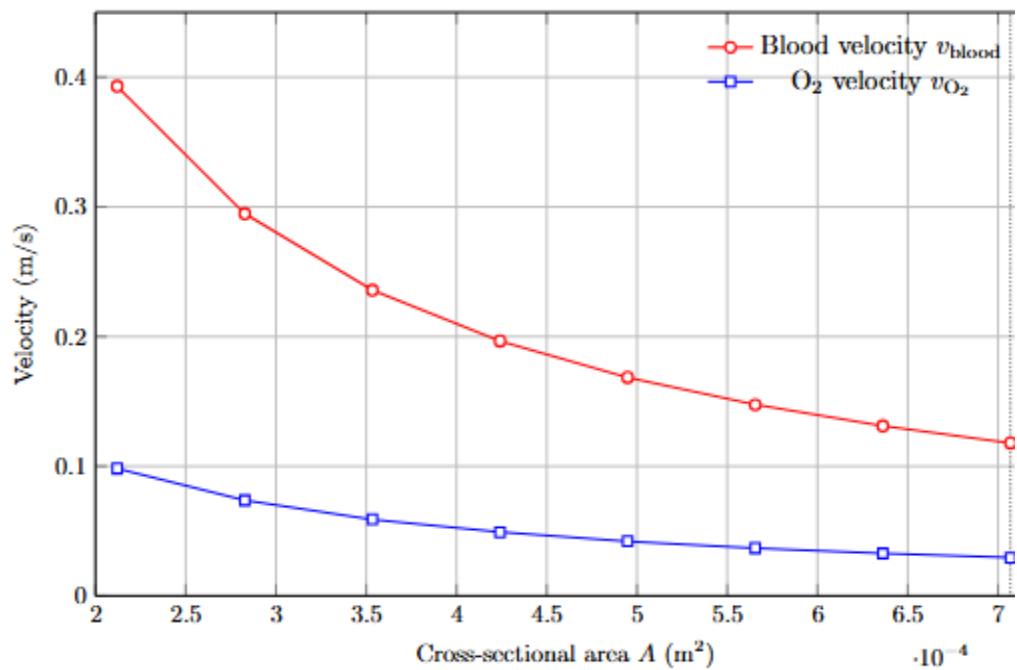
Results:

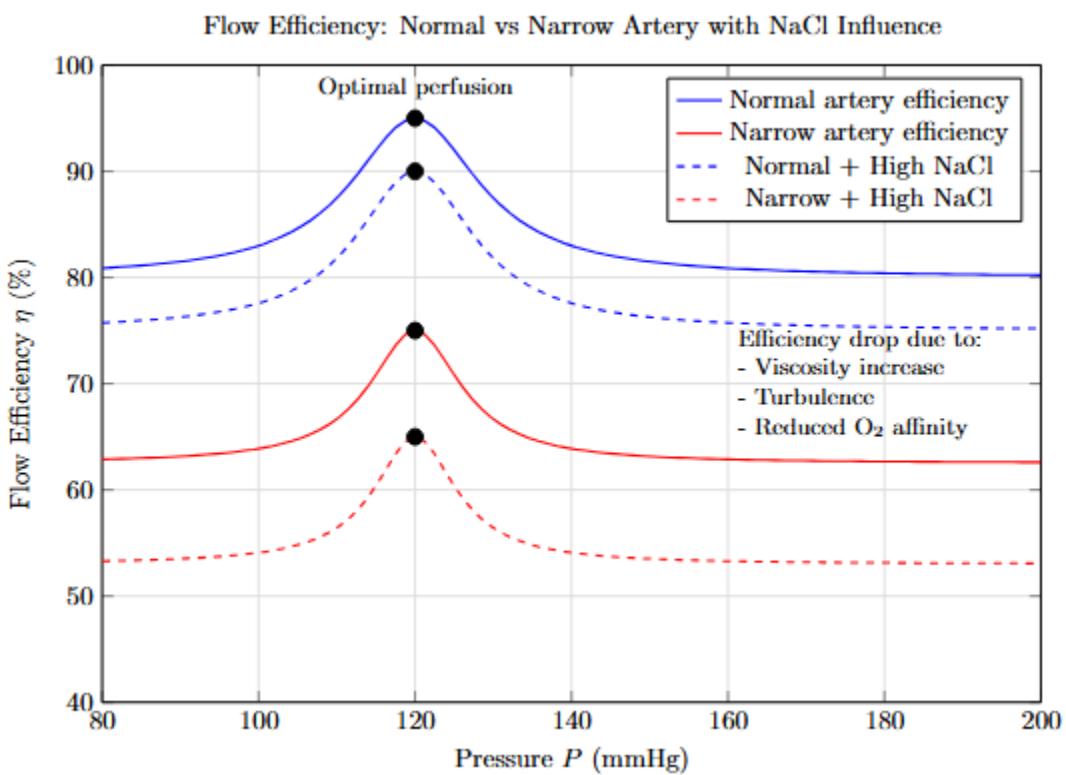
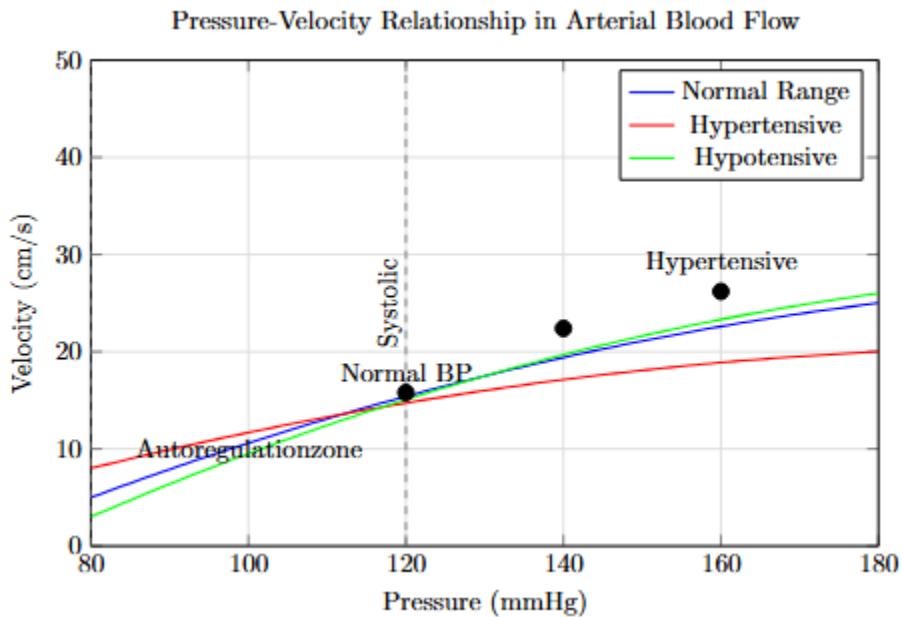


Inverse Relationship: Velocity vs Pressure in Arterial Flow(Bernoulli's Principle Application)



Blood and Oxygen Velocity vs. Artery Cross-sectional Area





Conclusion

This study explored the interaction of blood and oxygen flow in arteries under the effect of sodium chloride. The results indicate that elevated sodium intake decreases arterial elasticity and narrows the vessel lumen, leading to higher pressure differences and reduced efficiency of oxygen transport. These changes increase blood velocity while limiting oxygen delivery, which can compromise tissue perfusion. The findings emphasize the significance of sodium in vascular function and the importance of accounting for salt-related effects in arterial flow analysis. Managing sodium levels is therefore crucial not only for controlling blood pressure but also for ensuring adequate oxygen supply in circulation. Incorporating sodium-dependent vascular responses into multiphase flow models offers valuable insight into the relationship between dietary salt, arterial behavior, and gas exchange. Future work may expand this approach by including carbon dioxide dynamics, non-Newtonian blood properties, and patient-specific artery geometries for improved prediction accuracy.

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