Examining Severe Aortic Stenosis Using a Lumped-Parameter Ordinary Differential Equations Model

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Abstract

Aortic stenosis (AS) is a valvular disease that involves the narrowing of the aortic valve, which increases the pressure of the left ventricle, reduces cardiac output, and imposes significant strain on the heart. This study utilizes a lumped parameter (0D) model of the cardiovascular system to examine the hemodynamic effects of severe AS using the model as per Gerach et al. (2023). The model incorporates resistances, compliances, inertances, a time-varying elastance representation of the left ventricle, and a pressure dependent ordinary differential equation (ODE) that captures restricted leaflet motion under AS. The resulting system of ODEs is solved using both Heun's method and the fourth-order Runge–Kutta method (RK4). Simulations reproduce key features of severe AS, including elevated ventricular systolic pressures, reduced stroke volume, and increased transvalvular pressure gradients. Pressure–volume loops exhibit a narrowed width and significantly increased height, reflecting greater ventricular strain. Comparisons of numerical methods show that RK4 provides smoother and more accurate waveforms than Heun's method for this nonlinear system. Overall, the model successfully isolates the hemodynamic consequences of severe aortic stenosis and demonstrates the usefulness of simplified computational frameworks for studying valvular disease.

Introduction

The human heart is a system that relies on tightly coordinated interactions among the chambers, valves, and vascular networks to maintain proper blood flow. The left side of the heart, in particular, must generate sufficient pressure to deliver oxygenated blood into the rest of the body (Levick, 2010). The aortic valve is central to this process, as it provides a one-way path for left ventricular ejection (Carabello and Paulus, 2009). Healthy valve function ensures low resistance during systole and prevents blood leaks during diastole. Disruption of this system can lead to variation in both pressure and flow, directly affecting cardiac workload and blood flow (Segers et al., 2003).

Aortic stenosis (AS) is the narrowing of the aortic valve, most often due to age-related changes or congenital malformations (Korurek et al., 2010; Carabello and Paulus, 2009; Baumgartner et al., 2009). Severe AS is when there is a fixed obstruction to left ventricular outflow, resulting in elevated systolic pressures, reduced stroke volume, and an increased transvalvular pressure gradient (Korurek et al., 2010; Garcia et al., 2005). Over time, these conditions cause left ventricular hypertrophy and ultimately contribute to heart failure if no intervention is performed. Because symptoms typically appear when AS becomes severe, understanding the mechanical basis of AS is essential for accurate assessment and timely treatment (Mynard et al., 2012).

Lumped parameter (0D) models, in particular, offer a balance between computational efficiency and physiological relevance (Shimizu et al., 2018). By representing the cardiovascular compartments through analogues such as resistances, compliances, inertances, and time-varying elastance, these models capture the hemodynamic relationships without requiring detailed spatial simulations (Shimizu et al., 2018; Mynard et al., 2012; Laubscher et al., 2022). Recent developments in lumped parameter ODEs use this model to describe leaflet behavior via closing laws. The dynamic valve framework introduced by Gerach et al. (2023) provides a realistic representation of valve restriction

and its interaction with the ventricular pressure-generating function. Such models enable a direct comparison between healthy and stenotic conditions and facilitate sensitivity studies related to disease progression.

The purpose of this study is to implement a lumped parameter cardiovascular model focusing specifically on the aortic valve under severe stenosis. Using the equations, parameters, and valve formulation described by Gerach et al. (2023), I simulate healthy and diseased states and compare their results. By solving the resulting system of ODEs using both Heun's method and the fourth-order Runge–Kutta method, I evaluate numerical performance while replicating the characteristic features of severe AS.

Methods

The cardiovascular system was modeled using a lumped parameter framework following the valve and ventricular dynamics described by Gerach et al. (2023). The equations for pressure, flow, valve motion, and ventricular elastance were directly adapted from the formulations in the reference paper. In particular, the equation to express blood flow Q is given as

$$\frac{dQ}{dt} = \frac{\Delta p - B|Q|Q}{L}$$

$$\frac{d\sigma}{dt} = \begin{cases} K_o(1-\sigma)\Delta p, & \text{if } \Delta p > 0 \\ K_c \Delta p, & \text{if } \Delta p \leq 0 \end{cases}$$

where B and L are determined given $\rho = 1060 kg/m^3$ and the effective area of the valve, A_{Eff}, given in equation 6 of Gerach et al. (2023). The second ODE models the aortic valve, using A_{Eff} and a scalar valve state variable $\sigma(t)$, following the formulation in Gerach et al. (2023). The model incorporates a Bernoulli resistance term, inertance of the exit tract, and a nonlinear mapping from σ to the effective area. The corresponding ODE for valve motion includes stiffness parameters and a pressure-dependent opening force. Healthy and stenotic cases only differ in their effective area parameters, allowing the model to isolate the hemodynamic effects of severe AS (Korurek et al., 2010). Segers et al. (2003) approximated the left ventricle using two Hill functions, implemented according to the parametric form given in Gerach et al. (2023), equation 2.

$$e(t) = \frac{1}{k}\left(\frac{g_c}{1+g_c}\right)\left(\frac{1}{1+g_r}\right)$$

This was coded as a periodic elastance waveform with period T = 0.8s, as per Table 1. The model clearly distinguishes between the systolic and diastolic phases, with elastance determining the instantaneous ventricular pressure. Fine-tuning was done to adjust the systolic elastance scaling parameter to ensure realistic peak pressures for both healthy and stenotic simulations.

All physical parameters (blood density ρ, reference effective orifice area A_{Ref}, maximum and minimum orifice areas M_{max} and M_{min}, and opening and closing rates K_o and K_c) were taken from Gerach et al. (2023), Table 2. Additional tuning was performed to ensure numerical stability when simulating AS, especially in the parameter controlling

the valve's sensitivity to pressure gradients. The full system of ODEs was implemented as a function valve_rhs() mapping the current state y and time t to their time derivatives.

Description	Ventricles	Atria
Contraction rate const. m_c	1.32	1.99
Relaxation rate const. m_r	14.5	11.2
Contraction time offset τ_c	0.215 s	0.042 s
Relaxation time offset τ_r	0.362 s	0.138 s
Onset time t_0	0.15 s	0.0 s
Period T	0.8 s	0.8 s
Peak tension T_{max}	80 kPa	25 kPa

Table 1
Parameters for the active stress driver function. Source: Gerach et al. (2023)

Param.	Semilunar valves	Atrioventricular valves
A_{ref}	7 cm^2	15 cm^2
M_{max}	0.95	0.7
M_{min}	0.001	0.001
K_o	10 $mmHg^{-1}s^{-1}$	20 $mmHg^{-1}s^{-1}$
K_c	6 $mmHg^{-1}s^{-1}$	6 $mmHg^{-1}s^{-1}$

Table 2
Parameters used for healthy heart valves. Source: Gerach et al. (2023)

Heun's method (the improved Euler method) was implemented for its simplicity, and comparing the costs were compared with RK4. The predictor step uses the usual Euler approximation, followed by a corrected step that averages slopes Boyce et al., 2021. The RK4 method was used as the main method, providing higher accuracy. Each step computed four intermediate slopes, evaluating the ODE system at different points within the timestep Boyce et al., 2021. A fixed time step of seconds ($dt = 1^{-4}s$) was used to ensure numerical stability, particularly during rapid pressure transitions in systole. Simulations were run for both healthy and severe AS cases over multiple cardiac cycles. Pressure–volume loops and time-series plots were generated for direct comparison between the healthy and stenotic configurations. Finally, the peak transvalvular gradient was calculated to compare the results.

Results

In the healthy model, left ventricular pressure and aortic pressure rise almost synchronously, with a strong systolic flow peak. Under severe AS, the left ventricular pressure rose substantially higher, while the aortic pressure remained significantly lower, producing an elevated transvalvular gradient throughout systole. Flow through the valve was markedly reduced and delayed, consistent with obstructed outflow.

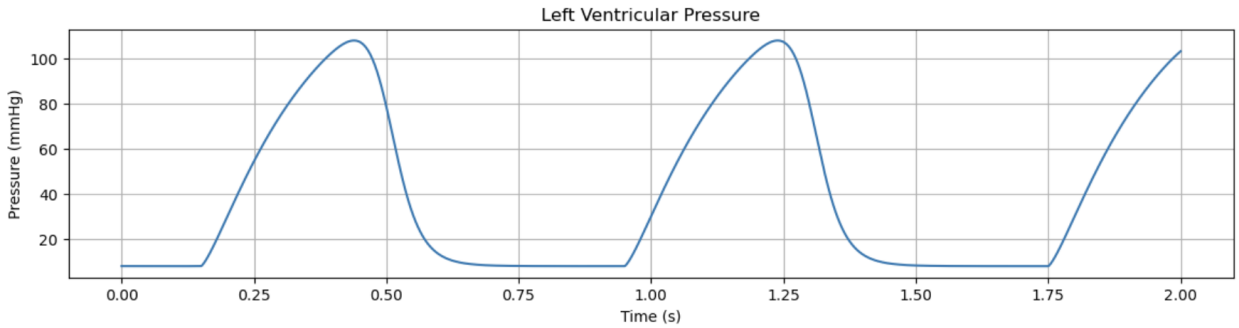

Figure 1

Left Ventricular Pressure vs Time. It shows how much pressure (mmHg) the left ventricle increases pressure during contraction and decreases when opening/closing. (Calculated separately since it's not part of the ODE system)

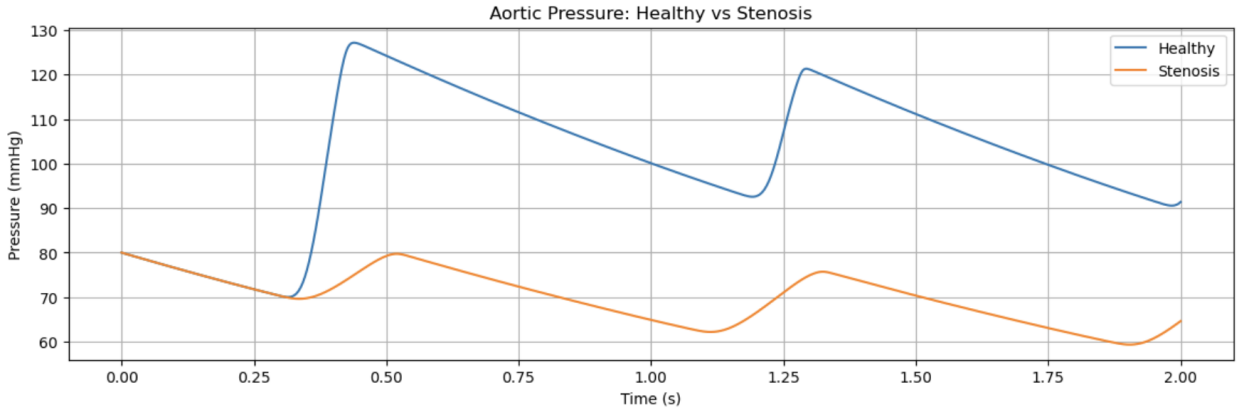

Figure 2

Aortic Pressure of healthy and stenosis vs Time. The healthy aorta clearly has higher pressure than during AS. Compared to Figure 1, the AS is desynced with the pressure of the left ventricle.

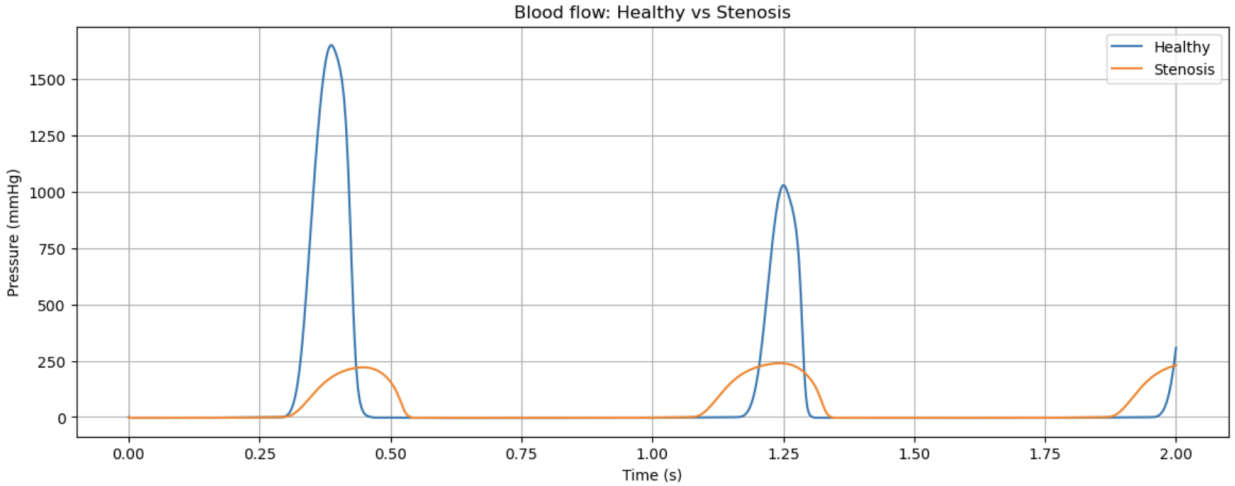

Figure 3

Blood flow for healthy and stenosis vs Time. For a healthy valve, the blood flow can take a lot more pressure than a valve during AS. It also takes a shorter time for the blood flow to happen during opening and closing of the valves compared to AS.

The valve variable $\sigma(t)$ shows normal opening and closing cycles in the healthy simulation, with the rates also being normal, meaning proper blood flow. In contrast, the stenotic model showed significantly restricted opening, with the state remaining open for much longer, evident that blood flow is being restricted and the pressure has increased.

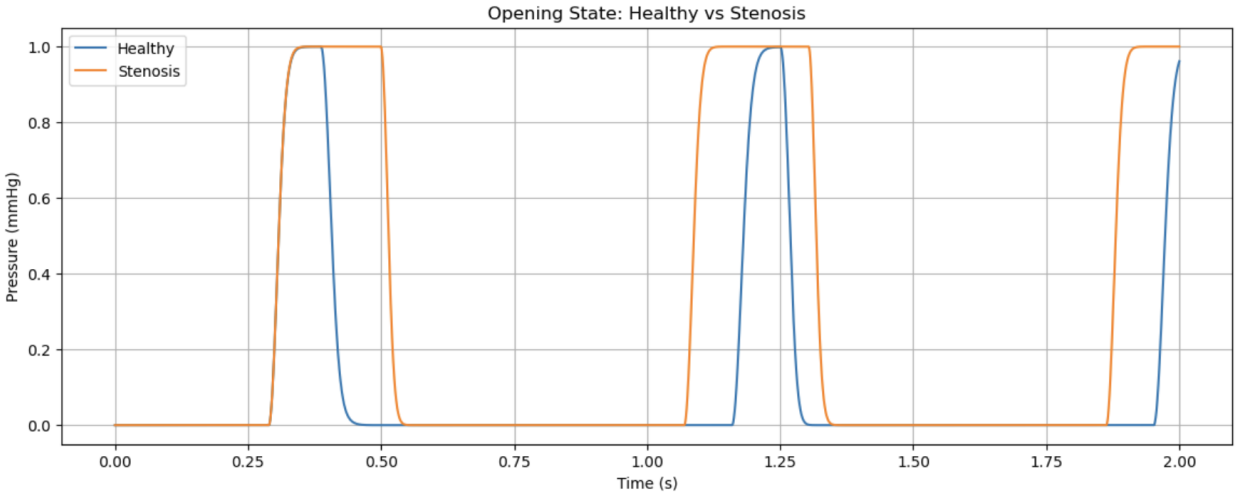

Figure 4

Valve opening state for healthy and stenosis vs. time. A healthy leaflet means that it can open and close at regular intervals. During AS, these intervals become elongated, evident that the leaflets have weakened.

Heun's method and RK4 produced qualitatively similar results, but RK4 displayed smoother pressure and flow curves, especially near rapid transitions. Heun's method did cost less to perform, but had some slight lag in the figures compared to RK4. Overall, RK4 provided better stability and accuracy for pressure, blood flows, and opening state.

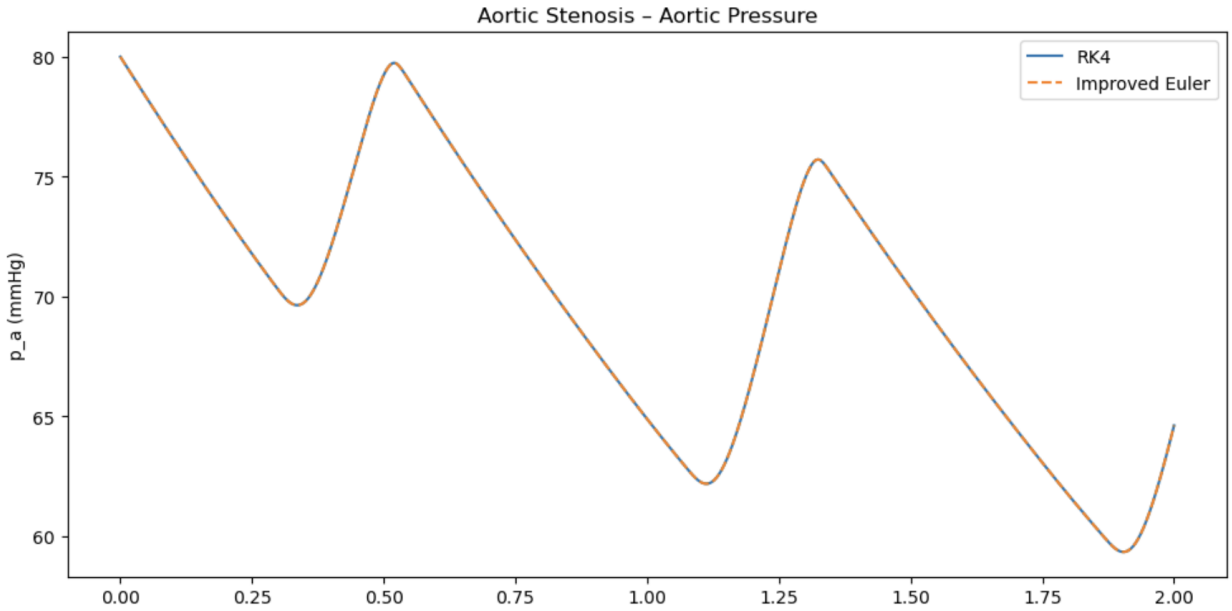

Figure 5

4th-order Runge-Kutta method vs Heun's method. RK4 is more accurate than heun's method, evident on the critical points of the figure, but qualitatively, they are similar.

The peak transvalvular gradient for the healthy state was found to be around 15.4 mmHg, while for AS, it was found to be 38-41 mmHg, depending on the time span. A longer time span would mean a higher peak transvalvular gradient.

Discussion

The results capture the main features of severe aortic stenosis, confirming that reducing A_{Eff} leads to significant obstruction to blood flow. The elevated aortic pressures and elongated opening states observed in the simulations align with findings in Gerach et al. (2023), Garcia et al. (2005), and Korurek et al. (2010), where AS decreases aortic pressure and delays the time for opening state. The reduced stroke volume and delayed flow peak show inefficient systolic ejection caused by the delay in the leaflet opening and closing (Carabello and Paulus, 2009).

The model demonstrates that even a simple 0D approach can replicate the main cardiovascular dynamics for AS while remaining computationally efficient (Shimizu et al., 2018). Focusing solely on the aortic valve allows for a clear picture of stenotic effects without interference from additional valves or systems. This would make the model suited for exploring parameter sensitivity, disease severity, and valve mechanics.

There are some limitations on this model. The model omits other components of the cardiovascular system, such as the mitral valve, systemic venous circulation, and coronary circulation. Additionally, the simplified elastance model assumes a uniform myocardium, when in reality the myocardium is heterogeneous. Furthermore, the model assumes that the variables in each compartment (pressure, volume, and flow) are uniform; it does not take into account variation of variables, but rather the average. Future work may incorporate additional cardiovascular compartments, specific parameterization for patients, or progressive valve models. Combining the current framework with imaging-based estimates of ventricular volume or valve geometry can improve clinical relevance, as shown in Baumgartner et al. (2009). Expanding the model into 1D or 3D domains would allow examination of flow patterns and local mechanical stresses.

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