THE BENEFIT OF CAROTID ENDARTERECTOMY has usually been assessed clinically by the prevention of further symptoms.\(^1,2\) It is only relatively recently that readily repeatable methods for objective follow-up assessment of the patency of carotid arteries have been available which have no significant complications. Carotid angiography is the standard method for assessing carotid artery disease but it is not entirely satisfactory since it may miss ulceration or lesions present in a different plane to that of the angiogram.\(^3,4\) It is also associated with complications\(^5,6\) and therefore is not routinely used to assess the patency of the internal carotid artery in asymptomatic patients following carotid endarterectomy.\(^7\)

Angiography performed soon after carotid surgery can provide unreliable information, which may be due to tissue fragmentation and oedema of the vessel wall.\(^8\) Postoperative angiograms were performed by Schultz and his colleagues\(^9\) who reported that of eleven internal carotid arteries shown to have irregular walls at this time, four were found to be smooth on angiograms performed after a further six months. Lougheed et al\(^10\) also reported a patient in whom a carotid angiogram, which was not performed until six months after carotid endarterectomy, showed the arterial wall to be irregular, whilst an angiogram performed more than two years later demonstrated a smooth wall.

Non-invasive tests have been developed in recent years to avoid the risks of angiography and to provide complementary information to the radiological investigation. These tests have included oculopneumoplethysmography,\(^11\) oculoplethysmography with photoangiography\(^12\) and the use of Doppler-shifted ultrasound.\(^13,14\)

The method used in this paper for assessing the carotid arteries, using spectral analysis of Doppler-shifted ultrasound, has been reported previously.\(^15,16\) The method is based on two parameters, that is, the temporal artery occlusion test (TAOT)\(^17\) and the ratio of the two peaks (A and B) that occur during cardiac systole in sonagrams from the supraorbital and common carotid arteries (fig. 1).\(^15\)

The TAOT (fig. 2) detects carotid artery disease sufficiently severe to cause a reduction in distal mean pressure in the internal carotid artery. The A/B ratio of sonagrams is used to detect less severe carotid lesions in addition to those that cause a pressure drop, although it can not be used to indicate the degree of stenosis.\(^16,18\) In a retrospective comparison between angiography and ultrasound in 201 carotid bifurcations it was shown that a combination of the TAOT and A/B ratio detected lesions in the internal carotid artery, of 25% and over reduction in lumen diameter, with a sensitivity of 92% and a specificity of 82%.\(^16\) The purpose of this paper is to report our experience using this method to assess the patency of the internal carotid artery in patients following carotid endarterectomy.

Patients

A total of 48 patients (37 male:11 female) were investigated, which represented 58 carotid endarterectomies; the mean age was 55 years, with a range of 37 to 76 years. A carotid endarterectomy was performed in all patients and in ten the operation was performed on both sides of the neck. The clinical features, with regard to cerebrovascular disease, at the time of carotid endarterectomy are shown in table 1.
DOPPLER ULTRASOUND ASSESSMENT OF ICA/Padayachee et al

Normal pulse age 64 years
Abnormal pulse age 69 years

FIGURE 1. A/B ratios in sonograms from the supraorbital arteries of a 64 year old man with a normal carotid arteriogram and a 69 year old patient whose arteriogram showed disease at the carotid bifurcation. (Sonograms show Doppler-shifted frequency on the ordinate, time on the abscissa and the trace blackness is related to the signal amplitude).

Method

Doppler ultrasound examinations were performed with patients lying supine on a couch. An 8MHz Sonicaid BV380 directional blood-velocimeter was used to obtain backscattered Doppler signals from the carotid and supraorbital arteries. These signals were recorded onto a stereo cassette tape recorder and processed by a spectral analyser. The signals were displayed in sonogram form (fig. 1) almost instantaneously, that is within five milliseconds. A hard copy print-out was made on ultra-violet sensitive paper (Kodak, linagraph 1801). This enabled precise measurement of the A/B ratios (fig. 1) using a programmed dedicated computer.

FIGURE 2. The temporal artery occlusion test: The response of supraorbital artery flow during compression of the ipsilateral superficial temporal artery is shown in sonograms from a normal subject and a patient with total occlusion of the internal carotid artery. "Off" and "on" indicate when the superficial temporal artery is compressed.

TABLE 1 Clinical Features with Regard to Cerebrovascular Disease of Patients Undergoing Carotid Endarterectomy

<table>
<thead>
<tr>
<th>Number of carotid endarterectomies</th>
<th>Asymptomatic LCB† no LCB</th>
<th>Cerebral ischaemic incidents LCB no LCB</th>
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<td>Number of patients</td>
<td>48</td>
<td>58</td>
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*Disease detected during bilateral angiography for contralateral symptomatic carotid artery disease.
†Localised carotid bruit.

(Pet 3032K, Commodore) interfaced to a ‘Bit-Pad one’ digitiser.

The screening procedure was performed on both internal carotid pathways. The supraorbital artery was insonated and the TAOT performed, as illustrated in fig. 2. The common carotid artery was then insonated at the base of the neck, following which the internal and external carotid arteries were insonated.

The presence of severe disease was indicated by a positive response to the TAOT, which was due to either total occlusion or severe stenosis of the internal carotid artery. These two conditions were then distinguished by examination of sonograms from the carotid arteries in the neck. Severe stenosis was indicated by turbulent, high blood-velocities in the internal carotid artery. Total occlusion was indicated when sonograms attributable to the internal carotid artery could not be detected. Further evidence confirming either total occlusion or severe stenosis was then obtained by imaging the carotid bifurcation.

The remaining patients had no evidence of severe disease and were categorised as normal or abnormal according to the value of the A/B ratio of supraorbital and/or common carotid sonograms. An abnormal test was indicated when A/B ratios of resting supraorbital and/or common carotid pulses were less than 1.05. All patients were assessed with ultrasound in the immediate post-operative period and further assessments were made as near as possible at yearly intervals, for up to six years with the mean follow-up period being thirty-four months.

Results

Mortality Following Carotid Endarterectomy

Three patients died soon after the carotid endarterectomy (6.3%). Post-mortem studies revealed the cause of death to be cerebral haemorrhage in one patient and cerebral infarction in the other two. Follow-up ultrasound assessments were therefore performed on 45 patients, which represented 54 carotid endarterectomies. During the follow-up period a further two patients died from myocardial infarction, the first at one year and the second at two years.

Forty-three patients (52 carotid endarterectomies) were alive at the end of the follow-up period.

Development of a Severe Lesion in the Internal Carotid Artery Following Endarterectomy (fig. 3)

In the immediate post-operative period the ultrasound investigation detected a severe lesion in the in-
NUMBER OF INTERNAL CAROTID ARTERIES

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Each box = one internal carotid artery
■ = associated cerebral ischaemic episode
□ = associated localised carotid bruit

FIGURE 3. Presence, and subsequent development, of a severe lesion in the ipsilateral internal carotid artery following carotid endarterectomy, demonstrated with ultrasound by the temporal artery occlusion test.

ternal carotid artery in seven of the 54 carotid endarterectomies. All seven were associated with a stroke, which occurred within hours of the operation. Two patients immediately returned to the operating theatre following the ultrasound investigation. A thrombectomy was performed in both patients and subsequent ultrasound examinations revealed patent internal carotid arteries. Surgery was not considered appropriate in the other 5 patients.

During the six year follow-up period the ultrasound examination showed that in addition to the 7 cases above, 7 of the remaining 45 endarterectomies (36 patients) had developed a severe lesion in the internal carotid artery. In 5 endarterectomies (5 patients) this lesion developed between one to two years after endarterectomy (fig. 3). Two of these 5 patients had developed an associated stroke and carotid bruit, two had TIA's, one of whom had a bruit. The fifth patient was asymptomatic. The remaining two patients developed a severe lesion in the fourth and fifth years following carotid surgery; in the former this was associated with TIA’s.

The ultrasound examination showed that 2 of these 7 patients had complete occlusion of the internal carotid artery, which was confirmed by angiography in both cases. One of these patients had an extra-intracranial bypass operation. Another patient underwent angiography which confirmed severe stenosis of the internal carotid artery but a second endarterectomy was not considered appropriate.

Presence of Severe Disease at the Contralateral Carotid Bifurcation to the Side of the Endarterectomy (fig. 4)

Thirty-eight patients had a carotid endarterectomy on one side of the neck only, 8 of whom had severe contralateral internal carotid artery disease at the time of carotid surgery. This was demonstrated by ultrasound and confirmed by angiography, both performed before surgery. The internal carotid artery was completely occluded in five of the eight patients.

Ultrasound examinations performed after endarterectomy on these 38 patients showed that a further 4 had developed severe disease in the contralateral internal carotid artery within the six year follow-up period. One of these patients had TIA's, whilst the remaining three were asymptomatic. A localised carotid bruit developed in one asymptomatic patient. The ultrasound investigation demonstrated complete occlusion of the internal carotid artery in the patient with symptoms. Angiography was not performed for any of these 4 patients.

A/B Ratios Following Carotid Endarterectomy (fig. 5)

Abnormal A/B ratios associated with a normal response to the TAOT were measured in sonograms from twenty-four of the ultrasound assessments performed immediately post-operatively. Four patients (4 endarterectomies) developed carotid bruits. Three of these had associated TIA’s post-operatively, which persisted in two. A further 4 patients had post-operative TIA’s. Subsequent ultrasound examinations revealed the development of severe internal carotid artery disease in seven of these twenty-four patients, that is seven endarterectomies. These included the two cases above with persistent TIA’s, one of which subsequently developed a carotid bruit. These seven cases have been referred to in the section on the development of severe disease in the internal carotid artery following endarterectomy (fig. 3).

Normal A/B ratios were obtained from 23 cases at the first post-operative assessment and of these, 19 remained normal at follow-up visits. None of these nineteen became symptomatic or developed severe disease during the follow-up period. Four cases developed abnormal A/B ratios and two were associated with TIA’s (fig. 5).
Arterial Disease in Other Regions of the Body

Nineteen of the forty-eight patients had symptomatic arterial disease in regions other than the carotid territory before carotid surgery. Two patients had ischaemic heart disease, 10 lower limb arterial disease and 7 were symptomatic in both these regions. Symptomatic arterial disease of the heart and/or lower limbs developed in five of the 43 patients who remained alive during the follow-up period.

Discussion

Reported figures for operative complications and long-term follow-up of carotid endarterectomies have varied. This may be due in part to differences in selection of patients, surgical procedure and the method of follow-up assessment. The study presented here shows that 7 of the 54 endarterectomies (13%) in the surviving 45 patients resulted in strokes within the first week of carotid surgery, and these were all associated with total occlusion of the internal carotid artery on the side of the operation. This figure for operative morbidity is relatively high when compared with some previous reports. However, comparison of surgical results can be misleading since in a number of studies different patient groups have been considered. It has been noted for example that patients who have had symptoms of cerebral ischaemia have a greater operative morbidity and mortality than those who have surgery for asymptomatic disease.

Surgical exploration of the carotid arteries for patients in whom strokes develop soon after endarterectomy has been frequently discussed. Treiman et al reported thrombosis at the site of carotid endarterectomy as the predominant cause of such strokes and advised surgical exploration as soon as the stroke had been diagnosed. In our series a successful thrombectomy was performed in two of the patients who developed a stroke soon after endarterectomy and had been shown by ultrasound to have a severe lesion in the internal carotid pathway. However, in both patients the neurological deficit was permanent.

Ortega et al found that the incidence of immediate post-operative occlusion of the internal carotid artery was three times greater when there was no severe disease present at the contralateral carotid bifurcation. They concluded that severe contralateral disease could result in a greater blood flow through the repaired artery, since this may then supply both the ipsilateral and contralateral hemispheres; the greater rates of blood flow which would then occur in the ipsilateral internal carotid artery may be responsible for the reduced incidence of post-operative thrombosis. However, in our series 3 of the 7 patients who developed a post-operative stroke were shown by ultrasound to have severe disease at the contralateral carotid bifurcation.

The long-term results of this study show that during the six year follow-up period the ultrasound demonstrated severe restenosis or total occlusion of a further 7 of the 45 internal carotid arteries assessed.

The interval between an endarterectomy and the development of occlusive lesions in the internal carotid artery has been related to differences in etiology. Stoney et al reported the results of histological studies performed on 29 specimens obtained during operations in which recurrent carotid lesions were excised. In 9 patients recurrent stenoses developed in the first post-operative year and were shown to be due to intimal fibrosis, whilst restenosis occurring more than two years after operation was shown to be due to atheroma. In our study 3 patients developed severe lesions within one year of endarterectomy and in a further four patients such lesions developed between two to five years.

The development of minor occlusive disease in the internal carotid artery after endarterectomy has not been reported in angiographic studies. Abnormal A/B ratios measured with ultrasound soon after endarterectomy cannot be interpreted as indicating minor disease since they could be due to distortion of the carotid bifurcation as a result of the surgical procedure. In our study abnormal A/B ratios were obtained in sonagrams from 24 cases after carotid endarterectomy, and these A/B values remained abnormal at subsequent visits. Seven of these cases developed severe disease as indicated by a positive response to the TAOT, 5 being associated with symptoms. In 4 instances normal sonogram A/B ratios were obtained post-operatively, but became abnormal at later assessments; two of these were associated with TIA's. In all 4 the response to the TAOT was normal, so the abnormal A/B ratios may have been due to the development of non-severe carotid artery disease.

Atherosclerosis is often considered to affect the whole vascular system, although in varying degrees. Our results show that of the forty-eight patients who had carotid artery disease twenty-three (48%) either
had symptoms of occlusive arterial disease in other regions of the body at the time of carotid endarterectomy or developed them within the follow-up period. This indicates that non-invasive assessment of asymptomatic regions in patients who present with symptoms in one region only may provide valuable clinical information.

The role of monitoring carotid endarterectomy with non-invasive tests requires further evaluation. In this study ultrasound has been used in the immediate post-operative period to provide information about the development of an occlusive lesion in the internal carotid artery. It has also been used for serial monitoring of patients who have undergone carotid endarterectomy, in order to provide information about the development of restenosis of the internal carotid artery and to assess the patency of the contralateral carotid bifurcation.

We suggest that the non-invasive screening method described is a useful adjunct to clinical assessment for the follow-up of patients who have undergone carotid endarterectomy. It provides objective information about the haemodynamic state of the repaired artery which complements clinical assessment.

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