Transient Cerebral Ischaemic Disease in Pakistan: Does Carotid Endarterectomy Help?

*S.M.A. BABAR**

A study pertaining to a small series of patients suffering from surgical cerebrovascular disease is presented in this paper. Since the study was not scientifically controlled prospectively, the techical charecteristics cannot be statistically generalised. Although the series contained a very few number of patients, 50% were found to be suffering from hypertension, and 50% had additional cardiovascular disease. 100% were suffering from symptomatic TIAs, 50% had a past history of stroke and Carotid bruit was heard in 83% of the cases. All patients were indiscriminately shunted during endarterectomy (10 with Brenner's shunt and 2 with tube shunt). One patient, additionally suffering from Takayasu's disease died on the 9th post-operative day from secondary haemorrhage.

This paper attempts to analyse the benefits and complications of Carotid endarterectomy and paves the way for an organised unbiased prospective study to assess the value of this operation for significant asymptomatic critical Carotid disease and for symptomatic patients with Carotid stenosis. The existing available literature is also reviewed in this light.

METHODS AND MATERIALS

All of the patients were referred from other specialties and had reported symptomatic Transient Ischaemic Attacks (TIAs). The duration of symptoms varied widely. 12 patients were treated in this study. 11 were males with an average age of 55 years (40-70). 1 was female aged 35. All patients underwent doppler assessment for Carotid stenosis. Serial BPs measured in supine and standing positions and fasting and 90 minute postprandial blood glucose levels constituted the main methods of categorising hypertension and diabetes respectively.

Angiography

Transfemoral percutaneous angiography was performed in 11 patients. Both Carotids were studied in all of these patients. Vertebral arteries were cannulated in 6 patients. In the remaining five the vertebral arteries could not be cannulated so that the Circle of Willis could not be properly studied. Cross compression tests were performed in only three patients. In one patient Carotid puncture angiography was performed only on the side of the lesion and contralateral study was not performed angiographically, but a doppler assessment was made.

Endarterectomy

All patients were subjected to Formal Carotid endarterectomies. The patient with Takayasu's disease additionally underwent a Caroticosubclavian bypass procedure on the same side. Extensive endarterectomy was carried out in all patients. Proximal and distal ends were tacked with 6/0 Prolene sutures. Plaques were not biopsied. Arteriotomy was sutured with continuous 5/0 Prolene. External irrigation with saline was performed prior to unclamping. Clamps were systematically removed so as to flush any debris into the External Carotid circuit.

Shunting

A shunt was used in every patient indiscriminately. 1 minute prior to clamping of the Carotid Artery, systemic Heparin, 5000 Units were administered. Brenner Shunts were used in 10 and straight improvised shunts were used in the remaining 2 Carotids.

Transient Cerebral Ischaemic Disease (TCID) has been treated under different nomenclatures and under different protocols for a number of

* Head of the Deptt. of Vascular Surgery
Dow Medical College and Civil Hospital, Karachi.

OBSERVATIONS

Pt	Sex	Age	TIA	Assoc CVS lesions	Diabetes	Hypertension	Past history of stroke
1	M	40	+	Nil	Nil	+	Nil
2	M	55	+	IHD	+	Nil	+
3	M	47	+	IHD	Nil	Nil	+
4	M	50	+	Nil	+	+	Nil
5	M	60	+	Nil	Nil	Nil	+
6	M	60	+	Nil	Nil	+	+
7	M	58	+	Nil	Nil	+	Nil
8	M	55	+	Nil	+	+	+
9	M	58	+	PVD	Nil	Nil	Nil
10	M	70	+	IHD	Nil	+	+
11	M	62	+	IHD	Nil	Nil	Nil
12	F	35	+	Takayasu	Nil	Nil	Nil

Table 1.
(IHD=Ischaemic heart disease. PVD=Peripheral vascular disease)
Relationship of Carotid bruit to haemodynamic stenosis:

Pt	Carotid bruit	Angiographic stenosis percentage (ICA)	Contralateral stenosis percentage (ICA)	Shunt Used
1	+	80	60	+
2	+	70	25	+
3	+	75	65	+
4	+	65	—	+
5	+	85	25	+
6	+	50	50	+
7	+	75	—	+
8	+	40 (Ulcer)	—	+
9	+	75	50	+
10	+	90	50	+
11	—	75	00	+
12	—	85	70	+

Table 2.

years in Pakistan. It is only recently that an organised collective effort is being made to reduce the incidence of "preventable" strokes in Pakistan. Internationally, since the advent and easy availability of modern non-invasive investigations and better intraoperative monitoring facilities to detect and counteract critical cerebral ischaemia, the results of surgical treatment have consistently improved.

REVIEW OF LITERATURE

Historical review

The relationship between stroke and extracranial Carotid atheromatous disease was recognised over a century ago and the earliest report came from Savory who in 1856 linked (through a post mortem) Left internal Carotid Artery and

Vertebral Artery with right hemiplegia and monocular blindness (1). In 1914 Ramsay Hunt (2) described "Cerebral intermittent claudication", which is now popularly known as "Transient Ischaemic Attack", and he was the first scientist to emphasise the importance of extracranial carotid lesions in stroke. In 1950, Strully (3), performed the first operation for a thrombosed Internal Carotid Artery. De Bakey (4) may have been the first person to have performed internal carotid endarterectomy in 1953 but did not report it until 1959. In 1954, Eastcott (5) performed Carotid artery bypass for atherosclerotic obstruction. In 1955 Carrea (6), resected a diseased Internal Carotid Artery and anastomosed it to the External Carotid Artery. However it is only in the last eleven years or so that the accurate methods of assessment have become available which have brought down the high earlier morbidity risk factors associated with contrast angiography (7). Many non-invasive investigations are now available which screen out high risk patients for selective invasive studies.

Incidence

Each year in the USA alone, some 500000 patients suffer from stroke and a substantial number of these patients dies from initial strokes. A classic 20-year study performed at the Mayo Clinic (8) showed that in the elderly, 38% of patients died of the initial stroke, 10% died as a result of subsequent stroke and 18% died of cardiovascular diseases. In those patients who survived the first stroke, the projected inference remains that 10% will have another stroke within one year and 42% in 5 years (9). Francis et al (10) reported in a large series taht upto 50% of stroke are caused by the extracranial carotid disease. The remainder result from intracranial disease, hypertension and cardiac disease.

Behavioural classification

Vascular occlusive transient diease tends to follow a stereotype pattern and based on the similarity of behavioural pattern, the Committee for the Classification of Cerebrovascular Diseases in 1975 (11) proposed the following classification of such ischaemic disorders.

1. Transient Ischaemic Attacks (TIAs). This is a transient focal neurovascular episode, lasting from 2-15 minutes and not longer than 24 hours and resolving fully with no deficits.

2. Reversible Ischaemic Neurological Deficit (RIND). This is a neurovascular focal episode, lasting from 24 hours to seven days but not leaving behind any residual deficits.

3. Recovered Stroke (RS). This is a focal neurovascular episode lasting for more than a week and recovering totally or partially in a variable period of time.

4. Amaurosis Fugax (AFx). This is a vascular episodic occlusion of the ophthalmic or the retinal artery, resulting in total or near total loss of vision, lasting for 2-15 minutes in one eye but not more than 24 hours, resolving fully and not leaving any deficits.

5. Combined Syndrome. This consists of TIAs or RIND and AFx usually occurring as a result of a lesion in a single Carotid Artery and both the cerebral and the visual effects are produced simultaneously. The course of the disorder and recovery time are variable.

Stereotyping pattern of lesions

TIAs are the most common manifestations of extracranial Carotid disease. They usually affect areas supplied by the Anterior or the Middle Cerebral Arteries producing contralateral lesions. Other forms of TIAs are Crescendo TIAs (where a second TIA occurs within 24 hours of the first episode), and Stroke-in-evolution (where the initial stroke is made worse by repetitive discrete exacerbations).

Ischaemic attack are generally stereotyped and involve the same vascular distribution. Millikin (12) injected small metal beads in Internal Carotid Arteries of monkeys and showed that such beads always stacked up in the same cortical branches. This resulted in the projection of the idea that embolisation from a plaque will almost always take place in the same cerebral arterial distribution.

Whisnant (13) conducted a trial between 1955-1969 evaluating untreated patients having

TIAs and found that the stroke rate was 23% at one year, 37% at three years and 45% at five years. The study also showed that 41% of patients developed stroke within the first year of the initial TIA (14). This study also indicated that 50% of patients with stroke will have had a previous warning TIAs ( ).

The most common lesion is an atherosclerotic plaque of the ICA. This accounts for about 90% of all cases of Cerebrovascular pathology in the Western world. 67% of 4748 patients studied angiographically were found to have extracranial carotid lesion in the region of the bifurcation (16). Although the exact mechanism of initial deposition of the plaque is not known, the behavioural progress subsequently follows a stereotypic pattern. The plaque grows and accumulates more material. Gradual stenosis of the ICA then occurs.

The Causes of Symptoms

There are three possible ways in which symptoms are produced.

1. The least accepted hypothesis explains it on the basis of ischaemic infarction produced as a result of reduced blood flow. The majority of vascular surgeons do not subscribe to this view because during the phase of carotid cross clamping under local anaesthetic, when the flow is drastically reduced, no focal TIAs are produced in 90% of the patients (17). Also, patients undergoing surgery under a general anaesthetic, during phases of hypotension induced by anaesthetic agents, do not show any crises on blood flow isotope studies or the intraoperative EEGs (18) and intra and perioperative stroke incidence does not correlate with reduced transient blood flow.

2. The other more plausible hypothesis suggests necrosis or haemorrhage in the centre of the plaque as an event of rapid progression. Loose fragments then embolise distally into the terminal branches producing ischaemic infarction.

3. The hypothesis most widely accepted suggests that as the plaque increases rapidly in size, there is central and some peripheral aseptic necrosis produced. The denuded area then attracts and binds platelets and susceptible formed elements of blood, which then embolise distally.

Causes of TIAs

"Stroke" is a heterogenous term which includes Cerebral infarction, haemorrgage, subarachnoid haemorrhage and Cerebral embolism. Atherosclerosis affects both the intra as well as the extracranial vessels, similarly factors producing TIAs act at both these two regions.

1. Embolism. The sources usually encountered are, extracranial vascular disease, Rheumatic heart disease, ischaemic heart disease, cardiomyopathies, Mitral Valve prolapse etc.

2. Reduced cerebral blood flow (cardiac dysrrythmia, intra and extracranial carotid atherosclerosis, extracranial vascular anomalies, compressive lesions in the neck and systemic hypotension.

3. Increased blood viscosity (polycythemia, dysproteinaemia etc).

4. Vasoconstriction (migraine, hypocapnoea, subarachnoid haemorrhage).

5. Cerebral oedema (hypertensive episodes etc).

6. Metabolic failure (hypoglycaemia, severe anaemia).

7. Stress factors precipitating TIAs.

   a. Unaccustomed severe exercise.

   b. Severe infection and fever.

   c. Emotional shock.

   d. Prolonged anaesthetic and invasive surgery.

   e. Trauma to neck, head or severe generalised trauma.

   f. Drugs (Thyroxine overdosage, stimulants etc.)

   g. Hypermetabolic states (Thyrotoxicosis, Cushings, etc.)

   h. Hypovolaemic shock.

8. Risk factors for TIAs and stroke

Transient cerebral ischaemic disease usually occurs in old age but increasingly more young patients are being documented. Its risk factors parallel those which precipitate ischaemic heart disease.

a. Hypertension.
b. Hyperlipidaemia (increased LDL).
c. Soft water consumption (increased hypertension).
d. Alcohol excesses (hypertension).
e. Diabetes Mellitus.
f. Nicotine.
g. Low levels of physical exercises.
h. Desrogen intake (probably haemodynamic).

RELEVANT SYMPTOMS AND SIGNS OF TRANSIENT CEREBRAL ISCHAEMIC DISEASE

1. Hemispheric symptoms. Dysphasia, hemiparesis, hemiplegia, altered level of consciousness and mentations, sensory disorders, cranial nerve dysfunctions etc. These are all transient and resolve depending upon the nature of the TCID.

2. Vertebrobasilar symptoms. These are rare and relate to Vertebral TIAs and correspondingly affect the gait, level of consciousness, basal cranial nerves and cerebellomedullary outflow. These are rare.

3. Visual disorders. These relate basically to disorders of Visual cortex or to disorders of Eye circulation. Of the Ocular symptoms the two most classical are Amaurosis Fugax (AFx) and Rubiosis Iridis. AFx is defined as total or partial blindness and is the manifestation of ophthalmic critical embolisation from an ICA embolus. The causative agent could be debris or platelet plugs. Generally the symptoms last for a few minutes and resolve fully in any case in less than 24 hours. Fundoscopy reveals Hollenhorst lesions (21). AFx merits aggressive work up for Carotid disease. Rubiosis Iridis occurs due to reduction in the blood flow of Ophthalmic artery resulting in neovascularisation of Iris which in turns results in congestive oedema and occasional obstructive glaucoma (19).

4. Carotid bruit. The most important clinical sign of Carotid stenosis is Carotid bruit, a murmur heard in the mid cervical region on the anterior border of the sternomastoid after exclusion of the venous hum, respiratory sound and transmitted sounds from the heart and from the great vessels on either side of the neck. In a recent series Murray et al (22) studied 400 Carotids. His findings showed that 34% of the vessels associated with symptoms had a bruit present and 23% of the asymptomatic patients also presented with a Carotid bruit. In another study, he examined 114 Carotid Arteries. Of these only 51 had significant haemodynamic disease while 63 did not have a significant haemodynamic disease in the ICA. Of the 63, 25% showed a haemodynamic disease in the ECA and the remainder did not show any objective stenosis to account for the bruit. These were then regarded as unexplained. Most workers would dispute with Murray's findings. At present it could be said that 60% of the patients presenting with a bruit in fact do have a significant haemodynamic stenotic lesion (23). This modern view is a result of better resolution of contrast radiography in defining fine lesions of the ICA. Roederer (24) studied 162 asymptomatic patients with bruit and observed their evolution in time with Duplex scanning. He found that patients with 80% or more stenosis had a 46% chance of developing a TIA/stroke in 1 year. In contrast he found that patients with stenoses of less than 80% had a 15% chance of TIA/stroke in 1 year. There is a considerable body of evidence present showing that in the arteriopaths (Carotid and concomitant Coronary Disease), who have an asymptomatic Carotid bruit present, perioperative stroke rate is significantly high with Coronary revascularisation procedures. This risk is significantly reduced by performing Carotid endarterectomy prior to Coronary revascularisation (25).

5. The Asymptomatic Carotid lesion. Since the advent and easy availability of non-invasive tools and in particular of the Duplex Scan, the behaviour pattern and natural historical progression of the Asymptomatic Carotid Disease have been well documented. There appear to be three categories of disease present.

a. Those with non stenotic ulcerated plaques.

b. Those with haemodynamically significant stenoses.

c. Patients with Carotid bruit on routine examination.

Moore et al (26) conducted a very good and valid study on asymptomatic patients who underwent contrast radiography and had their lesion typed. The lesions (72 out of 77) were of an ulcerated type. He categorized these as Minor, Large and Compound and used strict criteria. Patients were well matched and the findings were matched with the data of symptomatic patients who underwent carotid endarterectomy. He found that the stroke rate was 8 times higher in unoperated patients with large or compound ulcers. The stroke rate in patients with the minimum lesion was negligibly higher than in the control. Francis et al (27) using OPG criteria and Duplex scan found that patients with haemodynamically significant stenosis had a risk of developing stroke 7.5 times higher than in the patients with an insignificant disease. This increased risk was eliminated with Carotid endarterectomy (28).

INVESTIGATIONS OF CAROTID STENOSIS

Of the relevant practical investigations, currently, the best suited for the TCID are Duplex scanning, OPG, EEG, CT scanning and Contrast radiology. Other less attractive but upcoming methods include thermography, isotope studies of the blood flow, transcranial ultrasound scan, psychometry etc. In Pakistan at present doppler studies, EEG, CT Scan and angiography are available for assessment.

1. Duplex Scanning. This will soon be introduced in Pakistan and would considerably alter the protocol of investigations. It is clearly the first line investigation forming the basis for more invasive tests. It is also probably the most useful investigation for the follow-up of a patient who has either had surgery performed or who is on a follow-up after angiography, it utilises a real time B mode scan with a pulsed doppler ultrasound and gives a graphic image of the vessels. Individual cursors can be fixed on the screen and via an appropriate probe interpretation and study can be made of the mechanical lesions and of th flow characteristics of the pre and poststenoti segment. The spectral broadening gives a good idea of the degree to narrowing and the pulsed doppler helps to show the flow characteristics of any turbulence. Another development (Triplex, Dopscan) is to colour code the computer on the basis of directional flow characteristics. This, then colours the artery red and the vein blue, a clear and quick assessment is made of the dynamic lesion. On the basis of the Duplex, lesions are classed as Minor (10-15%), moderate (16-49%) and severe (50-99%). It has a 92% accuracy rate with an overall specificity of 96% and sensitivity of 95% (29).

2. Oculoplethysmography (OPG). This measures the pressure of the Ophthalmic artery and works on the principle that any narrowing of the ICA will be correspondingly reflected in the Ophthalmic artery pressure. Its accuracy has been variably reported alone and in conjunction with the Duplex as between 86-97% (30).

3. Electroencephalography (EEG). Its greatest benefit it is in its ability in differentiating between cerebrovascular disease and tumours, cerebral from brain-stem lesions and to a lesser extent between cerebral haemorrhage and infarction. In patients with stroke, the EEG abnormalities improve with time whereas with tumours, these deteriorate. Serial EEGs are therefore required for such assessment. Brain-stem lesions may demonstrate a normal EEG, or generalised slow activity, intermittent bouts of generalised beta activity (4 to below 8 c/s) and bitemporal changes. In cerebral haemorrhage widespread irregular high voltage and very slow delta activity are prominent. The feature on EEG after cerebral infarction tends to be less marked and produces moderately slow delta with regular smooth or triangular wave forms and an increase in the amplitude of abnormal alpha rythm on the side of the lesion. All of these changes tend to improve with time, leaving an almost normal EEG at six months upto 50% of the cases. The greatest value of EEG in respect of a Carotid disease lies in it being a sensitive tool to detect intraoperative signs of carotid territory cerebral ischaemia in susceptible patients during the phases of cross clamping of the Carotid. In a majority of patients such clamping does not cause any side effect. In cases where the Intraoperative EEG

does show significant distress and an impending infarction, the ischaemic crisis can be averted by immediately shunting the patient. Because of its transportability this is likely to become available as an intraoperative monitoring device with already emerging better co-operation between the vascular surgeon and the neurologist.

4. CT Scan. This is not considered to be a very important investigation, with some reservation. Its value lies in detecting any areas of previous ischaemic infarctions or unresolved haemorrhages. Many patients who present for surgery give a long history of TIAs and any one of these may have left a positive sign for the Scan. Concomitant lesions, such as an intracerebral aneurysm, a tumour etc. can be picked up pre-operatively. These will have a considerable bearing on the operability of the patient. Its biggest value lies in non-invasively assessing acute neurological problems. Following an endarterectomy if evidence of a large thrombus in the Carotid presents as an area of expanding infarction cerebrally, then re-exploration and a thrombectomy can be carried out within three hours of development of obstruction.

5. Digital Subtraction Angiography (DSA). With this study, X-ray images are taken of the parts required (neck, head, limbs). The information relating to the density characteristics are fed into a computer and stored. A dye is then injected peripherally into a vein. Images are taken again of the same parts and density characteristics are stored. From the later image the computer then subtracts the former image leaving behind the image of the dye in the vessel under study. With controlled timing and exposures and manipulation of the computer, good resolution images are obtained of the vessel. In high pressure areas such as the neck and the intracranial circulation, images are enhanced by selectively catheterising the Aortic arch and injecting the dye there. Thus with a very much smaller quantity of dye a high resolution image of the Circle of Willis or of the extracranial vessels can be seen. The morbidity associated with conventional angiography is reduced considerably. It has a sensitivity of 95%, a specificity of 99% and an overall accuracy of 97% (31). By its availability, the need for conventional angiography would drastically be reduced to specially selected cases which cannot be fully studied by the DSA. The DSA would shortly be available in Pakistan and the indication for angiography would become reduced correspondingly.

6. Conventional Angiography. It comprises Arch angiography for a general assessment of the major root vessels or specific selective angiography (general four vessels angiography). The transfemoral percutaneous approach has outdated needle puncture angiography. An injection into the Inominate Artery outlines the roots of the main divisions. Further selective catheterisation will produce detailed views of all four main vessels. The tip of the catheter into the Vertebral arteries should be withdrawn after every injection of the dye to prevent ischaemia. Carotid stump pressure after mechanical compression of the proximal Carotid provides information about reverse flow and allows a decision to be made regarding the use of a shunt during endarterectomy. Contralateral study, supplemented with a study of the Vertebrals, provides a full and necessarily desired information about the Circle of Willis. Angiography has a sensitivity of 97%, specificity of 99% and an overall accuracy of 98.5%.

7. Psychometric studies. Halstead Reitan tests etc. can reveal major cortical dysfunction however these relate to the experience of the psychologist and to obtain meaningful results, sufficient time must be spent periodically to evaluate the pre and the postoperative states of the patient.

8. Cerebral Blood Flow Measurements. Isotope studies have not become very popular, since the arrival into the arena of the DSA and the Duplex.

9. Magnetic Resonance Imaging (MRI). In the future this recently evolved tool may provide good information about the preinfarct ischaemia before any real damage has taken place.

INDICATIONS FOR CAROTID ENDARTERECTOMY

Surgery in a patient who is suffering from stroke is mainly a prophylactic procedure (except

for thrombectom). Revascularisation of dead tissue will not restore useful functions. Assuming that the general condition of the patient is adequate and that the angiography shows the lesion as a treatable local condition, the main indications for Carotid endarterectomy are:

a. Hemispheric TIAs, uncontrolled with medical therapy.

b. Amaurosis fugax and Central Retinal arterial occlusion.

c. Major TIAs and ulcerated plaques.

d. TIAs associated with stenosis of > 50%.

e. Asymptomatic Carotid stenosis of > 75% in patients undergoing major cardiovascular surgery.

f. Previous hemispheric stroke with good functional recovery with a critical stenosis in an asymptomatic patient.

g. Tandem lesions with focal symptoms or signs.

h. Subclavian steal.

i. Emergency surgery for acute stroke, stroke-in-evolution or crescendo TIAs. This implies performing a Carotid thrombectomy in a patient already hospitalised, within 3 hours. If indicated, angiography under Heparin control should precede the procedure. Such a contingency usually arises in iatrogenic thrombosis such as after a radiological or a surgical procedure. Revascularisation soon after this time is associated with a greater risk of haemorrhage from the freshly infarcted areas and such operations should be deferred for 4-6 weeks. Goldstone and Moore reported good results with this approach in a series of 25 patients where no deaths occurred and full neurological recovery followed (32).

CAROTID ENDARTERECTOMY

Universal preference for performing Carotid Endarterectomy is to use the general anaesthetic which allows good freedom of movement, appropriate mobilisation of the vessels, extension of wound and the ability to cope with unforeseen emergencies such as bleeding from the shunt sites etc. The advantage with local anaesthetic procedure is that the patient remains conscious and reports any untoward effects of clamping (dizziness, numbness, failing consciouness etc.). However there are drastic limitations of manoeuvres. Under a carefully controlled general anaesthesia where blood pressure adjustment is regulated within critical limits, through an anterior approach, the main stem of the Common Carotid Artery is exposed and a limited mobilisation carried out. Local anaesthetic is infiltrated at the site of the bifurcation to prevent reflex bradycardia. The ICA is exposed to the level of the Hypoglossal Nerve and under cover of a systemic load of Heparin (5-10,000 Units), pressures of the unclamped CCA and the ICA after clamping the CCA (stump pressure) are recorded. If the stump pressure is less than 1/3 of the systemic CCA pressure then the cross-flow from the opposite side via the Circle of Willis is inappropriate and a shunt is inserted. Selective shunting and intraoperative monitoring to avert an impending cerebral ischaemic crisis are the two modern methods which have helped to bring down the intra and perioperative stroke rate. If by these methods this rate is brought down to zero then many of the judicious objections for surgery will disappear. With or without a shunt, under clamps, an arteriotomy is made and endarterectomy performed. Frayed edges are tacked and the arteriotomy is sutured with a continuous Prolone(5/0) after systematic flushing of debris and clots outside and into the External Carotid circuit.

Should one routinely use the Shunt

The most unresolved controversy amongst the vascular surgeons pertaining to Carotid endarterectomy is whether or not to use a shunt during the procedure. There are three schools of thought:

1. To shunt in every patient undergoing endarterectomy. This is done in the belief that the risks of haemodynamic intolerance cannot be identified accurately with any of the intraoperative parameters (34) and that the use of the shunt carries with it minimal and acceptable risks for embolisation. Objectors point out that the shunt

interferes with surgical precision of endarterectomy by limiting accessability and may cause dissection in the proximal segment. Another valid point is that if facilities for intraoperative monitoring of critical parameters are not available then it would be quite appropriate to shunt every patient.

2. To not shunt in any of the cases. No shunt technique surgeons believe that the risks of clamp-induced ischaemia are exaggerated and that the actual cause of stroke is embolisation and not reduced blood flow (35). According to this argument the use of the shunt should by its technical presence and movements, precipitate embolisation. Objectors feel that some high risk patients do show a higher incidence of stroke unless adequate shunting is used.

3. Selective shunting. This attempts to offer a logical compromise (36) but the reported results are not altogether convincing. An excellent study was undertaken by Ferguson (37) to validate or invalidate the necessity of shunting, the criteria used involved continuous intraoperative monitoring by EEG, Carotid Stump pressure measurement and measurement of Cerebral blood flow (CBF) (38). In the past the use of the shunt was recommended if the intraoperative EEG showed significant changes, if the Carotid stump pressure was less than 50 mm Hg or if the CBF values were less than 20mm per 100 g/min. Measurement of somatosensory evoked responses have been to the intraoperative monitoring but these do not appear to have contributed greatly to the problem (39). Soon afterwards, Ferguson re examined the whole series in another better study and changed his views. He reported that neither the intraoperative monitoring nor the use of the shunt, in general prevented stroke. The main cause of the stroke being embolism, these were uninfluenced by the previously recommended measures. He postulated that during clamping, neuronal activity did suffer but that these were reversible (40). He did recognise a small subgroup of patients in whom the EEG changes were gross and/or the Stump pressure was 25mm Hg or less. These patients had a higher rate of stroke without shunting. Shunting was therefore recommended in these patients. Contradicting Ferguson, Gold described a reduction of stroke rate by selectively using the shunt on the hypothesis that the cerebral perfusion was otherwise compromised. He brought down the stroke rate to under 1% in his series. He used the CBF technique to monitor his patients. The modern majority now accepts that the intraoperative EEG does detect early signs of distress. Sundt (42) concluded that CBF measurements were fairly relevant parameters. Using selective shunting he found that 8% of the patients would have suffered a stroke had a shunt not been used. Ojemann (43) suggested a different approach. He argued that a general anaesthetic manipulation of blood pressure control, raising the systolic blood pressure to 170-180, during the period of occlusion (provided that there were no contraindications for doing this), would reduce the need for a shunt by greater perfusion (44). He too performed selective shunting for patients with poor collaterals who did not benefit from such "hypertension" (45), Summarising these two views, Hachinski (46) said that although Ferguson had earlier proposed a no-shunt technique, he changed his views to a selective shunting protocol by finding the subgroup of high risk patients and in the same ways, Ojemann now believes in selective shunting. "Surgical practice should be adjusted to account for facts as they become known".

In Pakistan, EEG is available as an outpatient investigation and it is now very likely that with a greater co-operation between the Vascular and the Neurological departments, it would become available as an intraoperative monitor. Since the CBF measurements are not likey to become available in the near future, the parameters of interest would be the EEG and stump pressure measurement. If these facilities are not available then all patients should be considered for routine shunting. It is the author's view that if monitoring facilities exist then selective shunting should be performed and where these are not available, indiscriminate shunting should be used.

Complications of Carotid endarterectomy:

1. The most feared and unfortunate complication of surgery is a stroke. This intra and perioperative complication has declined tremendously due to improved surgical technique, monitoring methods and shunting. It is widely suggested that a 3-5% rate of stroke should be acceptable (47). In any event this should not exceed 5%. The risks are increased in the elderly presumably because of diminished CBF capacity frequently seen in them. This renders the brain more sensitive to temporary

reduction of perfusion and accordingly higher stump pressure value would be accepted to avoid shunting (49). The best figures have shown a 2% stroke rate and a 1% death rate (50).

2. Myocardial infarction is the frequent cause of death (51).

3. Preoperative angiography carries with it a risk of 1.5% stroke rate in susceptible patients.

Strokes occur due to the following main post-operative complicating causes:

a. Reduced perfusion due to blockage of flow.
b. Embolisation of the pieces of debris, plaques etc.
c. Embolisation of platelet aggregates which adhere to the raw surface.

To detect thrombosis in the endarterectomised artery, some surgeons routinely perform post-operative on-table angiography. Because of its inherent risks, where facilities exist, instead of angiography, if neurological manifestations exist, then a Duplex scan and an OPG should be carried out. If the OPG pressure is low then a thrombus is more likely to have caused the problem and a reoperation should be performed urgently.

4. Other complications possibly pertaining to baroreceptors include hypotension (19%) hypertension (28%) (incidence of neurological complication is higher in patients who have postoperative hypertension (53).

5. Remote complications include wound infection and haematoma.

6. Haemodynamic flow disturbances due to kinking of the redundant endarterectomised artery.

7. Stroke rate is high if contralateral side shows a stenosis greater than 75%, in the Carotid artery. In such situations, a routine shunting is justified.

8. Ackroyd performed a study comprising 292 Carotid endarterectomised patients and reported in 1986 (54) using life time cumulative methods, that the restenosis rate was 15% at 5 years. Progression of disease on the contralateral side was 20% at five years. It was suggested that restenosis in the first year was caused by intimal hyperplasis (55). Inadequate endarterectomy and post-operative thrombosis constituted a significant percentage. Myointimal hyperplasis was also another factor. Ackroyd reported a higher incidence of restenosis in women but on later revaluation in an extended series she found a 1:1 ratio. However, in women the restenosis occurred earlier. She hypothesised that differential platelet behaviour was responsible for the disparity. This was further corroborated by others by demonstrating that the platelet response to antiplatelet agents was different in men and women. (58)

9. Recurrent TIAs were reported in 10% of patients after 5 years. Recurrent stroke rate was 6% (59, 60).

NON-SURGICAL TREATMENT OF CAROTID SURGICAL DISEASE

Apart from symptomatic treatment, the most popular agents showing a positive benefit to the patient are Aspirin and Warfarin (61). Warfarin is however inferior to endarterectomy and also precipitates bleeding. Dipyridamole and Sulfinpyrazone have been extensively studied. A combination therapy has now largely been abandoned in favour of a low dose intermittent aspirin (62) which shows a positive improvement.

REFERENCES

1. Savory WS, Case Report. Med Chir. Tr Lond. 1856: 39, 205-19

2. Hunt JR. Carotid arteries in lesions of brain. Am J Med Sci. 1914; 147, 704-13.

3. Strully KJ, Thromboendarterectomy for thrombosis of the ICA in the neck. J. Neurosurg. 1953. 10; 474-82.

4. De Bakey ME et al, Surgical considerations for occlusive diseases of Innominate, Carotid, Subclavian and Vertebral Arteries, Ann Surg. 1959, 149, 690-710.

5. Eastcott HHG et al. Reconstruction of ICA, Lancet 1954, 2; 994-6.

6. Carrea R et al. Report of a case. Acto Neurol. Latinoam 1955, 1; 71-8.

7. Summer DS et al. Noinvasive diagnosis of extracranial carotid disease. Arch Surg. 114;1222, 1979. Murie JA et al. Pulsed doppler Imaging and spectrum analysis for carotid disease. Angiology. 35;215. 1984.

8. Matsumoto N et al. Natural history of a stroke. Stroke 1973, 4; 20-9.

9. Sacco RL et al. Survival and recurrence after stroke. Stroke, 1982, 13, 290-5.

10. Mohr JP. TIAs and the prevention of strokes. N Eng 1978;299, 93-95, Pessin MS et al, Clinical and angiographic features of carotid TIAs. N Eng J Med 1977; 296; 358-62.

11. Committee on cerebrovascular disease. Classification. Stroke. 6;564., 1957.

12. Milliken C. Pathogenic mechanism of cerebral dysfunction. In Rutherford RB Vascular Surgery. Phil. Saunders. 1984; 1208-17.

13. Whisnant JP et al. Anticoagulant therapy in TIA. Mayo Clin proc. 1973; 48, 844-8.

14. Cartlidge et al. Carotid and vertebral-basilar TIA. Mayo Clin proc. 1977; 52, 117-20.

15. Yatsu FM et al. Stroke, Current Neurol, 1981; 3, 159.

16. Hass WK et al, Arteriography. Techniques, sites and complications, JAMA, 1968; 203, 961-8.

17. Moore WS et al. Carotid artery back pressure. Arch Surg. 1969; 99 702-10.

18. Kendall RE, et al. Role of hypotension in the genesis of TIAs. Brit Med J. 1963;2, 344-8.

19. Hauch et al. A report of iris neovascularisation. Surgery. 1984; 94, 358-62.

20. Baird RN, Carotid murmurs. Br J Surg. 70;83, 1983.

21. Rose FC et al. Radiological investigation and surgical treatment of sudden blindness due to extracranial vascular lesions. Trans Ophth Soc UK. 1971;91, 261-6.

22. Murie, JA Quin RD. Symptoms and signs of Carotid territory Transient cerebral ischaemia. Vasc Surg, 1986. Jan-Feb, 20-26.

23. Ochsner JL. Carotid bruit. The physician's dilemma. Postgrad Med, 70;57, 1981.

24. Roederer GO et al. The natural history of carotid disease in asymptomatic patients with cervical bruit. Stroke; 1984. 15; 605-13.

25. Bernhard VM et al. Carotid artery stenosis, Association for surgery with coronary artery disease. Arch Surg; 1972, 105, 837-40.

26. Moore WS et al. Natural history of nonstenotic asymptomatic ulcerative lesions of the carotid artery, Arch Surg, 1978; 113, 1352-9.

27. Sheng FL et al. Carotid surgery in stroke prevention AFP. 33;4, 1986. 109-24.

28. Busuttil et al. Carotid artery stenosis. Hameodynamic significance and clinical course. JAMA; 1981, 245, 1438-41.

29. Blackshear WM Jr et al. Detection of carotid occlusive disease by ultrasonic imaging. Surgery; 1979, 86, 698-706.

30. Gee W. Carotid physiology with OPG. Stroke, 1982; 13, 666-706.

31. Chilcote WA et al. DSA of the Carotid arteries, Radiol; 1981, 139, 287-95.

32. Goldstone J. Moore WS. Emergency carotid artery surgery in neurologically unstable patients, Arch Surg; 1976. 111, 1284-91.

33. Ferguson GG. Intraoperative monitoring and internal shunts. Are they necessary in Carotid endarterectomy Stroke; 1982, 13, 287–289, Stroke; 1983, 14; 116-117

34. Thompson JE, Complications of endarterectomy and their prevention. World J Surg. 1979; 3. 155-165.

35. Baker WH et al. Carotid endarterectomy. Is an indwelling shunt necessary, Surgery; 1977, 82, 321-6, Whitney DG, Carotid artery surgery without an indwelling shunt, 1977 consecutive cases, Arch Surg. 1980; 115, 1393-1399. Bland JE Lazar ML. Carotid endarterectomy without shunt, Neurosurg, 1981, 8, 153-157.

36. Crowell RM. Ojemann RG. Results and complications of carotid endarterectomy, Stroke and extracranial vessels. NY. Raven Press. 1984. pp. 203-212, Sundt TM et al. Correlation of CBF and EEG changes during carotid endarterectomy. Mayo Clin proc. 1981; 56, 533-543.

37. Ferguson GG. Carotid endarterectomy. To shunt or not to shunt. Arch Neurolog. 43, 1986. 615-617.

38. Ferguson GG et al. Carotid endarterectomy. Evaluation of results in relation to intraoperative monitoring in 282 consecutive cases. Abs 54, Am Assoc of Neurolog Surgeons Atlanta Apr 23, 1985.

39. Markand ON et al. Monitoring of Somatosensory evoked responses during carotid endarterectomy. Arch Neurol, 1984; 41, 375-8.

40. Ferguson GG, Extracranial carotid artery surgery, Clin Neurol. 1982; 29, 543-574.

41. Sundt TM Jr. Ischaemic tolerance of neural tissue and need for monitoring and selective shunting during carotid endarterectomy. Stroke; 1983. 14, 93-98.

42. Ricotta JJ et al. Determining criteria for shunt placement during carotid endarterectomy. EEG vs back pressure. Ann Surg; 1983, 198, 642-5, Cerebral haemodynamics in Carotid Surgery, Acta Neurol Scand Suppl 1973;52; 1-84.

43. Ojemann RG et al. Carotid endarterectomy. To shunt or not to shunt, Arch Neurol 43, June 1986. 617-618

44. Ojemann RG. Surgical management of cerebrovascular disease. Published in Baltimore Williams and Wilkins, 1983.

45. Baker WH et al. Carotid endarterectomy without a shunt. The control series. J Vasc Surg. 1984; 1. 50-56

46. Hachinski V. Shunting during carotid endarterectomy Arch Neurol. 43. June 1986. 618.

47. Hemmingssen R et al. Surgical treatment of stenosis of the ICA. A series of 257 patients, Ugeskr Lager 140;1727-1732, 1987. Less CD Hertzer NR, Post-operative stroke and late neurological complications after carotid endarterectomy, Arch Surg, 116; 1561-1568. 1981. White JS et al, Morbidity and mortality of carotid endarterectomy, Arch Surg; 116, 409-412, 1981.

48. Naritomi H et al. Effects of advancing age on regional cerebral blood flow, Arch Neurol; 36, 410-416, 1979.

49. Bernstein EF et al. Life expectancy and late stroke following carotid endarterectomy. Ann Surg. 198; 8--80-86, 1983.

50. Thompson JE et al. Carotid endarterectomy, Ann Surg 1976; 184, 1-15

51. De Bakey et al. Cerebral arterial insufficiency, Ann Surg. 1965, 161, 921.

52. Bove EL et al. Hypotension and hypertension as consequences of baroreceptor dysfunction following carotid endarterectomy. Surgery. 1979; 85, 633-7.

53. Towne JB Bernhard VM. The relationship of post-operative hypertension to complications following carotid endarterectomy, Surgery;1980;88. 575-80.

54. Ackroyd N et al. Carotid endarterectomy. Long term follow-up. J Cardiovasc Surg. 27; 1986. 418-425.

55. Stoney RJ String ST. Recurrent carotid stenosis. Surgery. 1976; 80 (6): 705-710. Imparato AM et al. Intimal and neointimal fibrous proliferation causing failure of arterial reconstruction. Surgery: 1972; 72; 1007-17.

56. Hertzer NR et al. Recurrent carotid stenosis after carotid endarterectomy, Surgery; 1979, 149, 360-4. Thomas M et al: Recurrent carotid artery stenosis following endarterectomy. Ann Surg; 1984; 200. 74-9.

57. Rutherford RB, Ross R. Platelet factors stimulate fibroblasts and smooth muscle cells quiescent in plasma serum to proliferate. J Cell Biol. 1976; 69; 196-203.

58. Canadian co-operative study group. A randomised trial of aspirin and dipyridamole in threatened stroke. N.E.J.M. 1978; 299. 53-9.

59. Bauer RB et al. Joint study of extracranial arterial occlusion. JAMA; 1969, 208, 509-18.

60. Thompson et al. carotid endarterectomy, Ann Surg. 1976. 184. 1-15, Allen GS, Preziosi TJ. Carotid endarterectomy. A prospective study of its efficacy and safety. Medicine (Baltimore) 1981; 60. 298-309. DeWeese JA et al. Results of carotid endarterectomy for TIAs—five years later. Ann Surg. 1973 178, 258-64. Sergeant PT et al. Carotid endarterectomy for cerebrovascular insufficiency. Long term follow-up of 141 patients followed upto 16 years. Acta Chir Belg. 1980; 79. 309-16.

61. Whisnant JP. The Canadian trial of Aspirin and Sulfinpyrazone in threatend stroke. Am Heart J. 1980; 99; 129-30.

62. The American Canadian co-operative study group. Persantin Aspirin trial in cerebral ischaemia. End point result. Stroke; 1985, 16, 406-15.