CASE PRESENTATION

The patient, a right-handed 64-year-old pakeha man (New Zealand European), was referred to the neurology clinic in June 2000. His family doctor had discovered bilateral carotid bruits and arranged an ultrasound examination. He was feeling very well; specifically, there were no symptoms of any kind which could be attributed to these bruits – no focal neurological symptoms, or even dizziness or lightheadedness – and he took regular vigorous exercise without difficulty.

Treatment of hypertension had begun more than a decade previously. In 1997 some recordings had been as high as 200/105 mmHg, but at the time of referral his blood pressure was controlled (150/80–90) with Atenolol 50 mg b.d. and Indapamide 2.5 mg daily. His family doctor had discovered bilateral carotid bruits and arranged an ultrasound examination. He was feeling very well; specifically, there were no symptoms of any kind which could be attributed to these bruits - no focal neurological symptoms, or even dizziness or lightheadedness - and he took regular vigorous exercise without difficulty.

The dilemma
After extensive dialogue over two appointments, conservative management was chosen. The patient had no strong preferences for, or against, carotid surgery. He declined an offer of a further opinion (although the choice of specialist may have been as difficult as the original advice). Two UK stroke medicine experts were provided with a brief synopsis and asked their opinions about bilateral carotid endartarectomy and gave contradictory advice. The patient was warned to report immediately if he noted any focal neurological symptoms and was prescribed aspirin 150 mg daily.

The stroke

Two months after his outpatient visit he collapsed at home just after 08.00 h with right-sided weakness and was brought directly to the emergency department. At 10.00 h he was dysarthric with dysphasic errors, had normal visual fields to confrontation and a right hemiparesis, face, arm and leg. The systolic blood pressure was over 200, the left carotid bruit remained audible. A brain CT scan was normal. After discussion of the risks and benefits it was elected to administer intravenous alteplase 0.9 mg/kg, 10% as a bolus and the remainder as an infusion over one hour, together with iv Labetalol to modestly reduce his blood pressure. Within 24 h of onset he was aphasic with oromotor apraxia, right visual inattention, a paralysed right arm but he had regained some movement in the right leg, which had near nor-
mal power. Aphasia prevented sensory examination, there was no important dysphagia. A repeat CT scan that day showed a small hypodense area in the left basal ganglia with mild oedema and lateral left frontal lobe ischaemic change. There was no haemorrhage. Repeat carotid Doppler ultrasound was essentially unchanged, the left carotid artery remained patent. Warfarin was started after 10 days. Two months later, following rehabilitation in hospital, first a left and then three months later a right carotid endarterectomy were performed, without complications. The surgeon considered the operative appearances to those of reasonably stable plaque, with a lot of atheromatous material in pits.

The consequences
Six months after his stroke, he still had a non-fluent dysphasia, making communication difficult and normal conversation impossible. There was little spontaneous speech but he could name most everyday items. Comprehension appeared normal, at least he could follow commands with complex grammatical construction. Walking was unhindered but the right arm remained essentially paralysed except for some proximal movement. He was able to live alone with limited support from a friend.

DISCUSSION
There is firm evidence that carotid surgery benefits selected patients with symptomatic carotid territory transient ischaemic attacks, but this example illustrates the difficulties in advising patients with asymptomatic carotid stenosis. Some physicians do not even embark upon investigations of carotid bruits yet, with the benefit of hindsight, my patient would have had a better outcome with elective, bilateral carotid endarterectomy.

In a resource-limited public health environment, such as New Zealand, surgery for asymptomatic carotid stenosis is rarely offered, as 50–80 patients of the kind entered into the trials of asymptomatic carotid surgery need to undergo surgery to prevent one stroke, over three years of follow up. However, the management of individual patients with asymptomatic stenosis remains controversial and a rigid symptomatic/asymptomatic dichotomy may lead to inappropriate decisions for both groups. What then was the risk/benefit analysis for this patient?

Using the New Zealand Heart Foundation risk stratification tables for asymptomatic patients (Dyslipidaemia Advisory Group 1996), he had an estimated 10–15% chance of a vascular event over the next five years (i.e. angina, transient ischaemic attack, myocardial infarction, stroke or coronary death). However this figure does not incorporate the influence of carotid stenosis. This risk is similar to the 2% per annum stroke rate of patients randomised to no surgery in asymptomatic carotid surgery trials (Chambers et al. 2001). The patient faced a perioperative stroke and death risk of about 2–3% for each operation, possibly less, based on local audit. The benefit of surgery is harder to estimate; patients in the trials achieved an absolute risk reduction of only about 1% (50% relative risk reduction) per annum over three years. However, an important consideration is the anticipated length of disease-free survival after successful surgery, clearly greater benefit will accrue for younger patients, assuming local complications such as neointimal hyperplasia do not develop. Furthermore, it is possible that the studies included in the Cochrane review are biased against surgery (Chambers et al. 2001), patients such as mine may not have been randomised in these trials if judged to be at higher risk by virtue of their disease severity. The trials of carotid surgery for asymptomatic patients may not be directly applicable to this patient, but surely must benefit some, albeit a minority of individuals, with asymptomatic carotid lesions. Although degree of stenosis, presence of bilateral disease, plaque morphology, gender and age, number of vascular risk factors or microembolic signals detected by transcranial Doppler, may be important variables, evidence that these factors enable clinicians to stratify risk and select patients appropriately for carotid surgery is still elusive. Unfortunately but inevitably, patients with asymptomatic stenosis may present with disabling stroke, rather than a transient ischaemic attack, yet it is difficult to improve the health of an asymptomatic individual (as opposed to ‘patient’). ‘Above all do no harm’ remains a sound principal but we also have a duty to prevent harm, if possible.

REFERENCES