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A Policy of Quality Control Assessment Helps to Reduce the Risk of Intraoperative Stroke During Carotid Endarterectomy

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Objectives: *a pilot study in our unit suggested that a combination of transcranial Doppler (TCD) plus completion angioscopy reduced incidence of intra-operative stroke (i.e. patients recovering from anaesthesia with a new deficit) during carotid endarterectomy (CEA). The aim of the current study was to see whether routine implementation of this policy was both feasible and associated with a continued reduction in the rate of intraoperative stroke (IOS).*
Materials and methods: *prospective study in 252 consecutive patients undergoing carotid endarterectomy between March 1995 and December 1996.*
Results: *continuous TCD monitoring was possible in 229 patients (91%), while 238 patients (94%) underwent angioscopic examination. Overall, angioscopy identified an intimal flap requiring correction in six patients (2.5%), whilst intraluminal thrombus was removed in a further six patients (2.5%). No patient in this series recovered from anaesthesia with an IOS, but the rate of postoperative stroke was 2.8%.*
Conclusions: *our policy of TCD plus angioscopy has continued to contribute towards a sustained reduction in the risk of IOS following CEA, but requires access to reliable equipment and technical support. However, a policy of intra-operative quality control assessment may not necessarily alter the rate of postoperative stroke.*

Key Words: Carotid endarterectomy; Transcranial Doppler; Angioscopy.

Introduction

Despite a proven role in the management of selected patients with symptomatic carotid artery disease, the International Trials have shown that carotid endarterectomy (CEA) is associated with a 2–4% risk of death or disabling stroke and a 5–7% risk of any stroke.[1,2] In the past, the majority of operation-related strokes were apparent upon recovery from anaesthesia (the intraoperative stroke) and must be directly attributable to some adverse event occurring during the procedure.[3–5] Evidence suggests that the commonest underlying cause of intraoperative stroke (IOS) is inadvertent technical error[4] due to retained intraluminal thrombus, residual stenoses and intimal flaps.

Prior to 1992, the risk of suffering an IOS during CEA in this Unit was 4%. In order to try and reduce this risk, we undertook a prospective audit of the causes of operative stroke and the role of various quality control methods including transcranial Doppler (TCD) monitoring, B-mode ultrasound imaging, continuous wave Doppler assessment and completion angioscopy. By the end of the pilot study, the incidence of IOS had fallen from 4 to 1% and we concluded that (for us) a combination of TCD monitoring plus completion angioscopy provided the maximum yield in terms of identifying inadvertent technical error.[6]

The aims of the current study were to see whether this apparent improvement in IOS risk could be sustained following the pilot study and how easy it was to continually implement this policy.

Materials and Methods

Between 1 March 1995 and 31 December 1996, 252 patients (median age 68 years) underwent either CEA ($n=243$) or saphenous vein carotid bypass ($n=9$) for the correction of severe (>70%) internal carotid artery (ICA) stenotic disease. Patients who underwent surgery for carotid body tumour, aneurysm, trauma, fibromuscular dysplasia or Takayasu disease were not included. Clinical presentation included stroke in 96 (38%), TIA/amaurosis in 129 (51%), while 27 patients (11%) were asymptomatic. Unilateral ICA stenoses were present in 161 (64%), bilateral severe

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ICA stenoses in 50 (20%), while 41 (16%) had a contralateral occlusion.

Carotid endarterectomy

Carotid endarterectomy was performed via a longitudinal arteriotomy in a standardised manner throughout the period of study by one of three consultants in vascular surgery (PB, RN, NL) or by a higher surgical trainee under consultant supervision using normocarbic, normotensive general anaesthesia and systemic (5000 IU) heparinisation. All patients underwent routine shunting (Pruitt–Inahara), the proximal and distal intimal steps were tacked down with 7:0 prolene (Ethicon) and all arteriotomies were closed with a Dacron patch (Vascutek). Following completion of the operation and recovery from anaesthesia, the patient was examined neurologically and transferred to the High Dependency Unit for 6 h of postoperative TCD monitoring (see below). Any new neurological deficit apparent upon recovery from anaesthesia was recorded and the patient assessed by a neurologist. Postoperative neurological complications (i.e. following normal recovery from anaesthesia and up to 30 days thereafter) were also documented. Our protocol required us to investigate all patients awakening from anaesthesia with a new neurological deficit by colour Duplex and TCD. The decision to re-operate was thereafter left to the discretion of the surgeon. All neurological deficits occurring in the postoperative period were investigated by colour Duplex, TCD and CT scan.

Intraoperative transcranial Doppler monitoring

Continuous TCD monitoring of blood flow velocity (time averaged mean) in the ipsilateral middle cerebral artery (MCA) was performed using a 2 MHz pulsed wave probe (via the transtemporal window), secured with an elasticated headband and connected to a Scimed PC842 transcranial system (Scimed, Bristol, U.K.). The probe was protected from dislodgement by a detachable, semicircular metal headguard. Recording started following induction of anaesthesia and continued until the surgical drapes were removed from the patient. Data were recorded onto digital audio tape for off-line analysis with particular emphasis on the number and character (air vs. particulate) of emboli detected throughout the procedure.[7,8] During the operation, one of the research fellows experienced in TCD (MG, JS, NL) were available to supervise minor revisions to the probe position and generally advise the surgeon of unexpected phenomena. In general, we aimed to keep MCA velocity ≥ 15 cm/s throughout the procedure, preferably ≥ 20 cm/s.

Completion angioscopy

Following endarterectomy and prior to closure of the patch, the shunt was removed, the carotid vessels flushed and irrigated with heparinised saline and then re-clamped. A 5 mm segment of the arteriotomy adjacent to the origin of the external carotid artery was left unsutured and through this an angioscopic assessment of the distal ICA, proximal CCA, ECA orifice and endarterectomy zone was performed prior to restoration of flow. In the original pilot study a 2.8 mm flexible multi-fibre angioscope (Olympus AF type 28C, Keymed, Southend on Sea, U.K.) was used.[6] Although there were no major problems with this angioscope during the pilot study, it subsequently suffered repeated fibre breakage and became unusable. In March 1995 this was replaced with a more robust 5 mm diameter flexible hysteroscope (Olympus 1070-48).

A standardised operation note required the surgeon to record any abnormal findings within the endarterectomy zone. The policy of the unit was to remove any fragments of thrombus and repair any intimal flap >3 mm.[6] Where necessary, an estimate of luminal sizing of an abnormality was based on comparison with the 2 mm diameter head of the forceps that could be passed down the instrument channel of the hysteroscope. If any abnormality required correction, the endarterectomy zone was routinely re-examined to confirm that there was no residual abnormality.

Postoperative TCD monitoring

From October 1995 onwards we introduced a policy of postoperative TCD monitoring where ipsilateral MCAV data were recorded for 10 min every 30 min for 6 h following restoration of flow. This policy change was based on previous studies in the unit which showed that embolic signals detected in the early postoperative period were exclusively particulate,[8] that sustained postoperative embolisation was highly predictive of stroke due to carotid thrombosis[9,10] and that selective intervention with Dextran-40 therapy could reduce the risks of progressing on to a postoperative carotid thrombosis.[11] Accordingly, any

Table 1. Ability to undertake TCD and completion angioscopy.

(i) Transcranial Doppler monitoring			
Successful TCD monitoring	No accessible TCD window	Simultaneous CEAs performed	Technical failure
229 (90.8%)	15 (6%)	7 (2.8%)	1 (0.4%)
(ii) Completion angioscopy			
Successful angioscopy	Equipment failure	Simultaneous CEAs performed	Other reason
238 (94.0%)	3 (1.2%)	3 (1.2%)	8 (3.2%)

patient who had ≥ 25 emboli detected during any 10 min period of monitoring was commenced on an incremental intravenous infusion of Dextran-40, starting at a rate of 20 ml/h. If the rate of embolisation did not diminish, the infusion was gradually increased by 5 ml/h every 10 min to a maximum of 40 ml/h. Once the Dextran infusion rate was stabilised, it was then continued at that dose for a further 12 h.

All patients were discharged home on aspirin therapy (150 mg daily), usually on the fifth postoperative day, and all were reviewed 4–6 weeks later in the Vascular Clinic.

Results

Ability to monitor

Continuous intraoperative TCD monitoring was achieved in 91% of patients undergoing CEA in this study. Technical failures (insonating the wrong artery, inexperience at monitoring, irretrievable probe dislodgement, equipment failure) fell from 3% in the pilot study[5] to 0.4% in the current study. The only residual barrier to routine TCD monitoring remains an inaccessible cranial window (affecting 6% of patients in both the pilot and current studies) and insufficient TCD machines to monitor more than two procedures at any one time (2.8%).

A successful angioscopic assessment was performed in 94% of patients (Table 1). The principal reasons for not performing angioscopy were equipment failure in three patients (1.2%), two theatres requiring the angioscope at the same time (1.2%), while eight of the nine patients undergoing carotid bypass were not angioscoped. The latter policy has been changed and all carotid bypasses are currently angioscoped prior to restoration of flow.

Prevention of inadvertent technical error

During this study, 94% of patients underwent a normal angioscopic assessment (Fig. 1), six patients (2.5%) underwent repair of an intimal flap, whilst six (2.5%) were noted to have residual luminal thrombus which was removed prior to restoration of flow (Fig. 2).

Impact on neurological morbidity

In this study, no patient recovered from anaesthesia with a new neurological deficit (Table 3) to give an intraoperative stroke rate of 0%. However, seven patients died or suffered a stroke in the 30-day postoperative period to give an overall death and disabling stroke rate of 1.6% and a death/any stroke rate of 2.8%.

The commonest cause of early morbidity was intracranial haemorrhage which was proven on CT scan in three patients. Two (day 3 and day 7) did not have an accessible TCD window and were not monitored with TCD, but blood pressure control had been normal throughout. The remaining patient underwent normal perioperative TCD and angioscopic assessment, was discharged home on day 5 but suffered a fatal intracranial haemorrhage on day 23.

Of the remaining four postoperative strokes (Table 3), one underwent an uneventful CEA but had to return to theatre 2 h later for evacuation of a neck haematoma. Immediately following this he became hypoxic and required ventilation, following which he awoke with an ipsilateral monoparesis.

Extracranial and transcranial Duplex studies were normal but a CT scan revealed a focal MCA territory infarction. This stroke occurred before implementing our policy of routine postoperative TCD monitoring, so that we do not know if he had TCD evidence of ongoing postoperative embolisation.

Three non-disabling strokes (with full recovery

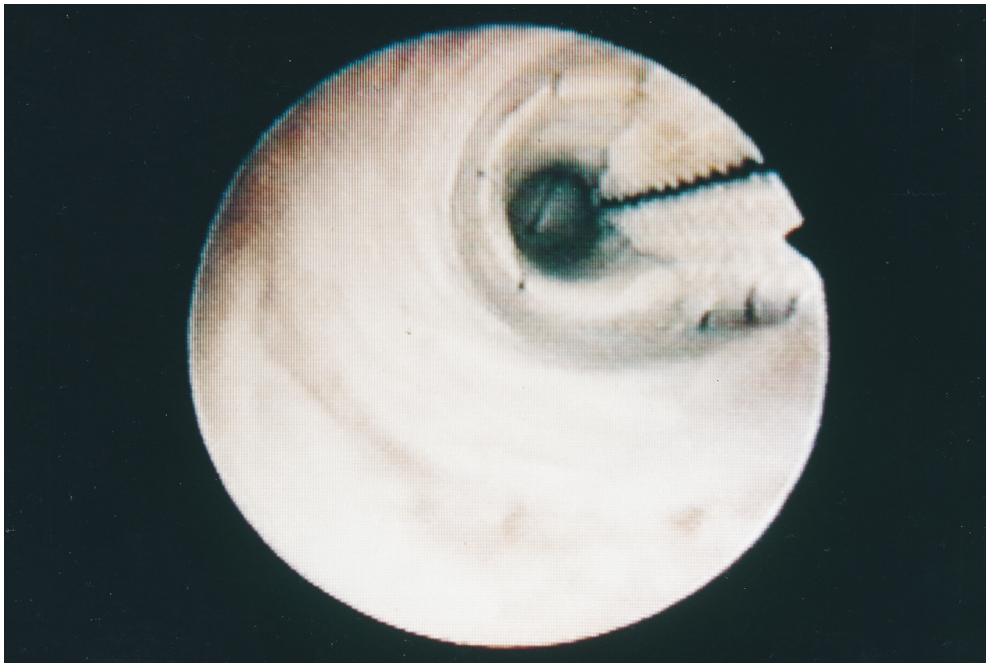

Fig. 1. Normal angioscopic assessment of the endarterectomy zone and distal internal carotid artery.

Fig. 2. Angioscopic assessment of the proximal common carotid artery. Note the presence of mural thrombus adherent to the intimal step.

Table 2. Angioscopy findings prior to restoration of flow.

Normal angioscopy	Intimal flap >3#mm	Luminal thrombus
226 (95%)	6 (2.5%)	6 (2.5%)

within 72 h) occurred in the postoperative period. The first followed thrombosis of the external carotid artery 5 h postoperatively. At re-exploration, there was a tongue of thrombus extending into the ICA which acted as a source of embolisation. This stroke again occurred prior to implementing our policy of postoperative TCD monitoring. A second patient was readmitted on day 7 with a classical hyperperfusion syndrome stroke (BP 240/160, MCAV 140 cm/s, normal Duplex, no intracranial occlusions on TCD, diffuse oedema on CT scan). The remaining patient was readmitted on day 9 with an ECG and enzyme proven MI (normal Duplex and TCD, focal MCA territory infarct on CT scan). In this patient we cannot reliably differentiate between a carotid or cardiac source for her focal embolisation.

Discussion

In order to develop a strategy for reducing the risk of stroke following carotid endarterectomy (CEA) it is important to audit underlying patterns of operative stroke with respect to timing and causation and thereafter use this data to design and implement a systematic approach to prevention and/or early therapeutic intervention.

Perioperative strokes can be divided into two subgroups. Intraoperative strokes (IOS) are apparent immediately following recovery from anaesthesia and must be attributable to some adverse event during the operation. Postoperative strokes can occur at any time after recovery from anaesthesia and usually follow thromboembolisation, hyperperfusion or haemorrhage.

Evidence suggests that the majority of perioperative strokes occur intraoperatively;[3] that patients presenting with a history of stroke, a residual neurological deficit, crescendo TIAs or who those with haemodynamic compromise or ipsilateral CT scan infarction are at particular risk of IOS[5], and that the principal underlying cause is usually inadvertent technical error.[4] Awareness of these factors led us subsequently to conclude that patients at highest risk of suffering an IOS were probably more vulnerable to the effects of hypoperfusion or microembolisation so that the margin for technical error was reduced or possibly non-existent.[12] The fact that despite his/her best efforts, the surgeon may be inadvertently responsible for causing an intraoperative stroke is an important concept to accept, but many surgeons are still reluctant to do so. Accordingly, few have adopted any strategy of QC assessment following carotid endarterectomy, despite the fact that most would undertake some form of completion assessment following femorodistal bypass!

In 1992, we undertook a pilot study to evaluate the role of QC assessment in reducing our rate of IOS which had previously been 4%. The study concluded that (for us) a combination of TCD and completion angioscopy fell to 1% by the end of the study.[6] However, although this study corroborated our own suspicions that experienced surgeons could be responsible for inadvertent technical error, there remains a degree of scepticism elsewhere as to whether QC assessment could ever reduce the rate of IOS long-term (i.e. not just during a pilot study) and whether it was feasible or practical to implement such a programme.

The current project therefore addressed these issues in a larger prospective study which was conducted 2 years after completion of the pilot study. During this 2-year period, the annual number of CEAs had increased seven-fold and more than 50% of CEAs were now being performed by vascular trainees under supervision.

The answer to the question as to whether QC assessment was associated with a sustained reduction in the rate of IOS appears to be yes. The rate of IOS, which was 4% prior to 1992, fell immediately to 1% during

Table 3. Perioperative neurological complications.

(i) Intraoperative strokes		
None		
(ii) Postoperative strokes and deaths		
Deaths ($n=3$)	Disabling stroke ($n=1$)	Non-disabling stroke ($n=3$)
Intracranial haemorrhage*	Unknown*†	External carotid thrombosis*
Intracranial haemorrhage*		Hyperperfusion stroke*
Intracranial haemorrhage		Postop MI/embolus

* Strokes which occurred prior to introducing policy of 6 h postoperative TCD monitoring.
† Stroke apparent following weaning from ventilator.

the pilot project and was 0% in the latest study. During the period of time concerned (1992–1996) the only consistent operative or technical factor that could have altered our rate of IOS was the introduction of routine QC assessment and any counter-argument that the improvement merely reflected increasing experience is unlikely given that the majority of our CEAs are currently performed by vascular trainees.[13] Although difficult to prove in the absence of a randomised trial, it is our opinion that the largest single factor in preventing IOS has been the identification and removal of residual luminal thrombus which accumulates on the highly thrombogenic endarterectomised surface during closure. In our own experience, the thrombus usually forms at the site of entry of the vasa vasorum into the endarterectomy zone.

The principle advantage of angioscopy is that it can be performed prior to restoration of flow so that intraluminal abnormalities (especially retained thrombus) can be corrected immediately and there is no requirement for technical support. In our own unit, the theatre staff set up the monitors and the surgeon performs the angioscopic assessment which in the latest study was successful in 94% of patients. Since we have now included vein grafts in the programme, the overall success rate should increase to about 97%. The original angioscope was, however, particularly vulnerable to fibre breakage and poor image quality, but this has been greatly improved by using the flexible hysteroscope. Image quality with the latter is excellent (Fig. 1) and the scope does not need to be advanced up the ICA in order to visualise the distal endpoint which could be a potential problem in small calibre arteries. Moreover, the instrument channel allows grasping forceps to remove adherent thrombus under direct vision if necessary. Some surgeons, however, remain concerned that an angioscopic assessment will unnecessarily prolong the procedure and could predispose towards haemodynamic stroke during the enforced period of carotid clamping. The latest study should therefore reassure those surgeons that this is not a problem. In our opinion, it is more likely that undetected luminal thrombus is more likely to cause an IOS than a minute or two of haemodynamic compromise whilst the angioscopic assessment is being performed.

In contrast to the ease of angioscopy, TCD can sometimes appear to be more trouble than it is worth. Surgeons (and anaesthetists) cite the need for technical support, inaccessible windows, delays to starting the operation, equipment getting in the way, bulky head probe systems, probe dislodgement and a widespread scepticism that TCD alters outcome in any tangible way. With experience, however, many of these irritating problems diminish and we were able to achieve continuous monitoring in 91% of our patients. A semicircular head-probe protection system rarely interferes with the operative field and as technicians become more experienced the incidence of inaccessible cranial windows reduces to about 6%, while the time taken to insonate the correct artery rapidly improves. In the future, some of the manpower problems could be overcome by designing centralised monitoring areas covering multiple theatres, involving the anaesthetists in the TCD monitoring, or have a vascular studies technician set up the equipment at the start of the operation and then leave to do other work.

From a practical viewpoint, we have found the TCD to be most useful in providing an early warning of unpredictable phenomena, whereupon the surgeon can take whatever action he/she feels appropriate to ensure the optimal outcome. For example, embolisation during the carotid dissection phase warns of unstable plaques and facilitates early distal ICA clamping,[14] whilst shunt malfunction (kinking, sustained embolisation, low flow) is immediately identified and corrected (exclusion of kinking, deflation of distal Pruitt shunt balloon or augmentation of blood pressure to improve shunt flow).

A further benefit from the QC programme has been the positive impact it has had upon training younger surgeons. It is always a salutary lesson to be conspicuously responsible for leaving residual luminal thrombus which would otherwise have embolised to the brain if left alone. Moreover, with increasing experience of TCD, we have found that it has been possible to modify our surgical technique and reduce the number of emboli detected during the operation by 40%.[15] Although it could be argued that the majority of emboli were gaseous and probably insignificant,[8] one must not overlook the potential clinical importance of micro-particulate emboli which may be associated with cognitive impairment[6] or the development of ischaemic lesions on MRI.[16] In 1992, the median number of particulate emboli detected during CEA was 21 (95% CI 16–29). By 1995, the median number of particulate emboli had fallen to nine per procedure (95% CI 7–14, $p = 0.00081$).[15]

In summary, the pilot study was implemented because of our concern at a 4% IOS rate during CEA prior to 1992. That study suggested that inadvertent technical error was probably the commonest underlying cause and that, for us, a combination of TCD and angioscopy provided the maximum yield in terms of identifying these and was associated with a fall in the rate of IOS to 1%. The present study has shown that

the ongoing discipline of a policy of QC assessment has been associated with a further reduction in the rate of IOS to 0% but that optimal enforcement of such a policy requires access to TCD technicians and reliable equipment.

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