Letters to the Editor

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- Include no more than 500 words of text, three authors, and five references
- Type with double-spacing
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Evaluation of surgical trauma and cardiopulmonary bypass as factors in inflammatory status after cardiac surgery

To the Editor:

I read with interest Prondzinsky and colleagues' article[1] "Surgical Trauma Affects the Proinflammatory Status After Cardiac Surgery to a Higher Degree Than Cardiopulmonary Bypass." Because there was no control group, cardiopulmonary bypass was considered as the sole factor in development of generalized inflammatory response after open procedures. In the past decade, coronary revascularization without cardiopulmonary bypass has become an alternative to the conventional on-pump technique. Patients operated on with this technique thus could represent a control group. Several studies have shown no difference between on-pump and beating-heart surgery with respect to oxygen metabolism after surgery, pulmonary dysfunction, and other aspects.[2,3,4] Different factors may play a role in development of postoperative inflammatory response, including surgical trauma itself, heparin use, anesthetic challenges, use of suction devices, and perioperative stress. Prondzinsky and colleagues[1] in their study supported the opinion that the cardiopulmonary bypass is not the sole factor in generation of postoperative inflammatory response. More large, randomized trials studying different inflammatory pathways are still required to confirm this finding.

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Reply to the Editor:

Thank you for the invitation to respond to Elsayed M. Elmistekawy's letter to the editor. The aim of our study was to show the effect of trauma and cardiac bypass on the systemic inflammatory response during coronary revascularization with and without cardiopulmonary bypass (CPB) to determine the contribution of CPB to the total amount of interleukin (IL) 6 release during the perioperative period.

Elmistekawy states that coronary revascularization without CPB should have been investigated as a control group. In our study we used patients without surgical trauma as a positive control group, comparing them with patients with surgical trauma. The additional IL-6 release during the perioperative period was used to define the contribution of CPB by "subtraction" of the trauma-induced inflammatory response. We agree an additional "negative control group" without CPB would have been helpful to determine the extent of the contribution. Nevertheless, despite the lack of a negative control group, the published data, in our opinion, clearly demonstrate that both aspects, trauma with all related factors and the CPB, contribute to the perioperative inflammatory response. These conclusions are in agreement with the findings of Hazama and colleagues,[1] who demonstrated that off-pump coronary artery revascularization leads to a significant decreased inflammatory response.

Considering these data by Hazama and colleagues and other published data, it is questionable that larger randomized trials

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.

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Radial artery for coronary artery bypass grafting

To the Editor:

I would like to comment on a recent editorial by Mussa and colleagues[1] 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 I[2] 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 colleagues[1] 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.

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Reply to the Editor:

We thank Dr Moran for his interest in our editorial concerning the use of radial arteries (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 associates[2] 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 colleagues[5] 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.

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