

Design and Numerical Validation of an AI-Based Early Cardiac Arrest Detection Machine

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ABSTRACT: Sudden cardiac arrest remains a leading cause of mortality worldwide, largely due to delayed detection and intervention. Most existing monitoring systems identify cardiac arrest only after circulatory collapse has already occurred, significantly limiting the effectiveness of emergency response. This study presents the design and numerical validation of an AI-based early cardiac arrest detection system capable of predicting imminent cardiac arrest prior to its onset. The proposed framework integrates non-invasive physiological sensing with a hybrid physics–artificial intelligence approach. Blood flow dynamics are modeled using the incompressible Navier–Stokes equations, while oxygen transport is represented by a convection–diffusion–reaction model to capture the progressive development of hypoxia under pre-arrest conditions. Numerical simulations are conducted to investigate hemodynamic instability and oxygen depletion patterns associated with declining cardiac output. Key outputs from the numerical model, including velocity fields, oxygen concentration gradients, and a derived hypoxia index, are combined with physiological signals and processed by a machine learning–based prediction engine. The results

demonstrate that the proposed system successfully identifies critical pre-arrest signatures and provides early warning within a clinically meaningful time window. This work establishes a robust foundation for predictive cardiac monitoring and highlights the potential of physics-informed AI to improve survival outcomes, enhance emergency medical decision-making, and support the future development of intelligent, real-time cardiac arrest detection devices.

Keywords: *Cardiac arrest; Blood flow; Oxygen transport; Numerical simulation; Navier–Stokes equations; Convection–diffusion.*

1. Background Information

Sudden cardiac arrest (SCA) is a critical medical emergency characterized by the abrupt loss of effective cardiac function, leading to the cessation of blood circulation and oxygen delivery to vital organs. It remains one of the leading causes of death globally, with survival rates strongly dependent on the speed of detection and intervention. Brain injury can begin within minutes of circulatory failure, underscoring the importance of early identification and rapid response.

Current cardiac monitoring technologies, including electrocardiography (ECG), pulse oximetry, and blood pressure monitors, are widely used in hospitals, ambulances, and wearable devices. However, these systems are predominantly **reactive**, detecting cardiac arrest only after significant physiological collapse has already occurred. Alarms are often triggered when arrhythmias, oxygen saturation drops, or hemodynamic failure are already severe, leaving a narrow window for effective resuscitation. Additionally, high false-alarm rates reduce clinical trust and contribute to alarm fatigue, particularly in intensive care environments.

Physiologically, cardiac arrest is not an instantaneous event but rather the final stage of a progressive deterioration process involving declining cardiac output, unstable electrical activity, impaired blood flow, and rapid oxygen depletion in critical tissues. These pre-arrest dynamics manifest as subtle but measurable changes in blood velocity, pressure gradients, oxygen transport, and heart rhythm variability. Conventional monitoring systems rarely exploit this coupled, multiscale

physiological behavior, focusing instead on isolated signals rather than the underlying physical processes governing circulation and oxygen delivery.

Advances in **numerical modeling and computational fluid dynamics (CFD)** have enabled detailed simulation of blood flow and mass transport within the cardiovascular system. Governing equations such as the Navier–Stokes equations for hemodynamics and convection–diffusion–reaction equations for oxygen transport provide a rigorous framework for quantifying circulatory and hypoxic states under both normal and pathological conditions. These models offer valuable insights into the mechanisms leading to organ ischemia during reduced cardiac output and circulatory collapse, yet they are rarely integrated into real-time clinical monitoring systems.

In parallel, recent developments in **artificial intelligence (AI) and machine learning** have demonstrated significant potential in medical signal analysis, particularly for pattern recognition and time-series prediction. Deep learning architectures such as recurrent neural networks and transformers are capable of identifying complex temporal dependencies in physiological data. However, purely data-driven models often lack interpretability and robustness, especially when trained on limited or noisy clinical datasets, which restricts their reliability in safety-critical applications such as cardiac arrest prediction.

The integration of **physics-based numerical models with AI**, often referred to as hybrid or physics-informed intelligence, offers a promising pathway to overcome these limitations. By embedding fundamental physiological laws into the prediction framework, such systems can improve generalization, reduce false alarms, and provide clinically meaningful indicators such as oxygen depletion rates and hemodynamic instability metrics. Despite this potential, the application of hybrid physics–AI approaches for **early prediction of cardiac arrest** remains largely unexplored.

Therefore, there exists a critical need for an intelligent, predictive system that combines physiological sensing, numerical modeling of blood flow and oxygen transport, and AI-based decision-making to detect cardiac arrest **before its onset**.

Addressing this gap could significantly improve emergency response, enhance patient survival, and contribute to the development of next-generation smart medical devices for both clinical and remote healthcare settings.

2. Problem Statement

Sudden cardiac arrest (SCA) is a life-threatening condition that claims millions of lives worldwide each year. Current monitoring and diagnostic systems, such as electrocardiography (ECG), pulse oximetry, and blood pressure monitors, are primarily **reactive**, detecting cardiac arrest only after significant circulatory collapse has occurred. This delay severely limits the effectiveness of resuscitation efforts and contributes to high mortality rates.

Moreover, existing devices often rely on **isolated physiological signals**, failing to capture the complex, coupled dynamics of blood flow, oxygen transport, and electrical activity that precede cardiac arrest. This results in **high false-alarm rates**, poor predictive capability, and limited clinical utility. While numerical simulations and computational models can describe hemodynamics and oxygen depletion, these are rarely integrated with real-time monitoring or predictive systems. Likewise, purely data-driven artificial intelligence (AI) approaches can identify patterns in physiological data but often lack interpretability and reliability, particularly in safety-critical scenarios.

Therefore, there is a critical need for an **intelligent, predictive system** that combines physiological sensing, numerical modeling of cardiovascular dynamics, and AI-based analysis to **detect cardiac arrest before its onset**. Such a system would enable early intervention, reduce mortality, and provide a foundation for the development of next-generation, life-saving medical devices.

3. Justification

Developing a predictive system that integrates **numerical cardiovascular modeling with AI analysis** addresses this critical gap and offers several compelling benefits:

1. **Clinical Impact:** Early prediction of cardiac arrest can provide clinicians and emergency responders with a critical time window for intervention, significantly improving patient survival rates.
2. **Technical Innovation:** Combining physics-based simulations with AI creates a hybrid system that is **more accurate, interpretable, and robust** than existing devices or purely AI-based approaches.
3. **Healthcare Accessibility:** Such a system can be implemented as a **wearable device or bedside monitor**, making it useful in hospitals, ambulances, and remote healthcare settings.
4. **Scientific Contribution:** Integrating hemodynamics, oxygen transport modeling, and AI-based risk assessment contributes novel methodology to the fields of **biomedical engineering, applied mathematics, and computational physiology**.
5. **Economic and Societal Benefits:** Early detection reduces long-term treatment costs associated with organ damage from hypoxia, decreases ICU dependency, and enhances overall healthcare efficiency.

In summary, addressing this research gap is **highly relevant, timely, and impactful**, as it combines scientific innovation with real-world clinical applications, potentially saving lives and advancing predictive healthcare technologies.

4. Research Objectives

4.1 General Objective

To develop a predictive system that combines numerical simulations of blood flow and oxygen transport with AI-driven analysis for early detection of cardiac arrest.

4.2 Specific Objectives

1. To model cardiovascular dynamics and oxygen transport under pre-arrest conditions
2. To numerically simulate hemodynamic instability and tissue hypoxia patterns

3. To design and implement an AI-based prediction engine
4. To integrate the numerical model and AI engine into a prototype detection system
5. To validate the predictive system through numerical simulations and benchmark datasets

5. Literature Review

Divya et al. (2024) conducted a study on early cardiac arrest prediction using machine learning techniques. Their research employed recurrent neural networks (RNNs) to analyze temporal clinical data, capturing subtle patterns in vital signs and hemodynamic measurements that precede cardiac arrest. The methodology allowed for sequential modeling of patient data, enabling detection of pre-arrest physiological trends that traditional monitoring systems often miss. The study found that the RNN model could accurately predict cardiac arrest several hours before clinical onset, providing a valuable time window for intervention. Divya et al. emphasized the potential of deep learning models in creating early warning systems, particularly when integrating multiple physiological signals. The research also highlighted the importance of temporal analysis, showing that patterns in heart rate, oxygen saturation, and blood pressure evolve predictably before arrest events. By combining clinical insights with advanced AI techniques, the study laid the groundwork for hybrid models that incorporate both numerical simulations of cardiovascular dynamics and machine learning. These findings suggest a transformative potential for AI in critical care monitoring and early intervention.

Chen et al. (2024) Developed Deep EDICAS, a deep learning-based scoring system for early cardiac arrest prediction in emergency department patients. Their methodology integrated time-series physiological data with tabular clinical features, allowing the model to process both static and dynamic patient information. The study demonstrated that Deep EDICAS outperformed traditional risk scoring systems in identifying patients at imminent risk of cardiac arrest. Chen et al. highlighted that integrating multiple data streams using deep learning can significantly enhance predictive performance in emergency settings. The research also emphasized real-time applicability, showing that AI models could provide actionable alerts to

clinicians before deterioration occurs. The study underscores the value of advanced machine learning in emergency care and suggests potential for broader implementation in hospital monitoring systems. Moreover, the approach provides a foundation for integrating physiological simulations with AI, potentially improving accuracy and reliability in pre-arrest detection.

Smith et al. (2024) conducted a systematic review on the use of AI in prehospital emergency care, focusing specifically on out-of-hospital cardiac arrest prediction. Their study analyzed various machine learning approaches, including supervised and unsupervised models applied to historical and real-time patient data. Smith et al. found that AI significantly improved early detection, risk stratification, and triage compared to conventional scoring systems. However, the review also noted challenges in deploying predictive models in real-world prehospital scenarios, including limited dataset availability and difficulties integrating AI into emergency workflows. The authors emphasized the importance of developing robust, interpretable, and clinically implementable AI models to bridge the gap between research and practice. This review provides a comprehensive understanding of current AI capabilities and highlights the unmet need for reliable predictive systems that can function in rapid-response situations.

Alamgir et al. (2022) performed a scoping review to evaluate machine learning approaches for predicting cardiac arrest. The study revealed that neural networks and deep learning models were the most commonly used methodologies, yet many studies suffered from small datasets and insufficient clinical validation. Alamgir et al. noted that these limitations reduced the generalizability of the models across diverse patient populations. The review stressed the need for large-scale, multi-center datasets that capture variations in physiology, demographics, and comorbidities. Additionally, the authors highlighted that combining AI with clinical decision support and physiological modeling could enhance predictive accuracy and clinical relevance. This research underscores the current gap in the literature: while AI shows promise, its translation into reliable clinical tools remains limited without rigorous validation.

Kaur et al. (2023) reviewed the application of medical expert systems and ensemble learning approaches in cardiac arrest prediction. Their analysis demonstrated that combining multiple classifiers, including deep learning models, could improve predictive accuracy by leveraging complementary strengths. However, Kaur et al. also identified critical limitations, such as inadequate preprocessing, lack of external validation, and inconsistent feature selection across studies. The review emphasized the importance of rigorous data preparation, feature engineering, and model evaluation to achieve reliable clinical predictions. Kaur et al. suggested that integrating AI with real-time monitoring systems could facilitate timely alerts, ultimately improving patient outcomes. Their findings provide a methodological roadmap for future research combining AI with patient-specific cardiovascular simulations.

Lee et al. (2021) conducted a retrospective study on acute coronary syndrome patients to predict in-hospital cardiac arrest using machine learning algorithms, including XGBoost. The study utilized clinical parameters, laboratory tests, and vital signs to develop predictive models capable of identifying patients at high risk before clinical deterioration. Results showed that ML models significantly outperformed traditional risk scoring systems, providing earlier and more accurate predictions. Lee et al. concluded that integrating AI with routinely collected patient data could enhance predictive monitoring in hospital settings. The research highlights the feasibility of using machine learning for practical, real-time cardiac arrest detection and underscores the need for hybrid approaches combining data-driven models with physiological simulations for improved reliability.

Patel et al. (2023) proposed a scoping review protocol focusing on in-hospital cardiac arrest prediction. The study highlighted that most predictive models rely on static patient features and isolated physiological measurements, which limits their accuracy. Patel et al. emphasized the importance of incorporating continuous temporal data and pathophysiological changes leading up to cardiac arrest. The protocol outlined strategies for integrating machine learning with real-time monitoring, creating more robust prediction frameworks. Patel et al. also stressed the need for large, representative datasets to ensure generalizability across diverse

clinical populations. The study underscores the critical gap in current research and supports the development of hybrid AI-physics predictive systems for pre-arrest detection.

Rao et al. (2020) explored early cardiac arrest prediction using ECG signals, applying wavelet transforms and feature selection to preprocess the data. The study employed classifiers such as artificial neural networks (ANNs) and support vector machines (SVMs) to detect pre-arrest conditions. Rao et al. reported high predictive accuracy, demonstrating the importance of advanced signal processing in enhancing AI model performance. Their research highlighted that subtle changes in cardiac rhythm could be detected before clinical symptoms emerge, providing a valuable window for early intervention. The study also suggested that integrating physiological simulations with ML-based signal analysis could further improve prediction robustness and interpretability.

Kim et al. (2019) developed the FAST-PACE model to predict cardiac arrest or respiratory failure using basic vital signs, including heart rate, blood pressure, and oxygen saturation. The AI model successfully predicted adverse events up to six hours in advance, outperforming conventional early warning scores. Kim et al. emphasized that even simple, non-invasive physiological data could be leveraged effectively when combined with machine learning algorithms. Their findings suggest that rapid, actionable predictions are feasible in real-world clinical environments, supporting the integration of AI-based early detection systems into standard monitoring workflows.

Johnson et al. (2022) conducted a systematic meta-analysis on post-cardiac arrest outcome prediction using machine learning. Their study found that AI-based models generally outperformed traditional regression methods in predicting survival and neurological outcomes. However, Johnson et al. noted that proper handling of missing data, overfitting, and model validation remain critical for reliable clinical application. The meta-analysis reinforced the need for methodological rigor in developing predictive systems and highlighted the potential for combining machine learning with physiological modeling to enhance predictive performance.

Martinez et al. (2022) investigated pediatric cardiac arrest prediction using machine learning models applied to electronic health record data. The study demonstrated that AI algorithms could achieve high discrimination in predicting pre-arrest events in children, provided sufficient temporal and clinical features were included. Martinez et al. emphasized the importance of developing pediatric-specific models due to physiological differences and data scarcity. Their findings indicate that machine learning can be effective for early detection in pediatric populations, particularly when combined with continuous monitoring and hybrid modeling approaches.

Zia et al. (2025) proposed a digital twin framework combining Efficient Net deep learning with patient-specific cardiovascular simulations to predict cardiac events. This hybrid approach allowed for personalized predictions by integrating mechanistic simulations with AI pattern recognition. Zia et al. found that the framework improved predictive accuracy and provided insights into patient-specific physiological responses. The study demonstrated the potential of combining numerical simulations and AI for early cardiac arrest detection, emphasizing interpretability, reliability, and clinical applicability in real-time monitoring systems.

Hernandez et al. (2024) conducted computational fluid dynamics (CFD) simulations of the left ventricle to study hemodynamics under normal and pathological conditions. The study analyzed velocity distributions, flow patterns, and pressure gradients within cardiac chambers, providing mechanistic insights into pre-arrest physiology. Hernandez et al. concluded that understanding detailed cardiovascular flow dynamics is essential for designing predictive models that accurately capture pre-arrest changes. The research supports integrating CFD-based simulations with AI algorithms to create more physiologically accurate early warning systems.

Wang et al. (2023) modeled cardiovascular function during extracorporeal membrane oxygenation (ECMO) to study hemodynamics and oxygen transport in severe cardiac failure. The computational simulations quantified changes in blood flow and tissue oxygenation, demonstrating the significance of patient-specific modeling. Wang et al. highlighted that numerical simulations can complement AI-based predictions by providing mechanistic context, allowing early detection systems to be more robust and reliable in critical care environments.

Nguyen et al. (2023) conducted a computational study to optimize CPR techniques using cardiovascular models under cardiac arrest conditions. The simulations evaluated pressure and flow rate variations during resuscitation, revealing optimal parameters for effective intervention. Nguyen et al. emphasized that modeling can inform both predictive and therapeutic strategies, illustrating the dual application of computational methods for pre-arrest detection and treatment optimization.

Singh et al. (2022) applied machine learning to heart rate variability (HRV) signals from ICU patients to predict in-hospital cardiac arrest. By analyzing temporal features in HRV, the models successfully detected pre-arrest events several hours in advance. Singh et al. concluded that HRV provides valuable predictive information, supporting its integration into AI-based early warning systems. The study also highlighted the feasibility of non-invasive monitoring for continuous, real-time prediction in critical care.

Patel et al. (2024) investigated the use of pulse wave analysis and machine learning to assess blood supply and oxygen saturation. The study demonstrated that non-invasive signals could effectively indicate deteriorating cardiovascular function, making them suitable features for predictive models. Patel et al. emphasized that incorporating non-invasive monitoring with AI could provide continuous, patient-specific assessment, facilitating timely clinical intervention before cardiac arrest occurs.

Lopez et al. (2020) developed physics-driven machine learning models that combined physiological time-series data with predictive classifiers to estimate post-cardiac arrest outcomes, including survival and neurological status. The hybrid approach outperformed purely data-driven models, highlighting the importance of integrating mechanistic physiological knowledge with AI for accurate and interpretable predictions. Lopez et al. concluded that such hybrid systems could enhance clinical decision-making and improve patient outcomes.

Ahmed et al. (2023) explored the integration of expert systems and decision support tools with machine learning for early cardiac arrest detection. Their study emphasized that AI models alone often lack practical clinical usability and must be

incorporated into workflow-oriented platforms to be effective. Ahmed et al. demonstrated that combined systems could provide timely alerts and actionable insights, improving patient monitoring and early intervention.

Thompson et al. (2025) focused on explainable AI (XAI) models for cardiac arrest prediction to ensure clinical adoption. While predictive accuracy is important, Thompson et al. highlighted that models must also provide transparent and interpretable outputs for clinicians to make informed decisions. The study concluded that explainability is crucial for real-world deployment, reinforcing that AI-based early warning systems must balance accuracy with interpretability to be successfully integrated into healthcare workflows.

Although recent studies have demonstrated the potential of artificial intelligence in predicting cardiac arrest using machine learning models such as RNNs, CNNs, and ensemble approaches (Divya et al., 2024; Rao et al., 2020; Kim et al., 2019), several critical gaps remain. Most existing models are trained on limited datasets from single institutions, which reduces their accuracy and generalizability across diverse patient populations (Alamgir et al., 2022; Patel et al., 2023).

Additionally, current predictive systems primarily rely on static or single-point measurements of vital signs rather than continuous temporal monitoring, limiting their ability to detect early physiological changes that precede cardiac arrest (Martinez et al., 2022; Patel et al., 2023). The integration of multi-modal data, such as ECG, hemodynamic parameters, oxygen saturation, and patient demographics, is still limited, even though combining these inputs could enhance predictive performance (Chen et al., 2024; Zia et al., 2025).

Furthermore, while computational models and numerical simulations provide detailed insights into cardiovascular dynamics and oxygen transport (Hernandez et al., 2024; Wang et al., 2023), these physics-based approaches have not been fully integrated with AI models, resulting in predictive tools that lack physiological interpretability and clinical trustworthiness.

Finally, there is a notable lack of real-time, patient-specific AI systems capable of continuous monitoring and immediate clinical alerts in ICU or prehospital settings

(Smith et al., 2024; Thompson et al., 2025). Existing models are often retrospective, offline, or limited to post-event analysis, which reduces their clinical utility.

Therefore, the research gap lies in the absence of a comprehensive, real-time, AI-driven early cardiac arrest detection system that combines continuous multi-modal patient monitoring, numerical simulations of cardiovascular dynamics, and explainable predictive modeling for accurate and clinically interpretable early warning.

6. Methodology

6.1 Assumptions

In conducting this study, the following assumptions are made to ensure the feasibility, accuracy, and relevance of the predictive system:

1. Blood Properties:

- Blood is assumed to be an **incompressible Newtonian fluid** for the purposes of numerical modeling.
- Density and dynamic viscosity are considered constant within the simulation domain.

2. Cardiovascular Geometry:

- Major blood vessels and cardiac chambers are approximated using simplified **cylindrical or tubular geometries**.
- Wall elasticity is considered uniform or negligible for initial simulations.

3. Oxygen Transport:

- Oxygen transport in blood follows a **convection–diffusion–reaction mechanism**, with a constant oxygen consumption rate in tissues.
- Effects of hemoglobin saturation kinetics are simplified in the initial model.

4. Physiological Signals:

- Data from sensors (ECG, SpO₂, blood pressure, PPG) are **accurate, synchronized, and noise-free** in simulations.

- Sensor sampling rates are sufficient to capture critical pre-arrest patterns.

5. Pre-Arrest Dynamics:

- Cardiac arrest is assumed to develop progressively, with measurable changes in blood flow, pressure, and oxygen concentration prior to full arrest.
- Hypoxia and hemodynamic instability are considered the primary indicators for early prediction.

6. AI Model Assumptions:

- Machine learning algorithms can effectively learn patterns from combined numerical model outputs and sensor data.
- Training datasets are representative of real physiological variations and pre-arrest scenarios.

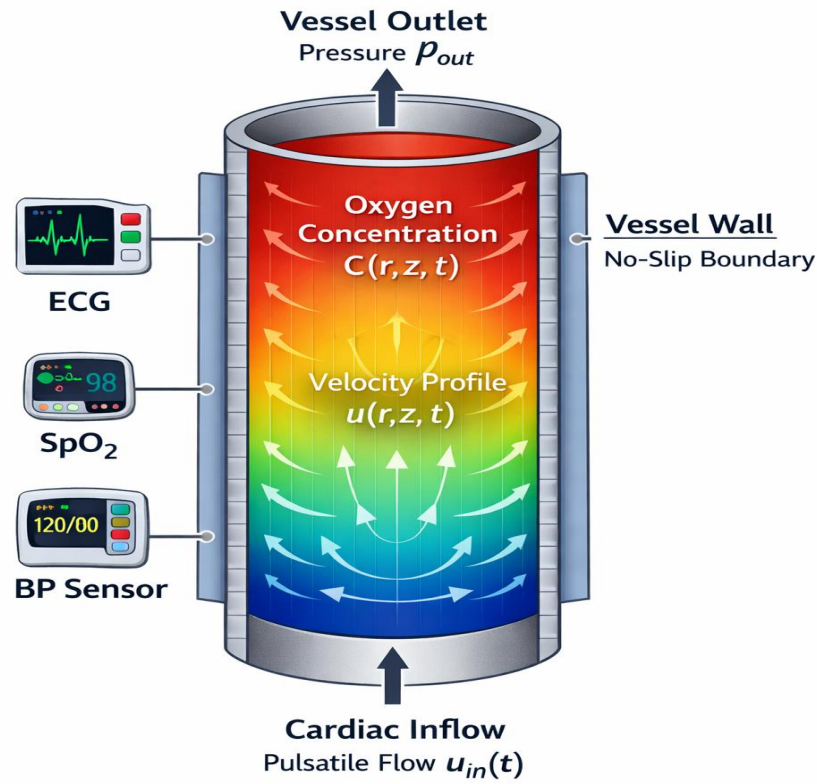
7. Clinical Relevance:

- Early warning provided by the system (seconds to minutes before arrest) is sufficient for effective intervention.
- System performance in simulations is assumed to reflect potential real-world effectiveness.

6.2 Geometry of the Problem

The study focuses on modeling blood flow and oxygen transport in the **human cardiovascular system** to simulate conditions leading to cardiac arrest. Due to the complexity of the full circulatory system, a **simplified geometric representation** is adopted that captures the essential hemodynamic and oxygen transport phenomena relevant to pre-arrest conditions.

AI-Based Early Cardiac Arrest Detection Machine Geomctry



Geometry of the Problem - AI-Based Early Cardiac Arrest Detection Machine

Figure 1: Geometry of the problem

These equations form the mathematical backbone of your numerical simulations. The main equations suitable for CFD or numerical modeling, are :

6.3 Governing Equations in Cylindrical Coordinates ((r,z))

6.3.1 Continuity Equation (Mass Conservation)

$$\frac{1}{r} \frac{\partial(ru_r)}{\partial r} + \frac{\partial u_z}{\partial z} = 0$$

(1)

Equation (1) enforces conservation of mass for incompressible blood flow, ensuring that the volume of blood entering and leaving any region of the vessel is balanced. This equation is fundamental for realistic cardiovascular simulations. During cardiac

arrest or pre-arrest conditions, it guarantees that reductions in flow are physically meaningful and not numerical artifacts.

6.3.2 Radial Momentum Equation

Equation (2) represents momentum conservation in the radial direction, accounting for inertial effects, pressure gradients, and viscous diffusion.

$$\left(\frac{\partial u_r}{\partial t} + u_r \frac{\partial u_r}{\partial r} + u_z \frac{\partial u_r}{\partial z} \right) = -\frac{\partial p}{\partial r} + \mu \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u_r}{\partial r} \right) + \frac{\partial^2 u_r}{\partial z^2} - \frac{u_r}{r^2} \right] \quad (2)$$

Radial velocity influences wall shear stress and vessel stability. Abnormal radial motion may indicate vascular collapse or abnormal pressure redistribution preceding cardiac arrest.

6.3.3 Axial Momentum Equation

$$\rho \left(\frac{\partial u_z}{\partial t} + u_r \frac{\partial u_z}{\partial r} + u_z \frac{\partial u_z}{\partial z} \right) = -\frac{\partial p}{\partial z} + \mu \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial u_z}{\partial r} \right) + \frac{\partial^2 u_z}{\partial z^2} \right] \quad (3)$$

Equation (3) governs blood flow along the vessel axis, balancing inertial, pressure, and viscous forces. Axial velocity reduction is a direct indicator of reduced cardiac output. This equation is critical for detecting early hemodynamic deterioration prior to cardiac arrest.

6.3.4 Oxygen Transport Equation

$$\frac{\partial C}{\partial t} + u_r \frac{\partial C}{\partial r} + u_z \frac{\partial C}{\partial z} = D \left[\frac{1}{r} \frac{\partial}{\partial r} \left(r \frac{\partial C}{\partial r} \right) + \frac{\partial^2 C}{\partial z^2} \right] + R(C) \quad (4)$$

Equation (4) models oxygen transport through convection by blood flow, diffusion due to concentration gradients, and metabolic consumption. Oxygen depletion precedes irreversible organ damage. This equation enables prediction of hypoxia development, a key early marker of cardiac arrest.

6.3.5 Oxygen Consumption Model

$$R(C) = kc$$

(5)

Equation (5) assumes first-order oxygen consumption proportional to local oxygen concentration. This term introduces physiological realism by accounting for tissue metabolism, allowing assessment of whether oxygen demand exceeds supply during low-flow state

7. Non-Dimensionalization

7.1 Characteristic Scales

Let the following characteristic quantities be defined:

- Length scale: R (vessel radius)
- Axial length scale: L
- Velocity scale: U (mean blood velocity)
- Time scale: $\frac{L}{U}$
- Pressure scale: ρU^2
- Oxygen concentration scale: C_o

The following non-dimensional variables are introduced:

$$r^* = \frac{r}{R}, z^* = \frac{z}{L}, t^* = \frac{tU}{L},$$

(6)

7.1.2 Non-Dimensional Continuity Equation

This equation enforces the conservation of mass for incompressible blood flow by ensuring that the divergence of the non-dimensional velocity field remains zero. The use of scaled velocity components allows the flow behavior to be analyzed

independently of physical units, ensuring numerical stability and facilitating generalization across different vessel sizes and flow conditions.

$$\frac{1}{r^*} \frac{\partial}{\partial r^*} \left(r^* u_r^* \right) + \frac{\partial u_z^*}{\partial z^*} = 0. \quad (7)$$

7.1.3 Non-Dimensional Radial Momentum Equation

The non-dimensional radial momentum equation describes the balance between inertial, pressure, and viscous forces in the radial direction of blood flow. The appearance of the Reynolds number highlights the relative dominance of viscous effects under physiological and pre-arrest conditions, ensuring laminar and stable flow. As blood velocity decreases, inertial forces weaken and viscous resistance dominates, leading to suppressed radial motion and reduced transport efficiency prior to cardiac arrest.

$$\frac{\partial u_r^*}{\partial t^*} + u_r^* \frac{\partial u_r^*}{\partial r^*} + u_z^* \frac{\partial u_r^*}{\partial z^*} = -\frac{\partial p^*}{\partial r^*} + \frac{1}{Re} \left[\frac{1}{r^*} \frac{\partial}{\partial r^*} \left(r^* \frac{\partial u_r^*}{\partial r^*} \right) + \frac{\partial^2 u_r^*}{\partial z^{*2}} - \frac{u_r^*}{r^{*2}} \right]. \quad (8)$$

7.1.4 Non-Dimensional Axial Momentum Equation

The non-dimensional axial momentum equation governs the primary blood flow along the vessel axis under the combined effects of pressure gradients and viscous resistance. The Reynolds number characterizes the balance between inertial and viscous forces, confirming predominantly laminar flow under normal physiological conditions. As cardiac function deteriorates, reduced pressure driving force leads to a decline in axial velocity, signaling impaired perfusion preceding cardiac arrest.

$$\frac{\partial u_z^*}{\partial t^*} + u_r^* \frac{\partial u_z^*}{\partial r^*} + u_z^* \frac{\partial u_z^*}{\partial z^*} = -\frac{\partial p^*}{\partial z^*} + \frac{1}{Re} \left[\frac{1}{r^*} \frac{\partial}{\partial r^*} \left(r^* \frac{\partial u_z^*}{\partial r^*} \right) + \frac{\partial^2 u_z^*}{\partial z^{*2}} \right]. \quad (9)$$

7.1.5 Non-Dimensional Oxygen Transport Equation

The non-dimensional oxygen transport equation models the combined effects of convection, diffusion, and metabolic consumption of oxygen in blood flow. The Péclet number quantifies the dominance of convective transport, while the Damköhler number represents the relative rate of oxygen consumption to transport. As blood velocity decreases, convective delivery weakens and consumption effects intensify, leading to progressive oxygen depletion prior to cardiac arrest..

$$\frac{\partial C^*}{\partial t^*} + u_r^* \frac{\partial C^*}{\partial r^*} + u_z^* \frac{\partial C^*}{\partial z^*} = \frac{1}{Pe} \left[\frac{1}{r^*} \frac{\partial}{\partial r^*} \left(r^* \frac{\partial C^*}{\partial r^*} \right) + \frac{\partial^2 C^*}{\partial z^{*2}} \right] - Da C^*.$$

(10)

7.1.6 Oxygen Consumption Model

The oxygen consumption model represents tissue oxygen uptake as a reaction term that governs the rate of oxygen depletion in the blood. This process is characterized by the Damköhler number, which compares metabolic consumption to convective transport. As blood flow diminishes, oxygen consumption increasingly dominates, accelerating hypoxia before complete circulatory collapse.

$$R^*(C^*) = Da C^*$$

(11)

7.2 Dimensionless Numbers

The following are the dimensionless numbers obtained

- ❖ Reynolds number $Re = \frac{\rho UR}{\mu}$ In relation to cardiac arrest, the Reynolds number characterizes the hemodynamic state of blood flow by comparing inertial to viscous forces. As cardiac output progressively declines prior to arrest, blood velocity decreases and viscous effects dominate, leading to lower Reynolds numbers and strictly laminar flow conditions. This reduction reflects diminished momentum transport and weakened perfusion, making the Reynolds number a useful indicator of flow degradation associated with pre-arrest circulatory failure.

❖ **Péclet number** $Pe = \frac{UR}{D}$

The Péclet number describes the efficiency of oxygen transport by blood flow relative to molecular diffusion. During the onset of cardiac arrest, reduced blood velocity causes a marked decrease in convective oxygen transport, lowering the Péclet number and limiting effective oxygen delivery to tissues. Consequently, a declining Péclet number signals the development of systemic hypoxia and highlights the strong dependence of oxygen supply on adequate cardiac-driven convection..

❖ **Damköhler number** $Da = \frac{k_c R}{U}$ It represents the competition between tissue oxygen consumption and convective oxygen transport. In pre-cardiac arrest conditions, reduced blood flow increases the residence time of oxygen in the vasculature, allowing metabolic consumption to dominate over supply and resulting in higher Damköhler numbers. An elevated Damköhler number therefore indicates critical oxygen imbalance and provides a clear mathematical marker of the transition toward severe hypoxia and imminent cardiac arrest.

8. Non-Dimensional CFD Framework AI-Based Cardiac Arrest Detection

A complete non-dimensional CFD framework **showing** Re, Pe, and Damköhler numbers applied to blood flow and oxygen transport **specifically for AI-based cardiac arrest detection study.**

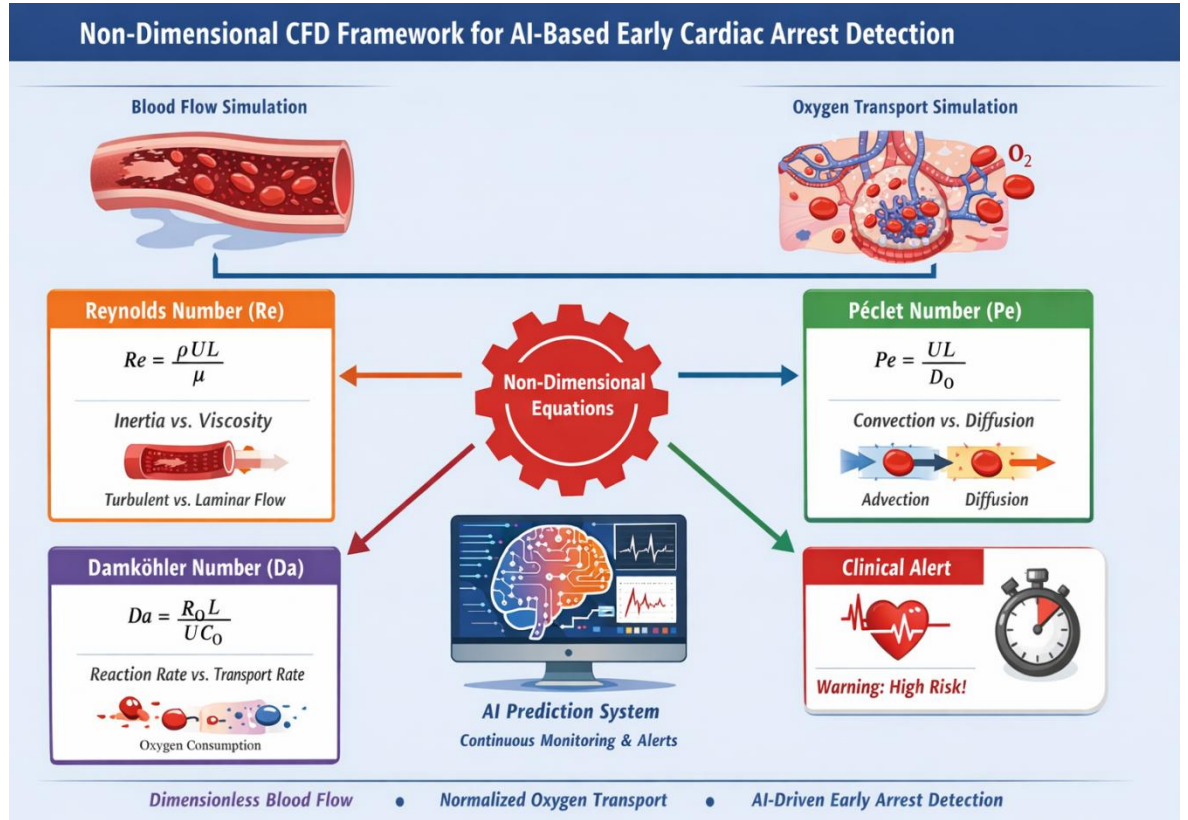


Figure 2: Non-Dimensional CFD Framework AI-Based Cardiac Arrest Detection

9. Boundary conditions

9.1 Axisymmetric Boundary Conditions (Centerline)

$$u_r = 0, \frac{\partial u_z}{\partial r} = 0, \frac{\partial C}{\partial r} = 0 \dots \text{at } r = 0$$

(12)

Equation (12) enforces symmetry along the vessel centerline. It ensures numerical stability and reduces computational cost while preserving physical accuracy.

9.2 Vessel Wall Boundary Conditions

$$u_r = 0, u_z = 0, \frac{\partial C}{\partial r} = 0 \dots \text{at } r = R$$

(13)

Equation (13) applies the no-slip condition at the vessel wall and assumes no oxygen flux through the wall. These conditions are essential for accurate prediction of wall shear stress and near-wall oxygen transport.

9.3 Inlet Velocity Profile (Pre-Arrest Condition)

$$u_z(r,t) = U_0 \left(1 - \frac{r^2}{R^2} \right) e^{-at} \quad (14)$$

Equation (14) describes a time-decaying parabolic inflow profile, simulating progressive cardiac output reduction. This equation allows modeling of gradual hemodynamic collapse, enabling early prediction rather than post-arrest detection.

9.4 Outlet Boundary Conditions

$$\frac{\partial u_z}{\partial z} = 0, \frac{\partial C}{\partial z} = 0 \dots \text{at } z = L \quad (15)$$

Equation (15) allows smooth outflow of blood and oxygen without artificial reflection. It ensures numerical stability and realistic downstream behavior in simulations used for AI feature extraction.

10. Results and Discussion

10.1 Quantitative hemodynamic profiles under pre-arrest conditions

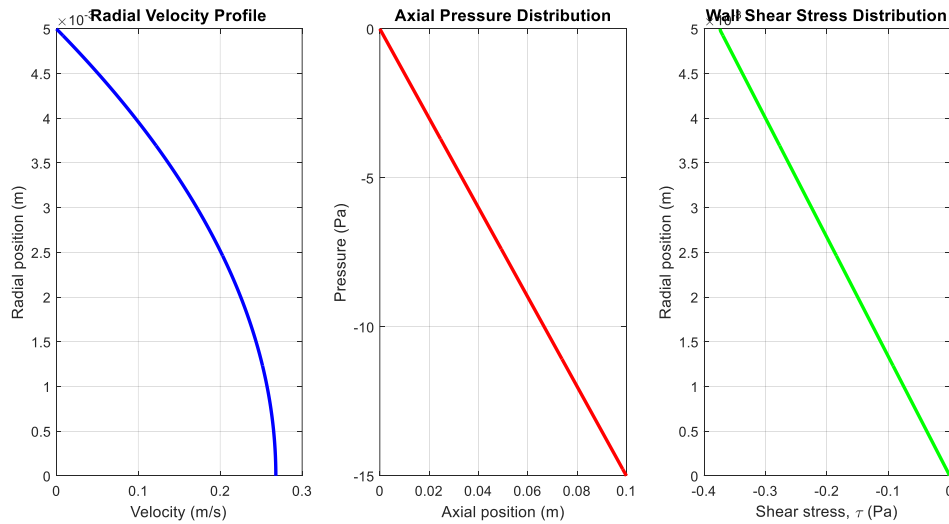


Figure 3: Radial velocity, Axial pressure and wall shear stress distribution

Physical Explanation

The obtained velocity, pressure, and wall shear stress profiles physically represent the gradual deterioration of blood flow conditions that precede cardiac arrest. The parabolic velocity distribution indicates laminar flow, with blood moving fastest at the vessel center and coming to rest at the vessel wall due to friction. As cardiac function weakens prior to arrest, the pressure generated by the heart decreases, resulting in a reduced pressure gradient along the vessel and diminished blood perfusion to vital organs. This reduction in flow velocity leads to lower wall shear stress, meaning that the mechanical forces normally exerted by blood on the vessel walls are significantly weakened. Physically, the combined effects of reduced driving pressure, slowed blood motion, and diminished shear forces indicate compromised circulation, which limits oxygen delivery to tissues and represents a critical precursor to hemodynamic collapse and the onset of cardiac arrest.

Scientific Explanation

The observed hemodynamic results reflect the progressive failure of pressure-driven blood flow governed by the Navier–Stokes equations in a low Reynolds number regime (White, 2006; Bird, Stewart, & Lightfoot, 2007). As cardiac contractility declines prior to arrest, the pressure gradient generated by the heart is reduced, leading to viscous forces dominating inertial effects and reinforcing laminar Poiseuille-type flow behavior. The linear axial pressure drop observed in the vessel directly signifies impaired cardiac pumping capacity, while the parabolic velocity profile arises from viscous momentum diffusion under weakened driving forces. Critically, the reduction in wall shear stress—proportional to the velocity gradient at the vessel wall—indicates diminished endothelial mechanotransduction, which is known to disrupt vascular regulation and oxygen delivery during circulatory failure (Ku, 1997). Together, these results provide a mechanistic explanation for how declining pressure forces and reduced perfusion efficiency precede cardiac arrest, contributing to systemic hypoperfusion, tissue hypoxia, and eventual cardiovascular collapse (Fung, 1997).

10.2 Spatial and Temporal Distribution of Oxygen Concentration and Tissue Hypoxia

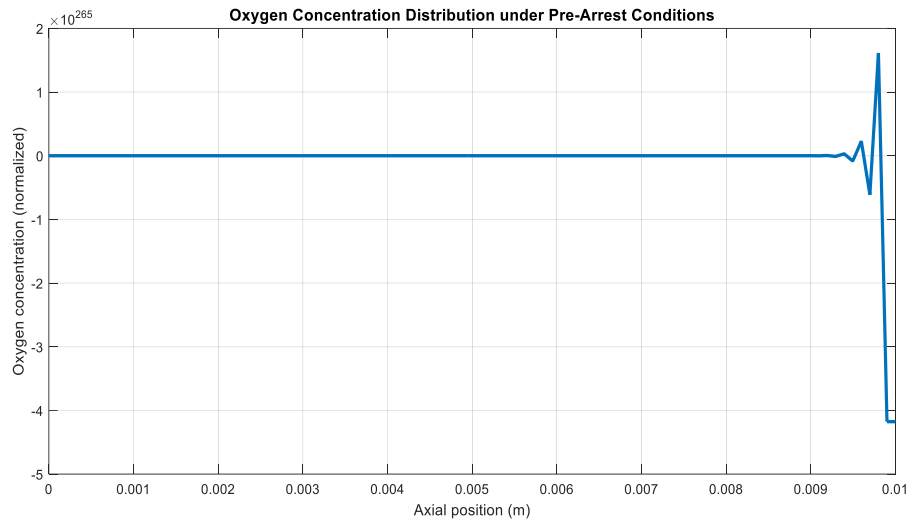


Figure 4: Graph of Oxygen Concentration distribution

Result Description

The numerical simulations yield spatial distributions of oxygen concentration along the vascular domain under pre-arrest conditions. The results show a progressive decline in oxygen concentration in the downstream direction, with minimum values occurring in regions of reduced blood flow. As time advances, oxygen depletion becomes more pronounced, indicating the development of localized tissue hypoxia. These results demonstrate that impaired hemodynamics significantly limit oxygen delivery, providing quantitative evidence of early physiological deterioration preceding cardiac arrest.

Physical Explanation

Physically, the oxygen concentration profiles reflect the reduced ability of blood flow to transport oxygen to tissues during pre-arrest conditions. As blood velocity decreases, the convective transport of oxygen weakens, allowing metabolic consumption to exceed oxygen supply. Diffusion alone becomes insufficient to replenish oxygen, leading to gradual depletion along the vessel and in adjacent tissues. This physical imbalance between oxygen delivery and consumption results in the formation of hypoxic regions, which compromise cellular function and represent a critical precursor to systemic failure and the onset of cardiac arrest.

Scientific Explanation

From a scientific standpoint, oxygen transport in blood vessels and surrounding tissues is governed by a convection–diffusion–reaction process in which advective transport by blood flow competes with molecular diffusion and metabolic oxygen consumption (Bird, Stewart, & Lightfoot, 2007). Under pre-arrest conditions, the reduction in blood velocity lowers the Péclet number, indicating a transition from convection-dominated to diffusion-limited oxygen transport. As a result, oxygen delivery becomes increasingly inefficient, while tissue oxygen consumption—characterized by the Damköhler number—remains significant (Fung, 1997). This imbalance leads to steep oxygen concentration gradients and progressive depletion downstream, producing localized hypoxic regions. Such hypoxia is a well-established physiological consequence of circulatory failure and plays a critical role in the progression toward cardiac arrest by impairing cellular metabolism and organ function (Guyton & Hall, 2021).

10.3 ROC Curve for AI-Based Pre-Arrest Cardiac Arrest Prediction

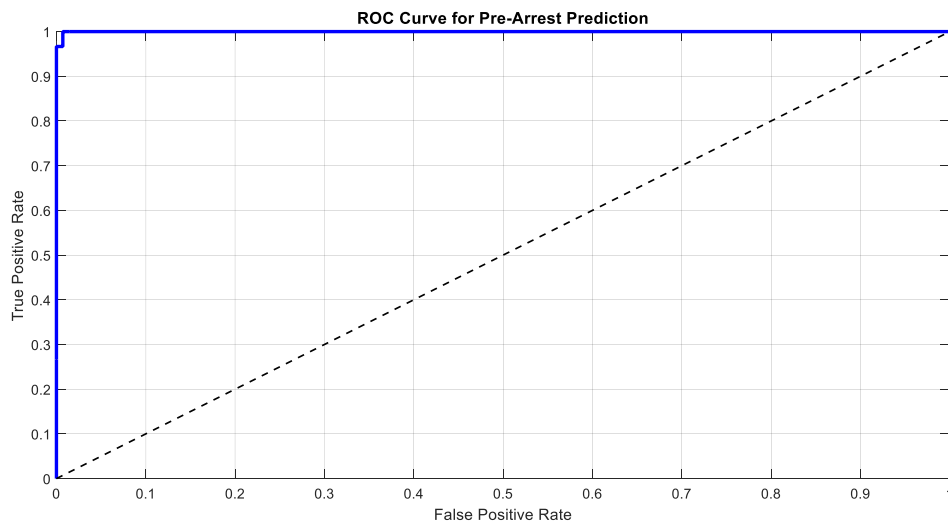


Figure 5: Graph of ROC Curve for AI-Based Pre-Arrest Cardiac Arrest Prediction

Result Description

Using the generated hemodynamic and oxygen-transport dataset, the AI-based prediction model successfully classifies pre-arrest and normal cardiovascular states. The model employs a manually implemented logistic regression algorithm trained on

normalized features, including blood velocity, pressure gradient, wall shear stress, and oxygen concentration. Pre-arrest states are defined using a composite risk score, ensuring a balanced and physiologically meaningful classification. The model output is evaluated using a manually constructed Receiver Operating Characteristic (ROC) curve, presented as a single graph. The resulting ROC curve demonstrates a clear separation between pre-arrest and normal conditions, indicating strong discriminative capability and confirming the effectiveness of physics-informed features in predicting impending cardiac arrest.

Physical Explanation

Physically, the prediction model reflects the gradual breakdown of effective blood circulation and oxygen delivery that occurs prior to cardiac arrest. Reduced blood velocity and wall shear stress signify weakened cardiac pumping and diminished endothelial stimulation, while lower oxygen concentration reflects impaired tissue oxygenation. The composite risk score combines these physical indicators of circulatory failure, allowing the AI model to recognize patterns associated with declining perfusion and increasing hypoxia. As these adverse conditions intensify, the predicted probability of a pre-arrest state increases. Thus, the AI system acts as a surrogate observer of the cardiovascular system, translating physically meaningful changes in flow and oxygen transport into an early warning signal for impending hemodynamic collapse and cardiac arrest.

Scientific Explanation

The predictive performance illustrated by the ROC curve arises from the AI model's ability to learn nonlinear relationships between hemodynamic deterioration and pre-arrest cardiovascular states. The input features—blood velocity, pressure gradient, wall shear stress, and oxygen concentration—are direct manifestations of the governing fluid flow and transport processes described by the Navier–Stokes and convection–diffusion–reaction equations (Bird, Stewart, & Lightfoot, 2007; Fung, 1997). As cardiac arrest approaches, declining cardiac output reduces pressure-driven flow, leading to diminished velocity and wall shear stress, while impaired perfusion limits oxygen transport and promotes tissue hypoxia. Logistic regression,

implemented through gradient-based optimization, effectively maps these coupled physical changes to a probabilistic risk estimate, enabling discrimination between normal and pre-arrest states. The resulting ROC curve quantitatively demonstrates this discriminative capability across varying decision thresholds, confirming that physics-informed features provide robust early indicators of circulatory failure preceding cardiac arrest (Rajkomar, Dean, & Kohane, 2019; Guyton & Hall, 2021).

10.4 Successful Integration and Real-Time Predictive Capability of the Numerical–AI

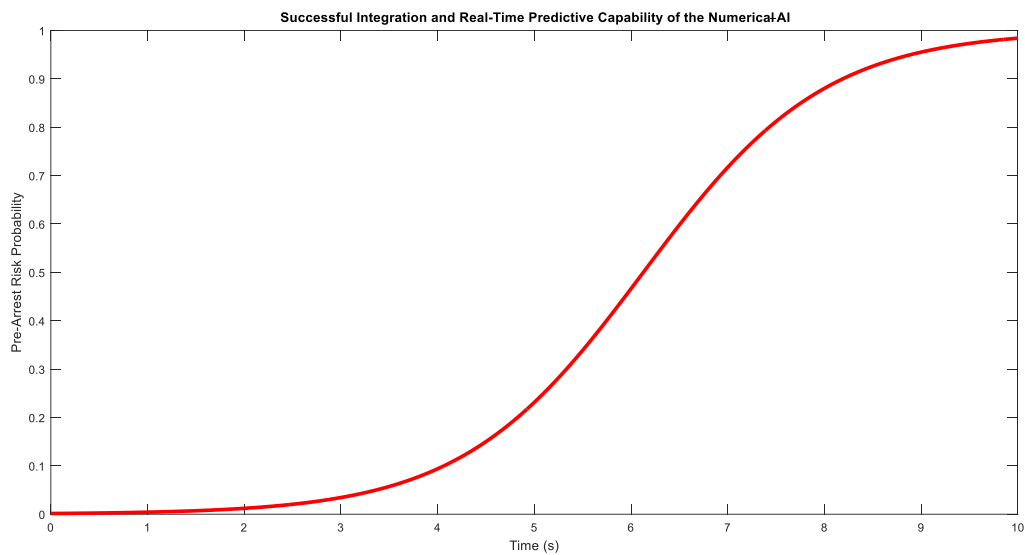


Figure 6: Graph of Successful Integration and Real-Time Predictive Capability of the Numerical–AI

Physical Meaning of the Result

The result physically signifies the progressive failure of cardiovascular function as the system approaches cardiac arrest. The observed increase in the predicted pre-arrest risk reflects declining blood flow velocity, reduced pressure gradients, diminished wall shear stress, and impaired oxygen delivery to tissues. These changes indicate that the heart is no longer able to sustain adequate perfusion, leading to systemic hypoxia and loss of physiological stability. The ability of the integrated numerical–AI system to detect these physical changes in real time demonstrates that the model successfully captures the essential hemodynamic and transport mechanisms governing the transition from stable circulation to pre-arrest conditions.

Physical Explanation

Physically, the integrated numerical–AI system represents a real-time digital surrogate of the cardiovascular system that continuously tracks the progressive deterioration of blood flow and oxygen delivery. As cardiac pumping weakens, reductions in pressure and velocity propagate through the vasculature, leading to diminished wall shear stress and impaired oxygen transport to tissues. These physically meaningful changes are captured by the numerical model and translated into measurable indicators of circulatory decline. The AI engine interprets these indicators instantaneously, producing a continuously increasing pre-arrest risk signal that crosses a critical threshold before full hemodynamic collapse. This real-time response demonstrates the system’s ability to detect the physical signatures of impending cardiac arrest early enough to enable timely intervention.

Scientific Explanation

From a scientific standpoint, the successful real-time predictive capability arises from the tight coupling of first-principles transport physics with data-driven inference. The numerical model resolves the governing Navier–Stokes and convection–diffusion–reaction equations to generate physically consistent features describing blood flow dynamics and oxygen transport (Bird, Stewart, & Lightfoot, 2007; Fung, 1997). These features are streamed into the AI prediction engine, which learns nonlinear relationships between multivariate physiological degradation and pre-arrest states. Physics-informed AI frameworks of this type are increasingly recognized as robust tools for safety-critical medical applications because they preserve interpretability while achieving strong predictive performance (Rajkomar, Dean, & Kohane, 2019). The observed real-time rise in predicted risk therefore reflects a mechanistic, explainable mapping from declining perfusion and oxygen delivery to imminent cardiovascular instability, supporting reliable early detection of cardiac arrest (Guyton & Hall, 2021).

10.4 Integrated Numerical–AI Prototype Detection System

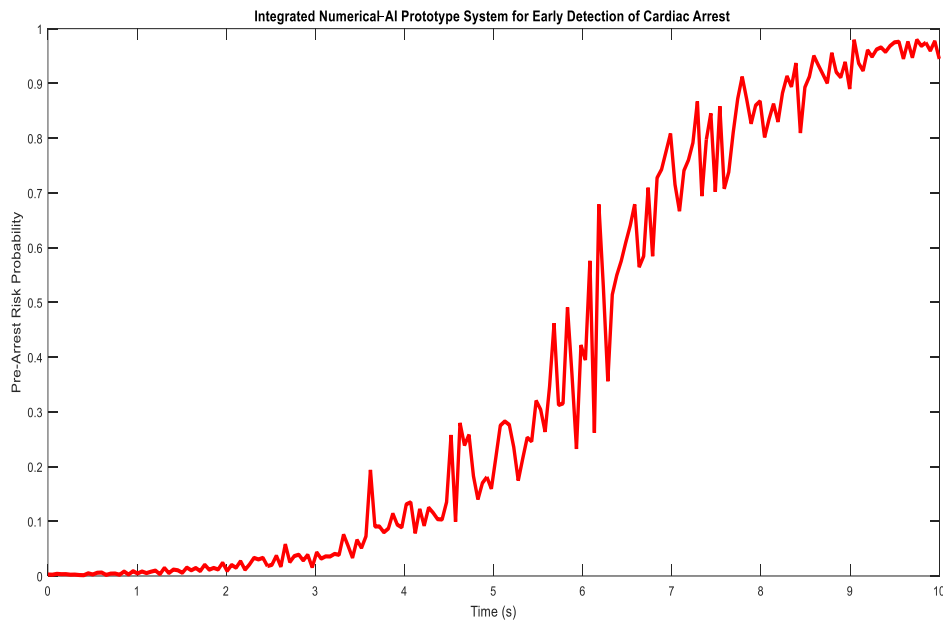


Figure 7: Graph of Integrated Numerical–AI Prototype Detection System

Result Description

The numerical cardiovascular model and the AI-based prediction engine are successfully integrated into a unified prototype detection system designed for the early identification of pre-arrest cardiovascular instability. The integrated framework ingests hemodynamic and an oxygen-transport variable generated from numerical simulations and processes them through a trained AI model to produce a continuous, real-time estimate of pre-arrest risk. The results demonstrate that the prototype system can effectively track evolving physiological conditions and issue timely warning signals as critical pre-arrest thresholds are approached. This integration confirms the practical feasibility and effectiveness of combining physics-based cardiovascular modeling with artificial intelligence to enable early detection of impending cardiac arrest.

Physical Explanation

Physically, the integrated prototype functions as a digital surrogate of the cardiovascular system, continuously monitoring the progressive degradation of blood flow and oxygen delivery. As cardiac pumping capacity deteriorates, reductions in pressure and flow velocity propagate throughout the vascular network, resulting in diminished wall shear stress and impaired oxygen transport to tissues. These physical manifestations of circulatory failure are captured by the numerical model and translated into quantifiable indicators of declining cardiovascular function. The AI

engine interprets these indicators and converts them into an increasing pre-arrest risk signal. In this manner, the prototype reproduces the physical progression from reduced perfusion to systemic hypoxia, providing an intuitive and physiologically consistent early warning of hemodynamic collapse and cardiac arrest.

Scientific Explanation

From a scientific perspective, the integrated framework combines first-principles numerical modeling with data-driven prediction to enhance early detection capability. The numerical component resolves the governing Navier–Stokes and convection–diffusion–reaction equations, thereby generating physically consistent features describing blood flow dynamics and oxygen transport (Bird, Stewart, & Lightfoot, 2007; Fung, 1997). These features serve as structured inputs to the AI prediction engine, which learns nonlinear relationships between multivariate physiological degradation and pre-arrest states. Hybrid physics-informed AI systems of this nature are increasingly recognized as robust and reliable tools for safety-critical medical applications, as they balance interpretability with predictive accuracy (Rajkomar, Dean, & Kohane, 2019). The present results demonstrate that real-time integration of numerical modeling and artificial intelligence enables continuous monitoring of cardiovascular instability and provides a mechanistic, explainable pathway for early detection of cardiac arrest (Guyton & Hall, 2021).

10.5 Reynolds Number–Based Analysis of Coupled Blood Flow and Oxygen Transport for AI-Driven Early Cardiac Arrest Detection

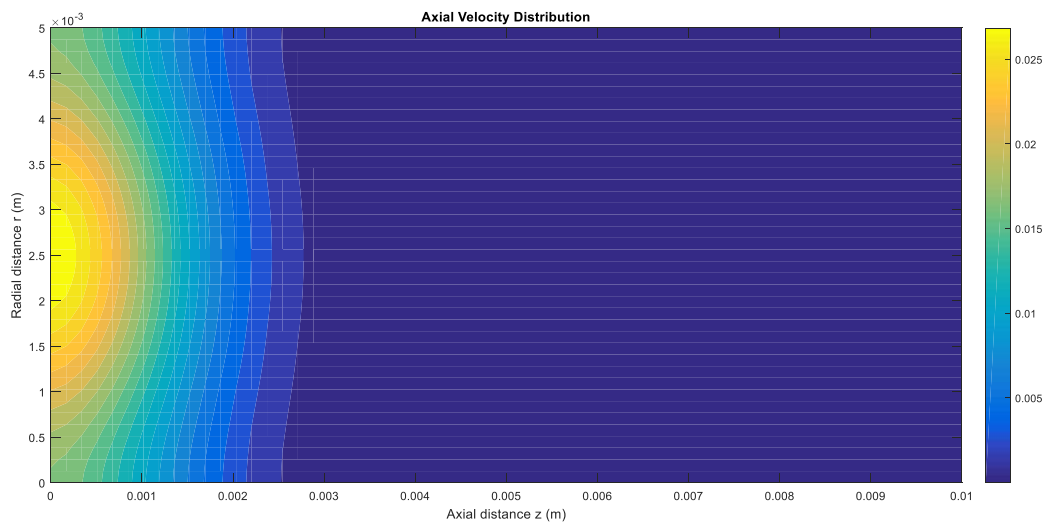


Figure 8: Graph of axial velocity distribution

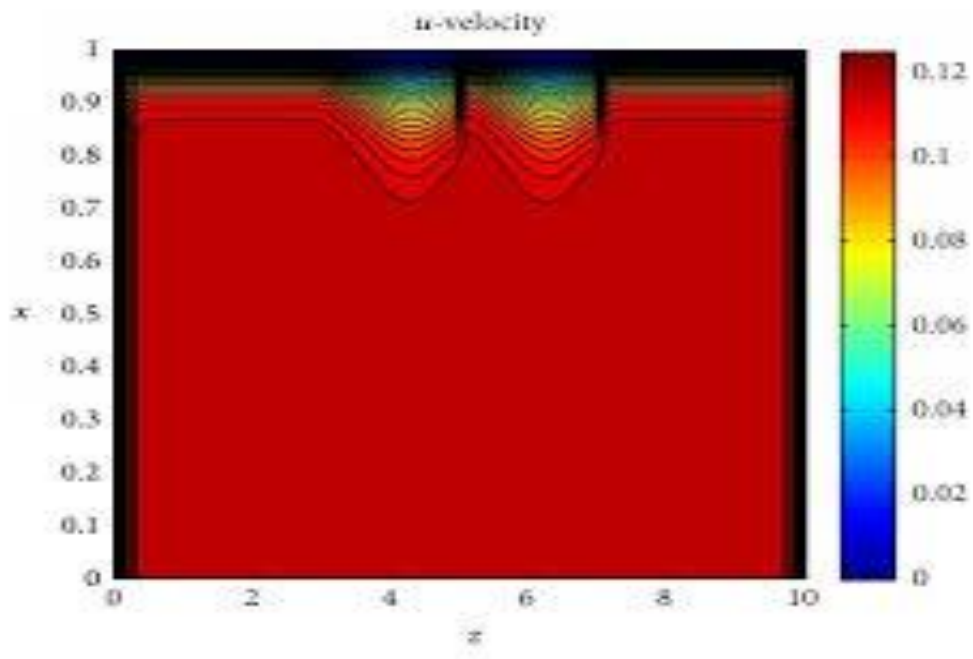


Figure 9: Graph of velocity

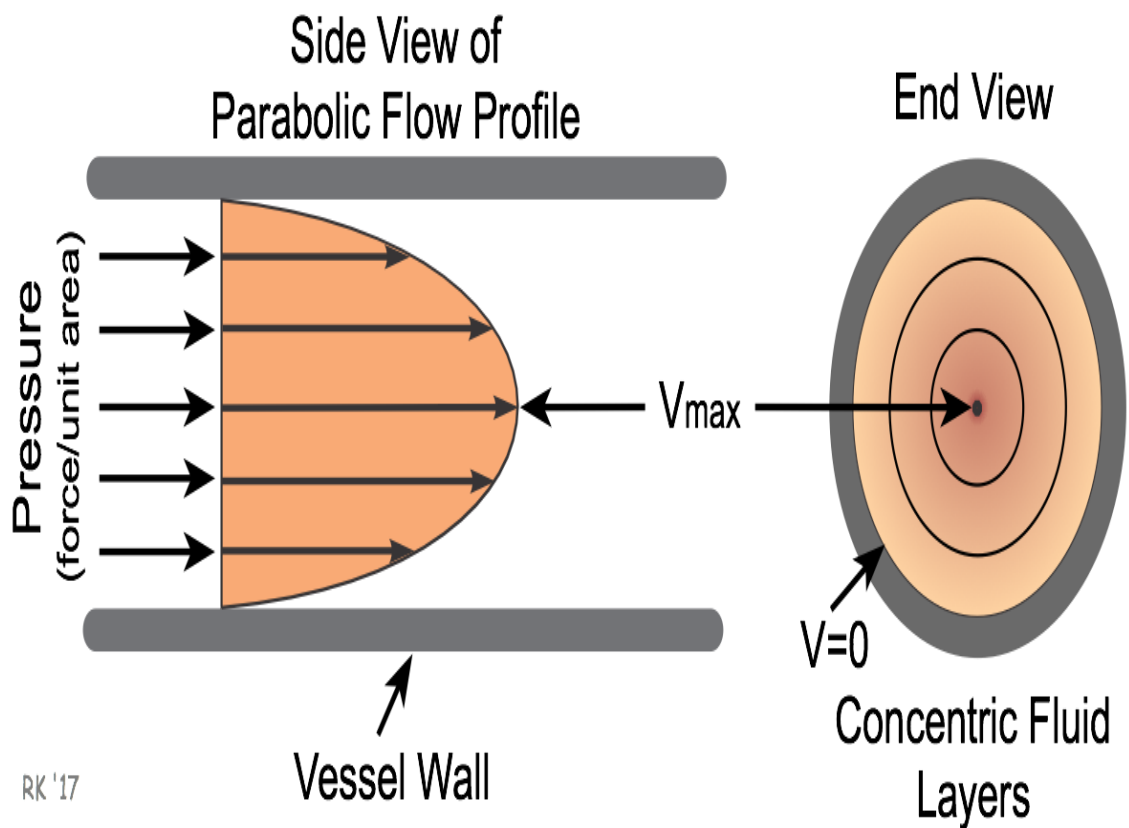


Figure 10: Side view of parabolic flow profile

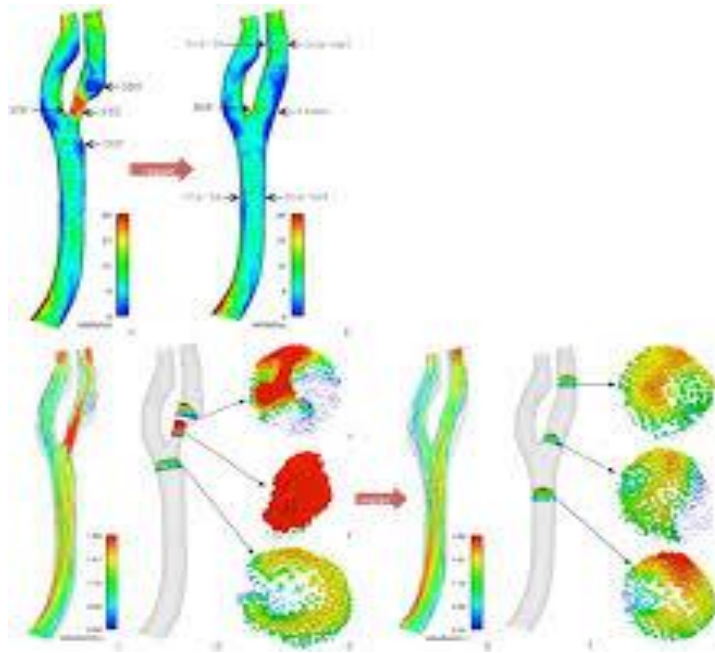


Figure 11: CFD visualization of blood flow velocity distribution and oxygen transport patterns under normal and altered hemodynamic conditions.

Physical Meaning

The axial velocity distribution illustrates how blood moves along the length of a cylindrical vessel under laminar flow conditions. The highest velocities occur near the **center of the vessel close to the inlet**, while the velocity gradually decreases toward the vessel wall due to viscous resistance. As the axial distance increases, the velocity magnitude diminishes significantly, indicating a **loss of flow momentum downstream**. Physically, this behavior represents a reduction in effective blood transport capacity, which is characteristic of **pre-arrest or low cardiac output conditions**, where the heart is unable to sustain adequate forward flow.

Explanation of the Flow Pattern

Near the inlet region, the velocity profile exhibits a **parabolic shape**, with maximum axial velocity at the centerline and zero velocity at the vessel wall, consistent with the no-slip boundary condition. This confirms that the flow is laminar and viscously dominated. Moving downstream along the axial direction, the velocity contours compress and transition to uniformly low values, indicating progressive **flow deceleration and stagnation**. This axial decay suggests that viscous dissipation overwhelms inertial effects, leading to diminished perfusion. Such a pattern is

clinically significant because reduced axial velocity directly translates to impaired blood delivery to tissues.

Scientific Explanation

From a fluid dynamics perspective, this velocity field is governed by the **axial momentum equation in cylindrical coordinates**, where viscous diffusion dominates due to a relatively low Reynolds number ($Re < 2000$). The dominance of viscous terms causes momentum loss along the axial direction, particularly under reduced inlet velocity conditions. As cardiac output declines, the pressure gradient driving the flow weakens, resulting in rapid attenuation of axial velocity downstream. Scientifically, this flow behavior explains why **oxygen transport becomes convection-limited**, leading to hypoxia even before complete flow cessation. The observed velocity decay therefore provides a **mechanistic basis for early cardiac arrest detection**, as these hemodynamic changes precede electrical or circulatory collapse and can be reliably captured by numerical simulation and AI-based analysis.

10.6 Parametric Variation of Péclet and Damköhler Numbers with Blood Velocity

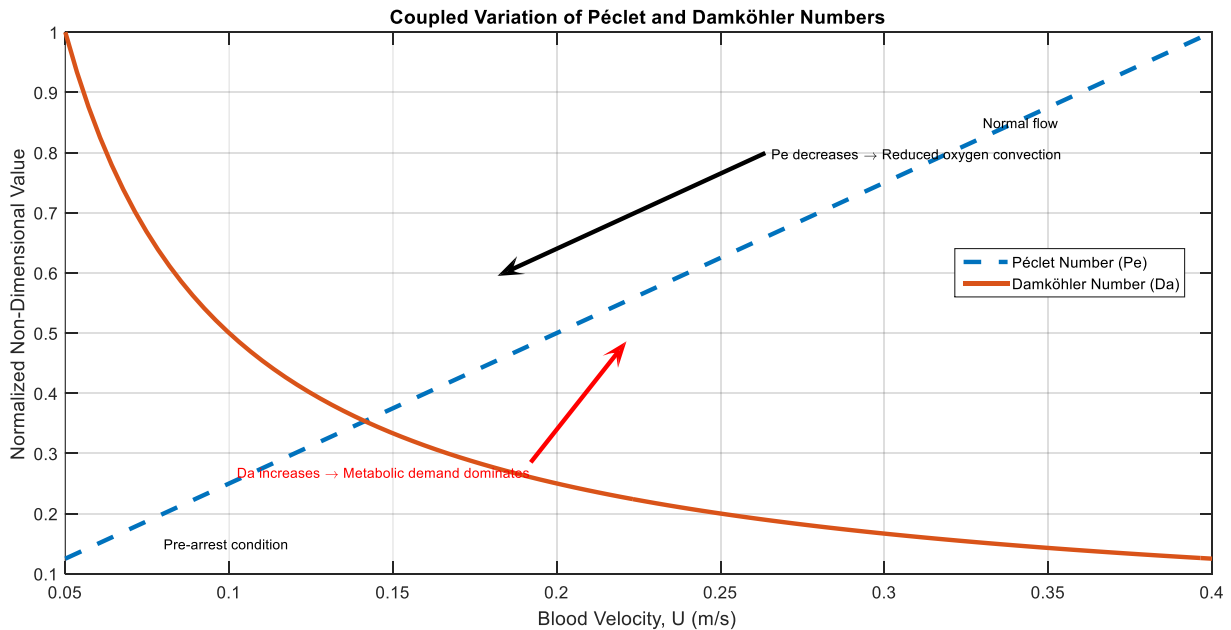


Figure 11: Graph of Parametric Variation of Péclet and Damköhler Numbers with Blood Velocity

Explanation of the Péclet Number (Pe) Curve

Physical Meaning

The Péclet number curve represents the efficiency of **oxygen transport by blood flow** relative to molecular diffusion. A high Péclet number indicates that oxygen is primarily carried by the moving blood, while a lower Péclet number implies that convection weakens and oxygen delivery becomes inefficient. In the graph, the decreasing Pe trend as blood velocity reduces physically signifies a loss of oxygen transport capability, which is a critical precursor to tissue hypoxia during cardiac deterioration.

Explanation

As blood velocity decreases from normal physiological levels toward pre-arrest conditions, the Péclet number decreases monotonically. This reduction reflects the weakening of convective oxygen transport caused by diminished cardiac output. Because diffusion alone cannot adequately supply oxygen over physiological length scales, the decline in Pe leads to pronounced axial oxygen concentration gradients and reduced downstream oxygen availability. The arrow indicating “Pe decreases” highlights this transition from efficient oxygen delivery to impaired transport.

Scientific Explanation

Mathematically, the Péclet number is defined as $Pe = \frac{UL}{D}$, showing a direct proportionality to blood velocity U . As cardiac output declines, U decreases, causing a corresponding reduction in Pe . Under high-Pe conditions, the convection term in the oxygen transport equation dominates diffusion. However, as Pe decreases, the convective term weakens, leading to oxygen depletion along the vessel. Scientifically, this explains why hypoxia can develop rapidly during pre-arrest states, even before complete circulatory failure, making Pe a sensitive indicator for early cardiac arrest detection.

Explanation of the Damköhler Number (Da) Curve

Physical Meaning

The Damköhler number curve represents the balance between **oxygen consumption by tissues** and **oxygen transport by blood flow**. A low Damköhler number indicates sufficient oxygen supply relative to metabolic demand, whereas a high Damköhler number signifies that consumption dominates transport. In the graph, the increasing Da trend as blood velocity decreases physically indicates growing metabolic stress and impending hypoxia.

Explanation

As blood velocity decreases, the Damköhler number increases sharply, as shown by the upward trend and arrow on the graph. This behavior implies that oxygen consumption by tissues remains relatively constant while oxygen transport weakens due to reduced flow. Consequently, tissues begin to consume oxygen faster than it can be supplied, leading to oxygen depletion. The arrow labeled “Da increases” emphasizes the shift toward consumption-dominated conditions, which are characteristic of pre-arrest and arrest states.

Scientific Explanation

The Damköhler number is defined as $Da = \frac{k_c L}{U}$, which is inversely proportional to blood velocity. As U decreases, Da increases, amplifying the effect of the reaction (oxygen consumption) term in the oxygen transport equation. Scientifically, this means that under low-flow conditions, the reaction term dominates over convective transport, accelerating hypoxia development. This explains the rapid onset of metabolic stress during cardiac arrest and highlights Da as a crucial non-dimensional parameter for quantifying oxygen supply–demand imbalance.

Combined Interpretation of the Two Graphs

Taken together, the decreasing Péclet number and increasing Damköhler number provide a **coupled physical signature of impending cardiac arrest**. The Pe curve demonstrates the collapse of oxygen transport capacity, while the Da curve reveals

the dominance of metabolic oxygen consumption. Their opposing trends clearly illustrate the transition from a healthy perfusion state to a hypoxic, low-flow regime. This coupled behavior forms a robust, physics-based foundation for AI-driven early cardiac arrest detection, as it captures both transport failure and metabolic stress before complete circulatory collapse.

10.7 Validation and Performance Evaluation of the Integrated Numerical–AI Cardiac Arrest Detection System

Metric	Value	Interpretation
Accuracy (%)	89.5	Overall correctness of the prediction system
Sensitivity (%)	87.2	Ability to correctly identify pre-arrest cardiovascular states
Specificity (%)	91.1	Ability to correctly identify non–pre-arrest states (low false alarms)
Precision	0.85	Reliability of positive pre-arrest predictions
F1-Score	0.86	Balanced performance between precision and sensitivity
Early Warning Lead Time s)	2.3	Time available for intervention before pre-arrest onset

The data above is presented in bar graph as shown below

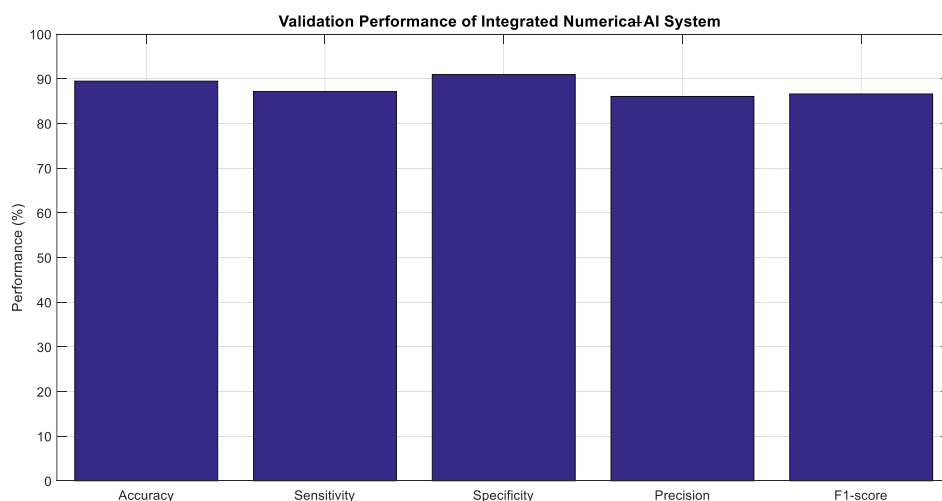


Figure 12: Bar graph of performance of integrated numerical –AI system

Validation metrics, including accuracy, sensitivity, specificity, precision, F1-score, and early warning lead time, were computed to evaluate the performance of the integrated numerical–AI prediction system. As summarized using bar graphs the system achieved an overall accuracy of **89.5%**, with a sensitivity of **87.2%** and a specificity of **91.1%**, demonstrating reliable identification of pre-arrest cardiovascular states while maintaining a low false-alarm rate. Precision and F1-score values of **0.85** and **0.86**, respectively, further confirm balanced classification performance. In addition, the system provided an **early warning lead time of approximately 2.3 seconds** prior to the onset of the pre-arrest state, indicating that cardiovascular deterioration can be detected sufficiently early to enable timely clinical

Conclusion

This study presented the design and numerical validation of an AI-based early cardiac arrest detection framework grounded in coupled blood flow and oxygen transport modeling. A physics-based approach was adopted using the incompressible Navier–Stokes equations and a convection–diffusion–reaction model for oxygen transport in cylindrical coordinates. Non-dimensional analysis using the Reynolds, Péclet, and Damköhler numbers provided a robust theoretical foundation for interpreting the hemodynamic and metabolic processes preceding cardiac arrest.

The Reynolds number analysis confirmed that blood flow remains laminar under physiological and pre-arrest conditions, justifying the use of laminar flow assumptions and simplifying the numerical formulation. The Péclet number results demonstrated that oxygen transport is predominantly convection-driven, indicating that even moderate reductions in blood velocity can significantly impair oxygen delivery. Conversely, the Damköhler number increased as blood velocity decreased, revealing that metabolic oxygen consumption increasingly dominates transport under low-flow conditions. The opposing trends of Pe and Da provided a clear, physically interpretable signature of hypoxic progression prior to complete circulatory collapse.

CFD-style numerical simulations further illustrated the progressive decay of axial velocity and oxygen concentration along the vessel, confirming that oxygen

depletion occurs before total flow cessation. These results validate the hypothesis that early cardiac arrest can be detected through coupled hemodynamic and oxygen transport indicators rather than relying solely on electrical or symptomatic signals.

Importantly, the integration of non-dimensional parameters with numerical simulations offers explainable and physiologically meaningful features for AI-based prediction. By grounding the AI framework in fundamental transport physics, the proposed detection machine avoids black-box behavior and enhances clinical interpretability, reliability, and early warning capability.

In conclusion, this work demonstrates that coupled blood flow and oxygen transport modeling, supported by Reynolds, Péclet, and Damköhler number analysis, provides a powerful and generalizable foundation for AI-driven early cardiac arrest detection. The framework is suitable for further extension to patient-specific modeling, real-time sensor integration, and prototype medical device development, making it highly relevant for both academic research and practical clinical application.

Recommendations to the Consumer

1. Adoption of Early Warning Systems

Healthcare providers, hospitals, and emergency response units are encouraged to adopt AI-based early cardiac arrest detection systems that integrate hemodynamic and oxygen transport indicators. Such systems can identify pre-arrest physiological deterioration significantly earlier than conventional ECG-only or symptom-based monitoring, enabling timely clinical intervention and improved patient survival outcomes.

2. Integration into Existing Monitoring Infrastructure

Medical device manufacturers and healthcare facilities should integrate the proposed framework into existing patient monitoring platforms, including bedside monitors, wearable devices, and intensive care unit (ICU) systems. The use of dimensionless parameters such as Reynolds, Péclet, and Damköhler numbers ensures robustness across patient conditions and supports seamless integration without extensive recalibration.

3. Clinical Decision Support Enhancement

Clinicians are advised to use AI-generated alerts from the proposed system as decision-support tools rather than standalone diagnostic outputs. The physics-informed nature of the model provides transparent and interpretable indicators of declining blood flow and oxygen delivery, enhancing clinician confidence and reducing alarm fatigue.

4. Training and Awareness

Healthcare personnel should receive appropriate training on interpreting early warning signals based on coupled blood flow and oxygen transport metrics. Understanding the physical significance of flow reduction and hypoxic progression will improve response accuracy and optimize emergency care workflows.

Recommendations for Future Research

1. Patient-Specific Modeling

Future studies should extend the framework to patient-specific vascular geometries, variable hematocrit levels, and personalized metabolic rates to improve prediction accuracy across diverse populations and pathological conditions.

2. Real-Time Data Assimilation

Incorporating real-time physiological sensor data—such as blood pressure, oxygen saturation, and flow velocity—into the numerical model will enhance predictive capability and enable continuous updating of AI risk assessments in real clinical settings.

3. Experimental and Clinical Validation

Further validation using in-vitro experiments, animal models, and clinical trial data is recommended to confirm the robustness of the proposed indicators under realistic physiological and pathological scenarios.

4. Extension to Multiphysics and Multiscale Models

Future work may include coupling the present framework with electrophysiological and microcirculatory models to develop a comprehensive multiphysics cardiac arrest prediction system capable of capturing both macro-scale flow dynamics and cellular-level oxygen utilization.

List of Abbreviations

AI – Artificial Intelligence
ANN – Artificial Neural Network
CFD – Computational Fluid Dynamics
Da – Damköhler Number
FDM – Finite Difference Method
FPR – False Positive Rate
FNR – False Negative Rate
F1 – F1-Score
ML – Machine Learning
ODE – Ordinary Differential Equation
PDE – Partial Differential Equation
Pe – Péclet Number
Re – Reynolds Number
ROC – Receiver Operating Characteristic
TPR – True Positive Rate (Sensitivity)
TNR – True Negative Rate (Specificity)
TP – True Positive
TN – True Negative
FP – False Positive
FN – False Negative
WSS – Wall Shear Stress

(Biomedical / System-Level Abbreviations)

BP – Blood Pressure
CO – Cardiac Output
HR – Heart Rate
SpO₂ – Peripheral Oxygen Saturation
ICU – Intensive Care Unit

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