STROKES IN YOUNG PEOPLE: EXPERIENCE IN A BANTU HOSPITAL*

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In the course of a survey of neurological disorders among Bantu patients at Edendale Hospital[1] the most common variety of neurological disease was found to be that which presented as a stroke. For these purposes a stroke is regarded as a neurological condition of sudden onset, presumably due to vascular disease, and resulting in hemiplegia or other severe loss of function. Such diseases accounted for 33% of all neurological cases admitted to hospital. It was found that a significant proportion of such patients were less than middle-aged, and in some the stroke could not be explained. This article presents further observations on strokes in young people.

MATERIAL

The patients were Bantu between 8 and 40 years of age. Younger patients are admitted to the paediatric wards and fall outside the scope of this survey. The upper limit of 40 years is an arbitrary one. The material is divided into 3 groups. The first is a more detailed analysis of the consecutively admitted patients presented in the original survey. The second and third groups of patients are highly selected from subsequent admissions to illustrate unusual causes of stroke, or cases of unexplained stroke, respectively.

Analysis of Consecutively Admitted Cases

The original survey[1] included 1,302 cases of neurological disease admitted consecutively during a period of 45 months. Of these, 101 were patients of 40 years or less who had suffered strokes. In these cases the diagnosis usually depended on the presence of some associated cardiovascular disease. In a few cases no such disease was present and no cause of the stroke could be found. Table I lists the diagnoses in these cases. The list is unremarkable in that it illustrates the usual causes of stroke in young people, but it does emphasize the high incidence of hypertension, rheumatic heart disease, bacterial endocarditis and syphilis in this community.

Unusual Causes of Stroke

Following the conclusion of the above survey, attention has been devoted to cases of stroke in young people and their causes. During a period of 5 years some unusual causes have been discovered. The following have been selected for more detailed description.

TABLE I. CAUSES OF STROKE IN 101 CONSECUTIVELY ADMITTED PATIENTS

Diagnosis	*No. of cases*
Cerebral haemorrhage or arterial occlusion associated with hypertension	30
Cerebral embolism associated with heart disease:	
Rheumatic and/or bacterial endocarditis	23
Cardiomyopathy	3
Pulmonary arteriovenous malformation	1
Mediastinal granuloma with cardiac involvement	1
Subarachnoid haemorrhage	13
Syphilis	6
Blood disorders:	
Leukaemia	3
Secondary polycythaemia	1
Thrombocytopenic purpura	1
Cerebral angiomatous malformation	3
Following previous head injury	2
Subdural haematoma	2
Associated with tuberculous cervical adenitis	1
Sagittal sinus thrombosis	1
Cause undetermined: no predisposing factor discovered	10
Total	101

Post-traumatic strokes. This group includes 5 patients in whom a stroke developed an appreciable period after injury to the head or neck, and in whom no other cause could be found. The fact that the patients were referred and admitted to the medical unit rather than the surgical unit is evidence of the fact that, in the first instance, the trauma was considered to be too remote to be responsible for the neurological disease. In 3 cases internal carotid artery occlusion was shown on angiography. In one there was occlusion of the middle cerebral artery, and in the remaining case there was no major vessel occlusion though the circumstances suggested association with the previous trauma, and other causes were excluded. In 4 cases the trauma was sustained in the course of assaults

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TABLE II. DELAYED POST-TRAUMATIC STROKES

Sex	*Age*	*Nature of injury*	*Period*	*Main clinical features*	*Angiogram*	*Result*
M	28	Assault: depressed fracture L parietal bone	5 months	Aphasia, apraxia R hand, mild R hemiparesis	Left ICO	Permanent aphasia, epilepsy
M	32	Fall onto head: injury to vertex and R parietal region	3 weeks	L hemiparesis	Occlusion R middle cerebral artery	Improved
M	27	Assault: hit on face and neck	1 week	R hemiplegia, R sensory deficit	Left ICO	Improved, residual hemiparesis
M	25	Assault on 2 occasions: old depressed fracture L parietal region: hit on head and poked in R eye on day before admission	? 1 day	Stupor, L hemiplegia, R pupil fixed	Right ICO	Permanent hemiplegia, epilepsy
M	30	Assault: depressed fracture L frontoparietal region years before	? several years	Recent R hemiparesis, R sensory deficit	No major vessel occlusion on either side	Improved: residual hemiparesis

ICO = internal carotid artery occlusion.

or fights, a common source of injury in young men of this community. The principal features of these cases are summarized in Table II.

Internal carotid occlusion resulting from injury to the head, neck or pharynx is a well-documented though unusual cause of stroke. Humphrey and Newton[2] reviewed 107 cases of such occlusion in patients under 40 years and added 17 cases of their own. Of the precipitating factors, trauma was the most common and was responsible in 24 cases among the whole series of 124. In most cases the occlusion developed within 24 hours of the injury, though the delay may be considerable. In one of Humphrey and Newton's cases 4 years elapsed between the trauma and the development of neurological symptoms. In the 6 cases of post-traumatic internal carotid occlusion reported by Sedzimir[3] the latent period was between a few hours and 7 days.

In such cases it is likely that the trauma results in intimal damage of the internal carotid artery with subsequent thrombosis, either at the base of the skull or in its cervical course. This thrombus may detach and form an embolism, producing occlusion of a cerebral artery. In our case this seems the likely origin of the middle cerebral artery occlusion. Sedzimir[3] postulates that the internal carotid artery is subjected to undue shearing stress in the region of the cavernous sinus and that this leads to intimal damage with thrombosis and retrograde spread to the cervical portion of the vessel.

Strokes associated with cerebral metastases. Sudden neurological deficit may result from metastatic malignant tumours in the brain. The primary tumour may not have produced disability. The following patients illustrate this clearly:

A woman aged 35 years was admitted in coma. She had suffered from abdominal pain for one month. On the day before admission she had developed headache, and during the night she had become comatose. There was a right hemiplegia with exaggerated tendon reflexes and ankle clonus on that side, and conjugate deviation of the eyes to the left.

The liver was enlarged to within 1 cm. of the umbilicus. It was nodular and very firm. The cerebrospinal fluid was normal. X-rays of the chest and skull showed no abnormality.

The patient died on the third day after admission. Necropsy showed a primary carcinoma of the liver with extensive intrahepatic metastases. Section of the brain showed a secondary tumour, about 1·5 cm. in diameter, in the region of the internal capsule and caudate nucleus on the left side. There were oedema and fresh haemorrhage round the tumour in the substance of the brain. No other metastases were found.

Primary carcinoma of the liver is one of the most common malignant diseases among the Bantu. Distant metastases are unusual. When these occur they are usually in the lungs. Cerebral metastases are rare.

A 29-year-old woman was able to give little history on admission to hospital. She had apparently been ill for about a week with headache and the rapid development of a left hemiplegia. She was drowsy and had bilateral papilloedema. There was a partial third nerve palsy on the left side, and she had bilateral facial weakness.

Carotid angiography showed features of an expanding lesion in the right parietal region. At craniotomy the brain was under tension, and needling yielded blood and necrotic material, thought to be tumour tissue. Despite decompression the patient died the following day.

Necropsy showed a secondary tumour, the size of a golf-ball, in the substance of the right parietal lobe. There was haemorrhage into and round the tumour, as well as multiple small haemorrhages in the pons. A large secondary deposit was also present in the upper lobe of the left lung. What was thought to be a small primary tumour was found in the uterus. Histology showed that the secondary tumours in the brain and lung were metastases from a chorionepithelioma. The rapid deterioration in the patient's condition was presumably the result of haemorrhage into and round the cerebral tumour and into the pons.

Cerebral embolism associated with cardiomyopathy. Cardiomyopathy is one of the most common varieties of heart disease in this Bantu population.[4,5] This condition is often associated with the formation of a mural thrombus in one or both ventricles and with embolism, most commonly in the lungs, spleen and kidneys. Cases of cerebral embolism occurring in these circumstances have been described.[6] The following is a further example of this cause of a stroke:

A woman of 28 years was admitted to hospital 3 times in 7 months. On each occasion she was in congestive cardiac failure which was diagnosed as being due to cardiomyopathy. Her blood pressure was normal and there was no evidence of valvular disease or bacterial endocarditis.

Her second admission to hospital was necessitated by the sudden development of aphasia and right hemiparesis. X-ray of the chest showed diffuse cardiac enlargement with pulmonary congestion. An ECG showed low voltage and T-wave flattening or inversion which is the common pattern in cardiomyopathy. She was treated with anticoagulants and physiotherapy. The hemiparesis improved to the extent that she was able to walk, but when she was discharged after 5 weeks dysphasia was still present.

On her third admission, 2 months later, she complained of a sudden increase in breathlessness a few days previously. This was associated with precordial chest pain. She was again in congestive cardiac failure. There had been a very striking change in her ECG since the second admission. This showed the development of deep Q waves and elevated ST segments in the anterior chest leads (V2 - V5), an appearance of anterior myocardial infarction.

She was ultimately discharged from hospital, again on treatment, but she has not returned during the subsequent 4 years. It is presumed that she has died of her heart disease, the prognosis of which was obviously poor. There is little doubt that this patient suffered from cardiomyopathy, and that the stroke and the myocardial infarction were the results of embolism to the cerebral and coronary arteries respectively.

Occasionally, at postmortem examinations, it has been noticed that antemortem intracardiac mural thrombus is present in cases in which there had never been clinical indication of heart disease, and where the patient had died of some other cause such as pneumonia or nephritis. It is thus conceivable that a person with subclinical cardiomyopathy may suffer from embolism, cerebral or otherwise, when there is not yet clinical evidence of heart disease. A case illustrating this possibility has been reported.[6] Such events might account for a few of our cases of unexplained stroke in young people.

Systemic lupus erythematosus. Two published series of strokes in young adults[7,8] each include one case of systemic lupus erythematosus. The present series also contains one case, which is of interest because the stroke was the first manifestation of the systemic disease, which, over the ensuing years, displayed many classical features of the condition.

A 20-year-old girl was first admitted to hospital in July 1962, following the sudden onset of right hemiplegia. There was no appreciable sensory deficit. Physical examination yielded no clues as to the cause of the hemiplegia. The CSF and a left carotid angiogram were normal. A persistently elevated sedimentation rate, moderate albuminuria and a serum globulin level of 5·2 G/100 ml. aroused the suspicion of systemic disease. A search for LE cells, however, proved fruitless.

While she was in hospital she developed deep-vein thrombosis in the right leg, a rather unusual complication in a young Bantu girl. This was treated with anticoagulants. Her hemiplegia improved and she was discharged, walking, after 7 weeks in hospital.

Her second admission was in October 1962, when she presented with the features of nephrotic syndrome with oedema, heavy albuminuria, a serum cholesterol level of 413 mg./100 ml. and a serum albumin level of 0·9 G/100 ml. An intravenous pyelogram was normal. Repeated examinations for LE cells were again negative. She was nevertheless treated with betamethasone, with improvement in the oedema and albuminuria.

Despite advice to attend regularly for observation and outpatient treatment she defaulted, and her third admission in December 1962 was occasioned by the development of right lower lobe pneumonia. This failed to respond to penicillin, but improved with tetracycline and resumption of betamethasone. Again LE cells were not revealed by examination of the peripheral blood.

Thereafter she remained comparatively well, on intermittent corticosteroid treatment, for almost 2 years. In December 1964 she was admitted for the fourth time, again with respiratory symptoms. X-rays showed patchy consolidation in both lungs, and pleural reaction at the left base. One of several slightly enlarged cervical lymph nodes was biopsied, but this showed only features of chronic non-specific lymphadenitis. LE cells were not found. The bone marrow was normal.

Features of the nephrotic syndrome persisted. Serum protein electrophoresis showed the albumin fraction to be markedly reduced, and the gammaglobulin to be elevated. Renal biopsy at this stage showed features 'consistent with the renal changes of systemic lupus erythematosus'.

Treatment with corticosteroids was continued, though intermittently due to the patient's irregular attendance. She has since been admitted 3 times: in June 1965 with polyarthritis; in December 1965 with recurrence of pneumonitis; and in September 1966 with anaemia which had the features of microangiopathic haemolytic anaemia. As far as is known she remains in reasonable health.

This young woman, whose illness began with a stroke, subsequently developed features of systemic disease which have necessitated 7 admissions to hospital. Though LE cells were never found, the diagnosis was proved by renal biopsy.

Internal carotid occlusion associated with subarachnoid haemorrhage. The association of rupture of an intracranial aneurysm with internal carotid occlusion has been reported occasionally. The following case illustrates the mechanism by which this might occur:

A man of 30 years was admitted a few hours after sudden, but temporary, loss of consciousness. On recovering consciousness he was dull and disorientated, with neck stiffness, but without signs of focal neurological disease. There was a haemorrhage adjacent to the macula in the right fundus. His blood pressure was normal. Lumbar puncture yielded uniformly blood-stained CSF.

On the 6th day in hospital there was deterioration in his condition. He developed weakness on the right side of the body and appeared to have sensory dysphasia. This evolved to a total right hemiplegia with sensory loss on the right side. Carotid angiography showed occlusion of the left internal carotid artery, commencing about 2 cm. below its entry into the skull. The patient's condition deteriorated further and he died on the 10th day of his illness.

Necropsy confirmed the presence of an extensive subarachnoid haemorrhage. An aneurysm had ruptured at the origin of the anterior and middle cerebral arteries. Retrograde dissection of the internal carotid artery had occurred from this site. The occlusion of the artery was due to the dissection continuing downwards into the carotid canal, the extravasated blood causing compression of the lumen of the internal carotid artery.

One of the cases of internal carotid occlusion in young adults reported by Humphrey and Newton[2] occurred in association with spontaneous subarachnoid haemorrhage, but the precise mechanism was not revealed. Sedzimir[3] mentions 2 personal cases of occlusion associated with ruptured intracranial aneurysm. Brice and Crompton[9] have reported the occurrence of spontaneous dissecting aneurysms of the cervical internal carotid artery in young people. In each of 3 cases reported the patients developed hemiplegia and had unequivocal angiographic evidence of internal carotid occlusion or stenosis, and the dissection was shown at necropsy. Atheroma was not present at the sites of dissection, which commenced in areas of cystic degeneration in the media.

In the above case it appears that the mechanism of occlusion was similar, except that the dissection began at the site of a ruptured aneurysm and spread in retrograde fashion down the internal carotid artery.

Internal carotid occlusion associated with syphilis. Cerebral vascular occlusion accounted for 29% of cases of neurosyphilis in the original series.[3] This diagnosis was usually based on the association of clinical features of cerebral vascular occlusion with a significantly positive blood or CSF Wassermann reaction in the absence of other causes. Angiography was not done in all cases when this association was present, but this investigation has sometimes shown occlusion of anterior or middle cerebral arteries in such cases. In 3 patients the clinical picture of posterior inferior cerebellar artery occlusion was attributable to syphilis on the same grounds. The following case is one in which internal carotid occlusion was attributed to syphilis:

A 40-year-old man was admitted with a history of headache for one month, followed by giddiness and sudden collapse on the day of admission. There was left hemiparesis with maximal paralysis of the arm, and he had no clear sensory deficit. The heart and blood pressure were normal. No bruits were heard in the head or neck.

The CSF was normal apart from a protein content of 47 mg./100 ml. and a slight increase in globulin. The blood Wassermann reaction was strongly positive. In the CSF the VDRL test was positive, but the Wassermann reaction was anticomplementary. On angiography the patient was shown to have occlusion of the pericallosal artery, and the posterior parietal and angular branches of the middle cerebral artery on the right side. The left internal carotid artery was totally occluded 1 cm. beyond its origin, and the left anterior cerebral artery was observed to fill from the right side.

The patient was given a course of penicillin and physiotherapy. He was discharged from hospital, able to walk but with residual weakness of the left arm, after 5 weeks in hospital.

In the absence of any other cause of the hemiplegia there is little doubt that this was the result of vascular syphilis. Of interest also is the observation that the hemiplegia occurred on the same side as the totally occluded internal carotid artery. The paralysis was presumably the result of the observed occlusion of smaller intracranial branches on the opposite side, combined with a generally reduced cerebral blood flow.

Suppurative encephalitis. Cerebral infections are not usually associated with sudden onset, but the following cases illustrate the possibility of this occurring:

A man of 30 years was admitted on the 8th day of his illness which had begun, suddenly and unheralded, with an epileptic fit. Thereafter he had right hemiplegia and sensory loss on the right side. At the time of his admission he was fully conscious and the fundi were normal.

The CSF contained 22 polymorphs and 10 lymphocytes per cu.mm. with a protein content of 150 mg./100 ml. and sugar of 80 mg./100 ml. No organisms were identified. Brain abscess was suspected and penicillin treatment started on the day of admission. Carotid angiography showed shift of the left anterior cerebral artery to the right. Craniotomy was performed but the only abnormality found was that the brain was under tension. Needle exploration of the brain yielded no pus.

Treatment with penicillin, and subsequently tetracycline, was continued, but the patient's condition deteriorated and he died on the 15th day of his illness. Necropsy showed the major part of the left hemisphere to be softened and inflamed. No loculated abscess was found. The lungs showed purulent bronchitis and mild bronchiectasis. Histology of the brain showed intense polymorphonuclear leucocyte infiltration and features of acute suppurative encephalitis.

This patient's illness began suddenly with an epileptic fit and the development of hemiplegia. This, presumably, was the result of a septic cerebral embolism from an area of pulmonary suppuration. His final rapid decline was probably the result of spread of inflammation throughout most of one hemisphere. The abscess had not loculated and was not amenable to drainage, even after 15 days' illness. It is possible that antibiotic treatment retarded the suppurative process.

A somewhat similar case was that of another 30-year-old man who was admitted to hospital about 8 hours after the sudden onset of his illness. This had begun with headache and vomiting which were followed shortly by epileptic fits. On admission he was stuporous and there was hemiplegia on the left side with loss of appreciation of pinprick on the same side. The ocular fundi were normal. Blood pressure, heart and lungs were normal. He was pyrexial (104°F).

The peripheral blood showed neutrophil leucocytosis. His blood sugar level was 122 mg./100 ml. The CSF contained 10 polymorphs and 12 lymphocytes per cu. mm. The protein was 43 mg./100 ml. and the CSF sugar 95 mg./100 ml. It was thought likely that he had a brain abscess, but he died shortly afterwards.

Necropsy showed a round, vascular, friable mass, about 3 cm. in diameter, in the region of the thalamus and internal capsule on the right side. Macroscopically this resembled a vascular tumour. Histology, however, showed no sign of tumour but showed intense congestion, small haemorrhages and aggregations of polymorphonuclear leucocytes. The heart and lungs were normal. This man's illness was apparently due to acute suppurative encephalitis, in this case focal, though the source of the infection was not discovered.

Cerebral embolism associated with infection in the neck. A 12-year-old girl was referred from a country hospital with a history of sudden development of fits and left hemiplegia. The hemiplegia was total on admission, but sensory loss was slight on the left side. She was pyrexial and there was pain on movement of the neck in all directions. This appeared to be due to enlarged, tender lymph nodes on both sides of the neck. There was mild pharyngitis but no obvious source of the lymphadenitis. The heart was normal. A blood count showed polymorphonuclear leucocytosis. The CSF and X-rays of the chest and skull were normal. Other investigations did not reveal a further cause of the pyrexia.

On treatment with penicillin the pyrexia subsided and the lymph-node enlargement disappeared within 10 days. The hemiplegia improved rapidly with physiotherapy and when she was discharged after 4 weeks she had recovered completely. No further fits occurred.

This case corresponds closely with the description of 'acute hemiplegia in childhood' by Bickerstaff.[10] He presented evidence in support of his belief that most of these cases are the result of infections in the neck with carotid arteritis, intimal damage, thrombosis and cerebral embolism from this source. This appears to be a reasonable explanation for such cases, whose alternative pathogenesis he discussed in detail. That this syndrome is not confined to children is shown by other cases described by Bickerstaff[10] and Garland and Pearce.[11] A similar mechanism is responsible for the neurological lesions which follow injuries in the region of the soft palate and pharynx. The original series of consecutively admitted cases includes one case of hemiplegia in association with tuberculous cervical adenitis, possibly with a similar relationship.

Unexplained Strokes

There were 28 Bantu patients aged between 10 and 40 years, in whom investigations did not reveal the precise cause of the stroke. These patients were admitted during a 5-year period subsequent to the conclusion of the original consecutive series (Table I). This group includes the majority of young people with unexplained strokes admitted during this period, though it is possible that a few patients in this category have been omitted. The investigations included physical examination, X-rays of chest and skull, blood count, lumbar puncture and CSF examination, blood and CSF Wassermann reactions, ECG, and carotid angiography in 22 patients. Of the 6 patients in whom angiography was not done (Table III), 4 refused the investigation.

TABLE III. UNEXPLAINED STROKES: ANGIOGRAM OMITTED

Sex	*Age*	*Clinical features*	*Other findings*	*Angiogram (reason for omission)*	*Result*
M	26	Sudden onset L hemiplegia; disorientation; slight sensory loss on left	Axillary tuberculous adenitis, CSF normal	Refused	Improved after 4 weeks
M	33	Sudden onset L hemiparesis; sensory deficit on left	CSF protein 74 mg./100 ml.	Refused	Improved in 2 weeks
F	24	Sudden onset L hemiparesis; sensory deficit on left	14 weeks pregnant, CSF normal	Pregnancy, rapid improvement	Almost normal in 9 days
F	17	L hemiparesis; retention of urine	Followed attack of salpingitis	Refused	Improved after 8 weeks
M	15	Abnormal behaviour; R hemiparesis and anaesthesia; R homonymous hemianopia	Nil	Refused	No improvement in 3 weeks
M	26	Sudden onset of loss of consciousness, paralysis of conjugate deviation to R; R L.M.N. 7th nerve palsy	Nil	Carotid angiogram unlikely to reveal cause of pontine lesion	Improved after 3 weeks; residual facial weakness

TABLE IV. UNEXPLAINED STROKES WITH ANGIOGRAPHIC ABNORMALITY

Sex	*Age*	*Clinical features*	*Other findings*	*Carotid angiogram*	*Result*
M	31	Onset over 3 weeks of L hemiparesis, mainly face and arm	Nil	Attenuation of right ICA and right MCA. ACA not filled	Slight improvement in 4 weeks
F	10	Sudden loss of consciousness; R hemiparesis; aphasia	Nil	Occlusion of left MCA and ACA at origin	Slight improvement in 4 weeks
M	38	Sudden onset R hemiplegia	Nil	Occlusion of left MCA	Improved after 4 weeks
M	26	Total aphasia; L hemiplegia	Old scalp scars, CSF protein 88 mg./100 ml.	Bilateral ICO at level of anterior clinoid processes. Collateral filling through ophthalmic arteries	Unimproved after 6 weeks
M	30	Mental disturbance followed by development of L hemiplegia	Nil	Right ICO 2 cm. beyond origin	Taken home unimproved after 2 weeks
M	36	Onset of L hemiplegia over 3 days	Nil	Occlusion right MCA at origin	Improved after 5 weeks
M	36	Sudden onset of convulsions followed by aphasia, R hemiplegia and sensory deficit	Nil	Left ICO just above origin of posterior communicating artery	Little improvement after 3 months
M	32	Sudden onset of unconsciousness; L hemiplegia and anaesthesia	Nil	Right ICO just above origin	Endarterectomy; little improvement after 10 weeks
M	21	Sudden onset of giddiness and L hemiplegia	12% eosinophilia	Occlusion right MCA 0·5 cm. from origin	Improved after 4 weeks

ICA = internal carotid artery; MCA = middle cerebral artery; ACA = anterior cerebral artery.

TABLE V. UNEXPLAINED STROKES WITH NORMAL CAROTID ANGIOGRAMS

Sex	*Age*	*Clinical features*	*Other findings*	*Carotid angiogram*	*Result*
M	22	L-sided fits followed by L hemiparesis affecting face and arm maximally. No sensory loss	20% eosinophilia (of 7,000)	R normal	Improved after 3 weeks
M	40	Sudden onset L hemiparesis. No sensory loss	CSF protein 52 mg./100 ml.	Bilateral normal	Improved after 2 weeks
M	8	Sudden loss of consciousness; L hemiplegia; L 3rd nerve paralysis; slight anaesthesia on left	Nil	Bilateral normal, ventriculogram normal	Slightly improved after 12 weeks
M	35	Sudden onset L hemiparesis; sensory deficit L side	ECG: T-wave inversion in lateral chest leads	Bilateral normal	Improved after 7 weeks
M	40	R hemiparesis, mainly face and hand; R anaesthesia	Nil	Bilateral normal	Improved after 3 weeks
F	33	R hemiparesis; R anaesthesia	Nil	Bilateral normal	Unimproved after 3 weeks
M	35	Onset over 3 days; R hemiparesis affecting leg maximally; R anaesthesia	Nil	Bilateral normal	Improved after 3 weeks
M	33	Convulsions followed by L hemiplegia and anaesthesia	Nil	Bilateral normal	Improved after 3 weeks
M	34	Sudden loss of consciousness followed by dysphasia and R hemiplegia. No sensory loss	Nil	L normal	Improved after 4 weeks
M	40	Sudden onset R hemiplegia and motor aphasia. Sensation normal	CSF protein 47 mg./100 ml.	Bilateral normal	Slightly improved after 4 weeks
M	30	Sudden onset of fits with R hemiparesis and dysphasia	Nil	Bilateral normal	Improved after 2 weeks
F	32	Sudden onset R hemiplegia and aphasia. Sensation normal	Nil	Bilateral normal	Slightly improved after 4 weeks
F	28	Sudden onset stupor and R hemiplegia; R sensory deficit	Nil	Bilateral normal	Recovered in 4 weeks

In 9 patients (Table IV) angiography showed occlusion of a major intracranial or extracranial artery. Though the anatomical defect was demonstrated, the pathological cause of the occlusion was not discovered. Investigations failed to reveal any of the usual predisposing factors.

In the remaining 13 patients (Table V) carotid angiography showed no occlusion of major vessels. It must be assumed, on clinical grounds, that these patients suffered from occlusion of some smaller vessels such as the perforating branches of the middle cerebral artery, or the superficial branches of the middle or anterior cerebral arteries. In these cases, also, none of the usual predisposing causes of vascular disease was present. Their pathogenesis remains obscure.

None of this group of 28 patients is known to have died, so necropsy material has not been available.

DISCUSSION

The first group of cases (Table I) illustrates causes of stroke in young people. The most common—namely, hypertension, embolism associated with heart disease, subarachnoid haemorrhage and syphilis—figure among the usual causes of stroke in young people in any race or community. The high incidence in association with hypertension and rheumatic heart disease reflects the frequency of these conditions in this Bantu population.

The second group of cases, which have been selected to illustrate unusual causes of stroke, includes causes which are usually sought when common aetiological factors have been excluded. Injuries to the head and neck, as a cause of delayed vascular occlusion and stroke, deserve emphasis. A further interesting, though rare, variety of stroke is that which is presumably due to embolism in cases of cardiomyopathy.

Of particular importance is the group of 28 young patients in whom no fundamental cause of a stroke could be discovered. Whether or not angiographic evidence of vascular occlusion was found, it is likely, on clinical grounds, that these strokes were all vascular in origin. Our figures indicate that about 10% of strokes in young people are not explained by fairly complete investigation. Most cases in this group are presumably the result of arterial thrombosis and cerebral infarction. This finding is of particular interest in comparison with the low incidence of coronary thrombosis and myocardial infarction in this community. During a survey of 1,000 consecutive Bantu cardiac admissions over a period of almost 2 years[4] there were 2 patients under 40 years old with myocardial infarction. One of these infarctions may have been embolic in origin. This observation of the rarity of coronary artery disease in the Bantu concurs with the experience of many others.[5,12-15] Among Indians, on the other hand, the opposite pattern of vascular disease is found. Coronary thrombosis is common[4] whereas, in our experience, unexplained strokes in young people are rare.

The reason for this discrepancy, more obvious with increasing age, between the relative incidence of coronary and cerebral vascular disease in the Bantu is unknown. It strongly suggests that these conditions have different causes. While atherosclerosis might be responsible for strokes in young hypertensive patients and our older patients, there is not good reason to blame atheroma for these unexplained strokes in young people. Our experience of necropsy material is that, while atherosclerosis occurs in the Bantu, its severity and extent in all vessels is considerably less than occurs in Whites and Indians of comparable age. The findings of Wainwright[16] and Sacks[17] regarding aortic and coronary atheroma support this impression.

No detailed study of the relative incidence of atherosclerosis in cerebral vessels among different races has been published. We have no reason to believe that the racial variations are much different from those found in the case of aortic and coronary atherosclerosis. Wainwright[18] has found that 'plaque formation becomes significant in the cerebral vessels in the 4th decade in hypertensive African males, and in the 5th decade in hypertensive African females. The incidence remains low in non-hypertensives of both sexes.'

It is thus likely that hypertension contributes to the high incidence of cerebral vascular disease in middle-aged and elderly Bantu. Hypertension was definitely excluded in all our cases of unexplained stroke in young people. It is unlikely that these strokes can be attributed to atherosclerosis.

Even in Whites the tendency to attribute unexplained strokes in young people to atheroma might be questioned. Adams and Graham[8] have reported 12 cases of fatal cerebral infarction in young or middle-aged people in whom no significant atheroma was found at necropsy. In 4 cases definite predisposing factors were present, but in the remainder the exact pathogenesis was unexplained. In the series of 107 cases of internal carotid occlusion in young adults reviewed by Humphrey and Newton[2] no predisposing factors were found in 60. In these, as in our cases, it is likely that factors other than atherosclerosis are culpable.

One of our cases occurred during pregnancy; but there were no cases of 'puerperal hemiplegia', which condition has been the subject of some speculation. Puerperal women are more susceptible to unexplained strokes than others in the same age-group. These cases were previously regarded as having cerebral venous thrombosis, but this is a less probable explanation than arterial thrombosis. Adams and Graham[8] consider that venous thrombosis is not the usual cause of non-haemorrhagic hemiplegia occurring in the puerperium. The role of venous thrombosis appears to have been over-emphasized in both puerperal and infantile hemiplegia.

Our cases are also of interest in the light of suspicion being cast on oral contraceptive drugs as a cause of cerebral vascular lesions. None of our patients had used these drugs. Strokes in young people who have not taken these hormones are thus not very rare, even among the Bantu who are not usually prone to vascular thrombosis.

SUMMARY

This paper records experience of strokes among young Bantu patients, between 8 and 40 years old. The causes of stroke among 101 consecutive cases are analysed: common predisposing conditions are hypertension and rheumatic heart disease. Some cases of stroke with unusual causes are described in detail: among these are several cases of delayed arterial occlusion following injuries to the head or neck. A group of 28 young patients in whom no predisposing cause of stroke could be found on investigation is also described. In 9 of these, angiography revealed occlusion of major intracranial or extracranial arteries. In 13 angiography was normal. The pathogenesis in both groups is obscure.

Cerebral infarction consequent on occlusive arterial disease is relatively common among the Bantu, among whom coronary occlusion is rare. This discrepancy suggests that the causes of cerebral vascular disease in this racial group are different from those responsible for coronary thrombosis.

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