



Commentary

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Articles attracting a large number of citations are regarded by many as high impact or “classic” contributions. Although limitations exist (regarding the use of citation counts to identify the most influential articles in vascular surgery), the article by Naylor et al.¹ must be considered a classic.

Patients with significant carotid disease face an increased risk of stroke during CABG. However, because of the risk of stroke associated with carotid endarterectomy (CEA) and the multitude of pathophysiological mechanisms responsible for post-CABG stroke, the role of prophylactic CEA is still of uncertain value. The unique importance of this review was that the authors approached the debate in an alternative way. Until then, most studies on prophylactic CEA hypothesized about the optimal order of procedures (synchronous/staged/reverse staged CEA). Instead, the authors focused on the actual role of carotid revascularization (as a potential cause of post-CABG stroke) and then questioned whether prophylactic carotid intervention could ever really impact on peri-operative stroke prevention.

It is first important to note that this review focused on 30-day outcomes and did not inform the debate about the potential benefits of prophylactic CEA (in terms of late stroke prevention) in patients scheduled for CABG. Their main findings were that, at the very most, only 40% of post-CABG strokes could be attributable to carotid disease and at least 50% of stroke sufferers did not have any significant carotid disease at all. They then provided five data-supported recommendations for future research with emphasis on the need to report the laterality of any stroke, the status of both carotids, and the timing of stroke onset (which is still of high interest).

After CEA, it is usually possible to determine the most likely cause of any procedure related stroke.² Similar

analyses are, however, difficult following CABG, largely because there are no reliable cerebral monitoring or quality control techniques for diagnosing stroke during CABG. Furthermore, the numbers of mechanisms of stroke after CABG are much greater than following CEA. Thirteen of the 59 series in Naylor’s review documented the timing of 484 strokes following 36,797 CABGs. A relative minority of strokes (38%) occurred within 24 h of surgery, while the majority happened at some postoperative moment, usually within the first seven days. In understanding the main causes of post-CABG stroke, intra operative hypoperfusion and microembolization still remain important mechanisms, but the majority cannot be simply ascribed to some adverse intra operative event.

The ISI web of knowledge and other electronic databases cite 12 further publications from the Leicester group regarding the management of patients with concurrent carotid and cardiac disease, confirming the excellent line of research over the years. Interestingly, there have been over 40 other contemporary papers describing surgical outcomes in patients with cardiac and carotid disease since Naylor’s paper was published, suggesting that research interests in this subject are still heavily influenced by the results of staged/synchronous interventions, rather than addressing the five research recommendations proposed by Naylor (i.e. to determine who really does benefit from prophylactic carotid revascularization?).

Some have suggested that carotid artery stenting (CAS) may become a safer and preferred intervention for patients with carotid disease who require CABG.³ However, simply showing that CAS can be performed with an acceptable complication rate is not the key issue. First, it has to be proven that the combination of CAS and CABG has a significantly lower stroke/death rate than by performing CABG alone (i.e. leaving the asymptomatic carotid artery untreated). Until then, any revascularization before CABG is unwarranted because it exposes patients to the risks of peri-operative stroke and myocardial infarction twice,

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without significantly reducing the risk of stroke. Furthermore, contemporary studies have now shown that the risk of stroke in asymptomatic patients with a unilateral 70–99% stenosis undergoing isolated CABG is extremely low.⁴ The overall risk of stroke after CABG is about 2% and after CABG plus either staged or synchronous CEA or CAS about 9% and these figures have probably not changed over the last decade. Attempts to set-up a CEA-CABG trial have failed so far. Since National and international guidelines still provide no consensus, the management of patients with concurrent carotid and cardiac disease remains enduringly controversial.

In Utrecht (prior to this review being published), our policy was to perform carotid revascularization in patients with concomitant carotid and coronary disease. Following the report, our policy changed and patients now undergo their cardiac procedure without carotid revascularization, unless the patient presents with a symptomatic carotid stenosis or contralateral occlusion. The authors have taught the world that the presence of coexistent carotid disease in

patients scheduled for CABG is not sufficient to warrant routine prophylactic carotid revascularization.

References

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