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catheters for dialysis, placement of inferior vena cava filters, and femoral or brachial artery needle placement during endovascular procedures. One may also consider incorporating the expertise of the noninvasive vascular laboratory technicians to help with this training. As with all techniques, repetition under an appropriate mentor should help minimize the learning curve, minimize complications, and lead to a positive training experience.

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Regarding "Trends and outcomes of concurrent carotid revascularization and coronary bypass"

The optimal management of patients with concomitant carotid and coronary artery disease remains an enduring controversy.¹⁻³ Timaran et al¹ described trends and outcomes in 27,084 concurrent carotid and coronary artery bypass grafting (CABG) revascularization procedures during a 5-year period. More than 96% of these patients received their carotid revascularization procedure for an asymptomatic carotid stenosis. The real debate is not about being staged or synchronous, nor about treatment type (carotid artery stenting [CAS] vs carotid endarterectomy), but whether treatment of asymptomatic carotid stenosis will reduce perioperative morbidity and mortality when combined with CABG at any stage.

In understanding the predominant cause of post-CABG stroke, hypoperfusion and microembolization remain important etiologic mechanisms. Patients with severe aortic disease have a 15% risk of perioperative stroke, paralleling the increased risk caused by severe carotid stenosis. It has to be realized that 50% of post-CABG stroke sufferers do not have carotid disease. Moreover, 60% of territorial infarctions cannot be attributed to carotid disease, confirming the multifactorial etiology of postcardiac surgery neurologic events. Even when prophylactic carotid revascularization would carry no additional risk at all, it can at most prevent 40% of procedural strokes.²

Timaran concludes with suggesting that CAS may provide a safer carotid treatment option for patients who require CABG. Van der Heyden³ recently found a combined death/stroke rate of 1.7% in CAS for asymptomatic carotid stenosis before surgery; however, the overall death/stroke/myocardial infarction rate of combined CAS/CABG still was 8.7%.

Showing that CAS can be performed with an acceptable complication rate is not the issue. First, it must be proven that the combination of CAS and CABG has a significant lower stroke/ death rate than CABG alone when the asymptomatic carotid artery is left untreated. Until then, any revascularization before CABG is unwarranted because it exposes patients to the risks of perioperative stroke and myocardial infarction twice, without significantly reducing the risk of stroke.

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Reply

We appreciate the letter from de Borst et al and do agree that the optimal management of patients with concomitant carotid and coronary artery disease has not been established, particularly for patients with asymptomatic severe carotid stenosis. We also consider that there is an urgent need to establish the role of any carotid intervention in the management of patients with asymptomatic carotid disease who need open coronary revascularization. Unfortunately, only a well-designed and conducted randomized clinical trial could provide the answer to this important clinical problem. Although several efforts to conduct such a trial have been made, to our knowledge, no ongoing study is trying to resolve this issue.

The purpose of our observational, population-based, crosssectional study was not to define the role of carotid interventions in the management of asymptomatic carotid stenosis in patients undergoing coronary bypass but to provide a detailed and descriptive status of the trends and outcomes of patients undergoing combined carotid and coronary interventions in the United States. In this regard, we have revealed that most patients undergoing combined procedures present with asymptomatic carotid disease. Moreover, those undergoing carotid stenting have better outcomes compared with those undergoing carotid endarterectomy. Whether performing carotid interventions for asymptomatic patients undergoing coronary bypass is right or wrong remains to be elucidated, and again, was not intended to be addressed by our study.

Although most cases of stroke during coronary bypass are secondary to embolism, not always related to carotid disease, the fact that up to 40% of periprocedural strokes could be prevented by concomitant carotid interventions, as suggested by de Borst et al, should not be ignored. In fact, it would be a sound justification for carotid interventions for patients with asymptomatic carotid disease. Moreover, specific instances of asymptomatic carotid stenosis that could potentially increase the risk of periprocedural stroke in patients undergoing coronary bypass should be specifically addressed, such as patients with contralateral carotid or vertebral artery occlusion, or both, incomplete circle of Willis, and proximal great vessel disease. Until it is proven that the combined treatment has or does not have a significant benefit compared with coronary bypass alone, neither offering nor denying combined treatment can be justified. Moreover, if combined treatment is offered for asymptomatic carotid stenosis, carotid stenting may be a safer option according to our results.

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