would be helpful to further elucidate this point.

We must stress that we did not postulate that CPB was the sole factor in the inflammatory response. Indeed, we documented that surgical trauma leads to higher IL-6 levels in comparison with the use of CPB without cardiac surgery.

We are in complete agreement that there are a number of different factors affecting IL-6 levels.

R. Prondzinsky

K. Werdan

Klinik und Poliklinik für Innere Medizin III

Universitätsklinik der Martin-Luther-

Universität Halle-Wittenberg, E.-Grube-Sr. 40,

D-06097 Halle/Saale, Germany

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doi:10.1016/j.jtcvs.2005.06.021

Radial artery for coronary artery bypass grafting

To the Editor:

I would like to comment on a recent editorial by Mussa and colleagues¹ regarding radial artery grafts for coronary artery bypass grafting. In their review of vasospasm prophylaxis, they state that in addition to antispasmodic agents during harvesting, oral calcium-channel antagonists have been recommended for as long as 1 year after surgery to prevent delayed vasospasm. However, my coworkers and I² recently reported a study evaluating the effects of calcium-channel blockers in two randomized groups of comparable patients. We found no differences between patients who received diltiazem and those who did not, especially regarding the development of vascular spasm and angiographic patency 1 year after surgery. Similar results have been published by other authors.^{3,4}

This evidence is relevant when we consider widespread use of the radial artery for myocardial revascularization in patients with different types of coronary disease. Antispasmodic agents are known to have adverse consequences, for example, in patients with acute hemodynamic instability or with poor left ventricular function.

I agree with Mussa and colleagues¹ when they state that there is accumulating evidence that grafting the radial artery to coronary targets with moderate stenosis (<70%) results in reduced patency. Angiographic evidence from our study, with a reproducible and objective method, demonstrated that the degree of native coronary stenosis was a strong predictor of radial artery patency ($P = .00001$; odds ratio 1.08). When the degree of stenosis in the native coronary artery is 70% or more, the radial artery graft patency approaches that of the internal thoracic artery at 1 postoperative year.²

I postulate that there is sufficient evidence with which to recommend the use of radial artery grafts for myocardial revascularization in patients with significant coronary artery stenosis (<70%). Topical antispasmodic agents should be used only during harvesting the conduit. There is no need for intravenous or oral calcium-channel blockers, either intraoperatively or during the first year of follow-up.

The definitive place of the radial artery compared with other conduits for coronary grafting will have to await the outcome of ongoing randomized trials.

Sergio V. Moran, MD

Cardiovascular Surgery

Catholic University Hospital

Marcoleta 367

Santiago, Chile

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doi:10.1016/j.jtcvs.2005.04.023

Reply to the Editor:

We thank Dr Moran for his interest in our editorial concerning the use of radial ar-

teries (RA) as coronary bypass conduits.¹ His letter invites us to expand on two important points covered in our article:

1. The use of oral calcium channel antagonists as prophylaxis for delayed vasospasm
2. Use of RA conduits as grafts to coronary vessels with proximal stenoses in excess of 70% favours improved long term patency

Concerning the first point, we would agree that despite previous anecdotal recommendations there is no substantial evidence for the use of oral calcium channel antagonists to prevent delayed vasospasm of RA conduits. Certainly, Gaudino and associates² randomized 120 patients receiving RA grafts to treatment with oral calcium channel antagonist therapy or not, and showed no difference in ischemic symptoms, scintigraphic evidence of ischemia, or RA angiographic patency at 5 years. This is consistent with the data published by Moran and coworkers.³

Concerning the second point, Moran and colleagues elegantly demonstrated the improved patency of RA conduits when anastomosed to target vessels with high-grade (>70%) proximal stenoses. This finding was subsequently confirmed in a larger series with a longer interval to angiographic follow-up.⁴

Graft patency is influenced not only by the biology and quality of the conduit but also by physical factors such as luminal blood pressure and runoff, which govern luminal blood flow. The concept of competitive flow suggests that graft flow is influenced by native coronary flow. Royse and colleagues⁵ have reported that blood flow through composite arterial grafts (left internal thoracic artery-RA T-grafts) fell by 44% on reintroduction of native coronary flow. Shear stress resulting from flow activates endothelial nitric oxide synthase and results in the production of nitric oxide.⁶ Intuitively, grafted conduits should fare better in conditions of poor native coronary flow typified by high grade coronary stenoses, as increased conduit blood flow will contribute to improved nitric oxide production.

Shafi Mussa, MA, MRCS

Bikram P. Choudhary, MRCS

David P. Taggart, MD(Hons), PhD, FRCS

Department of Cardiothoracic Surgery

John Radcliffe Hospital

Oxford, United Kingdom