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Competitive Flow Identification and Hemodynamic Analysis in Coronary Artery Bypass Grafting

1. Abstract

Background and Objective: Coronary artery bypass grafting (CABG) is the primary treatment for severe coronary artery disease. Competitive flow and anastomotic stenosis are two core etiologies leading to low graft flow. However, there is a lack of effective clinical methods to accurately distinguish between these two causes. This study aims to explore a novel approach to differentiate the etiologies of low-flow grafts after CABG based on fractional flow reserve (FFR) and intraoperative transit-time flow measurement (TTFM) waveform characteristics, and to quantitatively analyze the impact of competitive flow on the graft hemodynamic microenvironment. **Methods**: This retrospective study included data from 157 patients who underwent CABG and had intraoperative TTFM measurements. Patient-specific 0D-3D models were constructed based on postoperative CTA images and physiological parameters, and FFR and hemodynamic parameters were computed non-invasively using computational fluid dynamics (CFD). After excluding the gray zone (0.75-0.80), low-flow grafts were classified into a competitive flow group(FFR > 0.80) and an anastomotic stenosis group (FFR < 0.75) based on postoperative FFR. Binary logistic

regression was performed to evaluate the predictive value of preoperative FFR, pulsatility index (PI), and diastolic filling fraction (DF). Hemodynamic parameters, including wall shear stress (WSS) and relative residence time (RRT), were compared between the two groups. **Results**: After initial screening by TTFM, a total of 80 low-flow left internal mammary artery (LIMA) grafts and 80 low-flow saphenous vein graft (SVG) grafts were included. For LIMA grafts, preoperative FFR was an independent predictor of competitive flow (OR=1.418, 95%CI:1.086-1.851, P=0.010), with an area under the ROC curve (AUC) of 0.703. The competitive flow group exhibited characteristic systolic retrograde flow with a high-pulsatility waveform, along with significantly decreased mean WSS (WSS-mean, P=0.004), significantly decreased wall shear stress gradient (WSSG, P=0.019), and significantly prolonged RRT (P=0.005). For SVG grafts, DF was an independent risk factor for identifying anastomotic stenosis (OR=0.912, 95% CI: 0.844–0.985, P=0.020), with a model AUC of 0.768. However, no significant differences in hemodynamic parameters were found between the two groups, suggesting that venous grafts have relative tolerance to the adverse hemodynamic microenvironment induced by competitive flow. **Conclusion**: This study establishes a diagnostic system for low-flow graft etiology differentiation based on FFR and TTFM parameters, confirming that preoperative FFR independently predicts competitive flow in LIMA, while DF is more valuable for SVG, revealing significant heterogeneity in hemodynamic responses to competitive flow between arterial and venous grafts, providing a theoretical basis for individualized risk assessment and precise management after CABG.

[Keywords]: Competitive Flow; Fractional Flow Reserve (FFR); Hemodynamics

2. Background

Coronary artery bypass grafting (CABG) is a major clinical treatment for coronary artery disease, alongside medication and stenting. This procedure revascularizes the distal myocardium by transplanting autologous grafts to bypass occluded coronary arteries, thereby improving myocardial ischemia. Although this surgery significantly alleviates angina and improves survival rates, early graft failure remains a critical clinical challenge. Data indicate that the graft failure rate within the first postoperative year is as high as 15-30% for saphenous vein grafts (SVG) and 5-15% for left internal mammary artery (LIMA) grafts[1, 2], directly leading to recurrent myocardial ischemia. Apart from anastomotic technical defects, competitive flow is a major risk factor affecting postoperative graft patency.

Competitive flow refers to the phenomenon where graft blood flow is reduced or even reversed due to similar pressure levels or non-severe stenosis between the graft and the native coronary artery[3]. This phenomenon is particularly pronounced when the intraluminal pressures of the coronary artery and the graft reach near-equilibrium. The presence of competitive flow poses a significant threat to the efficacy of CABG. Studies have shown that competitive flow leads to decreased blood flow within the graft, potentially causing reverse flow, which may further induce the "string sign" and ultimately result in graft failure[4]. Long-term follow-up studies by Royse et al.[5] and Nakajima et al. [4]have further confirmed that the actual patency rate of grafts affected

by competitive flow is significantly lower than that of bypass grafts with antegrade flow. Notably, competitive flow not only originates from the native coronary artery but can also occur between different graft limbs in composite grafts (e.g., Y-grafts), thereby affecting the overall graft function[6]. Clinically, attempts have been made to induce maximal hyperemia using vasodilators (e.g., adenosine) or inotropic agents (e.g., dobutamine), and to observe whether graft perfusion is restored using intraoperative flow monitoring, as an indirect method to identify competitive flow. However, this approach is limited by its clinical complexity and susceptibility to interference from medications such as beta-blockers[3, 7].

Although the clinical significance of competitive flow has been preliminarily recognized, there remains a lack of standardized diagnostic criteria and assessment methods. Existing research often relies on retrospective judgment using postoperative angiography or TTFM waveform analysis, lacking preoperative predictive tools[8]. Studies have shown that the TTFM waveform characteristics of competitive flow typically include reduced mean flow, flattened peaks, and often accompanied by diastolic reversal[1, 2]. To quantify competitive flow, researchers have proposed various parameters and methods, such as the Competitive Flow Index (CFI) defined by Frank et al.[7, 9], the F0/H2 index based on Fast Fourier Transform analysis by Mao et al.[10], and the assessment of stenosis severity via quantitative coronary analysis (QCA). However, current research lacks in-depth investigation into the mechanisms of low graft flow caused by anastomotic stenosis related to surgical technique. Furthermore, it remains difficult to predict the presence of competitive flow preoperatively, and there

is still a lack of uniform, reliable, and fixed clinical indicators to guide intraoperative decision-making regarding grafting for moderately stenotic vessels[11].

It is noteworthy that although both competitive flow and anastomotic stenosis manifest as low graft flow, their hemodynamic mechanisms are distinctly different. The impact of competitive flow on graft patency is largely mediated by "reverse flow" altering the local hemodynamic microenvironment[12]. In contrast, anastomotic stenosis creates a high-velocity jet due to luminal narrowing, leading to a different set of hemodynamic alterations[13, 14]. Hemodynamic parameters, such as wall shear stress (WSS), oscillatory shear index (OSI), relative residence time (RRT), and time-averaged wall shear stress (TAWSS), are key indicators for assessing vascular endothelial function and predicting intimal hyperplasia and the progression of atherosclerosis[15]. The low or oscillatory flow caused by competitive flow can significantly increase OSI, disrupt endothelial cell function (e.g., the balance of NO and ET), thereby inducing intimal hyperplasia and ultimately compromising long-term graft patency[12].

In addition to the in-depth analysis of the local graft hemodynamic environment using microscopic parameters like WSS and RRT, macroscopic functional assessment tools have also garnered attention from many researchers for identifying competitive flow[3, 16, 17]. Fractional flow reserve (FFR), a crucial indicator for assessing the functional significance of coronary artery stenosis, has been extensively validated in percutaneous coronary intervention (PCI). In the field of CABG, preliminary studies suggest that grafting vessels with high FFR values (>0.80), indicating non-severe functional stenosis, may carry a higher risk of graft occlusion due to competitive flow[16,

[17]. This suggests that preoperative or postoperative FFR assessment may have potential value in identifying and preventing competitive flow, although current evidence is insufficient.

In summary, competitive flow is a key risk factor affecting graft patency after CABG. Current research still has limitations regarding real-time identification of competitive flow, specific analysis of its waveform characteristics, and its quantitative impact on graft hemodynamics. Therefore, this study aims to explore a safe and effective new method for identifying competitive flow, systematically analyze the TTFM waveform characteristics of grafts affected by competitive flow, and establish a waveform identification system. Using computational hemodynamic models, it seeks to quantitatively investigate the impact of competitive flow on the graft hemodynamic environment and elucidate the mechanical mechanisms leading to graft failure.

This study is expected to provide new theoretical foundations and tools for the precise clinical identification and management of competitive flow, ultimately improving the prognosis of CABG patients.

3. Results

3.1 Identification of Competitive Flow

3.1.1 Identification of Competitive Flow in LIMA Grafts

For LIMA grafts, multivariate binary logistic regression analysis was performed including PI, DF, preoperative FFR, graft length, and stenosis length. The results

showed that preoperative FFR (OR=1.418, 95% CI: 1.086–1.851, *P=0.010) was an independent predictor of competitive flow, while PI (OR=1.438，95%CI：0.910-2.270，P=0.120), DF (OR=0.989，95%CI：0.933-1.049，P=0.713), graft length (OR=0.942，95%CI：0.797-1.114，P=0.484), and stenosis length (OR=1.001，95%CI：0.794-1.262，P=0.993) were not statistically significant (Table 1 and Figure 1A).

The model demonstrated acceptable predictive value, with an AUC of 0.703(95% CI: 0.578-0.816, P<0.001). At the optimal cutoff value of 0.420, the sensitivity was 55.2% and specificity was 87.0% (Figure 1B), suggesting that this combination of indicators has a certain ability to identify competitive flow.

Variable	OR (95% CI: lower – upper)	P-value
Pre-FFR (per 0.1)	1.418(1.086-1.851)	*0.010
PI	1.438(0.910-2.270)	0.120
DF	0.989(0.933-1.049)	0.713
Graft length	0.942(0.797-1.114)	0.484
Stenosis_len	1.001(0.794-1.262)	0.993

Table 1 Multivariate logistic regression analysis of competitive flow vs. anastomotic stenosis in LIMA grafts

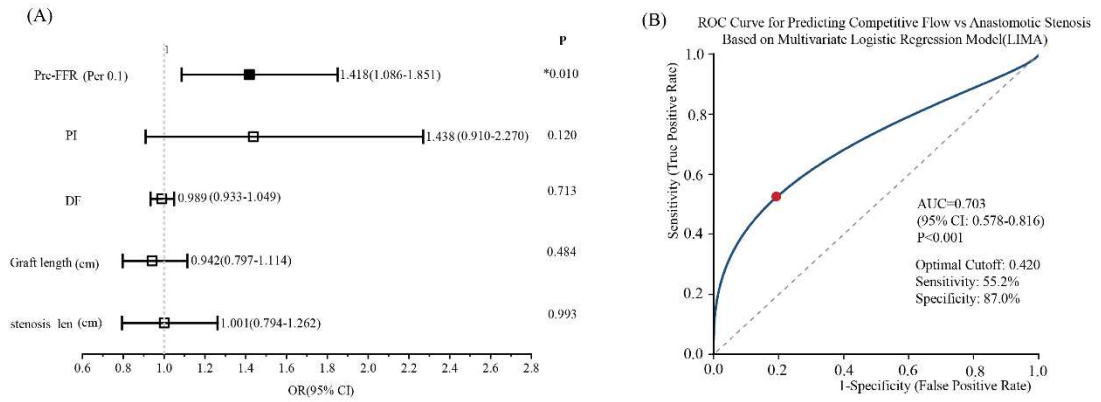


Figure 1 Multivariate logistic regression analysis and diagnostic performance evaluation for competitive flow vs. anastomotic stenosis in LIMA grafts. (A) Forest plot showing odds ratios (OR) and 95% confidence intervals (CI) from the binary logistic regression analysis. Pre-FFR was an independent predictor of competitive flow. The vertical dashed line indicates OR=1. *P<0.05。(B) ROC curve evaluating the predictive performance of the multivariate model. The area under the curve (AUC) was 0.703（95% CI: 0.578-0.816）, with an optimal cutoff value of 0.420, sensitivity of 55.2%, and specificity of 87.0.

Intraoperative TTFM monitoring revealed distinct waveform patterns in LIMA grafts under different flow conditions (Figure 2). Normal LIMA flow (Figures 2A, 2B) exhibited a typical biphasic pulsatile waveform, characterized by brief antegrade flow during systole followed by a rapid transition to sustained diastolic perfusion, with no retrograde flow component. The waveform was smooth with no sharp peaks. Normal grafts generally exhibited a mean flow > 50 mL/min, pulsatility index < 3, and diastolic filling fraction > 50%.

The competitive flow group (Figures 2C, 2D) demonstrated characteristic systolic retrograde flow with high pulsatility, marked by the presence of systolic waveforms significantly below the baseline, low mean flow, elevated PI, and numerous sharp peaks.

Both cases showed antegrade perfusion insufficiency and periodic flow direction reversal caused by competitive flow.

The anastomotic stenosis group (Figures 2E, 2F) presented a high-resistance waveform pattern, characterized by sharp peaks during diastole and flattened waveforms during systole, with low mean flow, elevated PI, and significantly reduced DF. Unlike competitive flow, the stenosis group exhibited flattened waveforms with reduced pulsatility and no obvious systolic retrograde flow, suggesting that the flow reduction resulted from hemodynamic obstruction rather than pressure competition.

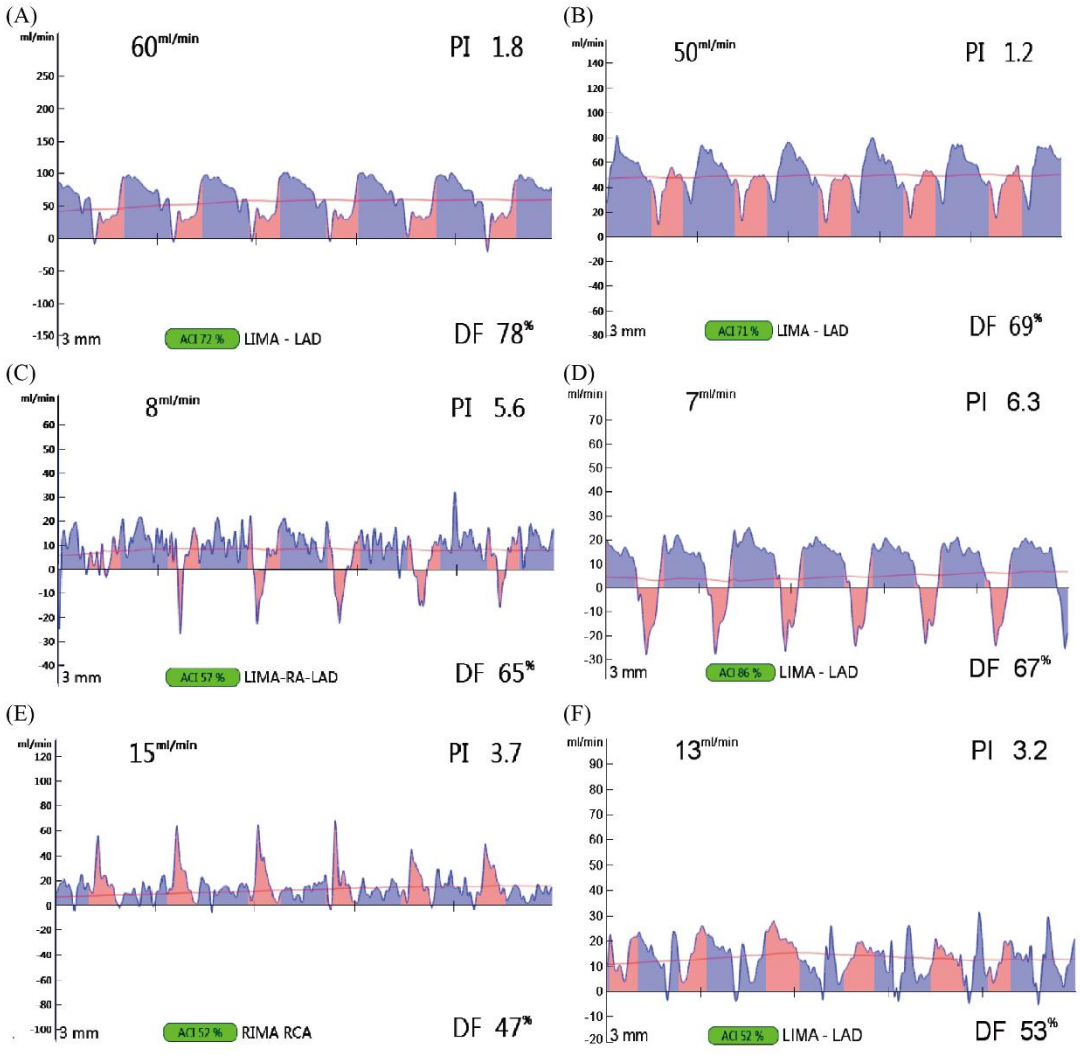


Figure 2 TTFM waveform characteristics of LIMA grafts. A-B: Normal flow waveform, typical biphasic pattern with predominant diastolic perfusion and no retrograde flow. C-D: Competitive flow (postoperative FFR > 0.80), showing pronounced systolic retrograde sharp peak waveforms and high pulsatility. E-F: Anastomotic stenosis (postoperative FFR < 0.75), showing high-resistance, flattened waveforms with significantly reduced DF and no obvious retrograde flow. Horizontal axis: time (seconds, normalized to one cardiac cycle); vertical axis: flow rate (mL/min). Above baseline: antegrade flow; below baseline: retrograde flow. HR: heart rate. Scale bar: 20 mL/min per grid (B, C, D) or 50 mL/min per grid (A, E, F).

3.1.2 Identification of Competitive Flow in SVG Grafts

For SVG grafts, multivariate binary logistic regression analysis was similarly performed including PI, DF, preoperative FFR, graft length, and stenosis length. The results showed that DF(OR=0.912, 95% CI: 0.844–0.985, *P=0.020) was an independent risk factor for anastomotic stenosis. Preoperative FFR showed a trend toward association with competitive flow, but did not reach statistical significance (OR=1.366，95%CI：0.966-1.932，P=0.078). Meanwhile, PI(OR=0.817，95%CI：0.640-1.043，P=0.105), graft length （OR=0.973，95%CI：0.859-1.102，P=0.665）, and stenosis length (OR=0.905，95%CI：0.661-1.240，P=0.535)showed no statistical significance (Table 2 and Figure 3A).

The model demonstrated good predictive performance, with an AUC of 0.768(95% CI: 0.621-0.869, P<0.001). At the optimal cutoff value of 0.796, the sensitivity was

74.6% and specificity was 78.6% (Figure 3B), suggesting that this combination of indicators has moderate ability to identify competitive flow.

Variable	OR (95% CI: lower – upper)	P-value
DF	0.912(0.844-0.985)	*0.020
PI	0.817(0.640-1.043)	0.105
Pre-FFR (per 0.1)	1.366(0.966-1.932)	0.078
Graft length	0.973(0.859-1.102)	0.665
Stenosis-len	0.905(0.661-1.240)	0.535

Table 2 Multivariate logistic regression analysis of anastomotic stenosis in SVG grafts

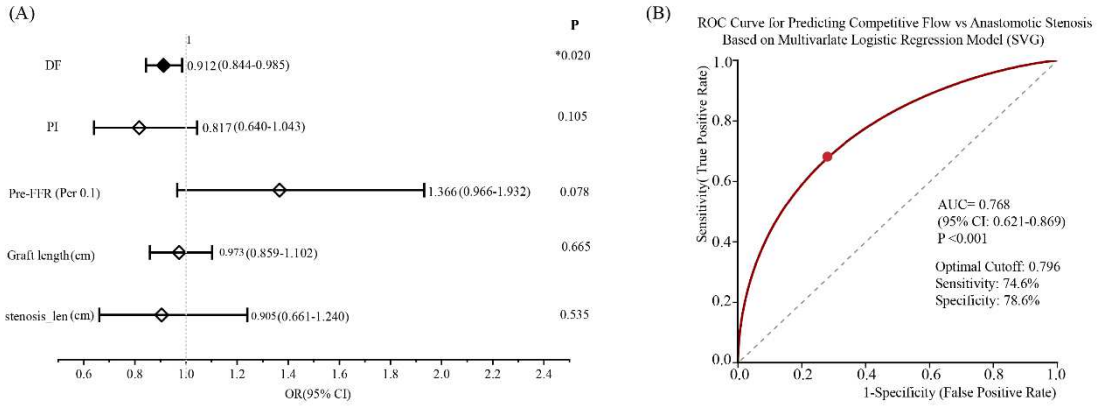


Figure 3 Multivariate logistic regression analysis and diagnostic performance evaluation for anastomotic stenosis in SVG grafts. (A) Forest plot showing odds ratios (OR) and 95% confidence intervals (CI) from the binary logistic regression analysis. DF was an independent predictor of anastomotic stenosis. The vertical dashed line indicates OR=1. *P < 0.05. (B) ROC curve showing a model AUC of 0.768（95% CI:

0.621-0.869）, with an optimal cutoff value of 0.796, sensitivity of 74.6%, and specificity of 78.6%.

Similar to normal LIMA grafts, normal SVG grafts also exhibited a biphasic waveform with sustained diastolic perfusion, no retrograde flow component, a smooth waveform, and no sharp peaks (Figures 4A, 4B).

Competitive flow in SVG grafts presented with low flow and high pulsatility characteristics (Figures 4C, 4D). This manifested as pronounced systolic retrograde flow, sharp peak waveforms below the baseline, elevated PI, and significantly reduced DF. Figure 4C shows moderate competitive flow (MGF=12 mL/min，PI=5.5，DF=46%), with a clearly visible systolic retrograde peak. Figure 4D shows severe competitive flow(MGF=9 mL/min， PI=10.1， DF=45%), exhibiting an extremely disturbed waveform with a sharp retrograde peak. Although the severity of competitive flow differed between the two cases, the diastolic filling fraction (DF%) was significantly lower compared to the anastomotic stenosis group (DF 45%-46%), which can serve as a key adjunctive parameter for etiological differentiation.

The anastomotic stenosis group (Figures 4E, 4F) presented a high-resistance, low-perfusion waveform. The waveform was complex with numerous sharp peaks, suggesting an adverse hemodynamic environment. Compared to the competitive flow group, the anastomotic stenosis group showed no retrograde flow component, and DF was within the normal range (> 50%).

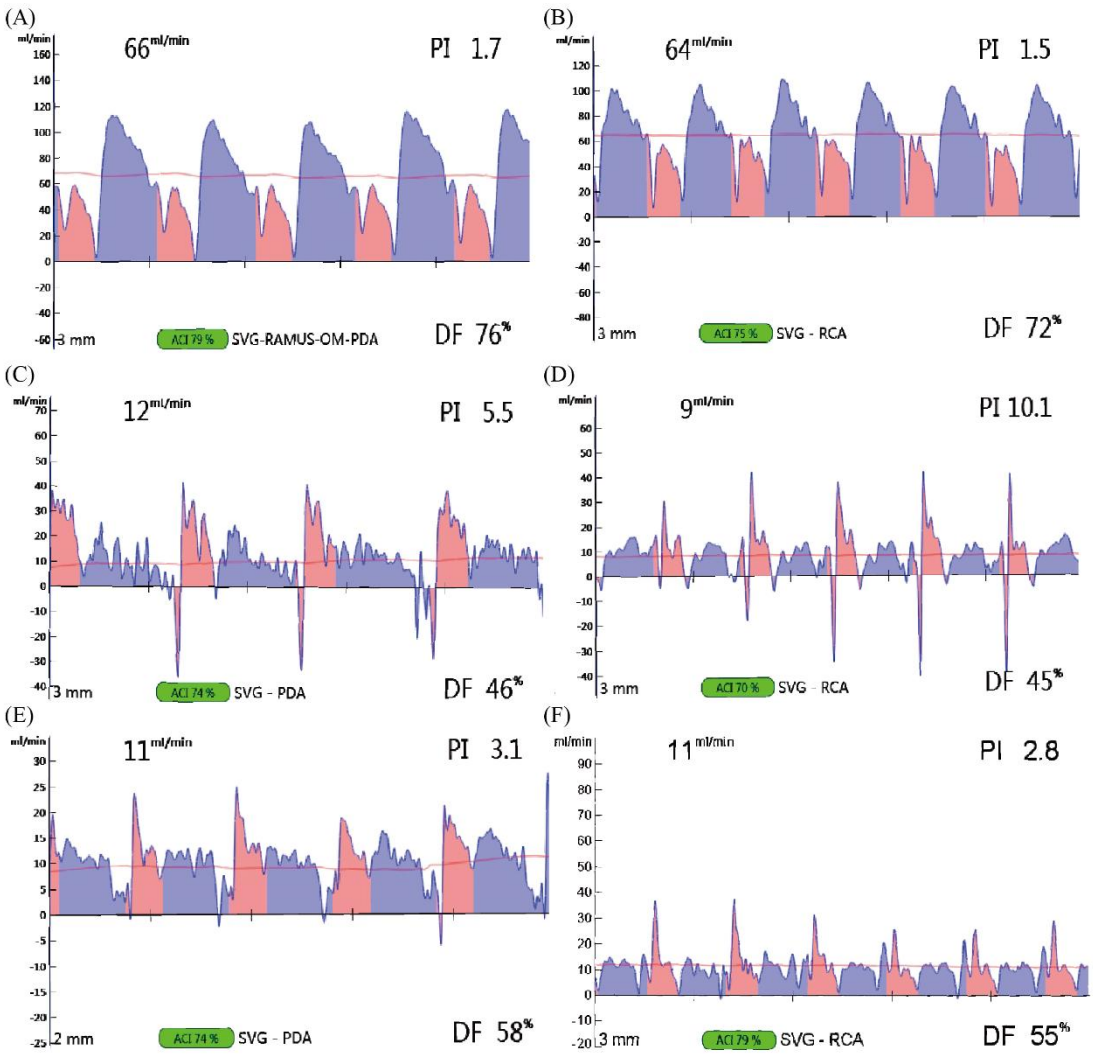


Figure 4 TTFM waveform characteristics of SVG grafts. A-B: Normal SVG flow, no retrograde flow. C-D: Competitive flow (postoperative FFR > 0.80), showing low flow, high pulsatility, significantly reduced DF, and systolic retrograde flow. E-F: Anastomotic stenosis (postoperative FFR < 0.75), showing a high-resistance, flattened waveform with no obvious retrograde flow or only minimal systolic retrograde flow, and normal DF percentage. Horizontal axis: time (seconds, normalized to one cardiac cycle); vertical axis: flow rate (mL/min). Above baseline: antegrade flow; below baseline: retrograde flow. HR: heart rate. Scale bar: 20 mL/min per grid.

3.2 Effect of Competitive Flow on Graft Hemodynamics

3.2.1 Analysis of Hemodynamic Parameters in LIMA Grafts

For LIMA grafts, univariate analysis showed that, compared to the anastomotic stenosis group, the competitive flow group had significantly higher preoperative FFR （0.53±0.24 vs 0.42±0.16, P=0.021）, significantly prolonged RRT （0.15 [0.06–0.26] vs 0.08 [0.03–0.15], P=0.005）， and significantly lower WSS-mean (9.67 [6.86–22.12] vs 16.18 [9.83–37.83] Pa, P=0.004） and WSSG （21.21 [13.29–47.73] vs 36.99 [18.36–101.94] Pa/mm, P=0.019） (Table 3 and Figure 5).

Variable	Competitive flow group (n = 36)	Anastomotic stenosis group (n = 44)	P-value
Pre-FFR	0.527±0.238	0.417±0.162	*0.021
RRT	0.148(0.056-0.262)	0.086(0.031-0.147)	**0.005
Wss-means	9.670(6.862-22.122)	16.180(9.826-37.829)	**0.004
WSSG	21.205(13.295-47.731)	36.994(18.359-101.943)	*0.019
OSI	0.035(0.005-0.061)	0.072(0.003-0.041)	0.142
Aps-means	353.681(121.616-570.450)	299.042(117.930-473.381)	0.468
PI	2.900(2.400-3.800)	2.900(2.200-3.375)	0.474
DF	71.500% (62.750%-79.250%)	74.000% (69.000%-79.750%)	0.090

Negative Flow Ratio	1.700% (0.400%-3.100%)	2.100% (0.200%-3.800%)	0.639
Graft Length	19.358cm (17.976-20.381cm)	19.516cm (17.858-20.769cm)	0.678
Stenosis Length	1.864cm (0.687-3.368cm)	1.161cm (0.418-4.189cm)	0.230

Table 3 Univariate analysis of hemodynamic parameters and TTFM indicators in LIMA grafts

*Note: Data are presented as mean ± standard deviation or median (interquartile range). Comparisons between the competitive flow group and the anastomotic stenosis group: preoperative FFR was analyzed using the independent samples t-test (normally distributed); other variables were analyzed using the Mann-Whitney U test (non-normally distributed). The same applies below. *P < 0.05 indicates statistical significance.

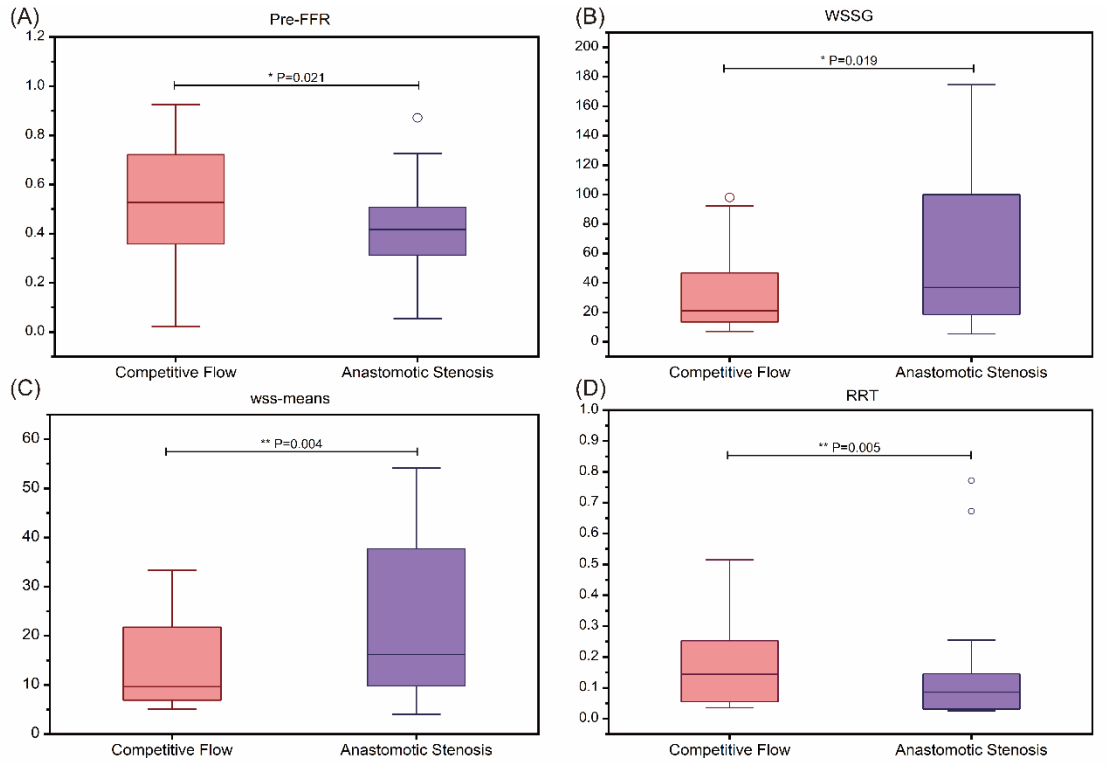
3.2.2 Analysis of Hemodynamic Parameters in SVG Grafts

For SVG grafts, univariate analysis showed that, compared to the anastomotic stenosis group, the competitive flow group had significantly higher preoperative FFR (0.57±0.21 vs 0.44±0.17, P=0.035) and significantly lower DF(62.0% [55.0%-69.3%] vs 68.0% [62.5%-77.5%], P=0.024) (Table 4 and Figure 6).

Variable	Competitive flow group (n = 66)	Anastomotic stenosis group (n = 14)	P-value

Pre-FFR	0.57±0.21	0.44±0.17	*0.035
DF	62.0%（55.0%-69.3%）	68.0%（62.5%-77.5%）	*0.024
RRT	0.280 (0.141-0.719)	0.530 (0.123-1.218)	0.429
WSS-means	5.674 (3.227-14.351)	4.201 (2.922-12.169)	0.583
WSSG	11.971 (7.944-36.618)	7.737 (6.056-30.879)	0.503
OSI	0.015 (0.004-0.066)	0.026 (0.004-0.102)	0.592
Aps-means	487.018 (289.656-817.631)	666.165 (401.264-812.153)	0.296
PI	3.100 (2.400-3.900)	2.800 (2.275-3.725)	0.702
Negative Flow Ratio	1.124% (0.470%-3.725%)	1.147% (0.344%-3.179%)	0.947
Graft Length	15.790cm (12.522-19.120cm)	14.151 cm (11.437-18.312 cm)	0.520
Stenosis Length	0.983cm (0.582-2.483cm)	1.538 cm (0.475-4.766 cm)	0.499

Table 4 Univariate analysis of hemodynamic parameters and TTFM indicators in SVG grafts



276

277 **Figure 5** Comparison of hemodynamic parameters between the competitive flow group
278 and the anastomotic stenosis group in LIMA grafts. (A) Preoperative fractional flow
279 reserve (Pre-FFR); (B) Wall shear stress gradient (WSSG); (C) Mean wall shear stress
280 (WSS-mean); (D) Relative residence time (RRT). Box plots show the interquartile
281 range (box), 1.5× interquartile range (whiskers), and outliers (scatter points). Pre-FFR
282 was normally distributed; the horizontal line within the box represents the mean, and
283 comparisons between groups were performed using the independent samples t-test. The
284 other parameters were non-normally distributed; the horizontal line within the box
285 represents the median, and comparisons between groups were performed using the
286 Mann-Whitney U test. Compared to the anastomotic stenosis group, the competitive
287 flow group showed significantly higher Pre-FFR and RRT, and significantly lower
288 WSS-mean and WSSG. *$P < 0.05$, **$P < 0.01$.

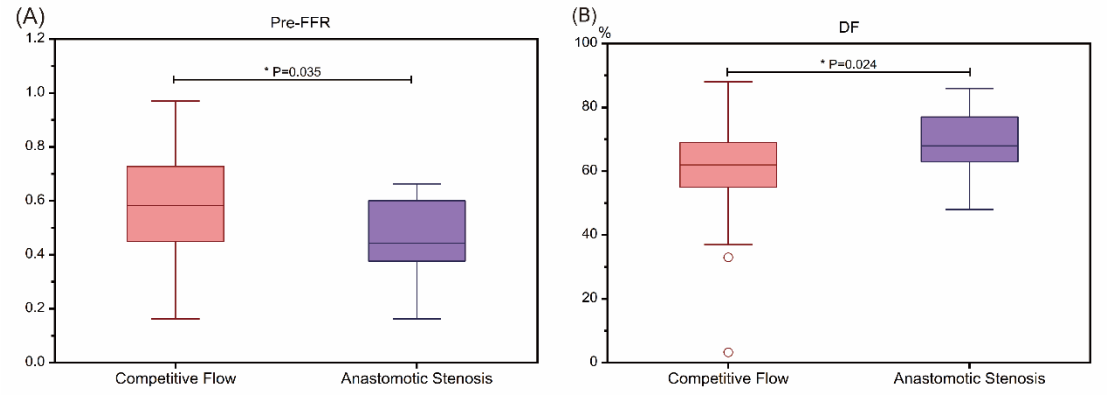


Figure 6 Comparison of hemodynamic parameters between the competitive flow group and the anastomotic stenosis group in SVG grafts. (A) Preoperative fractional flow reserve (Pre-FFR); (B) Diastolic filling fraction (DF). Pre-FFR was normally distributed; the horizontal line within the box represents the mean, and comparisons between groups were performed using the independent samples t-test. DF was non-normally distributed; the horizontal line within the box represents the median, and comparisons between groups were performed using the Mann-Whitney U test. Compared to the anastomotic stenosis group, the competitive flow group showed significantly higher Pre-FFR and significantly lower DF. *P < 0.05.

4. Discussion

This study has two main findings. First, regarding the identification of competitive flow, the preoperative FFR in the competitive flow group was significantly higher than that in the anastomotic stenosis group, and the intraoperative TTFM waveform showed characteristic systolic retrograde flow with high pulsatility, whereas anastomotic stenosis presented as a high-resistance, flattened, sharp-peaked waveform without obvious retrograde flow. Second, there are fundamental differences in the local graft

hemodynamic environment between competitive flow and anastomotic stenosis: competitive flow was primarily characterized by significantly decreased mean wall shear stress (WSS-mean) and wall shear stress gradient (WSSG), as well as significantly prolonged relative residence time (RRT), suggesting an oscillatory shear-induced endothelial injury mechanism. In contrast, anastomotic stenosis did not exhibit such features but rather presented as local hemodynamic alterations due to high-velocity jet flow at the anastomosis. These two conditions affect long-term graft patency through distinctly different pathophysiological mechanisms.

4.1 Identification of Competitive Flow and Its Clinical Significance

Intraoperative low graft flow is a relatively common hemodynamic abnormality in coronary artery bypass grafting. Its etiology may stem from mechanical obstruction due to anastomotic technical defects or from functional hypoperfusion due to competitive flow. However, currently, there is a lack of reliable clinical indicators to differentiate between these causes, which may lead to intraoperative misjudgment by surgeons. The risk of misjudgment is bidirectional: if anastomotic stenosis is misinterpreted as competitive flow, necessary technical revision may be delayed, leading to perioperative myocardial infarction (PMI) and significantly increasing 30-day postoperative mortality, with over 80% of PMI attributed to early graft failure[20, 21]. Conversely, if competitive flow is misinterpreted as anastomotic stenosis, it may lead to unnecessary anastomotic revision, potentially damaging an otherwise patent graft.

Therefore, this study integrated preoperative FFR, anatomical data, and intraoperative TTFM waveform characteristics to construct an etiological differentiation model for low-flow grafts, aiming to address the aforementioned clinical challenges. For LIMA grafts, preoperative FFR is an efficient independent predictor for identifying competitive flow: the higher its value, the higher the likelihood of competitive flow. This finding aligns with reports by Ishida et al.[16] and Kim et al.[17]. Combined with TTFM-derived DF and retrograde flow characteristics, this model provides a quantifiable intraoperative decision-making basis. For SVG grafts, this study found that DF is a key indicator for differentiating anastomotic stenosis, whereas preoperative FFR showed borderline significance for predicting competitive flow, consistent with the mechanism reported by Nakajima et al., whereby competitive flow impairs diastolic perfusion[4]. This difference reflects the fundamental differences in hemodynamic characteristics between venous and arterial grafts: as an elastic artery, the LIMA has good wall compliance; competitive flow primarily affects systolic flow components but has less impact on the proportion of diastolic antegrade flow, thus DF is relatively preserved[1]. However, its endothelial cells are extremely sensitive to changes in wall shear stress; low wall shear stress and high oscillatory shear index readily induce vascular intimal hyperplasia[15]. In contrast, as a venous graft, the SVG has a thinner wall and lower compliance, making it more sensitive to mechanical obstruction caused by anastomotic stenosis, manifested as elevated PI with decreased DF. Moreover, competitive flow can significantly impair its diastolic perfusion efficiency (significantly reduced DF, see Table 4). Therefore, intraoperative TTFM

waveform analysis to observe the presence of retrograde waveforms, combined with DF values, can precisely differentiate the specific etiology of low-flow grafts based on preoperative FFR assessment.

Regarding the determination of FFR cutoff values, this study innovatively used postoperative FFR as the core indicator for differentiating competitive flow from anastomotic stenosis. Postoperative FFR can more accurately reflect the pathophysiological microenvironment of low flow compared to angiography. Toth et al.[22] confirmed that low postoperative FFR is an independent risk factor for predicting graft occlusion and major adverse cardiovascular events (MACE). Furthermore, a clinical case study by Zhang et al.[20] further indicated that measuring FFR via the LIMA postoperatively can accurately identify functional stenosis distal to the anastomosis that is difficult to diagnose by angiography. Regarding the definition of competitive flow using a postoperative FFR > 0.80, this threshold is consistent with the functionally significant stenosis cutoff established by ESC guidelines. Moreover, multiple studies have confirmed that target vessels with an FFR > 0.80 have adequate native blood supply, making them prone to competitive flow after grafting. A recent CT-FFR study predicted an optimal cutoff value of 0.774 for postoperative competitive flow[17], validating the rationale for using FFR > 0.80. Regarding the definition of anastomotic stenosis, the cutoff value of postoperative FFR < 0.75 is recognized by the 2018 ESC/EACTS guidelines as a prognostic threshold for severe ischemia [17]. A CFD study by Kumar et al.[13] demonstrated that local geometric stenosis induces a sharp amplification of the pressure gradient across the lesion, with pressure drop being the

direct physical driver of hemodynamic deterioration. After CABG, FFR < 0.75 indicates significant flow-limiting physical obstruction at the distal anastomosis, whereas competitive flow corresponds to a high-pressure state with FFR > 0.80, forming a stark contrast between the two conditions[23].

From a clinical translation perspective, the differentiation system established in this study facilitates precise intraoperative management. For cases diagnosed as competitive flow, unnecessary anastomotic revision should be avoided; instead, graft perfusion should be improved through medications (e.g., vasodilators) or modified grafting strategies[2]. For cases diagnosed as anastomotic stenosis, timely technical revision should be performed to ensure long-term patency. Furthermore, this system also has value for preoperative strategy formulation: when the FFR of the stenotic coronary artery approaches or exceeds the gray zone (0.75-0.80), the risk of competitive flow in LIMA grafts is high. Bypass surgery may be avoided, or intensified medical conservative therapy may be prioritized. Given that venous grafts have better tolerance to competitive flow[23] and that two different revascularization strategies—bilateral internal thoracic artery (BITA) in situ grafting versus unilateral internal thoracic artery combined with saphenous vein (ITA+SVG)—are less disturbed by chronic competitive flow[4, 21], if revascularization of such vessels is deemed necessary, SVG grafts or special revascularization strategies are recommended, avoiding terminal anastomosis[4] to reduce the risk of early postoperative graft failure.

4.2 Hemodynamic Impact of Competitive Flow on Grafts and Its Clinical Translation Significance

In LIMA grafts, the competitive flow group exhibited significantly decreased WSS-mean and WSSG, significantly prolonged RRT, and a borderline significant increase in OSI. These findings are consistent with the conclusions of the analysis by Masoudi et al.[6]. Low WSS combined with high OSI constitutes a pro-thrombotic hemodynamic microenvironment, which can impair endothelial cell function (e.g., the balance of NO and ET), induce intimal hyperplasia, and compromise long-term graft patency[12]. In contrast, in SVG grafts, only DF showed a significant difference; parameters such as WSS-mean, WSSG, OSI, and RRT were not significantly different between the competitive flow group and the anastomotic stenosis group. This differential response based on graft type suggests that arterial and venous grafts have different sensitivities to competitive flow. As an arterial graft, the LIMA's endothelial cells are more sensitive to changes in wall shear stress and are prone to developing the "string sign." In contrast, the SVG, as a venous graft, is more sensitive to anastomotic technical defects but relatively tolerant to the hemodynamic changes induced by competitive flow. This finding is consistent with the previous conclusion that "venous grafts have better tolerance to competitive flow"[4]. Notably, previous hemodynamic studies (including those by Mao et al. and Ahmad et al.) have primarily focused on arterial grafts, implicitly assuming that the impact of competitive flow is universal. The present study found that SVG grafts showed no significant differences in key hemodynamic parameters, exhibiting significant heterogeneity compared to LIMA

grafts. This suggests that the previous conclusion derived from LIMA studies—that "OSI > 0.2 indicates competitive flow"[6]—is not applicable to venous grafts. SVGs are relatively tolerant to the oscillatory shear environment induced by competitive flow; their low flow primarily reflects anastomotic technical defects rather than the hemodynamic hazard of competitive flow. This finding corrects the previous misconception that treated the hemodynamic responses of arterial and venous grafts equivalently.

The differences in the hemodynamic environment revealed by this study provide important references for postoperative individualized management. For patients with low wall shear stress, high oscillatory shear index, and prolonged relative residence time caused by competitive flow—constituting a pro-thrombotic hemodynamic microenvironment—postoperative intensified antiplatelet therapy (e.g., dual antiplatelet therapy extended beyond one year)[8] and more intensive imaging follow-up (e.g., CT angiography within the first postoperative year)[2] may be necessary to prevent early graft failure. In contrast, for lesions with high-velocity jet flow due to anastomotic stenosis, once the local hemodynamic environment returns to normal after surgical revision, conventional anticoagulation regimens may suffice. This hemodynamic phenotype-based risk stratification strategy moves beyond the traditional, coarse management model that relies solely on graft type, offering a new approach for precise antithrombotic therapy after CABG.

4.3 Limitations and Future Directions

This study has several limitations. First, the sample size is relatively limited, particularly the small sample size in the SVG group, which may affect the stability and generalizability of the multivariate analyses. Second, postoperative FFR measurements were performed one week after surgery, a time point potentially influenced by early inflammatory responses or unstable postoperative physiological status, and thus may not fully reflect the true hemodynamic status of the graft during the stable phase. More critically, current research on the characteristics of competitive flow is limited to the first postoperative week, lacking medium- and long-term follow-up data. This may not fully capture the long-term hemodynamic competitive state and its cumulative effects on graft intimal hyperplasia[4].

Future research should focus on the following areas. First, expand the sample size, particularly by including more competitive flow cases in SVG grafts, to validate the stability of the differentiation model established in this study across different graft types. Second, extend the data collection period. Through CTA follow-up and FFR calculations at six months, one year, or even longer post-surgery, the long-term impact of competitive flow on graft patency can be observed. Establishing a quantitative relationship between the severity of competitive flow and the time to graft failure will further advance the study of competitive flow characteristics.

This study is the first to systematically establish an etiological differentiation system for low-flow grafts based on postoperative FFR and TTFM parameters. It

clarifies the differences in the mechanisms of low flow between LIMA and SVG grafts, providing important theoretical foundations and clinical tools for risk assessment and individualized management of graft failure after CABG.

5. Materials and Methods

This study aims to develop a comprehensive method based on computational fluid dynamics (CFD) by constructing a patient-specific 0D-3D geometric multiscale model. This model serves a dual purpose: first, to non-invasively calculate patient-specific FFR based on CTA data; second, to simulate the local postoperative hemodynamic environment, thereby revealing the fundamental differences in the local hemodynamic microenvironment between low-flow grafts caused by competitive flow versus anastomotic stenosis.

5.1 Patient Demographics and Inclusion/Exclusion Criteria

This study retrospectively collected data from X patients who underwent CABG at Peking University People's Hospital. The collected data included:

Preoperative imaging data: Coronary computed tomography angiography (CTA) data, with an image resolution of 512 * 512 pixels and a slice thickness of 1 mm.

Physiological parameters: Heart rate, systolic blood pressure, diastolic blood pressure, and cardiac output (measured by Doppler ultrasound).

Surgical information: Graft type (left internal mammary artery, LIMA / saphenous vein graft, SVG), anastomosis location, and intraoperative transit-time flow measurement (TTFM) waveforms.

Postoperative validation data: CTA performed one week after surgery.

5.2 Construction of the Geometric Multiscale Coupling Model

To differentiate the etiologies of competitive flow and anastomotic stenosis and to thoroughly analyze the fundamental differences in their respective hemodynamic microenvironments, this study constructed a patient-specific geometric multiscale coupling model based on the aforementioned postoperative CTA data. Using computational fluid dynamics methods, this model enables, on the one hand, the non-invasive calculation of FFR as a functional classification basis, and on the other hand, the quantitative acquisition of local hemodynamic parameters such as WSS and OSI[15], thereby establishing a comprehensive research framework that spans from macroscopic functional assessment to microscopic mechanistic analysis.

5.2.1 Three-Dimensional Model Reconstruction

Based on patient-specific postoperative coronary CTA data, we constructed personalized three-dimensional (3D) vascular models. All patients signed informed consent forms. Mimics 17.0 was used for 3D reconstruction. After importing the CTA images into the software, the aorta and coronary arteries were segmented using thresholding. Appropriate thresholds were selected to segment the aorta and main coronary arteries, while manual adjustments were made for the distal small coronary

arteries. After selecting the target regions, a 3D model of the coronary artery system was reconstructed (as shown in Figure 1D). Following the 3D model construction, ANSYS ICEM CFD software was employed for mesh generation and physical parameter settings. All meshes underwent sensitivity analysis to obtain mesh files suitable for finite element analysis.

(For the computational settings, the vascular wall was assumed to be rigid. Blood was simplified as an incompressible Newtonian fluid, with a density of 1050 kg/m³ and a dynamic viscosity of 0.0035 Pa·s. The blood flow was governed by the Navier-Stokes equations and assumed to be laminar.)

5.2.2 Construction of the Geometric Multiscale Coupling Model

The lumped parameter model in this study consists of three core modules (as shown in Figure 1A-C): the cardiac module, the systemic circulation module, and the coronary module.

Cardiac Part: A constant pressure source simulates left atrial pressure, while two diodes simulate the mitral and aortic valves, respectively. Resistance Rlv and inductance Lla simulate the resistance and inertia of blood flow through the mitral valve. Resistance Rlv and inductance L1 simulate the resistance and inertia of blood flow through the aortic valve, and capacitance C1 simulates aortic elasticity.

The variable capacitor C(t) is used to simulate left ventricular contraction, with its value adjusted by the left ventricular pressure-volume relationship. The relevant function is as follows:

$$E(t) = \frac{P(t)}{V(t) - V_0} \tag{2-1}$$

In the above function, E(t) is the time-varying elastic modulus, P(t) is the time-varying left ventricular pressure (mmHg), V(t) is the time-varying left ventricular volume (ml), and V_0 is the minimum left ventricular volume (ml).

Systemic Circulation Part: Resistance Rp represents arterial flow resistance, resistance Rd represents capillary and venous flow resistance, and capacitance C represents arterial elasticity.

Coronary Part: Resistance R represents coronary arterial flow resistance, resistance R_m represents coronary microcirculation resistance, resistance R_v represents coronary venous resistance, capacitance C represents arterial elasticity, and capacitance C_{im} represents coronary microvascular elasticity. A variable power source connected to C_{im} has a value equal to the left ventricular pressure.

Model parameters were personalized and optimized using a simulated annealing algorithm, with the objective function being the patient's measured aortic pressure waveform and cardiac output. This ensured that the model output was highly consistent with the individual's physiological state, matching the patient's clinical data for systolic pressure, diastolic pressure, and cardiac output.

5.2.3 Geometric Multiscale Coupling Model

To address the limitations of setting boundary conditions for the 3D model, we bidirectionally coupled the local 3D model with the lumped parameter model to

construct a geometric multiscale model (shown in Figure 7). Within this framework, the lumped parameter model provides flow boundary conditions at the inlet and pressure boundary conditions at the outlet for the 3D model. In turn, the 3D model feeds back the calculated interface pressure and flow to the lumped parameter model.

The coupling between the two dimensionally different models at each time step was calculated using Equations (2) and (3). This iteration was performed at each time step until convergence was achieved[18].

$$\overline{P_{3D,in}} = \frac{1}{A_{3D,in}} \int_{\Gamma_{in}} P d\gamma = P_{0D,in} \tag{2-2}$$

$$Q_{3D,out} = \rho \int_{\Gamma_{out}} \mathrm{u} \cdot \mathrm{n}_t d\gamma = Q_{0D,out} \tag{2-3}$$

After constructing the CABG multiscale model, calculations were performed using ANSYS-CFX software.

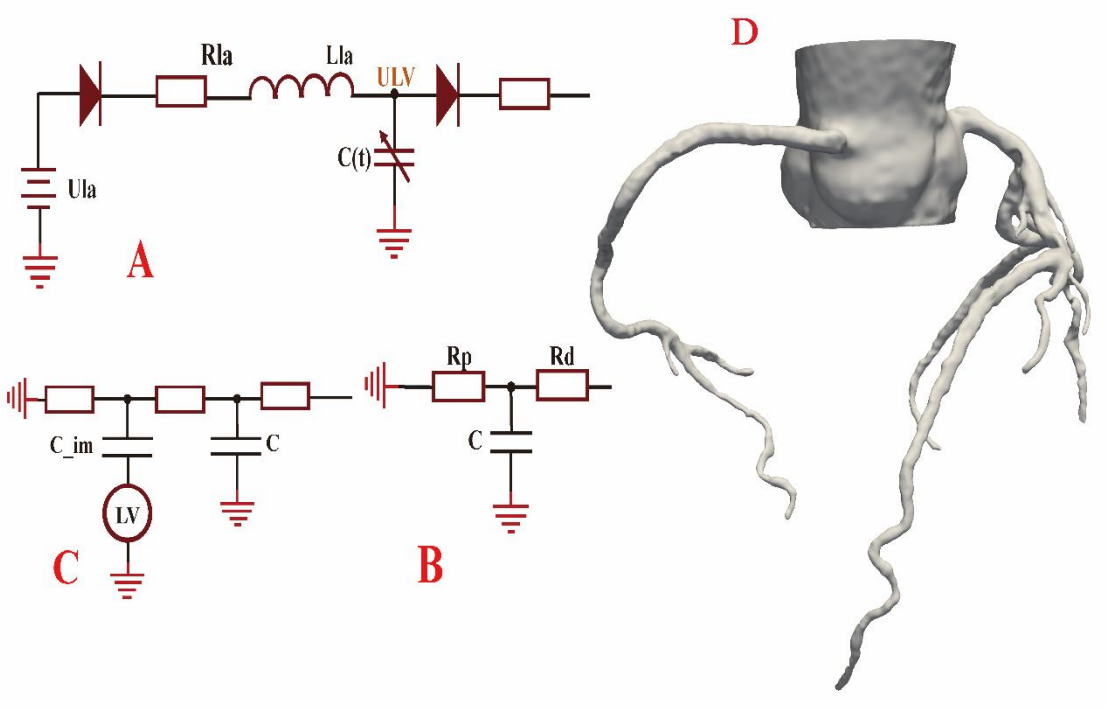


Figure 7 Schematic diagram of the geometric multiscale coupling model. (A-C) Lumped parameter model (0D): (A) Cardiac module; (B) Systemic circulation module; (C) Coronary module. (D) Patient-specific 3D reconstruction of the coronary artery. The red lines indicate the bidirectional coupling interfaces between the 0D and 3D models, where pressure and flow data are exchanged at each time step.

5.3 Calculation of FFR

After model construction, it was necessary to obtain functional graft assessment indicators to distinguish between competitive flow and anastomotic stenosis. FFR, the gold standard for assessing the functional significance of coronary stenosis, can effectively reflect the myocardial perfusion status downstream of the graft. Based on the geometric multiscale coupling model described above, this study reconstructed the 3D anatomical structures of the graft and target vessels using CTA images obtained one week post-surgery. After adjusting the parameters to the normal state, all coronary resistances were multiplied by 0.24 to simulate the maximal hyperemic state induced by adenosine injection during FFR measurement. Subsequently, the pressure at 3 cm distal to the stenosis and the pressure in the aorta were extracted, and their ratio was defined as the FFR value for that stenosis. The non-invasive FFR calculation model was thus established, and its accuracy has been clinically validated.

5.4 Screening of Low-Flow Grafts and Diagnostic Criteria for Competitive Flow

5.4.1 Preliminary Screening of Low-Flow Grafts Based on TTFM

Intraoperative transit-time flow measurement (TTFM) is the preferred method for clinically assessing immediate graft patency. Referring to relevant literature standards and clinical practice consensus[2, 16], this study defined a mean graft flow (MGF) of ≤ 20 mL/min as a low-flow graft. This threshold typically suggests high graft resistance or inadequate perfusion and is an indicator of high risk for potential graft failure. Based on this criterion, we preliminarily screened 130 cases of low-flow left internal mammary artery (LIMA) grafts and 171 cases of low-flow saphenous vein graft (SVG) grafts from the overall sample.

5.4.2 Etiological Differentiation and Grouping Based on Postoperative FFR

Although both competitive flow and anastomotic stenosis manifest as low graft flow, their underlying hemodynamic mechanisms are fundamentally different. Competitive flow originates from pressure competition between the native coronary artery and the graft, whereas anastomotic stenosis results from mechanical obstruction. Given that traditional morphological assessment (e.g., angiography) only reflects anatomical luminal stenosis and cannot truly represent the flow conduction efficiency of the revascularized pathway, this study innovatively employed postoperative computational FFR as the core indicator for etiological differentiation.

Competitive Flow Group: Postoperative FFR > 0.80. This cutoff is based on the ESC/EACTS guidelines on myocardial revascularization for diagnosing functionally significant stenosis (FFR ≤ 0.80)[8]. An FFR > 0.80 indicates preserved pressure reserve in the target vessel, suggesting that the low graft flow primarily results from the hydrodynamic opposition generated by robust antegrade flow in the native artery, rather than from physical obstruction at the anastomosis.

Anastomotic Stenosis Group: Postoperative FFR < 0.75. This cutoff is derived from the specific threshold established by Pijls et al.[19] that is highly correlated with myocardial ischemia (sensitivity 88%, specificity 100%). An FFR below this threshold indicates a low-pressure, ischemic state distal to the target vessel, where the low graft flow is caused by the high flow resistance resulting from mechanical stenosis at the anastomosis.

Data Screening and Final Enrollment: To ensure the purity of the groups, cases falling into the equivocal gray zone (FFR between 0.75 and 0.80) were excluded, as were outliers with poor image quality that prevented 3D reconstruction. The final valid data included for statistical analysis were: 80 cases (screened from 130 cases) of LIMA grafts (competitive flow and anastomotic stenosis combined), and 80 cases (screened from 171 cases) of SVG grafts (competitive flow and anastomotic stenosis combined).

5.5 Statistical Analysis Methods

SPSS 29.0 (IBM Corp., Armonk, NY, USA) was used to analyze the LIMA and SVG graft groups separately. First, the Shapiro-Wilk test was used to assess the

normality of continuous variables. Continuous variables following a normal distribution were expressed as mean ± standard deviation (Mean ± SD), and comparisons between groups were performed using a two-sided independent samples t-test. Variables not following a normal distribution were expressed as median (interquartile range) [M (IQR)], and comparisons between groups were performed using the two-sided Mann-Whitney U test. Categorical variables were expressed as frequency (percentage), and comparisons between groups were performed using the χ^2 test or Fisher's exact test.

For the identification of competitive flow, PI, DF, preoperative FFR, graft length, and stenosis length were included in a multivariate binary Logistic Regression Model (enter method), with competitive flow/anastomotic stenosis as the dependent variable, to analyze the independent predictive value of each indicator. Results were expressed as odds ratios (OR) with 95% confidence intervals (CI). The Hosmer-Lemeshow test was used to assess the goodness-of-fit of the model.

Based on the Logistic Regression Model, a receiver operating characteristic (ROC) curve was plotted, and the area under the curve (AUC) with its 95% CI was calculated to evaluate the discriminative performance of the model. All statistical tests were two-sided, and a P-value < 0.05 was considered statistically significant.

5.6 Hemodynamic Assessment of Vessels Associated with Competitive Flow

Competitive flow is recognized as a key pathological factor leading to long-term postoperative graft failure. The chronic damage it inflicts on the graft often begins with an adverse local fluid environment. Numerous studies have confirmed the significant impact of hemodynamic characteristics [e.g., wall shear stress (WSS), oscillatory shear index (OSI), and relative residence time (RRT)] on graft patency. Therefore, to elucidate the mechanism by which competitive flow leads to graft failure, this study employed hemodynamic parameters as surrogate indicators for assessing the risk of early vascular injury. Based on the simulation results from the multiscale coupling model described above, we calculated and extracted the flow waveform at the graft, as well as hemodynamic parameters including WSS and OSI.

$$WSS = -\mu \frac{du}{dr}$$

$$OSI = 0.5\left[1 - \frac{\left|\int_0^T \overrightarrow{WSS} dt\right|}{\int_0^T \left|\overrightarrow{WSS}\right| dt}\right]$$

6. Declarations

1. **Ethics Approval:**

Approval and consent to participate This study was reviewed and approved by the Medical Ethics Committee of Beijing University of Chinese Medicine (Ethics Approval No. 2022BZYL1009).

2. **Consent for publication:**

Informed consent forms have been signed by all participants.

3. Availability of data and materials:

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

4. **Conflicts of Interest:**

The authors declare no conflicts of interest.

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6. Authors contribution:

KC was responsible for constructing the coronary artery bypass grafting model and drafting the manuscript. GL was responsible for extracting the hemodynamic characteristics of the bypass grafts. ZZ was responsible for clinical data acquisition. YC was responsible for data analysis. BL was responsible for statistical analysis and data integration. YF assisted in extracting the hemodynamic parameters of the

bypass grafts. SY was responsible for generating the statistical figures. BM was responsible for supervision and guidance.

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