Neural Network-Based Digital Twin for Hypertension Simulation

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Abstract. Hypertension, a significant cardiovascular disease, necessitates continuous monitoring and precise predictive models to optimize treatment strategies. This research proposes a Digital Twin for Hypertension, integrating a mathematical model based on Olufsen's differential equations with artificial neural networks to dynamically adjust vascular resistance, arterial compliance, and blood flow inertia. This model is implemented in MATLAB®, utilizing the Runge-Kutta method to solve 11 differential equations.In contrast with conventional blood pressure monitoring techniques, which rely on discrete measurements, this approach provides real-time estimation of blood pressure fluctuations, allowing for improved risk stratification and personalized treatment recommendations. Expected results suggest that the use of the digital twin can enhance hypertension management by adapting to individual physiological conditions and predicting pressure variations more accurately than conventional methods. Compared to existing compartmental models, this approach leverages neural networks to optimize parameter estimation, improving prediction accuracy and adaptability. However, challenges remain about acquiring real-time data and achieving adequate computational efficiency, issues which will be addressed in future research. Potential applications include integrating wearable devices for continuous blood pressure monitoring and simulation of pharmacological interventions. It is submitted that this research makes a significant contribution to electronics and communication by demonstrating how mathematical modelling and artificial intelligence can be combined to enhance medical simulations. Future research will focus on real-time validation, optimization of computational performance, and extending the model's applicability to personalized medicine. The digital twin framework has been identified as a valuable instrument for enhancing hypertension diagnosis, treatment, and research in cardiovascular health.

Keywords: Digital Twin · Hypertension · Mathematical Modeling · Neural Networks · Blood Pressure Simulation · Cardiovascular System

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1 Introduction

Hypertension is the predominant risk factor for cardiovascular diseases and is recognized as a global public health issue. Current estimates suggest that more than 1.28 billion adults worldwide live with hypertension, while approximately 46 percentage of them are unaware of their diagnosis, according to the World Health Organization (WHO) [1]. In recent years, this has led to a growing need to monitor and control blood pressure to prevent complications such as heart attacks, strokes, and kidney failure. In this scenario, computational modelling methods based on tool development have enabled the creation of digital twins, which are virtual systems capable of modelling a patient's physiological behaviour and being actively updated with real-time individual data [2]. In instances of hypertension, for illustration, mathematical models have been shown to be conducive to the combination with artificial intelligence, thereby enhancing the capacity for prediction of physiological states and facilitating more informed medical decision-making. In this article, the development of a digital twin for hypertension monitoring is the focal point. This digital twin incorporates artificial neural networks, which are employed to predict blood pressure. Additionally, a detailed exposition is provided of the underlying mathematical model utilized for this purpose, in addition to a description of how it is implemented in MATLAB® environment. Furthermore, an examination of potential implications for early detection and personalized treatment of hypertension is undertaken. It also analyzes how relevant and beneficial such a method is in comparison to traditional blood pressure monitoring and control approaches. Following that, other sections provide an overview of relevant literature on the applicability of digital twins in healthcare, existing hypertension research, and artificial neural networks in this field. Then, there is a presentation of how the methodology was adopted, including a mathematical model, implementation process, evaluation methods, and validation. Subsequently, it shows the anticipated results and discusses their implications for medical practice. Finally, conclusions and future research directions in this field are outlined.

2 Literature Review

Advanced technology development in recent years has driven the rapid creation of digital twins across various fields, including medicine. A digital twin is a real-time virtual model representing a physical system, achieved through data collection and continuous self-updating to simulate and predict performance [4].

In medicine, these models offer valuable applications for personalizing diagnosis and treatment of chronic diseases, as deep learning has consistently improved clinical strategies' accuracy and preventive measures. In cardiology, orthopaedics, and neurology, recent studies have explored heart behaviour modelling, joint crush simulation, and prediction of neurodegenerative disease progression [5,6], respectively. Digital twin applications can help hypertension predict hypertensive episodes, assess real-time treatment impact, and offer a more adaptable, personalized approach to patient care.

Despite potential benefits, the complexity of the cardiovascular system—shaped by physiological, environmental, and genetic factors—makes hypertension modelling one of the most challenging tasks in biomedical research. Over recent decades, various mathematical models have been proposed to replicate blood pressure dynamics, using differential equations or analogue circuits to resemble electrical systems [7]. Among these, the Windkessel model has become a widely adopted approach for modelling blood pressure dynamics, drawing an analogy to a simple hydraulic system with resistive and capacitive components [8]. However, classical models struggle to capture interindividual variability and long-term treatment effects. Artificial neural networks have been suggested for hypertension modelling to address these limitations, as they can recognize nonlinear patterns in blood pressure data and enhance prediction accuracy [8,9].

So far, artificial neural networks have been applied in studies for clinical data analysis and cardiovascular disease (CVD) prediction. Recent research has demonstrated their ability to predict blood pressure from electrocardiography (ECG) and photoplethysmography (PPG) signals with greater accuracy than traditional models [10]. Additionally, deep learning algorithms have been introduced to detect patterns in data from hypertensive patients, enabling early risk factor recognition and optimized therapeutic strategies [11]. However, these advancements are rarely integrated into digital twin models based on neural networks for hypertension. Most studies rely solely on historical data analysis, lacking capability for dynamic simulation and real-time patient-specific treatment adaptation.

Although significant progress has been made in leveraging artificial intelligence for hypertension management, integrating these models into a digital twin capable of providing real-time insights into individual physiology remains challenging [8]. Current studies have primarily developed neural networks that predict trends based on historical data, yet they do not dynamically interact with patients or adjust treatments according to physiological responses. Additionally, many established models rely on population-based datasets, limiting their ability to personalize predictions and reducing their practical applicability in clinical settings. While numerous studies have demonstrated feasibility of implementing one or more components of a digital twin, lack of comprehensive clinical validations and challenges in merging multiple biomedical data sources into a unified framework continue to hinder adoption of a fully operational digital twin for hypertension management [11]. This study aims to bridge this gap by introducing a digital twin based on artificial neural networks. This twin simulates blood pressure evolution in hypertensive patients while optimizing treatment based on individual physiological responses. This approach enhances prediction accuracy by integrating a validated mathematical model with state-of-the-art machine learning algorithms. It offers an innovative tool for personalized hypertension treatment. I will schedule some time for us to connect.

3 Methodology

3.1 Description of Mathematical Model

Olufsen et al. (2005) proposed a compartmental system of ordinary differential equations (ODEs) to describe blood pressure and flow dynamics across multiple segments of the cardiovascular system. Developed to capture interindividual variability in hypertensive patients better, this model integrates cardiac contraction dynamics with portal and arterial circulation to reflect interactions between vessel dynamics and distribution [13].

Human cardiovascular function operates as a highly complex circuit, exhibiting pulsatile blood flow and variable blood pressure due to necessary cardiac contraction and relaxation, peripheral vascular resistance, and arterial capacitance. Olufsen's model extends classical approaches, such as Windkessel-based representations, by describing coupled arterial segments through differential equations.

As a compartmental model, it divides cardiovascular circulation into distinct arterial and venous compartments, each governed by a differential equation that describes pressure and blood volume evolution over time. This approach captures how pressure waves propagate through blood vessels and highlights the impact of arterial stiffness in individuals with hypertension.

Model Components Olufsen's model includes three main elements to represent the cardiovascular system: Resistance R represents opposition to blood flow due to friction between blood and arterial walls. Blood within the system behaves according to Hagen-Poiseuille's law, where vessel resistance is inversely proportional to the fourth power of vessel radius [12]. Slight variations in vessel diameter can lead to significant changes in flow resistance. This relationship is modelled using differential equations in Olufsen's model, which describe pressure drop along arterial circulation and allow assessment of how pressure changes in response to peripheral resistance [13].

Arterial capacitance C represents the ability of blood vessels to store blood and their elasticity, directly influencing blood pressure dynamics [14]. In Olufsen's model, this parameter is crucial for estimating pressure variations caused by changes in blood volume, which is particularly relevant in pathological conditions such as hypertension, where vessel stiffening prevents proper vascular adjustment to blood flow variations. Blood flow inertia Lmodels the delay in flow response caused by blood mass. These factors are key in studying pressure wave propagation in large arteries, affecting circulatory dynamics and interaction between blood ejection and vascular resistance. In Olufsen's model, inertia is incorporated into the equation governing flow dynamics to characterize how pressure and flow react swiftly to changes in heart rate and structural properties of blood vessels across arterial circulation [17].

Differential Equations for Blood Pressure: Each equation signifies this progression, delineating a distinct segment of the cardiovascular system.

$$\frac{dP_a}{dt} = \frac{\frac{(P_{lv}-P_a)}{R_{av}} - \frac{(P_a-P_{au})}{R_{au}} - \frac{(P_a-P_{ac})}{R_{ac}} - \frac{(P_a-P_{af})}{R_{af}}}{C_a} \tag{1}$$

$$\frac{dP_{au}}{dt} = \frac{\frac{(P_a-P_{au})}{R_{au}} - \frac{(P_{au}-P_{al})}{R_{al}} - \frac{(P_{au}-P_{vu})}{R_{aup}}}{C_{au}} \tag{2}$$

$$\frac{dP_{al}}{dt} = \frac{\frac{(P_{au}-P_{al})}{R_{al}} - \frac{(P_{al}-P_{vl})}{R_{alp}}}{C_{al}} \tag{3}$$

$$\frac{dP_{af}}{dt} = \frac{\frac{(P_a-P_{af})}{R_{af}} - \frac{(P_{af}-P_v)}{R_{afp}}}{C_{af}} \tag{4}$$

$$\frac{dP_{ac}}{dt} = \frac{\frac{(P_a-P_{ac})}{R_{ac}} - \frac{(P_{ac}-P_{vc})}{R_{acp}}}{C_{ac}} \tag{5}$$

$$\frac{dP_v}{dt} = \frac{\frac{(P_{vu}-P_v)}{R_{vu}} + \frac{(P_{vc}-P_v)}{R_{vc}} + \frac{(P_{af}-P_v)}{R_{afp}} - \frac{(P_v-P_{la})}{R_v}}{C_v} \tag{6}$$

$$\frac{dP_{vu}}{dt} = \frac{\frac{(P_{vl}-P_{vu})}{R_{vl}} + \frac{(P_{au}-P_{vu})}{R_{aup}} - \frac{(P_{vu}-P_v)}{R_{vu}}}{C_{vu}} \tag{7}$$

$$\frac{dP_{vl}}{dt} = \frac{\frac{(P_{al}-P_{vl})}{R_{alp}} - \frac{(P_{vl}-P_{vu})}{R_{vl}}}{C_{vl}} \tag{8}$$

$$\frac{dP_{vc}}{dt} = \frac{\frac{(P_{ac}-P_{vc})}{R_{acp}} - \frac{(P_{vc}-P_v)}{R_{vc}}}{C_{vc}} \tag{9}$$

Compartmental models, such as Olufsen's model, represent a system of 9 ordinary differential equations describing blood pressure evolution across different compartments of cardiovascular circulation. These equations establish relationships between pressure, flow, and resistance in specific arterial or venous regions. Nine differential equations governing how pressure influences blood circulation are described below. Differential equation (1) represents arterial pressure in systemic arterial circulation (aorta). This equation accounts for blood entering through the left ventricle and distributing into significant arteries. Resistance across the aortic valve R_{av} influences pressure at this stage, with blood flow redirected toward upper body arteries, capillary beds, and femoral arteries R_{au}, R_{ac}, R_{af}. Variations in these parameters help regulate overall blood pressure, increasing susceptibility to hypertension [13].

Second differential equation (2) corresponds to pressure in arteries supplying the upper body, specifically subclavian and carotid arteries. Blood is directed into this region from the aorta through resistance R_{au} and exits via R_{al} toward head and arm circulation. R_{aup} regulates arterial pressure in this region and influences redistribution of blood flow into superior venous circulation [13].

Equation (3) describes pressure in narrower upper-body arteries, which exhibit increased resistance due to progressive vessel constriction. This moist lung region lacks pressure in either pulmonary ventricle, where suction is determined by blood flow exiting P_{au} and entering superior venules P_{vl}, regulated by R_{alp}. Increased vessel stiffness in this region can result in localized hypertension in upper circulation [13].

Fourth differential equation (4) involves pressure in arteries supplying the lower body, specifically femoral arteries and branches. Blood flow from the aorta reaches this site through resistance R_{af}, while venous return from this area is regulated by R_{afp}. Reduced arterial compliance in this segment may contribute to increased systolic pressure in lower extremities [13].

Equation (5) governs transition toward capillary beds through filtration and fluid reabsorption mechanisms between arteries and veins. At this stage, blood pressure decreases significantly due to capillary resistance R_{ac} and outflow toward capillary venules R_{ac}. Microcirculatory disruptions can alter the fluid balance and contribute to pathologies such as capillary hyper flow [13].

Sixth differential equation (6) represents pressure in central venous circulation. Veins from various compartments, including upper circulation P_{vu}, capillary circulation P_{vc}, and femoral veins P_{af}, return blood to the heart through VNC, influenced by venous resistances $R_{vu}, (R_{vc}, (R_{fp}$. Central venous pressure is a key indicator of cardiac preload status and impacts cardiac function [13].

Seventh differential equation (7) describes pressure in superior veins, responsible for draining venous return from the head and arms. Flow rates from P_{vl} and P_{au} into P_{vu} are regulated by R_{vl} and R_{aup}, while outflow into systemic circulation occurs through R_{vu}. Alterations in venous return within this region can affect cerebral blood flow and venous pressure dynamics, influencing cerebral compliance [13].

Eighth differential equation (8) defines pressure in upper-body venules. Pressure in this region is determined by blood entering P_{al} and exiting through P_{vu}, where R_{alp} and R_{vl} regulate inflow and outflow, respectively. Variations in these resistances impact blood redistribution and venous pressure within the thoracic region [13].

Ninth differential equation (9) describes pressure in capillary venous circulation. Blood moves from P_{ac} to P_{vc} through R_{acp}, with venous return to P_v regulated by R_{vc}. Pressure within this compartment plays a crucial role in fluid balance, tissue perfusion, and systemic blood pressure regulation [13].

Each equation is fundamental in defining hemodynamic dynamics and arterial pressure distribution throughout circulation. Variations in R, C, or flow characteristics lead to alterations in overall hemodynamics, reinforcing Olufsen's model as a valuable tool for testing hypertension predictions and control strategies.

Equations for Ventricular Volume: Ventricular volume changes over time in response to pressure and resistance of the cardiac compartments:

$$\frac{dV_{lv}}{dt} = \frac{P_a - P_{lv}}{R_{mv}} - \frac{P_{lv} - P_a}{R_{av}} \tag{10}$$

$$\frac{dV_{la}}{dt} = \frac{P_v - P_{la}}{R_v} - \frac{P_{la} - P_{lv}}{R_{mv}} \tag{11}$$

Differential equation (10), which describes variations in left ventricular volume, is the primary driver of systemic blood pressure. The dynamic balance between inflow from the left atrium and outflow toward the aorta determines this. Blood flow occurs through the mitral valve, where resistance R_{mv} regulates the amount of blood transferred from the left atrium into the left ventricle. Consequently, blood ejection into systemic circulation occurs through the aortic valve, where resistance R_{av} determines the volume successfully delivered by the ventricle with each heartbeat [13].

Blood flow happens during this phase (ventricular filling) when left atrial pressure P_{la} exceeds ventricular pressure P_{lv}. Conversely, systole is when ventricular pressure rises sufficiently to open the aortic valve, allowing blood to be expelled into the aorta. This process is crucial for maintaining blood pressure and ensuring cardiac output, meaning that valvular resistance or ventricular contractility variations can influence stroke volume and organ perfusion [13].

On the other hand, differential equation (11) describes volume changes in the left atrium, a critical compartment for regulating blood pumping into the left ventricle. Atrial volume depends on inflow from PTX and outflow into the ventricle via MV. Resistance to blood returning from peripheral circulation R_v represents systemic venous resistance, while resistance to blood entering the left ventricle R_{mv} corresponds to mitral valve resistance [13].

During diastole, when P_{lv} decreases, the mitral valve opens, allowing the atrium to empty. However, increased mitral valve resistance or ventricular dysfunction can impair ventricular filling, reducing cardiac output and leading to pulmonary hypertension [13].

3.2 Integration with Artificial Neural Networks

Considering the complexity of cardiovascular function and interpatient variability in hypertension, an artificial neural network is integrated to enhance model performance in terms of mathematical model accuracy and real-time adaptability in management methods. This work has not yet determined the specific neural network type. However, different architectures will be explored to maximize blood pressure prediction and system response to representative physiological variations [14].

A neural network was trained to adapt parameters of Olufsen's model, including structural parameter elements related to vascular resistance R, arterial capacitance C, and blood flow inertia L. These parameters can be individualized, changing with age, arterial stiffness, or autonomic response. Consequently,

relying solely on a static model built with differential equations may not be sufficient to describe hypertension dynamics accurately [16].

While the specific architectural design may exhibit slight variations depending on the type of neural network, it is generally understood that input variables such as previous blood pressure readings, heart rate, and other clinical parameters are essential. These data facilitate the neural network's capacity to personalize Olufsen's model by adjusting its parameters, thereby producing enhanced flow predictions for each patient, tailored to their unique conditions [16].

During neural network training, the error function will be defined based on minimizing differences between experimentally obtained blood pressure values and those predicted by the hybrid model. This difference will be calculated using a mean squared error (MSE) metric [17].

$$\mathcal{L} = \sum_{i=1}^{N} (P_i - \hat{P}_i)^2 \tag{12}$$

Where P_i represents measured blood pressure, and $\hat{P}_i$ denotes blood pressure predicted by the system. Adjusting this multiple-input multiple-output (MIMO) process will refine the model, enhancing prediction accuracy and simulating physiological and pathological scenarios. These neural networks will be tested in different configurations:

- multilayer perceptron (MLP)
- recurrent neural network (RNN)
- convolutional neural network (CNN)
- deep neural network

during upcoming development stages.

Selection of the final architecture will be selected based on each model's performance in accuracy, stability, and generalization in blood pressure prediction [17]. Recent studies suggest that integrating classical mathematical methods with deep learning can help identify non-invasive patterns in blood pressure oscillations and improve health data mining to continuously monitor patients with hypertension [18].

3.3 MATLAB® Implementation

For simulation of a digital twin for hypertension, a model was developed based on 11 differential equations describing blood pressure evolution and volume changes across different compartments of cardiovascular circulation. MATLAB® was implemented using the ode45 integrator, which is suitable for solving first-order ordinary differential equation systems. The following section details the implementation process and analyzes the behaviour of the obtained results. [13]

Cardiovascular model was built following the mathematical formulation of Olufsen et al. (2005), incorporating physiological parameters that represent blood flow dynamics across different arterial and venous segments. Initial conditions were defined based on systemic blood pressure values, venous pressure, and volumes in the left ventricle and left atrium. [13]

Definition of Model and Physiological Parameters: This cardiovascular model presented in this study was developed based on a compartmental representation of arterial and venous circulation, divided into multiple physiological compartments (aorta, upper and lower arteries, capillary bed, and veins) [15]. Physiologically realistic values were assigned to initial conditions for arterial and venous pressures and volumes in the left ventricle and left atrium. Simulated parameters include vascular resistance, which represents hydrostatic opposition during blood flow through each compartment, and arterial and venous capacitance, which define the distensibility of vascular walls and the blood reservoir capacity itself. These parameters work together to establish cardiovascular load and simulate resting behaviour by relating pressures and volumes as a function of time [13].

Solving differential equations: Ode45, a differential equation integrator suitable for biological dynamic systems [12], was used to calculate the evolution of pressures and volumes over time. This method provided approximate solutions for each differential equation, illustrating how pressures in different compartments interact in response to changes in blood flow. Each differential equation describes how pressure in a compartment is influenced by inflows and outflows, which are regulated by vascular resistances. Similarly, equations for ventricular and atrial volumes show how these change based on blood entry from venous circulation and ejection into arterial circulation [13].

Viewing Results: Results were graphically represented as an evolution of pressures and volumes over time. Initial changes in pressure and volume dynamics at the beginning of the simulation, preceding eventual stability in the generated graph, indicate an adjustment period. During this phase, blood flow redistributes in response to the model's initial conditions. Pressure and volume curves stabilise over time as the system reaches a steady state. This behaviour is attributed to the absence of a periodic source of variability (e.g., heartbeats), resulting in pressures and volumes converging to constant values when no external stimuli are applied to the model [13].

System Behavior Analysis: This behaviour reflects a resting cardiovascular system, where vessel pressure and circulating blood volume reach equilibrium without external forces acting upon them [16]. In reality, blood pressure in a living biological system is not constant but oscillates with each ventricular contraction [12]. For a more realistic Coronary Blood Flow (CBF) simulation, a pulsatile signal is required to mimic the cardiac cycle. This generates oscillations that replicate human physiological blood pressures and enhance simulation accuracy. Using an artificial neural network to adjust model characteristics based on accurate data dynamically further personalizes the model, enabling more precise blood pressure predictions for hypertensive patients [18].

In order to facilitate numerical resolution of the differential equations, a cardiovascular model was implemented in MATLAB, thus enabling researchers to

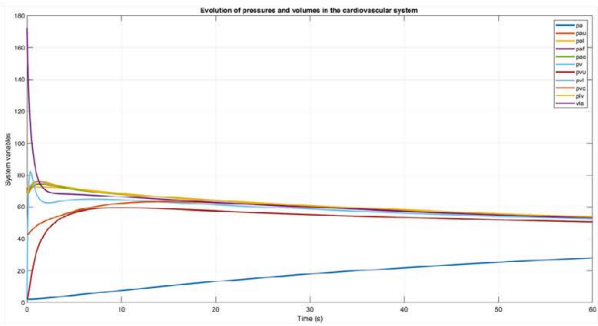

Fig. 1. Evolution of pressures and volumes in the cardiovascular system

obtain the evolution of pressures and volumes across different compartments. It was demonstrated by simulation results that, in the absence of external variations, the system tends to stabilise in a steady state. In a subsequent study, a periodic function representing heartbeats will be included, along with artificial neural networks, to enhance the model's accuracy and adaptability in clinical applications [17].

Evaluation and Validation Methods for Model: Ensuring the accuracy and applicability of a digital twin for hypertension requires evaluation and validation techniques that compare model predictions with clinical data or reference simulations. Olufsen's model, adapted with neural networks, will be validated at two levels: mathematical validation and physiological validation. Mathematical validation will focus on verifying that numerical solutions of the 11-equation differential system remain stable and consistent with known cardiovascular dynamics. Numerical stability of solutions obtained with ode45 will be analyzed, ensuring that the model does not exhibit divergences or non-physiological oscillations under minor variations in initial conditions or system parameters [12]. Additionally, model results will be compared with previously established solutions from studies on compartmental blood pressure models, ensuring that general simulation trends align with existing literature [13]. Model convergence will also be evaluated by analyzing whether arterial pressures and ventricular volumes reach a physiologically plausible steady state without external variations, indicating that the model accurately reflects cardiovascular homeostasis [14].

For physiological validation, model predictions will be compared with blood pressure recordings from prior simulations and databases of hypertensive patients. Evaluation will be performed using quantitative metrics such as mean squared error (MSE), which will measure differences between model-predicted blood pressures and reference values. A low MSE will indicate that the model accurately replicates expected values under normal and pathological conditions [15]. Pearson correlation coefficient R will also be analyzed to assess linearity between model predictions and reference data, ensuring that relationships be-

tween variables remain significant [16]. Additionally, Bland-Altman concordance index will be applied to evaluate error distribution and detect systematic biases in model predictions compared to actual data [17]. Since the model integrates an artificial neural network to adjust physiological parameters dynamically, assessing the impact of this integration on system accuracy is necessary. MSE values will be compared before and after neural network incorporation to determine whether parameter optimization enhances simulation accuracy [18]. Model adaptability to different hypertension profiles will also be evaluated, testing performance under varying vascular resistance and arterial stiffness to determine whether the neural network can adjust parameters in a physiologically coherent manner [13].

Computational efficiency of the system will be measured by comparing processing times for each simulation before and after artificial intelligence integration, ensuring feasibility for real-time applications or clinical simulations [14]. In order to assess model robustness under different clinical conditions, various pathological scenarios will be simulated, and system responses analyzed. Primary hypertension will be represented by increasing peripheral resistance, observing whether the model predicts a sustained rise in systolic and diastolic blood pressure, as seen in hypertensive patients [15]. Model behaviour under induced hypotension will also be analyzed by reducing vascular resistance and ventricular contractility to determine whether pressure drops similar to those observed in orthostatic hypotension episodes are simulated [16]. Additionally, administration of antihypertensive drugs, such as calcium channel blockers and vasodilators, will be simulated, verifying whether the model responds with a progressive reduction in blood pressure consistent with pharmacological effects reported in literature [17].

A digital twin for hypertension will be validated at multiple levels, ensuring that the model remains accurate and physiologically realistic. Integration with neural networks will be evaluated using quantitative metrics, determining its impact on prediction error reduction and system adaptability to different hypertension profiles. These processes will improve model reliability and potential application in treatment simulation and personalization for hypertensive patients.

4 Expected Results

A digital twin for hypertension promises to fundamentally transform how this condition is evaluated and managed by simulating blood pressure and cardiovascular system responses to various physiological and therapeutic scenarios. In this developed model, hypertension progression in patients is expected to be better predicted, enabling risk factor identification and personalized treatment [14]. An implemented digital twin has a significant advantage over static numerical modelling by dynamically adjusting vascular resistance, arterial capacitance, and blood flow inertia. These parameters change dynamically using artificial intelligence to reflect an individual's physiological state [15]. This approach captures

real-time variability and predicts responses to stimuli and treatments, unlike traditional blood pressure monitoring methods that rely primarily on discrete measurements and static models [16]. With integration into artificial neural networks, this model will adapt to specific data, providing more accurate simulations and representing a significant advancement over traditional compartmental models used in cardiovascular studies [17].

From a clinical perspective, the next step in utilizing a digital twin would be to improve risk stratification in hypertensive patients, aiding in more informed decisions regarding lifestyle modifications or pharmacological therapy adjustments [12]. Additionally, this tool supports research on therapeutic interventions by allowing evaluation of antihypertensive drug effects and blood pressure control strategies without requiring invasive procedures [13]. A digital twin would offer more predictive and adaptive capabilities than traditional hypertension measurements, such as ambulatory blood pressure monitoring (ABPM) or office-based sphygmomanometers [18]. Conventional methods rely on discrete readings that fail to capture blood pressure variability throughout the day accurately. However, differential equations and a machine learning framework embedded in this model will provide continuous and personalized estimates of cardiovascular dynamics [14]. This represents a crucial advantage, particularly in blood pressure management, where variability is key in cardiovascular event risk [16]. Furthermore, this proposed approach is unique in integrating artificial neural networks, allowing system parameter optimization and improving predictive accuracy compared to other compartmental mathematical models found in literature [13]. Unlike traditional models that require manual adjustments of physiological parameters to simulate variable pathological states, a digital twin can autonomously learn from clinical data, making it more adaptable for research and clinical environments [17].

Therefore, a digital twin for hypertension is envisioned as a far more precise, flexible, and predictive approach for evaluating this disease. It would enable continuous blood pressure monitoring and optimize therapy beyond classical methods [15]. This advancement could significantly impact clinical practice and hypertension research, enhancing patient quality of life and increasing efficiency in medical decision-making [18].

5 Discussion

Expected outcomes of a digital twin for hypertension suggest that this approach will provide a more precise and dynamic representation of blood pressure compared to traditional models, enabling a more detailed assessment of disease progression and personalized treatment strategies. The model's ability to dynamically adjust its parameters through artificial neural networks makes it an innovative tool with the potential to enhance hypertension behaviour prediction across different clinical scenarios. By considering interactions between vascular resistance, arterial capacitance, and blood flow inertia, this model will analyze the current state of cardiovascular function and anticipate physiological changes

under various conditions and treatments [12]. Despite its advantages, this study presents limitations that must be addressed in future research. One of the main limitations is the need for extensive and high-quality clinical data to train the artificial neural network, which may pose a challenge regarding availability and variability in physiological records. Model accuracy will largely depend on the diversity and quality of data used for calibration, making it essential to have representative databases that include patients with different hypertension levels and associated cardiovascular conditions [13]. Additionally, the current model does not incorporate a pulsatile source to represent heartbeats realistically. This may limit its applicability in studies where blood pressure variability throughout the cardiac cycle is a key factor. To improve cardiovascular dynamics representation, integrating a periodic signal that reflects pressure changes induced by left ventricular activity would be recommended [14].

Another significant limitation is computational complexity when integrating the model with artificial neural networks. While automatic parameter adjustment improves simulation accuracy, it also increases processing time and computational requirements, potentially complicating implementation in clinical settings where rapid and efficient responses are needed. Optimizing the model for real-time execution remains a challenge, and future research should focus on enhancing computational efficiency without compromising prediction accuracy [15]. Implications for future research span various aspects of cardiovascular modelling and artificial intelligence applications in healthcare. First, integrating a digital twin with continuous blood pressure monitoring devices could enable more precise and real-time calibration, improving its ability to adapt to patient profiles. This implementation would facilitate model validation in real clinical scenarios by comparing predictions with real-time measurements from wearable sensors or implantable devices [16]. Additionally, future studies could explore model applications in evaluating antihypertensive drug responses, analyzing how different medication classes influence vascular resistance and blood flow dynamics. Using a digital twin as a simulation tool for treatment personalization could lead to more effective therapeutic strategies and reduce the need for dosage adjustments through a mathematically driven predictive approach [17]. Another relevant research avenue involves improving the neural network architecture used for parameter adjustment. Exploring more advanced models, such as recurrent neural networks or hybrid frameworks combining deep learning with equation-based optimization, could further enhance prediction accuracy and computational efficiency [18].

Consequently, a digital twin for hypertension signifies a substantial advancement in cardiovascular system modelling and treatment personalisation. Nevertheless, challenges that necessitate future research persist. Enhancing data quality, optimising computational performance, and integrating with continuous monitoring devices will be pivotal for consolidating its clinical and research applications. Implementing this model in prospective studies could yield valuable insights into hypertension progression and its response to various therapeutic

strategies, thereby contributing to developing more effective disease management approaches.

6 Conclusions

This study integrates differential equations and artificial neural networks to develop a Digital Twin for Hypertension, marking a new cardiovascular simulation and prediction era. Extending Olufsen's framework, the model accurately represents interactions between vascular resistance, arterial capacitance, and blood flow inertia across cardiovascular compartments. Dynamically adjusting parameters via artificial intelligence enhances adaptability to individual conditions, improving personalized hypertension treatment [12].

A key contribution is its ability to provide real-time, informative blood pressure estimations compared to traditional static models. Unlike conventional monitoring methods, this approach integrates discrete measurements with continuous pressure evolution, offering superior predictive capabilities for cardiovascular risk stratification and clinical decision-making [14]. Additionally, its application in electronic and communication systems advances intelligent physiological modelling, fostering the development of software-based medical devices that integrate with monitoring sensors for continuous blood pressure estimation. AI-driven autonomous systems could further aid early diagnosis and treatment of cardiovascular diseases.

Future research should focus on integrating the digital twin with continuous monitoring devices for real-time validation, refining physiological parameterization, and incorporating pulsatile sources to improve simulation accuracy. Optimizing computational performance will enable its use in clinical environments requiring rapid decision-making [17]. Furthermore, evaluating its response to antihypertensive treatments and non-pharmacological interventions will provide deeper insights into cardiovascular dynamics, with prospective clinical validation confirming its potential in medical practice [18].

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