

to our study, only significant lesions were assessed.<sup>8</sup>

Finally, we are indebted to Sipahi *et al.* for the erratum found on the slopes of the regression lines of the second and third panels of *Figure 3*. Indeed, the value should be negative.

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

We read with great interest the article by Kolh *et al.*<sup>1</sup> dealing with the question about concurrent coronary artery bypass grafting (CABG) and carotid endarterectomy (CEA) surgery. Nevertheless, their report raises some concerns.

The article analysed factors influencing peri-operative outcomes, and ascending aortic calcification was found to be an independent predictor of both stroke and death. These data are considered a common finding in patients who underwent heart surgery.<sup>2</sup> However, in patients with concomitant severe carotid and coronary disease, the predictive role of aortic disease on stroke and death could be significantly overestimated. Carotid disease alone is a risk factor for stroke in patients who undergo surgical revascularization and stroke represents a known complication of CEA. In a recent large study, the adjusted stroke rate was 2.67-fold greater in the combined CEA-CABG group compared with CABG alone.<sup>3</sup> By its nature, this study did not evaluate the impact of CEA on stroke incidence and the confounding effect of carotid disease on mortality and morbidity does not emerge. Hence the predictive effect of aortic disease could be significantly altered. Further studies on patients with and without concomitant CEA are needed to unmask the effect of carotid disease on stroke and to evaluate the real predictive role of aortic disease.

In addition, the multivariate model cannot predict precisely the odds ratio for variables associated with operative mortality and morbidity, as confidence intervals are large, although *P*-values are significant.

Some data about cardiopulmonary bypass and surgical techniques are lacking. We think that it could be important to explain where the arterial cannulation was made in

patients with ascending aorta disease, as the number of no-touch aorta technique and off-pump surgery was lower than expected. Moreover, no explanation was given about surgical approach to 17 patients with aortic dilatation, as it seems that dilated ascending aorta was not substituted.

This study confirms that concurrent CEA and CABG can be performed with acceptable mortality and morbidity, but it represents only one of the surgical option.<sup>4</sup> We agree with the authors who consider treatment of symptomatic territory first reserving the combined simultaneous treatment for patients symptomatic in both territories.<sup>5</sup>

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

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'.<sup>1</sup> 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 occurring 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,<sup>2</sup> 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).<sup>1</sup> Our results are in concordance with the observation of John *et al.*<sup>3</sup> 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,<sup>1</sup> a moderately dilated ascending aorta (>3.5 cm) 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.

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