Concurrent coronary and carotid artery surgery: an open debate: reply

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We read with interest the letter from Barili *et al.* on our work 'Concurrent coronary artery surgery: factors influencing perioperative outcome and long-term results'. We thank Barili *et al.* for their comments that need our reply.

First, we share their doubts regarding the precise impact of asymptomatic carotid artery stenosis on cerebrovascular events occuring during coronary artery bypass grafting (CABG). This question could only be answered by randomizing patients with concurrent significant coronary and carotid artery lesions to either combined CABG and carotid endarterectomy (CEA) or CABG alone, with post-operative stroke rate being the primary endpoint. However, a very large number of patients would be required for such a study. Indeed, in the recent ACST trial,² a total of 3120 patients with asymptomatic carotid lesions were randomized to immediate CEA or indefinite deferral of any CEA to demonstrate a reduction of 5-year stroke risk from ~12 to ~6% in the immediate CEA group.

Therefore, to reach statistical significance, a randomized study comparing CABG and CEA with CABG alone would necessarily be multicentric (126 clinical sites from 30 countries participated in the ACST trial), with the inevitable differences concerning, for example, the use of a carotid shunt or the closure of the carotid arteriotomy with or without a patch.

Our policy has a more modest goal, which is to associate both surgical procedures during one anaesthesia when, in patients requiring CABG surgery, the carotid lesion meets the criteria accepted for surgical indication. We certainly are unsure that this policy significantly decreases the incidence of post-operative stroke. In our centre, the incidence of post-operative cerebrovascular events is higher after combined CEA-CABG when compared with CEA alone, and the incidence of post-operative myocardial infarction is higher after combined surgery than after CABG alone. This observation supports the notion that carotid artery lesions are a harbinger of severe atherosclerosis.

Secondly, we disagree with Barili *et al.* that our model was unable to accurately predict odds ratio for operative results. It is true that confidence intervals were large, but odds ratios were high (odds ratio for aortic calcifications was 6.3 for operative mortality and 5.2 for stroke). Our results are in concordance with the observation of John *et al.*³ that aortic calcifications were the leading risk factor for stroke, with an odds ratio of 3.0, in CABG patients.

Thirdly, arterial cannulation was usually performed in the ascending or transverse aorta, at a site of normal palpation, especially in the early part of the series. When the aorta was very hostile, arterial cannulation was performed in the femoral artery or in the axillary artery. In the series reported, a moderately dilated ascending aorta (>3.5cm) was left in place. When the ascending aorta was more severely dilated (>5 cm), it was replaced with a prosthesis, but those patients were not included in this study.

Finally, we agree that patients with coronary and carotid symptomatic lesions should be treated with concurrent surgical procedures.

References

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