Response to comment on "Patients undergoing cardiac surgery with asymptomatic unilateral carotid stenoses have a low risk of perioperative stroke"

Dear Sir,

Thank you for your comments regarding our study which concluded that patients with unilateral, asymptomatic carotid stenoses undergoing coronary artery bypass grafting (CABG) have a low risk of stroke.¹ You propose that CABG patients require routine evaluation of plaque morphology and that prophylactic carotid interventions should be offered to those with ‘vulnerable’ plaques. However, unless I am mistaken, no-one has conclusively shown that asymptomatic ‘vulnerable’ plaques pose any greater risk of procedural stroke during cardiac surgery.

Since our study was published, another has questioned the role of prophylactic carotid endarterectomy (CEA) or stenting (CAS) in neurologically asymptomatic patients undergoing CABG. Li screened 4335 CABG patients for carotid disease and evaluated the relationship between post-operative stroke and patterns of carotid disease.² The rate of post-CABG stroke was 1.8%, but 95% could not be attributed to carotid disease. More importantly, none of the 5% whose strokes might have been associated with carotid disease could have benefited from prophylactic CEA/CAS (internal carotid artery chronically occluded or <70% stenosis). Finally, 51 of their patients underwent isolated CABG with an asymptomatic 70–99% stenosis. None suffered a stroke. These findings are, of course, identical to those in our study.

You are not alone in believing that there is a causal association between carotid disease and post-CABG stroke. However, a growing body of literature suggests that the evidence may not support this position. It is important to remember that the rationale supporting prophylactic CEA/CAS in CABG patients is to prevent procedural stroke, not late stroke. Accordingly, a fundamental reappraisal of the appropriateness of CEA/CAS in CABG patients with asymptomatic, unilateral carotid disease is required. To paraphrase Roffi, we may be focussing on the wrong culprit (ie carotid stenosis) instead of addressing the true (and more difficult to treat) source of embolism, namely the ascending aorta.³

References