The Heart Surgery Forum

Heart Surg. Forum **2026**; 29(5): 48097
https://doi.org/10.31083/HSF48097

Review

Common Risk Factors, Treatment Strategies and the Current Situation of Anticoagulation Strategies for Postoperative Bleeding and Embolism After Left Ventricular Assist Device Implantation

Shengjie Ning[1,†], Chenguang Pan[1,†], Ruoyu Zhang[1], Li Yin[1], Zhibing Qiu[1,*]

[1]Department of Thoracic and Cardiovascular Surgery, Nanjing First Hospital, Nanjing Medical University, 210006 Nanjing, Jiangsu, China
*Correspondence: qiuzhibing2009@163.com (Zhibing Qiu)
[†]These authors contributed equally.

Academic Editor: Anton Sabashnikov
Submitted: 8 November 2025 Revised: 7 March 2026 Accepted: 17 March 2026 Published: 28 May 2026

Abstract

End-stage heart failure is the final stage of heart disease. Patients with end-stage heart failure receive much attention due to their often critical condition. Due to the shortage of donors for heart transplantation, left ventricular assist devices (LVADs) have become an important treatment method to replace the heart's pumping function. Post-operative bleeding and embolism are among the most common complications after LVAD implantation, seriously threatening the recovery and survival of patients. This article systematically classifies the risk factors of bleeding and embolism into either patient-related, LVAD-related, or exogenous. It analyzes the pathogenic differences of adverse events across different device types,exploring the treatment and prevention of gastrointestinal bleeding, stroke, and pump thrombosis. This article highlights the importance of adjusting anticoagulation regimens, endoscopic interventions, and imaging examinations, as well as preoperative optimization of high-risk patients and management of special clinical scenarios. The development ofthird-generation fully magnetically levitated LVADs make it possible to provide more flexible and precise anticoagulation regimens in clinical practice. Based on the use of unfractionated heparin for bridging in the early postoperative period and long-term administration of warfarin, treatment plans should be adjusted according to different clinical situations to increase the time in therapeutic range and the application of new anticoagulant drugs should be explored to improve the prognosis of LVAD patients, optimizing the anticoagulation strategy in this cohort.

Keywords: risk factors; treatment strategies; anticoagulation strategies; left ventricular assist device; pump thrombosis; device type

1. Introduction

Heart failure, as the end-stage of heart disease, attracts significant attention due to its severe prognosis. In China, approximately 8.9 million adults aged ≥35 years have heart failure, with a prevalence of 1.3% [1]. Heart transplantation remains the most effective treatment, but donor shortage, immune rejection, and matching limitations restrict its application to only a minority of patients. Left ventricular assist devices (LVADs) have thus become a critical intervention. With technological evolution from pulsatile to continuous-flow devices, and then to third-generation fully magnetically levitated centrifugal devices, the incidence of adverse events such as device failure and thromboembolism has been significantly reduced, but bleeding and embolism remain the most common and fatal complications after LVAD implantation. Postoperative bleeding and embolism are major challenges, with gastrointestinal bleeding affecting 5%–30% of patients [2] and stroke occurring in 6.7%–29.7% [3], both severely impacting outcomes. Identifying risk factors and strengthening preventive measures are therefore essential for improving patient prognosis. This article reviews perioperative risk factors, treatment strategies, and current anticoagulation protocols for bleeding and embolism after LVAD implantation, with a systematic analysis of the latest domestic and international guidelines, device-specific differences, and management of special clinical scenarios.

2. Common Risk Factors for Postoperative Bleeding and Embolism

The main causes of postoperative bleeding and embolism can be categorized into patient-related factors, LVAD-related factors, and exogenous factors, which influence intensive care unit stay, hospital stay, recovery quality, reoperation rate, and mortality. For example, reoperation for hemostasis occurs in 30% of adult LVAD patients and in up to 50% of pediatric patients [4]. Pump thrombosis and ischemic stroke, as severe embolism-related complications, carry a mortality rate of 50% [4]. Bleeding and thrombosis events are rarely caused by a single factor, but mostly by the synergistic effect of multi-dimensional factors. For example, the combination of preoperative liver dysfunction (patient-related), high shear stress of the device (LVAD-related), and excessive anticoagulation (exogenous) will significantly amplify the risk of fatal bleeding.

2.1 Preoperative Risk Factors (Patient-Related Core Factors)

Advanced Age: Older age is a predictor of LVAD outcomes. Compared with younger patients, those ≥75 years exhibit increased gastrointestinal bleeding rates but lower device thrombosis rates after LVAD implantation [5]. Advanced age is also associated with higher stroke incidence, lower survival, and greater risk of sequelae, though some studies suggest age <65 years may increase hemorrhagic stroke risk [6]. Mechanisms may include increased blood viscosity, vascular structural changes, coagulation-fibrinolysis disorders, prior antithrombotic use, altered blood flow patterns, and reduced vascular adaptability. Despite these risks, advanced age is not currently an exclusion criterion for LVAD implantation.

Gender: Female sex is an independent risk factor for postoperative bleeding. INTERMACS database analysis (2012–2017) of continuous-flow LVAD recipients showed higher intracranial and non-gastrointestinal/non-CNS bleeding risks in females after covariate adjustment, though no clear association with gastrointestinal bleeding was found [7].

Atrial Fibrillation (AF): Preoperative AF, a common arrhythmia, increases postoperative embolism risk, particularly affecting long-term cerebrovascular outcomes, though overall long-term survival is not significantly reduced [8]. Thrombogenesis is linked to hemodynamic changes, and left atrial appendage closure (LAAC) may reduce embolism risk. Computer simulation studies have verified the potential of LAAC in reducing thromboembolic events in LVAD patients [9], and a real-world retrospective study including 310 patients further confirmed that concomitant LAAC during LVAD surgery can significantly reduce the incidence of ischemic cerebrovascular accidents without increasing perioperative mortality and complications, and it is an independent protective factor for reducing postoperative ischemic stroke (HR 0.38, 95% CI 0.15–0.97, $p = 0.043$) [10]. Anticoagulation decisions can be guided by CHA2DS2-VASc (embolism risk) and HAS-BLED (bleeding risk) scores, with anticoagulation strongly recommended for patients with hypertrophic cardiomyopathy [11].

Malignancy: The relationship between cancer and LVAD-related embolism is complex. Tumor cells trigger coagulation, promoting thrombosis, while vascular changes (e.g., aneurysms, venous abnormalities) and anticancer therapies increase bleeding risk. Solid tumors are associated with higher mortality, major bleeding, and pump thrombosis [12].

Liver and kidney dysfunction: Liver and kidney dysfunction increases the propensity for bleeding and embolism after LVAD implantation. Studies demonstrate that chronic liver disease patients exhibit higher rates of major bleeding, platelet transfusions, and subarachnoid hemorrhage post-LVAD surgery, though with a lower risk of ischemic stroke [13]. Potential mechanisms include: ① reduced synthesis of coagulation factors and abnormal vitamin K metabolism in hepatic insufficiency, impairing vitamin K-dependent coagulation factor production and inhibiting platelet function via metabolic toxins; ② thrombopoietin abnormalities leading to platelet count reduction; ③ accumulation of toxic substances (e.g., elevated homocysteine) directly damaging vascular endothelium and disrupting vasoactive substance balance, causing vasomotor dysfunction and heightened bleeding risk. Consequently, LVAD implantation contraindications include TBIL >3 mg/dL, cirrhosis, or a Model for End-Stage Liver Disease (MELD) score >17 [11]. Additionally, renal dysfunction may impair drug metabolism or coagulation factor activation, increasing bleeding risk and necessitating early postoperative renal replacement therapy, thus rendering long-term renal replacement therapy a contraindication for LVAD [11].

Diabetes: As a common preoperative comorbidity, a unified consensus has not been reached in current literature regarding the impact of diabetes on postoperative adverse events. Crugnola *et al.* [14] found that it had no significant effect on postoperative gastrointestinal bleeding, but the composite endpoint incidence of all thromboembolic events was higher in the diabetes group. Additionally, retinal evaluation should be performed in diabetic patients before surgery to avoid retinal hemorrhage caused by postoperative anticoagulation [15].

Hematological disorders: Patients with hematological disorders significantly increase the risk of postoperative bleeding and embolism. Studies indicate that such patients have a high risk of bleeding, thrombosis, and neurological events during device support, leading to early mortality, with a two-year survival rate of only 49% [16]. It is recommended that postoperative anticoagulation strategies be adjusted under the guidance of hematology specialists.

Other preoperative clinical risk factors: Some patients with dilated cardiomyopathy have preoperative right-sided heart failure, which is associated with postoperative gastrointestinal bleeding [17]. Peripheral arterial disease, left ventricular noncompaction, a history of stroke, and nutritional status are also risk factors for postoperative adverse events. Obese patients (BMI ≥35kg/m^2) have a higher incidence of postoperative thrombosis, while patients with BMI <25kg/m^2 have significantly increased risks of bleeding and surgical failure [11,15]. Preoperative circulatory support, especially ECMO, depletes coagulation factors and platelets. Additionally, central venous pressure <12 mmHg, pulmonary artery wedge pressure <18 mmHg, and cardiac index >2.2 L/min/m^2 may reduce the impact of blood compatibility adverse events [18].

2.1.1 Preoperative Optimization for High-Risk Patients

Management of non-warfarin-related elevated international normalized ratio (INR): Preoperative elevated INR unrelated to warfarin is mostly caused by liver dysfunction,

vitamin K deficiency, severe malnutrition, or consumptive coagulopathy, which is an independent risk factor for postoperative fatal bleeding [11]. For such patients, systematic etiological assessment should be performed before surgery, including liver function grading, nutritional risk screening, and coagulation factor spectrum detection. Targeted optimization includes vitamin K supplementation (10 mg/day for 3–5 days for vitamin K deficiency), enteral/parenteral nutritional support for malnourished patients, liver function protection and coagulation factor correction for patients with liver insufficiency. The Chinese Expert Consensus on Pre-operative Evaluation of LVAD Candidates (2023) clearly states that persistent unexplained elevated INR is a relative contraindication for LVAD implantation, and surgery should be performed only after coagulation function is effectively corrected [11].

2.1.2 Control of Right Intracardiac Filling Pressures

Sustained elevation of right atrial pressure (RAP) is a core risk factor for postoperative gastrointestinal bleeding, stroke, and right-sided heart failure [19]. Preoperative optimization should aim to control RAP <12 mmHg, with strategies including optimized diuretic therapy, inotropic support (dobutamine, milrinone), pulmonary vasodilators (sildenafil, treprostinil), and preoperative temporary mechanical circulatory support (e.g., Impella, ECMO) for patients with refractory right heart failure. Intraoperative and postoperative maintenance of RAP <16 mmHg can significantly reduce the incidence of adverse blood compatibility events, as recommended by the Chinese Expert Consensus on Early Intensive Care Management After LVAD Implantation (2024) [20].

2.2 Intraoperative Risk Factors (Surgical and Technical Factors)

In cardiac surgery, the rational use of heparin and antifibrinolytic drugs is crucial for controlling the risk of postoperative bleeding and embolism. It is essential to precisely manage the dosage and timing of these medications to balance anticoagulation during extracorporeal circulation with postoperative hemostasis, thereby reducing the risk of postoperative complications. Extracorporeal circulation is generally initiated when the activated clotting time (ACT) exceeds 480 seconds. Notably, recent studies have suggested that thromboelastography may not accurately predict the risk of major postoperative bleeding [21].

The placement of inflow and outflow vessels affects hemodynamics, and improper placement can lead to blood flow stagnation or turbulence. Expert consensus recommends that the inflow vessel should be aligned with the mitral orifice and parallel to the interventricular septum, while the outflow vessel should be cut into a 45-degree bevel and anastomosed to the midpoint of the anterolateral wall of the ascending aorta, approximately 2 cm above the sinotubular junction [22]. Improper spatial relationship between the inflow cannula and the mitral orifice is a core technical risk factor for inflow cannula obstruction and pump thrombosis, which is detailed in Section 2.3.

The vast majority of LVAD implantations are performed on a beating heart under cardiopulmonary bypass without aortic cross-clamping, and thus do not cause global myocardial ischemia-reperfusion injury [22]. Local ischemia-reperfusion injury may only occur in a small number of cases where aortic cross-clamping is required (e.g., concurrent coronary artery bypass grafting, aortic valve surgery, or mitral valve intervention). In this scenario, during the ischemic phase, tissue energy metabolism is dominated by anaerobic glycolysis, leading to intracellular ATP depletion, ion pump dysfunction, and cellular edema; meanwhile, local accumulation of acidic metabolites causes vascular endothelial injury and basement membrane exposure, activating the intrinsic coagulation pathway. During reperfusion, massive production of oxygen-free radicals attacks vascular endothelium, increases vascular permeability, oxidizes platelet membrane glycoproteins, and alters their function. Additionally, this induces an inflammatory response, promoting inflammatory cell infiltration and releasing proteolytic enzymes, further damaging the vascular wall. These combined factors increase the risk of postoperative bleeding. For cases requiring aortic cross-clamping, standardized myocardial protection strategies (cold blood cardioplegia) are essential to reduce myocardial and endothelial injury; for conventional beating-heart LVAD implantation, the core of intraoperative management is hemodynamic stability and coagulation function protection, rather than global myocardial protection.

Inadequate myocardial protection measures (for cases requiring aortic cross-clamping) and prolonged extracorporeal circulation time increase the risk of postoperative bleeding, while the method of intraoperative blood salvage may affect the patient's coagulation function. Incomplete surgical suturing and vascular anastomosis are common human factors contributing to postoperative bleeding. Park *et al.* [23] proposed using 5-0 prolene sutures to anastomose the outflow graft to the aorta to reduce early pinhole bleeding, and decompressing the left ventricle before suturing the inflow cannula adapter to the left ventricular apex can also reduce the risk of postoperative bleeding.

2.3 Postoperative Risk Factors (LVAD-Related and Exogenous Factors)

After LVAD implantation, preventing thrombosis is critical, but the dosages of anticoagulants and antiplatelet drugs (such as warfarin, heparin, and aspirin) must be precisely controlled. Inadequate dosing can easily trigger embolism, obstruct blood flow, and endanger life, while excessive dosing can cause bleeding and affect prognosis. This is the core exogenous factor leading to bleeding and thrombosis events.

Mean arterial pressure (MAP) is a key factor in patient prognosis. Studies have shown that MAP <90 mmHg can reduce the risk of ischemic and hemorrhagic stroke. Compared with systolic blood pressure <100 mmHg, patients with systolic blood pressure >100 mmHg have a higher risk of stroke, with a 19% increased stroke risk for every 5 mmHg increase in systolic blood pressure [24]. However, a long-term MAP <75 mmHg also increases the risk of stroke, and the recommended postoperative MAP target for LVAD patients is 70–90 mmHg [20].

Advanced right-sided heart failure increases the risk of blood compatibility adverse events. Despite LVAD optimization, patients with high RAP have a lower rate of freedom from gastrointestinal bleeding (GIB) and stroke within 1 year. Low MAP (<70 mmHg), narrow pulse pressure (<35 mmHg), and RAP >16 mmHg are all associated with increased GIB risk. A significant decline in RAP waveform is also linked to higher incidences of GIB and stroke. Potential mechanisms for increased GIB risk in right ventricular dysfunction after LVAD implantation include: ① elevated venous pressure causing intestinal mucosal congestion; ② hepatic congestion with associated coagulopathy; ③ intestinal hypoxia increasing the risk of arteriovenous malformations [19].

Stress, a common postoperative condition, contributes to bleeding and embolism through multiple mechanisms. In terms of bleeding, stress enhances the inflammatory response, inhibits platelet production, increases platelet destruction, and releases hormones that disrupt platelet function. It also impairs hepatic synthesis of coagulation factors and causes imbalances in the coagulation-fibrinolysis system, leading to consumption of coagulation factors. Regarding embolism, stress activates the neuroendocrine system and inflammatory responses, triggering coagulation factors and platelet activation to induce hypercoagulability. Concurrently, sympathetic nerve excitation slows blood flow, while LVAD alters blood flow patterns to form eddy currents. Deposition of inflammatory mediators and immune complexes damages the vascular endothelium, promoting thrombus formation.

The high shear stress generated by LVAD operation is the core LVAD-related factor leading to coagulation dysfunction. In recent years, studies have shown that the reduction of three platelet receptors (GPIbα, P-selectin, and PECAM-1) and the increase in intracellular reactive oxygen species in platelets may play a role in postoperative bleeding patients with LVAD, helping to explain the high incidence of spontaneous bleeding during LVAD support [25].

Postoperatively, some patients develop acquired von Willebrand syndrome (AVWS), characterized by reduced von Willebrand factor (VWF), which may be associated with mechanical shear stress injury, inflammatory responses, and other factors. Changes in VWF activity have a strong predictive role for postoperative blood compatibility adverse events. Early postoperative ratios of VWF:Act/Ag and VWF:CB/Ag ≤0.7 can serve as biomarkers for blood compatibility adverse events during long-term LVAD support [26]. Hennessy-Strahs *et al.* [27] developed a bleeding risk score based on the severity of VWF multimer loss and VWF coagulation function, which has a high predictive value for LVAD-related bleeding.

Postoperative requirements for continuous transfusion of >4 units of packed red blood cells, unstable hemoglobin levels, and persistent elevation of lactate dehydrogenase (LDH) >500 IU/L indicate a bleeding risk [4]. Szymanski *et al.* [28] suggested that gastrointestinal bleeding may be associated with an increased risk of ischemic stroke. Perioperative red blood cell transfusion is also an independent exogenous risk factor for both bleeding and thrombosis, as it can induce an inflammatory response, platelet dysfunction, and allogeneic immune reaction, further disrupting the coagulation-fibrinolysis balance.

2.4 Device Type-Related Differences in Bleeding and Thrombosis Pathogenesis and Incidence

Device type is a core determinant of the incidence and pathogenesis of bleeding and thrombosis events, with significant differences across three generations of LVADs, as supported by the INTERMACS and EUROMACS registry data [2,3,29]. Table 1 summarizes the differences in pathogenesis and incidence of bleeding and thrombosis among different generations of LVADs.

First-generation pulsatile LVADs (e.g., HeartMate XVE): These devices mimic the physiological pulsatile flow of the native heart, with large-volume displacement and high mechanical wear. Clinical data show that pulsatile LVADs have a significantly higher incidence of stroke (12%–35% at 1 year), thromboembolic events, and device failure (20%–30% at 2 years), while the incidence of gastrointestinal bleeding is significantly lower (3%–10%) [29]. The pathogenesis of thromboembolism is mainly related to the high mechanical wear of the device, poor biocompatibility of the contact surface, and blood flow stagnation in the pump cavity; the low bleeding risk is due to the low shear stress in the pump cavity, which rarely causes AVWS and platelet receptor degradation. Due to the high incidence of adverse events, pulsatile LVADs have been essentially replaced by continuous-flow devices in clinical practice.

Second-generation axial continuous-flow LVADs (e.g., HeartMate II): These devices have a compact design with a high-speed axial rotor, which significantly reduces device failure rate (5%–8% at 2 years) and thromboembolic events (pump thrombosis incidence 6%–10% at 1 year) compared with pulsatile devices [3]. However, the incidence of gastrointestinal bleeding is significantly increased to 15%–30% [2]. The core pathogenesis is the continuous high shear stress generated by the high-speed rotor, which causes degradation of VWF high-molecular-weight multimers, leading to AVWS, and down-regulation of platelet receptors, resulting in platelet dysfunction. Meanwhile,

Table 1. Differences in pathogenesis and incidence of bleeding and thrombosis among different generations of LVADs.

Device type	Technical characteristic	1-year stroke incidence	2-year pump thrombosis incidence	Gastrointestinal bleeding incidence	Core pathogenesis of adverse events	Recommended INR target
1st-generation pulsatile LVAD (HeartMate XVE)	Pulsatile flow, large displacement, high mechanical wear	12%–35%	20%–30%	3%–10%	High mechanical wear, poor biocompatibility, pump cavity blood stasis; low shear stress rarely causes AVWS	2.5–3.5
2nd-generation continuous axial-flow LVAD (HeartMate II)	High-speed axial rotor, narrow flow gap	10%–20%	6%–10%	15%–30%	High shear stress induces AVWS and platelet dysfunction; narrow flow gap increases flow stasis	2.0–3.0
3rd-generation centrifugal fully maglev LVAD (HeartMate 3)	Maglev rotor, wide flow gap, artificial pulse design	6.7%–12%	<2%	8%–18%	Optimized hemodynamics reduces peak shear stress and AVWS incidence; no mechanical wear and flow stasis	1.7–2.3 (high bleeding risk); 2.0–2.5 (conventional)

LVADs, left ventricular assist devices; INR, international normalized ratio; AVWS, acquired von Willebrand syndrome.

the narrow flow gap of the axial rotor increases the risk of flow stagnation and pump thrombosis.

Third-generation centrifugal fully magnetically levitated LVADs (e.g., HeartMate 3): These devices are the current international mainstream, with a magnetically levitated rotor, wide flow gap, and artificial pulse design. Clinical data show that the pump thrombosis incidence is <2% at 2 years, and the 1-year stroke incidence is reduced to 6.7%–12% [30]. The incidence of gastrointestinal bleeding is also lower than that of axial flow devices, at 8%–18% [2]. The optimized hemodynamic design reduces the peak shear stress in the pump cavity, thereby reducing the incidence of AVWS and platelet dysfunction; the wide flow gap and magnetically levitated design eliminate mechanical wear and flow stagnation, significantly reducing the risk of pump thrombosis.

Correspondingly, the anticoagulation strategies for different device types are also significantly different: pulsatile LVADs require higher anticoagulation intensity (INR target 2.5–3.5) due to high thromboembolic risk; axial flow LVADs require a balance between bleeding and thrombosis risk, with a conventional INR target of 2.0–3.0; centrifugal fully magnetically levitated LVADs can adopt a more flexible low-intensity anticoagulation strategy, with an INR target of 1.7–2.3 for bleeding high-risk patients, as recommended by the International Society for Heart and Lung Transplantation (ISHLT) 2024 consensus [31].

2.5 Perioperative Risk Factors in Special Clinical Scenarios

The risk of bleeding and thrombosis after LVAD implantation varies significantly in special clinical scenarios, and targeted management is required to reduce adverse events, as detailed below:

2.5.1 Patients With Previous Coronary Artery Bypass Grafting (CABG) or Valve Surgery

Previous cardiac surgery leads to extensive mediastinal and pericardial adhesions, which significantly increase the risk of intraoperative bleeding, surgical injury, and prolonged cardiopulmonary bypass time, thereby increasing the incidence of postoperative bleeding and thromboembolism. For such patients, preoperative evaluation of adhesion severity with chest CT is recommended, and intraoperative use of antifibrinolytic drugs and blood salvage technology should be strengthened to reduce coagulation factor depletion.

2.5.2 Patients With Preoperatively Severe Right Ventricular Failure

Severe right-sided heart failure not only increases the risk of postoperative gastrointestinal bleeding and stroke, but also is the leading cause of early postoperative death [19]. For patients with RAP >16 mmHg that is refractory to medical therapy, preoperative temporary mechanical circulatory support (e.g., Impella, right-sided ECMO) is recommended to optimize right-sided heart function and reduce the risk of postoperative adverse events. Perioperative maintenance of right-sided heart function and control of right-sided filling pressure are the core of management.

2.5.3 LVAD Replacement Surgery Due to Pump Dysfunction

Reoperation for pump replacement has a significantly higher risk of bleeding and pump thrombosis [32]. The main mechanisms include adhesion-related surgical injury, coagulation factor depletion during reoperation, and preexisting hypercoagulable state caused by pump thrombosis. For such patients, preoperative anticoagulation bridging

strategy should be strictly formulated, and intraoperative hemostasis and coagulation function management should be strengthened.

2.5.4 Minimally Invasive LVAD Implantation (e.g., Left Thoracotomy, Subcostal Approach)

Compared with median sternotomy, minimally invasive surgery significantly reduces surgical trauma and intraoperative bleeding, with a lower incidence of postoperative major bleeding [33]. However, the limited surgical field may increase the risk of improper cannula placement, thereby increasing the risk of pump thrombosis. For minimally invasive surgery, intraoperative transesophageal echocardiography (TEE) is essential to monitor the position of the inflow cannula and ensure correct placement.

2.5.5 Concurrent Valvular Procedures During LVAD Implantation

Concurrent aortic valve surgery increases the complexity of the operation, prolongs the cardiopulmonary bypass time, and increases the risk of postoperative bleeding [34]. For patients undergoing concurrent valve surgery, the anticoagulation intensity should be appropriately adjusted according to the valve procedure, and the balance between bleeding and thrombosis risk should be strictly monitored.

2.5.6 Temporary Mechanical Circulatory Support (MCS) as a Bridge to Durable LVAD

Preoperative ECMO or Impella support leads to massive depletion of coagulation factors and platelets, endothelial injury, and inflammatory response, which significantly increase the risk of postoperative bleeding and thrombosis. For such patients, preoperative correction of coagulation function, reduction of the inflammatory response, and optimization of MCS parameters are essential to reduce perioperative adverse events.

2.5.7 LVAD as a Bridge to Heart Transplantation

For patients waiting for heart transplantation, the anticoagulation strategy needs to balance the risk of thromboembolism during LVAD support and the risk of perioperative bleeding during transplantation [35]. The ISHLT guideline recommends that the INR target should be maintained at 1.5–2.0 in the near term before transplantation, and anticoagulation should be reversed in a timely manner during the operation to reduce the risk of intraoperative bleeding. After transplantation, the anticoagulation strategy should be adjusted according to the cardiac function and coagulation status of the patient.

Table 2 summarizes the multidimensional risk factors for bleeding and thromboembolic events after LVAD implantation.

3. Treatment Strategies for Postoperative Bleeding and Embolism

Postoperative bleeding and embolism after LVAD implantation significantly affect patient prognosis, with gastrointestinal bleeding and stroke being particularly common. Gastrointestinal bleeding is mostly caused by anticoagulant therapy, necessitating timely adjustment of anticoagulation strategies and endoscopic intervention; stroke may be caused by thromboembolism or hemorrhagic stroke, requiring corresponding emergency management according to its type. Pump thrombosis is a fatal thromboembolic complication with a high mortality rate, requiring standardized graded management. This article briefly discusses the management strategies for gastrointestinal bleeding, stroke, and pump thrombosis after LVAD surgery to provide clinical guidance.

3.1 Postoperative Bleeding Management Strategies

Bleeding most commonly occurs in the upper gastrointestinal tract, particularly in the stomach and duodenum, with small intestinal arteriovenous malformations accounting for 29–44% of gastrointestinal bleeding [36]. For mild bleeding, antithrombotic drugs can be adjusted or discontinued, while closely monitoring the patient's condition, device parameters, and laboratory markers. Upon the first occurrence of bleeding, a gastroenterology consultation should be initiated, and proton pump inhibitors should be administered to protect the gastrointestinal mucosa. If bleeding persists, anticoagulation therapy must be reversed [36], and endoscopy may be necessary to identify the bleeding site and achieve hemostasis. After controlling the bleeding, the anticoagulation regimen should be reassessed, and the INR target range should be lowered according to the device type and patient's bleeding risk. For refractory bleeding, recombinant coagulation factor VIIa or VWF concentrate infusions may be considered, and repeated endoscopic evaluations are recommended, especially when previous treatment targets have been identified [36]. Medications such as octreotide, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and thalidomide, which reduce gastrointestinal bleeding, can be used. When hemoglobin is <80g/L, red blood cell transfusion may be considered. Desmopressin and antifibrinolytic drugs are effective for patients with platelet dysfunction or hyperfibrinolysis without increasing the risk of pump thrombosis [20]. Antiangiogenic therapies with intravenous bevacizumab or low-dose oral pazopanib have also shown benefit in LVAD-related gastrointestinal bleeding patients [37].

3.2 Management Strategies for Ischemic and Hemorrhagic Stroke

The management of ischemic stroke includes immediate assessment of neurological function, suspension of anticoagulant medications, and imaging examinations for definitive diagnosis. For patients with acute ischemic

Table 2. Summary of multidimensional risk factors for bleeding and thromboembolic events after LVAD implantation.

Risk factor category	Specific items	Core risk impact
Patient-related factors	Advanced age, female sex, atrial fibrillation, malignancy, liver and kidney dysfunction, diabetes mellitus, hematological disorders, preoperative right-sided heart failure, malnutrition, prior stroke, etc.	Increase baseline bleeding/thrombosis tendency, affecting long-term prognosis
LVAD-related factors	Device type (pulsatile/axial flow/centrifugal maglev), high shear stress, improper inflow/outflow cannula placement, pump dysfunction, etc.	Induces coagulation dysfunction, pump thrombosis, and hemodynamic disorders
Exogenous factors	Perioperative anticoagulation/antiplatelet therapy, prolonged cardiopulmonary bypass time, perioperative red blood cell transfusion, surgical operation factors, postoperative stress, etc.	Disrupts coagulation-fibrinolysis balance, increases perioperative bleeding risk
Special clinical scenarios	Previous cardiac surgery, severe preoperative right-sided heart failure, LVAD replacement surgery, minimally invasive implantation, concurrent valvular procedures, pre-MCS bridge, bridge to heart transplantation.	Amplifies bleeding/thrombosis risk, requiring targeted management strategies

MCS, mechanical circulatory support.

stroke, device parameters should be monitored to rule out pump thrombosis. Intravenous thrombolysis should be considered for those with an onset time <4.5 hours and no contraindications, but the bleeding risk must be evaluated [38]. Ischemic stroke patients who are ineligible for systemic thrombolysis within 24 hours of symptom onset should undergo urgent CT angiography and be considered for mechanical thrombectomy [36]. In the later stage, the intensity of anticoagulation should be reassessed, with adjustment of the INR target range or transition to low-molecular-weight heparin. For patients with recurrent embolism, combined antiplatelet therapy may be considered, while balancing the bleeding risk. For hemorrhagic stroke, anticoagulants should be discontinued immediately, with reversal of the anticoagulation effect (target INR <1.4) [39], and control of intracranial pressure and blood pressure. After the condition is stable, the restart time and intensity of anticoagulation should be evaluated according to the hematoma absorption, device type, and thromboembolic risk of the patient, as recommended by the Neurocritical Care Society guideline [39].

3.3 Management of Pump Thrombosis

Pump thrombosis is defined as thrombus formation in the inflow cannula, pump cavity, or outflow graft of LVAD, with an overall incidence of 2%–10% after LVAD implantation. Notably, the incidence of right ventricular assist device (RVAD) thrombosis is 2–3 times higher than that of LVAD, which is related to the lower flow rate of the right circulation, the complex anatomical structure of the right heart, and the higher coagulation activity of the systemic venous system [31].

3.3.1 Technical and Clinical Evaluation of Pump Thrombosis

Influencing factors: ① Anatomical and technical factors: small left ventricular cavity size, improper alignment between the inflow cannula and the mitral orifice, contact between the inflow cannula and the ventricular wall or mitral valve apparatus, the presence of intracardiac thrombi or dense myocardial trabeculations, and anastomotic stenosis of the outflow graft [22,31]; ② Patient-related factors: inadequate anticoagulation (Time in Therapeutic Range (TTR) <65%), previous gastrointestinal or cerebral bleeding leading to reduced anticoagulation intensity, driveline infection or systemic infection, hypercoagulable state, and hematological disorders [4,31]; ③ Device-related factors: axial flow devices have a higher thrombosis risk than centrifugal fully magnetically levitated devices, and pump dysfunction caused by mechanical failure increases the thrombosis risk [29].

Diagnostic evaluation system: ① Device parameter monitoring: persistent increases in pump power consumption, abnormal flow waveform, and reduced pulsatility are early signs of pump thrombosis; ② Laboratory examination: persistent elevation of LDH >2 times the upper limit of normal, accompanied by decreased hemoglobin and haptoglobin, is a core biomarker of pump thrombosis; ③ Imaging evaluation: TEE is the first-line examination, which can directly evaluate the position of the inflow cannula, the movement of the rotor, and the presence of thrombus in the pump cavity; cardiac CT angiography can evaluate the patency of the outflow graft and the presence of peripheral thromboembolism [31].

3.3.2 Graded Treatment Strategies

According to the ISHLT 2024 consensus, pump thrombosis is divided into three grades, with corresponding treatment strategies [31]: Table 3 summarizes the graded diagnosis and treatment strategies for LVAD-related pump thrombosis based on the ISHLT 2024 consensus.

• Grade 1 (suspected thrombosis): abnormal device parameters or elevated LDH, no clear thrombus on imaging. Treatment: optimize anticoagulation therapy, maintain

Table 3. Graded diagnosis and treatment strategies for LVAD-related pump thrombosis (based on ISHLT 2024 consensus).

Thrombosis grade	Core diagnostic criteria	First-line treatment strategies	Key monitoring indicators
Grade 1 (suspected)	Abnormal device parameters (elevated power consumption, abnormal flow waveform) OR persistent LDH elevation; no clear thrombus on imaging	Optimize anticoagulation: unfractionated heparin to maintain APTT 50–80 s	LDH level, device power consumption, flow waveform, TEE
Grade 2 (confirmed, no hemodynamic compromise)	Clear thrombus confirmed by imaging; stable device function and hemodynamics; no organ hypoperfusion	Low bleeding risk: intravenous thrombolysis (alteplase); High bleeding risk: intensified anticoagulation + antiplatelet therapy	Thrombus volume change, LDH, hemodynamic status, bleeding signs
Grade 3 (severe, with hemodynamic compromise)	Pump flow obstruction, cardiogenic shock, OR recurrent peripheral thromboembolism	Emergency surgical pump replacement; thrombolysis as bridging therapy for inoperable patients	Hemodynamic parameters, device function, end-organ perfusion

ISHLT, International Society for Heart and Lung Transplantation; LDH, lactate dehydrogenase; APTT, activated partial thromboplastin time; TEE, transesophageal echocardiography; OR, no specific definition.

activated partial thromboplastin time (APTT) 50–80 s with unfractionated heparin, and closely monitor LDH, device parameters, and imaging changes.

• Grade 2 (confirmed thrombosis without hemodynamic compromise): clear thrombus on imaging, stable device function and hemodynamics; no organ hypoperfusion. Treatment: for patients with low bleeding risk, intravenous thrombolysis (alteplase) can be considered; for patients with high bleeding risk, intensify anticoagulation and antiplatelet therapy, and closely monitor disease progression.

• Grade 3 (severe thrombosis with hemodynamic compromise): pump flow obstruction, cardiogenic shock, or recurrent peripheral thromboembolism. Treatment: emergency surgical pump replacement is the first-line treatment; for patients who cannot tolerate surgery, thrombolysis can be used as a bridging treatment.

For RVAD thrombosis, the diagnostic and treatment principles are the same as LVAD thrombosis, but the anticoagulation intensity should be appropriately increased, and the threshold for surgical intervention should be lower due to the faster progression of RVAD thrombosis.

4. Current Status of Anticoagulation Strategies

The third-generation fully magnetically levitated LVAD has become the international mainstream, featuring minimal surgical trauma, high durability, and excellent blood compatibility. Its magnetically levitated rotor and optimized hemodynamic structure have reduced the formation of pump thrombosis to a certain extent. To make its anticoagulation strategy more flexible and precise, it is necessary to formulate and adjust it individually according to the patient's intraoperative conditions and clinical status, as well as the device type.

According to different signs of postoperative active bleeding, anticoagulation strategies can be appropriately adjusted. If there is no active bleeding postoperatively, the hematocrit is stable, and the total drainage volume from the drainage tube is less than 40 mL within 3 hours, intravenous infusion of ordinary heparin can be used as a bridging anticoagulation regimen [22]. The target for APTT on the postoperative day is 40 seconds; if there are no signs of bleeding, APTT should be maintained at 40–60 seconds within 48–72 hours, and adjusted to approximately 50–60 seconds (not exceeding 80 seconds) after 2–3 days. Heparin should be discontinued after the INR reaches the target for 24 hours, and long-term oral warfarin anticoagulation should be initiated according to gastrointestinal function [20]. For patients with heparin-induced thrombocytopenia (HIT), fondaparinux can be used as a reasonable bridging agent [31]. According to the ISHLT 2024 consensus, for patients with HIT and high thrombosis risk, bivalirudin can also be used as an alternative bridging anticoagulant [31]. Table 4 summarizes the recommended standard anticoagulation strategies for the postoperative period after LVAD implantation.

Internationally, for patients without special conditions, INR is generally controlled at 2.0–3.0, with a typical starting dose of 2.5 mg or determined based on warfarin genotype. For different device types, the INR target should be adjusted: for axial flow LVADs, the conventional INR target is 2.0–3.0; for patients with centrifugal fully magnetically levitated LVADs (e.g., HM3 pump) at bleeding risk, the recommended INR range is 1.7–2.3 [31]. Studies have evaluated the feasibility of low-intensity anticoagulation after HM3 implantation, showing no increased risk of embolism events [31]. Some research has confirmed that with combined antiplatelet therapy, the INR range for fully magnetically levitated LVADs can be reduced to 1.8–2.5 [40,41].

Postoperatively, platelets should be closely monitored to maintain levels above 50×10^9/L and detect heparin-induced thrombocytopenia. When platelet counts exceed

Table 4. Recommended standard anticoagulation strategies for LVAD postoperative period.

Postoperative stage	Core anticoagulation regimen	Key monitoring indicators & target values	Special population/Device adjustment principles
Early postoperative period (no active bleeding)	Unfractionated heparin intravenous infusion as bridging therapy	APTT: 40 s on postoperative day 1; 40–60 s within 48–72 h; 50–60 s (max 80 s) after 3 days	HIT patients: switch to fondaparinux or bivalirudin
Transition to oral anticoagulation	Discontinue heparin when INR reaches target and maintains for 24 h; initiate warfarin oral therapy	INR: reach device-specific target; TTR ≥65%	Axial flow LVAD: INR target 2.0–3.0; Centrifugal maglev LVAD (high bleeding risk): INR target 1.7–2.3
Long-term maintenance phase	Warfarin as first-line agent; combined with aspirin in conventional population	INR: maintain stable target range; platelet count >50 × 10^9/L; LDH level	Bridge to heart transplantation: INR target 1.5–2.0 before surgery; recurrent thrombosis: adjust anticoagulation intensity
Stable follow-up phase	Regular monitoring of coagulation function, device parameters and hematological indicators	INR testing: at least once a week in the first 3 months; once every 2–4 weeks in stable phase	Aspirin can be reduced or discontinued in patients with repeated bleeding events (based on bleeding risk assessment)

HIT, heparin-induced thrombocytopenia; TTR, time in therapeutic range.

100 × 10^9/L, daily 100 mg aspirin combined with warfarin is recommended for antithrombotic therapy. In recent years, studies have shown that HM3 pump patients who discontinue aspirin have reduced bleeding risk without increased embolism risk [42]. However, no consensus has been reached on aspirin discontinuation, and more clinical evidence is needed. Most current guidelines still recommend aspirin for follow-up treatment, with dose reduction or discontinuation for patients with bleeding signs. Dual antiplatelet therapy is not recommended unless there is a clear risk of thrombosis, a history of prior pump thrombosis, or recent coronary revascularization [31].

TTR is a critical factor in postoperative adverse events - the higher the TTR, the better the prognosis. Studies show that age and distance to the hospital are positively correlated with TTR, while female gender, type 2 diabetes, and prior warfarin use are negatively correlated [43]. A TTR ≥65% is recommended, with point-of-care testing [20], and testing frequency potentially being key factors in reducing blood compatibility adverse events [44]. The Chinese Expert Consensus (2024) recommends that the INR testing frequency should be at least once a week in the first 3 months after surgery, and once every 2–4 weeks in the stable phase, to maintain TTR ≥65% [20].

Although some studies suggest novel anticoagulants have better outcomes than warfarin, they lack sufficient clinical trials and multicenter research, and thus are not recommended as first-line agents. The latest DOAC LVAD study shows that apixaban has good safety and efficacy in patients with fully magnetically levitated LVADs, but large-sample multicenter randomized controlled trials are still needed to confirm its long-term safety [45]. Currently, warfarin is still the first-line anticoagulant recommended by all domestic and international guidelines for LVAD patients [11,20,31].

5. Summary and Outlook

After LVAD implantation, a reasonable anticoagulation strategy is particularly important for preventing bleeding and embolism. Although the use of HM3 has effectively reduced the incidence of postoperative complications and progress has been made in optimizing LVAD anticoagulation strategies, there is still room for further improvement. This review systematically analyzes the multidimensional risk factors (patient-related, LVAD-related, exogenous) of bleeding and thrombosis after LVAD implantation, elaborates the differences in pathogenesis and management across different device types, discusses the management of special clinical scenarios, and integrates the latest recommendations of domestic and international guidelines, which provides a systematic reference for clinical practice.

Identifying and assessing risk factors before, during, and after surgery can effectively reduce the incidence of adverse events. Meanwhile, anticoagulation strategies should be further adjusted according to intraoperative conditions and patients' clinical indicators, device type, and special clinical scenarios, to achieve individualized management. Future research directions should include: (1) developing individualized anticoagulation regimens by combining genetic characteristics, clinical phenotypes, and device properties; (2) exploring the safety of new anticoagulants through large-sample multicenter randomized controlled trials; (3) optimizing postoperative monitoring and early intervention protocols for bleeding, thrombosis, and pump thrombosis; (4) improving LVAD design to enhance device blood compatibility and reduce shear stress-induced coagulation dysfunction. With the deepening of research and technological breakthroughs, the treatment protocols for LVAD patients will develop towards a safer, more efficient and individualized direction.

Author Contributions

SJN: Ideas; formulation or evolution of overarching research goals and aims. Preparation, creation, and presentation of the published work, specifically writing the initial draft (including substantive translation). CGP: Preparation, creation, and presentation of the published work by those from the original research group, specifically critical review, commentary, or revision – including pre- or post-publication stages. RYZ: Preparation, creation, and presentation of the published work by those from the original research group, specifically critical review, commentary, or revision – including pre- or post-publication stages. LY: Preparation, creation, revision, and presentation of the published work, specifically visualization/data presentation. ZBQ: Acquisition of the financial support for the project leading to this publication. Conducting research and the investigation process, specifically collecting data/evidence and revising the article. Oversight and leadership responsibility for the research activity planning and execution, including mentorship external to the core team. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

Acknowledgment

Not applicable.

Funding

This study was supported by the Key Medical Research Project of Jiangsu Provincial Health Commission (K2023039).

Conflicts of Interest

The authors declare no conflicts of interest.

References

[1] Hao G, Wang X, Chen Z, Zhang L, Zhang Y, Wei B, et al. Prevalence of heart failure and left ventricular dysfunction in China: the China Hypertension Survey, 2012-2015. European Journal of Heart Failure. 2019; 21: 1329–1337. https://doi.org/10.1002/ejhf.1629

[2] Han JJ, Acker MA, Atluri P. Left Ventricular Assist Devices. Circulation. 2018; 138: 2841–2851. https://doi.org/10.1161/CIRCULATIONAHA.118.035566

[3] Potapov EV, Antonides C, Crespo-Leiro MG, Combes A, Färber G, Hannan MM, et al. 2019 EACTS Expert Consensus on long-term mechanical circulatory support. European Journal of Cardio-thoracic Surgery : Official Journal of the European Association for Cardio-thoracic Surgery. 2019; 56: 230–270. https://doi.org/10.1093/ejcts/ezz098

[4] Lei B, Hu SS. Research Progress in Antithrombotic Therapy for Left Ventricular Assist Devices. Chinese Circulation Journal. 2020; 35: 200–204. (In Chinese).

[5] Caraballo C, DeFilippis EM, Nakagawa S, Ravindra NG, Miller PE, Mezzacappa C, et al. Clinical Outcomes After Left Ventricular Assist Device Implantation in Older Adults: An INTERMACS Analysis. JACC. Heart Failure. 2019; 7: 1069–1078. https://doi.org/10.1016/j.jchf.2019.10.004

[6] Wang Y, Li ZJ, Yi J, Li H, Chen M, Hou WM. Research Status and Future Prospects of Left Ventricular Assist Devices. Chinese Journal of Clinical Thoracic and Cardiovascular Surgery. 2024; 31: 1670–1678. (In Chinese).

[7] Vellanki N, Kennedy K, Grandin EW, Nunez J, Motiwala SR, Quintero PA, et al. Women have more bleeding events and intracranial hemorrhage than men post LVAD implantation. Circulation. 2020; 142: A16141. https://doi.org/10.1161/circ.142.suppl_3.16141

[8] Stulak JM, Deo S, Schirger J, Aaronson KD, Park SJ, Joyce LD, et al. Preoperative atrial fibrillation increases risk of thromboembolic events after left ventricular assist device implantation. The Annals of Thoracic Surgery. 2013; 96: 2161–2167. https://doi.org/10.1016/j.athoracsur.2013.07.004

[9] Ghodrati-Misek M, Schlöglhofer T, Gross C, Maurer A, Zimpfer D, Beitzke D, et al. Left atrial appendage occlusion in ventricular assist device patients to decrease thromboembolic events: A computer simulation study. Frontiers in Physiology. 2022; 13: 1010862. https://doi.org/10.3389/fphys.2022.1010862

[10] Nishida H, Jeevanandam V, Salerno C, Nemoto A, Song T, Onsager D, et al. Concomitant left atrial appendage closure during left ventricular assist device surgery can reduce ischaemic cerebrovascular accidents. Interdisciplinary Cardiovascular and Thoracic Surgery. 2023; 36: ivad112. https://doi.org/10.1093/icvts/ivad112

[11] Qing P, Du J, Zhou XT, Chen HB. Expert Consensus on Pre - operative Evaluation and Management of Candidates for Left Ventricular Assist Devices in China (2023). Chinese Circulation Journal. 2023; 38: 799–814. (In Chinese).

[12] Steinberg RS, Nayak A, Okoh A, Wang J, Matiello E, Morris AA, et al. Associations Between Preimplant Cancer Type and Left Ventricular Assist Device Outcomes: An INTERMACS Registry Analysis. ASAIO Journal (American Society for Artificial Internal Organs : 1992). 2024; 70: 272–279. https://doi.org/10.1097/MAT.0000000000002108

[13] Grant JK, Ebner B, Vincent L, Maning J, Olorunfemi O, Olarte NI, et al. Assessing in-hospital cardiovascular, thrombotic and bleeding outcomes in patients with chronic liver disease undergoing left ventricular assist device implantation. Thrombosis Research. 2021; 202: 184–190. https://doi.org/10.1016/j.thromres.2021.04.010

[14] Crugnola W, Cinquina A, Mattimore D, Bitzas S, Schwartz J, Zaidi S, et al. Impact of Diabetes Mellitus on Outcomes in Patients with Left Ventricular Assist Devices. Biomedicines. 2024; 12: 1604. https://doi.org/10.3390/biomedicines12071604

[15] Ono M, Yamaguchi O, Ohtani T, Kinugawa K, Saiki Y, Sawa Y, et al. JCS/JSCVS/JATS/JSVS 2021 Guideline on Implantable Left Ventricular Assist Device for Patients With Advanced Heart Failure. Circulation Journal : Official Journal of the Japanese Circulation Society. 2022; 86: 1024–1058. https://doi.org/10.1253/circj.CJ-21-0880

[16] Fried J, Levin AP, Mody KM, Garan AR, Yuzefpolsakaya M, Takayama H, et al. Prior hematologic conditions carry a high morbidity and mortality in patients supported with continuous-flow left ventricular assist devices. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2014; 33: 1119–1125. https://doi.org/10.1016/j.healun.2014.07.002

[17] Sparrow CT, Nassif ME, Raymer DS, Novak E, LaRue SJ, Schilling JD. Pre-Operative Right Ventricular Dysfunction Is Associated With Gastrointestinal Bleeding in Patients Supported With Continuous-Flow Left Ventricular As-

sist Devices. JACC. Heart Failure. 2015; 3: 956–964. https://doi.org/10.1016/j.jchf.2015.09.009
[18] Pinsino A, Castagna F, Zuver AM, Royzman EA, Nasiri M, Stöhr EJ, et al. Prognostic implications of serial outpatient blood pressure measurements in patients with an axial continuous-flow left ventricular assist device. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2019; 38: 396–405. https://doi.org/10.1016/j.healun.2018.11.003
[19] Rajapreyar I, Soliman O, Brailovsky Y, Tedford RJ, Gibson G, Mohacsi P, et al. Late Right Heart Failure After Left Ventricular Assist Device Implantation: Contemporary Insights and Future Perspectives. JACC. Heart Failure. 2023; 11: 865–878. https://doi.org/10.1016/j.jchf.2023.04.014
[20] Du J, Zou L, Chen Y, Zhou XT. Expert Consensus on Early Intensive Care Management after Implantation of Left Ventricular Assist Devices in China (2024). Chinese Circulation Journal. 2024; 39: 729–750. (In Chinese).
[21] Meesters MI, Burtman D, van de Ven PM, Boer C. Prediction of Postoperative Blood Loss Using Thromboelastometry in Adult Cardiac Surgery: Cohort Study and Systematic Review. Journal of Cardiothoracic and Vascular Anesthesia. 2018; 32: 141–150. https://doi.org/10.1053/j.jvca.2017.08.025
[22] Wang YP, Wang WY, Zuo ML, Fan D, Yu T, Zeng S, et al. Expert Consensus on the Whole - Process Management of Left Ventricular Assist Device Implantation (2023). Practical Journal of Clinical Medicine. 2024; 21: 43–55. (In Chinese).
[23] Park SY, Plambeck C, Joyce LD, Joyce DL. Bleeding After LVAD Implant: If Things Do Not Add Up, Take a Look! Innovations (Philadelphia, Pa.). 2021; 16: 488–490. https://doi.org/10.1177/15569845211042369
[24] Cho SM, Tahsili-Fahadan P, Kilic A, Choi CW, Starling RC, Uchino K. A Comprehensive Review of Risk Factor, Mechanism, and Management of Left Ventricular Assist Device-Associated Stroke. Seminars in Neurology. 2021; 41: 411–421. https://doi.org/10.1055/s-0041-1726328
[25] Klaeske K, Dieterlen MT, Eifert S, Scholz U, Garbade J, Jawad K, et al. Device-induced platelet dysfunction in patients after left ventricular assist device implantation. Journal of Thrombosis and Haemostasis : JTH. 2021; 19: 1331–1341. https://doi.org/10.1111/jth.15279
[26] Jahangiri P, Veen KM, van Moort I, Bunge JH, Constantinescu A, Sjatskig J, et al. Early Postoperative Changes in Von Willebrand Factor Activity Are Associated With Future Bleeding and Stroke in HeartMate 3 Patients. ASAIO Journal (American Society for Artificial Internal Organs : 1992). 2025; 71: 27–35. https://doi.org/10.1097/MAT.0000000000002250
[27] Hennessy-Strahs S, Kang J, Krause E, Dowling RD, Rame JE, Bartoli CR. Patient-specific severity of von Willebrand factor degradation identifies patients with a left ventricular assist device at high risk for bleeding. The Journal of Thoracic and Cardiovascular Surgery. 2024; 167: 196–204. https://doi.org/10.1016/j.jtcvs.2022.03.018
[28] Szymanski TW, Weeks PA, Patel CJ, Jezovnik MK, Gulbis B, Nathan SS, et al. Risk of pump thrombosis and stroke in patients with continuous-flow left ventricular assist devices and gastrointestinal bleeding. Artificial Organs. 2020; 44: 1171–1175. https://doi.org/10.1111/aor.13751
[29] Mehra MR, Uriel N, Naka Y, Cleveland JC, Yuzefpolskaya M, Salerno CT, et al. A Fully Magnetically Levitated Left Ventricular Assist Device - Final Report. The New England Journal of Medicine. 2019; 25: 1618–1627. https://doi.org/10.1056/NEJMoa1900486
[30] Mehra MR, Goldstein DJ, Uriel N, Cleveland JC, Yuzefpolskaya M, Salerno C, et al. Two-Year Outcomes with a Magnetically Levitated Cardiac Pump in Heart Failure. The New England Journal of Medicine. 2018; 378: 1386–1395. https://doi.org/10.1056/NEJMoa1800866
[31] Hollis IB, Jennings DL, Krim S, Ton VK, Ducharme A, Cowger J, et al. An ISHLT consensus statement on strategies to prevent and manage hemocompatibility related adverse events in patients with a durable, continuous-flow ventricular assist device. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2024; 43: 1199–1234. https://doi.org/10.1016/j.healun.2024.04.065
[32] Niamat J, Ramjankhan F, Van Der Kaaij N, Gianoli M, Van Laake LW, Mokhles MM. Outcome after left ventricular assist device exchange. European Journal of Cardio-Thoracic Surgery. 2024; 66: ezae317. https://doi.org/10.1093/ejcts/ezae317
[33] Ribeiro RV, Lee J, Singh S, Yau TM, Friedrich JO, Yanagawa B. Minimally Invasive Left Ventricular Assist Device Implantation: A Systematic Review and Meta-Analysis. The Journal of Heart and Lung Transplantation. 2021; 40: S457. https://doi.org/10.1016/j.healun.2021.01.1269
[34] Briasoulis A, Yokoyama Y, Kuno T, Ueyama H, Shetty S, Alvarez P, et al. In-Hospital Outcomes of Left Ventricular Assist Device Implantation and Concomitant Valvular Surgery. The American Journal of Cardiology. 2020; 132: 87–92. https://doi.org/10.1016/j.amjcard.2020.06.067
[35] Feldman D, Pamboukian SV, Teuteberg JJ, Birks E, Lietz K, Moore SA, et al. The 2013 International Society for Heart and Lung Transplantation Guidelines for mechanical circulatory support: executive summary. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2013; 32: 157–187. https://doi.org/10.1016/j.healun.2012.09.013
[36] Guglin M, Hirsch JR, Tanawuttiwat T, Akhtar N, Silvestry S, Ilonze OJ, et al. How to diagnose and manage emergency medical conditions in patients on left ventricular assist device support: A clinician's field guide. Trends in Cardiovascular Medicine. 2025; 35: 186–194. https://doi.org/10.1016/j.tcm.2024.11.004
[37] Inglis SS, Asleh R, Iyer VN, Schettle SD, Spencer PJ, Villavicencio MA, et al. Inhibition of angiogenesis in the management of refractory gastrointestinal bleeding in patients with LVAD support. Artificial Organs. 2024; 48: 646–654. https://doi.org/10.1111/aor.14694
[38] Kortekaas KA, den Exter PL, Beeres SLMA, Palmen M, Jukema JW, Huisman MV, et al. Systemic thrombolysis in the management of pump thrombosis in patients with left ventricular assist devices. Frontiers in Cardiovascular Medicine. 2022; 9: 969766. https://doi.org/10.3389/fcvm.2022.969766
[39] Frontera JA, Lewin JJ, Rabinstein AA, Aisiku IP, Alexandrov AW, Cook AM, et al. Guideline for Reversal of Antithrombotics in Intracranial Hemorrhage: A Statement for Healthcare Professionals from the Neurocritical Care Society and Society of Critical Care Medicine. Neurocritical Care. 2016; 24: 6–46. https://doi.org/10.1007/s12028-015-0222-x
[40] Marshall D, Sanchez J, Yuzefpolskaya M, Sayer GT, Takeda K, Naka Y, et al. Safety of reduced anti-thrombotic strategy in patients with HeartMate 3 left ventricular assist device. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2021; 40: 237–240. https://doi.org/10.1016/j.healun.2021.01.012
[41] Cosgrove RH, Khalpey Z. Comment on: Evaluation of low-intensity anti-coagulation with a fully magnetically levitated centrifugal-flow circulatory pump-the MAGENTUM 1 study. The Journal of Heart and Lung Transplantation : the Official Publication of the International Society for Heart Transplantation. 2018; 37: 1279. https://doi.org/10.1016/j.healun.2018.07.008

[42] Mehra MR, Netuka I, Uriel N, Katz JN, Pagani FD, Jorde UP, et al. Aspirin and Hemocompatibility Events With a Left Ventricular Assist Device in Advanced Heart Failure: The ARIES-HM3 Randomized Clinical Trial. JAMA. 2023; 330: 2171–2181. https://doi.org/10.1001/jama.2023.23204

[43] Henderson JB, Iyer P, Coniglio AC, Katz JN, Chien C, Hollis IB. Predictors of Warfarin Time in Therapeutic Range after Continuous-Flow Left Ventricular Assist Device. Pharmacotherapy. 2019; 39: 1030–1035. https://doi.org/10.1002/phar.2324

[44] Schlöglhofer T, Zapusek L, Wiedemann D, Riebandt J, Wittmann F, Dimitrov K, et al. International Normalized Ratio Test Frequency in Left Ventricular Assist Device Patients Affects Anticoagulation Quality and Adverse Events. ASAIO Journal (American Society for Artificial Internal Organs : 1992). 2021; 67: 157–162. https://doi.org/10.1097/MAT.0000000000001206

[45] Shah P, Looby M, Dimond M, Bagchi P, Shah B, Isseh I, et al. Evaluation of the Hemocompatibility of the Direct Oral Anticoagulant Apixaban in Left Ventricular Assist Devices: The DOAC LVAD Study. JACC. Heart Failure. 2024; 12: 1540–1549. https://doi.org/10.1016/j.jchf.2024.04.013