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Saphenous Vein Graft in Coronary Artery Bypass Grafting (CABG) Surgery: Combating Thrombosis, Intimal Hyperplasia and Atherosclerosis in Maintaining Long-Term Graft Patency

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SOUHRN

Cíl: Vzhledem k zájmu o používání safény jako štěpu pro koronární bypass (coronary artery bypass graft, CABG) bylo cílem této studie získat další poznatky umožňující zdokonalit léčebný postup, a zabránit tak dalšímu poškození žilního štěpu safény (saphenous vein graft, SVG).
Metody: Tento komplexní přehled popisuje současné standardy, aktuální postupy při léčbě a výsledky koronárního bypassu s použitím SVG; zároveň představuje řadu metod a nových postupů s potenciálem zlepšit jak bezprostřední, tak dlouhodobou průchodnost štěpu, přináší poznatky o patofyziologii poškození SVG i jak mohou tyto poznatky ukázat na nové možnosti léčby případného poškození.
Výsledky: Odhojení SVG může být výsledkem trombózy, intimální hyperplazie a aterosklerózy. K akutní trombóze dochází v důsledku hyperkoagulability, patologie v oblasti spojky nebo z technických důvodů, jako jsou poškození štěpu a defekty anastomózy. Hlavními patofyziologickými faktory v rozvoji intimální hyperplazie jsou migrace a proliferace buněk hladkého svalstva cév (vascular smooth muscle cell, VSMC). Prvním krokem, který celý proces spouští, je dysfunkce nebo poškození endotelu při poškození cév. Tento přehled popisuje postupy od odebrání SVG až po jeho implantaci včetně výběru štěpu, způsobu jeho odběru, jeho důkladného vyšetření, konzervaci a implantaci štěpu s použitím speciálních pomůcek a vybavení. Přehled rovněž seznamuje čtenáře s farmakoterapií v různých stadiích implantace SVG s cílem zabránit rozvoji neointimální hyperplazie a její případné léčby.
Závěr: Zlepšení dlouhodobé průchodnosti SVG vyžaduje propojení optimalizovaných chirurgických postupů, přísných farmakoterapeutických režimů a nových doplňkových strategií. Pro snížení počtu případů odhojení SVG a delší přežívání pacientů s CABG je naprosto nezbytné pokračovat v translačním výzkumu v oblasti cévní biologie a biotechnologií.

Keywords:
Atherosclerosis
Coronary artery bypass graft surgery
Coronary artery disease
Graft patency
Intimal hyperplasia
Saphenous vein graft failure

ABSTRACT

Aim: Gaining further insight to enhance treatment to stop the progression of saphenous vein grafts (SVG) disease, given the interest in using saphenous veins as grafts in coronary artery bypass graft (CABG).
Methods: This comprehensive review synthesizes the current standards, contemporary treatment, and results related to coronary arterial bypass grafting using SVG, exploring a variety of methods and developments that have the potential to improve both immediate and long-term graft patency. Understanding pathophysiology of SVG disease and where it leads to suggest potential new treatments for the condition.
Results: Failure of SVG can be the results of thrombosis, intimal hyperplasia, and atherosclerosis. Acute thrombosis caused by hypercoagulability, conduit-related pathology, or technical such as graft trauma and anastomotic defects. The migration and proliferation of vascular smooth muscle cells (VSMCs) in the tunica intima layer is the main pathophysiology of intimal hyperplasia. Endothelial dysfunction or damage from vascular injury is the first step that sets off the process. This review narrates SVG graft-to-implant strategies that include graft selection, harvesting technique, careful graft assessment techniques, graft preservation, and technology-assisted techniques. The review also covers pharmacotherapy that targets distinct stages of the SVG condition to prevent and treat neointimal hyperplasia.
Conclusion: Improving long-term SVG patency requires integration of optimized surgical techniques, rigorous pharmacological regimens, and novel adjunctive strategies. Continued translational research into vascular biology and biotechnology is crucial to reducing SVG failure rates and improving patient survival after CABG.

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Introduction

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide, with an estimated 17.9 million deaths annually, representing nearly one-third of all global deaths.[1] The global economic impact of CAD is immense, driven by costs of hospitalizations, loss of productivity, and long-term management.[2] Despite extensive progress in preventive cardiology—including statins, antihypertensive therapy, antiplatelets, and smoking cessation—CAD incidence continues to rise in parallel with aging populations, sedentary lifestyles, obesity, and diabetes.[3]

For more than four decades, coronary artery bypass grafting (CABG) has been the gold standard of surgical revascularization for patients with advanced or complex CAD, particularly those with left main stenosis, three-vessel disease, diffuse atherosclerosis, or diabetes mellitus.[4] The SYNTAX, FREEDOM, and EXCEL trials have consistently demonstrated the survival and repeat-revascularization advantage of CABG over percutaneous coronary intervention (PCI) in these populations.[5–7] While PCI offers less invasiveness, CABG remains superior for long-term durability of revascularization.

The choice of conduit is central to CABG outcomes. The left internal mammary artery (LIMA) to the left anterior descending artery (LAD) is universally considered the "gold standard" anastomosis, with patency rates exceeding 90% at 10–15 years.[8] However, the saphenous vein graft (SVG) continues to be the most widely used conduit globally because of its availability, length, ease of harvest, and ability to reach multiple distal coronary targets. In fact, more than 70–80% of CABG procedures worldwide still use SVGs in some capacity.[9]

Yet, SVGs represent the Achilles heel of CABG. While LIMA durability is excellent, SVG patency rates drop steeply over time—10–15% occlude within the first year, and up to 50% fail within 10 years.[10,11] The main drivers are acute thrombosis, intimal hyperplasia, and accelerated atherosclerosis. These processes are influenced by a combination of patient-related risk factors (e.g., diabetes, smoking), technical issues (harvesting trauma, preservation), and biological mechanisms (endothelial dysfunction, smooth muscle proliferation, oxidative stress).[12]

Understanding the mechanisms of SVG failure has driven numerous innovations, from no-touch harvesting techniques, advanced preservation solutions, and pharmacological strategies (dual antiplatelet therapy, statins, PCSK9 inhibitors), to novel adjunct technologies such as external stents and bioengineered grafts.[13–16] Yet, despite these efforts, SVG disease remains a major determinant of long-term CABG outcomes.

This review synthesizes recent global evidence on SVG disease, covering pathophysiology, risk factors, surgical strategies, pharmacological therapy, technological adjuncts, outcomes, and future perspectives, with a focus on both international data and regional experiences.

Historical perspective of CABG And SVG

The history of CABG is rooted in the pioneering work of René Favaloro at the Cleveland Clinic in the late 1960s, who performed the first successful CABG using autologous saphenous vein grafts. Early results were revolutionary, providing surgical treatment for previously untreatable coronary artery disease. During the 1970s and 1980s, CABG expanded worldwide, rapidly becoming one of the most common performed major surgeries. In those early decades, SVGs were the default conduit. They were easy to harvest, versatile in length, and familiar to surgeons. Initial short-term outcomes were excellent, with high rates of symptom relief and survival improvement. However, as long-term angiographic and clinical follow-up became available, it was increasingly evident that SVGs had limited durability compared with arterial conduits.[17]

By the 1980s, observational angiographic studies showed SVG patency rates of ~70% at 5 years, dropping to ~50% at 10 years, while LIMA patency exceeded 90% at 10 years. This realization spurred greater interest in arterial grafting strategies, particularly the right internal mammary artery (RIMA), radial artery, and gastroepiploic artery.[18]

Nevertheless, the SVG remained indispensable. Arterial conduits have limitations: the RIMA increases the risk of sternal wound complications, particularly in diabetic and obese patients; the radial artery is susceptible to vasospasm and is not suitable in all patients; and the gastroepiploic artery is technically demanding and infrequently used. Moreover, many patients requiring CABG have multivessel disease involving 3–4 targets, for which available arterial conduits are often insufficient. Consequently, current CABG strategies reflect a combination approach: a LIMA-to-LAD graft for durability, supplemented with SVGs to other coronary territories. Even in advanced centres advocating multi-arterial grafting, SVGs remain necessary in the majority cases.[19,20]

Pathophysiology of saphenous vein graft disease

SVG failure is a multifactorial, time-dependent process, classically divided into three phases:

1. Early failure (0–1 month): Acute thrombosis

Acute thrombosis occurs in ~10–15% of grafts. The most common causes were technical issues (conduit trauma, poor anastomosis), hypercoagulability, and low-flow states. The mechanism of acute thrombosis involved venous endothelium, graft that accustomed to low-pressure circulation undergoing injury under arterial pressure. The process causes endothelial denudation, platelet activation, and fibrin deposition (**Fig. 1**).[21,22]

2. Intermediate failure (1–12 months): Intimal hyperplasia

Intimal hyperplasia is the pathognomonic lesion of SVG disease. It represents the intermediate phase of graft failure, occurring after the initial risk of acute thrombosis but preceding late atherosclerosis. The process is primarily triggered by endothelial injury sustained during vein harvesting and implantation into the arterial circulation. The venous conduit, originally adapted to a low-pressure, low-flow environment, is suddenly exposed to arterial shear stress and pulsatile pressure, re-

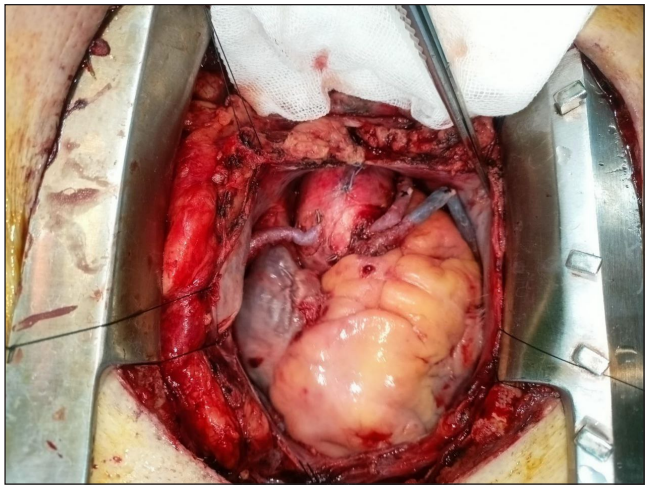

Fig. 1 – Aortocoronary bypass, operative field showing a saphenous vein graft with visible intraluminal thrombus.

sulting in endothelial denudation, platelet activation, and a cascade of inflammatory and proliferative signals. These early insults initiate vascular remodelling within the graft wall.[23]

Following endothelial injury, vascular smooth muscle cells (VSMCs) migrate from the media to the intima and undergo phenotypic modulation from a contractile to a synthetic state. In this state, VSMCs proliferate and secrete components of the extracellular matrix (ECM)—notably collagen, elastin, and proteoglycans—resulting in progressive intimal thickening. This neointimal formation narrows the graft lumen and increases vascular stiffness. Hemodynamic factors such as disturbed flow and oscillatory shear stress further promote VSMC proliferation and extracellular matrix deposition, reinforcing a cycle of maladaptive remodelling. Experimental and clinical studies have confirmed that the extent of intimal hyperplasia correlates strongly with graft stenosis and mid-term occlusion rates.[24,25]

Although intimal hyperplasia rarely causes acute graft occlusion, it establishes the pathological substrate for accelerated atherosclerosis within the vein graft. Thus, intimal hyperplasia is not only a hallmark lesion but also a pivotal transitional stage linking early mechanical and endothelial injury to the eventual development of graft atherosclerosis.

3. Late failure (>1 year): Accelerated atherosclerosis

Unlike native coronary atherosclerosis, SVG atherosclerosis is diffuse, concentric, and prone to plaque rupture. Histological features in SVG atherosclerosis are large lipid cores, thin fibrous caps, heavy inflammatory infiltrates. Late SVG failure clinically often presents as unstable angina, myocardial infarction, or distal embolization.[26]

Molecular mechanisms

The process of SVG disease begins with endothelial dysfunction, characterized by reduced bioavailability of nitric oxide (NO)—a key vasodilator—and concomitant upregulation of endothelin-1, reactive oxygen species (ROS), and other oxidative mediators. This imbalance leads to vasoconstriction, loss of anti-thrombotic surface properties, and promotion of a pro-inflammatory vascular milieu. The dysfunctional endothelium triggers inflammatory activation, during which cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and C-reactive protein (CRP) are released, inducing expression of adhesion molecules (VCAM-1, ICAM-1, and E-selectin). These molecules facilitate leukocyte adhesion, transmigration, and accumulation within the graft wall, initiating a chronic inflammatory response.[22,23]

Subsequently, vascular smooth muscle cells (VSMCs) within the media undergo phenotypic switching from a quiescent, contractile state to a synthetic phenotype capable of proliferation, migration into the intima, and secretion of extracellular matrix (ECM) proteins. This adaptive yet maladaptive remodelling response contributes to intimal hyperplasia, the hallmark intermediate lesion in SVG disease.[25]

Persistent oxidative stress further accelerates vascular injury: oxidized low-density lipoprotein (ox-LDL) particles are taken up by macrophages to form lipid-laden foam cells, which drive the development of atheromatous plaques within the graft. Over time, inflammatory cells and macrophages secrete matrix metalloproteinases (MMPs) that degrade ECM components such as collagen and elastin, leading to thinning of the fibrous cap and plaque instability, predisposing the vein graft to rupture and occlusive thrombosis.[23,27]

Hemodynamic factors

Veins are adapted to low-pressure and low-flow states. When saphenous veins are transposed into the arterial circulation, they are suddenly exposed to a hemodynamic environment for which they are not physiologically adapted. Normally accustomed to low-pressure and low-shear conditions, the venous endothelium experiences substantial mechanical stress when subjected to arterial shear and pulsatile pressure. Recent evidence demonstrates that this abrupt exposure induces endothelial injury and apoptosis mediated by mechanosensitive pathways such as the FGL2/FcγRIIB signaling cascade, leading to endothelial denudation and dysfunction. In addition, increased tangential stress and wall tension provoke maladaptive remodelling characterized by medial hypertrophy, smooth muscle cell activation, and extracellular matrix accumulation.[24,28]

Biomechanical and immunological studies further show that turbulent flow patterns at the graft–artery anastomosis amplify endothelial activation and inflammatory signaling, promoting smooth muscle migration and intimal hyperplasia. This combination of mechanical overload and inflammation drives concentric wall thickening and progressive luminal narrowing, setting the stage for vein graft disease. Together, these findings underscore that early hemodynamic mismatch—specifically increased shear stress, pulsatile arterial pressure, and disturbed flow—is the primary initiator of the structural and biological deterioration that ultimately compromises long-term graft patency.[29]

Risk factors for saphenous vein graft (SVG) failure

SVG patency is determined by a complex interaction between patient-specific, graft-specific, and surgical/technical factors. Understanding these risk factors enables surgeons to identify high-risk patients, tailor surgical strategies, and optimize postoperative care.

1. Patient-related risk factors

Diabetes mellitus
Chronic hyperglycaemia induces endothelial dysfunction, oxidative stress, and accumulation of advanced glycation end products, all of which accelerate smooth muscle proliferation and intimal hyperplasia. Histopathologic evidence further supports these findings, as patients with type II diabetes undergoing CABG exhibit characteristic structural changes within SVGs—such as intimal fibrosis, vacuolization of endothelial and smooth muscle cells, and pre-existing microvascular injury. Although the direct impact of these alterations on long-term graft patency remains to be fully clarified, they likely contribute to the heightened vulnerability of diabetic SVGs to early and progressive failure.[30]

Dyslipidaemia
Elevated low-density lipoprotein (LDL) cholesterol and persistent hypertriglyceridemia promote lipid infiltration and foam-cell formation within the SVG intima, accelerating graft atherosclerosis and luminal loss. Experimental and animal data show very rapid lipid accumulation and foam-cell development in veins graft exposed to hypercholesterolaemia, supporting a direct biological effect of circulating atherogenic lipoproteins on graft walls.[23,31]

Smoking
Smoking induces a cascade of vascular insults that directly compromise SVG integrity and long-term patency. Tobacco exposure increases oxidative stress and systemic inflammation, reduces endothelial nitric oxide bioavailability, and enhances platelet activation and aggregation, creating a prothrombotic and pro-inflammatory milieu within the graft. Clinical and observational studies consistently demonstrate that active smoking is associated with reduced graft patency and higher rates of adverse cardiovascular outcomes after CABG.[32]

Chronic kidney disease (CKD)
Chronic kidney disease promotes a pro-atherothrombotic vascular phenotype through persistent endothelial dysfunction (reduced NO signalling, impaired endothelial repair), chronic inflammation, and accelerated medial/intimal calcification. These mechanisms plausibly render saphenous vein grafts more susceptible to early neointimal hyperplasia and late atherosclerotic failure. Consistent with this biology, lower preoperative eGFR independently predicts higher SVG failure, contemporary reviews further confirm CKD as an adverse determinant of long-term post-CABG outcomes.[33,34]

Age and sex
Advanced age is associated with impaired saphenous vein graft (SVG) survival following coronary artery bypass grafting (CABG). Aging leads to progressive endothelial dysfunction characterized by decreased nitric oxide (NO) bioavailability, increased oxidative stress, and reduced endothelial regenerative capacity. Studies of human saphenous veins have shown that older donor age correlates with decreased endothelium-dependent relaxation and heightened susceptibility to intimal hyperplasia.[35]

Sex-based differences further influence graft outcomes. Several large observational cohorts have reported that women experience lower SVG patency and higher mortality after CABG compared with men. The underlying factors is poorly understood but this disparity maybe attributed in part to smaller coronary and conduit vessel diameters in women, which predispose to early graft thrombosis and competitive flow phenomena.[36–38]

Hypertension
Elevated and fluctuating arterial pressures expose the venous conduit—naturally adapted to low-pressure environments—to excessive hemodynamic stress, leading to endothelial injury, vascular smooth muscle cell proliferation, and maladaptive intimal hyperplasia. These processes accelerate graft wall remodelling and lumen narrowing. A recent retrospective study demonstrated that poor blood pressure control was significantly associated with lower long-term SVG patency following CABG, underscoring the role of hypertension as an independent predictor of graft failure. Chronic wall tension and disturbed flow patterns also promote atherogenesis and medial degeneration, further compromising graft durability.[39]

2. Graft-related risk factors

Vein quality
Pre-existing structural abnormalities in the saphenous vein, such as varicosities, thin vessel walls, or small luminal diameters, have been consistently associated with early thrombosis and late stenosis following coronary artery bypass grafting (CABG). These morphological changes reflect chronic venous remodelling and loss of medial smooth muscle integrity, predisposing the conduit to endothelial disruption and impaired adaptation to arterial pressure once grafted. Recent study reported that SVGs with diameters ≤2 mm exhibited markedly reduced long-term patency—approximately 55% at 10 years—compared with larger conduits (>2 mm, ≈88%), highlighting vessel size as a critical determinant of graft survival. Proper intraoperative vein assessment and selective harvesting of morphologically healthy segments are therefore essential to optimize long-term SVG performance.[23,40]

Conduit length and flow
Long and tortuous grafts are vulnerable mechanical-habitat risks: excessive graft length and tortuosity predispose the conduit to kinking, focal flow separation, and turbulence, which in turn amplify shear-stress abnormalities, endothelial dysfunction and focal intimal hyperplasia. Although conduit geometry is recognized as an im-

portant determinant of flow dynamics, recent literature highlights an ongoing evidence gap regarding the direct association between graft length and long-term patency. The specific contribution of graft length remains poorly defined and warrants further investigation. Preoperative ultrasonographic mapping of the great saphenous vein—assessing diameter, wall quality, and tortuosity—therefore remains a crucial step to optimize conduit selection, reduce wound morbidity, and potentially enhance long--term graft patency.[41,42]

3. Technical and perioperative factors

Harvesting technique

According to recent studies, conventional saphenous vein harvesting that strips the perivascular tissue and subjects the conduit to unregulated, high-pressure distension causes immediate structural and biological injury—mechanical trauma and endothelial denudation reduce nitric oxide bioavailability and expose subendothelial matrix, promoting platelet adhesion and acute thrombosis, while wall injury triggers vascular smooth muscle cell activation and intimal hyperplasia that accelerate graft stenosis. In contrast, "no-touch" or pedicled harvesting—where the vein is removed with its surrounding adipose and adventitia intact and handled minimally—has been associated with improved graft morphology and superior long-term patency, albeit at the cost of higher leg-wound morbidity in several series and registries. Endoscopic vein harvesting (EVH) reduces donor-site complications and wound morbidity compared with open techniques and is widely adopted, but operator technique matters: poorly performed EVH or excessive traction/distension during EVH can still produce endothelial injury.[43,44]

Therefore, many centres now emphasise a "soft-touch" conduit preparation strategy—defined by atraumatic handling, avoidance of high-pressure distension (use of low-pressure or no-pressure spasmolytic techniques), preservation of perivascular tissue when feasible, and standardised intraoperative graft assessment—to minimise endothelial and adventitial injury and optimise SVG performance. Contemporary guideline reviews and consensus statements recommend these pragmatic steps while acknowledging ongoing debates (and recent trial data) about the balance between patency gains and wound complications, and call for standardisation of harvesting and preparation protocols to improve reproducibility of outcomes.[45]

Preservation solution

Conventional storage media such as plain saline or heparinized autologous blood expose harvested SVG to a non-physiologic, unbuffered environment that promotes acidosis, oxidative stress, endothelial cell loss and impaired vasoreactivity, and several translational and clinical reports have linked saline storage to worse markers of endothelial injury. To mitigate these effects, investigators have evaluated a spectrum of preservation strategies such as simple buffered isotonic solutions and modified physiologic buffers that better maintain pH and ionic balance or specially formulated endothelial-damage-inhibitor (EDI) solutions such as DuraGraft that supply antioxidants and protective ions. Some others studied hospital-compounded recipes (for example the GALA solution: glutathione, L-ascorbic acid, L-arginine) tested in retrospective cohorts and early trials and organ-preservation-derived solutions (e.g., HTK/TiProtec-derivatives) that were developed to limit cold ischemia–reperfusion injury and have shown endothelial protection in experimental models. However, the clinical evidence remains heterogeneous and adequately powered trials with angiographic or functional graft--patency and hard clinical endpoints are limited.[46]

Anastomotic technique

Poorly constructed anastomoses—particularly those performed under excessive tension, with graft redundancy, twisting, or kinking—create local hemodynamic disturbances that manifest as flow separation, recirculation zones, and regions of low wall shear stress, which in turn accelerate early thrombosis and graft failure. Technical errors such as malalignment, adventitial injury, or mis--angle at the heel or toe of the graft further impair endothelial integrity and trigger platelet aggregation. Recent hemodynamic studies and clinical data confirm that non-physiologic geometry at the anastomosis correlates strongly with early occlusion risk. Competitive flow in terminal anastomoses of saphenous vein grafts, grafts with suboptimal configuration and moderate native coronary stenosis showed higher occlusion rates.[47,48]

In the realm of graft configurations, direct (single, end-to-side) SVG anastomoses remain the most common but may be vulnerable to competitive flow when the native target vessel stenosis is only moderate. Sequential grafting—where a single vein conduit supplies several distal anastomoses—has been shown to improve flow efficiency and reduce conduit waste in multivessel disease,

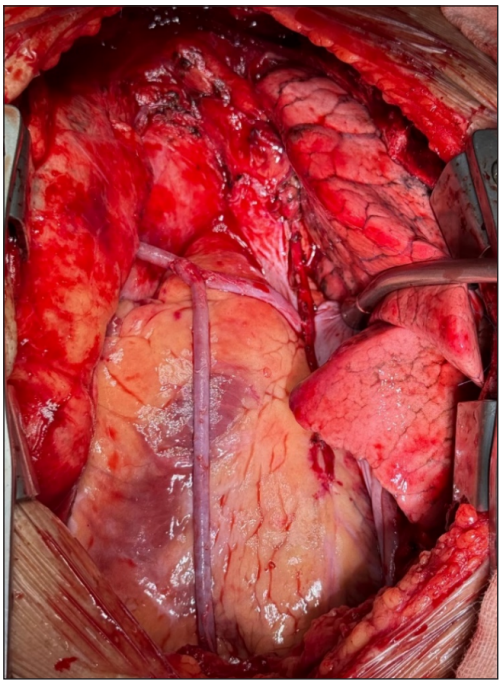

Fig. 2 – Y-graft of SVG in CABG.

though it demands technical precision. Composite graft configurations (e.g., Y- or T-grafts using a conduit such as the SVG or internal thoracic artery) are gaining interest for their potential to optimize inflow supply, reduce aortic manipulation and adapt to extended revascularization strategies. Recent found that a no-touch SVG used as a Y-composite graft demonstrated non-inferior one-year morphologic outcomes compared to an aortocoronary SVG configuration (**Fig. 2**).[49]

These findings reinforce that meticulous surgical technique—ensuring tension-free routing, minimal redundancy, proper alignment, anastomotic angle optimization, and configuration choice tailored to target morphology—is essential to minimize early thrombosis and preserve long-term SVG patency.

On-pump vs off-pump CABG

Off-pump coronary artery bypass grafting (OPCAB) may be associated with lower SVG patency when performed in the hands of less experienced surgeons or at lower-volume centres, although when performed in high-volume centres with experienced teams the long-term outcomes may be equivalent to on-pump CABG (ONCAB). Several randomised controlled trials (RCTs) and meta-analyses have shown that OPCAB is linked to a higher risk of graft occlusion with one updated meta-analysis reporting a relative risk (RR) of 1.40 (95% CI 1.23–1.59) for SVG occlusion in OPCAB vs ONCAB.[50]

The difference has been attributed in part to the technical challenges of performing distal anastomoses on a beating heart, potential incomplete revascularisation, fewer grafts per patient, and the steeper learning curve of OPCAB. Observational data also suggest that centres and surgeons with lower OPCAB volumes have worse outcomes and higher reintervention rates. On the other hand, recent analyses suggest that in centres with established OPCAB expertise and high annual volumes the SVG patency and overall outcomes approximate those of ONCAB.[51]

Therefore, when selecting OPCAB for a patient, the surgeon's experience and institutional volume should be taken into accounts, and intraoperative verification of graft quality (e.g., transit-time flow measurement) becomes particularly important to mitigate the higher risk of SVG failure.

Minimally invasive CABG and robotic CABG

In minimally invasive coronary artery bypass surgery, the use of SVG has demonstrated acceptable patency rates comparable to those achieved with conventional sternotomy approaches. Angiographic and computed tomography follow-up studies report early to mid-term SVG patency of approximately 80–90%, indicating that minimally invasive access does not compromise graft durability when proper harvesting and anastomotic techniques are applied.[52]

In robotic-assisted coronary artery bypass grafting (CABG) and totally endoscopic coronary artery bypass (TECAB), reported graft patency outcomes are generally favourable, primarily reflecting the routine use of the left internal mammary artery (LIMA) to the left anterior descending artery. The well-established durability of the LIMA conduit substantially influences overall patency results in these procedures. However, evidence specifically evaluating SVG patency in robotic CABG and TECAB remains limited. Although available data suggest acceptable early and mid-term SVG patency, further systematic follow-up is required to more clearly characterize long-term SVG outcomes in this minimally invasive context.[53]

Surgical strategies to improve SVG patency

Refinements in surgical technique are among the most effective interventions for improving SVG durability.

1. Vein harvesting techniques

Current studies emphasize that no touch harvesting is superior in terms of patency compared to conventional open harvesting. Endoscopic vein harvesting reduces wound complications and improves cosmetic. Early reports raised concerns about inferior patency, but other contemporary studies suggest equivalent outcomes when performed by experienced teams.

2. Preservation solutions

Traditionally, SVGs are stored in saline or blood during surgery. However, these solutions fail to prevent ischemic and oxidative injury. Buffered solutions (e.g., DuraGraft, Custodiol) provide antioxidant protection and maintain endothelial nitric oxide synthase (eNOS) activity, resulting in lower intimal hyperplasia.

3. Anastomotic technique

Sequential anastomosis can improve flow and reduce competitive flow, though carries risk of compromising multiple territories if the proximal graft fails. Individual grafting provides greater security but may sacrifice flow efficiency. Meticulous construction with avoidance of kinking, excessive tension, and precise suturing remains critical. Additionally, when a graft is anastomosed to a coronary vessel with only moderate stenosis (i.e., less-tight native flow resistance), the phenomenon of competitive flow from the native coronary reduces graft flow, increases the pulsatility index (PI) and retrograde flow fraction, and thereby augments the risk of early graft occlusion.

From a haemodynamic standpoint, computational modelling has demonstrated that increasing competitive flow (i.e., decreasing the severity of native stenosis) leads to lower time-averaged wall shear stress (TAWSS) and higher oscillatory shear index (OSI) in the graft, conditions known to promote endothelial dysfunction and subsequent occlusion.

4. Intraoperative graft flow measure

Intraoperative graft flow measurement has become a cornerstone in ensuring graft quality during CABG, especially for assessing the viability of SVGs and arterial grafts before chest closure. One of the most widely used tools is transittime flow measurement (TTFM), which quantifies the mean graft flow (MGF), calculates a pulsatility index (PI), assesses diastolic filling (DF) and may even incorporate advanced waveform analysis such as fast Fourier transform (FFT) to detect subtle anomalies.

Yet, intraoperative graft flow measurement should be considered a complement to, rather than a replacement for, surgical judgment. Surgeons are advised to measure flow under stable hemodynamic conditions, interpret the full waveform shape (not just single values), and revise grafts when persistently low flow, high PI or reverse flow are detected.

4. On-pump vs off-pump CABG

To improve SVG patency, the choice between on-pump and off-pump techniques should be guided by surgical expertise and patient characteristics. In the case of off--pump CABG, special attention should be paid to maintaining hemodynamic stability, avoiding target-vessel motion, and minimizing conduit trauma—factors that may explain the slightly higher graft failure rates when done in lower-volume centres. Indeed, a 2022 meta-analysis found that overall graft patency and SVG patency were significantly *poorer* in off-pump vs on-pump CABG (RR ~1.32 for any graft occlusion; RR ~1.37 for SVG occlusion). Meanwhile other trial found *non-inferiority* of off-pump vs on-pump graft patency when performed by experienced surgeons.[50,54]

5. External stents and conduit supports

External stenting places a flexible scaffold around the SVG to limit outward dilation, reduce wall tension, and thereby blunt the mechanotransduction cascade that drives smooth muscle cell proliferation and intimal hyperplasia. Early human and preclinical data consistently show that external support produces more uniform lumens, less focal dilation, and significantly smaller intimal-hyperplasia areas and intima–media thickness versus unsupported SVGs. However, the translation from favourable vessel remodelling to hard clinical benefit remains incompletely proven.[55]

6. Other surgical innovations

Composite grafting — for example an internal mammary artery-based Y-graft that supplies both the LAD and an attached SVG — offers important technical and physiologic advantages: it reduces aortic manipulation (fewer proximal aortic anastomoses), provides competitive LIMA flow that can pressurize downstream conduits, and can improve run-off and shear stress patterns in the distal coronary bed. Using arterial inflow (RITA or LIMA) to supply an SVG limb (composite-grafts), which may improve SVG behaviour by exposing it to arterial nitric-oxide production and more favourable hemodynamic.[56]

Pharmacological therapies for SVG patency

While surgical technique is crucial, pharmacotherapy is equally important in preventing graft failure. Drugs act on the thrombotic, inflammatory, and proliferative pathways that drive SVG disease.[57]

1. Antiplatelet therapy

In clinical practice following CABG, aspirin remains the foundation of antiplatelet therapy: begin as soon as haemostasis is secured (ideally within 6 hours) to reduce early graft thrombosis, and plan for long-term daily aspirin to minimise graft occlusion and major adverse cardiac events (MACE). For patients at higher risk of SVG failure (e.g., multiple SVGs, poor graft flow, or concomitant acute coronary syndrome), consider dual antiplatelet therapy (DAPT) with aspirin + a $P2Y_{12}$ inhibitor for 12 months, provided bleeding risk is acceptable. For example, the DACAB trial showed that aspirin + ticagrelor improved SVG patency and reduced MACE up to five years versus aspirin alone.[57]

On the other hand, the POPular CABG trial found no significant improvement in SVG occlusion at 1 year and an increased bleeding trend with DAPT. Consequently, the decision to use DAPT should be individualised: assess patient-specific factors (bleeding risk, graft count, graft flow quality, comorbidities) and aim for shared decision-making, monitoring carefully for bleeding. In lower-risk patients (stable CAD, single SVG, no ACS), monotherapy with aspirin may suffice; in higher-risk cases the incremental benefit of DAPT may justify the bleeding risk, provided close follow-up is ensured. Regular review of antiplatelet strategy and adjustment based on renal function, haemodynamic changes, and concurrent therapies is advised.[58]

2. Lipid-Lowering Therapy

Statins

Intensive statin therapy has biological plausibility for protecting SVGs. Statins lower LDL-C and exert pleiotropic actions (anti-inflammatory, antithrombotic, improved endothelial function) that reduce lipid deposition, blunt expansive remodelling and intimal hyperplasia in veins conduits. Observational and randomized data have linked statin use and lower LDL levels with less SVG disease and smaller intimal hyperplasia on imaging. However, some findings imply that early LDL lowering alone may be insufficient to prevent the multifactorial process of early SVG failure (thrombosis, technical factors) and that benefits on atherosclerotic progression may emerge later.[59]

PCSK9 inhibitors

Trials testing intensive LDL lowering beyond statins (PCSK9 inhibitors) have directly addressed whether deeper LDL reduction with adding evolocumab to background statin therapy did not show a reduction in early SVG failure.[60]

3. Anticoagulation

Routine postoperative warfarin or other full-dose oral anticoagulation solely to improve SVG patency is not recommended because randomized trials and pooled analyses have not shown a consistent patency advantage that outweighs the clear increase in bleeding risk. When anticoagulation is required (for AF, mechanical valves, LV thrombus, etc.), clinicians must explicitly plan antithrombotic combinations because adding antiplatelet therapy increases bleeding risk. In exceptional situations (extensive endarterectomy, early graft thrombosis), consider individualized short-term anticoagulation with careful risk–benefit assessment and local multidisciplinary input.[61]

Future research direction

Anti-proliferative such as sirolimus analogues or other anti-proliferative drugs have been investigated for preventing intimal hyperplasia, but more research is needed to evaluate its potensial for SVG disease.[62] Polymeric external stents, in some recent studies show reduced intimal hyperplasia, but though long-term clinical benefit remains questionable.[63]

Bioengineered conduits, developed using decellularized scaffolds or synthetic polymers seeded with endothelial or stem-derived vascular cells, are under active preclinical and early translational evaluation. Current studies show encouraging biomechanical strength, biocompatibility, and the ability to remodel and integrate into host tissue, but long-term durability and thrombosis resistance remain key hurdles before clinical adoption. Emerging tissue-engineered vascular grafts (TEVGs) must demonstrate not only safety and manufacturability at scale, but patency rates at least equivalent to, if not better than SVG to become a viable alternative for CABG in routine practice.[64]

Conclusion

SVG failure remains a critical limitation of CABG. Although numerous innovations show promising potential to improve saphenous vein graft patency, current evidence consistently demonstrates that the most decisive determinants of long-term outcomes remain the fundamentals: high-quality surgical technique, thorough preoperative optimization, and appropriate, guideline-directed pharmacotherapy. In other words, while adjunctive technologies may enhance results in the future, the cornerstone of successful CABG today continues to rely on meticulous operative practice and comprehensive medical management.

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Ethical statement
The work was carried out in compliance with the Declaration of Helsinki.

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