＂A STUDY OF CLINICAi AND LAEORATORY FEATURES
IN STROKE PATIENTS AT THE KENYATTA NATIONAL HOSPITAL＂．

## By

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DECLARATION

This dissertation is my own original work and has not been presented for a degree in any other University.

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## LIST OF ABBREVIATIONS

| KNH | Kenyatta National Hospital |  |
| :--- | :--- | :--- |
| ESR | - | Erythrocyte Sedimentation Rate |


| Great. | - | Creatinine |
| :---: | :---: | :---: |
| A.P. | - | Alkaline phosphatase |
| HDL-chol | - | High Density Lipoprotein cholesterol |
| LDI-chol | - | Low Density Lipoprotein cholesterol |
| Q1T | - | Gastro-intestinal tract |
| SOL | - | Space occupying leston |
| e.g. | - | for example |
| etc. | - | and so on |
| T.P. H, A. | - | Treponema pallidum |
|  |  | Haemagglutination test. |

## SUMMARY

This is both a retrospective as well as a prospective study conducted from January 1986 to January, 1987 at KNH involving 72 stroke patients. Thirty point four per cent of the patients were found to be hypertensive. The majority were found to be from the Central Province. More patients were rural dwellers than urban dwellers. The majority of the hypertensive patients were either first diagnosed at admission for stroke or had had ${ }^{\text {- }}$ poor control of their hypertension. Most hypertensive patients were old ( $>45$ years). Laboratory investigations showed hypertriglyceridemia, hyperuricemia, renal insufficiency, and high ESR to be common amongst stroke patients. Syphilis was not found in any of the patients. The young normotensive patients were examined and some possible aetiological factor identified in all except four.

Recommendations are made as to further work and prevention against stroke.

## INTRODUCTION

## HISTORICAL ASPECTS

The clinical syndrome of stroke has been known to mankined since time immemorial. In fact, it seems to be as old as man himself. The earliest evidence of brain disease is to be found in fossils of the New Stone Age (about 5,000 B.C.). In this period, there is evidence of skulls with marks of trephination (1). It was performed on men, women and children. They used flint-stone knives for this operation. The reasons for the trephination can only be presumed to have been strokes, epilepsy, etc., but these were attributed to demon possession and the trephination must have been to allow the demons to escare. The Bible also indicates man suffered strokes even then (1 Sam. 25: 37).

The Indiuns seem to have known about stroke as evidenced by tablets found in the Library of Ashurbanipal (2). The Egyptians also seem to have been well aware of the condition as shown by Papyrus Ebers which shows it might have caused the death of Weshptah (2). The Hippocratic school recognized stroke(apo plexy) and ascribed advancing age as the aetiological factor (1,2). Aretaeus of Cappadocia (2nd-3rd Century, A.D.) recognized hemiplegic paresis to be due to contralateral lesions of the brain (2).
early as 400 B.C. and ascribed it to cerebral hacmorrhage (2). One of the first scientific observations on strokes was done by Johann Jacob Wepfer (1620-1695) who, in 1658, published a work showing that apoplexy is due to haemorrhage from the cerebral vessels and worked up the anatomy of cerebral vessels (1,3). The modern description of stroke first appeared in Gower's book "Manua? of Diseases of the Nervous System" (1886-8) (1). He initiated a new era in the investigation of strokes. However, the big advances in the understanding of cerebrovascular accidents have come after the second world war. New techniques fn haematology have led to new knowledge of clot formation and the role of triglyceride. Fisher (4) has led the field in relating the clinical features of strok' to the anatomical leston. We have been alerted to the importance of the carotid vessels and the heart as sources of emboli. Computerised tomography has enabled us to further relate the clinical picture and the actual site of the lesion. Massive epidemiological sturies have emphasized the importance of hypertension, diabetes mellitus, obesity, and cigarette smoking as contributing factors in the production of strokes.

Prophylactic measures have proved to be effective and life expectancy has increased. Field (5) in his monograph "Collateral circulation of the brain", gives the importance that the circle
of Willis plays in the body's attempt to circumvent the effects of occlusion of the various arteries supplying the brain. The more effective the collateral circulation, the more complete the recovery that: is noted.

## CLINICAL PICTURE

Siroke is a term given to the clinical manifestation of ischaemia to the cerebrum following occlu= -sior or rupture cf any of the cerebral vessels (6,7). It may take any of the following forms:
a) Atherosclerotic thrombosis
b) Lacunar stroke,
c) Embolism
d) Hypertensive haemorrhage
e) Ruptured anerrysms and vascular malformations,
f) Transient ischaemic attacks,
g) Others.
i) Large vessel stenosis due to exirinsic causes.
ii) Radiation necrosis
iii) Post-operative encephalopathy
iv) Hypertensive encephalopathy

Transient ischaemic attacks are short-lived and are soon followed by full recovery so that many patients do not tend to come to hospital. It is therefore a diagnosis made retrospectiveiy from interrogation of the patients. Hence, it has not been included among the conditions investigated here.

The incidence of stroke in Kenya has nci been worked out yet. Bahemuka (8) in 1985 carried out a retrospective study of 207 stroke patients at KNH , Nairobi including the period 1975 through 1979, and found no history of transient ischaemic attacks, $48 \%$ had arterial hypertension, $11 \%$ had a prior history of stroke. There was no history of myocardial infarction, angina pectoris, or peripheral vascular insufficiency. On angiography, most lesions were found to be intracranial, usually in the middle cerebral artery or its branches. This suggested to him that atherosclerosis was not a major aetiological factor. In an analysis of all admissions to adult medical wards at Mulago hospital, Kampala, Billinghurst (9) in 1970 found npurological diseases to acr;ount for $9.6 \%$ of the 6144 crmissions with a male to female ratio of 2.7:1. Of trese, $34.0 \%$ were due to vascular disorders. Sharper \& Sharper (10) in a similar analysis earlier in Mulago found in 1957, that CNS diseases constituted $10.5 \%$ of all medical admissions; of these cerebrovascular diseases constituted 10.1\%. Dada et al (11) reported on the Nigerian neurological profile from Lagos University Teaching Hospital for the period 1962 to 1967 and in their literature review cited some of the following rates in various parts of Africa for neurological admissions:

Hutton (11) - 13\% Ior Kampala, Kaushik (11) $16.3 \%$ for Ibadan; Haddock (11) - 5.5\% for Tanzania;

Dads et al noted in the same paper that cerebio- vascular accidents form the second commonest of $a \geq 1$ neurological admissions with an incidence of $16.8 \%$. Osuntokun et al (12) made a similar survey and found non-embolic ischaemic disease to constitute $57.5 \%$, cerebral haemorrhage $26.4 \%$ and embolic ischaemic disease to constitute 6.I\% of all cerebrovascular diseases at the University Teaching Hospital in Ibadan, Nigeria.

The risk and aetiological factors associated with strokes are various and incIude hypertension which takes the top spot in most series, diabetes mellitus (7,9,12-15), rheumatic heart disease ( 8,13 ), pregnancy $(8,12,13)$, thyrotoxicosis (8), infections ( $8,9,13$ ), alcohol ingestion (8, 15-18), cavernous sinus tínombosis (9), sickle cell anaemia (9,12), chroni: myeiold leukemia (12), anaemia (12), obesity (12), syphilis (12), congestive heart failure (12), nephrotic syndrome (12), ryperlipidemia $(14,15,19)$, subacute bacterial endocarditis . (20) non-bacterial thrombっtic endocarditis (21), moyamoya disease (22), migraine (23,14), acute leukemia (13), metastatic cancer of the brain from stomach (13), trauma (14), oral contraception (14), mitral valve prclapse (14), atrial flutter/ fibrillation (l.4), abnormal platelets (14), cardiomyopathy (14), hypotension (14), ulcerative colitis (14) and smoking ( $15,17,20,24$ ). In all the series, -
there is always a substantial proportion of stroke patients in whom all the available and exhaustive investigations fail to reveal the underlying cause.

The clinical presentation is variable and depend on the underlying pathology and the site affected. Most patients present with hemiparesis or hemiplegia which may be purely motor, purely sensory, a mixture of the two, dysarthria, ataxia and/or signs of brainstem involvement. The features may be static, amy worsen with time with coma superveaing and in many instances death follows, or mav gradually regress and complete or near complete recovery may be expected in a minority of patients.

## AIMS AND ObJECTIVES OF THE STUDY

The previous two studies of strokes in Kenya reierred to $(8,13)$ were the only ones that could be traced in the literature. The first one dealt with a retrospective survey of hospital records and was, of necessity, incomplete in many aspects, especially as far as the clinical presentation and laboratory work up are concerned. The second one only looked at the normotensive young patients who form only a small proportion of patients with stroke seen at KNH. It has been reported in the literature that hypertension is the commonest cause of strokes. Whereas this is true in the western world, there are only few studies in Africa and only two studies in Kenya which gave two different figures of $48 \%$ and $20 \%(8,13)$ respectively. Hence, there was a need to carry out a prospective study of all stroke patients to see whether the situation at KNH is similar or different from the other places where similar studies have been carried out. The other felt need for the study was the clinical impression that there is a large number of young patients aged 45 years or less who are admitted to our medical wards in whom there is no obvious reason for them to have a stroke. Hence the objectives of this study were as follows:

1. To determine the frequency of stroke in terms of the total admissions and the total neurological cases admitted over the study period.
2. To determine the associated factors for stroke in our patients particularly in terms of frequency of hypertension and factors peculiar to the young normotensive patients.
3. To identify, where possible, any important clinical or biochemical peculiarities of stroke patients so as to suggest preventive strategies.

## MATERIALS AND METHODS

This was both a prospective as well as a retrospective study over the period January, 1986 to January, 1987. The retrospective part of the study consisted of going over the files of those who were known to have had hypertension to determine the level of blood pressure control. The subjects studied were those patients admitted to the medical wards at KNH over the period with a clinical diagnosis of "stroke" or "cerebrovascular accident". They were seen by the author personally and examined in the first instance to establish:
a) that they were indeed suffering from stroke,
b) whether they were hypertensive and
c) other accompanying physical findings. Stroke was definec as a sudden development of a focal neurological deficit of at least 24 hours' duration with or without loss of consciousness. The working definition excluded transient ischaemic attacks and subarachnoid haemorrhage. A patient was considered hypertensive if:
a) there was a history of hypertension prior to the incidence;
b) he/she had been treated for hypertension (13); defined as a diastolic pressure of 95 mmHg or greater in adults or 60 mmHg or greater in children (13);
c) repeated blood pressure readings were at least $165 / 95 \mathrm{mmHg}$ on at least two occasions taken for
at least three days after the stroke had been established (25);
d) there was evidence of ,therwise unexplained heart failure and raised blood pressure on admission (8);
e) there was severe retinopathy (grade III or IV) and raised blood pressure on admission (8).

The patients had a complete history including age, race, ethnicity, residence, occupation, duration of hypertension, whether on regular treatment, adequacy of blood pressure control (for those who were known hypertensive and whose files could be traced), whether actually taking the medication, history of other significant diseases, for example, diabetes mellitus, acute or chronic renal failure, phaechromocytoma, rushing's syndrome, etc. The presence or absence of neck bruits, fundoscopic changes, cardiac lesiors was also looked for. The laboratory investigations performed included a haemogram, blood sugar (2 hours post-prandial), VDRL, CXR, ECG, blood urea, creatinine, electrolyte levels, liver function tests, and serum lipids. It had earlier been intended to perform the catecholamine screening tests and urinary keto and hydroxysteroid levels but these had to be abandoned due to technician and organisational difficulties. A few patients had carotid/vertebral angiography done, and one had a computerised tomographic scan done. The serum lipid parameters looked at were:
a) cholesterol - using Boehringer/Mannheim (BM) Monotest Cholesterol chod-Pap method (cat. No. 29031);
b) high density lipoprotein cholesterol - using BM HDL. Cholesterol precipitant reagent (catalogue number 543004);
c) Low density lipoprotein cholesterol - using BM LDL - cholest:erol reagent catalogue number 726290;
d) triglycerides - using BM reagents catalngue number 701882.

The results were then subjected to statistical analysis to determine means and standard deviations. As this was not a comparative study, no attempts were made at a comparable analysis of the results.

The blood pressure control was classified
aroitrary as follows:-
Good control - average diastolic $\leqslant 95 \mathrm{mmHg}$
Fair control - average diastolic $>95 \leqslant 110 \mathrm{mmHg}$
Poor control - average diastolic $>110 \mathrm{mmHg}$.

## RESULTS

Seventy two consecutive patients were recruited into the study. There were 22 (30.6\%) hypertensives and 50 ( $69.4 \%$ ) normotensives. Twenty five (34.7\%) were 45 years of age and below, the age range being from 15 years to 88 years, mean 52 years. Twenty one ( $29.2 \%$ ) patients were from an urban setting while 47 ( $65.3 \%$ ) were from the rural areas. In 4 patients the residence was not known. 'The various provinces are represented on Table 1.

In terms of occupation, 33 ( $45.8 \%$ ) were farmers, $13(18.1 \%)$ were housewives, $5(6.9 \%)$ were students, $3(4.2 \%)$ were drivers, $4(5.6 \%)$ were businessmen, $2(3.8 \%)$ were watchmen or guards. There was 1 ( $1.4 \%$ ) each of a general worker, secretary, machine operator, waiter, fitter and housemaid. Four (5.6\%) were unemplnyed and the occupation of 4 others was unknown.

On questioning the following diseases, conditions and habits were elicited: hypertension in 11 (15.3\%), previous stroke in $7(9.7 \%)$, cigarette smoking in $12(16.7 \%)$, alcohol drinking in 14 ( $19.4 \%$ ), diabetes mellitus in 5 ( $6.9 \%$ ), probably trauma in 4 ( $5.6 \%$ ), congestive cardiac failure in 3 ( $4.2 \%$ ), duodenal ulcers in $2(2.8 \%)$, bronchial asthma in $3(4.2 \%)$, "heart disease" in 3 (4.2\%), pneumonia in 2 (2.8\%). One case each of the following was also found: sickle cell disease, chest pain, cold ischaenia of the foo' with gangrene, use of the oral contra-
ceptive pill, delivery one week before; chronic headaches probably migraine, dementia and mental depression, ulcerative colitis, cough with haemoptysis, gouty arthritis, deep vein thrombosis of leg, connective tissue disease (probably systemic lupus erythematosus), upper gastrointestinal bleeding, focal fits, pulmonary oedema, and malaria.

The patients with hypertension were further analysed. Fourteen had hypertension for less thár one year, 5 had a history of 1 to 2 years, 1 had had it for 3 years while 4 had it for more than 5 years. For those who had had hypertension for one year or more, 1 ( $10 \%$ ) had had good control, 7 (70\%) had fair control, while 2 ( $20 \%$ ) had had poor control of their hypertension.

Other findings on physical examination releaved the following: signs of atherosclerosis in 10 ( $45.5 \%$ ), grade I and II retinopathy in 19 ( $86.4 \%$ ) grades III and IV retinopathy in 11 (50.0\%), cardiomegaly in 13 (59.1\%), obesity in 4 (18.2\%), valvular heart disease in 1 and cataracts in 1 . Figure 1 shows the pattern of $\mathrm{Hb}, \mathrm{WBC}$ and ESR of the hypertensive patients.

The blood sugar in hypertensives ranged from 3.8 to $20.5 \mathrm{mmol} / \mathrm{l}$ with a mean of $7.6 \pm 4.2 \mathrm{mmol} / \mathrm{l}$, six ( $27.3 \%$ ) patients had blood sugar above 8 mmol/1: The serum sodium ranged from $122 \mathrm{mmol} / 1$ to $153 \mathrm{mmol} /$ I with a mean of $136 \pm 8 \mathrm{mmol} / \mathrm{I}$. There were 5 subjects $(23.8 \%$ ) with levels below 130 mmol/l and 1 with a level above $150 \mathrm{mmol} / 1$. The serum potassium levels ranged from 2.8 to $5.3 \mathrm{mmol} / 1$ with a mean of $4.0 \pm 0.7 \mathrm{mmol} / 1$. There were $7(31.8 \%)$ subjects with serum potassium levels below $3.8 \mathrm{mmol} / \mathrm{I}$ and none with levels above 5.5 mmolil. BUN levels ranged from 4.1 to $65 \mathrm{mmol} / 1$ with a mean of $14.4 \pm$ $22.1 \mathrm{mmol} / 1.13(59.1 \%)$ patients had BUN above $6.6 \mathrm{mmol} / 1$.

The serum creatinine ranged from $67 \mu \mathrm{~mol} / 1$ to $315 \mu \mathrm{~mol} / 1$ with a mean of $132 \pm 63 \mu \mathrm{~mol} / 1$. Six ( $35.3 \%$ ) had levels above $133 \mu \mathrm{~mol} / 1$ and it was not determined in 5 paitents.

Serum uric acid levels ranged from 110 jumol/1 to $1,395 \mu \mathrm{~mol} / 1$ with a mean of $417 \pm 321 \mu \mathrm{~mol} / 1$. Five
( $29.4 \%$ ) patients had levels above $420 \mu \mathrm{~mol} / 1$. Five patients did not have their uric acid levels det:ermined.

The total serum proteins ranged from $55 \mathrm{~g} / 1$ to $86 \mathrm{~g} / 1$ with a mean of $70 \pm 10 \mathrm{~g} / 1$. Six ( $22.7 \%$ ) had serum alkaline phosphatase levels which were higher than 15KA unit. Only one patient had a significantly raised SGOT level.

The triglycerides were markedly raised ir all the patients with a mean of $582 \pm 127 \mathrm{mg} \%$. The cotal serum cholesterol ranged from $108 \mathrm{mg} \%$ Eo 23? mg\% with a mean of $189 \pm 36 \mathrm{mg} \%$. No patient had levels above $250 \mathrm{mg} \%$. The serum high density lipoprotein levels ranged from $13 \mathrm{mg} \%$ to $48 \mathrm{mg} \%$ with a mean of $36 \pm 9 \mathrm{mg} \%$. No patient had levels above $55 \mathrm{mg} \%$ while 6 (28.6\%) had levels below $35 \mathrm{mg} \%$ 。

The serum low density lipoprotein levels ranged from 37 to $138 \mathrm{ag} \%$ with a mean of $96 \pm 18 \mathrm{mg} \%$. No patient had levels above $150 \mathrm{mg} \%$ 。

In the hyfertensive patients, the chest xray showed cardiomegaly in $16(72.7 \%)$ and was normal in 2. $(9.1 \%)$. It was not done in one patient. The electrocardiogram was abnormal in 15 (68.2\%) and normal in $3(13.6 \%)$. It was not done in 4 patients. The abnormalities noted were: tachycardia, left ventricular hypertrophy, left axis deviation, right ventricular hypertrophy and low voltage QRS complexes. Urine examination was abnormal in 6 (27.3\%) patients with hypertension. It was normal in 5 (40.1\%)
patients and was not done in 7 ( $31.8 \%$ ) patients. The abnormalities of the urine included glycosuria, and bacteruria (culturing Klebsiella, proteus, citrobacter, and salmonella typhimurium). Serological test for syphilis was not positive in any of the hypertensive patients. Blood cultures revealed septicaemia in one patient where a heavy growth of staphylococcus albus was obtained. Carotid angiogram was done in only one patient with hypertensive stroke and it was normal. Computerised axial tomography done in one patient showed infarction in the left cerebellar hemisphere and brainstem. In one patient ultrasonography showed enlarged kidneys with bilateral hydronephrosis. Three patients with hypertension were aged 45 years or younger.

The haemoglobin level ranged from $6.3 \mathrm{~g} / \mathrm{d} 1$ to $20.5 \mathrm{~g} / \mathrm{dl}$ with 2 mear of $14.0 \pm 2.0 \mathrm{~g} / \mathrm{dl}$. The white blood cell count in normotensive patients ranged from $4,1 \times 10^{9} / 1$ to $1066 \times 10^{9} / 1$ with a mean of $28.9 \pm 49.7 \times 10^{9} / 1$. This was due to a few of the patients who had extremely high WBC counts. No patient had a reading below $3.5 \times 10^{9} / 1$ while 5 ( $10 \%$ ) had readings abcve $11.0 \times 10^{9} / 1$, one of these had a reading of $32.0 \times 10^{9} / 1$ while the other one had the incredible figure of $1066.0 \times 10^{9} / 1$.

The erythrocyte sedimentation rate of the normotensive patients ranged from $1 \mathrm{~mm} / \mathrm{h}$ to $60 \mathrm{~mm} / \mathrm{h}$ with a mean of $29 \pm 20 \mathrm{~mm} / \mathrm{h}$. Five ( $15.2 \%$ ) of the
patients had an ESR of I or 2, while 20 (60.6.\%) had an ESR above 20. ESR's were not determined in 17 patients.

The blood sugar levels in normotensivas ranged from $2.1 \mathrm{mmol} / 1$ to $22.2 \mathrm{mmol} / 1$ with a mean of $4.9 \pm$ 3.1 mmol/1. One patient had a blood sugar level of 22.2 mmol/l, while the rest had blood sugar levels <ll mmol/1. Four patients had hypoglycaemia with blood sugar levels below $2.6 \mathrm{mmol} / \mathrm{l}$.

The serum sodium levels in normotensives ranged from $130 \mathrm{mmol} / \mathrm{l}$ to $160 \mathrm{mmol} / \mathrm{l}$ with a mean of $\mathrm{i} 35 \pm$ 21 mmol/1. Three patients (6.4\%) had levels above 150 mmol/l while none had levels below 130 mmol/ .

The potassium levels in normotensive patients ranged from $2.8 \mathrm{mmol} / 1$ to $5.6 \mathrm{mmol} / \mathrm{l}$ with a mean of $4.4 \pm 0.7 \mathrm{mmol} / 1$. One ( $2 \%$ ) had a reading above 5.5 mmol/1 while another $1(2 \%)$ had a level below 3.5 mmol/1. The blood urea nitrogen levels for normotensives ranged from $2.4 \mathrm{mmol} / 1$ to $16.3 \mathrm{rmmol} / 1$ with a mean of $5.4 \pm 2.5 \mathrm{mmol} / 1$. Ten ( $20 \%$ ) had BLN levels above 6.7 mmol/l.

The uric acid levels in serum of normotensives ranged from $160 \mu \mathrm{~mol} / 1$ to $608 \mu \mathrm{~mol} / 1$ with a mean of $376 \pm 36 \mu \mathrm{~mol} / 1$. Ten patients ( $31.2 \%$ ) had uric acid levels above $420 \mu \mathrm{~mol} / 1$. The serum creatinine levels ranged from $45 \mu \mathrm{~mol} / 1$ to $144 \mu \mathrm{~mol} / 1$ with a mean of $97 \pm 23_{\alpha} \mu m o l / 1$. Twelve (34.3\%) of patients had a serum creatinine level over $106 \mu m o l / 1$.

The liver function tests done included total
seruin proteins, albumin, glutamate-oxalo-acetate transaminase (SGCT), glutamic-pyruvic acid transaminase (SGPT), and alkaline phosphatase. The total serum protein in normotensive patients ranged from $38 \mathrm{~g} / \mathrm{l}$ with a mean of $70 \pm 19 \mathrm{~g} / \mathrm{l}$. Five ( $10 \%$ ) patients had levels above $85 \mathrm{~g} / 1$ while 7 (14\%) had levels below $60 \mathrm{~g} / 1$. The $\exists$ bumin levels ranged from $15 \mathrm{~g} / 1$ to $52 \mathrm{~g} / 1$ with a mean of $34 \pm 8 \mathrm{~g} / 1$. The SGOT levels ranged from 2 units/1 to 192 units/l with a mean of $46+40$ units/1. The range of SGPT was from 10 units/l to 197 units/l with a mean of $45 \pm 37$ units/1. The serum alkaline phosphatase levels ranged from 3.0-37.1 K.A. units with a mean of $15.4 \pm 6.3 \mathrm{~K} . \mathrm{A}$. units.

The lipid profile in normotensives showed that there was markedly raised triglyceridemia with a range from $450 \mathrm{mg} \%: 086 \mathrm{mg} \%$ with a mean of $595 \pm 116 \mathrm{mg} \%$. None of the patients had normal serum triglycerides. The total serum cholesterol ranged from $112 \mathrm{mg} \%$ to $293 \mathrm{mg} \%$ with a mean of $195 \pm 51 \mathrm{mg} \%$ 。

The high density lipoprotein cholesterol level ranged from $10 \mathrm{mg} \%$ to $72 \mathrm{mg} \%$ with a mean of $31 \pm 10 \mathrm{mg} \%$ while the low density lipoprotein cholesterol levels ranged from $22 \mathrm{mg} \%$ to $158 \mathrm{mg} \%$ with a mean of $94 \pm 30 \mathrm{mg} \%$ 。

The young normotensive patients were then aralysed. The cut off age was arbitrarily taken as 45 years. There were 10 males and 12 females
in this group. The youngest patient was 15 years of age. Six ( $27.3 \%$ ) were students: six ( $27.3 \%$ ) were farmers, $2(9.1 \%$ ) were unemployed and there was 1 (4.5\%) each of a secretary, waiter, housemaid, watchman and unknown occupation. Six ( $27.3 \%$ ) were urban dwellers while $16(72.7 \%$ ) were rural dwellers. Examination, history and laboratory work up indicated the following associated factors in these young normotensive patients: high ESR in 8 (36.4\%), mitral valve stenosis and regurgitation in $7(31.8 \%)$, anaemia in 4 ( $18.2 \%$ ), polycythemia in 3 ( $13.6 \%$ ), atrial fibrillation in 3 ( $13.6 \%$ ), heart palpitation in $2(9.1 \%)$. There was $1(4.5 \%)$ each of the following conditions: pneumonia, deep vein thrombosis, connective tissue disease, mitral valve prolapse, postpartum septicaemia, alcohol intake, cigarette smokin己, VDRL-positivity, sickle nell disease, arterio-venous malformation, hepatitis, malaria, an intracranial space occupying lesion and ulcerative colitis.

## NORMOTENSIVE PATIENTS:

Twenty two (44\%) normotensive patients were aged 45 years or below while 28 ( $36 \%$ ) were older than 45 years. Twenty three ( $46 \% 0$ were males while twenty seven (54\%) were females, 21 ( $42 \%$ ) were farmers, 9 ( $18 \%$ ) housewives, 5 ( $10 \%$ ) students, 2 ( $4 \%$ ) watchmen, 2 ( $4 \%$ drivers, and $2(4 \%$ ) were unemployed. There was one each of a businessmen, secretary, waiter and housemaid. In 5 ( $10 \%$ ) the
occupation was unknown. Seven (14\%) had history of alcohol consumption and cigarette smoking; 5 (10\%) ha! previous history of stroke, 4 ( $8 \%$ ) had history of heart disease, $3(6 \%)$ had history of chronic chest pain, cough and hemoptysis, 2 ( $4 \%$ ) had used contraceptive pills while 2 ( $4 \%$ ) had used intrauterine contraceptive devices, $2(4 \%)$ had pneumonia. The following occurred in one patient each: cold ischaemia of the foot, chronic headaches, recent parturition, trauma, ulcerative colitis, bronchial asthma, congestive cardiac failure, deep vein thrombosis, connective tissue disease, duodenal ulcer, dementia and mental depression. Physical examination in the normotensive patients showed that 20 (40\%) had left-sided stroke while 30 (60\%) had right-sided stroke and in one the stroke was bilateral. Signs of atherosclerosis was found in 11 ( $22 \%$ ) patients. Twenty two ( $44 \%$ ) had grade I or II retinopathy. $3(6 \%)$ had grade III or IV retinopathy, 6 (12\%) had atrial fibrillation, 10 ( $20 \%$ ) had cardiomegaly, 13 ( $26 \%$ ) had heart murmurs, and there was 1 each of tachycardia, and abdominal malignancy. On laboratory work up, chest x-ray showed cardiomegaly in 14 ( $28 \%$ ), the electrocardiogram was abnormal in 14 ( $28 \%$ ), urine examination was abnormal in 5 (10\%). VDRL test was positive in 2 (4\%), though both were negative for the more specific test of TPHA. Blood culture was positive in one patient; carotid angiogram was abnormal in

4 cases, the two dimensional echocardiogram was abnormal in two, the antinuclear factor and rheumatoid factor in one, the widal test was positive in one and $T_{3} / T_{4}$ levels were significantly raised in one.

The pattern of $\mathrm{Hb}, \mathrm{WBC}$ and ESR in normotensive patients is shown in Figure IF.

## DISCUSSION

As stated at the outset, the aim of the study was to document the occurreace of stroke in terms of clinical presentation and laboratory workup at Kenyatta National Hospital, i.e., it was a descriptive study and no attempt was made at a comparative analysis of any of the findings. Seventy two consequtive patients were enrolled in the study all coming from the medical wards. Only those who survived Iong enough to have the various clinical and Laboratory parameters studied were included. Hence, a number of stroke patients were excluded on the account of early demise following admissions. Other patients presenting to KNH with stroke but who got admitted to other wards, e.g., surgical, paediatric and gymiscological wards for other reasons were also exciuded. In that period, January, 1986 to January, 1987, there weze 4704 patients admitted to the adult medical wairds. 0 these, 1084 had neurological problems constituting $23 \%$ of all medical admissions and of those, 193 were recorded as having had CVA as described in the introduction. This was $18 \%$ of all neurological admissions. In 195?, Shaper and Shaper iound that neurological admissions constituted $10.5 \%$ of total medical admissions in Mulago Hospital (10). This was lower than in this present study. They found that CVA constituted $11.4 \%$ of all neuroiogical admissions. This is also a lower figure compared
with the findings in this study of $18 \%$. Dada et al (11) in a similar study at the University of Lagos, found CVA's to constitute $16.8 \%$ of all the neurological admissions. It is important to note that in this particular study, subarachnoid haemorrhage was included amongst the CVA's; and accounted for $13.6 \%$ of all CVA's. Billinghurst (9), about two decades̄ ago, studied the pattern of neurological admissions to Mulago Hospitai, Kampala and found that out of all 6144 medical admissions, neurological cases accounted for $9.6 \%$ This again is lower than the findings of the current sudy. In that study, CVA's constituted $34.0 \%$ of neurological cases. This is rather high compared to findings of the current study, but that is probabiy because no attempts were made by Billinghurse to subdivide the vascu?ar problems further; and the study included sucn entities as subdural haematoma, diffuse 3 theroscleoris, extradural haemorrhage and venous sinus thrombosis. These differences suggest that there is a higher incidence of neurological-admissions and CVA's. The aetiological factors to stroke were analysed and the patients were divided into two broad groups, namely: hypertensive and normotensive patients. The patients were examined in terms of the history, physical examination and laboratory findings including other investigative techniques from all these modalities of evaluation, it was noted that hypertension occurred in 22 patients
constituting $30.6 \%$ of all stroke patients. This is slightly lower than found by Bahemuka (8) at KNH ( $47.8 \%$ ) in a retrospective study of 207 cases in 1985. Billinghurst (9) found hypertension to constitute $45 \%$ of his cases. Osuntokun et al (12) found an even higher frequency of hypertension amongst cerebrovascular accident patients. They reported $53.6 \%$ at University College Hospital, Ibadan in 1969. The age distribution of the patients was from 15 years to 88 years with a mean of 52 years. The highest incidents were found in the sixth and seventh decades, both accounting for $44.4 \%$ of the patients. Of necessity there were no patients included below 12 years of age which is the operational dividing line between paediatric and adult admissions to KNH . No patients were seen older than 88 ycars. These findings are in desping with those of Bahemuka (9) who found a righer incidence in the fifth, sixth and seventh decades, the three totalling $59.3 \%$ of all his cases. Osuntokun (12) found a similar age distribution in Nigeria. Majority of the patients (65.3\%) in the current study were from a rural setting. This is a most interesting finding considering that hypertension has hitherto been thought of as an urban problem largely attributed to westernization, among other things. It is not clear whether this may be related to the other equally interesting finding patients
that the majority of the $\left(\begin{array}{l}(58.3 \%)\end{array}\right.$ came from the
central province of Kenya. Could this be a reflection of the level of "westernization" of the life style and foods and so on with its accompanying high level of "stress" etc? This calls for further scrutiny. It should be noted though that KNH is a National Referral hospital situated within the central province, so that the figures may only reflect ease with which patients get referred from the immediate environs, There was no literature available with which to compare these last two observations.

In terms of occupation, farmers occupied the first spot (45.8\%). This is, again, an interesting observation though it may probably be tied together with the preponderance of iural dwellers in the series. Housewives were second at $18.1 \%$. The group one would have expected to feature , rrominently, businessmen, only had 2 representatives constituting $2.8 \%$. This is again, an interesting observation as this group is usually thought to have risk factors such as stress. May be most of the businessmen with stroke would go to the private (paying) hospitals rather than to KNH . Of the hypertensive patients, $50 \%$ had a positive history. History of smoking was elicited in 12 (16.7\%). This is a very low incidence compared to the findings of a rate of $63.3 \%$ by Lederman et al (15) in the U.K. in 1985. The relationship between smoking and stroke has now been well established. Abbot et al (26) studied
this phenomen conclusively and found that cigarette smoking on a long term basis adversely affected the occurrence of stroke and probably had implications in prevention. A case control study was conducted by bonita, et al (24) in which they looked at a total of 132 cases of stroke and found that cigarette smokers had a three-fold increase in the risk of stroke compared to current non-smokers. This risk remained significant after adjusting for the risk from concurrent hypertension. Smoking and hypertension increased the risk to 20 -fuld compared to those who neither smoked nor had hypertension. Others have also noted the important contribution of smoking to stroke development $(20,27)$.

A history of alcohol consumption was elicited in 14 ( $19.4 \%$ ) of the patients. This is a recognised risk factor to the development of stroke as reported in several pape:s: (1.5-18). The study by Gill et al (18) indicates that the risk of stroke from al.cohol consumption is dependent on the sex and the anount of alcohol consumed. Among men, the relative risk of stroke (adjusted for hypertension, cigarette smoking and medication) was lower in light drinkers (those consuming 10 to 90 g of alcohol weekly) than in non drinkers, but four times higher in heavy drinkers (consuming $>300 \mathrm{~g}$ weekly) than in nondrinkers. There was not enough data to comment on the effect on women.

Diabetes mellitus was noted in $5(6.9 \%$ ) of the
patients. This is a well recognised risk factor to development of stroke $\exists 11$ over the world. The incidence compared well with other studies.

Bahemuika (8) found an incidence of $5.3 \%$, Billinghurst (9) found 3\%, Osuntokun et al (12) found: $8.5 \%$, Bahemuka (13) found an incidence of $4.2 \%$ young normotensive patients. Hilton-Jones and Warlow (14) found an incidence of $3.3 \%$, Schneider at al (20) found an incidence of $25 \%$. probably the 'highest incidence reported so far. Lederman et al (15) found an incidence of $9.5 \%$. Matenga et al (27). found an incidence of $3.2 \%$ in Harare; and many others have shown this correlation between diabetes and stroke.

Trauma to the head is an interesting association to stroke. The direct injury caused to the brain. with subsequent bleeding is obvious enough, but often patients who have had trauma to the head will present with stroke several weeks later and investigations may not reveal any blood clot to account for the ischaemia. In the current study, the only factor that could be found in $4(5.6 \%)$ patients was a history of trauma to the head several weeks before. In these 4 patients, carotid angiograms and skull x-rays were done and these did not show any abnormality. It was not clear how trauma led to stroke in these patients. Bahemuka (13) found this phenomenon in $11(30.6 \%)$ of 36 patients _ he studied with stroke. These 11 included those
in whom no possible cause for the stroke had been determined. Hilton-Jones and Warlow (14) found trauma to be the most common cause of stroke in the young at the Radcliff Infirmary in the U.K. They took patients below 45 years. They noted that the "trauma" may consist, as did in one of their patients, of being grabbed around the neck in a scuffle. The stroke developed the following day. There were no neck bruits to suggest dislodging of an atheromatous material.

Congestive cardiac failure was noted in 3 ( $4.2 \%$ ) of the patients. Bahemuka (8) found an unspecified number of his patients had cardiomyopathy with congestive cardiac failure, Billinghurst (9) did not find any, and in the other works, heart disease was reported in terms of the specific underlying cause other than as congestive cardiac failure. It would appear that the sluggishness of circulation may encourage thrombus formation in the heart chambers which may form source of the emboli. On the other hand, the failing heart's inability to pump enough oxygenated blood to the brain may be the underlying factor. No clear explanation can be offered for the association between stroke and duodenal ulcer disease found in 2 patients. The finding of an infective process (pneumonia), bronchial asthma, sickle cell disease, use of contraceptive pill, and
stroke occurring in the immediate post-partum is in conformity with other studies $(8,9,10,12,13)$. Bahemuka (13) found infections to be the most common of the associated factors in his series on the young normotensive patients. These included conditions like upper respiratory tract infection, cavernous sinus thrombosis, intracranial tuberculosis, malaria, typhoid fever, neurosyphilis, pyogenic meningitis. In this study, only one case could be ascribed to malaria. Three other cases had urinary tract infec; tion. One had pneumonia, one had septicaemia, two had positive VDRL but these were non-reactive to the more specific treponemal serological tests, one patient had hepatitis.

Chronic headaches thought to be secondary to migraine was elicited in one young normotensive patient. Migraine is a well recognized association with stroke. In the series by Hilton-Jones and Warlow (14) migraine was the second commonest association with stroke in the young normotensive patients accounting for $10 \%$ of the cases. Henrich et al (23) studied this association in some detail and found that migraine most often was associated with cerebral infarction type of stroke (244 patients) as opposed to cerebral haemorrhage (28 patients) or subarachnoid haemorrhage. This tended to suggest that migraine might be a direct causative factor via the vasospasm/vasodilatation phenomenon.

Hypertensive patients as a group tended to be older, only 3 being under 45 years. This is in contrast to elsewhere where the tendency has been for hypertensive complications to occur in the younger male biack patients. One of the postulates had been that probably if blood pressure control had been good then the risk of complications, including strokes might be lowered. This, however, may not be tenable in our local set up as $50 \%$ of the patients had not given previous history of hypertension prior to the event and were found to be hypertensive for the first time after admission for stroke. Matenga and colleagues (27) found that $44.9 \%$ of their patients presenting with stroke in Harare had no previons history, A further 44.9\% had previously been diagnosed but had then "absconded" from treatment, Bahemuka (25) found that only 6(13.3) had previous history of hypertension. In the present study of the patients who had hypertension for one year or more, $10 \%$ had good control as defined earlier, $70 \%$ had fair control, while $20 \%$ had either absconded from treatment and followup or had poor control of their blood pressure. From this, it would appear that a very strict control, of blood pressure is the only acceptable aim of treatment in terms of preventing strokes. But considering the average age of the patients presenting with hypertensive stroke, it is
not clear whether this is feasible as most elderly people would have diastolic pressure levels above 95 mmHg and yet not constitute a need for antihypertensive therapy. This opinion, namely, that the level of the blood pressure may not be the whole story in terms of neurological complications, is shared by many including Bahemuka (28) who in a study of 34 consecutive patients admitted to KNH from 1975 to 1979 with malignant hypertension and followed up upto 1982 , only 2 patients presented with stroke, and neuro-psychiatric syndromes were not observed in any of them. Against this is the fact that many reviewers think otherwise $(13,29)$. 'I'he frost common associated finding amongst hypertensives was an elerated uric acid level (54.5\%). This was followed by renal insufficiency (25.4\%) and a high ESR (31.8\%). It was difIicult to trace any literature discussing hypertensive stroke, high uric acid and ESR. The other findings were the expected ones, e.g., diabetes mellitus (18.2\%), history of cigarette smoking (18.2\%), family history of hypertension (18.2\%) and alcohol consumption (13.6\%).

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    Physical examination of the hypertensive
patients revealed signs of atherosclercsis (palpa-
ble radial artery, serpentigeous brachial artery,
etc) in lo (45.5%). This is in contrast to
previous studies which have tended to find no
signs of atherosclerosis in the African hypertens-
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ive patient (8,13). Grade I and grade II retinopathy was noted in $86.4 \%$ of the patients. The significance of this is not clear as in a concurrent. study by Ngumuta (30) a large majority of normotensive elderly people have Grade I \& II retinopathy. Fifty per cent of the patients had Grade III and IV retinopathy. This was thought to be related to the hypertension rather than to the development of stroke per se. Cardiomegaly, by clinicai examination, chest x-ray and ECG studies, was noted in $59.1 \%$ of the hypertensive patients. Again, this is explainable by the expected end-organ damage.

The haemoglobin in hypertensives ranged from $8.7 \mathrm{~g} / \mathrm{dl}$ to $18.8 \mathrm{~g} / \mathrm{dl}$ with a mean of $14.3 \pm 2.3 \mathrm{~g} / \mathrm{dl}$. This is in agreement with the local figures (31). Hence anaemia drses not seem to have played sn important part. Only 3 patients had a haemoclobin value below $12 \mathrm{~g} / \mathrm{dl}$. Six (27.3\%) had a haencglobin above $15 \mathrm{~g} / \mathrm{dl}$ and in fact 2 had a haemoglobin level above $18 \mathrm{~g} / \mathrm{dl}$. These may have had a part to play in the causation of stroke. Osuntokun (12) had 3 patients in his series with haemoglobin levels above $18 \mathrm{~g} / \mathrm{dl}$ but they all had normal WBC's. The WBC's in this series ranged from $3.9 \times 10^{9} / 1$ to $15.1 \times 10^{9} / 1$ with a mean $\pm$ Standard Deviation of $8.8 \pm 3.5 \times 10^{9} / 1$. Kyobe et al (32) in a study of normal subjects at Makerere College, Kampala, found the WBC to have a mean of $5.5 \times 10^{9} / \mathrm{L}$, But this was a much younger population.

Kasili et al (33), revicwing blood from donors found the WBC's to range from 2.0-10.7 $\times 10^{9} / 1$ with a mean of $5.0 \times 10^{9} / 1$. Allen et al. (34) had done a similar study in Kenyan Africans in 1959 and found the WBC to range from 3.0 to $9.1 \times 10^{9} / 1$. with a mean of $5.5 \times 10^{9} / 1$. Hence the current study indicates that the WBC counts were probably elevated. However it is not easy to compare this with the other studies quoted as the age range is not comparable. The ESR range was from $1 \mathrm{~mm} / \mathrm{hr}$ to $55 \mathrm{~mm} / \mathrm{hr}$ with a mean of $22 \pm 14 \mathrm{~mm} / \mathrm{hr}$. This tends to show that the majority of these patients had a raised ESR. Fifteen (68.2\%) had an ESR above $15 \mathrm{~mm} /$ nr. Whether this is a general response to the stroke event itself or a reflection of some underlying problem waich also led to the stroke is not clear. Whether the ESR was high beiore the stroke occurred is not clear either, If it could be shown that the ESR goes up just before the stroke occurs, then it might be a good screening test in those who are at risk of developing stroke. Kyobe, et al. (32) found the ESR in his series of healthy young adults to be $6 \pm 8 \mathrm{~mm} / \mathrm{hr}$. A study to show the effect of age on ESR and a controlled trial on ESR and stroke development might be interesting. The renal function tests showed most parameters to be within normal limits except the BUN which showed an elevation with a range from 4.1 to 65 mmol/l with a mean of $14.4 \pm 22.1 \mathrm{mmol} / 1$.

The serum creatinine was also elevated with a range from $67, \mu \mathrm{~mol} / 1$ to $: 15 \mu \mathrm{~mol} / \mathrm{l}$ with a mean of $132 \pm 63 \mu \mathrm{~mol} / \mathrm{I}$. The local laboratory range for normal serum creatinine is from 62 to $106 \mu \mathrm{~mol} / \mathrm{l}$ (31). The elevation of BUN and creatinine was expected from the effect of hypertension on the kidneys.

The total serun proteins in hypertensives was within normal limits and hyperproteinaemia did not seem to be an important factor in the aet lopathogenesis of stroke in the hypertensive patients. None was cilnically faundiced and none showed features of compromised liver function.

The total serum triglyceride levels were extremely elevater in all the patients. This was most surprising the ing several studies have shown that patients with hyper'lipidaemia have q high Iisk to development of stroke yet the figures were extremely high. There was really no reference material with which to compare these readings as the method used was the new fully enzymatic method, but the manufacturers recommend $200 \mathrm{mg} / \mathrm{dil}$ as the cut off point for definite hypertriglyceridaemia (35). Most of the readings obtained were three times higher than this. Further tests will need to be done using this method in order to establish appropriate normal levels for our local population hence, it would probably not be appropriate to make a categorical statement that triglyceride levels MEDical library
in all stroke patienis at least not at this stage. The total serum cholesterol ranged from $100 \mathrm{mg} / \mathrm{dl}$ to $237 \mathrm{mg} / \mathrm{dl}$ with a mean of $189+36 \mathrm{mg} / \mathrm{dl}$. This was within normal limits according to the recommendation of the manufacturers of the test kit who give an upper cutoff point of $260 \mathrm{mg} / \mathrm{dl}$ (35). There were no patients with readings above this level. Okelo, Kanja and Kyobe (36) looked at the serum lipid profile in diabetics and non-diabetic controls at the University of Nairobi and found the serum trigiycerides and cholesterol to be significantly elevated in comparison to controls. Ojwang, et al (37) worked out a reference level for serum lipids in Africans aged between 20 years and 40 years. They found that the levels for serum cholesterol was $4.73 \pm 2.7 \mathrm{~mm} . \mathrm{l} / \mathrm{l}$, for men and 4.46 $\pm 0.76$ for women while t'iat for serum triglyceride was $0.83 \pm 0.36 \mathrm{mmol} / 1$ for men and $0.66 \pm 0.39 \mathrm{mmol} / 1$ for women. The HDL-cholesterol in men was $1.0 \underline{f} \pm 0.34 \mathrm{mmol} / 1$ while that in women was $1.18 \pm 0.25 \mathrm{mmol} / 1$. These are closely similar to those found by Okelo et al and slightly lower than those in the current study except for the levels for serum triglycerides already referred to earlier. The normotensive patient with stroke is always a diagnostic problem especialiy if that patient is young and espectally in the developing world where the diagnostic options are limited. Sixty nine per cent of the patients in this series
were normotensive and of these 22 ( $44 \%$ ) were aged 45 years or below. There was a slight preponderance of males over females (54\%:46\%). Lederman et al (15) found males to be more $(88: 46)$ in his study of young stroke patients also. Bahemuka (13) also found a male preponderance of $2: 1$. There has been no attempt to explain this male preponderance of stroke, especially in the normotensive patient.s. The occupation distribution was similar to that of hypertensives. There were more right-sided hemiplegics than left-sided ( $60 \%: 40 \%$ ). This was only significant in that all the patients were right handed and a large proportion of these patients were aphasic. Whereas $46 \%$ had grade I cr II retinopathy, only $5 \%$ had grade III or IV retinopathy.

The haemogram in normotensive patients showed the Hb to range irom $6.3 \mathrm{~g} / \mathrm{dl}$ to $20.6 \mathrm{~g} / \mathrm{dl}$ with a mean of $14.0 \pm 2.6 \mathrm{~g} / \mathrm{dl}$. The majority had a range from $12 \mathrm{~g} / \mathrm{d} 1$ to $18 \mathrm{~g} / \mathrm{dl}$. Four ( $8 \%$ ) had a Hb above $\mathrm{L} 8 \mathrm{~g} / \mathrm{dl}$ and in these, the polycythemia was thought to have played some role in the causation of stroke. Anaemia might also be an important factor because upto 9 (18\%) had a Hb below $12 \mathrm{~g} / \mathrm{d} 1$. Bahemuka (13) did not find anaemia or polycythemia to be an important factor in his series. Hilton-Jones and Warlow (14) reported anaemia in $3(21.4 \%$ ) of the patients with ischaemic strokes. He did not find any patient with polycythemia. Lederman et al (15) reported no cases of anaemia but had $13 \%$ of his
normotensive patients with a ligh packed cell. volume. The white blood cell count in normotensive patients ranged from $4.1 \times 10^{9} / 1$ to $1066 \times 10^{9} / 1$ with a mean of $26.9 \pm 149.7 \times 10^{9} / 1$. The high mean was due to 3 patients who had extremely high WBC levels. One had a level of $1066 \times 10^{9} / 1$ and diagnosed to have a myeloproliferative disorder on bone marrow examination most probably granulocytic leukemia. The other one had a count of $19.4 \times 10^{9} / 1$, an infective process being considered the culprit, while the third one had a count of $32.0 \times 10^{9} / 1$. This was a 58 year old female who had a stroke six months before, she had atrial fibrillation with mitral stenosis and mitral regurgitation, she was on therapy with warfarin and she also had biochemically confirmed hyperthyroidism. The ESR in normotensive fatients was raised in 22 ( $44 \%$ ) of cases. The range ras $1 \mathrm{~mm} / \mathrm{hr}$ to $60 \mathrm{~mm} / \mathrm{hr}$, with a mean of $\angle 2 \pm 20 \mathrm{~mm} / \mathrm{ır}$. Hilton-Jones and Warlow (14) found a high ESk in 2 of 14 ( $14 \%$ ) of his patients.

The renal function tests showed that uremia occurred in $20 \%$ of the cases. It was not immediately apparent what caused this uremia and it was not easy to find any reference to uremia as a cause of stroke. The other interesting finding was that $31.2 \%$ of the normotensive patients had an elevated uric acid level $(>420$ umol/1). Whether this was related to the uremia or whetther it had a direct causative effect on stroke was not clear either and may need
further study. Ten per cent of normotensive stroke patients had total protein levels above $85 \mathrm{~g} / 1$. This may have the effect of making blood hyperviscous and this might in turn lead to stroke in patients who are predisposed.

The lipid profile of normotensive patients showed a similarly high levels of triglycerides as in hypertensives and similar comments apply. Eight (16\%) of the patients had significantly ellevated serum total cholesterol. These were not mecessarily those with renal insufficiency as only 2 of these 8 had elevated BUN or serum creatinine. Again the same problems as noted for hypertensive patients in terms of interpreting serum cholesterol results apply.

The young normorensive patients constituted 22 of the patients. There was an equal distribution of the sexes. Here there were more stuments. The diagnostic work-up was limited as has been judicated, but with what was available, not all the patients had a recognisable factor causing the stroke. Here again rural dwellers out-numbered urban dwellers ( $72.7 \%: 27.3 \%$ ). A. high ESR was noted in 8 ( $36.4 \%$ ). The other associated findings are similar to those noted in other studies. In one patient, echocardiography showed anterior myocardial infarction. This has not been reported before in the local literature.

## CONCLUSION AND RECOMMENDATIONS

The study carried out over about twelve months incorporated 72 patients. It was found that at KNH, stroke (cerebrovascular accident) constituted $18 \%$ of all neurological admissions. This is higher than in most other series. Hypertension constituted $30.6 \%$ of all stroke cases. The majority of the patients (58.3\%) originated from the central province. Sixty five point three per cent of the patiencs lived in the rural areas. Forty five point eight per cent were farmers, followed by housewives (10.1\%) 。

Fifteen point three per cent had a previous history of hypertension. Cigarette smoking and alcohol consumption were fuund to be important associated factors. This is in keeping with data now emerging from other parts of the worls indicating that these two habits, especially in the setting of hypertension are serious risk factors. Other coexisting conditions were noted but their weighting in terms of aetiology was not clear.

Good control of hypertension was seen to be possibly useful in preventing strokes by extrapolation though it was noted that there are reports throwing doubt on this assertation. It was noted that only about $50 \%$ of hypertensive stroke patients had a previous history of hypertension. Signs of atherosclerosis was noted in $45.5 \%$ of hypertensive patients. This has not been previously reported
from this part of the world.
Triglycerides were estimated using a new enzymatic technique and found to be elevated in all the patients. It was difficult to interpret this considering the method had not been previously used in Kenya and there was no data to compare it with.

No patient was found to have syphilis and the importance of this disease as a cause of stroke may be truly diminishing.

The young normotensive patients were found to have a raised ESR, mitral valve disease, anaemia polycythemia, urinary tract infection, atrial fibrillation, heart disease with palpitations, pneumonia, deep vein thrombosis, connective tissue disease, mitral valve prolapse, post-partum period, septicaemia, alcohol consumption, smoking, sickle cell disease, arterio-venous malformation, hepatitis, malaria, ulcerative colitis, and contraceptive pill use, as associated conditions. In four young normotensive patients, no cause or associated conditions could be found to explain the occurrence of stroke. It is recommended that:-

1. Treatment of hypertension, with follow-up and good control is still probably the single most important method of preventing strokes in patients attending K.N.H. The control would seem to be effective only when diastolic level is kept below 95 mm Hg .
2. The reason for the high incidence of strokes in patients from central province of Kenya should be studied to ascertain its statistical significance and to look for the possible reasons for its occurrence.
be
3. More attention should/ paid to the study and documentation of hypertension and stroke incidences in the rural areas of Kenya as it would seem from this study that they form the majority of our patients.
4. Cigarette smoking, alcohol consumption and hypertriglyceridaemia are important states which constitute risk factors to the occurrence of stroke and that the control of these may be an important step in preventing strokes. The first two may be achieved through intensive health education while the third may be achieved through both health education and drug therapy.
5. A further controlled trial should be undertaken to establish the status of serum lipids especially using the enzymatic method which is repcrted by the manufacturers to be highly sensicive, so local norms can be established particularly for the age range which suffers strokes and also to establish what levels require therapy.
6. The young normotensive stroke patient should be fully investigated as in the majority of cases, a treatable underlying cause
for the stroke can be demonstrated. In this rogard, it is hoped that computerized tomographic scanner will soon be available so that more accurate and safer evaluation can be made.

TABLE I: REGION OF ORIGIN OF STROKE PATIENTS

| (REGION) | NUMBER (\%) |  |
| :--- | :---: | :---: |
| Central | 42 | $(58.3)$ |
| Nyanza | 9 | $(12.5)$ |
| Western | 8 | $(11.1)$ |
| Eastern | 7 | $(9.7)$ |
| Coast | 2 | $(2.8)$ |
| Rift Valley | 1 | $(1.4)$ |
| North Eastern | 1 | $(1.4)$ |
| Nairobi | 1 | $(1.4)$ |
| Not known | 3 | $(4.2)$ |

TABLE II: AGE DISTRIBUTION IN STROKE PATIENTS

| AGE (YEARS) | NUMBER OF PATIENTS |
| :---: | :---: |
| $11-20$ | 4 |
| $21-30$ | 9 |
| $31-40$ | 8 |
| $41-50$ | 6 |
| $51-60$ | 17 |
| $61-70$ | 15 |
| $81-80$ | 8 |
| $91-100$ | 3 |
| Not known | 0 |
|  | 2 Both elderly |

TABLE III: OCCUPATIONS OF STROKE PATIENTS

| OCCUPATION | NUMBER (\%) |  |
| :--- | :--- | :--- |
| Farmer | 33 | $(45.8 \%)$ |
| Housewife | 13 | $(18.1 \%)$ |
| Student | 5 | $(6.9)$ |
| Businessman | 4 | $(5.6)$ |
| Driver | 3 | $(4.2)$ |
| Watchman/guard | 2 | $(2.8)$ |
| General worker | 1 | $(1.4)$ |
| Secretary | 1 | $(1.4)$ |
| Machine operator | 1 | $(1.4)$ |
| Waiter | 1 | $(1.4)$ |
| Fitter | 1 | $(1.4)$ |
| Housemaid | 4 | $(5.6)$ |
| Not known | 4 | $(5.6)$ |

TABLE IV: ASSOCIATED CONDITIONS IN STROKE PATIENTS

| CONDLTION | PERCENTAGE FREQUENCY IN HYPERTENSIVES | PERCENTAGE <br> FREQUENCY <br> IN NORMO- <br> TENSIVES |
| :---: | :---: | :---: |
| Diabetes Mellitus | 18.2 | 2.0 |
| Alcohol ingestion | 13.6 | 14.0 |
| Cigarette smoking | 18.2 | 14.0 |
| Urinary tract infection | 18.2 | 0 |
| Vulvular heart disease | 9.2 | 26.0 |
| Bronchial asthma | 13.6 | 2.0. |
| Signs of atherosclerosis | 45.4 | 22.0 |
| Previous stroke | 4.5 | 10.0 |
| GIT Bleeding | 4.5 | 2.0 |
| Atrial fibrillation | 0 | 12.0 |
| Chronic chest pain with hemoptysis | 0 | 6.0 |
| Pneumonia | 4.5 | 4.0 |
| Trauma | 4.5 | 2.0 |
| Congestive cardiac failure | 4.5 | 2.0 |
| Epilepsy | 4.5 | 0 |
| Intra-uterine contraceptive device | 0 | 4.0 |
| Dementia with depression | 0 | 2.0 |
| Deep vein thrombosis | 0 | 2.0 |
| Abdominal malignancy | 0 | 2.0 |
| Myocardial infarction | 0 | 2.0 |
| Ulcerative colitis | 0 | 2.0 |
| Gangrene (ischemic) foot | 0 | 2.0 |
| Migraine | 0 | 2.0 |
| Following child birth | 0 | 2.0 |
| Connective tissue disease | 0 | 2.0 |

TABLE V: BIOCHEMICAL PARAMETERS IN STROKE PATIENTS.


TABLE VI: FEATURES OF THE YOUNG NORMOTENSIVE STROKE PATIENTS

| CLINICAL FINDINGS | LAB FINDINGS | OCCUPATION |
| :---: | :---: | :---: |
| Mitral valve disease 7 | ESR---------8 | Farmer----6.6 |
| UTI---------------- 3 | Anaemia----4 | Student----6 |
| Atrial fibrillation--3 | High Hb--- 3 | Housewife - 3 |
| Heart disease -------2 | VDRL+VE ---- 1 | Secretary - - |
| Pneumonia-------------1 | $\begin{aligned} & \text { A-V malfor- } \\ & \text { mation }-----1 \end{aligned}$ | Waiter $-==-1$ |
| Connective tissue <br> disease---------------I | MaIaria -----1 | Maid $---=-=-1$ |
| Mitral valve prolapse---------------1 | SOL ---------1 | Guard-=-=--1 |
| Post-partum ----------I |  | Unemployed-2 |
| Septicemia---------1 |  | Not known==-1 |
| Smoking--------------1 |  |  |
| Smoking-------------1 |  |  |
| Sickle cell aisease----. ----------1 |  |  |
|  |  |  |
| The "Pill"-----------1 |  |  |
| DVT------------------1 |  |  |
| Alcohol consumption--1 |  |  |

Footnote: (1) 22 patients M - 10, F - 12
(2) Urban dwellers - 6, rural dwellers - 16
(3) Numbers refer to number of patients with each variable.


| KEY |  |
| :---: | :---: |
|  | $\mathrm{Hb} \mathrm{g} / \mathrm{dl}$ |
| $\begin{aligned} & x \times x \\ & x^{x} \times \\ & \hline \end{aligned}$ | WBC $\times 10^{9} / 1$ |
| [00 0 | ESR mm/hr. |

FIGURE II: SHOWING PATTERN OF Hb , WBC AND ESR


| KEY |  |
| :---: | :---: |
|  | Hb. g/dl |
| $\begin{aligned} & x \times x \\ & x \times x \end{aligned}$ | WBC $\times 10^{9} / 1$ |
| $\|$0 0  <br> 0 0  <br> 0 0  <br> 0 0 0 | ESR mm/hr. |

1. Singer, C., Underwood, E;A::

Short history of medicine (2nd edition). Oxford at the Clarendon Press pp. 1~3; 239-273; 1962.
2. Brothwell, D., Sandison, A.T, (Editors), Diseases in Antiquity = A survey of the Diseases; Inquiries, Injuries and Surgery of Early populations: Charies $\bar{C}$. Thomas, Springfield, Iifineis, U. S:A:, $2 \overline{3} 4=235$, 477-479, 1979,
3. Major R, H:

A short history of medicine: Charies C. Thomas, Springfield, Iilinois, $\mathrm{U}_{\mathrm{S}} \mathrm{S} . \mathrm{A}: \quad 220-556,888=$ 890: 1954.
4. Fisher, C.M., Ccciusion of the interrai carotid artery, Arch. Neurol. Psychiat., 65; 346, 1951.
5. Field, WS: Bruetman, M.E., Weibel, J., Collateral circulation of the brain. The Williams and Wilkins Co; Baltimore,220, 1965.
6. Isselbacher K.J., Adams, R.D. Braurwald, E., Petersdorf,•R,G., Wilson, J.D. (Editors):

Harrison's Principles of Internal Medicine (10th editon). McGraw-Hi11 Kogakısha Ltd., Tokyo, 1911-1942, 1983.
7. Aioraham, J.:

An understanding of the pathophysiology and management of cerebrovascular disease in man based on experimental data.

Neurology India 33, 1-24, 1985.
8 Bahemuka, M.:
Cerebrovascular accidents in 207 Kenyans:
General peculiarities and prognosis of stroke in an urban medical centre.
E.Afr.Med. J. 62, 315-321, 1985.
O. Billinghurst, J.R.:

The pattern of adult neurological admissions to Mulago Hospital, Kampala (June 1966 to May 1968). E.f̊fr. Med. J. 47: 633-653, 1970. .
10. Shaper, A.G., Shaper, L.:

Analysis of medical admissions to Mulago Hospital, 1957.
E. Afr. Med. J. 35: 647-678, 1958.
11. Dada, T.O., Johnson, F.A., Araba, A,B.,

Adegbite, S.A.:
Cerebrovascular accidents in Nigerians - A review of 205 cases.
W. Afr. Med. J. 18:95-108, 1969.
12. Osuntokun, B.O., Odeku, E.L., Adeloye, R.B.A.:

Cerebrovascular accident in Nigerians: A
study of 348 patients.
W. Afr, Med. J. 18: 160-173, 1969.
13. Bahemuka, M.:

Cerebrovascular accidents (strokes) in young normotensive Africans: A preliminary report of a prospective survey.
E.Afr. Med. J. 56: 661-664, 1979.
14. Hilton-Jones, D., Warlow, C.P.:

The causes of strokes in the young. J. Neuro. 232: 137-143; 1985:
15. Ledermann, J.A., Murphy P.J., Hamilton, M, ; Hoffbrand, B.I.:

A Survey of non-embolic stroke in adults under 50 years of age:
J.R. Coll. Physicians Lond, 19: 163-165; 1985:
16. Taylor, J.R;

Alcohol and strokes $=$ letter to the edtur: N.Eng1. J. Med. 306: 1111, 1977:
17. Wolf, P.A.:

Cigarettes, Alcohol and stroke (editoriai):
N. Eng1. J. Med. 315: $1087=1088$, 1986 :
18. Gill, J.S., Zezulka, A.V., Shipley, M:J:; Gill, S.K., Beevers, D.G.:

Stroke and alcohol consumption.
N.Eng1. J. Med. 315: 1041-45, 1986.
19. Goldbourt, U., Holtzman, E, Neufeld, H.N.:

Total and high density lipoprotein cholesterol In the serum and risk of mortality: evidence of a threshold effect.

Brit. Med. J. 乞90: 1239-42, 1985.
20. Schneider, R., Korber, N., Zeumer, H., Kiesewetter, H., Ringelstein, E,B., Brockmann, M.: The haemorheological features of lacunar strokes. J. Neurol. 232: 357-362, 1985.
21. Pauranik, A., Maheshwari, M.C., Dinda, A., Chopra, P.: Non bacterial thrombotic endocarditis presenting as fatal cerebral haemorrhagic infarction in a twelve year uld healthy boy - case report and review of literature. Neurology India 33: 229-235, 1985.
22. Ashok, P.K., KaJ.ra, G.S., Mishra, N.K., Maheshwari, M.C.:

Moyamoya disease: Presentation as a recurrent thrombotic stioke in an adult.

Neurology India 32: 49-52, 1984.
23. Henrish, J.B., Sandercock, P.A.G., Warlow, C.P., Jones, L.N.:

Stroke and migraine in the Oxfordshire Community Stroke Project. J.Neurol. 233: 257-262, 1986.
24. Bonita, R., Scragg, R., Stewart, A., Jackson, R., Beaglehole, R.:

Cigarette smoking and risk of premature stroke in men and women.

Brit. Med. J. 293: 6-8, 1986.
25. Carey, R.M., Reid, R.A., Ayers, C.R., Lynch, S.S., McLain III, W.L., Vaughan Jr., E.D.:

The Charlottesville Blood pressure survery;
Value of repeated blood pressure measurements.
JAMA 236: 847-851, 1976.
26. Abbott, R.D; Yin, Y; Reed, D.M; Yano, K:; Risk of stroke in male cigarette smokers: N. Engl. J. Med. 315; 717-20, 1986:
27. Matenga, J., Kitai, Levy, £.: Strokes among black people in Harare, Zimbabwe: results of computed tomography and associated risk factors.

Brit. Med. J. 292: 1649-51, 1986:
28. Bahemuka, M., Malignant hypertension: A review of the neurological features in 34 consecutive patients:
E. Afr. Med. J. 62; 560-565, 1985 :
29. Peart, S; Sir; (Chairman), Medical Research Council Working Party. NRC trial of treatment of mild hypertension: principal results. Brit. Med. J. 291; 97-104, 1985.
30. Ngumuta, A.N., Personal Communication, 1987.
31. Orinda, D.A.U; Personal Communication, 1986.
32. Shaper, A.G; Kyobe, J.; Stansfield D., Haematologi.cal observations in an East African student population. E. Afr. Med. J. 39; 1-4, 1962.
33. Kasili, E.G; Cardwell, C.L.; Taylor, R.J., Leucocyte counts on Blood Donors in Nairobi. E.Afr. Med. J. 46́; 677-679, 1969.
34. Alien, E.F; Cruickshank, A; Whittaker, R.L., Leucocyte counts in Africans in Nairobi. E, Afr. Med. J. 36; 274, 1959.
35. Schettler, G; Nussel, E., Test serum cholesterol using the CHOD-PAP enzymatic colourimetric
method. Arbeitzmed. Sozialmed. Praventivmed. 10; 25. 1975.
36. Okelo, G.B.A.; Kyobe, J; Kanja, C., Fasting glucose and lipid profiles in diabetic and nor-diabetic subjects in Kenyatta National Hospitai, Nairobi, Kenya. Medicom 2; 15-18, 1980.
37. Ojwang, P.J., Ogada, T; Maina, F.W; Sekadde-Kigondu, C.E; Mati, JKG., Reference values for serum lipid and lipoprotein cholesterol in adults and cord blood of Keny'n Africans. E. Afr. Med. J. 61; 367-371, 1984.
38. Lore, W; Kyobe, J., Fibrinolytic activity (euglobulinalysis time) and lipid profile in adult hypertensive natients at the Kenyatta National Hospital, Nairobi (Kenya). Trop. Card. 9; 87-91, 1983.

