schools or through schemes like the one which is being run at Monash's Gippsland Medical School, where students are paired with researchers at local hospitals and paid for their time while working on research projects.

As a student, surgical specialties are becoming more competitive and it feels as if research experience is becoming increasingly important to be competitive for acceptance into postgraduate surgical training. Therefore, I believe that postgraduate medical schools offering research programmes will be the schools that will graduate students which are the most competitive students for postgraduate surgical training programmes.

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Conflicts of interest

None declared.

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Nicolas R. Smoll, BAppSc(CompMed), MClinChiro, MB BS (candidate)
Gippsland Medical School, Monash University, Churchill, Victoria, Australia

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Do patients with asymptomatic carotid stenoses still benefit from surgical intervention?

The Carotid Endarterectomy Trialists have convincingly demonstrated that symptomatic patients with a carotid artery stenosis of equal or greater than 70% benefit from carotid endarterectomy (CEA), and decreases the risk of stroke and death by 16% over 5 years, with greater benefit in men, patients aged over 75 years, and those within 2 weeks of their index neurological event (Figs 1,2).[1]

Up to 9% of the population over the age of 75 years will have a 50% or greater carotid artery stenosis,[2] and carotid intima-media thickness is now considered a surrogate marker for risk of systemic atherosclerotic occlusive disease, and correlates well with vascular events.[3,4] Although the concepts of the vulnerable plaque is now well established, the triggers that activate an asymptomatic carotid plaque to become symptomatic remains obscure and identification of the 'at-risk patient' remains difficult. In controlled trials for symptomatic carotid disease, a firm relationship between the severity of stenosis and the risk of stroke has been acknowledged.[1] But, although longitudinal studies in asymptomatic patients have suggested that patients with a greater carotid stenosis is at an increased risk of stroke, this correlation has not been as well defined, and no clear relationship between carotid stenosis and stroke exists; and uncertainty surrounds the failure to find any difference between the higher and lower grades of stenosis, as assessed by ultrasound in trials of asymptomatic carotid artery stenosis.[5,6]

Randomized controlled trials for surgery in asymptomatic carotid artery stenosis (including *ACST* and *ACAS*) were published with stroke rates for medically treated patients ranging from 1.3% to 3.3% per annum.[7] In a prospective population-based study of transient ischaemic attack and stroke, Marquardt *et al*. demonstrated an annual event rate of 0.34% for any ipsilateral ischaemic stroke and 0% for disabling stroke for patients on medical therapy.[8] The *ACST* data were pooled with the *ACAS* trial, and demonstrated a 5-year reduction in stroke from 11.8% to 6.4% in the surgical arm.[5,6] The results were qualified; benefit from surgery was seen in men more than women and also in patients less than 75 years of age. The absolute stroke risk reduction was around 5.4% over 5 years, i.e. an annual risk reduction from around 2% to around 1% with best medical therapy. The benefits of intervention for asymptomatic carotid stenoses were not seen for several years, and patients over the age of 75 were five times more likely to die from a non-neurological (stroke) cause.[9] It has been estimated in *ACAS* that the average risk reduction of ipsilateral stroke with surgery was 1.18% and the perioperative stroke or death rate was 2.3%.[5]

The advent of carotid artery stenting (CAS) has added a new paradigm to the management of carotid artery stenosis (Fig. 3). Randomized controlled trials of patients with symptomatic carotid artery occlusive disease were recently discussed in this journal.[10]

Fig. 1. Severe left internal carotid artery stenosis (with occluded external carotid artery).

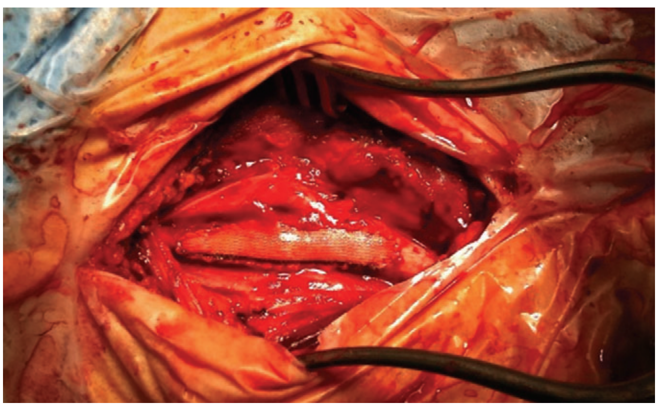

Fig. 2. Internal carotid artery, after endarterectomy and patch (CEA).

CAS 30-day outcome for stroke and death was found to be equivalent to surgery (CEA), 10% versus 9.9% in the *CAVATAS* study.[10] Other non-inferiority-designed trials demonstrated stroke and death rate of 6.8% (CAS) versus 6.3% (CEA) in the *SPACE* trial, and 9.6% (CAS) versus 3.6% (CEA) in the *EVA-3S* trial.[10] The *ICSS* trial demonstrated stroke and death rate of 8.5% (CAS) versus 4.7% (CEA); and a sub-study further demonstrated magnetic resonance cerebral imaging (clinically silent) ischaemic changes in 50% (CAS) versus 17% (CEA).[10] The *SAPPHIRE* study included stroke death and myocardial infarction as end point in 334 patients (71% asymptomatic), and demonstrated 12.2% event rate (CAS) versus 20.1% (CEA), the difference mainly due to coronary events in the CEA group.[11] The recently published *CREST* trial (47% asymptomatic) demonstrated 7.2% (CAS) versus 6.8% (CEA) for stroke death or myocardial infarction, and a stroke rate of 4.1% (CAS) versus 2.3% (CEA).[12]

Fig. 3. Internal carotid artery, after angioplasty and stenting (CAS).

SAPPHIRE and *CREST* were the two randomized trial of CAS to include significant numbers of asymptomatic patients.[11,12] In *CREST*, the overall 4-year rate of stroke or death was 6.4% in the CAS group as compared with 4.7% in the CEA group ($P = 0.03$); the respective rates were 8.0% and 6.4% for symptomatic patients ($P = 0.14$), and 4.5% and 2.7% among asymptomatic patients ($P = 0.07$).[12] In *SAPPHIRE*, there were 117 asymptomatic patients in the stenting group and 120 in the endarterectomy group; as determined in post hoc analyses, the rates of stroke at 3 years were 10.3% (12 of 117 patients) in the stenting group and 9.2% (11 of 120 patients) in the endarterectomy group.[13]

Medical therapy for asymptomatic, significant carotid artery stenosis in the pooled *ACST/ ACAS* data is 11.8% risk of stroke over 5 years or 2.4% per annum.[5,9] There is as yet no randomized data comparing CAS and medical therapy, and trials to look at this question, has yet to attract funding. The data that do exist suggest that asymptomatic patients remain with a similar or higher stroke risk after CAS compared to the natural history of the carotid stenosis itself.[12,13] Benefit from surgery in *ACST/ACAS* was limited to a select group of patient, but the degree of stenosis associated with stroke remains ill-defined and the data are further confounded by consideration of contralateral stroke in the trial cohort.[5] The true ipsilateral stroke rate in the medical group, however, was only 3.9%.[5] *ACAS* and *ACST* were interpreted as CEA being protective against stroke in asymptomatic patients, but the procedural stroke and death rate had to be kept to a minimum (approximately 3% or less) for a benefit to be seen, and perhaps not replicated in real world surgery.[5,6]

The evolution of best medical therapy and conscientious regulation of risk factors has also not been considered fully; in *ACST*, the use of anti-hypertensives and statins was not standardized with a gradual uptake over the years of follow-up, and the effect of this evolution of medical treatment with time was not measured.[5] Over the years, there

has been a significant change to the threshold values used to define risk factor disease states, for example, the definition of hypercholesterolemia, diabetes and hypertension, and the introduction of newer more effective drugs (e.g. clopidorel and statins).[7,8] Analysis of medically treated patients over many years demonstrated a decrease in stroke rate over time with best medical therapy; the *SMART* study further demonstrated a continuation of the downward trend among patients receiving medical intervention for vascular disease, with the annual risk of stroke from asymptomatic carotid stenosis of less than 1%;[14] and medical intervention alone was calculated to be at least 3 to 8 times more cost-effective in stroke prevention.[7]

While patients with symptomatic carotid artery stenoses benefit from intervention, improvements in medical therapy now mean that the same probably does not hold true for asymptomatic carotid stenoses with stroke rates of less than 1%. There has also not been a concurrent reduction in surgical risk, which in many centres exceed 2–3% for both CAS and CEA.[7] It is also interesting that the magnetic resonance imaging changes after CAS is not considered in the same light as a troponin rise (indicating myocardial infarction) after CEA. When offering intervention to patients with asymptomatic carotid stenoses with marginal benefit at best, vascular interventionalists should adhere to evidence and guidelines as recently discussed in this journal.[10] But it is perhaps time for surgeons to stop taking comfort from the relative risks of intervention, and historic studies of medical management, and instead look to future well-designed studies of modern medical management of asymptomatic carotid stenoses.

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Irwin V. Mohan, MB BS, MD, FRCS, FEBVS, FRACS
Shannon D. Thomas, BMedSc, MB BS
Westmead Hospital, University of Sydney, Sydney, New South Wales, Australia

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A pilot training program in surgical communication, leadership and teamwork

The Royal Australasian College of Surgeons (RACS) has defined nine competency domains regarded as integral to being a surgeon.[1] Competencies such as technical expertise and clinical decision-making can be acquired by established educational and training methods, usually in the workplace. Other competencies relating to professionalism, communication, collaboration, management and leadership largely rely on the ability of a trainee to recognize good role modelling of relevant skills. Furthermore, trainees must then have the willingness to integrate appropriate behaviours into clinical practice and the ability to improve performance with or without feedback.

The competencies concerning professionalism, communication, collaboration, management and leadership are sometimes referred to as 'non-technical skills' (although this term is misleading because elements of the competencies are in fact highly technical). Similarly, these competencies are often regarded as being less tangible, less easy to formally teach and less objective to measure. However, the evidence indicates that poor communication and interpersonal skills and failure to demonstrate leadership and responsibility with patient care are significant contributors to poor patient outcomes.[2] Deficiencies in these skills also result in patient dissatisfaction, complaints and medico-legal claims.[3–5]