protein and possesses NOS activity [2]. This finding is of particular relevance to the improved early- and long-term patency rates shown in patients receiving saphenous vein grafts prepared using a 'no-touch' method of harvesting [3] where the vein is removed complete with its cushion of surrounding tissue, much of which is fat. Indeed, a long-term prospective follow-up study (mean time 8.5 years) showed the patency of 'no-touch' vein grafts comparable to the ITA [3].

While the PVT is likely to play an important role in the improved performance of 'no-touch' saphenous vein grafts its contribution to the superior patency of the ITA in CABG patients may be questionable. As mentioned by Malinowski et al., the ITA is traditionally harvested as a pedicle, complete with surrounding tissue. In some centres, ITA skeletonisation is performed that provides a longer graft with superior flow and reduced postoperative sternal wound infection. However, a review of the ITA graft points out that data from long-term angiographic studies comparing pedicled with skeletonised ITA is not currently available. Furthermore, in this review, Del Campo [4] suggests that skeletonisation of the ITA might adversely affect the long-term patency of this conduit since in this preparation the vasa vasorum, innervation and lymphatic drainage of the vessel might be compromised. Again, the preservation of PVT in 'no-touch' saphenous vein grafts is likely to have a protective role since the capillary network contained within the surrounding cushion of fat and the underlying vasa vasorum are not damaged. The identification of eNOS associated with perivascular fat and endothelial cells of the capillaries and vasa vasorum accompanied by the finding that it possesses NOS activity indicates its potential to release NO when used as a bypass conduit. We suggest that the PVT of 'no-touch'-harvested saphenous vein plays an important role in its superior patency rate comparable to the ITA and agree with Malinowski et al. that skeletonisation of vessels used for CABG should be re-evaluated.

References


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doi:10.1016/j.ejcts.2008.03.021

Reply to the Letter to the Editor

Reply to Dashwood et al.

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Received 18 March 2008; accepted 19 March 2008

Keywords: Internal thoracic artery; ADRF; CABG; Perivascular tissue; Nitric oxide; Saphenous vein

We thank Dashwood et al. for their interest and comments regarding our manuscript [1]. We used to be enthusiastic regarding ITA skeletonization but with growing awareness of the active role of perivascular tissue we are becoming less and less so [2]. Our study showed that the factor responsible for anticontractile properties of perivascular tissue (PVT) in human internal thoracic artery (ITA) acts independent of NO and PGI2 [3]. It suggests that PVT releases agents, different to these well known relaxing factors that clearly affect vascular reactivity, adventitia/adipocyte derived relaxing factor (ADRF).

We appreciate Dashwood et al.’s findings on the importance of perivascular tissue of saphenous vein [4,5]. Their papers clearly show that preserving perivascular fat results in improved long term patency rates of SV grafts. Dashwood et al. proved that PVT of SV possesses strong NOS activity and argue it might contribute to the improved patency of SV harvested as a pedicle. Still, we do not know if this high NOS activity is found in PVT, nor whether SV PVT releases ADRF. Likewise, it remains to be shown if ADRF, similarly to NO, has the ability to affect patency rates of the vessels.

Meanwhile, we have analyzed the influence of internal thoracic artery’s PVT on the function of other vessels such as radial artery and saphenous veins and we failed to find any anticontractile effect. This may suggest that ADRF may be a vessel specific agent. (These data will be presented during the 57th ESCVS International Congress in Barcelona). It is crucial now to establish the nature and precise mechanisms of action of ADRF and check if this is truly a vessel specific factor which may affect clinical outcome of non-skeletonized grafts.

References

Sivelestat and its role in tissue reperfusion injury

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Received 7 January 2008; accepted 11 March 2008

Keywords: Sivelestat; Tissue reperfusion injury; Multiple organ failure

The recent article by Mori et al. is highly interesting [1]. Recent studies have shown that sivelestat decreases and prevents tissue reperfusion injury not just in the lung but in other organs also.

For instance, Kotake et al. have recently shown that sivelestat can effectively decrease the effects of ischemia reperfusion injury in the hepatointestinal region [2]. Similarly, it has been shown that sivelestat attenuates the effects of ischemia reperfusion injury in the bladder [3]. Kambe et al. have even reported that sivelestat decreases myocardial damage secondary to tissue reperfusion injury in the heart [4]. These beneficial effects were noted even if the drug was administered after the onset of the myocardial ischemia. In another recent study, Hoshi et al. compared the mortality in critically ill patients admitted in an intensive care unit who were treated with sivelestat to the mortality in those who did not receive sivelestat [5]. The mortality in critically ill patients who were treated with sivelestat was 6% compared to 33.3% in those who did not receive sivelestat therapy.

These studies clearly indicate that sivelestat may have a major role to play in the management of tissue reperfusion injury, especially in patients with multiple organ failure, in the near future. Further studies, especially in humans, are needed so as to fully avail these beneficial effects.

References


The authors of the original paper [1] were invited to reply to this Letter to the Editor but they did not respond.

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doi:10.1016/j.ejcts.2008.03.009