
To the Editor:

We read with interest the article by Webb et al,1 demonstrating that, 5 years after coronary surgery, radial artery (RA) grafts have a preserved flow-mediated vasodilatation, whereas saphenous vein (SV) grafts have not. We agree with the authors that these results may provide insight into the more favorable patency of the RA graft over the SV graft, because the RA grafts preserved their ability to autoregulate their diameter in response to changes in myocardial flow physiology. However, some considerations about the SV graft should be highlighted.

First, although the results represent a situation 5 years after the surgery, it is important to know the baseline situation of these patients. The authors have already shown a reduced dilatation to acetylcholine but preserved dilator response to nitrate in SV grafts 3 months after surgery.2 According to these published results, the authors stated that they have now found a loss of the dilator response to both acetylcholine and nitrate. However, it is intriguing and difficult to argue a worsening in endothelial-independent vasomotion of SV grafts, especially when different patients have been evaluated in 2 different studies without a paired comparison. Moreover, at 3 months with nitrates infusion, there was not a significant increase in the mean diameter of SV graft (only 3.4%) compared with baseline; in addition, the authors had already found no vasodilatation of SV grafts in response to adenosine infusion, but a significant increase in blood flow.2 Thus, the knowledge of the preexisting vasomotor response of those grafts is relevant to determine whether endothelial dysfunction or inability of SV grafts to autoregulate the flow is present at follow-up or was already present at baseline.

Second, the baseline anatomic differences in amount and sensitivity of smooth muscle cells between a RA and a SV graft should be accounted for the behavior of the SV graft as an inert conduit of blood flow. The data from literature about the ability of the SV graft to dilate in response to acetylcholine and nitrates in an in vivo model are varied. Werner et al3 have already shown the functional superiority of the internal mammary artery over SV grafts; SV grafts showed a less pronounced cholinergic vasoconstriction, and only half of them showed a significant vasodilatation to nitrates. Hanet et al4 have also demonstrated that SV grafts do not dilate in response to nitrates. The difference in vasomotor response and, in particular, in vasodilatation response to nitrates between a RA and a SV graft, derived by these contrasting results, could reflect their heterogeneity in the sensitivity of vascular smooth muscle cells to nitrate or some differences in the baseline tone.5

With these considerations, we think that, although the results obtained by the authors are noteworthy and represent the longest follow-up available in the literature, they are not new and should be interpreted carefully, considering that, probably from the beginning, SV grafts are already poor responders to vasodilators and act as inert conduits of blood flow, unable to autoregulate their diameter in response to changes in myocardial flow physiology.

Disclosures

None.

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References

Letter by Brugaletta et al Regarding Article, "Vascular Reactivity and Flow Characteristics of Radial Artery and Long Saphenous Vein Coronary Bypass Grafts: A 5-Year Follow-Up"
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