

Transcranial Doppler detected cerebral microembolism following carotid endarterectomy

High microembolic signal loads predict postoperative cerebral ischaemia

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Summary

Cerebral ischaemia, the most frequent serious complication of carotid endarterectomy (CEA), usually occurs in the early postoperative period and is often the result of thromboembolism. We hypothesized that the early postoperative detection of microembolic ultrasonic signals (MES) with transcranial Doppler ultrasound (TCD) may be of value in identifying patients at risk of postoperative cerebral ischaemia and that the MES rate may be an important determinant in risk prediction. Sixty-five patients undergoing CEA were studied at intervals up to 24 h postoperatively with TCD insonation of the middle cerebral artery ipsilateral to the operation side. Study design was open and prospective with blinded off-line analysis of MES counts. End-points

were any focal ischaemic neurological deficit and/or death up to 30 days postoperatively. MES were detected in 69% of cases during the first hour postoperatively with counts ranging from 0 to 212 MES/h (mean 19 MES/h; SEM \pm 4.5; median 4 MES/h). In seven cases (10.8%) counts were >50 MES/h. Five of these seven cases developed ischaemic neurological deficits in the territory of the insonated middle cerebral artery during the monitoring period. The positive predictive value of counts >50 MES/h for cerebral ischaemia was 0.71. Frequent signals (>50 MES/h) occur in \sim 10% of cases in the early postoperative phase of CEA and are predictive for the development of ipsilateral focal cerebral ischaemia.

Keywords: microemboli; carotid endarterectomy; perioperative stroke

Abbreviations: CEA = carotid endarterectomy; MCA = middle cerebral artery; MES = microembolic ultrasonic signal; ICA = internal carotid artery; TCD = transcranial Doppler ultrasound; TIA = transient ischaemic attack

Introduction

Carotid endarterectomy (CEA) is of proven benefit in stroke prevention for patients with high grade symptomatic carotid artery stenosis (North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991; European Carotid Surgery Trialists' Collaborative Group, 1991). However, the overall benefits of surgery are critically dependent on perioperative morbidity and mortality. Strategies capable of reducing the risk of perioperative stroke have the potential to improve the risk/benefit ratio and cost-effectiveness of the operation. This is particularly relevant

where the margin of benefit from surgery is modest, as is the case in high grade asymptomatic carotid artery stenosis (Executive committee for the Asymptomatic Carotid Atherosclerosis Study, 1995).

Studies using transcranial Doppler ultrasound (TCD) to examine the occurrence of microembolic ultrasonic signals (MES) distal to high grade carotid artery stenoses suggest that clinically silent microembolism correlates with recent symptomatic cerebral ischaemia, carotid plaque ulceration and intraluminal thrombosis (Siebler *et al.*, 1994; Sitzer *et al.*,

1995; Valton *et al.*, 1995). In this setting it is postulated that MES may provide an indirect marker of carotid plaque instability and thereby predict the likelihood of clinically significant cerebral embolism. Prospective studies examining the risk of cerebral ischaemia associated with MES are, however, not yet available.

The frequency of MES has been shown to decline significantly when measured beyond day 4 post-CEA (Siebler *et al.*, 1993; Van Zuilen *et al.*, 1995). However, the natural history of microembolism in the early postoperative period has not been systematically studied. As perioperative stroke occurs most frequently during this early postoperative phase and thromboembolism is often implicated (Krul *et al.*, 1989; Riles *et al.*, 1994), early postoperative TCD monitoring may provide information relevant to perioperative stroke prevention. In support of this concept is the evidence from case reports of an association between early postoperative microembolism and cerebral ischaemic events (Spencer *et al.*, 1990; Gaunt *et al.*, 1994a, b).

We hypothesized that the detection of postoperative MES may be of value in the identification of patients at risk of postoperative embolic cerebral ischaemia and that the frequency of MES may be an important determinant in risk prediction. Based on the published case reports (Spencer *et al.*, 1990; Gaunt *et al.*, 1994a, b) and our own preliminary monitoring studies, counts of >50 MES/h were considered likely to be associated with the development of cerebral ischaemia.

To test these hypotheses we studied patients prospectively with postoperative TCD monitoring and clinical neurological assessments during the first 24 h following CEA. Clinical end-points were re-assessed at 30 days postoperatively.

Methods

Subjects

A total of 75 consecutive patients who underwent CEA with TCD monitoring were entered into this open prospective study over the period from November 1993 to October 1995. Sixty-five cases (87%) were successfully studied with postoperative TCD monitoring and 59 cases (79%) were successfully monitored both intraoperatively and postoperatively (six intraoperative technical failures). Signal quality was inadequate in eight patients because of poor temporal ultrasonic windows. CEA was abandoned as the internal carotid artery (ICA) was found to be thrombosed at operation in one patient and in the other case the ICA was ligated following an intraoperative distal intimal dissection and postoperative monitoring was not performed.

Patients enrolled in the study gave informed consent and were screened for operative suitability by the Neurovascular Service, Austin and Repatriation Medical Centre. The same six vascular surgeons performed all procedures. Preoperative digital subtraction angiography was used to delineate vascular anatomy in 63 cases, carotid duplex ultrasonography alone

in one case and CT angiography in one case. The study was approved by the Austin and Repatriation Medical Centre Ethics Committee.

Transcranial Doppler ultrasound monitoring

Thirty-minute monitoring sessions were performed at the 0–1 h phase, 2–3 h phase, 4–6 h phase and at 24 h post-operatively. Time zero was defined as completion of skin closure. The initial 17 cases underwent a single recording commencing during the first postoperative hour. The monitoring protocol was then extended and the subsequent 48 cases underwent serial postoperative recordings.

Two megahertz pulsed wave TCD systems (EME-Nicolet TC 2–64B, EME-Nicolet TC 2020, EME-Nicolet, Uberlingen, Germany or DWL MultiDop T, DWL Elektronische Systeme GmbH, Sipplingen, Germany) were used to insonate the MCA (insonation depth 45–55 mm) ipsilateral to the operation side. Power output and gain settings were adjusted to provide an optimal signal-to-noise ratio. The sample volume length was varied between 10 and 15 mm depending on signal quality. The instrument's maximum sweep speed was used for on-line MES counting. An elasticized headband was used to fix the probe in position.

Doppler signal recording and analysis

The analogue Doppler signal was recorded continuously on digital audio tape (Sony TCD-D10 Pro II, Sony Corporation) and gain settings adjusted to provide a constant signal recording level within and between each recording. The MES counting for analysis was performed at an off-line review of the Doppler recordings (EME-Nicolet TC 2020; sweep duration 5.1 s). An intensity threshold of >6 dB above peak background intensity was used for off-line reporting of MES in conjunction with previously defined signal criteria (Consensus Committee of the Ninth International Cerebral Haemodynamic Symposium, 1995). Intensities were estimated after saving the spectral display (128 point fast Fourier transformation) to hard disk (EME-Nicolet TC 2020). Measurement of the peak intensity of the MES was performed by visually thresholding the intensity of the spectral display in 1 dB increments using the instrument gain-setting adjustments. Intensities were averaged for each embolic signal over three replays of the tape. Measurement of the peak background intensity at the same point in either the preceding or following cardiac cycle was performed similarly. Only signals assessed as 'definite' and fulfilling the criteria at off-line review (CL) were accepted and reported as embolic signals (Fig. 1).

Operative and medical treatment details

Fifty-seven cases underwent standard CEA (six under general anaesthesia) and eight cases underwent eversion CEA (six under general anaesthesia). A common carotid to internal

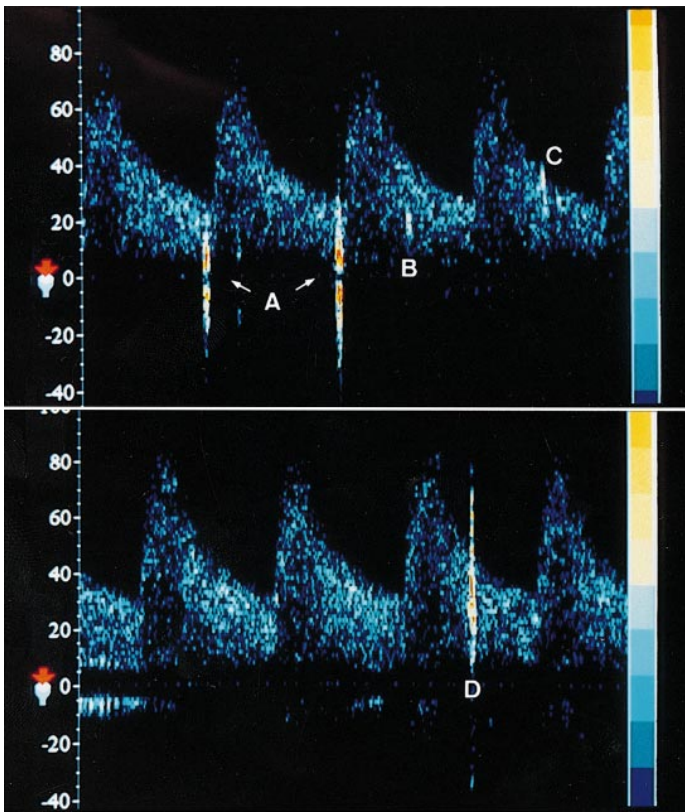


Fig. 1 Postoperative transcranial Doppler blood flow velocity spectra (128 fast Fourier transformation, sweep 5.1 s, signal intensity shown by colour scale) reproduced off-line from digital audio tape recording. This patient (Case 43) developed early postoperative transient ischaemia. Examples of MESs and a probe movement artifact are illustrated. Probe movement typically results in low frequency high intensity signals that are bi-directional and centred on the zero line (A). The MESs are typically of higher frequency, are located within the spectral display and have a characteristic auditory signature (B, C and D). MESs of higher intensity can exceed the dynamic range of the instrument resulting in extraspectral artifact (D).

carotid artery shunt was inserted in 48 of the cases undergoing standard CEA and in two of the eversion endarterectomy cases. Patch angioplasty was performed in 24 cases (37%). All patients received aspirin perioperatively and all cases received an intraoperative bolus dose of heparin. Protamine sulphate was given following arteriotomy closure in 36 cases (55%). In the event of the development of a clinical outcome event during the first 30 days postoperatively, the management options pursued were at the discretion of the treating surgeon and neurologist and included either operative re-exploration, intravenous heparinization and/or infusion of 10% dextran 40.

Clinical and radiological assessments

All patients underwent detailed preoperative neurological examination and serial detailed postoperative clinical assessments immediately following each TCD monitoring

session (four assessments within the first 24 h). A further clinical assessment was performed at 30 days postoperatively. Postoperative cerebral ischaemia was defined as the occurrence of new focal neurological signs following a normal or unchanged initial recovery room neurological assessment. Particular attention was given to new signs of cortical dysfunction including visual or tactile inattention, dysphasia, limb drift and impairment of fine finger movements. The clinical outcomes assessed prospectively were death, postoperative ischaemic stroke or witnessed transient ischaemic attack (TIA) within 30 days of surgery.

In 48 cases hard copies of intraoperative angiograms were available for review. Reporting was performed blind to other information. In selected cases with high embolic signal counts postoperative neuroimaging and carotid duplex ultrasonography were performed.

Statistical methods

MES counts are represented as mean and SEM values, or median values with interquartile ranges. Univariate analysis of embolic signal loads in different groups and for different outcomes were made using the χ^2 test with significance set at $P = 0.05$. Performance of multivariate analysis was not possible as only a single patient had an outcome event not associated with MES. Correlation between on-line and off-line MES reporting by a single observer (C.R.L.) during the first postoperative monitoring session was assessed using a Spearman rank correlation test.

A proportion of agreement (Fleiss, 1981; Markus *et al.*, 1996) was calculated to assess inter-observer agreement for MES counting at the same point in time by three observers (C.R.L., C.F.B. and H.M.O.; 60 min of blinded, independent tape review). Proportion of agreement was also calculated to assess test versus re-test agreement for MES counts reported at the same point in time by a single observer (CL, 60 min of blinded, off-line tape review, counting sessions separated by 2 weeks). The proportion of agreement measures the probability that a second or third observer of the same MES will register the same result as the first observer. This method was chosen as observation points at which no MES were counted could not be compared prospectively as reviewers remained blinded to the reports of the other observers. The absence of prospectively registered time points at which all observers detected no MES does not permit the use of Cohen's kappa statistics. In addition to a classification of 'definite' MES, a category of 'possible' MES was included in off-line counting for inter-observer agreement. Possible MES were defined as those with suggestive features but intensity <6 dB above peak background intensity or those with atypical audio-visual features.

Results

The indications for surgery, and the patients' angiographic status and demographic details are outlined in Table 1.

Table 1 Clinical, angiographic and risk factor characteristics of the patient group

	<i>n</i>	%
Clinical presentation		
Transient cerebral/retinal ischaemia	38	58
Ischaemic stroke	11	17
Asymptomatic	16	25
Angiographic stenosis		
<70%	4	6
70–79%	8	13
80–89%	12	19
90–99%	39	62
Risk factors		
Hypertension	43	66
Current smoking	13	20
Prior smoking (<10 years)	24	37
Peripheral vascular disease	21	32
Ischaemic heart disease	26	40
Diabetes mellitis	6	9
Atrial fibrillation	2	3

For the patient group, $n = 65$ (50 males and 15 females, mean age 68 years, range 44–85 years). Intra-arterial digital subtraction angiography was performed in 63 of 65 cases. The degree of stenosis was determined using the North American Symptomatic Carotid Endarterectomy Trial (1991a) measurement system (NASCET). CEA was undertaken on the four patients with <70% angiographic carotid artery stenosis following randomization to the surgical arm of the 30–69% stenosis NASCET.

Transient cerebral or retinal ischaemia preceded surgery in 58% of cases. Seventeen percent of patients had had a preceding ischaemic stroke and 25% of cases were asymptomatic. The majority of cases (62%) had 90–99% ipsilateral carotid artery stenosis using the North American symptomatic carotid endarterectomy trial measurement system (Steering Committee of North American Symptomatic Carotid Endarterectomy Trial Collaborators, 1991).

Postoperative MES and clinical outcomes

At the 30 day assessment there was one major ischaemic stroke and three minor ischaemic strokes (overall stroke rate 6.1%; major stroke rate 1.5%). All cerebral ischaemic events were ipsilateral to the CEA. The major stroke was associated with prolonged MCA signal loss after shunt insertion. A further three cases developed transient ischaemic deficits in the recovery room with clinical signs lasting <4 h. There was one sudden cardiac death on day 2 postoperatively (death rate 1.6%).

The median values, ranges and quartile rankings for the postoperative MES counts at each phase are shown in Fig. 2. MES counts for each patient at the 0–1 h postoperative phase are shown in Fig. 3. A strong association was evident between counts of >50 MES/h during the 0–1 h phase (seven cases) and the development of ischaemic stroke ($n = 3$) and TIA ($n = 2$) (total of five cases, $\chi^2 = 45.9$, $P < 0.0001$). The positive predictive value for cerebral ischaemia of detecting counts of >50

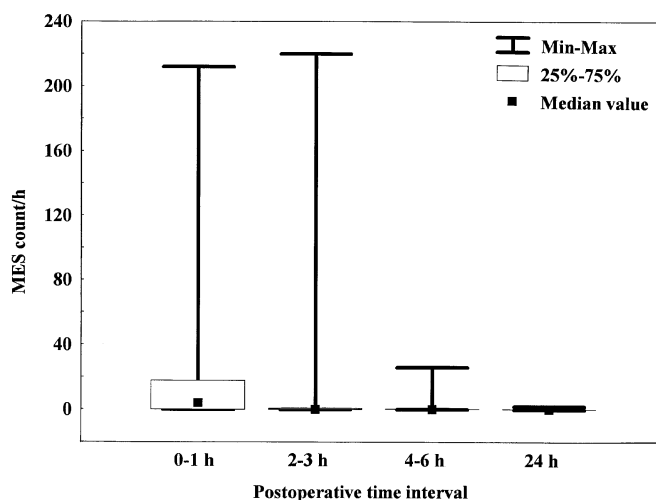


Fig. 2 This plot shows the range of MES counts detected during each postoperative time period. MESs were counted at off-line review of Doppler recordings. The minimum–maximum counts (bars), the 50% interquartile range (open box) and the median counts (filled square) are shown for each phase. Sixty-five patients are included at the 0–1 h phase, and 48 patients in each of the other phases.

MES/h was 0.71, and the negative predictive value for counts of <50 MES/h was 0.98.

The clinical signs in the five patients who developed focal ischaemic deficits were evident at the detailed neurological examination following the initial 30 min TCD monitoring session. In two of the three cases of stroke, specific questioning and observations suggested focal neurological dysfunction was developing during the monitoring period. A gradual worsening of the neurological deficit was noted over 30 min to 1 h following its initial documentation in the three stroke cases. At this stage interventions were commenced. One of the patients was returned to the operating theatre and at arteriotomy dense platelet clumps adherent to the exposed subintimal surface were removed, no imperfection in the ICA was noted and patch angioplasty performed. The other high MES-load cases were treated with full dose intravenous heparin, 10% dextran 40 or a combination of these two therapies. The MES rates were noted to return to <20 MES/h at 1–2 h post-intervention. No significant changes were noted in blood flow velocities or pulsatility indices in the MCA carrying frequent MES. Twenty-four hour postoperative carotid duplex ultrasound scanning in three cases did not identify thrombus on the endarterectomy bed.

High MES loads were not associated with requirement for patch angioplasty ($P = 0.89$) or the use of protamine for reversal of intraoperative heparin therapy ($P = 0.69$). Operative angiographic results were available in six of the seven cases with frequent postoperative MES. A suboptimal operative result defined as a postoperative stenosis of >30% with the European carotid surgery trial stenosis measurement system (European Carotid Surgery Trialists' Collaborative Group, 1991) was associated with a trend towards the subsequent occurrence of counts of >50 MES/h ($\chi^2 = 3.3$,

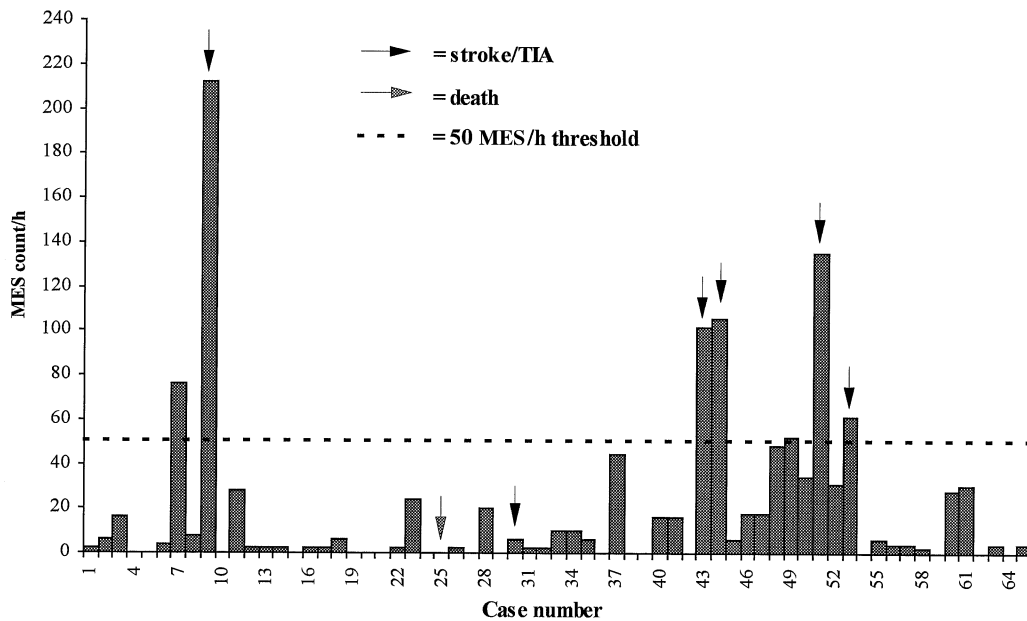


Fig. 3 This histogram shows MES counts and clinical outcomes (patients arrowed) during the 0–1 h postoperative phase for each of the 65 patients. MES were counted at off-line review of Doppler recordings blind to clinical outcomes. The hypothetical pathogenic threshold of 50 MES/h is shown.

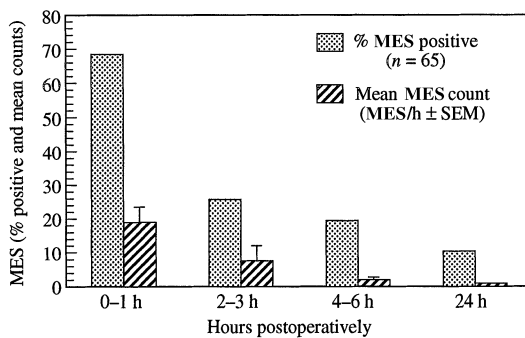


Fig. 4 This histogram shows the proportion of patients who were MES positive (left column) and the mean MES counts with standard errors (right column) at each postoperative phase ($n = 48$ –65).

$P = 0.07$). Female sex was significantly associated with high MES loads ($\chi^2 = 5.13$, $P = 0.023$).

The proportion of MES-positive patients and the mean MES counts at each postoperative phase are shown in Fig. 4. MES were most prevalent in the period immediately following the return of the patient to the recovery room (69% MES positive). In the patients with >50 MES/h, the signal events were random in occurrence and spread throughout the 30-min monitoring period with no apparent clustering. The proportion of positive cases declined to 25% at 2–3 h (one case only with a count of >50 MES/h), 19% at 4–6 h (no cases of >50 MES/h) and 10% at 24 h (no cases of >50 MES/h). The mean MES count (\pm SEM) was 19 ± 4.5 MES/h in the 0–1 h postoperative phase falling to 0.2 ± 0.09 MES/h at 24 h.

In 16 of 65 cases (25%) no embolic signals were detected at any stage during the first 24 h postoperatively. One major ischaemic stroke occurred in this patient group in association

with prolonged MCA signal loss following shunting. In this case hemiparesis was evident on the patient regaining consciousness postoperatively.

Postoperative brain imaging

Two minor stroke cases and two TIA cases associated with high postoperative MES counts underwent pre- and postoperative brain CT and/or MRI. MRI demonstrated new ischaemic lesions in the corona radiata in one of the two patients with transient ischaemia. In both of the cases of ischaemic stroke, infarction was seen in the external watershed zone on CT and MRI (Fig. 5).

Reliability of MES reporting

The correlation between the total MES counts on-line versus off-line in 48 patients during the first postoperative hour was excellent, with $r = 0.99$. The test versus re-test agreement for definite MES reported at the same points in time was 0.80. The results of counting definite or possible MES reported at the same point in time by the three independent observers are summarized in Table 2. The proportion of agreement between these three observers for definite MES occurring at the same points in time was 0.83.

Discussion

Intraoperative high risk time periods for gaseous and particulate brain embolism have been identified with TCD monitoring (Spencer *et al.*, 1990; Jansen *et al.*, 1993; Gaunt *et al.*, 1994a; Jansen *et al.*, 1994; Gavrilescu *et al.*, 1995)

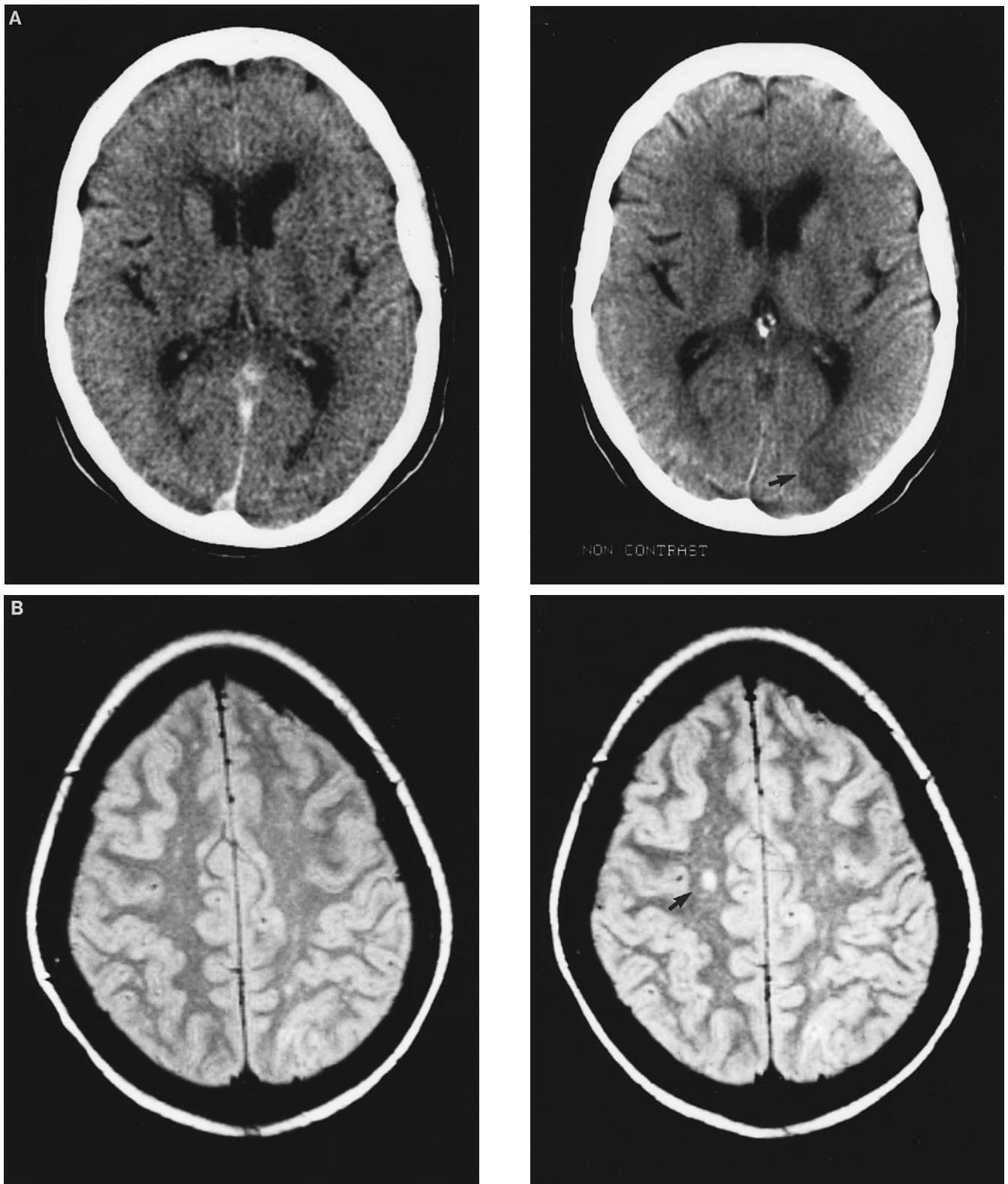


Fig. 5 (A) Preoperative (*left*) and postoperative (*right*) CT brain scans showing the development of infarction in the watershed zone between middle cerebral and posterior cerebral arterial territories of the left hemisphere (arrow). This patient (Case 51) developed expressive dysphasia and right hemiparesis 1 h postoperatively following detection of 136 MES/h. The patient made a complete motor recovery with residual dyslexia only at day 30 postoperatively. (B) Preoperative (*left*) and postoperative (*right*) proton density MRI brain scans showing the development of infarction in the subcortical white matter of the right hemisphere (arrow). This patient (Case 43) developed transient left upper limb weakness following the detection of 82 MESs in the first postoperative hour.

Table 2 Inter-observer agreement on MESs counted at identical time points

Observer			Total MESs (A, B and C)
A	B	C	
+	+	+	40
+	+	(+)	3
+	+	-	4
+	(+)	+	1
+	(+)	-	1
+	-	+	3
+	-	(+)	1
+	-	-	3
(+)	+	-	1
(+)	(+)	-	1
(+)	-	-	2
-	+	-	1
Total			61

Classification of MES: '+' indicates definite MES(s) counted by observer; '(+)' indicates possible MES(s) counted by observer; '-' indicates no MES counted by observer. Summary of the agreement between three independent, blinded observers for definite post-CEA MESs (typical audio-visual signal with intensity ≥ 6 dB above peak background intensity) or possible post-CEA MESs (typical audio-visual signal with intensity < 6 dB above peak background intensity or atypical audio-visual signal) counted at the same point in time during 60 min of off-line tape review. The overall proportion of agreement between observers for definite MESs is 0.83.

and an association between MES and the development of postoperative neuropsychological impairment has been reported (Gaunt *et al.*, 1994a). It has also been noted that the occurrence of > 10 MES during surgical exposure of the carotid artery is associated with the development of new ischaemic lesions on MRI (Ackerstaff *et al.*, 1995).

Spencer (1990) first observed that cerebral ischaemia complicating CEA may be associated with frequent postoperative MES. In his series of 91 patients, two were monitored with TCD postoperatively, both developed frequent MES and both subsequently developed early postoperative cerebral infarction. Recent reports identify a further three patients with cerebral ischaemia associated with postoperative MES (Gaunt *et al.*, 1994a, b). We aimed to examine in more detail the clinical relevance and natural history of postoperative MES.

Our results demonstrate that postoperative counts of > 50 MES/h are significantly associated with and predictive of the acute development of focal cerebral ischaemia. The positive predictive value of high MES counts and the delayed and gradual development of focal ischaemic signs encountered in our patients suggest embolus detection may have a role in the identification of patients at high risk of postoperative stroke.

Counts of > 50 MES/h occurred most often during the first hour postoperatively and were detected in 10.8% of patients overall. The incidence and frequency of MES declined

progressively after the first postoperative hour in the 48 patients monitored serially with only one patient developing high MES counts during the second monitoring period. Of the 17 patients who underwent only the 0–1 h postoperative embolus detection monitoring, one developed evidence of cerebral ischaemia; however, all others were neurologically unchanged at serial clinical assessment. Given the consistency of the observed trend for MES counts to decline with time, it is unlikely that these cases developed unrecognized high MES rates at later stages. In no cases were MES detected at a frequency of < 50 MES/h associated with the development of clinical evidence of cerebral ischaemia (negative predictive value = 98%); however, measures of subclinical ischaemia detection such as pre- and postoperative MRI were not systematically used in this study. No cases developed frequent MES after 3 h postoperatively, suggesting the 0–3 h time period is the important epoch in which to monitor for this specific variety of carotid thromboembolism. The positive predictive value for cerebral ischaemia of detecting counts of > 50 MES/h was 0.71, and the negative predictive value for counts of < 50 MES/h was 0.98.

The topography of the infarction demonstrated in the high MES-load cases that underwent postoperative neuroimaging supports earlier observations that watershed infarction can be the result of microembolism (Torvik and Skullerud, 1982; Torvik, 1984; Pollanen and Deck, 1989). Although cerebral hypoperfusion is the usual cause of watershed infarction (Brierley *et al.*, 1980) evidence that glass microspheres of 150–210 μm diameter distribute preferentially to the watershed zones when perfused into the cadaveric brain suggests that the border zones may be selectively vulnerable to ischaemic injury from particles in this size range (Pollanen and Deck, 1990). The lack of clinical evidence of cerebral ischaemia in the majority of patients with TCD detected microembolism implies that infrequent microemboli usually do not exceed the reserve capacities of the microcirculation. Our evidence suggests, however, that higher rates of microthromboembolism can result in a gradual mode of onset of focal ischaemic signs, presumably the result of failure of the microcirculation caused by frequent microembolic occlusions. The gradual mode of onset of cerebral ischaemia associated with platelet microembolism is supported by observations of a latency of up to 20 min in the development of neurophysiological evidence of cerebral ischaemia in a baboon model where radioactively labelled platelet microemboli were generated from an extracorporeal dacron graft (Kessler *et al.*, 1992). Although these microemboli were detected in the extracranial carotid artery using Doppler ultrasound, MES rates were not reported.

Our hypothesis that rates of MES may influence the development of clinical signs of cerebral ischaemia is supported by the strong association between rates exceeding 50 MES/h and focal ischaemic signs. We acknowledge however, that the hypothetical 50 MES/h 'threshold' is based on very limited evidence (Spencer *et al.*, 1990; Gaunt *et al.*, 1994a, b) and the existence of such a threshold remains

speculative. Of the patients in whom frequent MES were associated with cerebral ischaemia, four of five demonstrated rates >100 MES/h. It is likely therefore, that the microcirculation has considerable reserve capacity for platelet microembolism and that the capacity may be influenced by variables such as microembolus size, blood rheology, collateral flow and native anti-coagulant and thrombolytic pathway activities in addition to microembolism rates.

Assumptions as to the nature and the source of embolism in this study group are based on the evidence from the one case that underwent operative re-exploration. The endarterectomy site was implicated and the cases reported by Gaunt *et al.* (1994a, b) also support this view. Although surgical imperfection is a potential cause for early thrombosis on the endarterectomized surface, a suboptimal operative angiographic result was only noted in one of the seven cases with high microembolic loads. Possible causative factors such as increased thrombogenicity from a deeper endarterectomy plane or heightened platelet aggregability in these patients remains to be explored. Surgical or medical intervention and possible modification of MES rates after the detection of focal neurological signs was ethically necessary. As the five patients concerned had reached a clinical end-point, this did not influence the principle results of the study.

When focal neurological signs did develop in this series they were quite obvious (three cases with a fixed neurological deficit). However, the signs that developed in the cases of transient ischaemia tended to be more subtle and would have been overlooked had it not been for the protocol requirement that all patients were assessed prospectively at frequent intervals over the first 24 h by a clinician experienced in the neurological examination. The importance of audit methodology has been recently emphasized in a meta-analysis of reports of morbidity and mortality of surgery for symptomatic carotid artery stenosis (Rothwell and Warlow, 1995; Rothwell *et al.*, 1996). The authors estimated a stroke and death rate of 7.7% where postoperative assessment was performed by a neurologist (nine studies, 2605 operations) compared with 2.3% where assessment was by a single surgeon (five studies, 1849 operations). It is likely that a proportion of the ischaemic neurological events identified in our series may not have been identified by a surgical audit at 30 days postoperatively. Moreover, the MRI evidence of new ischaemic injury in one of the two TIA cases imaged suggests that silent perioperative infarction may be significantly underestimated (Vanninen *et al.*, 1996).

The agreement between the three independent observers on definite MES counted at the same points in time (0.83) is slightly inferior to the 0.90 reported by Markus *et al.* (1996). However, the majority of MES were agreed upon and a discrepancy rate of 0.17 would not have significantly effected the conclusions of the study.

Recovery room TCD monitoring in the early postoperative course of carotid endarterectomy has the potential to identify a population at high risk of embolic stroke. Further studies

in this setting to examine preventive strategies and interventions are required.

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