superoxide (generated by neutrophils) to produce peroxynitrite, a highly toxic free radical (4).

Raimondo Ascione, MD
Jamie Y. Jeremy, MSc, PhD, FRSH
G. D. Angelini, MD, MCH, FRCS
Bristol Heart Institute
Bristol Royal Infirmary
Bristol
BS2 8HW
United Kingdom

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REPLY

We thank Dr. Ascione et al. for their interest in our study (1), in which internal mammary arteries (IMA) and saphenous veins (SV) were obtained from patients undergoing coronary bypass surgery. Care was taken during harvesting of the blood vessels not to touch the inner surface of the blood vessels. The SV were harvested by using a no-touch technique, and the dilution procedure was avoided, whereas IMA were harvested as pedunculated graft. Each graft was sectioned carefully to minimize artifact induced by handling and cutting, particularly in the intimal surface. Presence of endothelium was evaluated by light microscopy in six nonadjacent sections of each graft, and endothelial cell confluence was observed over the entire examined surfaces. The concern that elevated concentrations of NO donors could affect directly neutrophil function is not justified for at least two reasons. First, after incubation with L-arginine and nisipride, the vessel segments were repeatedly washed and transferred with fresh Krebs-Henseleit solution immediately before the neutrophil adherence assay. Second and most important, previous studies have clearly demonstrated, using an experimental model similar to that used in our study, that exogenous NO donors do not directly affect the ability of neutrophil to adhere.

Niu et al. (2) demonstrated that incubation of neutrophils with sodium nitroprusside (1.0 mmol/L) for 30 min did not modify the amount of their adhesion to endothelium. In a similar fashion, Ma et al. (3) demonstrated that incubation of PMNs, rather than vessel segments, with L-arginine (3 mM) for 20 min, did not significantly decrease PMN adhesion to coronary artery segments subject to 20 min of reperfusion, indicating that the inhibitory effect of L-arginine on PMN adherence occurs on the endothelium.

Many studies in the literature clearly show that, other than technical failures, acute thrombosis and delayed intimal hyperplasia are the two processes most closely linked with vein graft failure.

However, caution should be recommended in extending the result of animal studies (4,5) to the humans. Actually, although the differences between the porcine data and human vein data might be attributed to the different time periods that the veins are arterialized (7 to 12 years for humans, much less for pigs), it appears equally likely that heterogeneity of endothelium-dependent responses between different species have a significant role because the relevance of porcine vein graft data to human bypass is uncertain. It may be hypothesized that the short period of time (four weeks) involved in the animal vein graft study cited by Dr. Ascione (4), does not allow for the return of endothelial function seen in human vein grafts. Moreover, the anatomic position of the vein graft in the arterial circulation affects the phasic composition of flow. Grafts in the peripheral arteries, like the model used by Dr. Angelini (4), receive flow primarily in systole, whereas graft in the left coronary circulation receive flow principally in diastole. The different flow patterns and shear stress may significantly influence the results. A recent clinical study (6) has in fact revealed that, when compared with human saphenous veins studied before implantation, venous coronary bypass grafts develop somewhat more pronounced endothelium-dependent vasodilation to acetylcholine after implantation, although the potency of these responses is still markedly less than in arterial grafts.

Finally, we would like to point out that, as stated in the introduction of the report, the main purpose of our study was to evaluate the relation between the pattern of neutrophil-endothelial adhesion in SV and IMA and the endothelial production of NO. No therapeutic solutions have been proposed. We therefore agree with Dr. Ascione and coworkers by suggesting prudence in administering NO donors for the prevention of graft failure.

Massimo Chello, MD
Pasquale Mastororoberto, MD
Via S. Giacomo dei Capri 29
80128 Napoli
Italy

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Corrections


Erenberg FG, Banerjee A. Systolic and Diastolic Properties in Children With Single Ventricles. Abstract—Pediatric Cardiology, Session 1061–145. J Am Coll Cardiol 1999;33:521A. This abstract was presented at the 48th Annual Scientific Session of the American College of Cardiology, New Orleans, Louisiana. The results section of the printed abstract, line 4, should have read as follows: “In the SV group an abrupt prolongation of $\tau$ occurred at a HR of 150 ± 12 bpm, the ‘critical heart rate’ (HR crit).”