iCirculation
https://www.sciltp.com/journals/iCirculation

Review

What Is the Fate of Saphenous Vein Graft Vasa Vasorum after Coronary Artery Bypass Surgery: An Open-and-Shut Case?

Michael R. Dashwood

Surgical and Interventional Sciences, Royal Free Hospital Campus, University College Medical School, London NW3 2PF, UK; m.dashwood@ucl.ac.uk or mickeydash@hotmail.com; Tel: +44-(0)20-7794-0500

How To Cite: Dashwood, M.R. What Is the Fate of Saphenous Vein Graft Vasa Vasorum after Coronary Artery Bypass Surgery: An Open-and-Shut Case? *iCirculation* **2026**, *1*(1), 2. https://doi.org/10.53941/icirculation.2026.100002

Received: 20 December 2025
Revised: 16 January 2026
Accepted: 25 February 2026
Published: 18 March 2026

Abstract: The vasa vasorum, a network of microvessels providing the vessel wall with oxygen and nutrients, is more dense and penetrates deeper in veins than in arteries. The saphenous vein is the most commonly used conduit for coronary artery bypass surgery but its patency is inferior to the 'gold standard' internal thoracic artery. Considerable vascular damage is caused during conventional preparation of the saphenous vein that effects all vessel layers, the endothelium and intima, the media and the adventitia. Graft patency is dramatically improved when the vein is harvested intact and with minimal trauma using the no touch technique. Among the structures preserved is the vasa vasorum that is suggested to play a role in the superior patency rate of no touch saphenous vein grafts. Histological studies have identified regions of the saphenous vein that are proposed to represent termination of vasa vasorum in the lumen. Since the vasa vasorum of the explanted saphenous vein graft is disconnected from its arterial blood supply *in vivo* an inside out/outside in communication has been hypothesised based on *ex vivo* perfusion studies. Such a novel transport system may play an important role in maintaining blood supply to the vessel wall, extending to the outermost capillary network of the perivascular fat. Also, bidirectional transport of vasoprotective factors such as nitric oxide may contribute to the underlying mechanisms involved in the improved performance of no touch saphenous vein grafts.

Keywords: saphenous vein; vasa vasorum; CABG; blood flow; patency

1. Saphenous Vein and Graft Patency: Surgical Implications

1.1. Clinical Results of the No-Touch Technique

Coronary artery disease accounts for >7 million deaths per year worldwide with >800,000 coronary artery bypass graft (CABG) procedures performed annually [1]. The three main conduits used for myocardial revascularization are the internal thoracic artery (ITA), radial artery (RA), and saphenous vein (SV) [2]. The choice of conduits has been the topic of many reviews and here follows a brief overview of the selection and rationale for their use. The SV is the most commonly used conduit for cardiac revascularization in patients undergoing CABG due a number of practical advantages: it is expendable, since lower limb venous drainage can rely solely on the deep venous system; its long length allows its use for multiple grafts and its superficial position renders it easily accessible, facilitating its exposure at harvest [3]. However, it is generally accepted that the performance of the SV is inferior to the ITA [4,5]. In the past, many groups favoured the use of total arterial grafting combining both the ITA (as a single or bilateral, left ITA and right ITA) and RA as bypass conduits [6]. However, the review by Schwann et al. [7] describes a reduction in the use of total arterial grafts, driven in particular by the decreased use

of the RA. Nevertheless, Audisio et al. [8] suggest that the RA is superior to the SV with its use becoming standard of care for the treatment of patients with multivessel coronary artery disease [8]. In contrast to conventional (CT) harvesting, where the perivascular fat (PVF) is removed and adventitia damaged, the no touch (NT) technique of SV harvesting, first described by Souza, preserves the cuff of surrounding tissue [9]. Here, the course of the SV is marked on the skin pre-operatively using duplex ultrasound enabling an incision to be made directly over the SV and minimising wound complications. The SV is then harvested using diathermy and scissors to include a surrounding fat pedicle of about 0.5 cm. The visible side-branches are ligated and divided at the same distance from the SV. After removal, the SV is stored in heparinised blood from the aortic cannula. During anastomoses, the SV is handled only via the surrounding PVF tissue, avoiding direct contact with the vein, a procedure that prevents spasm and obviates the need for high pressure intraluminal distension [10]. In follow up studies the patency of NT grafts is superior to CT grafts at up to 16 years and comparable to the ITA [11–14]. In general the comparison between the use of arterial and SV grafts has been based on data obtained from CT SV grafts used in CABG. In a study where the RA was compared with NT SV, NT SV grafts were shown to have an excellent patency similar to that of RA grafts after 8 years with the additional benefit that NT SV grafts can be used in situations that are not ideal for RA grafts [15]. When commenting on early RA vs. SV trials it has been suggested that these could be described as comparing apples with oranges since NT data was lacking [16]. Furthermore, in their Editorial commenting on the analysis of the RADIAL trial that compared RA and SV data [17], Kopjar et al. emphasize the fact that when comparing data on RA and SV grafts NT SV data was excluded [18]. When the angiographic patency data of the five trials with protocol driven angiography were supplemented with NT SV data the difference in risk of graft occlusion between the RA and SV grafts dissipates ($p = 0.35$). More recently, those proponents of RA grafts for CABG appear to accept the potential advantage of SV grafts where the NT technique has been used [19,20], a situation supported by a number of recent meta analyses [20–25].

Minimally invasive endoscopic vein harvesting (EVH) was also introduced in 1996, the same year as the NT SV harvesting technique. This method reduces leg wound complications, infection rate, and pain and improves cosmetic results [26] and used in many centers worldwide and in the majority of CABG in the United States [27]. However, there have been concerns regarding the quality of EVH-harvested grafts following issues regarding decreased graft patency, increased reoperation rate, and myocardial infarct, problems that are associated with vascular trauma caused when using this technique [18,28,29]. EVH is usually performed with carbon dioxide insufflation where small incisions are made under physical retraction. In order to introduce the endoscope and to harvest the SV, a subcutaneous tunnel is formed where the carbon dioxide pressure of ~15 mm Hg allows an easier separation from the SV surrounding tissue. Once the dissection is complete side branches of the SV are ligated using bipolar scissors or ligaclips and the SV removed. While EVH reduces leg wound complications the effects of traction, adventitial stripping, and venous compression cause vascular damage including that to the endothelium [30] and to the vasa vasorum (VV) [31]. Although EVH is the method of choice in the USA and other countries the question arises, is endoscopic or NT SV Harvesting for CABG best for the patient [32]? Since recognising the superiority of the NT technique a number of recent studies have focused on minimally invasive methods of NT SV harvesting in an attempt to reduce wound healing issues [33–36].

Although a number of recent trials show an improved patency of NT SV grafts in CABG [37–40] there are those sceptical of NT SV graft superiority [41–45]. For example, the most recent SwedeGraft Trial, based on data from a total of 902 CABG patients and operated in 10 centers in Sweden and Denmark, showed that, at 3.5 years, 19.8% NT versus 24.0% CT SVGs were occluded. On the basis of this data the authors concluded “No-touch vein graft harvesting for CABG was not superior to conventional open harvesting” [44]. This was a poorly-conducted trial for a number of reasons with Gaudino and Sandner raising a number of critical issues. In their Editorial is mentioned the use of outdated vein graft failure data which “represents a critical flaw in sample size calculation” and the low imaging follow-up rate in this trial [46].

1.2. Significance of Saphenous Vein Vasa Vasorum during and after CABG

Vasospasm occurs during harvesting in a high proportion of grafts that may have both immediate and long term adverse effects [47–49]. The patency of the SV as a conduit in patients undergoing CABG is improved when harvested with minimal trauma using the NT technique [11,13,14]. This technique differs from the CT technique [9,50] that was first described by Favorolo over 50 years ago [51,52]. Here the PVF is removed, the vein distended and adventitia and endothelium damaged. By contrast NT SV are harvested atraumatically with no distension used, the PVF intact and endothelium and adventitia undamaged [53–55]. NT SV harvesting provides a graft with and improved patency compared with CT grafts at up to 16 years and comparable to the ‘gold standard’ ITA [12,13,56]. A variety of factors underlying the improved patency of NT SV grafts have been described

including a reduction of vascular damage thus maintaining normal SV architecture. Consequently, preservation of the cushion of PVF and an intact endothelium and VV is achieved [57]. While the potential role of the PVF and endothelium has been described previously [58–60] here is presented an overview of studies and a hypothesis on the possible role of the VV in the improved patency of NT SV grafts used in CABG.

The pronounced cushion of PVF surrounding NT SV protects against the high pressure saline distension used during CT harvesting [3,57,61]. At CABG, when harvesting the SV, the 'manual' pressures used may reach extremely high levels [62] and in their elegant study measuring intraluminal pressure, Roubos et al. showed that the peak pressures required to prevent spasm of SV at CABG approaches 500 mmHg [63]. Early studies on NT SV examined the effect of 300 mm Hg saline distension pressure on CT SV, where the PVF is removed, revealed significant damage to the luminal endothelium as demonstrated by both immunohistochemistry [61,64,65] and ultrastuctural studies [66]. This endothelial damage is accompanied by reduced endothelial nitric oxide synthase (eNOS) protein expression and nitric oxide (NO) production [61,67]. The cushion of PVF therefore possesses a protective, buffering, role where it provides mechanical support that opposes the effect of high distension pressures used at harvesting. Since this protective effect is seen at 300 mm Hg intraluminal pressure, the preservation of PVF on NT SV grafts would be expected to protect the graft once implanted into the coronary arterial circulation where the graft endothelium is subjected to a pressure of ~100 mm Hg [61,68].

At CT harvesting there is often damage to the adventitia when following the original preparation instructions, "Care must be taken to dissect only the vein, avoiding as much as possible the adventitia that surrounds it" [52]. When stripping the adventitia the VV is damaged. This microvascular network provides oxygen and nutrients to the media and is more prominent and penetrates deeper in veins than in arteries [11,69–71]. By harvesting the SV using the NT technique the adventitia is not damaged and the VV remains intact [11,72]. Apart from providing blood to the SV vessel wall it has been suggested that the VV may also act as a transport system from the PVAT through the SV wall terminating in the lumen [59,60,73] (Figure 1). In support of early ultrastructural studies [74], openings suggested to represent the termination of VV in NT SV lumen have been observed using scanning electron microscopy (SEM) [75]. An ultrastructural study using transmission electron microscopy (TEM) has revealed altered shape changes in CT vs. NT SVs where medial and adventitial CT VV collapse with erythrocyte clumping occurring in some regions [66] (Figure 2).

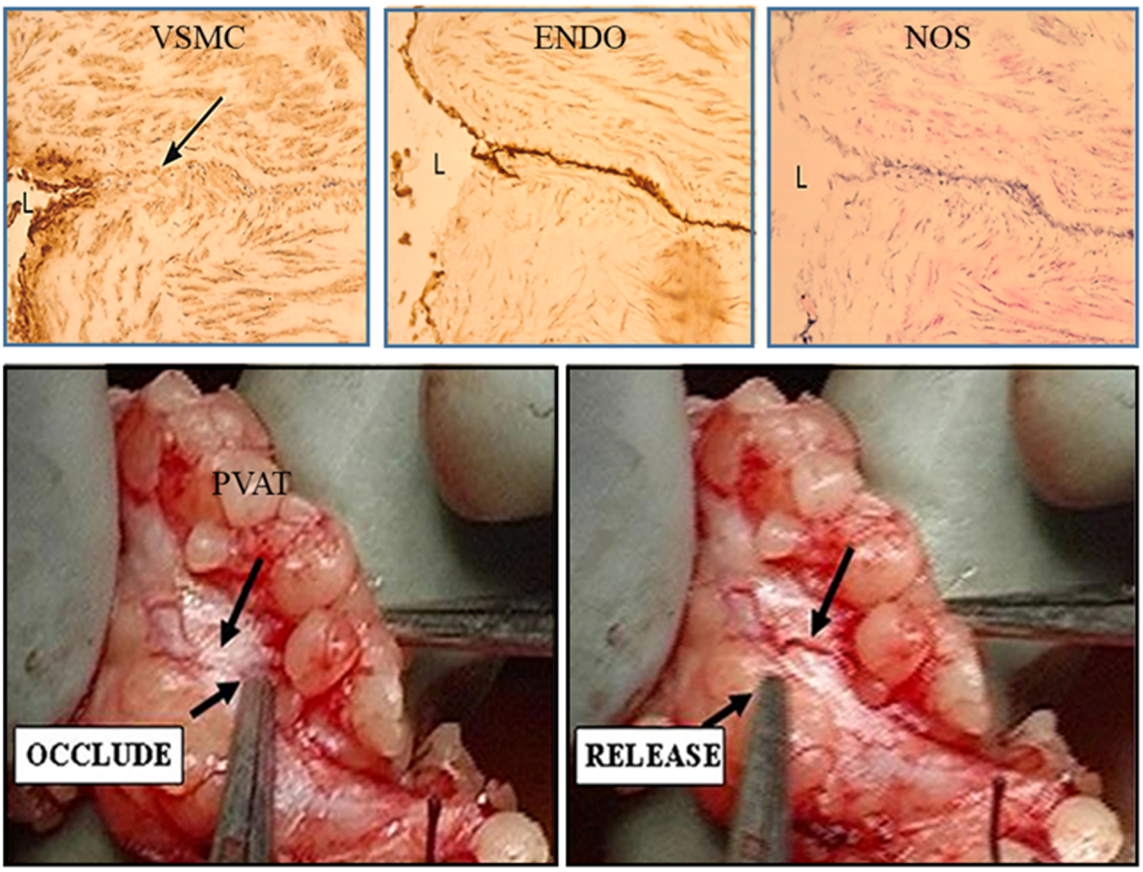

Figure 1. Retrograde flow of blood from lumen to adventitia of no touch saphenous vein. Top panels show a 'channel' through the vessel wall that terminates in the lumen (L) of a no touch SV. The **left panel** shows vascular

smooth muscle cells (VSMC) identified by alpha smooth muscle staining (dark brown staining). The non-stained channel is shown by the arrow. The **middle panel** shows the endothelial cells on an adjacent section (ENDO) lining the channel using CD31 antibody—brown stain. The **right panel** shows nitric oxide synthase (NOS) staining (blue) using NADPH-diaphorase histochemistry that is endothelium dependent (modified from [76] with permission. Copyright 2004, Sage Publications). Lower panels show blood flow from the cardiopulmonary bypass machine in an isolated segment of no touch saphenous vein perfused from the lumen. **Left**: Prevention of blood flow through adventitial vasa vasorum caused by pressure applied by tip of forceps (OCCLUDE—between arrows). **Right**: Restoration of flow at release of forceps—between arrows (from [11] with permission. Copyright 2011, Elsevier).

By contrast the VV in NT SV appear patent and contain normal shaped erythrocytes within the VV lumen. The potential of the VV connecting the lumen of the SV with the outermost SV layers is suggested to explain the retrograde blood flow to the adventitial VV that is observed at completion of proximal anastomosis and removal of vascular clamps at conclusion of CABG as described by Dreifaldt et al. [11]. Based on these observations at surgery an 'inside out/outside in' system of transport through the SV wall has been proposed [59,60,73]. This system has also been demonstrated experimentally where *ex vivo* India ink perfusion via the lumen of isolated NT SV explants stains the luminal endothelium and VV within the media and adventitia extending to the capillary network within the perivascular fat [59,60] (Figure 2).

As a microvascular network providing the vessel wall with oxygen and nutrients, the VV is more prolific and penetrates deeper in veins than in arteries [11,69–71]. Interestingly, an observation over 50 years on SV used for arterial reconstruction states, "the vasa vasorum in the arteries only penetrate to the media, whereas the saphenous vein vasa vasorum are 5–8 times more numerous and penetrate through the entire wall thickness into the lumen" [70]. In a pig experimental model, occlusion of the VV in arteries by a close-fitting external cuff causes neointimal hyperplasia (NIH) and eventual appearance of atherosclerotic lesions due to transmural ischaemia [77]. Furthermore, removal of the adventitia of the rabbit carotid artery causes NIH which is reduced on the appearance of neovascularization and neoadventitia [78]. A cinematographic study performed over 40 years ago on post-mortem atherosclerotic human coronary arteries identified microvessels growing from native VV that extended from the adventitia to the thickened intima of atherosclerotic vessels. These were suggested to represent a beneficial process in the restoration of blood flow to the vessel wall and improved oxygen and nutrient supply [79]. A study of the distribution of VV in failed SV grafts has been performed by Stingl et al. [80]. Here, the authors describe various changes that occur to vascular smooth muscle cells, the thickness of the different layers of the vessels and to the VV in post-implanted SV grafts at different time periods. Interestingly, in this study, it was shown that the density of VV increases and that the VV penetrates deeper towards the lumen over time and in relation to plaque formation. In the study by Dreifaldt et al. [11], endothelial cells of the VV were identified on histological sections by immunohistochemistry where the total area of VV was significantly greater in the media and adventitia of NT vs. CT SV. A later study compared the density and distribution of VV in the ITA, RA and CT SV vs. NT SV [72]. Here, it was confirmed that the density of VV was significantly greater in the SV than both the ITA and RA and that the SV VV penetrates deeper towards the lumen. In addition, the density and size of the VV was greater in the adventitia than in the media of the SV and there was a significant reduction in the density of VV in CT compared with NT SV due to removal of, or damage to, the adventitia at harvesting when using CT SV for CABG. It has been proposed that what appear as intimal folds in NT SV may in some instances represent VV endings or terminations in the lumen based on histological observations [76] (Figure 1). While this suggestion has been queried by Stingl et al. [80], it is supported by SEM studies on NT SV [74,75] and by the *ex vivo* demonstration of transmural blood and India ink flow perfused from the lumen to the adventitia of NT SV [59,60] (Figure 2).

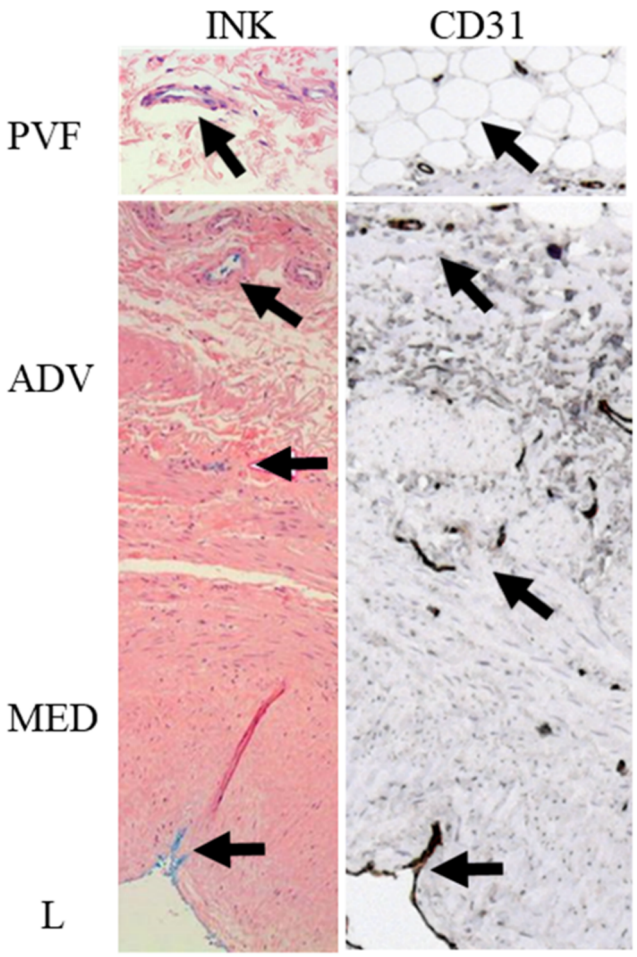

Figure 2. NT SV VV; inner to outer connection? **Left**: India ink staining (blue) from lumen (L) via the media (M) and adventitia (ADV) to capillaries in the perivascular fat (PVF) in *ex vivo* perfused segment of NT SV (from [59] with permission. Copyright 2016, Bentham Science Publishers). **Right**: Endothelial cells stained with CD31 (dark staining) of the luminal (L) endothelium and VV within the M and ADV extending to the capillaries in the PVF [81]. Arrows indicate endothelial cells of the lumen and vasa vasorum.

Here we might consider certain aspects of the study by Stingl et al. [80] where time-related changes in SV grafts were assessed from both post mortem samples and grafts obtained from patients undergoing reoperation after CABG due to graft failure. Clearly, these are 'abnormal' SVs when compared with normal SVs used as conduits for CABG. In the methods section the vague short statement, "[the SV was]… dissected as carefully as possible…" is made. Presumably the SV was harvested by the CT technique and therefore distended and perhaps treated with anti-spastic agents. From the histology examples provided it seems that the PVF was removed, a procedure that in many cases damages the outer adventitia. For standard histology the SV grafts were fixed; was immersion or perfusion fixation used? If perfusion fixation was used, what pressure was applied? Also, this study was performed on SV grafts following CABG in SVs that had presumably been surgically manipulated and distended at harvesting as well as being subjected to coronary arterial pressure of ~100 mmHg over different time periods. Such conditions not only alter the density and distribution of the VV but also effects other features of CT SV grafts [57].

The following section focuses on NT SV before CABG and harvested with minimal trauma and where normal architecture is preserved compared with those harvested by 'damaged' CT grafts. Blood flow is conserved in NT SV VV *in vivo* and *ex vivo* with recent evidence of maintained patency of adventitial NT SV VV at 2 weeks after CABG.

1.3. SV Vasa Vasorum: SEM and TEM Data

Based on SEM studies of corrosion casts of human SV, a complex architecture of the vein VV was observed and where no direct openings of the VV to the lumen were detected [82,83]. Instead, the venae vasorum emptying into the terminal segments of largest tributaries of SVs were identified [84]. An earlier SEM study on NT SV grafts

used in CABG showed that the luminal endothelium of these grafts was preserved whereas the endothelium of CT SV grafts was damaged [10], as supported in the later study by Vasiliakis et al. [75]. A novel finding was the detection of openings close to the SV lumen, possibly terminations of VV [11,74,75] (Figure 3).

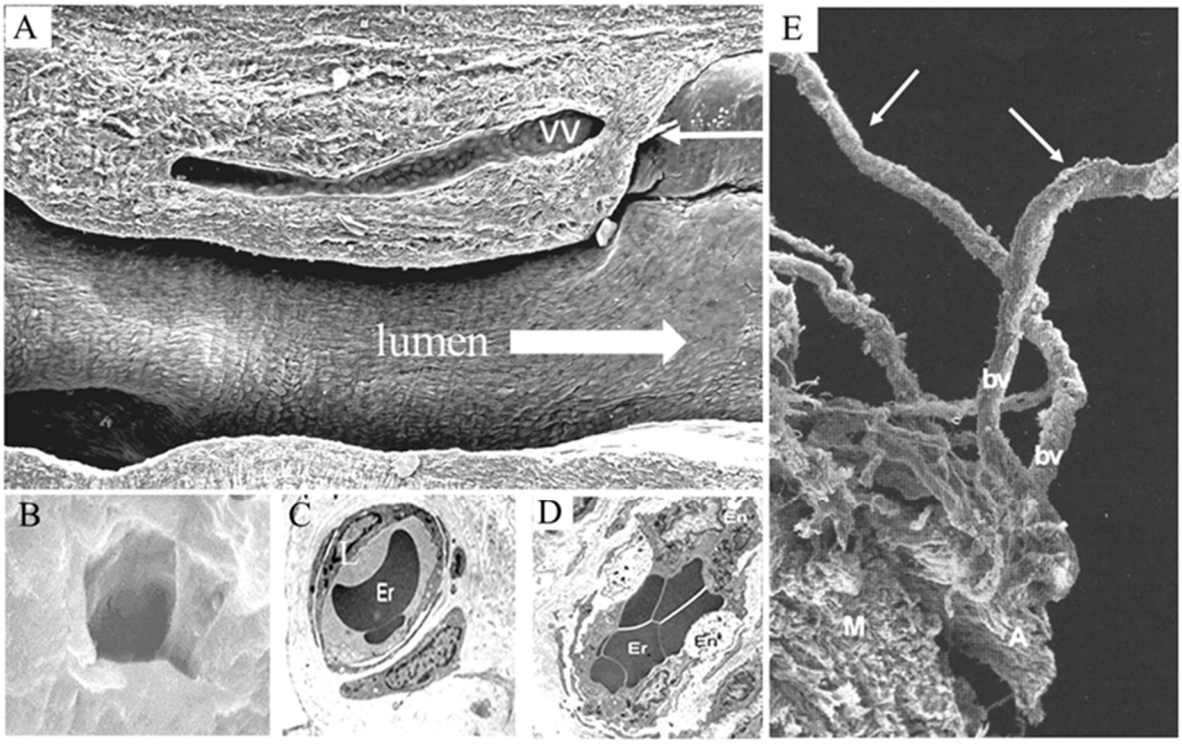

Figure 3. Saphenous vein vasa vasorum: scanning and transmission electron microscopy. (**A**) SEM of a longitudinal section of no touch saphenous vein showing an intact luminal endothelium with direction of blood flow (large arrow). Possible retrograde flow through the vasa vasorum (VV) shown by the small arrow (from [74] with permission, Copyright 1999, Taylor & Francis). (**B**) SEM showing an opening (of about 5 μm diameter), a possible termination of vasa vasorum within the intact luminal endothelium of a no touch saphenous vein (from [11] with permission, Copyright 2011, Elsevier). (**C**) TEM of vasa vasorum in the media of a no touch saphenous vein. Erythrocytes (Er) within an open vasa (of about 10 μm diameter) with surrounding normal endothelial cell (from [66] with permission, Copyright 2004, Sercrisma International S.L.). (**D**)TEM of vasa vasorum in the media of a conventional saphenous vein. There is a clump of Er occluding the lumen of a vasa (of about 20 μm diameter) with an abnormally shaped endothelial cell (En) (from [66] with permission, Copyright 2004, Sercrisma International S.L.). (**E**) SEM of the media (M) and adventitia (A) border of a conventional saphenous vein where the vasa (bv; of about 20–25 μm diameter) have been disconnected at harvesting (from [75] with permission, Copyright 2004, Vascular Disease Prevention, Bentham Science Publishers).

Anatomically, the VV originate from feeding vessels in the adventitia and penetrate the media to form an elaborate network of different sized arterial and venous vessels and capillaries that reach deep into the inner media [84,85]. In addition to supplying oxygen and nutrients to the vessel wall the VV plays a role in preserving vein wall elasticity in response to intraluminal pressure changes [84]. The structural changes of the CT SV VV observed at the ultrastructural level are likely in response to high pressure saline distension and surgical trauma used in these grafts at harvesting [66,75] and that may influence the performance of SV grafts used in CABG. Also, such changes may affect the formation of the corrosion casts produced when mapping the human VV, particularly in those SV obtained either post-mortem or in SV grafts used in CABG reoperation [84].

The VV of veins follow a more tortuous path than those of arteries [86], and the VV identified by positive CD31 immunostaining in SVs may represent the “meandering” course taken by a single microvessel running along the vein or passing through the media and terminating in the vessel lumen [11,76] (Figure 2). It is also notable that such points of entry to the vessel lumen are infrequent. It has been shown that, in canine SV, the VV is made up of a network of structurally separate units, each with an annular or collar distribution in the vein [87,88]. Also, in the dog, the luminal termination of VV have been demonstrated after 14 days in the neointima of experimental SV grafts using silicone rubber casts. These VV were frequently observed along the region of anastomosis running throughout the media and adventitia and connecting to the original VV. After six months those VV terminating in

regions of neointimal hyperplasia (NIH) of the lumen formed a network of microvessels once NIH was more than 250 μm thick [89]. If there is a similar distribution in human SV their identification at the light microscope level may be more challenging and even more so using electron microscopy [76].

Subsequent SEM studies have described intimal features suggesting the existence of such direct openings into the human SV lumen [11,73,90]. It is unclear, however, if these openings contribute to retrograde blood flow to the SV graft wall. Various aspects of this issue have been discussed previously [73,91]. Regarding the corrosion cast studies [84], the VV may not fill *ex vivo* as the viscosity of the resin used is around 5–10 times higher than that of blood. Also, in this study, the SV samples used were 12–24 h post mortem: such dead tissue will be affected by necrotic changes and the release of constricting hypoxic factors. Those SV samples obtained at harvesting during CABG were stored in ice-cooled heparinized saline and cast within 6–10 h and then examined under SEM. These conditions may affect the flow of resin through VV due to time delay after SV excision as well as cold- and hypoxic factor-induced constriction. Perhaps oxygenated physiological Krebs solution, such as such as that used in (functional) organ bath studies, should have been used for storage and transport [73]. The recent review by Loesch [92] discusses in detail the "intimal openings" of the SV. Here the author's previously published and unpublished data is presented describing observations in SVs from CABG patients obtained using traditional histology as well as SEM and TEM. The openings discussed and illustrated range from 5 μm to 20 μm and are suggested "to be a part of blood vessels, such as branches of tributaries, rather than terminals of the vasa vasorum". While the proposal that certain intimal folds observed at the light microscope level represent the luminal termination of VV [76] is questioned there is functional evidence, both *ex vivo* and *in vivo,* demonstrating the communication from the lumen via the media and adventitial VV to the capillary network of the PVF [59,60].

A recent study compared the morphological features of the specimens of healthy and varicose SVs in order to determine any significant differences between the groups. Variations were identified where there was a highly significant difference in maximum venous diameter between the control group (mean 0.52 cm) and varicose vein group (mean 0.68 cm) (p-value of 0.0002). Also, wall thickness in the varicose veins group (mean 0.206 cm) was significantly higher than in the control group (mean 0.138 cm) (p-value of 0.014). Of particular relevance to this review was the observation that there was a significant difference in VV in the adventitia of varicose SVs compared to the control group ($p = 0.050$) leading to the authors' 'plea to avoid the use of varicose veins as a graft' [for CABG] [93].

Another study compared SV conduits in 60 CABG patients where in 30 patients the SV was harvested using the classical method described by Favaloro (1969) [52] and in the other 30 patients the SV was "procured in the composition of perivascular adipose tissue without hydraulic dilatation" (i.e. NT SVG). On microscopic examination of SV sections at different magnifications, endothelial damage was assessed as well as the preservation of the VV. The SV harvested with PVF intact (NT SV) exhibited a higher density of VV and a reduction in endothelial damage compared with the 'Favaloro-harvested' (CT) SV [94], confirming the previously published results of Dreifaldt et al. [72]. A similar study investigated the preoperative incidence and severity of NIH and level of blood supply of arterial and venous conduits used in CABG [95]. Here, paired ITA and SV segments were harvested and visualised by SEM in back-scattered electrons to measure thickness of NIH and number/area of VV using imaging software. Although NIH was observed more frequently in SV versus the ITA this did not reach significance. However, the maximal-to-minimal NIH ratio correlated with the percentage of stenosis ($p < 0.0001$) as well as the area ($p = 0.023$) and the number ($p = 0.015$) of the VV in both the conduits. Based on these results it was concluded that NIH correlates with the area and number of the VV in the conduits studied. Furthermore, this study shows that the SV possesses a larger number and higher density of VV when compared with the ITA, and that the number of VV is correlated with stenosis of the VV more closely than with stenosis of the ITA.

2. Prevention of Vasospasm Encountered at SV Graft Harvesting

As previously discussed, vasospasm is encountered in a high proportion of SV grafts when harvested by the CT. This spasm is due to vascular damage and is avoided using NT SV as the PVF remains intact and vascular damage is minimized. An enhanced formation of VV has been demonstrated after the implantation of drug-eluting stents that is associated with coronary hypoconstriction *in vivo* in pigs, a response that is elicited through the activation of Rho-kinase [96]. In a study by the same group VV formation was assessed in vasospastic angina patients using optical frequency domain imaging and if there was a correlation between the extent of VV and that of coronary vasoconstriction. Intracoronary optical frequency domain imaging was performed along the coronary artery, after administration of intracoronary isosorbide dinitrate after the spasm provocation test with morphometric analysis of images performed at regular intervals along the artery. It was shown that VV formation

was markedly enhanced at the spastic artery in a patient with vasospastic angina compared with a control subject. VV area density was significantly greater in the angina group compared with the control group ($p < 0.001$). Also, in the angina group, there was a significant positive correlation between VV area density and the extent of coronary vasoconstricting responses to ACh (<0.001) as well as a significant positive correlation between VV area density and Rho-kinase activity in circulating leukocytes ($p < 0.001$). On the basis of these results the authors conclude that adventitial VV formation is enhanced at the spastic coronary segment in angina patients suggesting the involvement of adventitial VV formation in the pathogenesis of the spasm [97]. In a study using 18F-fluorodeoxyglucose positron emission tomography/computed tomography, ACh-induced diffuse spasm in the left anterior descending artery in patients with angina was compared with subjects with suspected angina but without organic coronary lesions or coronary spasm [98]. It was shown that coronary PVF volume and coronary perivascular 18F-fluorodeoxyglucose uptake significantly increased in the angina group compared with the non-angina group. Also, OCT showed that formation of VV was increased significantly in the angina compared with the non-angina group as did Rho-kinase activity. These results were concluded to provide evidence that coronary spasm is associated with inflammation of coronary adventitia and PVF and that 18F-fluorodeoxyglucose positron emission tomography/computed tomography may be useful for disease activity assessment.

2.1. Effect of Perivascular Fat

Vasospasm is often encountered at harvesting, a condition that has both immediate and long term effects on graft performance. Spasm may be abolished or reduced when PVF is left intact at harvesting and this is particularly the case for the NT SV. According to a retrospective review of CABG patients, vasospasm and graft occlusion is less common in arterial grafts with PVF intact than in patients receiving SV grafts with PVF removed [99,100]. Preservation of PVF is linked to better graft patency and reduced number of adverse cardiovascular events when comparing the results of pedicled versus skeletonized arterial grafts [56,101]. PVF releases a range of adipocytokines and chemokines that modulate vascular tone and reactivity through the release of several biologically active substances, including perivascular relaxing factors and perivascular contractile factors [102]. Primary perivascular relaxing factors include adiponectin, angiotensin (Ang) 1–7, leptin, omentin, NO, and hydrogen sulfide, while perivascular contractile factors include superoxide and angiotensin II (Ang II) [103,104]. NO has a variety of recognized effects on blood vessels where NO released from endothelial cells dilates the vessel by acting on adjacent vascular smooth muscle cells. Furthermore, NO reduces inflammation and inhibits oxidative stress in the vessel wall [105]. PVF also reduces vascular tone in response to noradrenaline through the involvement of L-type Ca^{2+} channels and by releasing both PGE2 and prostacyclin (PGI2) [106,107]. Moreover, PVF of the SV functions as a source of PGE2 and PGI2, which facilitate vasorelaxation of SV via EP4 and IP receptors, respectively [107,108].

2.2. Pharmacological Agents

A variety of antispastic strategies and/or drugs have been developed including submersion in heparinised blood, saline containing papaverine, nitroglycerine, NO donors and mixed anticontractile cocktails [47,109]. Nitrovasodilators such as glyceryl trinitrate, sodium nitroprusside and isosorbide dinitrate, are commonly used in patients undergoing CABG that act by releasing NO [48]. Recently, Hou et al. [110] assessed the antispastic effect of a combination of the RhoA/Rho-kinase inhibitor, fasudil, with and without nitroglycerin in the ITA. Here, fasudil fully relaxed certain vasoconstrictor-induced contractions and diminished protein levels of ROCK2 in the ITA. Also, when a combination of fasudil and nitroglycerin were used together, a more pronounced impact was observed than when using either vasodilator alone. Calcium antagonists such as nifedipine, diltiazem and verapamil used alone or in combination with GTN have been used as antispastic drugs in arterial grafting [111–113]. The calcium antagonists that are particularly effective in preventing or curing potassium-induced contraction in the ITA [114] or RA [115] that cause vasorelaxation of vessels by reducing calcium influx by blocking voltage-gated (L-type) calcium channels. Other calcium channel blockers used are Diltiazem, which is less effective than nifedipine in the ITA [114] with little effect on the RA [115]. This observation revitalized the use of RA grafts after a period of about 20 years due to significant spasm issues [116]. Injectable verapamil exhibits a greater potency than diltiazem in human vasculature and is therefore commonly used in antispastic protocols [48,117]. A cocktail of nicardipine and nitroglycerin provides a useful antispastic procedure on human ITA and RA having a quick onset, complete vasorelaxation and superior prophylactic impact [118]. The opioid derivative, papaverine, causes vasodilation by inhibiting phosphodiesterase resulting in a reduction of calcium influx and the inhibition of intracellular Calcium release [119] but is not recommended for systemic or topical application due to its acidity which may impair endothelial function [49]. The onset in vasodilation by papaverine is more gradual compared to other vasodilators [112,113]. Phosphodiesterase

inhibitors are clinically important with their positive inotropic and vasodilator effects [115]. Milrinone, a PDE-III inhibitor, increases intracellular cAMP, inhibits myosin light chain kinase and causes vasodilatation in the vascular system [119] and has been shown effective on the ITA [114] or RA [115].

Given the abovementioned effects of PVF (via perivascular adipocyte-derived factors) and the various pharmacological agents used it may be proposed that, apart from their beneficial anti-contractile properties on the conduit in question (SV, ITA or RA), they protect the vessels' VV at harvesting during CABG. In doing so transmural flow would be maintained, medial ischaemia prevented and NIH reduced and/or abolished.

3. Hypothesis: The Vasa Vasorum and SV Graft Patency

Two decades ago it was hypothesised that preservation of an intact VV may play a role in improving SV graft patency [76]. This hypothesis is based mainly on the fact that the major cause of the high failure rate of CT SV grafts is the degree of vascular trauma inflicted during SV harvesting and at graft insertion. Manipulation of the outer layer of the SV (stripping) during CT surgery induces vasospasm and is prevented using 'manual', uncontrolled, high-pressure intraluminal saline distention causing severe damage to the endothelium and VV. There are clear indications strongly suggesting the importance of the VV for the maintenance of a healthy graft, as can be observed both experimentally and in surgery. Examples include *in vivo* observations using NT SV in CABG where blood flow to the adventitial VV occurs at completion of graft insertion and removal of vascular clamps [11] (Figure 1). This is taken as evidence of retrograde blood flow from the lumen to the adventitia and supported by subsequent *ex vivo* studies on excess segments of NT SV using the luminal perfusion of blood [11] or India ink [59] that appeared in the adventitial VV even extending to the capillary network within the preserved cushion of PVF of NT SV (Figure 2). Taken together, these observations may reveal a novel 'inside out/outside in' system that not only maintains transmural blood supply in NT SV grafts but that may potentially transport a variety of tissue-derived factors throughout the vessel wall [59,73].

As mentioned previously, proliferations of the VV/neovascularization has been described in atherosclerotic coronary arteries post mortem [79] and that there is an increased density of 'neo vasa vasorum' associated with increased NIH thickening and atherosclerotic plaque formation. These features are proposed to be a mechanism by which transmural blood flow is maintained [120]. A more recent study by Stingl et al. [80] suggests that a similar scenario occurs *in vivo* where the density of VV in the adventitia and media of SV grafts increases and the VV penetrates deeper through the vessel wall towards the lumen over time in patients after CABG. Again, this proliferation of VV may represent a means by which transmural blood supply is maintained in (failing) grafts. The appearance of the histological sections provided in this study, indicates that CT (damaged) SV grafts were used since the PVF has been removed, a procedure potentially causing damage to the outer adventitia and VV. In NT SV normal architecture is preserved, including the VV. Since retrograde filling of adventitial VV has been demonstrated *in vivo* and *ex vivo* [59,60,72] it is possible that transmural oxygen supply is provided both by diffusion via luminal coronary arterial blood and through an intact VV system (Figure 4).

The SEM and light microscope studies provide evidence of a potential connection from the SV lumen to the adventitia, possibly even to the PVF, via the VV. These findings are likely to explain the retrograde blood flow observed at the time of NT SV graft implantation at CABG as well as the experimental observations in isolated NT SV segments using perfused blood and India ink. Another potentially important finding using SEM was the 'disconnection' of VV at the media/adventitia border of CT SV grafts (Figure 3) [75]. This is as a result of the vascular trauma at CT SV harvesting where the PVF is removed and the adventitia partially damaged, as occurs in many cases. Furthermore, ultrastructural observations reveal VV in the wall of CT SV that appear occluded by clumps of erythrocytes and with abnormal surrounding endothelium and vascular smooth muscle cells whereas NT SV VV show an open lumen with normal endothelium containing individual erythrocytes [66] (Figure 3).

Taken together, what might these observations suggest regarding the role of the VV in the improved patency of NT SV grafts? After CABG is the VV of NT SV grafts preserved and is transmural flow maintained [72]. If so, this would be expected to reduce intimal hyperplasia/plaque formation as demonstrated by intravascular ultrasound [121] as well as by angiography [13], computed tomography angiography [44,122] and electrocardiogram-gated enhanced computed tomography [40,123,124].

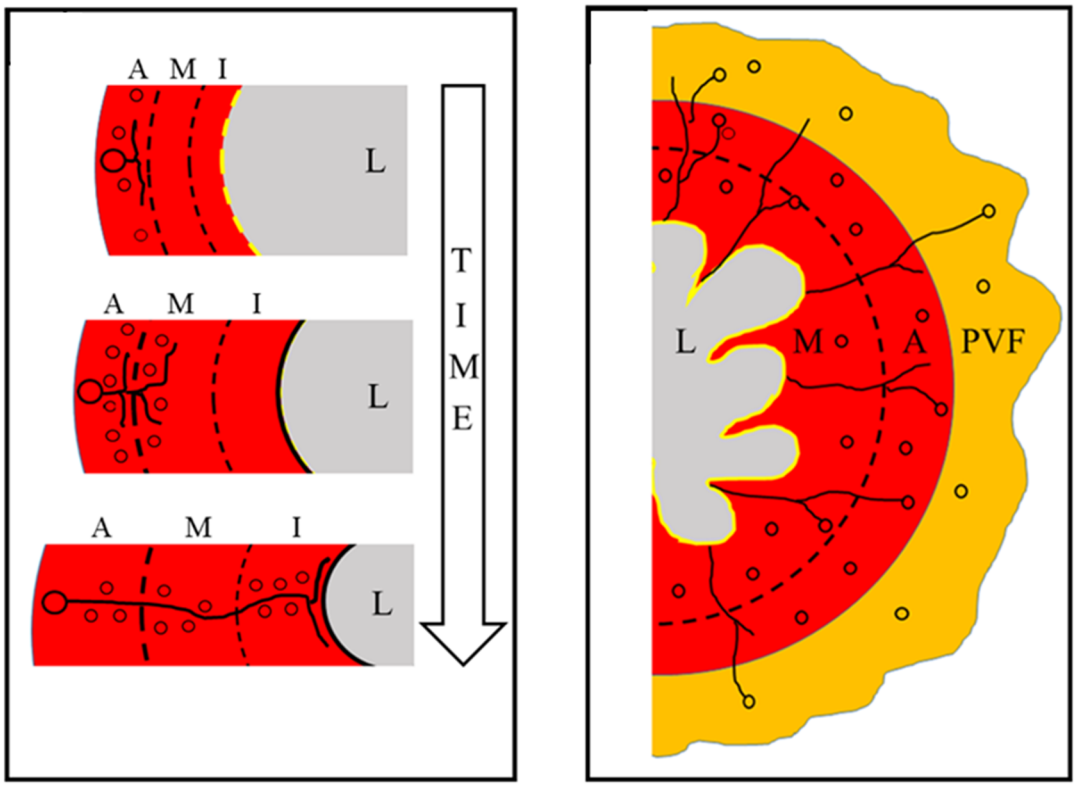

Figure 4. Potential blood flow in NT SV vasa vasorum after CABG. **Left panel**: Diagram of density and distribution of vasa vasorum in failing SV grafts after CABG. There is a time-dependent increase in wall thickness and reduction in lumen diameter. There is also a time-dependent increase in density and depth of penetration of the vasa vasorum through the vessel wall towards the lumen. **Right panel**: Proposed distribution of vasa vasorum in no touch SV after CABG. Vasa vasorum/capillaries are present within the perivascular fat (PVF) which is preserved in NT SV but removed in CT SV. There is also a greater density of (preserved) VV in the M and A of NT SV. A proportion of oxygen requirement of the vessel wall is by diffusion from arterial blood from the lumen with an additional supply provided via VV terminating in the lumen (irregular lines) some of which reach the M, A and PVF. The endothelium (continuous yellow line at the L/M border) in NT SV is undamaged before implantation. Circles = vasa vasorum, black broken lines = borders (between A, M and I). Black continuous irregular lines = course of VV ('outside in' for post CABG CT SVG and 'inside out' for NT SVG). A = adventitia, M = media, I = intima, L = lumen.

How are these questions to be addressed? The preservation of an intact/'healthy' endothelium have been shown on NT SV grafts at up to 16 years post-mortem [13]. Is the same true for the VV and are appropriate post mortem samples available to confirm this? If so, histological and ultrastructural studies are recommended. Are the necessary, sensitive, methods available to study the distribution and blood flow in the VV in patients' SV grafts to determine their potential role in graft patency? The recent study by Sugaya et al. [124] identified VV in the adventitia/PVF area of NT SV grafts *in vivo* in patients 1 to 2 weeks after CABG using frequency-domain optical coherence tomography (Figure 5) [124]. The volume of VV per mm was significantly greater in NT than in CT SV grafts. Interestingly, the *in vivo* distribution of VV in NT vs. CT SV grafts is very similar to that described in histological sections of comparable grafts from CABG patients taken at harvesting and before implantation [11,72] as well as in early failed SV grafts [80]. Although the recent *in vivo* data was obtained over a short follow up period [124] it shows that the VV remain viable and supports the suggestion that transmural blood supply is maintained in NT SV grafts after CABG, based on previous *in vivo* and *ex vivo* perfusion studies [59,60,72]. Clearly, once harvested, the VV are disconnected from their 'normal' arterial blood supply *in vivo* via the external iliac arteries. The images obtained *in vivo* using frequency-domain optical coherence tomography show the presence of adventitial VV at 2 weeks after CABG that exhibit a circular morphology indicating that they are under [arterial] pressure. In terms of the potential importance of VV in SV graft performance is the striking similarity in their density and distribution in NT versus CT SV (Figure 5). Whereas the VV of NT SV remain intact and 'open', VV of CT SV exhibit signs of damage at the adventitial border as well as constriction and occlusion by erythrocytes in the media (Figure 3). Taken together these conditions support the suggestion that the VV play an important role

in the superior patency of NT SV grafts where transmural flow is maintained via an inside-out blood flow from the lumen via the media to the adventitia and PVF [60,73].

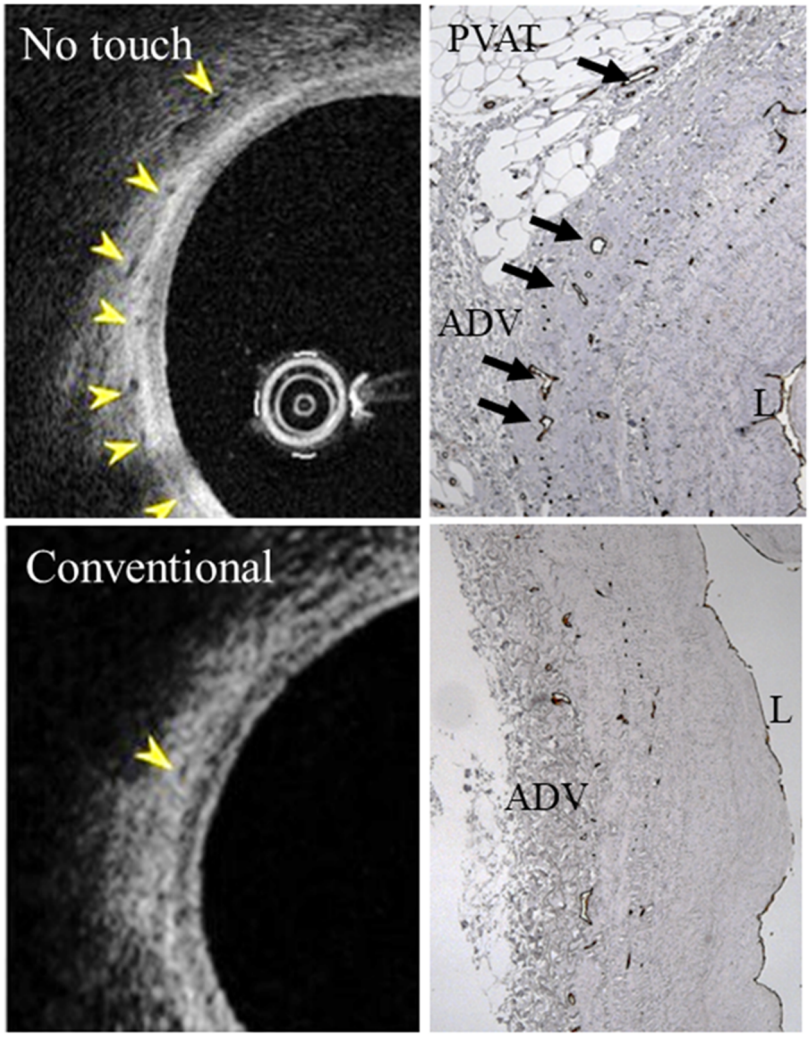

Figure 5. OCT images and histology of vasa vasorum of no touch and conventional saphenous vein. **Left**: The *in vivo* frequency-domain optical coherence tomography images in the left panels show adventitial NT and CT saphenous vein vasa vasorum at 1–2 weeks after CABG (yellow arrowheads (from [124] with permission, Copyright 2024, Oxford University Press). Despite being 'disconnected' from their feeding arteries at harvesting they appear circular, *in vivo*, once inserted as grafts. This indicates that they remain 'open' and that blood flow is maintained at coronary arterial pressure from the graft lumen. **Right**: Histology showing the vasa vasorum (black arrows) in the adventitia (ADV) (dark CD31 immunostaining identifying endothelial cells) where the distribution is similar to the frequency-domain optical coherence tomography images. The vasa vasorum is preserved after CABG since it exhibits the same distribution shown in the histology of SV before implantation as grafts. The density of VV in NT SV is greater than in CT SV [11], Copyright 2011, Elsevier.

4. Conclusions

Over recent years there has been increased interest in the VV, particularly in relation to the SV grafts used in CABG. Most data has been obtained using histology, immunohisto-chemistry, SEM and TEM and generally on SV samples obtained at harvesting and before preimplantation. However, little is known regarding any changes that occur and/or the role of the VV in the performance of grafts in patients after CABG. While there may be conflicting opinions concerning the luminal termination of VV, transmural blood flow from the innermost to the outermost layers of the SV has been demonstrated experimentally and in patients at completion of CABG. When using the NT technique, preservation of the PVF provides mechanical support that protects the endothelium and is a source of vasculoprotective adipocyte-derived factors. Also, by reducing surgical trauma and vascular damage, the VV is preserved and blood supply to the vessel wall restored. There is recent evidence that the VV remains intact in patients receiving NT SV grafts after CABG. In addition to the protection of the endothelium and

preservation of PVF, maintained transmural blood supply via the VV may further contribute to the improved performance of NT SV grafts. Future *in vivo* follow up studies are required on CABG patients in order to identify the VV, to examine VV distribution and to detect/measure VV blood flow over time.

Funding

This research received no external funding.

Acknowledgments

Andrzej Loesch is gratefully acknowledged for his important contribution to VV research and for his critical comments on earlier drafts of this manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

Abbreviations

ADV	Adventitia
bv	blood vessel
CABG	Coronary Artery Bypass Graft
CT	Conventional technique
en	endothelial cell
ENDO	endothelium
er	erythrocyte
EVH	endoscopic vein harvesting
I	Intima
NIH	Neointimal hyperplasia
ITA	Internal Thoracic Artery
L	Lumen
M	media
NO	Nitric Oxide
NOS	Nitric Oxide Synthase
NT	No touch
OCT	Optical Coherence Tomography
PVAT	Perivascular Adipose Tissue
PVF	Perivascular Fat
RA	Radial Artery
SEM	Scanning Electron Microscopy
SV	Saphenous Vein
TEM	Transmission Electron Microscopy
VV	Vasa Vasorum

References

1. World Health Organization. Cardiovascular Diseases (CVDs). Available online: http://www.who.int/mediacentre/factsheets/fs317/en/ (accessed on 22 December 2025).
2. Dimeling, G.; Bakaeen, L.; Khatri, J.; et al. CABG: When, why, and how? *Clevel. Clin. J. Med.* **2021**, *88*, 295–303.
3. Tsui, J.C.; Dashwood, M.R. Recent strategies to reduce vein graft occlusion: A need to limit the effect of vascular damage. *Eur. J. Vasc. Endovasc. Surg.* **2002**, *23*, 202–208. https://doi.org/10.1053/ejvs.2002.1600.
4. Alzghari, T.; Dimagli, A.; An, K.R.; et al. Conduits and Strategies for Arterial Revascularization in CABG. *Rev. Cardiovasc. Med.* **2023**, *24*, 188. https://doi.org/10.31083/j.rcm2407188.
5. Gaudino, M.F.L.; Spadaccio, C.; Taggart, D.P. State-of-the-Art Coronary Artery Bypass Grafting: Patient Selection, Graft Selection, and Optimizing Outcomes. *Interv. Cardiol. Clin.* **2019**, *8*, 173–198. https://doi.org/10.1016/j.iccl.2018.11.007.
6. Taggart, D.P. Current status of arterial grafts for coronary artery bypass grafting. *Ann. Cardiothorac. Surg.* **2013**, *2*, 427–430. https://doi.org/10.3978/j.issn.2225-319X.2013.07.21.
7. Schwann, T.A.; Tatoulis, J.; Puskas, J.; et al. Worldwide Trends in Multi-arterial Coronary Artery Bypass Grafting Surgery 2004–2014: A Tale of 2 Continents. *Semin. Thorac. Cardiovasc. Surg.* **2017**, *29*, 273–280. https://doi.org/10.1053/j.semtcvs.2017.05.018.

8. Audisio, K.; Halbreiner, M.S.; Chadow, D.; et al. Radial artery or saphenous vein for coronary artery bypass grafting. *Trends Cardiovasc. Med.* **2022**, *32*, 479–484. https://doi.org/10.1016/j.tcm.2021.09.007.
9. Souza, D. A new no-touch preparation technique: *Technical notes. Scand. J. Thorac. Cardiovasc. Surg.* **1996**, *30*, 41–44. https://doi.org/10.3109/14017439609107239.
10. Souza, D.S.; Arbeus, M.; Botelho Pinheiro, B.; et al. The no-touch technique of harvesting the saphenous vein for coronary artery bypass grafting surgery. *Multimed Man Cardiothorac Surg.* **2009**, *2009*, mmcts-2008.
11. Dreifaldt, M.; Souza, D.S.; Loesch, A.; et al. The "no-touch" harvesting technique for vein grafts in coronary artery bypass surgery preserves an intact vasa vasorum. *J. Thorac. Cardiovasc. Surg.* **2011**, *141*, 145–150. https://doi.org/10.1016/j.jtcvs.2010.02.005.
12. Johansson, B.L.; Souza, D.S.; Bodin, L.; et al. No touch vein harvesting technique for CABG improves the long-term clinical outcome. *Scand. Cardiovasc. J.* **2009**, *43*, 63–68. https://doi.org/10.1080/14017430802140104.
13. Samano, N.; Geijer, H.; Liden, M.; et al. The no-touch saphenous vein for coronary artery bypass grafting maintains a patency, after 16 years, comparable to the left internal thoracic artery: A randomized trial. *J. Thorac. Cardiovasc. Surg.* **2015**, *150*, 880–888. https://doi.org/10.1016/j.jtcvs.2015.07.027.
14. Souza, D.S.; Bomfim, V.; Skoglund, H.; et al. High early patency of saphenous vein graft for coronary artery bypass harvested with surrounding tissue. *Ann. Thorac. Surg.* **2001**, *71*, 797–800. https://doi.org/10.1016/s0003-4975(00)02508-x.
15. Dreifaldt, M.; Mannion, J.D.; Geijer, H.; et al. The no-touch saphenous vein is an excellent alternative conduit to the radial artery 8 years after coronary artery bypass grafting: A randomized trial. *J. Thorac. Cardiovasc. Surg.* **2021**, *161*, 624–630. https://doi.org/10.1016/j.jtcvs.2019.09.177.
16. Dashwood, M.R.; Loesch, A. Arterial versus venous conduits in coronary artery bypass surgery: Comparing apples with oranges. *J. Thorac. Cardiovasc. Surg.* **2019**, *157*, e253–e254. https://doi.org/10.1016/j.jtcvs.2018.11.047.
17. Gaudino, M.; Benedetto, U.; Fremes, S.; et al. Radial-Artery or Saphenous-Vein Grafts in Coronary-Artery Bypass Surgery. *N. Engl. J. Med*. **2018**, *378*, 20.
18. Kopjar, T.; Dashwood, M.R.; Dreifaldt, M.; et al. No-touch saphenous vein as an important conduit of choice in coronary bypass surgery. *J. Thorac. Dis.* **2018**, *10*, S3292–S3296. https://doi.org/10.21037/jtd.2018.08.
19. Dell'Aquila, M.; Prapas, S.; Falco, G.; et al. Saphenous Vein Graft and Nitric Oxide: Strategies to Prevent Graft Failure and Enhance Patency in Coronary Artery Bypass Grafting. *Front. Cardiovasc. Med*. **2026***, 12*, 1745260. https://doi.org/10.3389/fcvm.2025.1745260.
20. Sandner, S.; Hirofuji, A.; Mantaj, P.; et al. Outcomes of No-Touch Vs Conventionally Harvested Saphenous Veins for Coronary Artery Bypass Surgery: A Meta-Analysis of Randomized Trials. *Eur. J. Cardio-Thorac. Surg.* **2025**, *67*, ezaf297. https://doi.org/10.1093/ejcts/ezaf297.
21. Ali, M.A.; Alam, U.; Khattak, F.; et al. Comparative efficacy and safety of no-touch versus conventional vein harvesting techniques in coronary artery bypass grafting: A systematic review and meta-analysis. *Open Heart* **2025**, *12*, e003391. https://doi.org/10.1136/openhrt-2025-003391.
22. Chen, X.J.; Zhang, J.; Li, W.D.; et al. Interpretation on Expert systematic review on the choice of conduits for coronary artery bypass grafting endorsed by the European Association for Cardio-Thoracic Surgery and the Society of Thoracic Surgeons in 2023. *Chin. J. Surg.* **2023**, *62*, 45–49. https://doi.org/10.3760/cma.j.cn112139-20230917-00126
23. Gaudino, M.; Hameed, I.; Robinson, N.B.; et al. Angiographic Patency of Coronary Artery Bypass Conduits: A Network Meta-Analysis of Randomized Trials. *J. Am. Heart Assoc.* **2021**, *10*, e019206. https://doi.org/10.1161/JAHA.120.019206.
24. Liu, C.; Zhang, M.X.; Guan, X.L.; et al. Techniques for harvesting the saphenous vein in coronary artery bypass grafting: A network systematic review and meta-analysis. *Open Heart* **2025**, *12*, e003728. https://doi.org/10.1136/openhrt-2025-003728.
25. Yokoyama, Y.; Takagi, H.; Kuno, T. Graft Patency of a Second Conduit for Coronary Artery Bypass Surgery: A Network Meta-Analysis of Randomized Controlled Trials. *Semin. Thorac. Cardiovasc. Surg.* **2022**, *34*, 102–109. https://doi.org/10.1053/j.semtcvs.2021.02.002.
26. Lumsden, A.B.; Eaves, F.F.; Ofenloch, J.C.; et al. Subcutaneous, video-assisted saphenous vein harvest: Report of the first 30 cases. *Cardiovasc. Surg.* **1996**, *4*, 771–776.
27. Dacey, L.J.; Braxton, J.H.; Kramer, R.S.; et al. Long-term outcomes of endoscopic vein harvesting after coronary artery bypass grafting. *Circulation* **2011**, *123*, 147–153.
28. Kopjar, T.; Dashwood, M.R. Endoscopic Versus "No-Touch" Saphenous Vein Harvesting for Coronary Artery Bypass Grafting: A Trade-Off between Wound Healing and Graft Patency. *Angiology* **2016**, *67*, 121–132. https://doi.org/10.1177/0003319715584126.
29. Kopjar, T.; Dashwood, M.R. Towards Endoscopic No-Touch Saphenous Vein Graft Harvesting in Coronary Bypass Surgery. *Braz. J. Cardiovasc. Surg.* **2022**, *37*, 57–65. https://doi.org/10.21470/1678-9741-2022-0144.
30. Kiani, S.; Poston, R. Is endoscopic harvesting bad for saphenous vein graft patency in coronary surgery? *Curr. Opin. Cardiol.* **2011**, *26*, 518–522.

31. Nowicki, M.; Buczkowski, P.; Miskowiak, B.; et al. Immunocytochemical study on endothelial integrity of saphenous vein grafts harvested by minimally invasive surgery with the use of vascular mayo stripers. A randomized controlled trial. *Eur. J. Vasc. Endovasc. Surg.* **2004**, *27*, 244–250.
32. Kopjar, T.; Ivankovic, S.; Lima, M.L.; et al. Endoscopic or No-Touch Vein Harvesting for CABG: What is Best for the Patient? *Braz. J. Cardiovasc. Surg.* **2016**, *31*, 461–464. https://doi.org/10.5935/1678-9741.20160091.
33. Hayashi, I.; Kashima, I.; Yoshikawa, E. Use of the No-Touch Saphenous Vein Harvesting Technique via Small Incisions. *Innovations* **2020**, *15*, 81–84. https://doi.org/10.1177/1556984519886549.
34. Katayama, Y.; Miho, T.; Nogami, E.; et al. Endoscopic Pedicle Saphenous Vein Graft Harvesting. *Ann. Thorac. Cardiovasc. Surg.* **2024**, *30*, 23–00054. https://doi.org/10.5761/atcs.nm.23-00054.
35. Yoshino, K.; Abe, K.; Suzuki, K.; et al. A Novel Technique of Endoscopic Vein Harvesting with Preserved Perivascular Tissue. *Innovations* **2020**, *15*, 475–477. https://doi.org/10.1177/1556984520948139.
36. Yoshino, K.; Kikuchi, K.; Tamaki, R.; et al. A Novel Technique of No-Touch Endoscopic Saphenous Vein Harvesting. *Innovations* **2023**, *18*, 589–591. https://doi.org/10.1177/15569845231210299.
37. Kim, J.; Sohn, S.H.; Choi, J.S.; et al. Graft Patency of No-Touch Saphenous Veins Used as Aortocoronary Bypass Grafts. *J. Chest Surg.* **2023**, *56*, 313–321. https://doi.org/10.5090/jcs.23.027.
38. Milutinović, A.; Klajević, J.; Živković, I.; et al. Early Experiences of Serbian Surgeons Using No-Touch Technique for Vein Conduits in CABG Patients: A Follow-Up Study with Multi-Slice CT Angiography. *Medicina* **2024**, *60*, 1427. https://doi.org/10.3390/medicina60091427.
39. Tian, M.; Wang, X.; Sun, H.; et al. No-Touch Versus Conventional Vein Harvesting Techniques at 12 Months After Coronary Artery Bypass Grafting Surgery: Multicenter Randomized, Controlled Trial. *Circulation* **2021**, *144*, 1120–1129. https://doi.org/10.1161/CIRCULATIONAHA.121.055525.
40. Tian, M.; Wang, X.; Feng, W.; et al. No-touch versus conventional vein in coronary artery bypass grafting: Three year follow-up of multicentre randomised PATENCY trial. *BMJ* **2025**, *389*, e082883. https://doi.org/10.1136/bmj-2024-082883.
41. Angelini, G.D.; Johnson, T.; Culliford, L.; et al. Comparison of alternate preparative techniques on wall thickness in coronary artery bypass grafts: The HArVeST randomized controlled trial. *J. Card. Surg.* **2021**, *36*, 1985–1995. https://doi.org/10.1111/jocs.15477.
42. Deb, S.; Singh, S.K.; de Souza, D.; et al. SUPERIOR SVG: No touch saphenous harvesting to improve patency following coronary bypass grafting (a multi-centre randomized control trial, NCT01047449). *J. Cardiothorac. Surg.* **2019**, *14*, 85. https://doi.org/10.1186/s13019-019-0887-x.
43. Ragnarsson, S.; Janiec, M.; Modrau, I.S.; et al. No-touch saphenous vein grafts in coronary artery surgery (SWEDEGRAFT): Rationale and design of a multicenter, prospective, registry-based randomized clinical trial. *Am. Heart J.* **2020**, *224*, 17–24. https://doi.org/10.1016/j.ahj.2020.03.009.
44. Thelin, S.; Modrau, I.S.; Duvernoy, O.; et al. No-touch vein grafts in coronary artery bypass surgery: A registry-based randomized clinical trial. *Eur. Heart J.* **2025**, *46*, 1720–1729. https://doi.org/10.1093/eurheartj/ehaf018.
45. Weiss, M.G.; Nielsen, P.H.; James, S.; et al. Clinical Outcomes After Surgical Revascularization Using No-Touch Versus Conventional Saphenous Vein Grafts: Mid-Term Follow-Up of Propensity Score Matched Cohorts. *Semin. Thorac. Cardiovasc. Surg.* **2023**, *35*, 228–236. https://doi.org/10.1053/j.semtcvs.2021.12.002.
46. Gaudino, M.; Sandner, S. The SWEDEGRAFT trial: When absence of evidence is not evidence of absence. *Eur. Heart J.* **2025**, *46*, 1730–1732. https://doi.org/10.1093/eurheartj/ehaf043.
47. Dashwood, M.R.; Celik, Z.; Topal, G. Reducing vasospasm of vein and arterial conduits used in coronary artery bypass surgery: Are solutions the solution or is preserved perivascular fat the answer? *Front. Physiol.* **2025**, *16*, 1539102. https://doi.org/10.3389/fphys.2025.1539102.
48. He, G.-W.; Taggart, D.P. Spasm in Arterial Grafts in Coronary Artery Bypass Grafting Surgery. *Ann. Thorac. Surg.* **2016**, *101*, 1222–1229. https://doi.org/10.1016/j.athoracsur.2015.09.071.
49. Zheng, D.; Zhang, L.; Yang, M.D.; et al. Use of Radial Artery Grafts for Coronary Artery Bypass Grafting. *Heart Surg. Forum* **2022**, *25*, E385–E390. https://doi.org/10.1532/hsf.4697.
50. Rueda, F.; Souza, D.; Lima, R.D.C.; et al. Novel no-touch technique of harvesting the saphenous vein for coronary artery bypass grafting. *Arq. Bras. Cardiol.* **2008**, *90*, 356–362. https://doi.org/10.1590/s0066-782x2008000600002.
51. Favaloro, R.G. Saphenous vein autograft replacement of severe segmental coronary artery occlusion: Operative technique. *Ann. Thorac. Surg.* **1968**, *5*, 334–339. https://doi.org/10.1016/s0003-4975(10)66351-5.
52. Favaloro, R.G. Saphenous vein graft in the surgical treatment of coronary artery disease. Operative technique. *J. Thorac. Cardiovasc. Surg.* **1969**, *58*, 178–185.
53. Dashwood, M.R. No-Touch Saphenous Vein—Vascular Damage and the London Connection. *Braz. J. Cardiovasc. Surg.* **2022**, *37*, 1–6. https://doi.org/10.21470/1678-9741-2022-0024.
54. Gomes, W.J.; Kim, K.B.; Pinheiro, B.B.; et al. The No-touch Saphenous Vein Graft in Coronary Artery Bypass Surgery. Towards a New Standard? *Braz. J. Cardiovasc. Surg.* **2022**, *37*, I–II. https://doi.org/10.21470/1678-9741-2022-0955.

55. Pinheiro, B.B.; Dashwood, M.; Souza, D.S.R. The "No-touch" Harvesting Technique Revives the Position of the Saphenous Vein as an Important Conduit in CABG Surgery: 30-year Anniversary. *Braz. J. Cardiovasc. Surg.* **2021**, *36*, I–III. https://doi.org/10.21470/1678-9741-2021-0959.
56. Souza, D.S.; Johansson, B.; Bojö, L.; et al. Harvesting the saphenous vein with surrounding tissue for CABG provides long-term graft patency comparable to the left internal thoracic artery: Results of a randomized longitudinal trial. The *J. Thorac. Cardiovasc. Surg.* **2006**, *132*, 373–378. https://doi.org/10.1016/j.jtcvs.2006.04.002.
57. Dashwood, M.R.; Tsui, J.C. 'No-touch' saphenous vein harvesting improves graft performance in patients undergoing coronary artery bypass surgery: A journey from bedside to bench. *Vasc. Pharmacol.* **2013**, *58*, 240–250. https://doi.org/10.1016/j.vph.2012.07.008.
58. Dashwood, M.R.; Dooley, A.; Xu, S.-W.; et al. Does periadventitial fat-derived nitric oxide play a role in improved saphenous vein graft patency in patients undergoing coronary artery bypass surgery? *J. Vasc. Res.* **2007**, *44*, 175–181. https://doi.org/10.1159/000099833.
59. Fernandez-Alfonso, M.S.; Souza, D.S.; Dreifaldt, M.; et al. Commentary: Perivascular Fat and Improved Vein Graft Patency in Patients Undergoing Coronary Artery Bypass Surgery. *Curr. Vasc. Pharmacol.* **2016**, *14*, 308–312. https://doi.org/10.2174/1570161114666160513150444.
60. Fernández-Alfonso, M.S.; Gil-Ortega, M.; Aranguez, I.; et al. Role of PVAT in coronary atherosclerosis and vein graft patency: Friend or foe? *Br. J. Pharmacol.* **2017**, *174*, 3561–3572. https://doi.org/10.1111/bph.13734.
61. Dashwood, M.R.; Savage, K.; Tsui, J.C.; et al. Retaining perivascular tissue of human saphenous vein grafts protects against surgical and distension-induced damage and preserves endothelial nitric oxide synthase and nitric oxide synthase activity. *J. Thorac. Cardiovasc. Surg.* **2009**, *138*, 334–340. https://doi.org/10.1016/j.jtcvs.2008.11.060.
62. Angelini, G.D.; Passani, S.L.; Breckenridge, I.M.; et al. Nature and pressure dependence of damage induced by distension of human saphenous vein coronary artery bypass grafts. *Cardiovasc. Res.* **1987**, *21*, 902–907. https://doi.org/10.1093/cvr/21.12.902.
63. Roubos, N.; Rosenfeldt, F.L.; Richards, S.M.; et al. Improved preservation of saphenous vein grafts by the use of glyceryl trinitrate-verapamil solution during harvesting. *Circulation* **1995**, *92*, II31–II36. https://doi.org/10.1161/01.cir.92.9.31.
64. Tsui, J.C.; Souza, D.S.; Filbey, D.; et al. Preserved endothelial integrity and nitric oxide synthase in saphenous vein grafts harvested by a 'no-touch' technique. *Br. J. Surg.* **2001**, *88*, 1209–1215. https://doi.org/10.1046/j.0007-1323.2001.01855.x.
65. Tsui, J.C.; Souza, D.S.; Filbey, D.; et al. Localization of nitric oxide synthase in saphenous vein grafts harvested with a novel "no-touch" technique: Potential role of nitric oxide contribution to improved early graft patency rates. *J. Vasc. Surgery* **2002**, *35*, 356–362. https://doi.org/10.1067/mva.2002.121072.
66. Ahmed, S.R.; Johansson, B.L.; Karlsson, M.G.; et al. Human saphenous vein and coronary bypass surgery: Ultrastructural aspects of conventional and "no-touch" vein graft preparations. *Histol. Histopathol.* **2004**, *19*, 421–433. https://doi.org/10.14670/HH-19.421.
67. Saito, T.; Kurazumi, H.; Suzuki, R.; et al. Perivascular Adipose Tissue Is a Major Source of Nitric Oxide in Saphenous Vein Grafts Harvested via the No-Touch Technique. *J. Am. Heart Assoc.* **2022**, *11*, e020637. https://doi.org/10.1161/JAHA.120.020637.
68. Dashwood, M.R. Keeping NO in No-Touch Saphenous Vein Bypass Grafts. *Braz. J. Cardiovasc. Surg.* **2026**, *41*, e20250040. https://doi.org/10.21470/1678-9741-2025-0040.
69. Ham, A.W. *Histology*, 7th ed.; J. B. Lippincott & Co.: Philadelphia, PA, USA, 1974; pp. 577–581.
70. Jackson, D.R. Living homologous saphenous vein. A new graft conduit for use in arterial reconstruction. *Angiology* **1970**, *21*, I–II. https://doi.org/10.1177/000331977002100101.
71. Phillippi, J.A. On vasa vasorum: A history of advances in understanding the vessels of vessels. *Sci. Adv.* **2022**, *8*, eabl6364. https://doi.org/10.1126/sciadv.abl6364.
72. Dreifaldt, M.; Souza, D.; Bodin, L.; et al. The vasa vasorum and associated endothelial nitric oxide synthase is more important for saphenous vein than arterial bypass grafts. *Angiology* **2013**, *64*, 293–299. https://doi.org/10.1177/0003319712443729.
73. Loesch, A.; Dashwood, M. Vasa vasorum inside out/outside in communication: A potential role in the patency of saphenous vein coronary artery bypass grafts. *J. Cell Commun. Signal.* **2018**, *12*, 631–643. https://doi.org/10.1007/s12079-018-0483-1.
74. Souza, D.S.; Christofferson, R.H.; Bomfim, V.; et al. "No-touch" technique using saphenous vein harvested with its surrounding tissue for coronary artery bypass grafting maintains an intact endothelium. *Scand. Cardiovasc. J.* **1999**, *33*, 323–329. https://doi.org/10.1080/14017439950141362.
75. Vasilakis, V.; Dashwood, M.R.; Souza, D.S.R.; et al. Human saphenous vein and coronary bypass surgery: Scanning electron microscopy of conventional and 'no-touch' vein grafts. *Vasc. Dis. Prev.* **2004**, *1*, 133–139. https://doi.org/10.2174/1567270043405204.
76. Dashwood, M.R.; Anand, R.; Loesch, A.; et al. Hypothesis: A potential role for the vasa vasorum in the maintenance of vein graft patency. *Angiology* **2004**, *55*, 385–395. https://doi.org/10.1177/000331970405500405.

77. Barker, S.G.; Talbert, A.; Cottam, S.; et al. Arterial intimal hyperplasia after occlusion of the adventitial vasa vasorum in the pig. *Arterioscler. Thromb. J. Vasc. Biol.* **1993**, *13*, 70–77. https://doi.org/10.1161/01.atv.13.1.70.
78. Barker, S.G.; Tilling, L.C.; Miller, G.C.; et al. The adventitia and atherogenesis: Removal initiates intimal proliferation in the rabbit which regresses on generation of a 'neoadventitia'. *Atherosclerosis* **1994**, *105*, 131–144. https://doi.org/10.1016/0021-9150(94)90043-4.
79. Barger, A.C.; Beeuwkes, R., 3rd; Lainey, L.L.; et al. Hypothesis: Vasa vasorum and neovascularization of human coronary arteries. A possible role in the pathophysiology of atherosclerosis. *N. Engl. J. Med*. **1984**, *310*, 175–177. https://doi.org/10.1056/NEJM198401193100307.
80. Stingl, J.; Musil, V.; Pirk, J.; et al. Vasa vasorum of the failed aorto-coronary venous grafts. *Surg. Radiol. Anat.* **2018**, *40*, 769–778. https://doi.org/10.1007/s00276-018-2036-y.
81. Samano, N.; Loesch, A.; Dashwood, M.R. What is the impact of preserving the endothelium on saphenous vein graft performance? Comments on the 'NO' touch harvesting technique. *J. Cardiovasc. Surg.* **2021**, *16*, 21. https://doi.org/10.1186/s13019-021-01397-y.
82. Herbst, M.; Hölzenbein, T.J.; Minnich, B. Characterization of the Vasa Vasorum in the Human Great Saphenous Vein: A Scanning Electron Microscopy and 3D-Morphometry Study Using Vascular Corrosion Casts. *Microsc. Microanal.* **2014**, *20*, 1120–1133. https://doi.org/10.1017/S1431927614001287.
83. Lametschwandtner, A.; Minnich, B.; Kachlik, D.; et al. Three-dimensional arrangement of the vasa vasorum in explanted segments of the aged human great saphenous vein: Scanning electron microscopy and three-dimensional morphometry of vascular corrosion casts. *Anat. Rec. Part A Discov. Mol. Cell. Evol. Biol.* **2004**, *281*, 1372–1382. https://doi.org/10.1002/ar.a.20098.
84. Kachlik, D.; Baca, V.; Stingl, J.; et al. Architectonic arrangement of the vasa vasorum of the human great saphenous vein. *J. Vasc. Res.* **2007**, *44*, 157–166. https://doi.org/10.1159/000099142.
85. Kachlík, D.; Lametschwandtner, A.; Rejmontová, J.; et al. Vasa vasorum of the human great saphenous vein. *Surg. Radiol. Anat.* **2003**, *24*, 377–381. https://doi.org/10.1007/s00276-002-0067-9.
86. Brock, W.H. Vasa vasorum of veins in dogs and man. *Angiology* **1977**, *28*, 351–360. https://doi.org/10.1177/000331977702800507.
87. Crotty, T.P. The role of vasa vasorum in atherosclerosis. *Med. Hypotheses* **1989**, *28*, 233–243.
88. Crotty, T.P. The path of retrograde flow from thelumen of the lateral saphenous vein of the dog. *Microvasc. Res.* **1989**, *37*, 119–122.
89. Ohta, O.; Kusaba, A. Development of vasa vasorum in the arterially implanted autovein bypass graft and its anastomosis in the dog. *Int. Angiol. J. Int. Union Angiol.* **1997**, *16*, 197–203.
90. Loesch, A. Vasa Vasorum in Saphenous Vein for CABG: A Review of Morphological Characteristics. *Braz. J. Cardiovasc. Surg.* **2023**, *38*, e20230045. https://doi.org/10.21470/1678-9741-2023-0045.
91. Loesch, A.; Dashwood, M.R. A Brief Comment on Vasa Vasorum of Human Saphenous Vein: Relevance for Coronary Artery Bypass Surgery. *Braz. J. Cardiovasc. Surg.* **2021**, *36*, 106–111. https://doi.org/10.21470/1678-9741-2020-0066.
92. Loesch, A. Great Saphenous Vein Lumen: Intimal Openings. *Braz. J. Cardiovasc. Surg.* **2026**, *41*, e20240382. https://doi.org/10.21470/1678-9741-2024-0382.
93. Părău, A.F.; Manzur, A.R.; Mihaicuta, S.; et al. The Controversy of Using Insufficient Great Saphenous Veins in Coronary Artery Bypass Grafting: A Comparative Morphological Analysis of Healthy and Insufficient Veins Related to the Possibility of Using Them as a Graft. *Biomedicines* **2024**, *12*, 476. https://doi.org/10.3390/biomedicines12030476.
94. Bazylev, V.V.; Tungusov, D.S.; Mikulyak, A.I.; et al. Morphological assessment of autovenous conduits in the composition of perivascular adipose tissue. *Angiol. Vasc. Surg.* **2022**, *28*, 104–109. https://doi.org/10.33029/1027-6661-2022-28-1-104-109.
95. Frolov, A.V.; Terekhov, A.A.; Bogdanov, L.A.; et al. Comparative study of vasa vasorum and neointima in conduits for coronary artery bypass grafting. *Angiol. Vasc. Surg.* **2021**, *27*, 121–126. https://doi.org/10.33529/ANGIO2021218.
96. Nishimiya, K.; Matsumoto, Y.; Takahashi, J.; et al. Association of adventitial vasa vasorum and inflammation with coronary hyperconstriction after drug-eluting stent implantation in pigs in vivo. *Circ. J.* **2015**, *79*, 1787.
97. Nishimiya, K.; Matsumoto, Y.; Takahashi, J.; et al. Enhanced Adventitial Vasa Vasorum Formation in Patients with Vasospastic Angina: Assessment with OFDI. *J. Am. Coll. Cardiol.* **2016**, *67*, 598–600. https://doi.org/10.1016/j.jacc.2015.11.031.
98. Ohyama, K.; Matsumoto, Y.; Takanami, K.; et al. Coronary Adventitial and Perivascular Adipose Tissue Inflammation in Patients with Vasospastic Angina. *J. Am. Coll. Cardiol.* **2018**, *71*, 414–425. https://doi.org/10.1016/j.jacc.2017.11.046.
99. Mikami, T.; Furuhashi, M.; Numaguchi, R.; et al. Comparison of Phenotypes in Subcutaneous Fat and Perivascular Adipose Tissue Surrounding the Saphenous Vein in Coronary Artery Bypass Grafting. *Circ. J.* **2023**, *87*, 791–798. https://doi.org/10.1253/circj.CJ-22-0740.

100. Mikami, T.; Dashwood, M.R.; Kawaharada, N.; et al. An Obligatory Role of Perivascular Adipose Tissue in Improved Saphenous Vein Graft Patency in Coronary Artery Bypass Grafting. *Circ. J.* **2024**, *88*, 845–852. https://doi.org/10.1253/circj.CJ-23-0581.
101. Ferrari, G.; Loayza, R.; Azari, A.; et al. Superior long-term patency of no-touch vein graft compared to conventional vein grafts in over 1500 consecutive patients. *J. Cardiothorac. Surg.* **2024**, *19*, 570. https://doi.org/10.1186/s13019-024-03057-3.
102. Sigdel, S.; Udoh, G.; Albalawy, R.; et al. Perivascular Adipose Tissue and Perivascular Adipose Tissue-Derived Extracellular Vesicles: New Insights in Vascular Disease. *Cells* **2024**, *13*, 1309. https://doi.org/10.3390/cells13161309.
103. Rami, A.Z.A.; Hamid, A.A.; Anuar, N.N.M.; et al. Exploring the Relationship of Perivascular Adipose Tissue Inflammation and the Development of Vascular Pathologies. *Mediat. Inflamm.* **2022**, *2022*, 2734321. https://doi.org/10.1155/2022/2734321.
104. Zhang, Y.Y.; Shi, Y.N.; Zhu, N.; et al. PVAT targets VSMCs to regulate vascular remodelling: Angel or demon. *J. Drug Target.* **2021**, *29*, 467–475. https://doi.org/10.1080/1061186X.2020.1859515.
105. Miao, C.Y.; Li, Z.Y. The role of perivascular adipose tissue in vascular smooth muscle cell growth. *Br. J. Pharmacol.* **2012**, *165*, 643–658. https://doi.org/10.1111/j.1476-5381.2011.01404.x.
106. Ford, C.A.; Mong, K.; Tabrizchi, R. Influence of tangential stress on mechanical responses to vasoactive agents in human saphenous vein with and without perivascular adipose tissue. *Can. J. Cardiol.* **2006**, *22*, 1209–1216. https://doi.org/10.1016/S0828-282X(06)70961-8.
107. Ozen, G.; Topal, G.; Gomez, I.; et al. Control of human vascular tone by prostanoids derived from perivascular adipose tissue. *Prostaglandins Other Lipid Mediat.* **2013**, *107*, 13–17. https://doi.org/10.1016/j.prostaglandins.2013.06.002.
108. Foudi, N.; Kotelevets, L.; Gomez, I.; et al. Differential reactivity of human mammary artery and saphenous vein to prostaglandin E_2: Implication for cardiovascular grafts. *Br. J. Pharmacol.* **2011**, *163*, 826–834. https://doi.org/10.1111/j.1476-5381.2011.01264.x.
109. Dashwood, M.; Souza, D.; Topal, G. Advancing coronary artery bypass grafting: The fasudil-nitroglycerin cocktail as a potential antispastic solution. *J. Thorac. Dis.* **2024**, *16*, 2694. https://doi.org/10.21037/jtd-23-1953.
110. Hou, H.T.; Wang, Z.Q.; Wang, J.; et al. Antispastic Effect of Fasudil and Cocktail of Fasudil and Nitroglycerin in Internal Thoracic Artery. *Ann. Thorac. Surg.* **2023**, *115*, 1152–1161. https://doi.org/10.1016/j.athoracsur.2021.11.079.
111. Chanda, J.; Brichkov, I.; Canver, C.C. Prevention of radial artery graft vasospasm after coronary bypass. *Ann. Thorac. Surg.* **2000**, *70*, 2070–2074. https://doi.org/10.1016/s0003-4975(00)02001-4.
112. He, G.W.; Yang, C.Q. Use of verapamil and nitroglycerin solution in preparation of radial artery for coronary grafting. *Ann. Thorac. Surg.* **1996**, *61*, 610–614. https://doi.org/10.1016/0003-4975(95)00920-5.
113. He, G.W. Verapamil plus nitroglycerin solution maximally preserves endothelial function of the radial artery: Comparison with papaverine solution. *J. Thorac. Cardiovasc. Surg.* **1998**, *115*, 1321–1327. https://doi.org/10.1016/S0022-5223(98)70215-6.
114. He, G.W.; Rosenfeldt, F.L.; Buxton, B.F.; et al. Reactivity of human isolated internal mammary artery to constrictor and dilator agents. Implications for treatment of internal mammary artery spasm. *Circulation* **1989**, *80*, I141–I150.
115. He, G.W.; Yang, C.Q. Comparative study on calcium channel antagonists in the human radial artery: Clinical implications. *J. Thorac. Cardiovasc. Surg.* **2000**, *119*, 94–100. https://doi.org/10.1016/s0022-5223(00)70222-4.
116. Acar, C.; Jebara, V.A.; Portoghese, M.; et al. Revival of the radial artery for coronary artery bypass grafting. *Ann. Thorac. Surg.* **1992**, *54*, 652–660. https://doi.org/10.1016/0003-4975(92)91007-v.
117. Jett, G.K.; Arcici, J.M., Jr.; Hatcher, C.R., Jr.; et al. Vasodilator drug effects on internal mammary artery and saphenous vein grafts. *J. Am. Coll. Cardiol.* **1988**, *11*, 1317–1324. https://doi.org/10.1016/0735-1097(88)90298-7.
118. He, G.W.; Fan, L.; Furnary, A.; et al. A new antispastic solution for arterial grafting: Nicardipine and nitroglycerin cocktail in preparation of internal thoracic and radial arteries for coronary surgery. *J. Thorac. Cardiovasc. Surg.* **2008**, *136*, 673–680.e2. https://doi.org/10.1016/j.jtcvs.2007.12.019.
119. Pillai, V.V.; Sreekanthan, R.; Karunakaran, J. Quantitative estimation of LIMA blood flow between extraluminal papavarine vs. extraluminal papavarine plus intraluminal vasodilator cocktail in CABG patients. *Ann. Card. Anaesth.* **2020**, *23*, 414–418. https://doi.org/10.4103/aca.ACA_164_19.
120. Kamat, B.R.; Galli, S.J.; Barger, A.C.; et al. Neovascularization and coronary atherosclerotic plaque: Cinematographic localization and quantitative histologic analysis. *Hum. Pathol.* **1987**, *18*, 1036–1042. https://doi.org/10.1016/s0046-8177(87)80220-4.
121. Johansson, B.L.; Souza, D.S.R.; Bodin, L.; et al. Slower progression of atherosclerosis in vein grafts harvested with 'no touch' technique compared with conventional harvesting technique in coronary artery bypass grafting: An angiographic and intravascular ultrasound study. *Eur. J. Cardio-Thorac. Surg.* **2010**, *38*, 414–419. https://doi.org/10.1016/j.ejcts.2010.02.007.
122. Samano, N.; Geijer, H.; Bodin, L.; et al. The no-touch saphenous vein graft in elderly coronary bypass patients with multiple comorbidities is a promising conduit to substitute the left internal thoracic artery. *J. Thorac. Cardiovasc. Surg.* **2017**, *154*, 457–466.e3. https://doi.org/10.1016/j.jtcvs.2017.03.048.

123. Kurazumi, H.; Suzuki, R.; Nawata, R.; et al. Impact of the no-touch harvesting technique on the vessel diameter of saphenous vein grafts for coronary artery bypass grafting. *JTCVS Tech.* **2022**, *15*, 87–94. https://doi.org/10.1016/j.xjtc.2022.07.005.
124. Sugaya, A.; Uesugi, S.; Doi, M.; et al. Vasa vasorum of the no-touch saphenous vein graft observed using frequency-domain optical coherence tomography. *Interdiscip. CardioVascular Thorac. Surg.* **2024**, *39*, ivae167. https://doi.org/10.1093/icvts/ivae167.