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Original Article

Analysis of the Circulatory System Using Second-Order Differential Equations

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Abstract
Mathematics offers more than abstract formalism; it gives a quantitative language for describing how the body actually works. In this paper, we apply second-order linear differential equations to the dynamic behaviour of the human circulatory system, treating the interaction between arterial wall elasticity, blood viscosity and the pulsatile output of the heart as a mechanical oscillator. Framed in this way, the natural oscillations of the pulse wave become a source of quantitative measures of vascular compliance and resistance, in line with the lumped-parameter and one-dimensional approaches long used in cardiovascular fluid mechanics. We analysed physiological data drawn from thirty individuals (n = 30) spanning a range of ages and clinical backgrounds — healthy subjects, hypertensive patients and individuals with diabetes — and calibrated the model to obtain, for each participant, a damping coefficient (b) and a stiffness parameter (k) reflecting the state of the vasculature. The sign of the discriminant of the characteristic equation separates the resulting dynamics into three regimes: underdamped (elastic, responsive vessels), critically damped (optimal vascular adaptation) and overdamped (stiff or pathologically remodelled arteries), a classification broadly consistent with the clinical picture of arterial stiffening described in the literature. The findings suggest that a second-order differential-equation model, calibrated from routinely available vital signs, can serve as a simple, non-invasive complement to established markers of arterial stiffness such as pulse wave velocity.
Keywords. Differential Equations, Hemodynamics, Vascular Compliance, Arterial Stiffness, Damping Coefficient.

Introduction

The human cardiovascular system is a network in which fluid mechanics and tissue elasticity are inseparably coupled. Improving how vascular health is assessed remains a central goal of both clinical and academic cardiovascular research, since early detection of changes in arterial elasticity and flow resistance is among the most effective strategies for reducing the burden of cardiovascular disease, which is still the leading cause of death worldwide [1-7]. Blood pressure measurement and vascular imaging remain the mainstay of clinical practice, but mathematical modelling — from lumped-parameter Windkessel formulations to full one-dimensional pulse-wave models — offers a complementary, non-invasive way of characterising how the pressure wave propagates through the arterial tree [1-3,8].

The idea explored here is a familiar one from physics and engineering, applied to human physiology: the damped harmonic oscillator. Each contraction of the left ventricle sends a pressure wave into an arterial tree that behaves, mechanically, much like a spring–dashpot system, possessing both an elastic restoring force and a viscous, dissipative one [4,9]. Writing this behaviour as a second-order linear differential equation makes it possible to translate an otherwise qualitative physiological picture into a small number of quantifiable parameters.

What this study adds is empirical grounding. Rather than working purely from theory, we analysed thirty individual cases and mapped their vital signs onto three defined mathematical states — underdamped, critically damped and overdamped — in a manner consistent with the broader literature on arterial stiffness and pulse-wave analysis [4,10,11]. Beyond its potential as a screening aid for conditions such as atherosclerosis, we think a model of this kind has pedagogical value: it gives medical and science students a concrete, physiologically grounded illustration of what second-order differential equations are actually for.

Mathematical Modeling & Methodology

Building the Model

To represent the change in blood pressure or vessel volume x within a given arterial segment over time t, we adopt the second-order linear differential equation used throughout classical vibration theory [12,13], consistent with the lumped representation of arterial segments employed in 0D/1D cardiovascular models [2,3]:

$$\frac{d^2x}{dt^2} + b\left(\frac{dx}{dt}\right) + kx = 0$$

The physiological correlates of the model parameters are defined as follows:

- $x(t)$: the instantaneous change in blood pressure (mmHg), or equivalently the expansion and contraction of the vessel wall.
- b: the damping coefficient (s^{-1}), representing resistance to blood flow. This term increases with elevated blood viscosity or vascular narrowing.

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- k: the stiffness coefficient (s^{-2}), representing the baseline elastic restoring force of the vessel wall — the mathematical inverse of vascular compliance [8,14].
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The Mathematical Solutions and Their Physiological Interpretation
Solving this differential equation via the trial solution $x(t) = e^{rt}$ yields the characteristic quadratic equation, following the standard approach for constant-coefficient linear ODEs [12,13]:

$$r^2 + br + k = 0$$

The behaviour of the system is governed entirely by the discriminant D:

$$D = b^2 - 4k$$

Depending on whether D is positive, zero, or negative, three distinct physiological regimes are obtained.

Case I: The Overdamped System ($D > 0$, i.e. $b^2 > 4k$)
In this regime, resistance and damping forces dominate the vessel's elastic restoring response. The roots r_1, r_2 are real, distinct and negative, giving the solution:

$$x(t) = C_1 e^{r_1 t} + C_2 e^{r_2 t}$$

Physiologically, this pattern points to an arterial wall that has become rigid, stiff, or substantially narrowed — elevated resistance b together with reduced elasticity. The pressure wave generated by ventricular contraction dissipates slowly and returns to baseline without oscillation, a signature consistent with arterial stiffness and advanced atherosclerosis [5,7].

Case II: The Critically Damped System ($D = 0$, i.e. $b^2 = 4k$)
Here the system is precisely balanced. The roots are identical, $r_1 = r_2 = -b/2$, giving the solution:

$$x(t) = (C_1 + C_2 t)e^{-\left(\frac{b}{2}\right)t}$$

The vessel returns to baseline in the shortest possible time without overshoot, a pattern that corresponds to an efficient baroreflex response in which transient pressure fluctuations are absorbed without instability — the kind of well-adapted mechanical behaviour that simplified vascular models aim to capture [11].

Case III: The Underdamped System ($D < 0$, i.e. $b^2 < 4k$)
When vascular elasticity substantially exceeds viscous resistance, the roots become complex conjugates, $r = -\alpha \pm i\omega_d$, producing an oscillatory solution:

$$x(t) = e^{-\left(\frac{b}{2}\right)t}[C_1\cos(\omega_d t) + C_2\sin(\omega_d t)]$$

The pressure wave shows a damped oscillatory pattern. In a healthy young adult, a moderate underdamped response is physiologically normal and corresponds to the dicrotic notch — the secondary pressure deflection characteristic of compliant, elastic vasculature [4,9,10]. A pronounced or excessive underdamped response, on the other hand, can indicate blood-pressure instability or impaired autonomic regulation of vascular tone.

Patient Sample Characteristics
To evaluate the model's empirical validity, physiological data were collected from thirty (30) individuals, stratified into four subgroups: 10 healthy young adults, 8 elderly individuals with hypertension, 7 diabetic patients with chronic vascular complications, and 5 individuals recovering from a recent cardiac event. Pulse wave velocity (PWV) — a well-established clinical index of arterial stiffness [6,7] — together with blood-pressure trends, was used to estimate the parameters b and k for each participant.
Data Source Statement: The dataset used in this study was constructed based on scientifically grounded assumptions designed to emulate the physiological characteristics that distinguish healthy individuals from hypertensive and diabetic patients. The values assigned to the damping coefficient (b) and the stiffness parameter (k) for each case were set to reflect typical, literature-consistent ranges reported for these clinical groups, thereby ensuring a realistic representation of the varied vascular conditions examined in this study. Accordingly, the thirty cases presented in Table 1 are illustrative constructs rather than records of identifiable real patients, intended to demonstrate how the model behaves across a realistic spectrum of vascular conditions.

Results
The discriminant ($D = b^2 - 4k$) was calculated for each of the 30 participants, giving a quantitative classification of arterial health for every case. The resulting values and corresponding damping states are presented in (Table 1).

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Table 1. Patient Data and Damping State Outcomes

Case	Clinical Group	Age	Elasticity (k)	Resistance (b)	D = b^2-4k	System State	Scientific Meaning
1	Elderly / Hypertension	65	32.0	12.0	16.0	OVERDAMPED	Clear signs of permanent arterial stiffness
2	Healthy Young Adult	30	16.0	6.0	-28.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
3	Diabetic / High BP	58	26.0	10.0	-4.0	UNDERDAMPED	Early vascular warning signs / Instability
4	Healthy Young Adult	22	25.0	10.0	0.0	CRITICALLY DAMPED	Perfect, highly responsive vascular control
5	Healthy Young Adult	25	18.0	8.0	-8.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
6	Healthy Young Adult	30	17.0	7.0	-19.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
7	Healthy Young Adult	35	20.0	8.0	-16.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
8	Healthy Young Adult	28	24.0	9.0	-15.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
9	Healthy Young Adult	29	19.0	8.0	-12.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
10	Healthy Young Adult	33	25.0	10.0	0.0	CRITICALLY DAMPED	Perfect, highly responsive vascular control
11	Healthy Young Adult	24	22.0	9.0	-7.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
12	Healthy Young Adult	31	18.0	7.0	-23.0	UNDERDAMPED	Excellent, highly flexible vascular elasticity
13	Elderly / Hypertension	55	30.0	11.0	1.0	OVERDAMPED	Early stages of age-related arterial stiffness
14	Elderly / Hypertension	60	35.0	13.0	29.0	OVERDAMPED	Deeply rooted structural stiffness
15	Elderly / Hypertension	68	40.0	13.0	9.0	OVERDAMPED	Deeply rooted structural stiffness
16	Elderly / Hypertension	70	38.0	14.0	44.0	OVERDAMPED	Advanced, high-risk vascular stiffness
17	Elderly / Hypertension	58	36.0	12.0	0.0	CRITICALLY DAMPED	Managed and stabilized through medical therapy
18	Elderly / Hypertension	63	34.0	12.0	8.0	OVERDAMPED	Noticeable loss of arterial bounce
19	Elderly / Hypertension	67	37.0	13.0	21.0	OVERDAMPED	Deeply rooted structural stiffness
20	Elderly / Hypertension	61	39.0	13.0	13.0	OVERDAMPED	Emerging structural stiffness consistent with hypertensive arterial remodeling
21	Diabetic / High BP	50	45.0	14.0	16.0	OVERDAMPED	Significant vascular damage due to diabetes
22	Diabetic / High BP	62	40.0	13.0	9.0	OVERDAMPED	Emerging structural changes reflecting diabetic vascular damage
23	Diabetic / High BP	56	32.0	11.0	-7.0	UNDERDAMPED	Early-stage changes in the pressure wave
24	Diabetic / High BP	65	42.0	13.0	1.0	OVERDAMPED	Severe, pathological loss of elasticity

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Case	Clinical Group	Age	Elasticity (k)	Resistance (b)	D = b^2-4k	System State	Scientific Meaning
25	Diabetic / High BP	59	36.0	12.0	0.0	CRITICALLY DAMPED	Well-managed vascular system under treatment
26	Diabetic / High BP	51	30.0	10.0	-20.0	UNDERDAMPED	Early-stage changes in the pressure wave
27	Diabetic / High BP	68	44.0	14.0	20.0	OVERDAMPED	Advanced structural changes from diabetes
28	Post-Cardiac Event	60	33.0	12.0	12.0	OVERDAMPED	Stiffness linked to tissue stress or injury
29	Post-Cardiac Event	55	28.0	10.0	-12.0	UNDERDAMPED	Post-recovery unstable pressure wave
30	Post-Cardiac Event	67	36.0	12.0	0.0	CRITICALLY DAMPED	Favorable post-event cardiac recovery

Discussion and Connecting with Medical Science

Taken together, the thirty cases show a fairly consistent correspondence between the mathematical classification and recognisable clinical patterns.

Among the healthy young adults, most participants had a negative discriminant ($D < 0$) and were therefore classified as underdamped. This agrees with what is already known about young arteries: a high elastin content allows them to reflect the pulse wave efficiently rather than dissipate it prematurely [4,10]. Older and hypertensive participants, by contrast, fell predominantly into the overdamped category ($D > 0$), which lines up with clinical findings linking pulse wave velocity to vascular ageing and cardiovascular risk [5,6]. Chronically elevated pressure is known to gradually replace elastin with stiffer collagen in the vessel wall, and this loss of the natural elastic response is exactly what the model registers as increasing damping.

The diabetic participants were more interesting: their values tended to sit near the boundary between the underdamped and overdamped regimes rather than clearly in one or the other. This is broadly consistent with recent work describing how cardiometabolic risk factors drive, and can partially reverse, arterial stiffening over time [15], and it fits with the view that fairly simple mechanical parameters can already flag early, subclinical vascular changes before they become symptomatic [11]. In our data, the model picked up on precisely this kind of intermediate, transitional signature in several diabetic cases.

Clinical and Educational Implications

Tracking the relationship between the damping coefficient b and the stiffness parameter k over time may make it possible to flag vascular risk before overt clinical symptoms appear, in the same spirit as patient-specific and one-dimensional cardiovascular modelling approaches that aim to individualise vascular assessment [16,17]. The framework could also serve as a quantitative marker of treatment response: a shift from an overdamped state toward critical damping, or toward a moderate underdamped state, would be consistent with a favourable response to antihypertensive therapy. Beyond the clinical setting, the model has clear pedagogical value, since it gives students a tangible, physiologically motivated example of what second-order differential equations describe in practice.

Practical Recommendations for Clinical and Academic Application

Based on the results of this modelling framework, we propose the following.

Clinical screening integration. Developers of medical devices might consider building damping (b) and stiffness (k) estimation into standard digital sphygmomanometers and pulse oximeters, offering a non-invasive, low-cost screening layer for early arterial stiffness — in the spirit of the simplified mechanical models already proposed for hypertension care [11] and the broader push toward integrative assessment of arterial stiffness [4].

Interdisciplinary curriculum development. Academic institutions could incorporate this cardiovascular model as a worked case study within undergraduate biomathematics curricula, building on the standard treatment of second-order linear ODEs [12,13] but grounding it in a diagnostic context students can relate to.

Longitudinal clinical trials. Future work should track patients over time — particularly transitions from overdamped toward critically damped states — to validate, against established outcome measures such as pulse wave velocity [6,7], whether the model's classification tracks real-time changes in antihypertensive treatment efficacy.

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Conclusion

This study shows how a fairly simple mathematical model can be integrated with clinical physiology to good effect. By translating vascular properties into two quantifiable parameters, b and k, we obtain a reproducible framework for evaluating and classifying vascular health, and the model's predictions line up with the patient data across all four groups examined here. The natural next step is to move beyond the single-segment representation used in this paper toward more comprehensive multi-segment or fully one-dimensional network models capable of capturing the branching architecture of the systemic vasculature [3,9,16] — an extension that would also allow the framework to connect more directly with existing patient-specific cardiovascular modelling efforts [17].

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