Transient Cerebral Ischemic Attacks, Association with Hypertension and Atherosclerosis.

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SUMMARY

Six cases of Transient cerebral Ischaemic attacks (TIAs) within the carotid system territory and 10 cases with TIAs in the Verterbal basilar system territory were followed up, salient clinical features, differential diagnosis, complications and their mechanism have been described.

Transient cerebral ischaemic attack (TIA) is defined as an episodic focal cerebral vascular insuffiency and retinal ischaemia (amaurosis fugax) with abrupt onset and rapid clearance. Each episode lasts seconds, minutes and occasionally an hour or more, resolves in 24 hours completely and is due to an alteration in the cerebral circulation to the limited region of the brain either in the carotid or vertebrobasilar arterial territory. Minor stroke is defined similar neurological symptoms lasting upto one week while major longer than one week.

Clinically TIA presents as either transient monocular blindness (TMB or Amaurosis fugax) or transient cerebral ischaemic attacks (TIA). In TMB there is periodic loss of the vision in one eye which may come as an episodic "snowing up" or like a "curtain coming down" which lasts only a few seconds or minutes, one hour or more. Permanent loss of vision may indicate embolization from the carotid bifurcation.

Carotid TIA manifests as weakness, or numbness of the face, arm or leg lasting for few minutes often accompanied by contralateral transient ocular blindness (TMB or Amaurosis Fugax) with or without carotid bruit, The classic but uncommon finding is the presence of glistening cholesterol crystal emboli in the retinal arterioles or often an embolus at the bifurcation (Hollenhorst plaque).

Studies have shown that carotid TIA are followed by a complete stroke in 23-45% of patients after one, three and five years. The risk of stroke is greatest in the first two months.

When build up of symptoms is slow migraine complex may be considered (Fisher, 1980). Monocular visual loss may also be complicated by cranial arteritis, papilloedema, glaucoma and retrobulbar neuritis, blood disorders such as polycythaemia (Millikan et al 1960) thrombocytosis (Preston et al 1979) thrombocytopenic purpura (Mundall et al 1972).

Cardiac lesion e.g. prolapse mitral valve, (Shapiro et al 1981) ; atrial myxoma (Sandok et al 1980) bicuspid aortic valve, (Pleet et al 1981) particularly with an episode lasting longer than hour (Harrison et al 1978). In practice artery-to-artery embolism accounts over 90% of all cases (Heyman et al 1979, Parkin et al 1982). Carotid thrombosis and athero sclerosis is the main source of platelets embolism.

Transient visual disturbance may also be caused by vertebrobasilar insuffieiency which is usually associated with turning or rotation of the head. Transient vertigo on turning of the neck to one side or in its hyperextension is caused by vertebral artery insufficiency due to pressure of the transverse process of the Atlas Head facing straight, the vertebral artery is passing through transverse process of the vertebra. The internal carotid artery passes in front of the transverse process of the atlas. Head turned to the right, the

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vertebral artery is carried forward with the transverse process and the angle, between the portion above and below the vertebra, becomes acute.

The common symptoms of vertebrobasilar TIAs are: Vertigo, diplopia, hemianopia, paraesthesiae around the face and the lips, cerebellar ataxia and slurred speech, numbness of both arms or legs or of one side. A variant picture is the "subclavian steal syndrome", in which symptoms are produced by exercise of the arm which increases the demand for blood, then the narrow subclavian artery "Steals" blood from the vertebral artery. Taka Yasu disease (arteriopathy of unknown origin) is also associated with TIAs.

These clinical features are being illustrated herewith by the presented cases. There is no other report in the Pakistan medical literature.

PATIENTS AND CLINICAL METHODS

Six out of 120 hypertension cases were followed-up who had TIAs in the carotid territory during the past five years (about 1% per year).

One of the cases had established hypertension, transient coronary ischaemic attacks, TIAs, paroxysmal supraventricular tachycardia associated with cervical spondylosis. Investigation in London revealed prolapse mitral valve on echocardiogram and angiography showed 50% stenosis of diagonal branch of left anterior descending artery and irregular right coronary artery. Two cases of this series had ischaemic heart disease and one had coronary death without cerebral stroke. One normotensive had supraventicular tachycardia with heart block and TIA during the attack. Two hypertensives had significant carotid bruit.

One established hypertensive case had TIA with aphasia and right hemiplegia which improved within 48 hours but residual dysphasia cleared in 3 weeks time.

10 cases out of 120 cases of cervical spondylosis with transient vertebro-basilar insufficiency were followed up during the past 10 years. Four cases of this group had hypertensive disease as well and two of them had healed cardiac infarction. One case had subclavian stenosis (arteriopathy — Taka Yasu type disease.).

The clinical findings were analysed carefully and followed up regularly — cases in this study included were fulfilling the criteria of TIA either in the carotid or vertebro-basilor territory. All cases were x-rayed for cervical spondylosis with special emphasis on apophysial joints and routine examination including: complete blood picture, lipids, electrolytes, ECG were arranged and special investigation such as carotid and vertebral angiography, brain scan, Electroencephalogram and aortic arch angiogram were also arranged when considered essential.

TABLE — 1

AGE AND SEX INCIDENCE

Age	Number of Cases	Sex
45 — 55	8 cases	Male
56 — 65	5 cases	Male
66 — 70	3 cases	Male

TABLE — 2

CAROTID TRANSIENT ISCHAEMIC ATTACK PRESENT SERIES.

1.	Amaurosis Fugax	4 cases
2.	Transient Paresis (Hemi)	3 cases
3.	Hemi paresis with paraesthesiae	3 cases
4.	Dysarthria	3 cases
5.	Monocular visual symptoms	3 cases
6.	Binocular visual symptoms	1 case
7.	Loss of consciousness with supraventricular tachycardia-cum-block	1 case
8.	Supraventricular tachycardia with prolapse mitral valve.	1 case

TABLE — 3

VERTEBRAL BASILAR TRANSIENT ISCHAEMIC ATTACK PRESENTING SYMPTOMS IN 10 CASES (PRESENT SERIES).

Vertigo	5 cases
Ataxia	2 cases
Drop attack	1 case
Hyperacusis	1 case
Tinnitus	1 case

ILLUSTRATIVE CASES

(A) CAROTID TRANSIENT ISCHAEMIC ATTACK :

Case — 1.

Male aged 50 years note-examiner was seen at this Medical Centre on 24-9-1974 with complaint of intermittent dysphasia, profound weakness of the right arm, less weakness of the right leg associated with paraesthesiae preceded by palpitation, retrosternal oppression and sweating lasting for 2-5 minutes, occurring 2-3 times a day and complete recovery in between attacks. He was having these episodic attacks at home for the past 3 days. He was conscious during these attacks. These attacks were followed by light headache. Past history revealed no abnormality except suffering from high blood pressure: 180-160/110-100 mm Hg noted on 9-6-1973 and he was on antihypertensive therapy since then.

On examination during attack he was apprehensive and frightened unable to lift the right arm and the right leg, cranial nerves were normal. Examination of the nervous system revealed no significant abnormality except expressive dysphasia. Sensory components were normal. Deep and superficial reflexes were present. After recovery of these attacks he described that he felt paraesthesiae of the right arm and leg, palpitation and retrosternal oppression followed by difficulty in speech and inability to lift right arm and the right leg. Left carotid pulsation was weak and no bruit was heard on both the carotids. His BP was 150/90 mm. Hg., pulse rate with regular rhythm was 100 per minutes. Heart and lungs were within normal limit, systemic examination revealed no abnormality.

INVESTIGATION

ECG and x-ray chest x-ray skull were normal, Blood count: Urea, sugar, electrolytes, lipids were normal. Special investigation: Electro encephalogram on 5-11-1974 showed "slow activity on the left side of probable vascular origin-relative ischaemia of the left frontal lobe" (Courtesy Jinnah Post-Graduate Medical Centre EEG Unit and Professor of Neurosurgery).

Carotids angiography was advised but due to some technical difficulty was not done. Brain Scan showed no abnormality (Courtesy Jinnah Post-Graduate Medical Centre Radioistope Unit).

In view of these findings and transient attacks of dysphasia, and right hemiparesis more marked in the right upper limb associated with hypertension he was diagnosed as a case of left carotid insufficiency probably due to atheromatous lesion and platelets — fibrin embolism in the middle cerebral artery. Neuro-Surgeon was consulted who advised medical management to be continued.

He was put on antiplatelets and antihypertensive regime keeping BP at 150-140/90-80 mm. Hg, low fat and high fibre diet, nitroglycerine for his chest oppression. He was followed up regularly first weekly for two months then bimonthly and finally monthly.

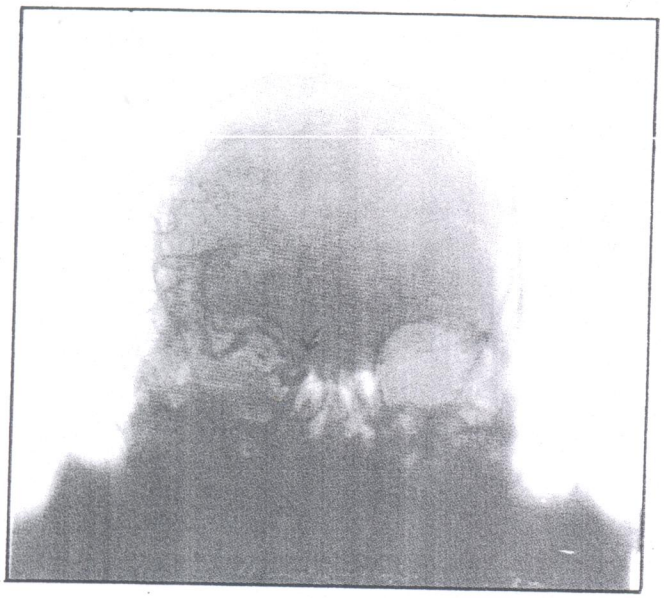
FOLLOW-UP — In spite of treatment he was having these transient cerebral ischaemic attacks 2-3 times a day lasting 2-5 minutes for further two weeks and then the attacks subsided with some residual speech defects for which speech therapy was arranged. He made a good recovery and complete stroke was averted during follow up period of three to six months on medical management.

His BP in 1975 was fluctuating between 180/100-150/90 and 140/80 mm. Hg. Repeated ECG, x-ray chest and CNS during follow up and in 1975 revealed no change. He remained well with some dysphasia. There was no deterioration in CNS system and no further TIA's were observed.

During 1976-1982 follow up his BP was well maintained between 160/90-150/90 mm. Hg. He had antihypertensive drugs: Methyldopa (Aldomet) later on Propronolol (Inderal) 20 mg. B.D. Dipyridamol (Persantine 50 mg. T.D.S. Aspirin 300 mg. daily and Isordil 5 mg. Sublingually T.D.S. for his anginal pain. His repeated ECG showed no evidence of infarction but revealed some ischaemic changes. He died suddenly at home with coronary thrombosis but no hemiplegia was observed during follow up period of 8 years. He was a hypertensive case and possibly had atherosclerosis both of the coronary and carotid arteries associated with transient cerebral and coronary ischaemic attacks.

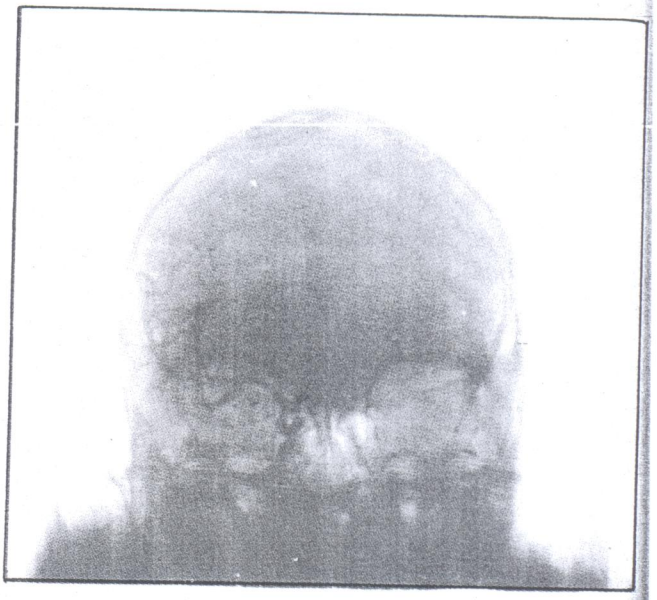
Case — 2.

Male aged 50 years Canteen Cook was seen at this Medical Centre on 29-3-1981 morning at



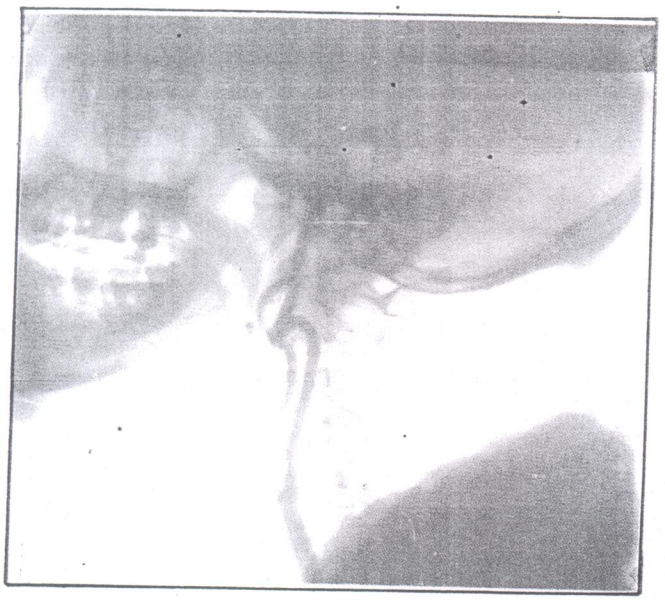
CASE NO. 2. FIGURE (a)

Right carotid angiogram (arterial phase) A. P. view showing non filling of anterior cerebral artery.



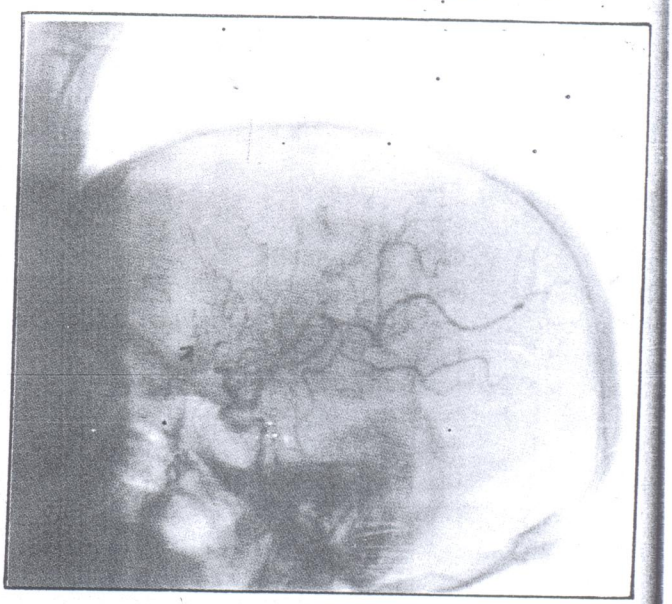
CASE NO. 2. FIGURE (b)

Right carotid angiogram after cross compression showing non filling of anterior cerebral artery (A. P. view).



CASE NO. 2. FIGURE (c)

Right carotid angiogram (neck).

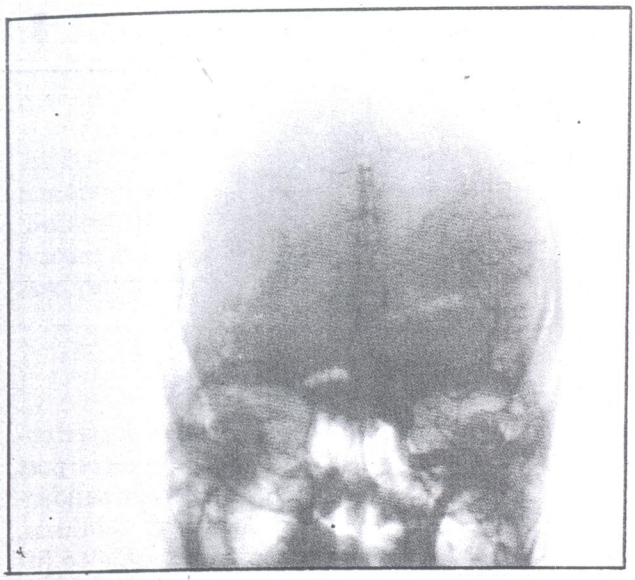


CASE NO. 2. FIGURE (d)

Right carotid angiogram (Lateral view) cerebral artery filled from other side.

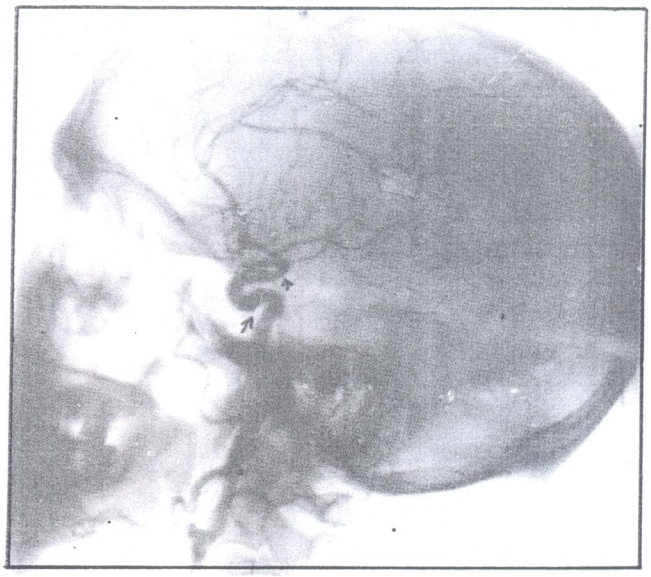
8.00 A.M. who complained that while at work in the early monring at 6.00 A.M., he felt suddenly loss of power of the left arm, left leg associated with visual hallucination and transient snowing in the left eye and expressive dysphasia for few minutes with complete recovery. He had ten such transient attacks of left hemiparesis, left visual hallucination and ocular blindness for an hour, preceded by palpitation and retrosternal oppression and then complete recovery in between attacks as described by the patient.

ON EXAMINATION AT 8.00 A.M. – there,



CASE NO. 2. FIGURE (e)

Left carotid angiogram showing anterior and middle cerebral arteries.



CASE NO. 2. FIGURE (f)

Left Carotid Angiogram (Neck).

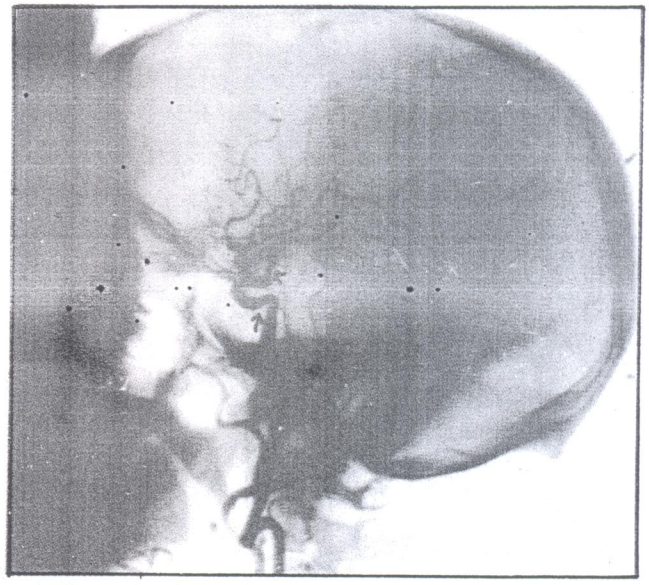
was no evidence of any deficit in the central nervous system. His pulse was 1 00 p.m. regular in rhythm and B.P. was 320/120 mm. Hg. Heart and lungs and other system examination revealed no abnormality. Right Carotid pulsation was weaker than the left without carotid bruit.

INVESTIGATION – Blood examination for sugar, urea, lipids and electrolytes was normal and so was urine. ECG showed no evidence of rhythm disorder, and ischaemia, x-ray chest was normal so was x-ray of the skull. Cervical spine x-ray showed cervical spondylosis.

Inview of the above clinical findings TIA in the right carotid artery territory with hypertension was diagnosed possibly due to atherosclerosis of the right carotid and subsequently platelete fibrin emboli. (artery to artery embolism). The contralateral visual disorder in the left eye was an unusual finding, possibly as a sympathetic mechanism. He was put on Dipyridamol (Persantin) 50 mg. T.D.S., Aspirin 300 mg. T.D.S. Isordil 5 mg. sublingually T.D.S. and Methyldopa (Aldomet) 250 mg. B.D. to keep B.P. at a reasonable level and to avoid any disturbance in the compensatory autoregulation of the cerebral vascular circulation. He was put on low fat and high fibre diet with low salt in take.

SPECIAL INVESTIGATIONS

His brain scan and electroencephalogram



CASE NO. 2. FIGURE (g)

Right carotid angiogram. Lateral view showing non filling of anterior cerebral artery, narrow and atheromatous internal carotid artery intracranially particularly above carotid syphon.

were within normal limits—Carotids angiography was arranged and reported as: "Right common artery is punctured just near the bifurcation and contrast medium is injected

On the right side the internal carotid artery remains slightly narrowed through out the extra

cranial portion. The internal carotid artery remain narrow intracranially particularly above the carotid syphon.

Throughout examination anterior cerebral artery is not visualised although same was repeated 3 times with cross compression. (Fig. 1 a, b, c).

Left common carotid is punchtured and contrast medium is injected. The internal carotid artery is normal – normal intracranial circulation of the internal carotid artery. Normal anterior and middle cerebral artery. The right anterior cerebral artery is filled from the left side. Cross compression film is also taken but this does not improve the situation".

CONCLUSION

As the intracranial portion of right carotid is narrow throughout examination, this may be due to ahteromatous disease. Angiographic evidence supported the clinical diagnosis of TIA in the right carotid territory.

On 1-4-1981 B.P. 160/85 and he was feeling better and there was no further TIAs or any manifestation of hemiplegia. He was followed up, regularly, weekly initially for first two months, then bimonthly and monthly and was stable uptil January 1983. His B.P. was varying between 170/130, 150/90, 140/90 mm. Hg. ..... ng.

On 8-10-1981 his BP was 230/130, and complain of vertigo as he had defaulted antihypertensive treatment. He was put on Viskaldix 1 tablet daily. Persantin 50 mg. T.D.S. Aspirin 300 mg. daily Isordil 5 mg. Sublingually as he used to get anginal attacks off and on. He was finally put on Adalat 10 mg. T.D.S., Persantin 100 mg. B.D. and Solublė Aspirin 150 mg. daily, followed by low dose (75 mg.) Aspirin indefinitely.

COMMENTS

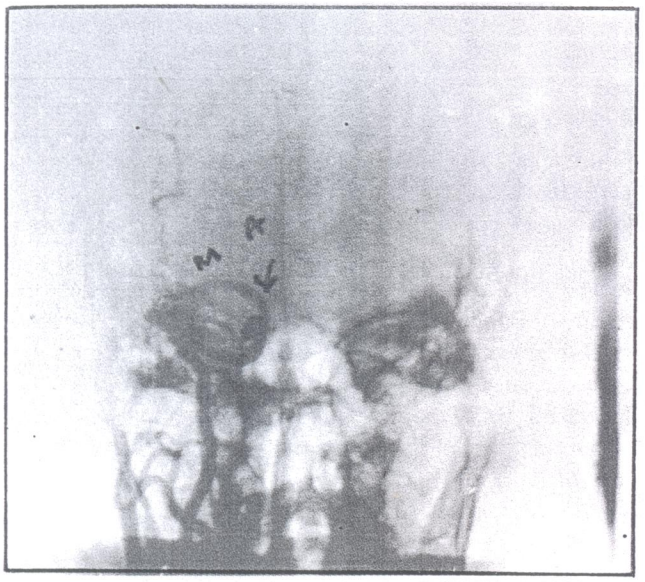
This case resembles the case of a house wife aged 66 described first from London in 1953 by East Cott, Pickering and Rob – In which case carotid angiogram showed atheromatous obstruction at the bifurction of the common carotid artery, delayed filling of internal carotid artery and non filling of the anterior cerebral artery at its origin and was filling from other side. She had 33 major attacks lasting 10 minutes to half an hour in all of which there was loss of vision left eye, right hemiparesis and aphasia preceded by palpitation and heavy feeling in chest. She wa successfully operated on occluded segment o internal carotid artery with reconstruction by direct anatomosis between common and interna carotid artery.

In the presented case compensatory mechanism within the circle of Willis had played well and on medical regime complete stroke was averted follow up exhibited no further TIAs, and carotid stenosis was less than 70% therefore surgery was deferred in this case.

Case – 3.

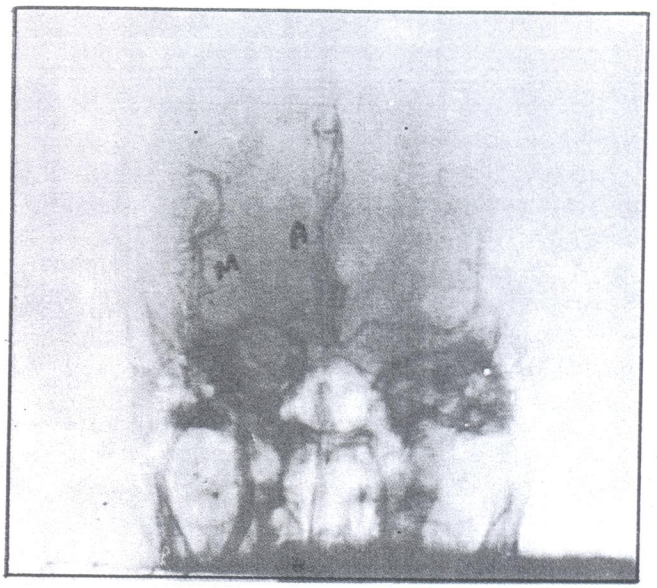
Male employee aged 53, a known hypertensive and on antihypertensive treatment since past 2 years, well till 28-5-1983 evening when suddenly dropped down following feeling of giddiness, vertigo and ataxia to left and was unable to lift left arm and leg associated with loss of speech. Few hours later he felt snowing in the left eye and weakness of the right arm. Motor power of the limbs and snowing in the left eye improved partially within 24 hours but speech defect persisted for 48 hours.

He was seen at the P.S.P.C. Medical Centre on 3-5-1983 A.M. On examination he was found walking with support and ataxic to the left side. He was dyphasic can utter one or two words = Yes or No. Cranial nerves were normal. Left upper limb was weaker than the lower. Right hand grip was weak and was unable to write. He was right handed. There was no sensory defect. Deep reflexes were present and plantars were flexor. There was no carotid bruit in the neck. His BP was 170/110. Cardiovascular other systems were normal. ECG showed normal limit. He was diagnosed as bilateral carotid TIAS due to platelet emboli from atheromatous carotids to intra cranial vessels (artery to artery emboli). He was put on Aspirin 300 mg. three times a day after meals, Dipyridamole (Persantin) 100 mg. morning and evening on empty stomach half hour before meal and Nifedipine (Adalat capsules) 10 mg. three times a day. On this regime he improved much in three days, the stroke in evolution was arrested and complete paralysis was averted during the follow up period of two months. His speech improved to near normal – he was able to walk freely. He showed writing defect in spite of complete recovery of motor power and exhibited construction apraxia and spatial agnosia = parietal lobes syndrome.



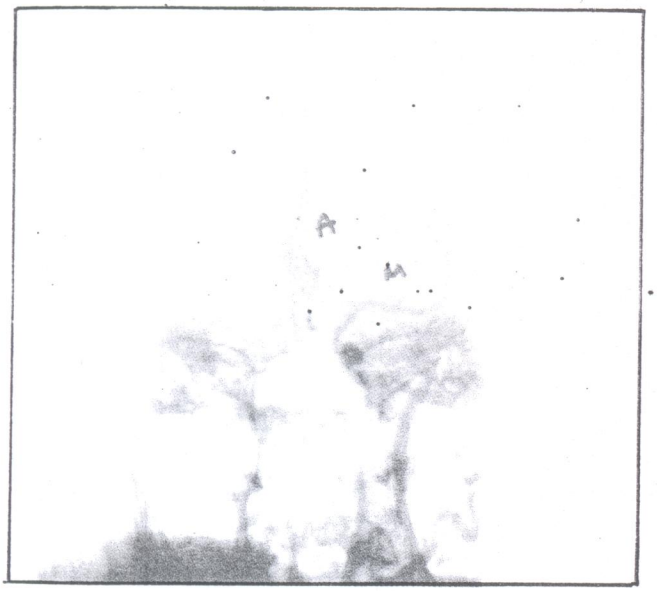
CASE NO. 3. FIGURE 2 (a)

Right carotid angiogram A. P. view showing non filling of anterior cerebral artery.



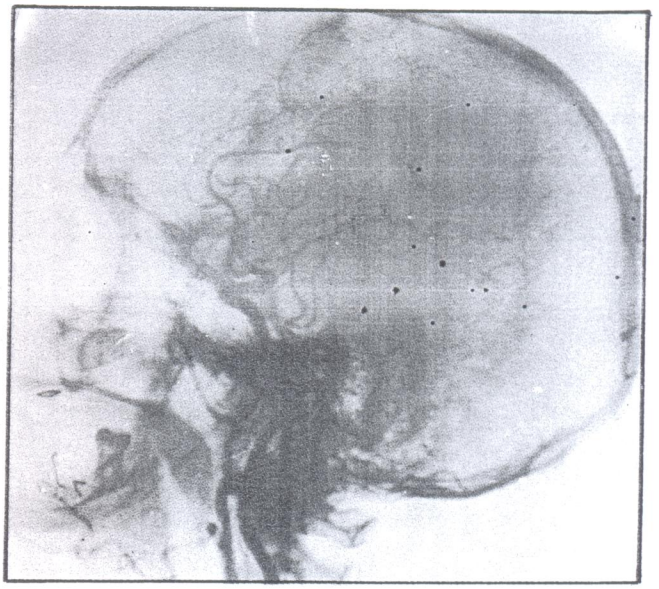
CASE NO. 3. FIGURE 2 (b)

Cross compress shows filling of right anterior cerebral artery.



CASE NO. 3. FIGURE 2 (c)

Left carotid angio – showing filling of anterior and middle cerebral artery (A. P. view).



CASE NO. 3. FIGURE 2 (d)

Left carotid angiogram neck (Lateral view) showing normal internal and external carotid.

Special investigation: EEG showed bilateral temporoparietal lesion (probably due to ischaemia).

Bilateral carotid angiography was arranged at Karachi and reported as : ; :

"Left carotid angiogram is attempted first. the right carotid artery was punctured at the level of C5-6. The inracranial circulation of left carotid artery is normal. No displacement or space occupying lesion.

However, ont the right side, there is evidence of spasm specially in right external carotid artery and also in right anterior cerebral artery.

On the AP film of the right carotid angiog-

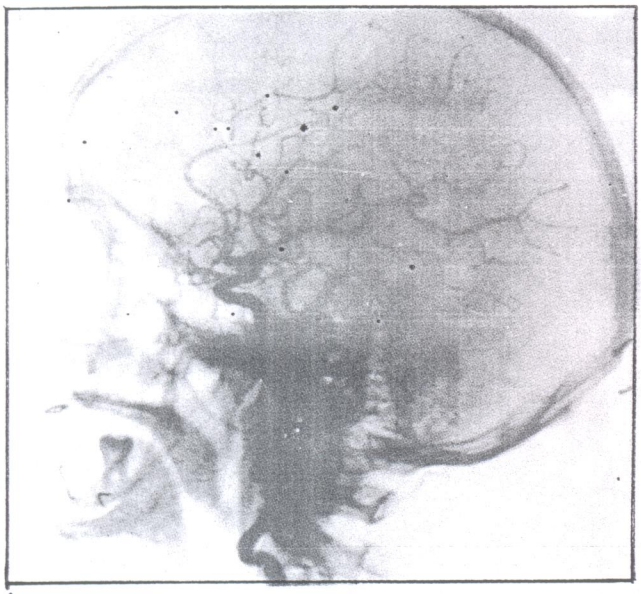
ram, the anterior cerebral artery could not be seen but it is outlined satisfactorily on the lateral view. A film is taken with cross compression on the left carotid artery and this outlines the right anterior cerebral artery completely.

On the lateral view of right carotid angiogram, the right external carotid artery is not outlined but it is fully outlined in the AP projection.

No evidence of abnormality in the intracranial circulation of right carotid angiogram as well.

CONCLUSION

"Apart from some spasm in the right anterior cerebral artery and right external carotid artery, no other abnormality is seen". (Fig. 2. a, b, c.)



CASE NO. 3. FIGURE 2 (e)

Right carotid angiogram — external carotid is not outlined, suggestive of atheromatous changes and spasm.

He felt much better after angiography and speech and other residual neurological disorder improved further is suggestive of spasm plus platelete aggregation and its clearance possibily by the angiographic procedure. He was advised to continue Aspirin 75 mg. and Dipyridamole 75 mg. and Nifedipine (Adalat capsule) 10 mg. three times a day for hypertension indefinitely to avert stroke and coronary thrombosis and to control hypertension.

COMMENT

These attacks of TIA of the brain in the carotid system resembled the "TIAs of the heart". Many patient with coronary atherosclerosis experience spontaneous non exertional angina, both intermittent vasospasm and platelete aggregation have been implicated (Hirsch, 1981). When coronary arteriography is performed during a period of spontaneous coronary flow reduction, a non-opacified intracoronary mass was observed in the region of stenosis which was dislodged by vigorous injection of the arteriographic dye. It appeared to disperse the platelet aggregation and no vessel occlusions were observed to occur down stream.

Blood flow through experimental stenosed coronary arteries in dogs were monitored where 80% stenosis was produced by plastic ring constriction. This resulted in intermittent transient reductions of blood flow. Antiplatelet drugs such as Ibuprofen, Indomethacin and Aspirin abolished the flow reduction, where as vasodilators had no effect. Heparin blocks "red thrombus" formation but does not impair platelet aggregation.

During low blood flow through stenosed artery, pinching the stenosed artery suddenly restore normal blood flow. This experiment support and confirms the platelet aggregation plus spasm of intracranial arteries in TIAs. It is now agreed that platelet aggregation and spasm may occur in sequence. It is well known that cerebral TIAs can result from platelet aggregation and this probably happened in the presented case.

Platelet aggregation releases thromboxane A_2, an extremely potent vasconstrictor. Aspirin even in low dose of 40 mg. every 48 hours can prevent this release of vasconstrictor by preventing aggregation of platelet.

Case — 4.

CAROTID OCCLUSION PRESENTED AS DISSEMINATED SCLEROSIS

A female aged 49 was seen in 1949 at Queen Square National Hospital London under care of Neurologist with the complaint of blurring of vision left eye followed by, 3 months later, speech defect, six weeks later her right arm become useless associated with frontal headache and then right leg become weak.

Examination showed paralysed right arm, weak right leg and right lower facial weakness. She had right hemianaesthesia. Right plantar was extensor. B.P. was 135/90. Left carotid pulsation and left temporal pulsation were absent. His W R was negative. EEG showed left fronto-sylvian lesion, cerebral angiography — showed left common carotid occlusion — possibly due to atheromatous lesion and artery to artery emboli. This case initially was misdiagnosed and referred as disse-minated sclerosis a common neurological disorder in U.K. and inview of slow evolution and progressive neurological disorder.

(B) CERVICAL SPONDYLOSIS AND TIAS IN VERTEBRO-BASILAR TERRITORY

Case — 1.

A manager aged 58, used to get episodic attacks of giddiness, unsteadiness, vomiting deafness, tennitus of the left ear especially in the morning on getting up which were first considered to be due to inner ear (MENIERE'S) syndrome. He used to get paroxysmal hypertension and used to count his pulse with the pulsation in the ear. He had dysphagia to solids. Clinical and biochemical investigation were within normal limit, except spondylosis of cervical 4-7 vertebrae with large sharp hooked anterior ostophytes (3-4-67 vertebrae) pressing on the oesophagus. Anterior ligment was dense and ankylosed at anterior surface of cervical 3-7 vertebrae suggestive of rheumatoid arthritis as well (Fig. 3). He was also investigated for pheochromocytoma without any conclusive biochemically and radiologically evidence. He was given Meniere's syndrome treatment without any effect for one year. Temporal artery biopsy for Giant cells arteries was inconclusive.

It was considered that his cervical spondylosis was interfering vertebral artery circulation associated with brain stem transient ischaemia on change of position of the neck. He was prescribed cervical collar. Few months later his attacks were subsided which supported the clinical diagnosis of TIAS in the vertebrobasilar system.

Case — 2.

A senior executive manager aged 65 used to get vertigo at the time of going to bed whenever his head and neck were turned to the right. There was no such attack when his neck is kept straight or to the left while going to bed.

Examination revealed no abnormality in the nervous system and cardiac vascular system. He had coronary thrombosis 12 years ago but recent ECG showed no evidence of infarction or ischaemia — His x-ray cervical spine showed advanced osteoarthritis — It was considered that on head turning to right his vertebral artery circulation was impaired due to cervical spondylosis. He was prescribed soft night collar to the neck to keep his neck straight while going to bed and attack was relieved and subsequently he was advised to keep neck straight while going to bed without night cervical collar. This posture stopped the nocturnal vertigo on head rotation while going to bed.

Case — 3.

Another case aged 45 years used to get vertigo when neck as turned to left side while going to bed. His x-ray neck showed cervical spondylosis. Systemic examination was negative. He was advised to keep neck straight while going to bed and thus attacks were stopped.

Case — 4.

Carpenter aged 60 used to get vertigo on looking up associated with sweating while at work. Examination central nervous system, cardiovascular system including ECG and other system were normal except cervical spondylosis. He was advised to avoid looking up or use cervical collar to neck, because due to change of position of his neck vertebral arteries circulation was impaired due to cervical spondylosis.

Case — 5.

General Manager aged 60 used to get vertigo on turning neck to right side. He had coronary thrombosis 10 years ago but his recent ECG was normal and there was no evidence of arrhythmia or block. His cardiovascular and central nervous system were normal. His x-ray cervical spine showed cervical spondylosis. He was prescribed cervical collar to his neck and no further attacks

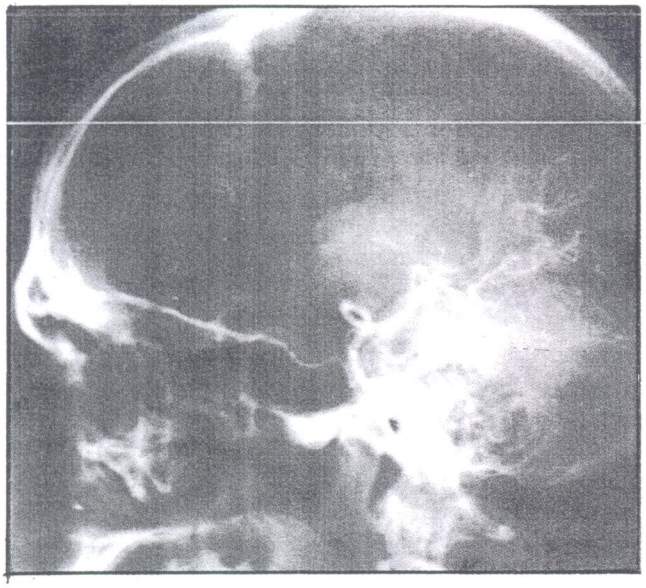


FIGURE NO. 4.

Vertebral angiogram with cervical osteo arthritis the left vertebral artery is continued as basilar artery after looping around transerse process of the atlas.

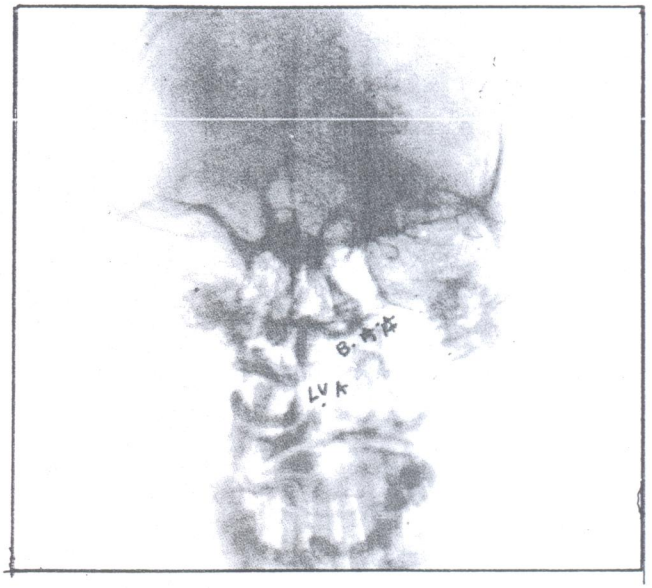


FIGURE NO. 4 (b)

Left vertebral arter angiogram, A. P. view, the artery is continued as basilar artery.

were experienced by him and was advised to avoid head turning to side when he is not in the cervical collar. Such cases of healed coronary lesion case with good collateral circulation in the coronary system demands ambulatory ECG monitoring to identify the arrhythmias. He used to get short runs of ventricular tachycardia and sometimes bidirectional ventricular extra systoles which were well controlled on medical management.

Case — 6.

Carpentor aged 55 used to complain giddiness — vertigo and snowing before eyes intermittently while at work especially on head rotation. On examination of eyes and central nervous and cardiovascular and other systems were within normal limit except high B.P. 180/110 mm. Hg. In view of radiological evidence of cervical spondylosis and apophysial joints arthrosis with posterio-lateral osteophytes vertebral artery insufficiency was suspected.

Bilateral carotid angiograms were normal. Bilateral vertebral angiography was reported, "a very difficulty procedure = left vertebral artery was showing looping around the transverse process of the atlas instead of crossing over it and the right vertebral artery could not be catherterised in spite of repeated attempt (Fig. 4.).

In this case the left vertebral artery continued as basilar artery being larger than the right. The later being small could not be catheterized even by repeated attempts, Gowers pointed out that the left vertebral artery is larger than the right, the latter being very small in some person and the basilar artery is a continuation of the left vertebrate artery. This variation is important and can cause severe disproportionate effects on cerebral circulation when one is compressed. In this case head movement can cause the impairment of cerebral circulation. His symptoms disappeared completely after angiography and is suggestive of spasm plus platelets emboli from early atheromotous lesion of the carotid — vertebral arteries systoms.

Case — 7.

Male 60 was having giddiness, vertigo on turning head associated with stiff neck. On examination central nervous system and cardiovascular system showed no abnormality except high blood pressure of 180/110 mm. Hg. Marked

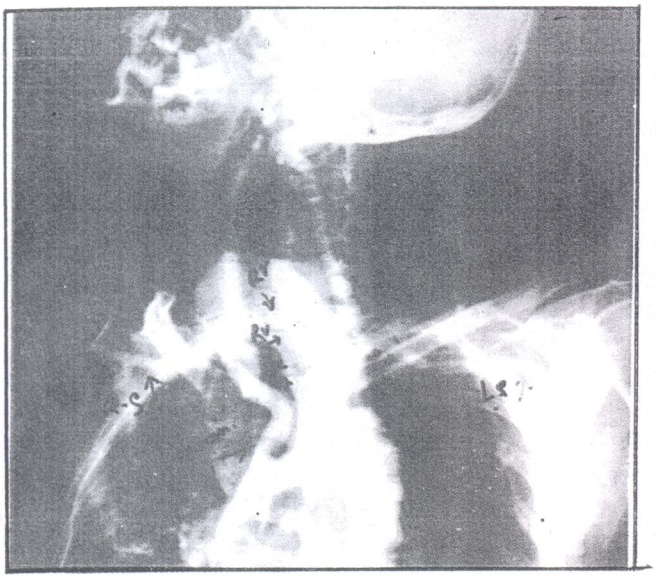


FIGURE NO. 5.

Arch aortogramm showing normal carotids, vertebrals arteries but right subclavian shows gradual narrowing from its origin towards peripherally compared with left subclavian thus going rise to reduction of blood flow peripherally.

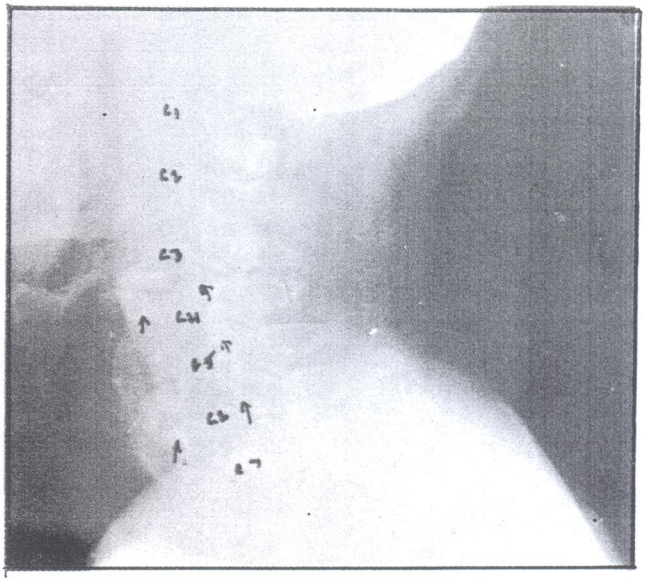


FIGURE NO. 6.

X-ray cervical spine showing aged crumbled spine with large hooked osteophytes.

carotid bruit was heard on the right common carotid artery which was hard on palpation suggestive of the atherosclerosis with reduced lumen (50% carotid stenosis). Carotid and peripheralarteries pulsation were comparatively soft. X-ray cervical spine showed crumbled aged spine and large hooked anterior osteophytes — clinically he presented symptoms suggestive of carotid and vertebro basilar insufficiency. He was advised cervical collar to neck at work and low dose aspirin 75 mg. (Fig. 6). He improved very much on this regime.

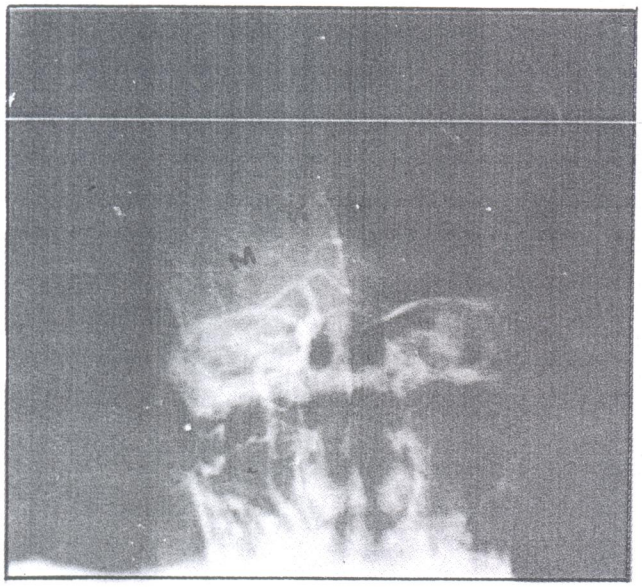
Case – 8. (Subclavian Idiopathic Arteriopathy).

Male aged 58 used to get drop and fainting attacks with vertigo — weakness fatigue and paraesthesiae right index finger. On examination central nervous system cardio vascular system was normal and so other system. There was a bruit on the right supra clavicular area with good palpable carotid. B.P. on right side was 80/60 mm. Hg. and 120/80 mm. Hg. on the left side with reduced pulse volume on the right side. X-ray cervical spine showed spondylosis. In view of right supraclavicle bruit, inequality of the pulses and difference of systolic blood pressure in the arms "subclavian stenosis" was suspected with vertebral TIA. Arch aortogram showed normal carotids, vertebrals but smooth attenuation of the right subclavian artery to below 2 mm. in the axilla thus reducing blood flow in the arm significantly suggestive of Takayasu disease — Idiopathic arteriopathy, possibly, of autommune origin (Fig. 5.).

DISCUSSION

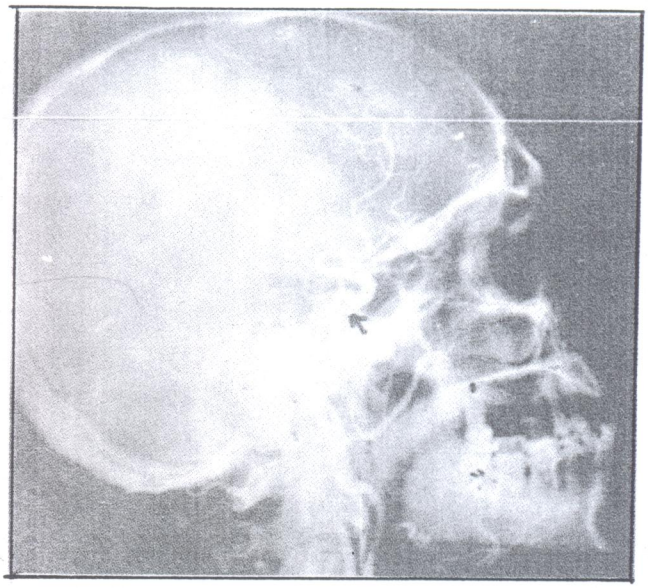
There are three separate streams of arterial blood supplying the brain: one from the basilar artery and one from each internal carotid artery separated from the another by dead points, in the posterior and anterior communicating arteries at which points the pressure of the two opposing stream is balanced.

The most common cause of internal carotid obstruction is atherosclerosis and the site is usually in the neck within a centimetre of the origin of the artery. Before total obstruction there is a gradual narrowing of the lumen which favours the establishment of collateral circula-



CASE NO. 5. FIGURE NO. 1 (a)

A. P: view of the right carotid angiogram showing partial obstruction and narrowing of the middle cerebral artery circulation is markedly poor in the middle cerebral artery.



CASE NO. 5. FIGURE NO. 1 (b)

Right carotid angiogram lateral view showing atheromatous changes in the internal carotid artery in the neck and carotid syphon.

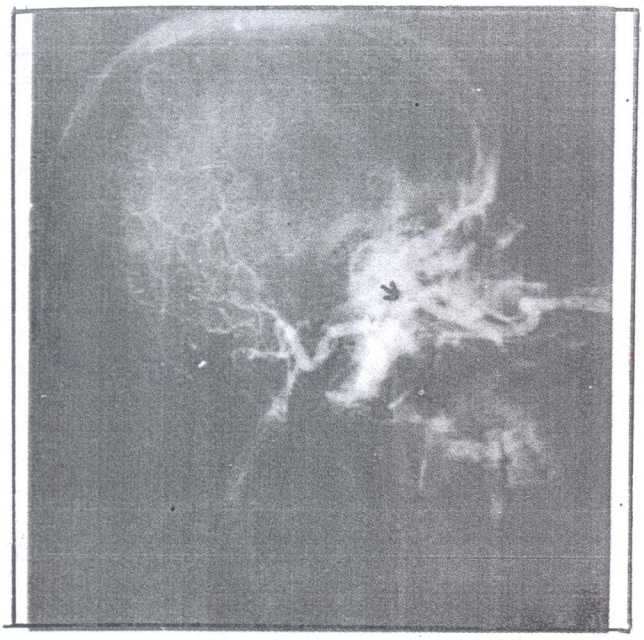
tion. We can compare this event with what happens when there is atherosclerosis of the coronary arteries, for in the heart as in the brain, occlusion may occur without infarction, if the process is slow and there is time for a good collateral circulation to develop.

In the brain there is opportunity for widening of anastomotic channels between the superficial branch of the posterior and the middle cerebral arteries and as we know from angiography the communication in orbit between branches of external carotid and the ophthalmic branch of the internal carotid artery can short circuit the obstruction of the carotid in the neck. The direction of flow in the ophthalmic artery being then reversed, it receives the blood from the external carotid above the obstruction and fills the middle cerebral artery. But the circle of Willis must provide the most important source of compensatory flow. When pressure on the obstructed side falls there must be a shift of dead point in the anterior communicating artery and a flow of blood from the unobstructed side to fill the anterior and probable the middle cerebral arteries on the obstructed side.

This had exactly happened in the presented case 2 and thus hemiplegia was averted inspite of obstruction of right anterior cerebral artery possibly by an atheromatous embolism from narrowed and atheromatous right internal carotid artery. When cerebral function remain intact after internal carotid occlusion one can assume that various compensatory mechanism have come into action, but situation is still precarious. Haemodynamic crisis is not a transient affair. There is a perpetual struggle both for the maintenance of total cerebral blood flow and for its equitable distribution, a fall of blood pressure or a temporary upset of balance between vasconstriction and vasodilation may cause local ischaemia or infarction with reversible or irreversible loss of function.

The equitable distribution of the blood to the two cerebral hemiaphere after occlusion of one internal carotid requires an anterior communicating artery of adequate diameter and also alteration of calibre in the peripheral vessels of two hemiphere must play a part.

The local effect of cerebral ischaemia is vasodilation from accumulation of CO_2 etc. The hemisphere on the obstructed side may claim more than equal share of blood with a corres-



CASE NO. 5. FIGURE NO. 2.

Left carotid angiogram lateral view showing narrowing of the internal carotid artery at pituitary fossa region and narrowing of the internal carotid artery in the carotid syphon. Atheromatous changes are seen in the internal carotid artery in the neck as well.

ponding vasoconstriction in the hemiphere on the unobstructed side. This finding is supported by observation, immediately after common carotid ligation, of abnormal waves in the electro-encephalogram in both the hemisphere and occasionally only in the hemisphere on the unobstructed side. This explains the transient loss of function in the limbs on the ipsilateral or obstructted side of the body in certain cases. This may also explain the Amaurosis-Fugax on the unobstructed side in case no. 2 of the presented series instead of amaurosis fugax on the ipsilateral or obstructed side. Therefore inview of the above facts the episodic loss of cerebral function in the presented case 1-2 is not due to any fresh vascular obstruction but to a temporary failure of the compensatory mechanism.

Attacks of transient blindness a well recognised symptoms (Amaurosis Fugax) is due either to transient retinal ischaemia, as retinal arteries has no anastomosis, a clinical effects following the happenings in the cerebral hemispheric circulation or due to passing of cholesterol crystals through retinal arteries giving rise to transient retinal ischaemia.

The effects of narrowing or occlusion of the vertebral arteries upon cerebral circulation is well known (Lord Brain 1963) but cervical spondylosis might make impairment of blood flow through both vertebral arteries and can intensify the symptoms of vertebro-basilar ischaemia due to the atheromatous lesion, which has a similar age incidence of cervical spondylosis, and makes displacement of vertebral arteries by osteophytes, a potentially serious state. Osteophytes projecting from the tips, of the inter vertebral joints (neuro-central joints) displace vertebral arteries both anteriorly and laterally. Osteophytes from the apophysial joints are particularly likely to distort the vetebral arteries between second and third cervical vertebra, because the vessels is moving laterally from here to reach the transverse process of the Atlas. Normal Vessels are well able to accommodate changes in their course. There seems no doubt that cervical spondylosis can intensify the symptoms of vertebro-basilar ischaemia due to the presence of atheromatous lesion. A supporting clinical example is reported in Lord Brain's patient who was put in collar for severe cervical myelopathy and developed symptoms of cerebellar deficiency due to impaired circulation through his vertebral arteries as his neck was more extended in collar than without it. Gowers pointed out that the left vertebral artery is larger than the right, the latter may be very small in some person so basilar artery is a continuation of the left vertebral artery. This variation has some importance that if one vertebral artery is small, compression of the other can cause severe disproportionate effects on cerebral circulation. This possibly happened in case 6 where the right vertebral artery was difficult to catheterize, while the left vertebral artery was catherized and this continued as basilar artery which was looped around the transverse process of the atlas and this defect can impair vertebral circulation on head turning. (Fig. 4.).

The effects of head movement on the blood flow through vertebral arteries have been studied by various workers (Lord Brain 1963) in the cadaver and by vertebral angiography and conclude that even in a normal person head rotation to one side imapirs or even abolish the cerebral blood flow through the atlanto-axial segment of the contra-lateral vertebral artery. Since the vertebral artery passess through the transverse

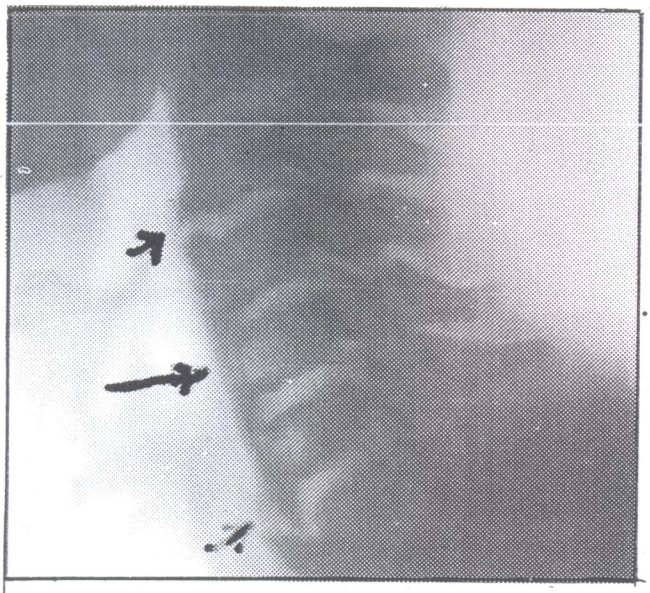


FIGURE NO. 3.

X-ray cervical spine showing advanced cervical osteo-arthritis with large anterior osteophytes with thick fibrosed anterior ligament.

process of the atlas (Brain 1963) it has to move with it, since it is relatively fixed, where it enters the foramen magnun, rotation of the head to the opposite side carrying the atlas with it, puts traction on the vertebral artery and actually pulls it out of its canal in cervical spine (Lord Brain 1963).

Angiography study shows that remarkable tortuousity may occur in the vertebral artery in its course through cervical spine, the effect of disc degeneration is to shorten the cervical spine and since the vertebral artery remains in its original length, it must become tortuous and kinked. Moreover atheroma increases the liability of the vessels to become more tortuous and narrow, hence obstructed by the head rotation. In the presence of cervical spondylosis, if patient is also atheromatous, the head rotation to one side may impair cerebral circulation through both vertebral arteries, severity of symptoms depends upon the patency of the internal carotid arteries.

Though symptoms are produced by extension of the neck but angiographic evidence are lacking and anatomical studies on the effects of head rotation suggest that the obstruction is not at atlanto-axial segment, Obviously some torsion of the brain-stem must occur on head rotation, it is difficult to assess the effects, when circulation is already impaired by vertebro-basilar ischaemia due to atheromatus lesion of these vessels, head rotation might be a contributing factor in causing transient additional failure in circulation and platelet fibrin embolism from the atheromatous lesion caused by repeated trauma to the atheromatous lesion of the vessels on head rotation and thus can give rise to transient symptoms. It had been observed by several workers and (Lord Brain 1963) that head rotation can impair circulation in the contralateral carotid artery by compression of this vessel against the lateral process of the atlas deep to which it can be seen passing and it also has been shown that kinking and stenosis of the cervical portion of a tortuous internal carotid artery may be produced on head rotation either on the ipsilateral or on the contralateral side.

Transient focal ischaemia of the cerebral hemisphere on head roation are uncommon but illustrated by a patient of right hemiplegia due to cerebral vascular deficit, who used to lose speech when head turned to the right side but could talk to some body head turned to the left, the loss of speech was possibly, due to transient cerebral ischaemia of the cerebral portion of speech area on head turning (Lord Bran 1963).

Amaurosis Fugax on the opposite side of carotid stenosis in one of the case can be explained by this mechanism on head rotation and also as a compensatory mechanism in the circle of Willis as in case no. 2.

MECHANISM OF SYMPTOMS (PATHOPHYSIOLOGY) IN V-B DISEASE

Hutchinson and Yates (1961) pointed out that normal vessels accommodate the change in the course of vertebral arteries, it is the presence of atheroma, which has similar age incidence of cervical spondylosis, which makes the displacement of the vertebral arteries by osteophytes potentially serious. Hutchinson and Yates (1961) mention, particularly in this connection osteophytes projecting from the tips of the intravertebral joints (neuro-central joints) displace the vertebral arteries both anterially and laterally. Scandinavinan worker Kovaes (1985) had first drawn attention to the fact that apophysial joints may be idented by vertebral arteries and osteophytes, from these joints, are particularly like to distort the vertebral artery between second and third cervical vertebra, as the vessel is here moving

laterally to reach the transverse process of the atlas, Cervical spondylosis can intensify the symptoms of vertebrobasilar ischaemia due to atheroma. In support Lord Brain described a case of cervical myelopathy after being put in collar developed symptoms of cerebellar deficiency. In the collar his neck was extended more than without it, an this posture, presumably, impaired vertebral arteries circulation.

Lord Brain (1963) had described a case of brain stem infarction after manipulation of the neck. One case developed left homonymous hemianopia two weeks after the neck traction. She had severe cervical spondylosis especially of upper apophysial joints. Treatment of the neck caused obstruction of the right posterior cerebral artery, possibly, due to detachment of a thrombusi at the site of compression of the one vertebral artery by an osteophytes. Lord Brain described two more cases of cerebellar deficit due to cervical spondylosis. In one case there was severe extensive cervical spondylosis and in another severe arthritis of the upper apophysial joints. The vertebral artery passess these joints on its way to the atlas.

Ford and Clark (1956) described thrombosis of the basilar artery with softening of cerebellum and brain stem due to manipulation of the neck. Yoga exercise, (Hanus et al 1977) neck manipulation, particularly in younger patient, chiropractice and osteopathic manipulation have been identified as causes of the vertebral artery damage at C2 level. Damage to the artery either by traumatic dissection or simple endothelial injury appears setting the stage of immediate apoplexy or for the development of events of recurrent nature and sometimes after a delay.

The possibilities of platelets fibrin embolisation in such cases is very real (Barnet 1979) it has been established that osteophytes at neurocentral joints may interfere with the circulation and the course of the vertebral arteries in patients with severe cervical spondylosis may intrude on these vessels with sufficient severity or to produce atheroma (Scheehan et al 1969, and Yates and Hutchinson 1961), and eventually platelets fibrin embolisation (Barnett 1979). Extra cranial portion of vertebral artery stenosis is often protected by development of an extensive collateral systems of vessels from throcevical, costo cervical and accipital arteries and remain asymptomatic.

RHEUMATOID ATLANTO-AXIAL SUBLUXATION

Atlanto-axial subluxations of rheumatoid nature may interfere intermittently with the vertebral arters as it passes through foramen magnum and produce recurrent ischaemic events (Jones and Kaufmann 1976).

PROLAPSE MITRAL VALVE

Several series have been reported in which prolapsing mitral valve was the only associated abnormality to account for the cerebral ischaemic events (Hanson et al 1978, Barnett 1976-77, 1978-1979, 1974, Hirsowitz and Saffer, 1978 Kostuk et al 1977, Wilson et al 1977).

Myxomatuous degeneration of the mitral valve once thought benign, it is now known as a cause of progressive mitral regurgitation, predisposing factor for bacterial endocarditis and is known to have an association with cardiac arrhythmia, including serious ventricular arrhythmia leading to sudden death and lately has been indentified, in association with TIAS. Thromboembolism (Platelets fibrin deposition) is thought to be the main cause. Radiological events supports emboli rather than arterial disease.

ATRIAL MYXOMA

Atrial myxoma is a rare and treatable condition, frequently has been associated with TIA (Yufe et al 1976). It is easily recognized by two dimensional Echocardiogram and by a variety of new cardiac imaging techniques. Bacterial or non-bacterial thrombotic endocarditis may be recognised first time by TIA. Embolic cerebral ischaemic events from recent myocardial infarction and rheumatic mitral stenosis tends to result in stroke when emboli are large.

CARDIAC ABNORMALITIES (MISC.)

A variety of abnormalities of heart walls, chambers, and valves are being recognised with increasing frequency as sites for TIAS or persistent and minor cerebral event, cardiac imaging and sophisticated investigation procedures are available so that the sources of potential emboli can be ascertained in cases of TIA or stroke. All cases of stroke should be submitted to a good cardiac history and examination of the heart when doubt exists about cause of TIA when carotid angiography is normal.

BLOOD DISORDER

Intermittent or persistent ischaemic events occur in haemolytical crisis = polycythaemia, sickle cell disease, thrombo cytopenia, Thrombocytosis — This also applied to contraceptive pills and high dose of steroids. Amaurosis has been observed in idiopathic thrombocythaemia, emboli passing through retina had ceased with busulfan therapy.

NON ARTERIOSCELEROTIC — vasculopathies = In young patients TIA may be due to postraumatic lesion (atheroma or endothelial damage) and subsequent platelets — fibrin embolism, granulomatous angiitis, polyarteritis, vasculitis, hypersensitive angiitis, lupus erythematosus and fibromuscular hyperplasia of arteries.

Study has demonstrated no reduction of the carotid artery blood flow or its pressure gradient change unless carotid artery lumen is reduced below 2 mm. diameter both the flow and pressure gradient fell regardless of the length of stenosis (Brice et al 1964).

FIBROMUSCULAR DYSPLASIA OF INTERNAL CAROTID ARTERIES

A well recognised cause of TIAS, is usually bilateral, involves the middle third of extra cranial part of internal carotid arteries.

Several pathological varient have been described and in most of them the primary lesion in overgrowth of media in a segmental distribution producing irregular zones of arterial narrowing, concentric rings giving rise to radiological appearance of a "String beads".

The most frequent complaint is abnormal noise in the ear synchronous with the heart beat case no. (1-V) presented this symptoms. An uncommonly loud bruit high in the neck may be only physical findings on vascular examination. Two of the presented cases exhibited this bruit high in the neck with hard arteriosclerotic carotid with TIAS.

NON-INVASIVE PROCEDURES

Venous Digital Subtraction Angiography

Digital intervenous angiography offers a relatively safe and noninvasive method of obtaining substantial information about cervical cerebral system, the visualization of carotids and other major intracranial arteries is sufficient in most cases for assessment of gross lesion such as stenosis, large ulcers, occlusion, abnormal cross flow pattern and large aneurysm.

Ultra Sound Scanning

Duplex Doppler scanning is one of the most useful techniques available for evaluation of carotid stenosis, both physiological and anatomical data can be obtained. Duplex scanner can estimate volume blood flow, the extent of arterial stenosis can be calculated by the ratio of the relative flow velocities proximal to and within the stenosis and as well as from analysis of the extent of flow disturbance distal to stenosis.

Neuclear Magnetic Resonance Imaging (NMR).

NMR imaging is a new recent technique which will have extensive application in future. This NMR imaging procedure is still in its infancy and will be a very useful noninvasive procedure in future. NMR imaging depends on the ability of strong magnetic signals, transmitted into the body to change the spine of electrons, protons in the molecules of the proteins, carbohydrate and other component of the tissues. The magnetic induced changes produce electrical signals which can be detected, amplified and used to construct image of the part of the body.

COMPLICATIONS

(1) Venous Stasis Retinopathy — was first described by Hedges (1962) Kearn and Hollenhorst (1963) and recently by Kearn et al 1979 from Mayo clinic. This type of retinopathy consists of microaneurysm, punctate areas of capillary dilatation. Small retinal haemorrhages and in severe cases gross dilation, darkening and irregularity of calibre of the major retinal veins. It resembles diabetic retinopathy and the mechanism is same: ischaemia and hypoxia but site of obstruction differs. In diabetes the obstruction is caused by altered haemodynamics in retinal microcirculation whereas in carotid occlusion the

cause is reduction in perfusion pressure from atheromatous obstruction proximal to retinal microcirculation. Diabetic retinopathy is bilateral whereas venous retinopathy in unilateral unless there is bilateral occlusion of the carotids. These best differentiating point is low retinal artery pressure in carotid occlusion. Venous stasis retinopathy is confused with occlusion of central retinal vein. Venous occlusion have more haemorrhages, the vein are dark and engorged. They do not become irregular in caliber as they do in venous stasis retinopathy, retinal artery is normal.

Kearn et al 1979, reported a case of right carotid occlusion with venous stasis retinopathy. A right superficial temporal middle cerebral artery and astomosis was done. In spite of successfully by Diamox, bypass had alleviated venous stasis retinopathy.

(2) Collateral Flow Retinopathy.

Neupert et al 1976 and Kearn et al 1979 described a peculiar changes throught to be due to colletral blood flow. High percentage of carotid occlusion shows retrograde filling of the anterior and middle cerebral artery by way of external carotid, orbital and opthalmic arteries. In collateral flow retinopathy fine new vessels emerges extending from the optic disc to vitreous cavity associated with dilated retinal veins irregular in calibre, retinal haemorrhages and microaneurysm (venous stasis retinopathy). Neupert et al's (1976) patient had occluated left internal carotid artery and 99% stenosis of right internal carotid artery. After endarterectomy the neovascularisation disappeared, Kearn et al 1979 described a case of left carotid occlusion with collateral and venous stasis retinopathy. A superficial – temporal – middle artery anatosis was done – Venous stasis retinopathy disappeared with lessening hypoxia but collateral retinopathy remained. Mayo clinic study (Kearn et al 1979) shows that superficial temporal artery-middle cerebral artery bypass does not return the retinal artery pressure to normal as does a successful internal carotid artery endarterectomy. They measured retinal artery pressure before and after this bypass only a slight improvement in retinal artery pressure was noted.

In Mayo clinic study venous stasis retinopathy was present in 20% cases of carotid occlusion and some decrease of retinopathy was observed by this bypass. Progressive venous stasis retinopathy with its subsequent neovascular glaucoma is an indication for such a bypass (Kearn et al 1979).

(3) Ischaemic Orbital Pain – Kearn and Hollenhorst (1963) described ischaemic pain in carotid artery occlusion and it is also associated with venous stasis retinopathy. It may be confused with the pain of glaucoma. In carotid occlusion pain is out of proportion to slight elevation of intra ocular pressure. Kearn et al (1979) described a case of carotid occlusion, venous stasis retinopathy for which by pass was done – The patient developed glaucoma and mature cataract one year later following successful bypass procedure. Mayo clinic study group believes orbital pain in carotid occlusion is an indication of bypass procedure, they also think that progressive venous retinopathy with its subsequent neovascular glaucoma is an indication for such a bypass. In few patients with internal carotid occlusion they found that new vessels have developed from retinal circulation or retinal vessels have become dilated and tortuous, which they termed collateral flow retinopathy. The operative risk, long term reduction in stroke and increased survival are as yet unclear. This operation remain unproved procedure but it is hoped that it will become a useful therapy in select small group of patients.

In view of 21% risk of stroke or death associated with endarterectomy, the incidence of angiographic complications, carotid endarterectomy appears most appropriate for patients with frequent, recurrent carotid system TIAS and who are willing to accept these modest risks to obtain symptomatic relief alone.

PROGNOSIS IN TIA

The risk of stroke in TIA is in the neighbourhood of 5 to 6% a year. The risk is greater in 3 months after first TIA. There is some evidence that in patients with bright plaques in the retina the prognosis is worse than others, 15% were dead within one year, 54% dead in 7 years, (Pfaffenbach and Hollenhorst 1973) in a follow up study of 208 cases. In the presented cases Case No. 1, died of coronary thrombosis after 7 years follow up but not in stroke and case no. 2 did not developed stroke either in two years follow up and is free from any TIA or neurological deficits.

TREATMENT OF TIA

Since cerebral ischaemic events have a variety of causes therefore no single therapeutic prog-

ramme can be advised to prove effective for all cases. The important and a wise plan is to eliminate risk factors. The increased risk of stroke in the hypertensive individual demands its vigilant therapy, the most important single factor to prevent stroke. This was applied to the presented cases and prevented stroke. Other important risk factors are: Cigarette smoking, coronary artery disease, hyperlipidaemia, diabetes but ones heredity cannot be changed. Two of the presented cases had coronary artery disease and hypertension and one died of coronary artery disease in spite of long term treatment for hypertension and ischaemic heart disease.

Specific therapy is indicated in variety of conditions which may threaten stroke. A cardiac pacemaker may be needed, cardiac emboli from prosthetic valves may require anticoagulant, nonsclerotic vasculopathies may need steroids, antihypertensives may need adjustment to correct orthostatic hypotention or may need to be instituted in lacunar brain infarction and finally anti platelets and antithombic drugs in cereberal arterial disease with artery to artery thrombo embolism or endarterectomy of an atheromatous affected artery.

In practice artery to artery embolism occur in 90% case. In patients under the age of 55, ninety per cent of the men have extracranial arterial disease but 70% of the women have coagulation disorders (Mettinger et al 1978) and such women patients are generally younger and on contraceptive pills. About a third of patients in the amaurosis fugax may go to stroke within 5 years. None of our patients had stroke within 5 years follow up period.

Angiography should be performed in all cases of amaurosis fugax over age of 50 with or without carotid bruit. As these patients manifests treatable risk factors: lipid disorder, hypertension, diabetes and smoking habits while other sources of emboli or haematological disorders should be sought first in younger patients and need angiography if there is evidence of hypertension, old cardiac infarct, carotid bruit and transient ischaemic attacks. Angiographically abnormal carotid artery is that with greater than 70% stenosis of its lumen, usually in the area of carotid bifirctation. If TIAS are associated with a carotid bruit on the appropriate side, the angiographic abnormality of the ipsilateral carotid increases to 85%. If stenosis greater than 70% exists in the carotid artery in a surgically accessable site without significant stenosis in the intracranial vessels such cases are candidate of carotid endaterectomy. The greatest problem in treatment is the choice of surgery anticoagulation or antiplatelets treatment.

No consensus can be found on the value of endarterectomy. Toole and his colleagues, (1978) in a 225 cases study of TIA, found no difference related to cerebral infarction, or transient ischaemic attacks among patients treated by medical or surgical or not treated, the results confirming those of american joint study (Fields et al 1978).

Mayo group believes that carotid endarterectomy can reduce symptoms of TIA (Sandok et al 1978) and may lower risk of stroke in selected patients. In the presence of cardiac disease (ischemic and hypertensive heart) multiple or incaccessible arterial lesion, or carotid accessible arterial lesion, or carotid occulusion on both side, fixed neurological deficit, advancing age, obesity and pulmonary disease, all these factors greatly increase the operative risk.

In U.K. — TIA Aspirin trial (1983) 32% under went angiography. Ten of 41 (24%) patients undergoing carotid surgery after randomisation had a peri-operative stroke and four of them died. Therefore some U.K. neurologist believe that if surgical risk of strokes or death is less than 5% patients with TIA should be exposed to the risk of angiography with a view to carotid surgery.

The most suitable patients are those with TIA (Amaurosis fugax) and unilateral carotid stenosis of greater than 50% but no residual neurological findings. Several of these patients were also given antiplatelet agent even postoperatively (Parkin et al 1982), and among older normotensive cases (Fields et al 1977, 1978, Reuthan and Domdorf 1978). A low dose aspirin to all patients and even after endarterectomy, it may be given permanently (Jaffe and Weksler 1979), the value of anticoagulant is not proven but if symptoms persist or increased on the above regime, one or two months course (initial heparin followed by oral anticoagulants) may be advised with strict follow up and vigilance to avoid intracerebral bleeding.

Common regimes employed are aspirin 600 mg. twice daily or 300 mg. daily dipyridamole 75 mg. three times a day and sulphinpyrazone 100-200 mg twice daily. Two cooperative studies were undertaken in Canada and United States. At 26 months, with follow up rate of 99%, aspirin reduced the risk of stroke or sudden death by

48% among men and no benefit was seen in women. Sulphinpyrazone was not effective. The conclusive evidence exists that at least one anti-platelet drug (Aspirin) prevent subseqjent stroke or death and reduces or abolishes TIAS with an acceptably low complication rate.

Hanley et al (1982) studied the effect of different regimes of 40 mg. aspirin on platelet thromboxane A_2 synthesis and vascular prostacyclin synethesis. Aspirin 40 mg. taken at interval of 48 hours consistently reduced platelet thromboxane A_2 synthesis to a level at which it failed to support platelet aggregation. Aspirin 40 mg. every 48 hours may have the maximum antithrombotic effect. In the present study 75 mg. aspirin daily was used as a safe dose for indefinitely.

MICRO SURGERY – A new surgical procedure, anastomasis of superficial temporal artery to the middle cerebral artery (Yasargil procedure), is being performed with increasing frequency – During the period (1971-1977), 121 patients have undergone this new procedure at Mayo clinic, progressive venous stasis collateral flow retinopathy and ischaemic orbital pain are main indication for this bypass surgery. 90 per cent or more stenosis in the affected carotid artery produces lowering of the retinal artery pressure. Measuring of retinal artery pressure (opthalmodynamometry) may be of great help in identifying occlusion of the internal carotid artery. Fibromuscular disease of the carotid artery significantly lowers the retinal artery pressure.

PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY FOR BASILAR ARTERY STENOSIS

Gruntzing in 1978 first reported on successful transluminal balloon catheter dilatation of a coronary stenosis resulting in improved myocardial perfusion and performance associated with relief in symptoms.

Sundt et al in 1980 described first time the successful use of transluminal balloon-catheter dilatation (angioplasty) for a high grade basilar atherosclerotic stenotic lesions. The report describes initial favourable experience in two patients with severe brain stem, cerebellar and posterior cerebral symptoms unresponsive to medical treatment and bypass graft of superficial temporal artery to anterior temporal branch of posterior cerebral artery.

ACKNOWLEDGEMENT

I wish to record my sincere thank to Rear Admiral R. M. Shaikh, Chairman and Managing Director Pakistan Security Printing Corporation for providing necessary facilities and financial aid to carry out this work.

I would also like to acknowledge the help of Neurosurgeon Prof. O. V. Jooma, Dr. Mehmood of Karachi X-Ray and Vascular Surgeon Dr. Aftab Ahmed Khan.

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