

Computational Modeling of Cerebral Arterial Hemodynamics in Embolic Ischemic Stroke Under Varying Mean Arterial Pressure

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KEYWORDS

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ABSTRACT

Cerebral hemodynamics plays a pivotal role in determining ischemic injury severity following embolic stroke events. Variations in mean arterial pressure (MAP) can exacerbate ischemic vulnerability through altered perfusion and autoregulatory mechanisms. This study develops a computational framework to simulate cerebral arterial flow under varying MAP conditions using lumped-parameter models and three-dimensional hemodynamic analysis. We integrate patient-specific parameters to examine perfusion heterogeneity and the interplay between blood pressure variability and hemispheric ischemic susceptibility. Results demonstrate nonlinear interactions between MAP fluctuations and regional perfusion, highlighting critical thresholds beyond which autoregulatory mechanisms fail. The proposed modeling approach provides a predictive platform for individualized clinical interventions and stroke risk assessment.

INTRODUCTION

1 Problem Statement

Ischemic stroke is a leading cause of morbidity and mortality globally, often precipitated by embolic occlusion in major cerebral arteries (Elsheikh et al., 2024). The cerebral vasculature's ability to maintain perfusion in response to fluctuating mean arterial pressure (MAP) is critical in determining infarct extent and functional outcome (Bateman & Bateman, 2024). Traditional clinical assessment lacks the granularity to quantify regional perfusion changes, limiting individualized therapeutic

optimization.

1.2 Research Relevance

Recent advancements in computational modeling facilitate the examination of cerebrovascular dynamics at both systemic and microvascular scales (Seidenstein et al., 2015; Liu et al., 2016). Integrating patient-specific anatomical and physiological parameters enables predictive simulations of ischemic vulnerability, bridging a critical translational gap between hemodynamic

theory and clinical practice (Chen et al., 2024).

1.3 Objectives

This study aims to:

1. Develop a computational model of cerebral arterial hemodynamics under varying MAP conditions.
2. Quantify the impact of MAP fluctuations on regional cerebral perfusion and ischemic risk.
3. Investigate the influence of autoregulatory mechanisms on hemodynamic stability in embolic stroke scenarios.

1.4 Scope and Significance

The modeling framework encompasses both lumped-parameter simulations and three-dimensional hemodynamic analysis within the circle of Willis. It emphasizes clinical relevance by simulating patient-specific stroke events and quantifying blood pressure variability effects on perfusion heterogeneity (Dicker et al., 2006; Gilmore et al., 2006). Findings inform optimized blood pressure management strategies, potentially mitigating ischemic damage in acute stroke care.

LITERATURE REVIEW

Cerebral autoregulation maintains stable perfusion despite fluctuations in systemic blood pressure. Bateman and Bateman (2024) demonstrated through lumped-parameter models that autoregulatory failure can lead to regional ischemia even in the presence of apparently adequate systemic pressure. Complementing this, studies of blood pressure variability in acute ischemic stroke reveal hemispheric-dependent differences in perfusion response (Dicker et al., 2006), emphasizing the necessity for individualized hemodynamic assessment.

Elsheikh et al. (2024) and Dzeshka et al. (2017) underscore the intersection of atrial fibrillation, hypertension, and stroke pathophysiology,

highlighting how arrhythmogenic variability complicates cerebral perfusion. Clinical studies further demonstrate that increased ventricular rates during atrial fibrillation elevate the risk of hypoperfusion, suggesting a mechanistic basis for modeling hemodynamic instability (Saglietto et al., 2019).

From a computational perspective, lumped-parameter modeling offers a tractable approach to simulate systemic and cerebral vascular dynamics (Bateman & Bateman, 2024; Seidenstein et al., 2015). Three-dimensional hemodynamic models provide detailed insight into flow distribution within the patient-specific circle of Willis, revealing nonuniform perfusion and potential ischemic zones (Liu et al., 2016). These approaches have been integrated with numerical control strategies for heart rate variability and arterial stiffness to evaluate cerebrovascular response under stress conditions (Nakao et al., 2001; Kobayashi & Musha, 1982).

While these models have advanced understanding, critical gaps persist. Most studies either focus on systemic hemodynamics or isolated cerebral perfusion, lacking an integrated framework linking MAP fluctuations to spatial perfusion heterogeneity during embolic events. This study addresses this gap by combining lumped-parameter and three-dimensional modeling to simulate embolic ischemic stroke scenarios under varying MAP conditions (Bateman & Bateman, 2024; Sun et al., 2024).

METHODOLOGY

3.1 Computational Framework

The model integrates a lumped-parameter representation of the cerebral arterial network with patient-specific three-dimensional arterial geometry (Bateman & Bateman, 2024; Liu et al., 2016). Lumped-parameter elements—**resistors**, capacitors, and inductors—represent vascular resistance, compliance, and inertial effects, respectively, allowing rapid simulation of dynamic perfusion under variable MAP.

Mathematically, the system is governed by:

$$C \frac{dP}{dt} = Q_{in} - Q_{out} \quad P = C(Q_{in} - Q_{out})$$

where P is arterial pressure, Q_{in} and Q_{out} are inflow and outflow rates, and C is compliance. Autoregulatory mechanisms modulate R (resistance) to maintain target perfusion.

3.2 Model Parameterization

Patient-specific arterial geometry was extracted from MRI data (Józsa et al., 2023), including vessel diameters and lengths. Hemodynamic parameters, including baseline MAP, heart rate, and stroke volume, were calibrated using literature-derived physiological ranges (Biel et al., 2001; Moore et al., 2005). Blood pressure variability was modeled as stochastic fluctuations superimposed on baseline MAP, reflecting clinical observations (Dicker et al., 2006; Gilmore et al., 2006).

3.3 Simulation of Embolic Occlusion

Embolic events were simulated by introducing localized resistive elements to mimic arterial blockage. Flow redistribution was computed iteratively across the network to quantify regional perfusion deficits. Three-dimensional simulations of the circle of Willis enabled visualization of ischemic zones and collateral flow compensation (Liu et al., 2016; Sun et al., 2024).

3.4 Validation and Sensitivity Analysis

Model outputs were compared against documented hemodynamic responses in patients with atrial fibrillation and acute ischemic stroke (Elsheikh et al., 2024; Dzeshka et al., 2017). Global sensitivity analysis quantified the influence of MAP fluctuations, vascular resistance, and compliance parameters on regional perfusion (Iooss & Lemaître, 2015).

3.5 Analytical Metrics

Perfusion heterogeneity was assessed via coefficient of variation across arterial territories. Autoregulatory failure thresholds were identified as MAP deviations causing >20% reduction in cerebral blood flow relative to baseline (Bateman & Bateman, 2024).

RESULTS

4.1 Perfusion Patterns

Simulations indicate that moderate MAP reductions (10–20 mmHg) are largely compensated by autoregulatory mechanisms. However, MAP reductions exceeding 30 mmHg induce pronounced perfusion deficits in distal MCA and ACA territories, consistent with clinical observations of embolic stroke severity (Bateman & Bateman, 2024; Liu et al., 2016). Three-dimensional mapping illustrates collateral flow recruitment through the circle of Willis, which partially mitigates ischemic vulnerability.

4.2 Hemodynamic Variability

Introduction of stochastic MAP fluctuations reveals nonlinear amplification of regional perfusion heterogeneity. Hemispheric differences emerge, with the dominant hemisphere experiencing greater perfusion compromise, reflecting documented clinical patterns (Dicker et al., 2006; Gilmore et al., 2006).

4.3 Autoregulatory Limits

Autoregulatory mechanisms fail under combined embolic obstruction and extreme MAP deviations (>30 mmHg reduction). Sensitivity analysis confirms that vessel compliance and baseline resistance are critical determinants of ischemic thresholds (Bateman & Bateman, 2024).

4.4 Model Validation

Predicted perfusion deficits correspond closely to observed patterns in patients with acute embolic stroke and atrial fibrillation (Elsheikh et al., 2024; Saglietto et al., 2019). Cross-comparison with previous computational studies demonstrates the

model's predictive accuracy in both lumped-parameter and three-dimensional domains (Seidenstein et al., 2015; Sun et al., 2024).

DISCUSSION

The findings underscore the critical interplay between systemic MAP fluctuations and regional cerebral perfusion in embolic ischemic stroke. Computational modeling elucidates mechanisms underlying ischemic vulnerability that are difficult to capture in vivo.

Autoregulatory capacity is not uniform across the cerebral vasculature; distal territories are more susceptible to perfusion deficits, emphasizing the need for individualized hemodynamic monitoring (Bateman & Bateman, 2024). Variability in MAP, as observed in atrial fibrillation, further exacerbates ischemic risk, aligning with clinical observations linking arrhythmia to stroke severity (Dzeshka et al., 2017; Saglietto et al., 2019).

The integration of lumped-parameter and three-dimensional modeling offers a comprehensive platform for exploring intervention strategies, such as blood pressure optimization or targeted thrombolysis. Nevertheless, limitations include assumptions of rigid vessel walls in 3D simulations and simplified stochastic modeling of MAP fluctuations. Future refinements could incorporate patient-specific vessel compliance and autoregulatory feedback loops for enhanced predictive fidelity.

CONCLUSION

This study demonstrates that computational modeling of cerebral arterial hemodynamics under varying MAP provides critical insights into embolic ischemic stroke pathophysiology. Key findings include:

1. Nonlinear amplification of perfusion deficits under MAP fluctuations.
2. Differential autoregulatory capacity across

cerebral territories.

3. Validation of lumped-parameter and 3D modeling frameworks for patient-specific predictive simulations.

The approach supports individualized stroke risk assessment and optimization of therapeutic strategies, with future extensions toward real-time integration with clinical monitoring systems.

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