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Effect of Left Ventricular Assist Device Pulsatility Mode and Cannula Angle on Aortic Blood Flow Characteristics, Blood Renewal Efficiency, Hemolysis, and Platelet Activation

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Abstract

Purpose: This study aimed to investigate the effects of left ventricular assist device (LVAD) pulsatility mode, outflow graft angle, and partial recovery of native cardiac function on aortic hemodynamics, blood renewal, and thrombosis risk.

Methods: A patient-specific normal aortic model implanted with an LVAD was established for computational fluid dynamics simulations. Different outflow-graft angles, support modes (continuous and pulsatile flow), and cardiac recovery conditions were investigated. Hemodynamic characteristics were evaluated using wall shear stress-related indices, blood washout behavior, hemolysis, and platelet activation metrics.

Results: Outflow-graft angle primarily determined the organization and impingement behavior of the LVAD-induced jet within the ascending aorta. Pulsatile support periodically reconstructed the flow field and alleviated persistent low-velocity regions observed under continuous-flow support, thereby improving blood washout characteristics. Progressive cardiac recovery enhanced large-scale flow mixing between native ventricular ejection and LVAD outflow and improved overall blood transport within the proximal aorta. However, recovery conditions also generated increasingly complex oscillatory flow structures near the aortic root because of intensified dual-source flow interaction. In addition, pulsatile support reduced sustained high-shear exposure and demonstrated lower hemolysis and platelet activation levels compared with continuous-flow conditions.

Conclusions: Aortic hemodynamics under LVAD support are governed by the coupled interaction between graft orientation, pulsatile support, and native cardiac recovery. Different support conditions exhibit competing effects on blood transport, wall shear distribution, and hemocompatibility. These findings may provide theoretical guidance for individualized LVAD implantation and support optimization.

Keywords: Left Ventricular Assist Device (LVAD); Aortic hemodynamics; Blood flow characteristics; Platelet activation; Thrombosis risk; Hemolysis.

1. Introduction

Heart failure (HF) remains a major global health burden, affecting approximately 64 million individuals worldwide, with 5-year mortality in end-stage populations exceeding 50% [1]. For patients with advanced HF, heart transplantation remains the gold-standard therapy; however, its application is severely limited by the persistent shortage of donor organs [2]. Consequently, left ventricular assist devices (LVADs) have become an essential mechanical circulatory support strategy, serving both as bridges to transplantation and as destination therapy for patients who are not transplant candidates [3,4].

The development of LVAD technology has progressively shifted from early pulsatile displacement pumps toward modern continuous-flow (CF) centrifugal devices. Although contemporary CF-LVADs substantially improve survival and durability, their non-physiological flow characteristics significantly alter cardiovascular hemodynamics and remain associated with complications including thrombosis, stroke, gastrointestinal bleeding, hemolysis, and aortic insufficiency [5-7]. The HeartMate 3 (HM3), a fully magnetically levitated centrifugal LVAD incorporating intrinsic artificial pulse modulation, represents a major advance in hemocompatibility and pump durability. The MOMENTUM-3 trial demonstrated significant reductions in pump thrombosis and stroke compared with previous-generation devices [8]. Nevertheless, systemic complications such as gastrointestinal bleeding and thrombo-inflammatory events remain prevalent despite improvements in pump design [9,10]. This discrepancy suggests that although contemporary LVADs have substantially reduced pump-internal blood trauma, they still fail to fully restore physiological aortic hemodynamics and pulsatile flow patterns, allowing pathological flow environments outside the pump to persist during long-term mechanical support.

Increasing evidence indicates that abnormal aortic hemodynamics may contribute substantially to these complications. Under physiological conditions, pulsatile ventricular ejection generates organized flow structures that promote blood transport, near-wall washout, and vascular homeostasis within the ascending aorta. After LVAD implantation, however, the high-momentum jet introduced through the outflow graft substantially modifies native flow organization, resulting in altered wall shear stress distributions, abnormal washout behavior, and disturbed flow pulsatility [11]. These hemodynamic alterations have been implicated in thrombus formation, platelet activation, endothelial dysfunction, and acquired von Willebrand factor degradation associated with gastrointestinal bleeding [12].

Among the factors influencing LVAD-supported aortic hemodynamics, outflow-graft orientation represent an important surgically modifiable parameter. Previous computational and experimental studies demonstrated that graft angle significantly affects jet impingement behavior, recirculation structures, blood residence characteristics, and regional shear-stress distribution within the ascending aorta [13,14]. Current ISHLT guidelines recommend an anastomosis angle of approximately 45–60° onto the lateral aspect of the proximal ascending aorta; however, recent studies suggest that the optimal graft configuration may vary depending on the hemodynamic metric evaluated and the interaction between graft geometry and patient-specific anatomy [12]. In addition, increasing attention has been directed toward the role of pulsatile support in restoring physiological flow characteristics. Recent studies demonstrated that LVAD speed modulation can substantially alter flow mixing, helicity distribution, viscous energy dissipation, and blood transport behavior within the aorta [15].

Besides graft configuration and pulsatile support, cardiac recovery during LVAD assistance may further alter the aortic flow environment. Clinical studies have demonstrated that prolonged ventricular unloading can promote reverse remodeling and partial recovery of native cardiac function in selected patients [16,17]. As native ventricular ejection progressively recovers, the aortic flow field gradually transitions from a predominantly pump-driven system toward a coupled dual-source flow environment governed by both LVAD outflow and native ventricular pulsation. However, most previous computational studies focused on fixed-support conditions and investigated graft angle or pulsatility independently, while the coupled effects of graft orientation, pulsatile support, and cardiac recovery on aortic hemodynamics remain poorly understood.

Therefore, the present study employed computational fluid dynamics (CFD) simulations in a patient-specific normal aortic model to systematically investigate the effects of different outflow-graft angles, support modes, and cardiac recovery conditions on aortic hemodynamics. Hemodynamic metrics including wall shear stress-related indices, blood washout characteristics, hemolysis, and platelet activation were analyzed to elucidate how graft orientation, pulsatile support, and ventricular recovery jointly reconstruct flow organization and hemocompatibility within the LVAD-supported aorta. This study aims to provide mechanistic insight for individualized LVAD implantation optimization and physiologically informed support strategies.

2. Materials and methods

2.1. Geometrical model construction

A patient-specific normal aortic model was reconstructed from clinical computed tomography (CT) images using medical image processing software. The reconstructed geometry included the ascending aorta, aortic arch, descending aorta, and supra-aortic branches. An LVAD outflow graft was subsequently attached to the ascending aorta to establish the post-implantation model.

To investigate the influence of graft orientation on aortic hemodynamics, multiple outflow-graft configurations with five implantation angles were established. The implantation angle was defined as the angle between the graft centerline and the local tangent direction of the ascending aorta at the anastomosis location. All geometrical models were smoothed and optimized prior to mesh generation to eliminate local irregularities and ensure numerical stability.

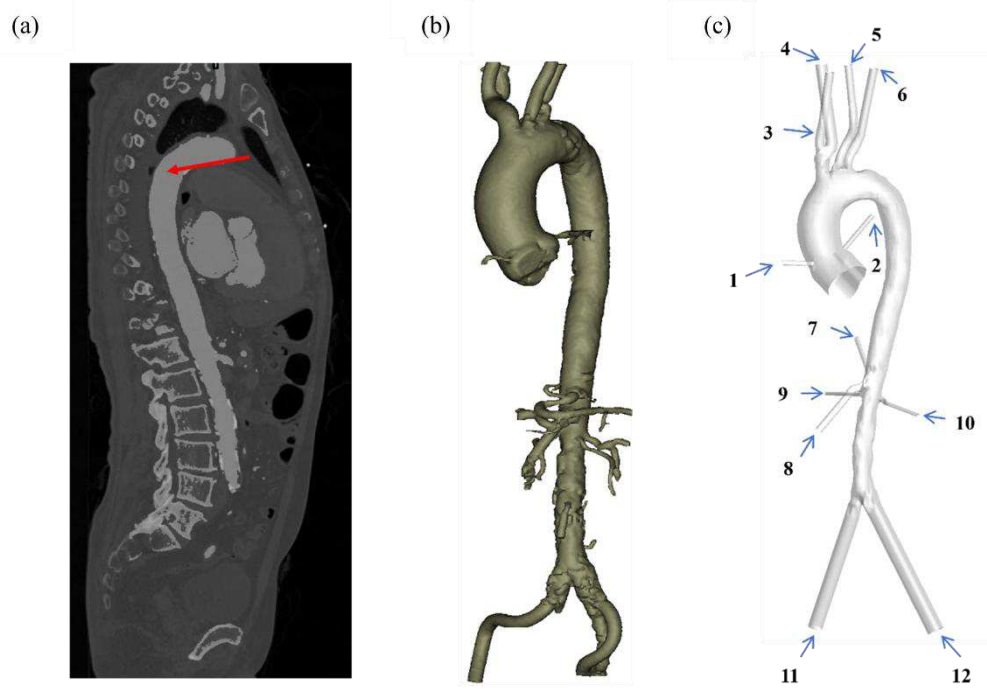


Fig 1. Study model: (a) Schematic representation of the sagittal CT cross-section, demonstrating the anatomical region used for reconstructing the aortic blood-flow domain (indicated by the red arrow). (b) Aortic model reconstructed from CT data. (c) Diagram of the aortic wall and 12 outlets: Right Coronary Artery (RCA - outlet 1), Left Coronary Artery (LCA - outlet 2), Left Subclavian Artery (LSA - outlet 3), Left Common Carotid Artery (LCCA - outlet 4), Right Common Carotid Artery (RCCA - outlet 5), Right Subclavian Total Artery (RSTA - outlet 6), Abdominal Aorta (ATA - outlet 7), Superior Mesenteric Artery (SMA - outlet 8), Right Renal Artery (RRA - outlet 9), Left Renal Artery (LRA - outlet 10), Right Iliac Artery (RIA - outlet 11), and Left Iliac Artery (LIA - outlet 12).

2.2. Mesh generation and numerical simulation

The computational domains were discretized using unstructured tetrahedral meshes with prism boundary layers near the vessel wall to accurately resolve near-wall flow behavior. A mesh independence study was performed to ensure that further mesh refinement produced negligible variations in velocity and wall shear stress distributions. The final mesh size was selected by considering both computational accuracy and efficiency.

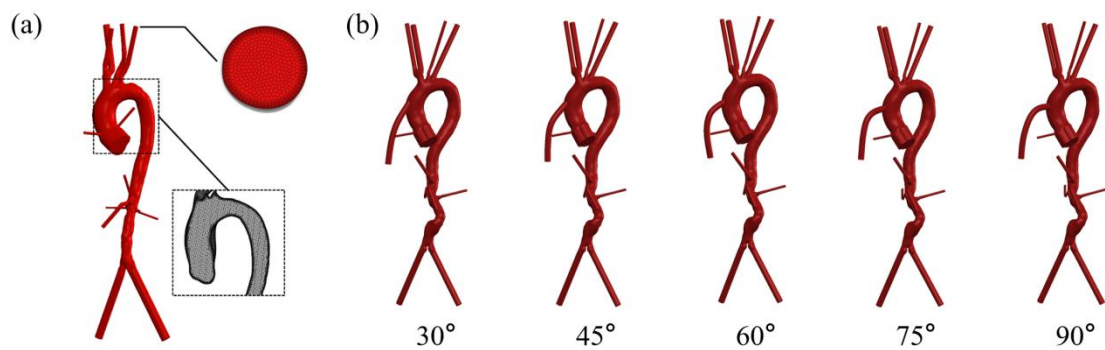


Fig 2. Computational model and mesh generation. (a) Reconstruction of the normal aorta and

the computational mesh. (b) LVAD implantation models with five implantation angles (30°, 45°, 60°, 75°, and 90°).

Given that the high rotational speed of the blood pump subjects virtually the entire fluid domain to shear rates well above 100 s⁻¹ [18], the non-Newtonian shear-thinning behavior of blood is rendered physiologically insignificant. In this study, treating blood as a Newtonian fluid is a justified approximation under these operating conditions. Blood was modeled as an incompressible Newtonian fluid with a constant viscosity of 0.0035 Pa s and a density of 1050 kg/m³ [15]. Under this assumption, the flow field is governed by the incompressible Navier–Stokes equations, expressed as Eq.1 and Eq.2, where $\mathbf{u}$ represents the velocity vector, μ is the viscosity, ρ denotes the fluid density, p is the pressure. The solutions were prepared using commercial software ANSYS Fluent (ANSYS Inc., Canonsburg, PA, USA). The SST k - ω turbulence model was used for transient simulations [19]. The time step was set to 0.005s.

$$\nabla \cdot \mathbf{u} = 0 \quad (1)$$

$$\rho \left(\frac{\partial \mathbf{u}}{\partial t} + \mathbf{u} \cdot \nabla \mathbf{u} \right) = -\nabla p + \mu \nabla^2 \mathbf{u} \quad (2)$$

2.3. Boundary conditions and support conditions

Physiological pulsatile flow conditions are established at the inlet boundary to simulate the natural ventricular ejection state [11].

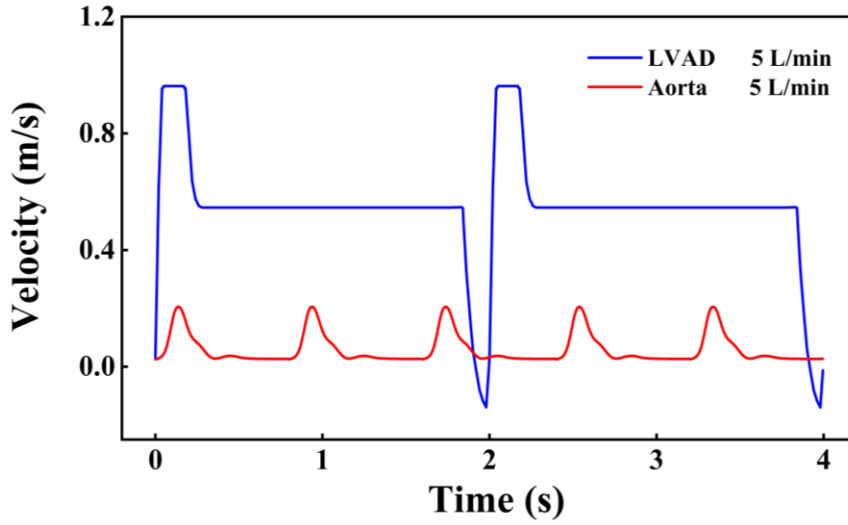


Fig 3. Pulsatile velocity waveforms of the aortic inlet and LVAD outlet.

A three-parameter Windkessel model is employed as the pressure outlet boundary for achieving 0D-3D coupling, implemented using the UDF function; the mathematical expression is presented in Eq.3, with specific parameter values listed in Table 1.

$$P(t) = P(0)e^{-\frac{t}{RC}} + r \left(Q(t) - e^{-\frac{t}{RC}} Q(0) \right) + C^{-1} \int_0^t Q(s) e^{-\frac{t-s}{RC}} ds \quad (3)$$

This study examines different LVAD support modes, including continuous flow support and pulsed flow support. For pulsed flow support, blood flow pulsatility is generated by periodically

adjusting pump speed while maintaining an average flow velocity comparable to that under continuous flow conditions.

Table 1: Parameters of the three-element lumped parameter model for the aorta.

Outlet Number	1	2	3	4	5	6	7	8	9	10	11	12
Rp (10 ⁷ Pa·s·m ⁻³)	3.39	3.39	0.99	0.51	1.21	1.01	5.56	1.68	7.75	5.85	0.37	0.35
Rd (10 ⁸ Pa·s·m ⁻³)	5.71	5.71	1.67	0.87	2.03	1.71	9.37	2.84	13.0	9.86	0.62	0.60
C (10 ⁻⁹ m ³ ·Pa ⁻¹)	0.31	0.31	1.06	2.03	0.87	1.03	0.19	0.62	0.13	0.18	2.83	2.95

To investigate the influence of cardiac recovery, multiple recovery states were modeled by adjusting the relative contributions of native ventricular output and LVAD support flow. Under full-support conditions, the majority of systemic flow was provided by the LVAD, whereas under recovery conditions the native ventricular contribution was increased to represent partial restoration of cardiac function. Different combinations of graft angle, support mode, and cardiac recovery state were systematically investigated. Detailed operating conditions for all simulated cases are summarized in Table 2 and Table 3.

Table 2: The research conditions for 5 implantation angles under full support mode of LVAD

Pulsating and steady	Flow rate (L/min)	Aorta	30°	45°	60°	75°	90°
5 implantation angles	Aorta	5	0	0	0	0	0
	Graft	none	5	5	5	5	5

Table 3: The research conditions for 4 LVAD support levels under 45°implantation angle

Pulsating and steady	Flow rate (L/min)	Aorta	100%	70%	60%	50%
4 LVAD support levels	Aorta	5	0	1.5	2	2.5
	Graft	none	5	3.5	3	2.5

2.4. Hemodynamic and bio-mechanic indicators

Computational analyses of aortic hemodynamics commonly rely on wall shear stress–based descriptors to quantify the near-wall flow environment and assess its potential physiological and pathological implications. Among these metrics, time-averaged wall shear stress (TAWSS), oscillatory shear index (OSI), and relative residence time (RRT) are widely used, because they jointly characterize the magnitude, directionality, and residence behavior of wall shear stresses over the cardiac cycle. The time-averaged wall shear stress (TAWSS) was computed to quantify the overall shear loading exerted on the endothelial surface across the entire cardiac cycle. TAWSS is defined as:

$$TAWSS = \frac{1}{T} \int_0^T ||\tau_{wall}|| dt \quad (4)$$

TAWSS reflects the sustained mechanical stimulus on the vessel wall and serves as a key indicator of endothelial homeostasis.

To characterize the directional change of wall shear stress, the oscillatory shear index (OSI) was calculated as:

$$OSI = \frac{1}{2} \left(1 - \frac{\left| \int_0^T WSS dt \right|}{\int_0^T |WSS| dt} \right) \quad (5)$$

OSI ranges between 0 (unidirectional flow) and 0.5 (purely oscillatory flow) and has been widely used to identify flow reversal or disturbance near bifurcations and graft anastomoses. Elevated OSI is strongly correlated with endothelial dysfunction and inflammation in regions of complex aortic geometry.

2.5. Platelet activation and hemolysis risk prediction

The scalar shear stress (τ) was calculated from the shear stress components using the following equation which was implemented with a user defined function:

$$\tau = \left[\frac{1}{6} \sum (\tau_{ii} - \tau_{jj})(\tau_{ii} - \tau_{jj}) + \sum (\tau_{ij}\tau_{ij}) \right]^{\frac{1}{2}} \quad (7)$$

Platelet activation can occur purely under mechanical shear, even in the absence of biochemical agonists. Under elevated shear stresses, platelets exhibit increased activation tendencies, which enhances their pro-coagulant potential. In numerical studies, shear-induced platelet activation is typically described using a power-law relationship that depends on the local shear stress and exposure time. In this work, platelet activation (PA) was modeled using the Eulerian scalar transport equation:

$$\frac{\partial \left(PA^{\frac{1}{\alpha_1}} \right)}{\partial t} + \mathbf{u} \cdot \nabla \left(PA^{\frac{1}{\alpha_1}} \right) = (C_1 \cdot \tau^{\beta_1})^{\frac{1}{\alpha_1}} \quad (8)$$

where PA is the platelet activation level, $C_1 = 4.08 \times 10^{-5}$, $\alpha_1 = 0.8$, $\beta_1 = 1.56$ are model coefficients fitted by experimental results [20].

Hemolysis was solved using the Eulerian scalar transport equation:

$$\frac{\partial \left(hb^{\frac{1}{\alpha_2}} \right)}{\partial t} + \mathbf{u} \cdot \nabla \left(hb^{\frac{1}{\alpha_2}} \right) = (HB \cdot C_2 \cdot \tau^{\beta_2})^{\frac{1}{\alpha_2}} \quad (9)$$

where hb is the plasma free hemoglobin as a percentage of the total blood hemoglobin. HB is the total blood hemoglobin concentration (10 g/dL). C_2 , α_2 , and β_2 are empirical coefficients taken as $C_2 = 1.8 \times 10^{-6}$, $\alpha_2 = 0.765$, $\beta_2 = 1.991$ [21].

The hemolysis index (HI) is defined as the volume-averaged Δhb to HB:

$$HI(\%) = \frac{\Delta hb}{HB} \times 100\% \quad (10)$$

For all simulated cases, PA and HI were evaluated based on the results obtained at the end of the tenth cardiac cycle.

2.6. Old blood washout prediction

Old blood washout was evaluated using a transient pulse-tracer method. A transport equation for a passive scalar, denoted by ϕ and defined as the old blood index (OBI), was solved:

$$\frac{\partial \phi}{\partial t} + u \cdot \nabla \phi = S(t) \quad (11)$$

where $S(t)$ denotes the time-dependent source term. To model the instantaneous distribution of resident blood, $S(t)$ was defined as an impulse-like function:

$$S(t) = \begin{cases} 1, & t = t_0 \\ 0, & t \neq t_0 \end{cases} \quad (12)$$

The theoretical mean residence time of blood flowing from the inlet to the outlet of the aorta was estimated as 5.9 s, calculated as $t = V/Q$, where V is the aortic volume and Q is the volumetric flow rate. At time t_0 , the entire fluid domain was initialized as old blood ($\phi=1$). As blood flow progressed, the OBI field was gradually washed out by incoming fresh blood. The washout process was simulated over eight cardiac cycles to ensure convergence of the transient behavior, and the OBI results at the end of the eighth cardiac cycle were used for subsequent statistical analysis. Six planes were created to observe the scalar distribution within the aorta.

3. Results

3.1. Alterations in aortic flow organization and helical characteristics

At smaller implantation angles (30° and 45°), the LVAD jet remained relatively aligned with the aortic axis and produced no obvious flow separation or recirculation. A low-velocity region was observed in the ascending aorta because of the limited contribution of the LVAD inflow. At 30° , the jet impinged near the brachiocephalic artery, whereas at 45° the impingement location shifted distally along the outer curvature of the aortic arch, accompanied by the formation of a localized high-velocity region.

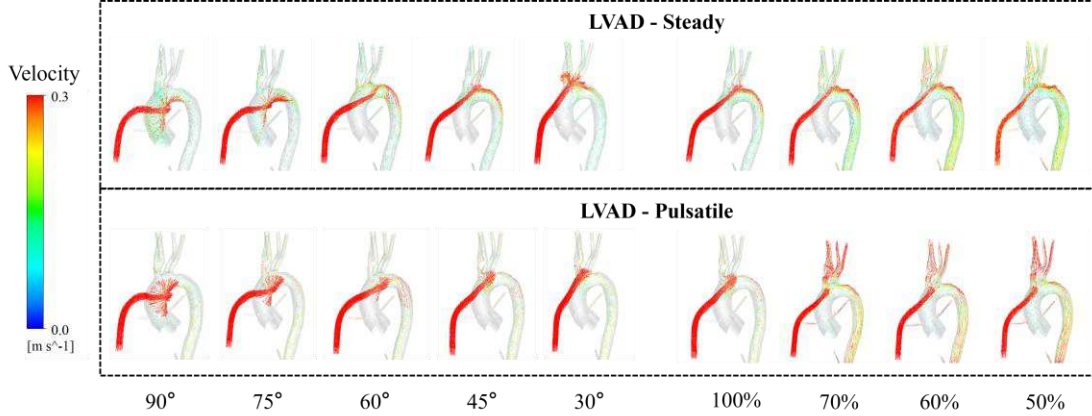


Fig 4. Streamline distributions following LVAD implantation at the peak systolic velocity.

As the implantation angle increased from 60° to 90° , the LVAD jet progressively deflected toward the inner curvature of the ascending aorta. Wall impingement became increasingly pronounced, and a larger recirculation region developed near the aortic root, with the strongest flow disturbance observed at 90° . Under pulsatile support, both jet impingement and flow recirculation were more evident than under continuous support.

During ventricular function recovery, the native left ventricular outflow increasingly altered the trajectory of the LVAD jet. Under continuous support, the jet weakened and the high-velocity region near the ascending aortic wall gradually diminished with decreasing pump flow. Under pulsatile support, the interaction between the native outflow and the LVAD jet displaced

the impingement location away from the vessel wall and promoted greater flow into the supra-aortic branches at peak systole.

3.2. Variations in wall shear stress (WSS) environment

3.2.1. Peak WSS distribution

The spatial distribution of peak WSS varied markedly with implantation angle. Under continuous support, most of the aortic wall remained exposed to relatively low WSS, whereas a localized high-WSS region developed at the LVAD jet impingement site. As the implantation angle increased, both the location and extent of the high-WSS region changed, with the largest high-WSS area observed at 90°. At peak systole, pulsatile support altered the instantaneous WSS distribution. Compared with continuous support, pulsatile flow reduced low-WSS regions in the descending aorta, whereas limited changes were observed in the ascending aorta and aortic root, where low-WSS regions remained evident. During ventricular function recovery, the high-WSS region gradually contracted as LVAD flow decreased. Under pulsatile support, the jet impingement location shifted away from the vessel wall, resulting in a more uniform instantaneous WSS distribution than under full-support conditions.

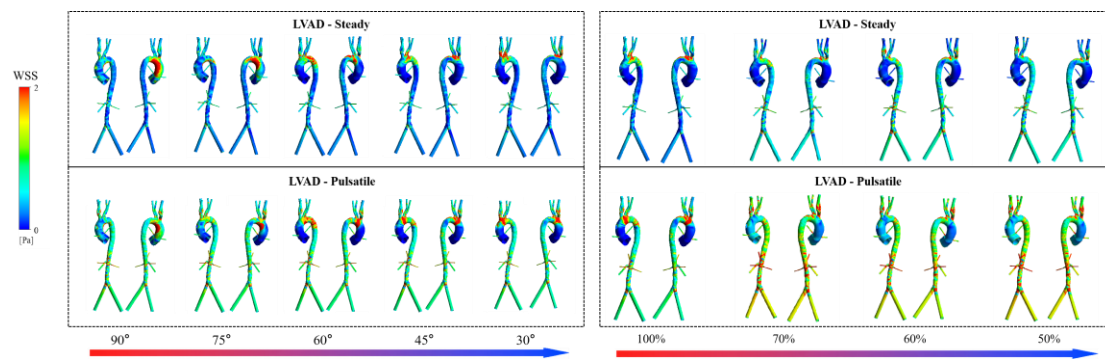


Fig 5. Wall shear stress (WSS) distributions at the peak systolic velocity. (a) Different LVAD implantation angles. (b) Different LVAD support flow rates.

3.2.2. Hemodynamic Parameters of TAWSS, OSI and RRT distribution

Compared with the physiological baseline, LVAD support increased the area-averaged TAWSS under all implantation angles. The highest value was observed at 90°, followed by a gradual decrease with decreasing implantation angle, although a slight increase was found at 30°. A similar trend was observed for the high-TAWSS area. During ventricular recovery, the area-averaged TAWSS gradually decreased with the reduction in LVAD flow. At 70% ventricular recovery, the overall TAWSS approached the physiological baseline, indicating that the wall shear environment became comparable to that of the native aorta.

Although the area-averaged TAWSS was comparable between continuous and pulsatile support, pulsatile flow reduced the extent of low-TAWSS regions during ventricular recovery, whereas limited improvement was observed in the ascending aorta and aortic root.

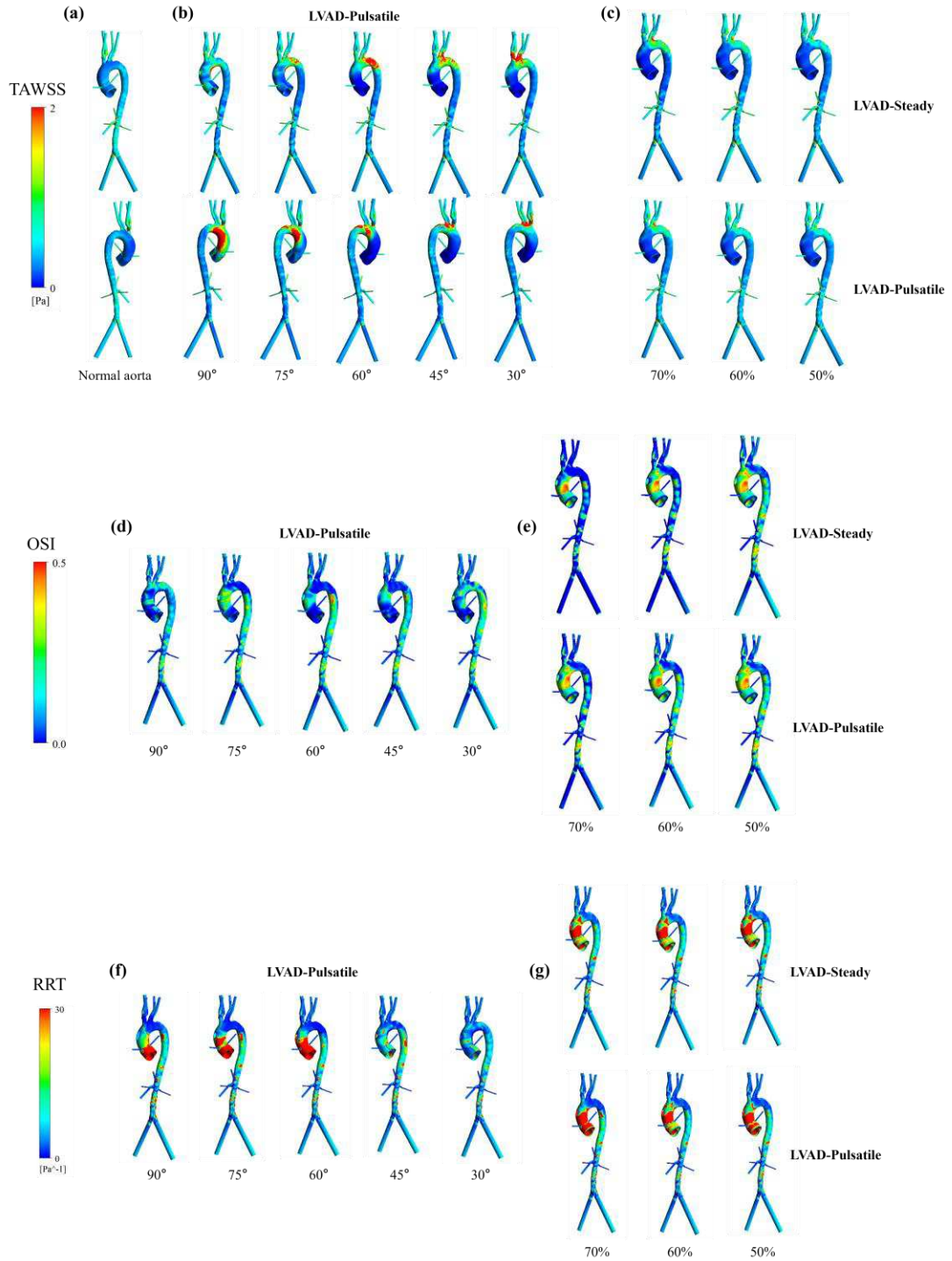


Fig 6. TAWSS, OSI, and RRT results. (a) Baseline TAWSS distribution in the normal aorta. (b) TAWSS under different LVAD implantation angles. (c) TAWSS under different LVAD support flow rates. (d) OSI under different LVAD implantation angles. (e) OSI under different LVAD support flow rates. (f) RRT under different LVAD implantation angles. (g) RRT under different LVAD support flow rates.

High OSI values were primarily distributed around the graft anastomosis and the downstream thoracic aorta. As the implantation angle increased, the spatial distribution of OSI changed markedly. At larger implantation angles, the high-OSI region around the ascending aorta

became progressively reduced, whereas intermediate implantation angles exhibited more extensive oscillatory flow downstream of the graft. During ventricular recovery, high-OSI regions gradually expanded toward the ascending aorta as LVAD flow decreased. The spatial extent of oscillatory flow became more pronounced under reduced support, particularly in the ascending aorta.

The spatial distribution of RRT was generally consistent with that of OSI. Under full LVAD support, elevated RRT was mainly observed around the aortic root and the downstream thoracic aorta. With increasing implantation angle, the high-RRT region in the ascending aorta gradually diminished, whereas lower implantation angles were associated with more pronounced blood residence near the aortic root. During ventricular recovery, the overall distribution of RRT remained relatively stable despite the reduction in LVAD flow. Compared with the marked redistribution observed for OSI, only minor changes in the extent and location of high-RRT regions were observed throughout the recovery process.

3.3. Hemolysis and platelet activation Analyzation

LVAD support substantially increased HI compared with the physiological baseline under all simulated conditions. As the implantation angle increased, the high-HI region progressively expanded within the ascending aorta, accompanied by an increase in the volume-averaged HI. Under pulsatile support, the HI increased from 1.85×10^{-4} at 45° to 2.60×10^{-4} at 90° , whereas the corresponding steady-flow values increased from 1.76×10^{-4} to 2.40×10^{-4} . During ventricular recovery, the high-HI region gradually contracted and became confined to the proximal ascending aorta. Correspondingly, the volume-averaged HI decreased continuously from 2.90×10^{-4} to 1.50×10^{-4} under pulsatile support and from 2.60×10^{-4} to 1.42×10^{-4} under steady support.

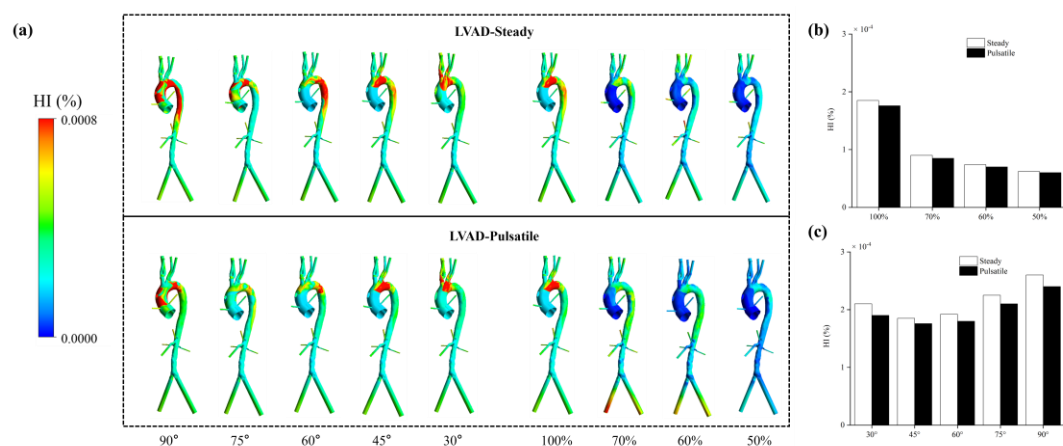


Fig 7. Hemolysis results. (a) Contours of the hemolysis index (HI). (b) Volume-averaged HI under different LVAD support flow rates. (c) Volume-averaged HI under different LVAD implantation angles.

A trend similar to that of HI was observed for PA. The volume-averaged PA increased with implantation angle, rising from 3.00×10^{-5} at 30° to 3.50×10^{-5} under pulsatile support, compared with an increase from 2.70×10^{-5} to 3.30×10^{-5} under steady support. During ventricular recovery, PA progressively decreased with the reduction in LVAD flow, from 2.90×10^{-5} at full support to 1.50×10^{-5} at 50% recovery under pulsatile support, while the

corresponding steady-flow values decreased from 2.60×10^{-5} to 1.42×10^{-5} . Consistent with these quantitative results, regions of elevated PA became progressively smaller as ventricular function recovered.

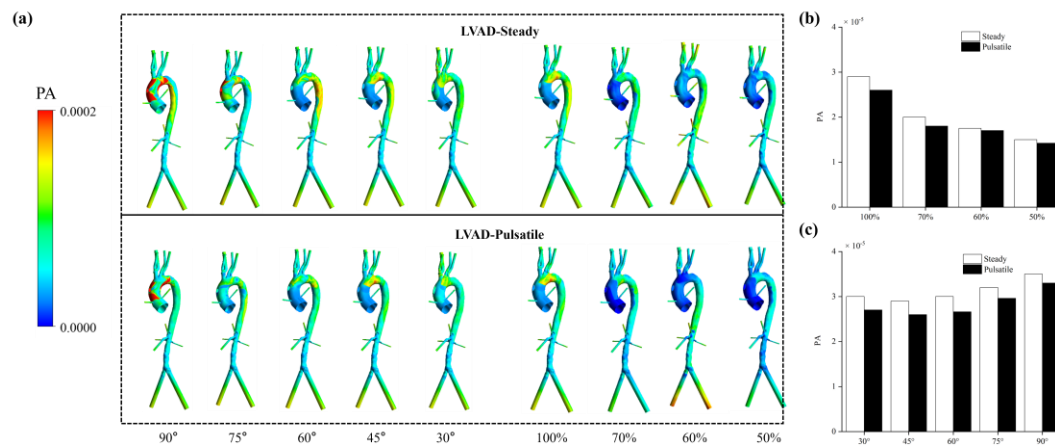


Fig 8. Platelet activation results. (a) Contours of the platelet activation level (PA). (b) Volume-averaged PA under different LVAD support flow rates. (c) Volume-averaged PA under different LVAD implantation angles.

3.4. Blood renewal efficiency

Two representative cross-sectional planes were defined along the aorta to evaluate blood renewal efficiency (Fig.9). The OBI calculated at these planes was used to quantify the temporal replacement of blood under different graft angles, support modes, and cardiac recovery conditions.

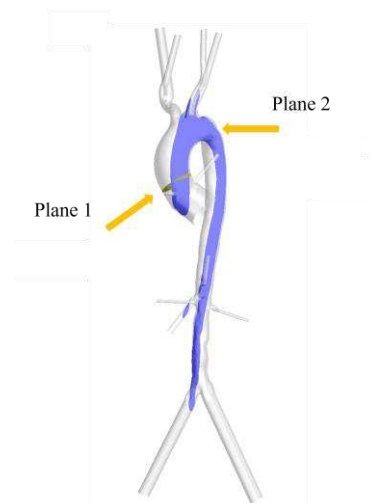


Fig 9. Locations of the two cross-sectional monitoring planes (Plane 1 and Plane 2) used for the evaluation of blood renewal efficiency.

The volume-averaged OBI decreased progressively with increasing implantation angle and ventricular recovery (Fig.9). At the 45° implantation angle, the OBI under steady support decreased from 0.29 at full support to 0.19 at 50% recovery, while the corresponding value under pulsatile support decreased from 0.275 to 0.18. Similarly, under full support, increasing

the implantation angle from 30° to 90° reduced the OBI from 0.31 to 0.21 under steady support and from 0.30 to 0.21 under pulsatile support. For all implantation angles and recovery stages, pulsatile support consistently produced slightly lower OBI values than steady support.

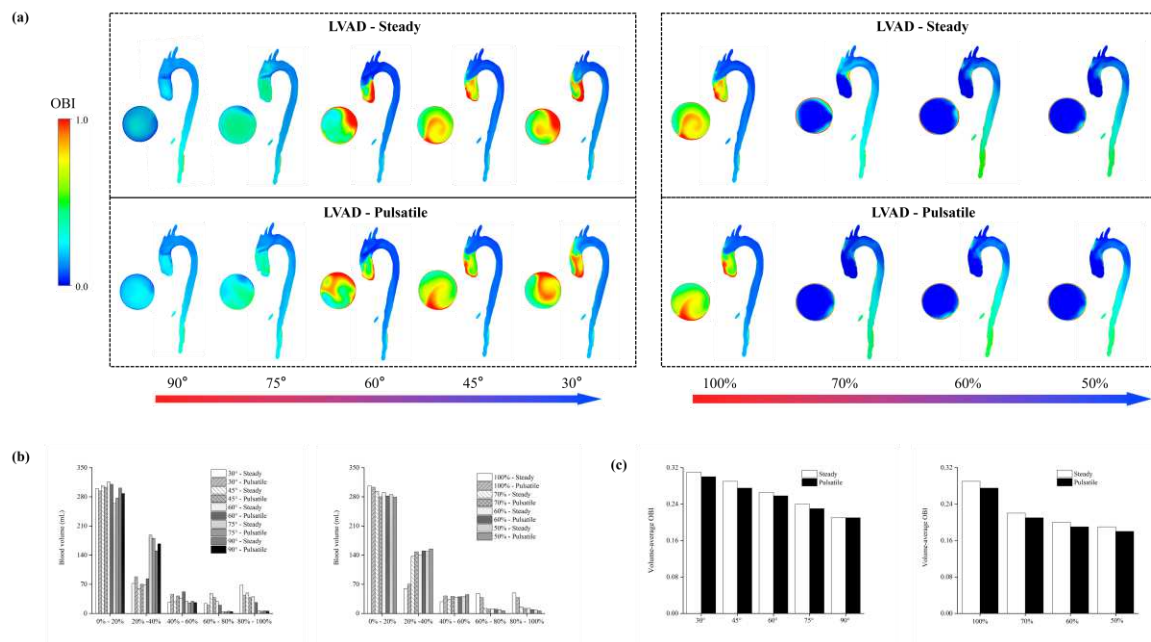


Fig 10. Blood washout efficiency. (a) OBI distributions on two cross-sectional planes. (b) Volume distributions across different OBI ranges. (c) Volume-averaged OBI.

The OBI volume distribution further showed that the decrease in the average OBI was mainly associated with the reduction of highly stagnant blood rather than changes in freshly renewed blood (Fig. 9). Across all implantation angles and recovery stages, the volume within the 0–20% OBI interval remained relatively stable. In contrast, the high-OBI regions (60–80% and 80–100%) were markedly reduced. At the 45° implantation angle, the combined volume of these two intervals under steady support decreased from 97 mL at full support to 17 mL at 50% recovery, while that under pulsatile support decreased from 76 mL to 12 mL. A similar trend was observed with implantation angle. Under full support, increasing the implantation angle from 30° to 90° reduced the combined high-OBI volume from 92 mL to 11 mL under steady support and from 63 mL to 10 mL under pulsatile support. These results indicate that the improvement in washout efficiency was primarily characterized by the progressive contraction of highly stagnant blood regions, whereas the volume of freshly renewed blood remained largely unchanged.

4. Discussion

Left ventricular assist device implantation fundamentally reconstructs physiological aortic flow by introducing a high-momentum outflow jet that interacts with native ventricular ejection. The resulting hemodynamic environment is governed by the coupled effects of graft orientation, pulsatile support, and cardiac recovery, which regulate the spatial trajectory, temporal evolution, and interaction intensity of the composite jet. Previous computational studies have mainly investigated these factors separately under fixed support conditions, focusing on individual parameters such as wall shear stress or flow stagnation. In contrast, the present study integrates

graft angle, support mode, and progressive cardiac recovery within a unified framework and demonstrates that alterations in wall mechanics, blood transport, and hemocompatibility originate from continuous reorganization of the composite jet. These findings emphasize the importance of multi-objective evaluation during LVAD optimization.

The present results indicate that graft orientation is the primary determinant of composite-jet organization. Smaller implantation angles directed the LVAD jet toward the outer curvature of the ascending aorta, causing localized wall impingement, recirculation, and low-velocity regions. Increasing the graft angle redirected the jet toward the inner curvature and distal aorta, enhancing momentum transport and reducing proximal blood stagnation. Similar angle-dependent effects on jet impingement and wall shear distribution have been reported previously [22,23]. However, the present study further demonstrates that graft angle regulates not only local wall mechanics but also the global transport pathway of the composite jet. Larger angles improved washout and reduced residence time but simultaneously increased localized shear exposure and blood damage due to stronger jet-wall interaction, indicating an inherent trade-off between transport efficiency and hemocompatibility.

The evaluated hemodynamic parameters exhibited strong coupling rather than independent variation. Improved blood renewal at larger implantation angles was accompanied by reduced RRT, indicating enhanced transport efficiency and reduced stagnation. Conversely, regions with prolonged recirculation showed lower TAWSS and higher OSI, reflecting unfavorable flow conditions associated with thrombosis risk. Although larger angles promoted washout, increased jet impingement elevated shear-related hemolysis and platelet activation. Therefore, LVAD implantation strategies should balance flow transport, wall mechanical environment, and blood compatibility rather than optimize a single hemodynamic index.

Pulsatile support primarily modified the temporal evolution of the composite jet rather than its overall spatial distribution. Cyclic acceleration and deceleration disrupted persistent recirculation structures and enhanced momentum exchange between the jet core and surrounding flow, thereby improving blood mixing and washout without markedly altering global TAWSS patterns. Similar improvements in transport efficiency under pulsatile modulation have been reported previously [24]. Moreover, pulsatility reduced cumulative hemolysis and platelet activation by shortening the duration of high-shear exposure during deceleration phases. However, periodic flow acceleration also increased temporal flow variation near the ascending aorta, resulting in localized elevation of oscillatory shear. Thus, pulsatile support improves circulation not simply by increasing pulsation intensity but through redistribution of transport efficiency and shear exposure.

Cardiac recovery further reshaped the flow environment by increasing the contribution of native ventricular ejection. Under full support, aortic flow was dominated by the LVAD jet, whereas recovery transformed the circulation into a dual-source system driven by both pump output and ventricular ejection. This transition weakened LVAD dominance, enhanced flow mixing, and improved blood renewal, resulting in reduced hemolysis and platelet activation. However, recovery did not uniformly improve all hemodynamic parameters, as competing inflow sources increased temporal flow variation and elevated OSI near the ascending aorta. Therefore, cardiac recovery represents a redistribution of flow organization rather than complete normalization, highlighting the need for continuous evaluation during LVAD weaning. Collectively, this study demonstrates that LVAD-supported aortic hemodynamics result from

coupled regulation of composite-jet organization, wall mechanical loading, and blood transport. Larger implantation angles enhance downstream transport and washout but increase localized shear-mediated blood damage. Pulsatile support improves blood renewal while reducing cumulative blood trauma through temporal modulation of shear exposure, whereas cardiac recovery weakens LVAD dominance and introduces more complex oscillatory patterns. These findings suggest that future LVAD optimization should adopt patient-specific multi-objective strategies considering transport efficiency, thrombosis risk, and hemocompatibility simultaneously.

Several limitations should be acknowledged. First, only one representative aortic geometry was analyzed, and anatomical variability among patients was not considered. Second, rigid vessel walls were assumed, neglecting fluid–structure interaction effects on wall shear and flow evolution. Third, cardiac recovery was represented through modified boundary conditions rather than realistic ventricular motion and valve dynamics. Finally, blood was modeled as a homogeneous Newtonian fluid without explicit consideration of thrombosis or cellular-scale transport. Future studies incorporating patient-specific anatomy, fluid–structure interaction, ventricular mechanics, and thrombosis models may further improve physiological accuracy and support individualized LVAD optimization.

Ethics approval and consent to participate:

No ethical approval is required.

Consent for publication

All authors have given their knowledge and consent to publication.

Competing interests

There is no conflict of interest in this paper.

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Authors' contributions:

Kangli Sun drafted the article, wrote the publication, has done the conception and design of the work, evaluated the results, analyzed the data, and critically revised the article. Yue Han, commented on the manuscript, and critically revised the article. Gaoyang Li and Zengsheng Chen provided the funding needed for this study. All authors read and approved the final manuscript.

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All data generated or analyzed during this study are included in this published article and its supplementary information files. The original datasets used and analyzed during the current study are available from the corresponding author on reasonable request

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