Letters to the Editor

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Surgical damage of the saphenous vein and graft patency

To the Editor:

It is 37 years since Favaro's article¹ introducing the saphenous vein as a bypass graft appeared in the *Journal of Thoracic and Cardiovascular Surgery*. A novel "no-touch" technique of preparing this vessel has been described,² and the long-term follow-up study recently reported in the *Journal* shows that, with this technique, the patency rate of the saphenous vein may be improved to a rate equal to that of the internal thoracic artery.³ Although the saphenous vein is the most commonly used conduit for coronary artery bypass grafting, its patency rate is poor, with 15% to 30% of grafts occluding in the first year and over 50% within 10 years.⁴ Along with many others, as basic research scientists, we are surprised by the degree of damage inflicted on the saphenous vein during conventional bypass surgery. It seems, when harvesting this vessel as a graft, many surgeons are in effect preparing a "tube" or "pipe" (common definitions of conduit) with no regard to its many vital vascular structures. The contribution of tissue damage to graft occlusion has been recognized for some time, and various attempts at reducing surgical trauma have been introduced, ranging from the use of a system of "bent rod of rubber and sutures"⁵ to the use of local vasodilators⁶ and the "Souza no-touch technique."²

In conventional methods of harvesting, the vein is isolated from its surrounding connective tissue, trauma that causes a high proportion of vessels to go into spasm. Although vasodilators may be used to overcome spasm, high-pressure intraluminal distention is mostly used, resulting in damage to the luminal endothelium (Figure 1). This influences endothelium-derived factors, such as nitric oxide, affecting vessel tone, cell proliferation, and thrombus formation.⁶ In addition, the width of the vein wall is markedly reduced (Figure 1) and the vas-

cular smooth muscle cells are distorted. Last, the outer vessel wall layer, the adventitia, is generally removed or damaged during harvesting of the vein. This may play an important part in the subsequent processes involved in graft occlusion because the adventitia does not merely provide structural support for the media, but contains the vasa vasorum, a microvascular network responsible for the exchange of gases and supply of nutrients to the vein wall. Clearly, damage to segments of the saphenous vein during harvesting as a graft is inevitable. Although this isolated segment of vein is "disconnected" from its vascular bed, the supply of blood to medial and adventitial structures will be maintained by the retrograde blood flow observed through the vasa vasorum in "no-touch" vein grafts at implantation.³ Furthermore, the connective tissue surrounding the vein when the "Souza no-touch" technique is used may act as a buffer against coronary arterial hemodynamics, "protecting" the graft in very much the same way that has been suggested with the use of experimental external stents.⁴

We propose that damage to the saphenous vein during conventional coronary artery bypass grafting is a major contribution to graft failure and suggest that improved patency may be achieved simply by reducing vascular damage by use of less traumatic harvesting techniques.

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Figure 1. Vascular damage to saphenous vein harvested for coronary artery bypass grafting. Representative micrographs are shown of conventional (*CONV*) and no-touch (*NT*) preparations of the same vein from a patient undergoing bypass grafting. The *top panels* show the general structural differences where the adventitia is removed from the conventional vein segments and, as a result of saline distention, the intimal folds seen in the “no-touch” vein are absent and the vessel wall thickness is reduced (elastic-van Gieson staining). The *middle panels* indicate a continuous lining of endothelial cells (CD31 immunostaining) in a “no-touch” vein segment, whereas there are regions of endothelial denudation in the conventional sample (*arrow*). The *bottom panels* show ultrastructural changes to the vasa vasorum at the adventitial/medial border where a microvessel is contracted and there is clumping of red blood cells in the conventional sample, whereas in “no-touch” veins the lumen is open. Original magnifications: *top panels* $\times 20$, *middle panels* $\times 100$, *bottom panels* $\times 3000$. *L*, Lumen.

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Restaging patients with N2 (stage IIIa) non-small cell lung cancer after neoadjuvant chemoradiotherapy: A closer look at redo mediastinoscopy *To the Editor:*

On the basis of the data obtained by the analysis of 93 locally advanced cases of non-small cell lung cancer clinically restaged after induction therapy, Cerfolio and coworkers¹ conclude that when repeat positron emission tomography (PET)/computed tomography (CT) is adopted either in the staging or restaging process, the percentage decrease in maximum standardized uptake value (maxSUV) of the primary tumor and involved lymph nodes is predictive of pathology, but pathologic assessment is still required because persistently high maxSUV “does not equate to residual cancer.” We commend the authors for their valuable study. Along the line of discussion, Cerfolio and coworkers state that “repeat mediastinoscopy often is inaccurate and potentially dangerous, especially after chest irradiation” and that endoscopic ultrasonography-guided fine-needle aspiration (EUS-FNA) biopsy, despite being more precise and accurate, is “available only in few centers.” We would like to amicably address the authors on this point on the basis of our own personal experience and confidence with redo mediastinoscopy. Pathologic reassessment of the mediastinum is strongly advisable in the setting of induction therapy for locally advanced non-small cell lung cancer because persistent N2 disease heralds a poor prognosis.

Shortly after the introduction of mediastinoscopy, redo procedures were considered to be technically impossible be-