Seizures After Carotid Endarterectomy: Hyperperfusion, Dysautoregulation or Hypertensive Encephalopathy?

A. R. Naylor[*][1], **J. Evans**[1], **M. M. Thompson**[1], **N. J. M. London**[1], **R. J. Abbott**[2], **G. Cherryman**[3] **and P. R. F. Bell**[1]

The Departments of [1]Vascular Surgery, [2]Neurology and [3]Radiology, Leicester Royal Infirmary, Leicester, U.K.

Objectives: *presentation, management and outcome following seizure after carotid endarterectomy (CEA).*
Materials and Design: *prospective audit.*
Results: *Eight patients (0.8%) suffered a seizure (three bilateral) <30 days following 949 CEAs. Seizure was not associated with age, gender or presentation. Seven were treated hypertensives but four had labile BP pre-operatively. Five had severe bilateral carotid disease and four had vertebral/subclavian stenoses. Six had a >50% drop in middle cerebral artery blood flow velocity (MCAV) with clamping. Only three had >100% increase in MCAV with flow restoration. Five required treatment for post-operative hypertension. Two suffered seizures <36 hrs of CEA, the remainder were at 3–8 days. All eight had significantly elevated blood pressure at onset of seizures. Four underwent immediate MCAV monitoring and each was elevated. Emergency CT scanning/autopsy showed normal scans (n = 3), white matter oedema (n = 3), oedema and diffuse haemorrhage (n = 1), intracranial haemorrhage (n = 1). Seven developed a post-ictal neurological deficit (stroke = 5, TIA = 2). Overall, two patients either died or suffered a disabling stroke.*
Conclusions: *post-CEA seizure was associated with adverse outcome. Most were labile hypertensives with severe bilateral carotid/vertebral disease. MCAV changes suggested poor collateral recruitment, but no consistent pattern of early hyperperfusion emerged. It remains uncertain whether high MCAVs and severe hypertension after seizure onset are cause or effect. Clinicians treating these patients in acute medical units were generally unaware of the "post-CEA hyperperfusion syndrome" and tended to treat the hypertension less aggressively.*

Key Words: *Seizures; Carotid endarterectomy; Hyperperfusion; Hypertensive encephalopathy.*

Introduction

In 1981, Thoralf Sundt[1] described a series of patients with varying combinations of headache, seizure and focal neurological signs within days of a successful carotid endarterectomy (CEA). The term hyperperfusion syndrome (HS) has thereafter been used to describe this phenomenon, although a uniformly agreed definition still remains elusive. It is hypothesized that large increases in cerebral blood flow, possibly due to impaired cerebral autoregulation in the early post-operative period, contributes towards a cascade of intracranial microcirculatory changes. These culminate in seizure or, in severe cases, a neurological deficit that may be haemorrhagic or ischaemic.[2,3]

However, not all patients with symptoms considered "typical" of HS have elevated cerebral blood flow.[4] Moreover, there has been surprisingly little attention directed towards understanding the pathophysiology of the grossly elevated blood pressure frequently observed in patients labelled as HS (is this cause or effect?). Hypertensive encephalopathy (HE) is a well recognized cardiovascular phenomenon characterised by rapidly evolving signs and symptoms that include headache, seizure, confusion and either ischaemic or haemorrhagic stroke.[5] Evidence increasingly suggests that both phenomena may be closely inter-linked in a common clinical pathway.

The current audit evaluated patients suffering seizure after CEA. The aim was to correlate clinical and other demographic factors with particular emphasis on the relationship between "hypertension" and "hyperperfusion".

Materials and Methods

A prospective audit identified 8 patients suffering seizure (sudden onset of involuntary tonic or clonic

---

*Please address all correspondence to: A. R. Naylor, Consultant Vascular Surgeon and Honorary Reader in Surgery, Robert Kilpatrick Clinical Sciences Building, Leicester Royal Infirmary, Leicester, U.K.

muscular contractions) within 30 days of 949 CEAs in the vascular unit at Leicester Royal Infirmary between 01.01.1995 and 30.06.02. Case notes and CT scans were thereafter reviewed to extract demographic and outcome data. Patients suffering seizure *after* a post-operative stroke were excluded.

Pre-operative assessment

Patients were seen in a single-visit clinic where risk factor assessment and duplex examination were performed. Unit policy was to avoid elective CEA in patients with uncontrolled hypertension (systolic blood pressure >180 mmHg).

Peri-operative management

CEAs were performed under normocarbic, normotensive general anaesthesia with transcranial Doppler (TCD) monitoring, routine shunting, patching and completion angioscopy. From 01.10.1995, all patients underwent 3 h of post-operative TCD monitoring to guide selective Dextran therapy in those with high-grade embolisation.[6] Middle cerebral artery blood flow velocity (MCAV) was recorded onto digital audiotape for off-line analysis.

The management of post-operative hypertension (thresholds, choice of therapy) was at the discretion of the surgeon and anaesthetist. The "aim" was to maintain systolic blood pressure within 10% of pre-operative levels while avoiding sustained pressures >160 mmHg. Medications were resumed on the evening of surgery. Between 1995–1999, most patients were discharged on day 3. After 2000, the majority were discharged on day 1 or 2. All survivors were seen for outpatient review at 30 days.

Management of post-op-operative seizures

The management of seizure after CEA depended on the mode of presentation. Those notified from neighbouring hospitals were instructed to control the seizure/hypertension and return the patient to the vascular unit as soon as possible. Clinicians admitting patients to the emergency medical units in the three Leicester hospitals were asked to inform the on-call vascular team immediately. Seizures were usually controlled with titrated boluses of intravenous Diazepam. A Guedel airway optimised oxygenation. TCD monitoring was performed (where possible) to quantify MCAV and exclude ongoing embolisation.

All patients underwent duplex examination to confirm patency of the operated ICA. Severe hypertension was reduced using titrated boluses of Labetolol and continued as an infusion where necessary. Alternative anti-hypertensive regimes were thereafter employed upon advice from the intensive care unit where all patients were nursed. Three intravenous doses of 8 mg Dexamethasone were administered empirically to "minimise" cerebral oedema. Each patient underwent a CT scan as soon as possible after admission/onset of seizures to exclude intracranial haemorrhage. A neurologist (RJA) reviewed all CEA patients suffering a seizure. The CT scans were independently reassessed by a consultant radiologist (GC).

Results

Seizures (three bilateral and five unilateral (i.e. contralateral to the side of operation)) affected all age groups, gender and modes of presentation. All bar one was on treatment for hypertension. Each patient had a normal blood pressure documented in outpatients, but four had an admission BP that would not ordinarily be classified as "controlled" (cases 2, 3, 5, 6; Table 1). Five of the eight had severe bilateral carotid disease (>70%/occlusion). Only one patient had a unilateral carotid stenosis. Four also had unilateral/bilateral subclavian or vertebral disease (cases 1, 3, 4, 6; Table 1).

Clamp times ranged from 4 to 11 min, shunt times were 29 to 87 min. Six had a mean MCAV <15 cm/s during clamping, including two with no detectable flow (Table 1). All had an MCAV >15 cm/s during shunting consistent with flows in excess of the threshold for loss of cerebral electrical activity.[7] Six had a >50% fall in MCAV following clamping (Table 2). Mean MCAV increased by more than 100% in three patients (pre-clamp vs restoration of flow MCAV (cases 1, 5, 8; Table 2). Only three had a >100% increase in MCAV at 3-h (cases 2, 5, 8; Table 3).

Five patients destined to suffer seizures required anti-hypertensive therapy in the post-operative period. Table 1 summarises the maximum daily systolic pressure in the early post-operative period. In retrospect, case 2 was allowed home too early (day 1) before his blood pressure was adequately controlled.

Table 3 summarises management and outcome after seizure. Six patients developed focal neurological symptoms and signs *after* the onset of the seizures (ipsilateral motor/sensory = 5, dysphasia = 1). Duplex confirmed ICA patency with no evidence of any technical defect. The first recorded blood pressure after onset of the seizure (whether on the ward or in the

Table 1. Peri-operative patient data.

	Age/ sex	Clin pres	HT Rx	BPOA mmHg	Carotid stenosis OP/NON-OP	Vertebral/ subclavian	Clamp/shunt Time (mins)	Mean MCAV (cm/s)				HT Rx recovery	Daily systolic BP		
								Pre	Clamp	ROF	3 h		1	2	3
1	44F	TIA	Y(+)	145/85	95%/90%	Subclavian occ	11/87	34	21	84	n/a	Y	140	150	130
2	62M	TIA	Y(+)	190/90	90%/OCC	Normal	8/43	29	14	33	88	Y	200		
3	78M	TIA	Y	210/110	90%/90%	Unilat vert occ	4/38	37	0	59	28	N	170	170	160
4	59F	TIA	Y	140/80	95%/60%	Bilat vert sten	5/42	86	10	105	126	N	187	176	190
5	64F	TIA	Y	200/98	95%/OCC	Normal	5/44	33	10	89	74	N	155		
6	70M	ASY	Y(+)	185/104	90%/OCC	Unilat vert sten	5/29	41	0	41	30	Y	140	175	190
7	74M	ASY	N	134/64	95%/0%	Normal	5/39	41	26	58	79	Y	145	170	
8	75F	CVA	Y(+)	125/75	90%/0%	Normal	7/45	16	6	57	67	Y	120	130	140

clin pres = clinical presentation, TIA = transient ischaemic attack, CVA = stroke, ASY = asymptomatic. BPOA = blood pressure on admission (+) indicates if a physician was involved in antihypertensive therapy, MCAV = mean middle cerebral artery blood flow velocity in cm/s, Key to MCAV data: pre = pre-clamp MCAV, clamp = MCAV immediately following clamping, ROF = MCAV immediately following restoration of flow, 3 h = MCAV 3 h post restoration of flow. HTRx = patients requiring anti-hypertensive therapy in the first 3 post-operative hours, n/a = not available.

Table 2. Percent change in mean MCAV (compared to pre-clamp values).

Case	% fall with clamping	% increase with restoration of flow	% increase 3 h after restoration of flow
1	−38	+147	n/a *
2	−52	+14	+200
3	−100	+59	−24
4	−88	+22	+47
5	−70	+170	+124
6	−100	+0	−27
7	−37	+41	+93
8	−63	+256	+318

* n/a = no data available.

referring hospital) was elevated in six patients (cases 1, 2, 5, 6, 7, 8) and borderline in two. Systolic and mean MCAV were measured in four patients *prior* to reducing blood pressure (cases 1, 2, 7, 8). Mean MCAV was >200% higher than pre-clamp values in four patients, >300% in two and >400% in one.

CT scans were performed within 6 h of seizure onset apart from case 3 (Table 3). Three were reported as normal following independent review for this study (cases 1, 2, 5), while four had varying degrees of white matter oedema in the anterior and posterior circulations (cases 4, 6, 7, 8). Case 4 had evidence of white matter oedema in the right posterior circulation and a petechial haemorrhage in the right frontal region (Fig. 1). Case 7 developed seizures 32 h after an uneventful CEA. The seizures were followed 10 min later by dysphasia then a mild hemiparesis. Twenty minutes later became rapidly hemiplegic and obtunded. The CT scan (performed 40 min after onset of the seizures) revealed extensive oedema throughout the right anterior circulation territory with diffuse haemorrhage (Fig. 2).

Outcomes are detailed in Table 3. Only one patient had no further seizures and no neurological deficit

(case 2). Three patients made a full neurological recovery within 24 h (cases 4, 5, 8) and were classed as having suffered a "TIA". Two patients (cases 1 and 6, Table 3) suffered minor, non-disabling strokes. Overall, five of the eight patients in this series died (n = 1) or suffered a non-fatal stroke (n = 4). Two patients died or suffered a disabling stroke (cases 3 and 7, Table 3).

The most important practical observation from this audit was the fact that medical colleagues tended to be unaware of the post-CEA hyperperfusion syndrome and its sequelae. Case 3 was admitted to the emergency medical unit at the Infirmary with seizures seven days after CEA. These were treated and the patient discharged the following morning without informing the vascular team. He was readmitted later the same day with an ultimately fatal intracerebral haemorrhage. Cases 1 and 8 were only referred from peripheral hospitals to the vascular unit once the families contacted the on-call vascular team. Case 1 had gross hypertension (220/170 mmHg) and the clinicians were reluctant to treat the hypertension aggressively in case it aggravated the neurological deficit. Case 8 suffered a seizure followed by onset of expressive dysphasia 17 h after CEA, whilst still on the ward. The dysphasia recovered within 24 h and she was kept in hospital until the seventh post-operative day. By the time of discharge, she had been normotensive with normal MCAV for four days. However, within 12 h of discharge, she developed status epilepticus (but no significant hypertension) and was admitted to her district general hospital. By the time of transfer back to the vascular unit, she was hemiparetic.

Discussion

In 1981, Sundt suggested there might be a causal/ synergistic link between HS and HE.[1] However,

Table 3. Clinical parameters and outcome after onset of seizures.

	Seizure	Neuro deficit	BPOA mmHg	MCAVOA peak/ mean cm/s	Admitted to	CT scan	Outcome
1	4 days*	Hemiparesis	220/170	140/116	Other med	Normal	Hemiparesis recovered <72 h. OHS = 0 at 30 days
2	5 days*	None	206/97	170/93	Vasc unit	Normal	No recurrent seizures, no neurological deficit
3	7 days	None	170/90	n/a	Med unit	Not done	Discharged next day, fatal ICH 26 h later
4	5 days	Hemiparesis	180/80	n/a	On ward	PCO + APH	Hemiparesis recovered <24 h
5	8 days*	Hemiparesis	210/110	n/a	Vasc unit	Normal	Hemiparesis recovered <12 h
6	3 days	Hemiparesis	209/90	n/a	On ward	PCO	Hemiparesis recovered <48 h. OHS = 0 at 30 days
7	32 h	Hemiparesis	220/112	240/174	On ward	ACO + ICH	No significant improvement, OHS 5 at day 30
8	(i) 17 h	Dysphasia	200/150	194/94	On ward	ACO	Dysphasia resolved <24 h. Discharged home on day 7.
	(ii) 8 days	Hemiplegia	142/75	134/74	Other med	ACO	OHS 2 at 30 days

* indicates patients with bilateral seizures, BPOA indicates first BP reading after onset of symptoms, MCAVOA = first MCAV reading after onset of symptoms which may not be the same time as reading BPO. CT reports: PCO = posterior circulation white matter oedema, APH = anterior circulation petechial haemorrhage, ACO = anterior circulation white matter oedema, ICH = intracranial haemorrhage, OHS = Oxfordshire handicap Scale (REF). OHS 0–2 = non-disabling stroke, 3–5 = disabling stroke.
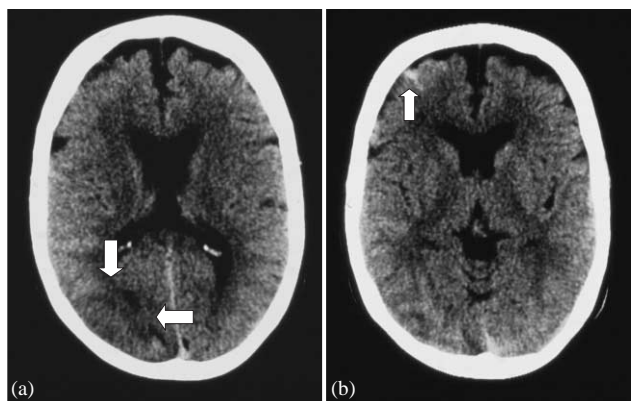
Fig. 1. CT scans of case 4 who underwent a right CEA. Five days post-operatively, she suffered unilateral seizures and was noted to have a left-sided weakness. Scans revealed an area of white matter oedema in the right posterior circulation (a) and a focal petechial haemorrhage in the right frontal region (b). She made a full neurological recovery within 24 h.

Fig. 2. CT scan of case 7 who underwent a right CEA. Thirty-two hours post-operatively, he suffered unilateral seizures followed 10 min later by dysphasia and hemiparesis. Twenty minutes thereafter he became profoundly hemiplegic and obtunded. The CT scan revealed extensive oedema throughout the right anterior cerebral territory with diffuse haemorrhage (Fig. 2). Although his conscious state improved, he made no significant neurological recovery.

there has generally been a tendency to diagnose one or other condition should patients suffer a seizure after CEA. It does seem increasingly likely, however, that the two are interlinked and may represent synergistic triggers for a subsequent "common pathway". Considering the two syndromes together might explain some of the anomalies observed to date. First, if this is purely a hyperperfusion phenomenon, why should some patients develop severe secondary hypertension? Second, if autoregulation is defective from the outset, why do most of these patients have a surge in MCAV following clamp release that declines over the next 10 min (suggesting that autoregulation is intact)? Similarly, if pre-operative dysautoregulation predisposes to post-operative hyperperfusion and secondary seizure, why are there not consistently large increases in MCAV 3 h post clamp release in patients destined to suffer seizure? Third, if this is purely a high flow problem, why have some series reported no increase in blood flow when the patient becomes symptomatic?[4] Fourth, how does HS or HE account for the patchy white matter oedema (now increasingly described in recent series where flow is either normal or increased) and why do patients exhibiting this abnormality predominantly do so in the posterior (rather than the carotid) circulation?

What then is the evidence linking HS/HE in a common pathway? First, the clinical sequellae of HS and

HE are virtually identical (headache, confusion/agitation, seizure, visual impairment, neurological deficit etc.). Second, both conditions exhibit near identical pathological features, which in the more severe forms include oedema, fibrinoid necrosis, arteriolar thrombosis, micro-infarcts and petechial/breakthrough haemorrhages.[3,8] Third, imaging of the brain shows remarkably similar features in HS and HE.

The principal abnormalities in HS and HE includes white matter oedema, predominantly affecting the posterior cerebral circulation (especially the occipital lobes). More advanced cases display focal ischaemic infarction, petechial haemorrhage and more overt ICH. These abnormalities are clearly demonstrated in Figures 1 and 2 in the current series. Diffusion weighted MR[4,8] indicates that the oedema is vasogenic (as opposed to cytotoxic) and is consistent with breakthrough of the autoregulatory mechanism rather than ischaemia.[5,8] Extravasation of protein and oedema fluid creates the subtle white matter changes. Moreover, because autoregulation is now absent, a secondary hyperperfusion response inevitably occurs, further aggravating the microcirculatory changes with increasing breakdown of the blood brain barrier (i.e. here HS may supersede HT). Unchecked, HS/HE triggers sequential impairment in endothelial permeability, activation of the coagulation cascade and inhibition of endothelial fibrinolysis.[8] Accordingly, patients with severe HE or HS can suffer ischaemic or haemorrhagic infarction (or both).

Two important issues require clarification. First, how can we be sure that the white matter oedema on CT and T2 weighted MR is not simply an evolving ischaemic infarct. Second, if the operation has been performed on the carotid circulation, why does the white matter oedema predominantly affect the vertebrobasilar territory?

The emergency CT scans of four patients in this series were originally reported as showing "evolving infarction" even though, in retrospect, this was highly unlikely to have become apparent within 40–60 min of onset of seizures. Subsequent independent review for this study reclassified each of these as probable white matter oedema. Breen[3] has documented exactly the same scenario. Although previous studies' using early generation CT scans in patients with HS or seizure after CEA were normal, a few did note white matter oedema into which haemorrhage would later occur.[9–11] More recent studies, have greatly clarified the situation. First, diffusion weighted MR indicates that the oedema is not cytotoxic, which would otherwise be expected with an evolving infarction[4,8] Second, Baptista recently described a case identical (in many respects) to the eight in this series. CT and

T2 weighted MR suggested an infarct but diffusion weighted MR clearly showed no evidence of an ischaemic lesion and MR perfusion-imaging indicated no change in local cerebral blood volume. TCD showed no evidence of increased MCAV. Unfortunately, the blood pressure was not documented.[4] Paradoxically, Schwartz has demonstrated increased perfusion on SPECT in the area of brain corresponding to the hypodense lesion on CT and the increased T2 signal on MR.[5]

To illustrate the sequence of changes in HS, Figure 3 illustrates a patient from our unit who was readmitted with a severe headache (but no seizures or neurological deficit) seven days after left CEA. Figure 3a shows an early CT scan after he suffered a right carotid territory stroke in June 2000. Duplex showed a symptomatic right ICA occlusion and an asymptomatic left 90% stenosis. Note the evolving right MCA territory infarct, but no abnormality in the left posterior circulation. By August 2000, the patient had reported left carotid territory TIAs and underwent a left CEA. Seven days post-operatively he was readmitted with severe left sided headache, elevated MCAV and severe hypertension (Fig. 3b). Note the old established infarct in the right hemisphere and the new area of "white matter oedema" in the left posterior circulation (initially diagnosed as an infarct). T2 weighted MR imaging six months after Figure 3b shows no evidence of infarction in the area corresponding to the white matter oedema in the posterior circulation (Fig. 3c). There was, however, a small focus of ischaemic change adjacent to the lateral ventricle. These sequential scans indicate the presence of a transient phase of white matter oedema in the ipsilateral posterior circulation territory in a patient with early HS/HE (headache only).

There is no consensus as to why the white matter oedema predominantly affects the posterior circulation. The prevailing view is that this may reflect regional variations in sympathetic innervation within the brain.[5,8] The carotid territory and its downstream arterioles have a much denser network of sympathetic neural control than the vertebrobasilar territory. Accordingly with worsening HS or HE, the posterior brain (especially the occipital cortex) may suffer autoregulation breakthrough and the secondary microcirculatory changes before the anterior circulation.

In conclusion, patients suffering seizure after CEA may have either HE or HS. How much one "drives" the other remains to be seen but warrants further research. It has been suggested that CEA performed under locoregional anaesthesia may be associated with a lower incidence of post-operative hypertension.[12] Accordingly, one might hypothesize that
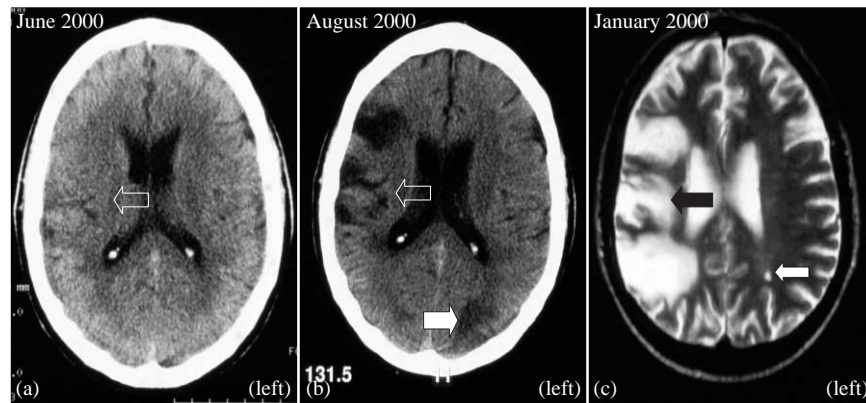
Fig. 3. CT scans of a patient with severe headache (no seizures), high MCAV and severe hypertension seven days after left CEA. (a) Early CT scan after suffering a right carotid territory stroke in June 2000. Duplex showed a symptomatic right ICA occlusion and an asymptomatic left 90% stenosis. Note the evolving right MCA territory infarct (open arrow). (b) By August 2000, the patient had suffered left carotid territory TIAs and underwent a left CEA. Seven days post-operatively he was readmitted with severe left sided headache, elevated MCAV and hypertension. Note the old established infarct in the right hemisphere (open arrow) and the area of white matter oedema in the left posterior circulation (white block arrow). (c) T2 MR scan six months after (b). Note there is no evidence of infarction in the area corresponding to the white matter oedema noted in (b), but there is a small focal lesion at its periphery (small arrow).

HE and HS may be less of a problem. However, this is unlikely to be borne out, as there are already reports in the literature of ICH, seizures and HS following carotid angioplasty in the absence of significant hypertension.[13,14] Accordingly, some high-risk patients undergoing angioplasty with extensive extracranial disease may need longer periods of in-patient observation, especially if there is any evidence of worsening hypertension.

In this audit, important lessons were learnt regarding the effectiveness of existing protocols for peri-operative blood pressure management. Most importantly, patients with seemingly well-controlled hypertension pre-operatively may still have labile hypertension on admission. In the presence of severe multi-vessel extracranial disease, surgery should probably be deferred until a review of blood pressure management has been undertaken. Secondly, colleagues in medical units tended to view aggressive control of hypertension as being of lesser importance to control of seizures. The principal reason cited was the general recommendation that blood pressure should not be reduced in patients with acute neurological deficits. However few had heard of the hyperperfusion syndrome and they were generally unaware of the pathophysiology and clinical implications of untreated HS and HE after CEA.

References

1 Sundt TM, Sharborough FW, Piepgras DG, Kearns TP, Messick JM, O'Fallon WM. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy. *Mayo Clin Proc* 1981; **56**: 533–543.

2 Naylor AR, Ruckley CV. The post-carotid endarterectomy hyperperfusion syndrome. *Eur J Vasc Endovasc Surg* 1995; **9**: 365–367.

3 Breen JC, Caplan LR, DeWitt LD, Belkin M, Mackey WC, O'Donnell TP. Brain edema after carotid surgery. *Neurology* 1996; **46**: 175–181.

4 Baptista MV, Maeder P, Dewarrat A, Bogousslavsky J. Conflicting images. *Lancet* 1998; **351**: 414.

5 Schwartz RB, Jones KM, Kalina P *et al.* Hypertensive encephalopathy: Findings on CT, MR imaging and SPECT imaging in 14 cases. *AJR* 1992; **159**: 379–383.

6 Naylor AR, Hayes PD, Allroggen H *et al.* Reducing the risk of carotid surgery: A seven year audit of the role of monitoring and quality control assessment. *J Vasc Surg* 2000; **32**: 750–759.

7 Halsey JH, McDowell HA, Gelmon S *et al.* Blood flow velocity in the middle cerebral artery and regional blood flow during carotid endarterectomy. *Stroke* 1989; **20**: 53–58.

8 Vaughan CJ, Delanty N. Hypertensive emergencies. *Lancet* 2000; **356**: 411–417.

9 Schroeder T, Sillesen H, Boesen J. Intracerebral haemorrhage after carotid endarterectomy. *Eur J Vasc Surg* 1987; **1**: 51–60.

10 Harrison PB, Wong MJ, Belzberg A, Holden J. Hyperperfusion syndrome after carotid endarterectomy. *Neuroradiology* 1991; **33**: 106–110.

11 Reigel MM, Hollier LH, Sundt TM, Piepgras DG, Sharbrough FW, Cherry KJ. Cerebral hyperperfusion syndrome: A cause of neurologic dysfunction after carotid endarterectomy. *J Vasc Surg* 1987; **5**: 628–634.

12 Tangkanakul C, Counsell C, Warlow CP. Local versus general anaesthesia in carotid endarterectomy: a systematic review of the evidence. *Eur J Vasc Endovasc Surg* 1997; **13**: 491–499.

13 Morrish W, Grahovac S, Douen A *et al.* Intracranial haemorrhage after stenting and angioplasty of extracranial carotid stenosis. *AJNR* 2000; **21**: 1911–1916.

14 Ho DS, Wang Y, Chui M, Ho SL, Cheung RT. Epileptic seizures attributed to cerebral hyperperfusion after percutaneous transluminal angioplasty and stenting of the internal carotid artery. *Cerebrovasc Dis* 2000; **10**: 374–379.