"SPONTANEOUS SUBARACHNOID HAEMORRHAGE AT KENYATTA NATIONAL HOSPITAL (K.N.H.)"

(A Prospective and retrospective study of patients seen in K.N.H. from July 1983 to July 1984 and July 1974 to June 1983 respectively).

By

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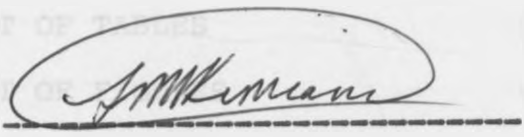
A Thesis submitted in part fulfilment for the degree of Master of Medicine (Medicine) in the University of Nairobi

1985

DECLARATION

'This Thesis is my original work and has not been presented for a degree in any other University'

Signature 

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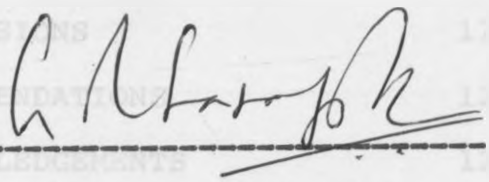
Signature 

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SUMMARY

A total of 109 patients with spontaneous subarachnoid haemorrhage were included in this study. 80 of these patients belonged to retrospective group covering a ten year period from July 1974 to June 1983 and 29 patients formed a prospective group covering a thirteen months period from July 1983 to July 1984. The two groups of patients were analysed separately and the results of analysis compared.

Males were more than females and the male to female ratio was 2.5:1 and 2.6:1 in retrospective and prospective group respectively. Age range was 15 to 90 years in retrospective group and 20 to 90 years in prospective group. Males were slightly older than females. The mean age was 47.52± 18.72 and 36.50± 15.02 in males and females respectively in the prospective group. The peak incidence was in 20 - 50 age group which comprised over 60 percent of the patients. The younger the patient the better was the outcome with best recovery below 40 years. There was no difference in outcome between the sexes.

In prospective study, alcohol was observed to be the leading factor of association in 35 percent of patients. Hypertension was found in 24 percent and smoking found in 17 percent. Both alcohol and smoking were associated with the largest number of intracranial

vascular abnormalities (4 out of 9 aneurysms and one arteriovenous malformation).

The largest group of patients had normal angiographic findings and formed 44 percent in prospective group, 70 percent in retrospective group and 61 percent when the two groups were combined. Aneurysms were the second largest group of subarachnoid haemorrhage. They formed 33 percent in prospective group, 20 percent in retrospective group and 24 percent when the two groups were combined. Intracerebral haemorrhage was third. It formed 19 percent in prospective group, 5 percent in retrospective and 10 percent when the two groups were combined. Arteriovenous malformation was last and formed 4 percent in prospective group, 3 percent in retrospective and 4 percent when the two were combined. Normal angiographic findings had best prognosis followed by the group with aneurysms. Intracerebral haemorrhage had grave prognosis with 100 percent mortality in prospective group.

The commonest site of aneurysm in each separate group and when combined was posterior communicating artery which had 45 percent of the aneurysms. Anterior cerebral and anterior communicating artery was second with 25 percent, carotid artery had 15 percent, middle cerebral artery 10 percent and basilar artery 5 percent.

Electrocardiographic changes were found in 73 percent of all the patients investigated, in 68 percent of the control group and in 78 percent of the propranolol group.

Patients on propranolol showed a trend of short recovery period compared with control group (8 and 13 days respectively). There was also a trend of better outcome for the propranolol group than the control group.

The serum cholesterol levels, urinary 17-ketosteroid, 17-Hydroxysteroid, vanillyl mandelic acid and catecholamines were normal.

INTRODUCTION AND LITERATURE REVIEW

DEFINITION

Symonds in 1924 used the term spontaneous subarachnoid haemorrhage to describe "those instances of subarachnoid haemorrhage not due to trauma", the only implication of the term being that the haemorrhage was not traumatic. He recognised that the principal cause of non-traumatic subarachnoid haemorrhage was ruptured cerebral aneurysms, but in 41 of the 124 cases he reviewed, no satisfactory cause could be found (1). An arterial aneurysm was defined by Allock in 1974 as an "abnormal localized dilatation" of the vessel (2).

ANATOMY AND PATHOLOGY OF CEREBRAL VESSELS

The cerebral vessels belong to the medium sized or muscular group of arteries. Muscular defect of the media at the vessel bifurcation is the principal cause of cerebral arterial aneurysms. On the other hand, degeneration of the internal elastic lamina due to continual overstretching under ordinary conditions of circulation is said to be the final stage in the production of the sac (3). There are six types of aneurysms whose difference lie in anatomical location and cause. They include congenital or berry aneurysms, atherosclerotic, dissecting, infective, traumatic and neoplastic aneurysms (2,4,5).

PATHOPHYSIOLOGY

Growth of Aneurysms

Aneurysms have been shown to enlarge with time using serial angiography. This is as a result of the water hammer effect of the pulse which causes ballooning of the normal gap in internal elastic lamina found at an arterial bifurcation. Proliferation of adventitia thickens the neck of the aneurysm while the fundus, poorly bolstered by adventitia, expands in response to transmural pulsatile forces (5).

ANEURYSM RUPTURE

The pressure of the pulse finally pierces a tiny hole in the fundus of the aneurysm. Bleeding is

probably very brief in most instances, leading to headache, nausea, vomiting and less frequently, loss of consciousness. In some cases, massive haemorrhage quickly fills the basal cisterns and ventricular system, leading to coma and death (5). The rupture is followed immediately by intense spasms of the vessels in continuity with the aneurysm. Although the spasm is crucial in stopping haemorrhage and saving life, it is harmful in rendering the brain ischaemic in the territory of the supply by the particular vessel in spasm. The neurological signs depends on extent and severity of the spasm (6).

The vasospasm is due to mechanical stimulation of the arteries by clot and contact with platelet products (serotonin, prostaglandins and thromboxane) as well as vasoconstrictors (oxyhaemoglobin, methaemoglobin and bilirubin) released by lysed erythrocytes (6,7).

INCREASED INTRACRANIAL PRESSURE

This is a common sequel of acute subarachnoid haemorrhage and may be associated with impaired consciousness. The raised intracranial pressure may also act to retard recurrent subarachnoud haemorrhage (5).

HYPOTHALAMIC IRRITATION

Hypothalamic irritation by subarachnoid bleeding may lead to a variety of systemic abnormalities. Generalized discharge of catecholamine which inturn constrict cerebral arteries is observed in some patients. Electrolyte imbalance due to inappropriate secretion of antidiuretic hormone and abnormal levels of hydroxycorticosteroids are other associated systemic abnormalities (5).

CARDIAC ABNORMALITIES

The electrocardiographic abnormalities associated with subarachnoid haemorrhage were first documented by Byer, Ashman and Toth in 1947. They have been reported in 50 to 80 percent of patients in various studies. The pathological changes in the heart were however first described by Koskelo in 1964. Myocardial injury proven on histology has been described as early as 17 hours after subarachnoid haemorrhage (9). Various abnormalities in the electrocardiogram (ECG) have been reported. These are: tall and peaked P wave (exceeding 2.5mm in Lead II), prolonged or shortened P-R interval, pathological Q wave, left ventricular hypertrophy (SV_1 + RV5 exceeding 35 mm), elevated or depressed ST segment, peaked, flattened or inverted T wave, prolonged or shortened Q-T interval, tall (exceeding 1mm) or inverted u wave and arrhythmias (sinus tachycardia and bradycardia,

extrasystole) (8-11). The abnormal ECG changes revert to normal within two to six weeks after initial cerebrovascular insult (8).

Myocardial injury as well as electrocardiographic abnormalities have been attributed to the electrolyte disturbances, in particular low serum potassium, increased catecholamine production via the autonomic nervous system, irritation of area 13 on the orbital surfaces of the frontal lobes where vagus nerve has cortical presentation, and the metabolic effect of norepinephrine released locally into the extracellular space (8-10).

RECURRENT SUBARACHNOID HAEMORRHAGE

The risk of recurrent haemorrhage reaches its peak 10-14 days after initial rupture. The daily incidence rises from an initially low level to a sharp peak and then declines steadily. The explanation which has been given for this phenomenon is that the initial rise is caused by progressive lysis of the fibrin plug which has sealed the hole in the aneurysm and the subsequent decline by the maturation of the definitive fibroblastic repair. A mortality of up to 90 percent due to rebleeding has been reported. In patients with multiple aneurysms followed for several years, the risk of rupture of a second aneurysm of up to 80 percent has been reported (12-15).

Understanding of mechanism of rebleeding makes the rationale of use of various antifibrinolytic drugs such as tranexamic acid and e-amino caproic acid self explanatory.

AETIOLOGY

Several aetiologies have been suggested for spontaneous subarachnoid haemorrhage. Intracranial aneurysms are the leading cause and account for 80 percent of spontaneous subarachnoid haemorrhage (6). Intracranial arteriovenous malformation on the other hand accounts for 10 percent (13). Others which have been mentioned include; hypertension, physical strain, oral contraceptives, cigarette smoking, alcohol intoxication, tumours, infections, disorders of coagulation, angiopathies, non-alcoholic intoxications, vitamin deficiency especially vitamins C and K and pregnancy (16-25).

Despite refinements in investigations, including angiography and computerized transverse axial tomography (CT) of the brain, the cause still cannot be determined in 20 to 30 percent of patients. The explanation given for this group of subarachnoid haemorrhage is the rupture of pial vessel or thrombosed "berry aneurysm", atherosclerotic aneurysm, or cryptic arteriovenous malformation, thrombosed or otherwise obliterated by haemorrhage (1,16,24).

CLINICAL GRADING OF PATIENTS WITH SUBARACHNOID HAEMORRHAGE:

Grading or classifying patients with subarachnoid haemorrhage on the basis of clinical status is helpful in their management. The common ones are Hunt and Hess grading system, Nishioka grading system(26) and cooperative aneurysm study grading system of neurological conditions (27).

HUNT AND HESS GRADING SYSTEM

GRADE	DESCRIPTION
1	Asymptomatic, or minimal headache and slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, no neurological deficit (except cranial nerve palsy).
3	Drowsiness, confusion / or mild focal deficit
4	Stupor, moderate to severe hemiparesis, possible early decerebrate rigidity and vegetative disturbances.
5	Deep coma, decerebrate rigidity, moribund.

In Hunt and Hess grading system, serious systemic disease such as hypertension, diabetes mellitus, severe arteriosclerosis, chronic pulmonary disease, and vasospasm on angiography result in placement of patients in next less favourable category.

NISHIOKA GRADING SYSTEM

GRADE	DESCRIPTION
1	Symptom free
2	Minimally ill, complaining of headache but alert and responsive
3	Moderately ill (a) Lethargic with headache and neck stiffness but without neurological deficit (b) Alert, recovered from effects of subarachnoid haemorrhage, but having a hemispheric neurological deficit
4	Seriously ill (a) Severely obtunded without major neurological deficit (b) Lethargic or poorly responsive with hemispheric deficit (hemiparesis, dysphasia, confusion)
5	Moribund: decerebrate or unresponsive to stimuli.

COOPERATIVE ANEURYSM STUDY GRADING SYSTEM OF NEUROLOGICAL CONDITIONS

GRADE	DESCRIPTION
1	Symptom free
II	Minimally ill
III	Moderately ill
IV	Seriously ill
V	Moribund

INVESTIGATIONS

Investigations can be broadly divided into those helping in confirming the diagnosis and those pointing to complications resulting from subarachnoid haemorrhage.

INVESTIGATIONS TO CONFIRM DIAGNOSIS.

LUMBAR PUNCTURE

Lumbar puncture still plays an important role in the evaluation of subarachnoid haemorrhage particularly in cases of subarachnoid haemorrhage without neurological signs. Lumbar subarachnoid spinal fluid sampling can be diagnostic and lead to appropriate management. This approach is particularly appropriate in hospitals where computed tomography (CT) is not readily available (5,7).

Computed tomography.

Computed tomography (CT) has revolutionized the investigation of subarachnoid haemorrhage. This scanning technique often makes diagnosis of subarachnoid bleeding without the risks involved in Lumbar puncture. The advantage of CT scanning is particularly appropriate in patients who may suffer from increased intracranial pressure, that is, patients in grades 3 to 5 with significant neurological deficit. In some patients with multiple cerebral aneurysm or cerebral aneurysm and cerebral arteriovenous malformation, CT scan may specify which of the various lesions has actually bled. Computed tomography is also useful for the diagnosis of complications of subarachnoid haemorrhage, including intracerebral or intraventricular haematoma, hydrocephalus, and cerebral infarction.

To permit the rapid diagnosis of these adverse developments, CT scanning should be performed on admission for all patients with subarachnoid haemorrhage and promptly after any deterioration (5). Computed tomography gives highest positive results when performed on the first two days after the bleed. The yield declines from 100 percent on the first two days to about 0 percent after 3 weeks (28).

SKULL X-ray

Plain films of the skull seldom provide helpful information, however, the examination should never be omitted. Occasionally aneurysms are calcified, but the vast majority bleed before this healing process takes place. On the other hand, the much rarer atherosclerotic aneurysms often produce changes in plain x-ray of the skull (4). Calcification which occurs in approximately one percent of cases is often very characteristic, with a fine ring or egg shell appearance. It occurs in larger aneurysms and indicates thrombosis, but does not imply that the aneurysm is no longer capable of rupture.

Angiography

Angiography remains the procedure of choice for the precise characterization of bleeding intracranial lesions. In most cases, angiography may be deferred

until just before surgery, as vasospasm can be evaluated in close temporal relation to operation. This strategy of timing for angiography avoids immediate study on admission. A search for vasospasm is the other major indication for angiography; definite evidence of vasospasm can only be gained when narrowing of cerebral vessels is seen on angiographic investigation (5).

It is now generally accepted that angiography for suspected aneurysm is carried out via retrograde catheterization of the femoral artery (2). Comprehensive angiography includes injection of all major vessels from which aneurysms arise. In practice, this is usually both common or internal carotid arteries and one vertebral artery (usually left); the second vertebral artery generally fills in a retrograde fashion beyond the origin of the posterior inferior cerebellar artery (2). Where transfemoral, carotid and vertebral angiography is not possible, direct bilateral carotid angiography is carried out first and vertebral angiography should be performed after an interval of about 48 hours (4). Three angiographic projections - lateral, antero-posterior and oblique are considered the minimum for demonstration or exclusion of most aneurysms. The lateral projection is the most informative as regards disturbances of the cerebral circulation (2).

An aneurysm appears at angiography as an additional blob of intravascular contrast medium, sitting on or connected by a neck to an artery, usually near the birfurcation. The diameter varies from about 2 to 20 mm in the majority of cases (2).

Radionuclide studies

Radionuclide dynamic flow studies may indicate the presence of local diminution of flow which suggest deferral of surgical intervention (5). Such studies have shown that cerebral blood flow falls with advancing age. Similar studies have also shown that cerebral blood flow falls progressively below normal during the first week after subarachnoid haemorrhage and remains abnormally low for at least another two weeks. For each separate day during this period the oldest patients tend to have the lowest cerebral blood flow (29).

INVESTIGATIONS POINTING TO COMPLICATIONS.

Electrocardiogram and chest x-rays. These should be done because myocardial infarction and pulmonary oedema are known complications of subarachnoid haemorrhage and may kill the patient if unnoticed and therefore untreated (5,30,31).

Serum electrolytes and blood urea nitrogen.

These are helpful in pointing out the syndrome of inappropriate secretion of antidiuretic hormone. This is known to cause disturbed consciousness (5).

24 hour urinary catecholamines (adrenaline, nor-adrenaline, 3-methoxy-4-hydroxymandelic acid and metadrenalline)and steroid (17-ketosteroid and 17-hydroxysteroid).

These indicate hypothalamic irritation, sometimes very high levels of catecholamines similar to those observed in pheochromocytoma are produced. These may induce heart failure, as in the cardiomyopathy associated with pheochromocytoma. Steroids are also increased together with catecholamine through hypothalamic adrenal axis (5,30,31).

Electroencephalogram. This may be useful in cases of seizures.

MANAGEMENT

The management of patients with subarachnoid haemorrhage can be divided into supportive, medical and surgical.

SUPPORTIVE MANAGEMENT

Supportive management is very important in the acute phase and very sick patients, possibly from grade 3 to 5. It is similar to the management of any unconscious patient or patient with stroke. This involves supporting respiration, treating shock when present, bladder and bowel care, proper feeding and maintenance of water and electrolyte balance; physiotherapy, occupational therapy and rehabilitation at later stages.

MEDICAL MANAGEMENT This group involves bedrest and use of various drugs.

BEDREST

Bedrest has traditionally been used to avoid recurrent subarachnoid haemorrhage. "Aneurysm precautions" usually include a darkened quiet room, limited visitors, and avoidance of radio and television. Stool softeners are administered to avoid straining at stool, which can lead to recurrent subarachnoid bleeding. Sedatives such as diazepam are administered to avoid upset and consequent hypertension. Anticonvulsants, such as diphenylhydantoin are administered to prevent seizures which could be harmful (5).

ANTIHYPERTENSIVE AGENTS.

Antihypertensive therapy is used in many centres to prevent recurrent bleeding. Drugs used include hydrallazine, propranolol and nitroprusside among several others (5, 29).

ANTIFIBRINOLYTIC AGENTS

Antifibrinolytic agents have provided a major advance in the medical management of ruptured aneurysm. Since fibrinolysis in cerebrospinal fluid lyse the clot sealing the bleeding site in aneurysm, antifibrinolytic therapy is a logical approach to prevent the recurrent bleeding which commonly occurs in the first two weeks after the initial haemorrhage. Several studies indicate that the incidence of rebleeding is

substantially diminished in patients who receive the antifibrinolytic agent, epsilon aminocaproic acid (EACA). The recommended dose of EACA is 30 to 36g a day in six divided doses, administered intravenously until surgery or for 21 days if surgery is not performed. Some authors have recommended oral therapy. It acts by competitive inhibition of the activator that converts plasminogen into the proteolytic enzyme plasmin and in so doing stabilizes the clot making it mature (5,32,33).

More recently, good results have been obtained with tranexamic acid. It's mechanism of action is similar to that of EACA. The recommended dose is 6g per day in four divided doses. It can be given intravenously for the first 7 days and thereafter orally (33).

The commonest side effects of EACA and tranexamic acid are diarrhoea and vomiting. The theoretical side effects of thromboembolism and communicating hydrocephalus from obstruction of the subarachnoid space are not commonly encountered (5,32-35). The antifibrinolytic drugs should be given for at least 4 weeks to cover the period during which primary or secondary fibrinolysis continues and the risk of break through bleeding is highest.

CEREBROVASCULAR VASOSPASM

Cerebrovascular vasospasm remains a difficult problem in management. A large number of experimental and clinical therapies have been used in the treatment of vasospasm.

Isoproterenol and Lidocaine hydrochloride

It is now clear that cerebral blood vessels contain both alpha and beta adrenergic endings (36). Isoproterenol is almost purely a beta adrenergic drug and therefore its action is similar in many respects to that of alpha blockers. It dilates vascular smooth muscle, in so doing, it tends to slightly decrease the peripheral blood pressure, which is in part compensated for by increased cardiac output. Lidocaine hydrochloride is administered concurrently to counteract cardiac irritability (5,36).

Propranolol

Propranolol has been shown to improve clinical state and to reduce complications which follow subarachnoid haemorrhage. The advocated dose is 80 mg 8 hourly for 3 weeks. Propranolol is a highly potent, non-selective beta-adrenergic blocking agent with no intrinsic sympathomimetic activity. The suggested mechanisms of action include; the blocking of increased catecholamine effects in general and reduction of

plasma renin by blocking B_1 receptors. Catecholamines and renin levels are raised in some patients with subarachnoid haemorrhage and the former has been associated with cerebral vasospasm. In addition, propranolol reduces cerebral oxygen requirements in patients with stroke or cerebral ischaemia by prevention of uncoupling of oxidative phosphorylation, normally caused by catecholamines. It also shifts the oxygen dissociation curve to the right, making more oxygen available to the tissues. The cardioprotective effect of propranolol is another additional advantage (30,31,37,38).

AMINOPHYLLINE

Aminophylline combined with isoproterenol has been reported helpful. Aminophylline which is a methylated xanthine acts by controlling the levels of the cyclic nucleotide phosphodiesterase which breaks cyclic AMP to 5'-AMP. Twelve patients were treated with infusion of these drugs, and 9 patients improved in one clinical series (5,36,39).

RESERPINE AND KANAMYCIN

Reserpine and kanamycin reduce blood serotonin, an agent known to cause vasoconstriction. Prophylactic administration of these agents has been reported to diminish the incidence of delayed ischaemic deficit particularly after operation (5).

CALCIUM ANTAGONISTS

Recently, nifedipine and nimodipine have been shown to be useful in management of cerebral ischaemia in general and cerebral vasospasm in particular (40).

SURGICAL MANAGEMENT

Microsurgical obliteration of aneurysms offers a safe and effective means of preventing dangerous recurrence of subarachnoid bleeding (5). In experienced hands, the complication rate for such intracranial surgery is now less than 5 percent for good risk patients. Given a high rate of recurrent bleed (as high as 30 percent in the first year), intracranial direct obliteration of aneurysm may be confidently recommended for most patients. A wide variety of aneurysmal clips are available for obliteration of intracranial aneurysms. When clipping of an aneurysm is impossible, certain rapidly polymerizing tissue adhesives may invest the aneurysm with an impenetrable plastic coating (5).

In days prior to microsurgery, common carotid and later internal carotid artery ligation was a popular method for giant or inaccessible aneurysms (5).

Innovative alternative approaches to aneurysm surgery have been developed such as stereotactic

obliteration of intracranial aneurysms and open introduction of fine thrombogenic wire into certain giant aneurysmal lesions. Stereotactic introduction of ferromagnetic particles into aneurysms, with local magnetic attraction to hold the thrombogenic particles within the aneurysmal lumen has been used (5,41).

In case of multiple aneurysms, the unruptured aneurysm still has the risk of rupturing. The mortality rate after rebleeding during an average follow up of 16 years has been found to be 11.5 percent. Mortality rate when operating on a ruptured aneurysm has been found to be 4.2 percent and therefore operation for unruptured aneurysms, have a slight edge over conservative treatment (42) In view of this information, many surgeons advocate surgical treatment of all aneurysms in low risk patients in two stages if necessary (42).

Patients with arteriovenous malformation of the brain are subject to disabling or fatal recurrent haemorrhage. The definitive treatment is total surgical excision or embolization. The patients considered unsuitable for conventional therapy, usually because of the location, size or operative risk of lesions are treated with stereotactic bragg-peak proton beam therapy. This is

intended to induce subendothelial deposition of collagen and hyalline substance which narrows the Lumen of small vessels and thickens the wall of the malformation during the first 12 to 24 months after the procedure (12,42,43). A pregnant woman with an aneurysm or arteriovenous malformation is at a special risk towards the end of her pregnancy. Craniotomy and obliteration or excision is sometimes recommended (6). The other alternative is caesarian section at about 34 weeks and sterilization as soon as the family is of acceptable size (12).

The timing of surgery for obliteration of aneurysms remains controversial. For grade 1 and 2 patients, operation after 1 to 2 weeks of medical therapy is generally advocated. For patients in grade 3 and 4, surgery is generally deferred until recovery or at approximately 3 to 4 weeks after bleeding. During this time maximum recovery can occur undisturbed by the trauma of surgery however minimal. For patients in grade 5, surgery is not recommended because of their moribund condition (5).

Some clinicians advocate early surgery to avoid the complications of recurrent bleeding and vasospasm. Saito et al (44) have given good results of aneurysmal direct surgery in the first 3 days after subarachnoid haemorrhage. They advocate that vasospasm has not set in the first 3 days.

Emergency surgery is sometimes indicated for the complications of subarachnoid haemorrhage such as intracerebral haematoma and hydrocephalus (5).

SURGICAL VERSUS MEDICAL (CONSERVATIVE) MANAGEMENT

Various authors have compared medical (conservative) with surgical management as regards the outcome. The mortality of 50 percent has been given in the medical group by many authors. Mortality in patients treated by surgery on the other hand ranges from 50 percent to as low as3.6 percent. Mckissock et al (13) compared the outcome of various aneurysmal locations in both methods of management and found consistent lowering of mortality rate in the surgically treated subgroups. He cited the anterior communicating or middle cerebral artery aneurysms which when the clinical state is very bad have a natural death rate of over 90 percent and said that if surgical measures can restore a few patients to reasonable health, then the operative mortality should be assessed against the natural death rate of 90 percent and so even a 50 percent operative mortality would be acceptable. Logue (6) in his comparison of surgical and medical management had similar conclusion as Mckissock.

Forster et al (45) compared surgical and medical management of patients with arteriovenous malformations, who were followed up for a mean period of over 15 years. The result was in favour of operated cases.

THE COURSE OF SUBARACHNOID HAEMORRHAGE

MEDICAL (CONSERVATIVE) GROUP

It is difficult to obtain a truly unselected series in this condition; for selection, natural selection, starts with the patient outside hospital - the most severe cases going straight to the mortuary and the mildest cases not coming to the hospital at all and continues at each step through hospital (6).

Before surgical treatment was available, 65 percent of patients with ruptured aneurysms died, 43 percent from the initial haemorrhage and 22 percent from recurrent bleeding during the following year. It is estimated that approximately 15 percent of patients with ruptured intracranial aneurysms die before reaching a hospital (46).

Adams et al (47) in their review of Literature quoted Parkarinen's study of patients managed conservatively, which showed that 35 percent of the survivors of the initial haemorrhage would be expected to die of a recurrence within a year, and 51 percent would be dead in five years, the remaining survivors dying at 3.3 percent a year there after. Recurrences are found occuring up to 21 years after the initial haemorrhage. Total disability occurs in 11 percent of survivors and partial disability in 19 percent.

SURGICAL GROUP

Adams et al (47) found that when surgery was carried out in the first week the mortality was 22 percent; when surgery was carried out in the second week the mortality fell to 5 percent, and when the interval between subarachnoid haemorrhage and surgery was greater than 2 weeks there were no deaths. The patients who were operated on in the second week had a higher incidence of poor results (23 percent) than those who underwent operations in the first week (7 percent).

Lack of information on this subject in our set up prompted me in undertaking this study.

AIMS AND OBJECTIVES

1. Carrying out a prospective study of adult patients admitted to Kenyatta National Hospital with subarachnoid haemorrhage.
2. Finding out incidence, age, sex, occupation and tribal distribution of patients with subarachnoid haemorrhage.
3. Finding out causes and associations if possible.
4. Evaluating the usefulness of propranolol therapy in management of these patients in the acute stage of the illness.
5. To compare the comparable information from prospective study with retrospective study.

MATERIAL AND METHODS

The prospective study covered thirteen months period from July 1983 to July 1984. The material included patients of 15 years and above of both sexes admitted to the medical wards of the Kenyatta National Hospital. All patients were seen by the author within 24 hours of admission.

A detailed history was taken from patients, relatives or friends by the author himself, and then a detailed physical examination was carried out as shown in the attached proforma. Diagnosis was confirmed by a careful lumbar puncture to avoid trauma, using a thin needle and removing minimum cerebrospinal fluid after obtaining uniformly blood stained or uniformly xanthochromic cerebrospinal fluid (CSF). To be sure that the bloody CSF was not due to trauma, a CSF sample was centrifuged in the ward laboratory and the xanthochromic supernatant viewed against a white background to a certain uniformity in at least three specimens. The lumbar puncture was done by the author in most of the patients and a few by the admitting doctor. Where the author was not satisfied with the lumbar puncture findings he repeated it himself. The cerebrospinal fluid obtained was cultured for pyogenic bacteria, fungal and acid fast bacili, sugar and protein were estimated and kahn and VDRL tests were carried out.

Soon after confirming diagnosis of subarachnoid haemorrhage, blood specimen was taken for complete haemogram, serum cholesteral, urea and electrolytes. Liver function tests, blood sugar (fasting and post-prandial), kahn test and coagulation screen were carried out. 24 hour specimen of urine was collected for 17-ketosteroid, 17-Hydroxysteroid, catecholamines and vanillyl mandelic acid (VMA); Urine was also tested for sugar and proteins.

Electrocardiogram was done as soon as possible as well as chest and skull x-rays. The patients were then booked for bilateral carotid and brachial angiography between 7th and 10th day after onset of subarachnoid haemorrhage. Intravenous urography was also done to rule associated renal cysts and other renal malformations.

Haemogram and coagulation screenwere done in the department of haematology laboratories while urea and electrolytes in the department of Chemical Pathology.

Serum cholesterol, urinary 17-ketosteroid, 17-hydroxysteroid, catecholamine and vanillyl mandelic acid were done in the department of Medicine.

Serum cholesteral was done by Boehringer Kit method (Watson D). The estimation of the cholesterol level in the serum was done in duplicate. The normal range by this method is 3.1 to 6.47 mmols/L (140-250 mg percent).

Both 17-ketosteroid and 17-Hydroxysteroid in 24 hour urine specimen were estimated using Zimmermann reaction as outlined by Varley (48). The estimation of each was done in duplicate. The normal range by this method is, 17-ketosteroid 21 to 70 umol in 24 hours and 17-hydroxysteroid 21 to 87 umol in 24 hours.

The catecholamines in 24 hour urine specimen were estimated using semi-quantitative method of catecholamines as outlined by Varley (49). The estimation of each was done in duplicate. The normal range by this method is 0.05 to 0.9 umol in 24 hours (10 to 180 ug in 24 hours).

Vanillyl mandelic acid (VMA) estimation was done using VMA screening method, S.E. GITLOW (50). This estimation was also carried out in duplicate. The normal range is upto 36 umol in 24 hours (upto 7mg in 24 hours).

Electrocardiography was done using cardiostat 701 made by Siemens company of West Germany. This was done at least 72 hours of admission and repeated after 10 days. In both groups (Beta-blockers group and control) where this was possible, 12 leads electrocardiography was done: The interpretation of the electrocardiography was done as outlined in principles of clinical electrocardiography by Goldman (51).

Angiography to locate the possible sites of bleed was carried out between 7th and 10th day. Bilateral carotid followed by one brachial angiography were done on different days with a minimum of 48 hours period apart to avoid excess dose of radiopaque medium.

The patients were investigated under general anaesthesia. Common carotid was injected with a short bevelled Lindigren needle Number 16 or 18 depending on patients size. The contrast used was urograffin 60 percent. 10mls of the radioopaque medium was used for each projection anteroposterior, lateral and oblique when required to demonstrate aneurysmal neck. For brachial angiogram 25 mls of the medium was used for each projection, anteroposterior lateral and oblique when required. Two films were used for anteroposterior projection and three films for lateral projection. Physiological saline was perfused through the connection tube and needle between injections to prevent clotting in the system. A standard schonader skull table with serial hand changers was used. Where aneurysms or malformations were found, they were assumed to be the source of bleed.

The brachial angiography was done to fill the vertebral artery in retrograde and therefore demonstrate the posterior part of circle of Willis. The vertebral artery on the same side of the brachial artery inject-

ed, the basilar artery and its branches were demonstrated. The upper part of vertebral artery on the opposite side was also demonstrated as it filled in retrograde from opposite vertebral artery. The area where majority of aneurysms on posterior part of circle of Willi's was demonstrated. It is the policy of the department of radiology in this hospital to do only one side of brachial angiography when demonstrating vertebral circulation. The side does not matter unless specified. In this series either side right or left brachial angiography was done depending on radiologists choice. Bilateral carotid angiography and brachial angiography were carried out because the table for the popular transfemoral four vessel angiography was not operating during the period of this study.

Soon after collecting 24 hour urine specimen, the patients were randomly allocated into two groups. One group received bedrest, diazepam 5mg twice a day, dexamethasone 4mg 8 hourly and laxatives to soften the stool. The other group received the above treatment plus propranolol 80mg 8 hourly orally for three weeks. Those who were unconscious or unable to accept oral medication were given the same dose by nasogastric tube. Propranolol was avoided in patients with branchial asthma, heart failure, diabetes or any other condition where propranolol was thought to have deleterious effects. Those who presented with high blood

pressure were given antihypertensives other than propranolol unless they fell in the group which was receiving propranolol. Patients were taken to be hypertensive if on admission were already known hypertensive and on treatment or were found to have a persistent diastolic pressure above 100mmHg and systolic pressure above 160mmHg for at least 3 readings after admission with or without hypertensive changes such as cardiomegally or retinopathy among others.

The two groups of patients were examined daily by the author and pulse and blood pressure taken. Presence and severity of headache, neck stiffness, Kernig's sign were noted and the level of consciousness indicated. The end point of recovery was taken as the time when there was no headache, no meningeal irritation signs (neck stiffness and Kernig's sign) and patient had regained full consciousness. That is when fully orientated in time, place and person when addressed by the author as well as other members of staff. The total number of days taken for this to be achieved by each patient in both groups was recorded. Those who by the end of three weeks of drugs had not regained full consciousness, and those who died before 3 weeks were excluded from each group when calculating the recovery period. The recovery time in the two

groups was compared at the end of the study. The patients who had identifiable aneurysms or arteriovenous malformations were referred to neurosurgeons for the opinion and decision whether to operate or not. At the time of discharge the outcome was assessed into complete recovery (where there was no neurological deficit), partial recovery for those with some neurological deficit but independent (This group included those with hemiparesis, isolated cranial nerve paresis and slight confusion) and disabled for those who had severe neurological deficit and could not walk or use their hands and were to be dependent on other people even on feeding. Death was also included in the outcome.

RETROSPECTIVE STUDY

The retrospective study was over a period of 10 years from July 1974 to June 1983. The files of patients diagnosed and confirmed by lumbar puncture as having subarachnoid haemorrhage at Kenyatta National Hospital over this period were studied. As much as possible information concerning the patients was extracted and put in the proforma form attached. The information about age, sex, tribe, angiographic findings and outcome were compared with those of prospective study.

RESULTS

PROSPECTIVE STUDY

INCIDENCE: A total of 29 patients were seen during the study period. There were 21 males and 8 females giving a male to female ratio of 2.6:1. Of the 29 patients, 9 patients (31 percent) had aneurysms, 5 (17 percent) had intracerebral bleed, 1 (3.45 percent) had arteriovenous malformation and 12 patients (41.4 percent) had normal angiographic or postmortem findings. Two patients were neither investigated nor postmortem done.

During the same period of the study 4393 patients were admitted and treated in adult medical wards of Kenyatta National Hospital. This gave the incidence of spontaneous subarachnoid haemorrhage at Kenyatta National Hospital Medical wards as 6.6 per 1000 patients. The incidence of aneurysmal subarachnoid haemorrhage was 2 per 1000, intracerebral bleed was 1.14 per 1000, arteriovenous malformation was 0.23 per 1000 and the incidence of normal angiographic findings was 2.7 per 1000. A total of 106 patients with cerebrovascular diseases were admitted over the study period. Subarachnoid haemorrhage contributed 27.4% of this: Aneurysms contributed 8.5%, intracerebral bleed 4.7% and arteriovenous malformation 1%.

MORTALITY

Out of the 29 patients, 9 died giving a case mortality of 31 percent. Five patients (56%) of the dead had intracerebral bleed, one patient (11%) had aneurysm, one patient (11%) had normal angiographic findings and the other two (22%) had not been investigated and therefore findings in them remained unknown. Eight of the dead (89%) were due to the first bleed and one (11%) due to second bleed.

Four patients (44%) died in the first 72 hours, three patients (33%) died after 72 hours but within the first week and 2 patients (22%) died after second week but within four weeks. A total of 49 patients died of cerebrovascular diseases over the study period and subarachnoid haemorrhage contributed 18.4 percent.

RECURRENT BLEEDING

Two patients both with aneurysms had a second bleed on 26th and 28th days. One of the patient died while the other was left disabled. This gave recurrent bleeding in total subarachnoid haemorrhage patients as 7 percent. For the aneurysmal group, recurrent bleeding was 22 percent. Mortality due to recurrent bleeding was 50 percent.

CLINICAL PRESENTATION.

Of the 29 patients confirmed to have subarachnoid haemorrhage, 26 patients (90%) had uniformly

blood stained cerebrospinal fluid and 3 patients (10%) had uniformly xanthochromic cerebrospinal fluid. They presented with various signs and symptoms. Headache was the leading symptom in about 80 percent of the patients, followed by neck pains in 48 percent and then nausea and vomiting in 41 percent. Neck stiffness was the leading sign in 86 percent of the patients, followed by Kernig's sign in 72 percent of the patients. The other symptoms and signs are as shown in table 0.

TRIBAL DISTRIBUTION

Kikuyu tribe had largest number of patients. It contributed 55 percent of the total patients. Kikuyu male constituted 52 percent of the total males and 38 percent of the total patients while the females constituted 63 percent of the total females and 17 percent of the total patients. Male to female ratio in this particular tribe was 2.2:1. The other tribes are as shown in table 1a. Table 1b shows tribal population rates and admission rates to Kenyatta National Hospital.

AGE AND SEX DISTRIBUTION

There were no patients below age of 20 years. The peak incidence was in 20-29 age group followed by 40-49 age group. The age group 20-50 (3rd, 4th and 5th decades) formed 66 percent of the patients.

TABLE O THE FREQUENCY OF SYMPTOMSAND SIGNS

SIGNS AND SYMPTOMS	FREQUENCY	PERCENTAGE %
SYMPTOMS:		
HEADACHE	23	79%
NECK PAINS	14	48
NAUSEA AND VOMITING	12	41
BACKACHE	9	31
CONVULSIONS	5	17
VERTIGO	5	17
DISTURBED VISION (Photophobia, Blindness)	4	14
GENERAL WEAKNESS	3	10
SIGNS		
NECK STIFFNESS	25	86
KERNIGS SIGN	21	72
INABILITY TO TALK (APHASIA)	8	28
UNCONSCIOUSNESS	8	28
RIGHT SIDED HEMIPLEGIA/PARESIS	7	24
LEFT SIDED HEMIPLEGIA/PARESIS	6	21
HYPERREFLEXIA	5	17
DILATED PUPILS	5	17
DISORIENTATION (CONFUSION)	4	14
PAPILLOEDEMA	4	14
ATAXIA	2	7
HYPOTONIA	2	7
DIPLOPIA	2	7
SUBHYOID HAEMORRHAGE	1	3.5
DYSARTHRIA	1	3.5
FEVER	1	3.5
CRANIAL NERVE PALSY/PARESIS		
SEVENTH NERVE	13	45
THIRD NERVE	2	7

TABLE 1 a THE TRIBAL DISTRIBUTION OF PATIENTS IN PROSPECTIVE STUDY.

TRIBE	MALE	%	FEMALE	%	TOTAL	%
Kikuyu	11	38	5	17	16	55
Luo	5	17	0	0	5	17
Kamba	3	10	1	3.4	4	14
Meru	2	7	0	0	2	7
Luhya	0	0	1	3.4	1	3.4
Taita	0	0	1	3.4	1	3.4
Other	0	0	0	0	0	0
Total	21	72	8	28	29	99.8

Table 1b Tribal population rates and admission rates to Kenyatta National Hospital as shown by KASILI E. (52).

TRIBE	POPULATION RATES %	KNH ADMISSION %
KIKUYU	20.1	33.9
KAMBA	10.1	19.8
LUHYA	13.3	9.2
LUO	13.9	17.2
MERU	5.6	3.9
MASAI	1.9	3.3
KALENJIN	10.9	1.1
MIJIKENDA	4.8	1.5
EMBU	1.1	1.7
KISII	6.4	2.2
SOMALI	2.6	4.9
OTHER	6.8	1.4
UNKNOWN	-	-

TABLE 2 THE AGE AND SEX DISTRIBUTION OF PATIENTS IN PROSPECTIVE STUDY.

AGE GROUP IN YEARS	MALE	FEMALES	TOTAL	%
10 - 19	0	0	0	0
20 - 29	4	4	8	27.6
30 - 39	4	0	4	13.8
40 - 49	4	3	7	24.1
50 - 59	2	0	2	6.9
60 - 69	4	1	5	17.2
70 - 79	2	0	2	6.9
80 - 89	0	0	0	0
90 - 99	1	0	1	3.4
TOTAL	21	8	29	99.9

M:F = 2.6:1

RANGE Male 21 - 90 years; Mean ($\bar{x}$) = 47.52; SD=18.72

Female 20 - 60 years; Mean ($\bar{x}$) = 36.50;SD=15.02

Mean (M+F) = 44.48;SD=18.22

The other age groups are as shown in table 2. Men had age range of 21 - 90 years and mean age of 47.52 ± 18.72. Females had age range of 20 - 60 years and mean age of 36.50 ± 15.02. The total males and females had mean age of 44.48 ± 18.22. Females therefore had lower age range and mean age.

OCCUPATION DISTRIBUTION

The largest number of patients were unemployed which formed 24 percent. Business was the leading occupation followed by house wifely and clerical work. The four groups of patients formed nearly 70 percent of the study group. Various other occupations are as shown in table 3.

DISTRIBUTION OF ASSOCIATED FACTORS BETWEEN SEX

One factor association was found in 68 percent while more than one factor was in 32 percent of the patients. Male patients with one factor association formed 43.0 percent while those with more than one factor formed 28.6 percent. Females with one factor formed 25 percent of the total patients while those with more than one factor formed 3.6 percent of the total patients. Alcohol was the leading single factor of association and was incriminated in 35.0

TABLE 3 OCCUPATION DISTRIBUTION OF PATIENTS IN PROSPECTIVE STUDY

OCCUPATION	FREQUENCY	PERCENTAGE
NONE	7	24.1
BUSINESS	4	14.0
HOUSE WIFELY	3	10.3
CLERICAL	3	10.3
HOUSE BOY/GIRL	2	7.0
TECHNICIAN	2	7.0
PEASANTRY	2	7.0
MASON	1	3.4
COBBLER/SHOE MAKER	1	3.4
PRINTER	1	3.4
DRIVING	1	3.4
WATCHMAN	1	3.4
TAILOR	1	3.4
TOTAL	29	99.9

TABLE 4a DISTRIBUTION OF ASSOCIATIONS ACCORDING TO SEX.

ASSOCIATION	MALE	FEMALE	TOTAL
UPSET	2	1	3
HYPERTENSION	1	2	3
ALCOHOL	2	0	2
WALKING	1	1	2
WIPPING FLOOR	0	2	2
DIGGING	1	0	1
COITUS	1	0	1
DEFECATING	1	0	1
EATING	1	0	1
JOGGING & SICKLE CELL	0	1	1
DIABETES	1	0	1
SMOKING	1	0	1
ALCOHOL + SMOKING	4	1	5
ALCOHOL + HYPERTENSION	3	0	3
UPSET + HYPERTENSION	1	0	1
NONE	1	0	1
TOTAL	21	8	28

ASSOCIATION	Male	FEMALE	TOTAL
ONE FACTOR	12	7	19
MORE THAN ONE FACTOR	8	1	9
TOTAL	20	8	28

percent of the patients alone or incombination with others. Hypertension was the second single factor of association found in 24.0 percent of the patients. Activity related (walking,wiping floor, digging coitus, defecating, eating and jogging) association was found in 31.0 percent. Other associations are as shown in table 4a.

ASSOCIATIONS AND INTRACRANIAL VASCULAR ABNORMALITIES.

These are as shown in table 4b. Alcohol and smoking were associated with the largest number of intracranial vascular abnormalities which made up 26.7 percent of the total abnormalities (4 out of 15). 20 percent of these was made up of aneurysms and 6.7 percent made of arteriovenous malformation. The two factors were associated with 33.3 percent of the total number of aneurysms found (3 out of 9).

Alcohol alone was associated with 33.3 percent of the total abnormalities found (5 out of 15). 20 percent of these was made up of aneurysms while 13.3 percent was made up of intracerebral haematoma.

Hypertension was associated with 26.7 percent of the total abnormalities all of which were intracerebral haematomas. It was associated with 80 percent of the intracerebral haematomas found (4 out of 5).

TABLE 4 b DISTRIBUTION OF ASSOCIATIONS AND INTRACRANIAL VASCULAR ABNORMALITIES.

ASSOCIATIONS	ABNORMALITIES				
	NONE	ANEURYSMS	INTRACEREBRAL HEMATOMA	ARTERIO-VENOUS MALFOR-MATIONS	TOTAL
UPSET	1	2	0	0	3
HYPERTENSION	1	0	1	0	2
ALCOHOL	2	0	0	0	2
SMOKING	1	0	0	0	1
WALKING	0	1	0	0	1
WIPPING-FLOOR	1	1	0	0	2
DIGGING	1	0	0	0	1
COITUS	0	1	0	0	1
DEFECATING	1	0	0	0	1
EATING	0	0	1	0	1
JOGGING + SICKLE CELL	0	1	0	0	1
DIABETES	1	0	0	0	1
ALCOHOL + SMOKING	1	3	0	1	5
ALCOHOL + HYPERTENSION	1	0	2	0	3
UPSET + HYPERTENSION	0	0	1	0	1
NONE	1	0	0	0	1
TOTAL	12	9	5	1	27

Two patients with aneurysms ruptured them while emotionally upset. Four other people with aneurysms ruptured them while one was walking, one while wiping the floor, the other while having coitus and the last one while jogging. The details of other associations and abnormalities are shown in table 4b.

DISTRIBUTION OF INTRACRANIAL VASCULAR ABNORMALITIES IN ANATOMICAL SITES AND SEX.

Abnormalities were identified by angiography and postmortem. 16 patients had bilateral carotid and brachial angiography with positive results in 6(5 aneurysms and 1 arteriovenous malformation). Three patients had bilateral carotid angiography only and all were positive (posterior communicating aneurysms in the three). Two patients had right carotid angiography only. One had aneurysm while the other one was normal. Seven patients had postmortem done including one who had complete angiography with positive findings. For the six who had no angiography 5 were found to have intracerebral haematoma. One had evidence of subarachnoid haemorrhage but no identifiable source. Two patients neither had angiography nor postmortem and their cause of bleed was not known.

Among the aneurysms group, posterior communicating aneurysms was leading, contributing 44.4 percent. Left side formed 75 percent of the posterior communicating aneurysm group.

It was found to be more common in males who formed 75% of patients with posterior communicating aneurysms. The others are as shown in table 5-9.

Normal angiographic or postmortem findings formed largest number 44.44 percent, followed by aneurysms which were found in 33.33 percent of the total patients investigated, while intracerebral haemorrhage was third with 18.52 percent and arteriovenous malformation last with 3.7 percent. Normal angiographic findings and aneurysmal group of subarachnoid haemorrhage formed 78 percent of the total patients investigated and male to female ratio in each group and combined was 2:1. Intracerebral bleed (haemorrhage) was found only in males while the single arteriovenous malformation was found in a male. These findings are as shown in table 10.

Both aneurysms and intracerebral haemorrhage were more common on the left than right side but the incidence of the latter was more than the former. Aneurysms were found on left side in 62.5 percent and right side in 37.5 percent of the patients. Intracerebral haemorrhage was found on left side in 80 percent and right side in 20 percent of patients. For combined abnormalities, left side had 69 percent and right side 31 percent. These are as shown in tables 6 and 11.

TABLE 5 : DISTRIBUTION OF INTRACRANIAL VASCULAR ABNORMALITIES SITE ACCORDING TO SEX.

ABNORMALITY	MALE	FEMALE	TOTAL	PERCENTAGE
RIGHT PCOAA	1	0	1	3.7
RIGHT ACAA	1	1	2	7.4
LEFT PCOAA	2	1	3	11.1
LEFT MCAA	2	0	2	7.4
BASILAR AA	0	1	1	3.7
RIGHT AVM	1	0	1	3.7
RIGHT ICH	1	0	1	3.7
LEFT ICH	4	0	4	14.8
NONE	8	4	12	44.4
TOTAL	20	7	27	99.9%

Key

Right PCOAA = Right posterior communicating artery aneurysm.

Right ACAA = Right anterior cerebral artery aneurysm.

Left PCOAA = Left posterior communicating artery aneurysm.

Left MCAA = Left middle cerebral artery aneurysm.

Basilar AA = Basilar artery aneurysm.

Right AVM = Right arteriovenous malformation.

Right ICH = Right intracerebral haemorrhage.

Left ICH = Left intracerebral haemorrhage.

Note:

Two patients had neither angiography nor postmortem and have been omitted in the table.

TABLE 6 DISTRIBUTION OF ANEURYSMS AND INTRACEREBRAL HAEMORRHAGE (ICH) IN BOTH RIGHT AND LEFT CEREBRAL HEMISPHERES.

SIDE	ANEURYSM	%	ICH	%	TOTAL	%
RIGHT	3	37.5	1	20	4	31%
LEFT	5	62.5	4	80	9	69%
TOTAL	8	100	5	100	13	100%

TABLE 7 FREQUENCY OF VARIOUS ANEURYSM SITES.

ANEURYSM LOCATION	FREQUENCY (NUMBER)	PERCENTAGE
POSTERIOR COMMUNICATING ARTERY ANEURYSM (PCOAA)	4	44.4
ANTERIOR CEREBRAL ARTERY ANEURYSM + ANTERIOR COMMUNICATING ANEURYSM (ACAA+ACOAA)	2	22.2
MIDDLE CEREBRAL ARTERY ANEURYSM (MCAA)	2	22.2
INTERNAL CAROTID ARTERY ANEURYSM (ICAA)	0	0.0
BASILAR ARTERY ANEURYSM (BAA)	1	11.1
OTHERS	0	0.0
TOTAL	9	99.9

TABLE 8 COMPARISON OF SITE OF ANEURYSM IN THIS STUDY AND THAT OF MAGEE 1943 (3)

SITE OF ANEURYSM	PRESENT STUDY		MAGEE STUDY 1943	
	NUMBER	%	NUMBER	%
LEFT ANTERIOR CEREBRAL ARTERY	0	0.0	2	4.7
RIGHT ANTERIOR CEREBRAL ARTERY	2	22.2	5	11.6
LEFT MIDDLE CEREBRAL ARTERY	2	22.2	5	11.6
RIGHT MIDDLE CEREBRAL ARTERY	0	0.0	7	16.3
ANTERIOR COMMUNICATING ARTERY	0	0.0	8	18.6
LEFT VERTEBRAL ARTERY	0	0.0	1	2.3
RIGHT VERTEBRAL ARTERY	0	0.0	0	0.0
BASILAR ARTERY	1	11.1	4	9.3
LEFT POSTERIOR CEREBRAL ARTERY	0	0.0	0	0.0
RIGHT POSTERIOR CEREBRAL ARTERY	0	0.0	2	4.7
LEFT POSTERIOR COMMUNICATING ARTERY	3	33.3	0	0.0
RIGHT POSTERIOR COMMUNICATING ARTERY	1	11.1	2	4.7
SITE NOT SPECIFIED	0	0.0	7	16.3
TOTAL	9	99.9	43	100.1

TABLE 9 COMPARISON OF PRESENT STUDY ANEURYSM DISTRIBUTION WITH THOSE OF

1. MCKISSOCK 1956 (13)
2. ADAMS 1976 (47)
3. BARTON 1982 (53)

SITE OF ANEURYSM	PRESENT STUDY		MCKISSOCK		ADAMS		BARTON	
	No.	%	No.	%	No.	%	No.	%
ANTERIOR CEREBRAL & ANTERIOR COMMUNICATING	2	22.2	71	28.5	24	24	226	26.9
POSTERIOR COMMUNICATING ARTERY	4	44.4	62	25	21	21	0	0
MIDDLE CEREBRAL ARTERY	2	22.2	42	17	21	21	152	18.1
INTERNAL CAROTID ARTERY	0	0.0	31	12	10	10	216	25.7
VERTEBROBASILOR ARTERY	1	11.1	15	6	3	3	42	5.0
DISTAL ANTERIOR CEREBRAL ARTERY	0	0.0	7	3	1	1	0	0
MULTIPLE SITES	0	0.0	19	7.5	20	20	181	216
SITE UNVERIFIED	0	0.0	2	1.0	0	0	22	2.6
TOTAL	9	99.9	249	100	100	100	829	99.9

While most of the authors report the anterior cerebral artery and anterior communicating artery as the leading site of aneurysm, this present study shows that the leading site is posterior communicating artery. See also table 8.

TABLE 10 DISTRIBUTION OF TOTAL SUM OF INTRACRANIAL VASCULAR ABNORMALITIES ACCORDING TO SEX.

ABNORMALITY	MALE	FEMALE	TOTAL	PERCENTAGE
NONE	8	4	12	44.44
ANEURYSMS	6	3	9	33.33
INTRACEREBRAL HAEMORRHAGE	5	0	5	18.52
A.V.M.	1	0	1	3.70
TOTAL	20	7	27	99.99

KEY - AVM - ARTERIOVENOUS MALFORMATION.

TABLE 11. DISTRIBUTION OF ANEURYSMS IN BOTH RIGHT AND LEFT CEREBRAL HEMISPHERES.

SIDE	MALE	FEMALE	TOTAL	PERCENTAGE %
RIGHT	2	1	3	37.5
LEFT	4	1	5	62.5
TOTAL	6	2	8	100.0

INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME

These are shown in table 12 and 13. Disabled patients were those who were left bed ridden and could not walk or feed themselves and were totally dependent. Patial recovery included those with hemiparesis but could walk and use their weak limbs, or had isolated cranial nerve paresis or slight confusion irrespective of whether they went back to their former occupation. Those with complete recovery had no neurological deficit at all and went back to their previous jobs.

Patients with normal angiographic findings had best recovery. Complete recovery was 66.6 percent and partial recovery 16.7. Good outcome therefore was 83.3 percent in this group. Mortality in this group was 8.3 percent and contributed 14.4 percent of the total deaths.

Patients with aneurysms were second best in recovery. Complete recovery was 33.3 percent, partial recovery 33.3 percent and therefore good outcome (complete + partial) was 66.6 percent. 22.2 percent in this group were disabled. Mortality was 11.1 percent contributing also 14.3 percent of the total death.

The group with intracerebral haemorrhage had 100 percent mortality contributing 71.4 percent of the total death. One patient with arteriovenous malformation had complete recovery.

TABLE 12 INTRACRANIAL VASCULAR ABNORMALITIES

SITES AND OUTCOME

ABNORMALITY SITE	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL
(PCOAA) RIGHT POSTERIOR COMMUNICATING ARTERY ANEURYSM	0	1	0	0	1
RIGHT ANTERIOR CEREBRAL ARTERY ANEURYSM (ACAA)	0	1	0	1	2
LEFT POSTERIOR COMMUNICATING ARTERY ANEURYSM	0	0	2	1	3
LEFT MIDDLE CEREBRAL ARTERY ANEURYSM(MCAA)	1	0	1	0	2
LEFT INTRACEREBRAL HAEMORRHAGE (ICH)	4	0	0	0	4
RIGHT INTRACEREBRAL HAEMORRHAGE (ICH)	1	0	0	0	1
RIGHT ARTERIOVENOUS MALFORMATION (AVM)	0	0	0	1	1
BASILAR ARTERY ANEURYSM (BAA)	0	0	0	1	1
NO LESION	1	1	2	8	12
LESION NOT LOOKED FOR	2	0	0	0	2
TOTAL	9	3	5	12	29
PERCENT %	31	10.3	17.2	41.4	99.9

TABLE 13 TOTAL INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME

ABNORMALITIES	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL	%
NONE	1	1	2	8	12	44.4
ANEURYSMS	1	2	3	3	9	33.3
INTRACEREBRAL HAEMORRHAGE	5	0	0	0	5	18.5
ARTERIO VENOUS MALFORMATION	0	0	0	1	1	3.7
TOTAL	7	3	5	12	27	99.9
PERCENT %	26	11.1	18.5	44.4	100	

AGE AND OUTCOME

Age and outcome is shown in table 14. The results indicate that the younger the patient with subarachnoid haemorrhage, the better the outcome. The best recovery was in third and 4th decades (20-40 years). Good outcome (complete and partial) in each decade separately and when combined was 75 percent while poor outcome (death and disabled) was 25 percent. The two decades made 41 percent of the total patients. From 5th decade onwards, good outcome was less than 50 percent. Mean age was lower for those with complete recovery as compared to those who died (41.7 and 52.78 respectively). This was however not statistically significant ($P>.10$).

SEX AND OUTCOME

These are illustrated in table 15. Females with good outcome (complete recovery and partial recovery) formed 50 percent. Poor outcome (disabled and death) also formed 50 percent. Mortality in females was 25 percent which made 22 percent of the total death. Males with good outcome formed 62 percent, and poor outcome 38 percent. Mortality in males was 33.3% and made 78% of the total death. These results indicate that females had lower mortality than males while male survivors had better recovery than female survivors. This difference was not statistically significant ($P>0.5$).

TABLE 14 AGE AND OUTCOME OF PATIENTS IN PROSPECTIVE STUDY.

AGE GROUPS	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL
10 - 19	0	0	0	0	0
20 - 29	1	1	2	4	8
30 - 39	1	0	0	3	4
40 - 49	3	1	1	2	7
50 - 59	1	0	1	0	2
60 - 69	2	1	0	2	5
70 - 79	0	0	1	1	2
80 - 89	0	0	0	0	0
90 - 99	1	0	0	0	1
TOTAL	9	3	5	12	29
MEAN AGE	52.78	45.0	45.0	41.7	46.03

TABLE 15 : SEX AND OUTCOME OF PATIENTS IN PROSPECTIVE STUDY.

SEX	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL	%
FEMALE	2	2	1	3	8	28
MALE	7	1	4	9	21	72
TOTAL	9	3	5	12	29	100.0
PERCENT %	31.0	10.3	17.2	41.4	99.9	

ECG CHANGES IN TOTAL PATIENTS AND IN CONTROL AND PROPRANOLOL GROUPS

Out of 29 patients in study group, 22 had ECGs done and analysed. Abnormal changes were found in 73 percent of the patients. The various ECG changes are as shown in table 16. T wave changes were the leading abnormality forming 58 percent of the total changes. Flat T wave was the leading single change giving 31.6 percent of the total ECG changes and 55 percent of the T wave changes. Others are as shown in the table 16.

In control group, ECG changes were found in 69 percent of the ECGs done in this group and formed 56 percent of total ECG changes. In the propranolol group, ECG changes were found in 78 percent and this formed 44 percent of the total ECG changes (see table 17). T wave changes especially flat T wave occurred almost at the same rate in propranolol patients as in control group.

More than one ECG change occurred in some patients examples of this are:- patient P2, a 26 year old non hypertensive man who had right anterior cerebral aneurysm near origin had Q wave in avF and lead III and flat T wave in lead III. P15, a 58 year old businessman who was also hypertensive for 10 years and had normal angiography, had inverted T-wave and depressed ST segment on V5 and V6 leads. He also had left ventricular hypertrophy and left axis deviation. He was on propranolol.

Two female patients P24 and P29 aged 20 and 23 years respectively and having left posterior communicating artery aneurysm and right anterior cerebral artery aneurysm respectively had inverted and flat T waves in their ECGS.

One female patient 23 years old who had basilar artery aneurysm had a prominent Q-waves 2mm in lead II, III, and avf of her 72 hour ECG and after 10 days of propranolol her ECG showed large Q waves 3.5 mm in the same leads.

TABLE 16 FREQUENCY OF VARIOUS ECG CHANGES IN THE PROSPECTIVE STUDY.

ECG CHANGE	FREQUENCY	PERCENT
FLAT T WAVE	6	31.6
INVERTED T WAVE	2	10.5
TALL T WAVE	3	15.8
DEPRESSED ST SEGMENT	2	10.5
PATHOLOGICAL Q WAVE	2	10.5
HYPERTROPHY (LVH)	1	5.3
ARRHYTHMIA (ATRIAL FIBRILLATION)	1	5.3
PROLONGED PR INTERVAL (0.24 SEC)	1	5.3
SHORTENED P-R INTERVAL (0.10)	1	5.3
TOTAL	19	100.1

KEY

ECG - Electrocardiogram

LVH - Left ventricular Hypertrophy

TABLE 17 ECG CHANGE DISTRIBUTION BETWEEN PROPRANOLOL AND CONTROL GROUP.

GROUP	ECG CHANGE	NORMAL ECG	TOTAL	PERCENT %
CONTROL	9	4	13	59%
PROPRA-NOLOL	7	2	9	41%
TOTAL	16	6	22	100%
PERCENT	73%	27%	100%	

KEY

ECG - Electrocardiogram

RECOVERY PERIOD IN PROPRANOLOL AND CONTROL GROUPS.

Of 29 patients in prospective study, 11 were randomly put on propranolol and 14 were used as control. 4 patients could not be put in any of the group as they died within 48 hours of admission. One patient in propranolol group developed hypotension and was withdrawn from the group. The other patient died of rebleeding of left middle cerebral artery aneurysm after he had completed 3 weeks on propranolol and had not regained consciousness. This was not included when calculating recovery period. The third patient in propranolol group recovered fully after 28 days, 7 days after he had completed taking propranolol. The propranolol group was finally left with 8 patients. In the control group 4 patients died from the effect of the first bleed before completing the 21 days course and were excluded from the analysis of recovery period. Only 10 patients completed the study course.

The mean recovery period for the control group was 13 days. The mean recovery period for the propranolol group was 9 days and when patient with 28 days was excluded it was 8 days.

After statistical analysis including patient with 28 days using the conditional Chi square test with X^2 (1) = 1.176 and P value $>$ 0.25, it was found out that there was no significant differences in the recovery period between the two groups.

However, when the recovery period between the two groups was tested excluding patient with 28 days who had gone beyond study period, $X^2(1) = 2.763$ and P value $<$ 0.05. The result was found to be significant at 5% level for a one sided test. See tables 18 and 19a & 19b.

TABLE 18 NUMBER OF DAYS IN BOTH PROPRANOLOL AND CONTROL GROUPS TAKEN FOR FULL CONSCIOUSNESS TO BE RESTORED AND HEADACHE, KERNIG'S SIGN AND NECK STIFFNESS TO RESOLVE.

GROUP	NUMBER OF DAYS TAKEN BY VARIOUS PATIENTS.	SUM	MEAN
CONTROL	2,7,7,7,14,14,16,18,21, 21, 4 DIED	127	13
PROPRA-NOLOL	2,3,7,7,8,10,10,14,28 1 DIED.	89	9

TABLE 19a & b FIGURES USED FOR STATISTICAL ANALYSIS FOR DAYS TAKEN BY TWO GROUPS USING CONDITIONAL CHI-SQUARE TEST (a) WITH PATIENT HAVING 28 DAYS INCLUDED AND (b) PATIENT WITH 28 DAYS EXCLUDED.

(a)

DAYS	X	PROPRA-NOLOL	CONTROL	TOTAL
< 5	2.5	2	1	3
5-10	7.5	3	3	6
10-15	12.5	3	2	5
15-20	22.5	1	2	3
> 20	22.5	1	2	3
Sum	n	9	10	19
		87.5	130	217.5
Mean		9.72	13	11.45

$X^2(1) = 1.176$, $P > 0.25$ $T = 1.08$

(b)

DAYS	X	PROPRA-NOLOL	CONTROL	TOTAL
<5	2.5	2	1	3
5-10	7.5	3	3	6
10-15	12.5	3	2	5
15-20	17.5	0	2	2
> 20	22.5	0	2	2
Sum	n	8	10	18
		65	130	195
Mean		8.13	13	10.83

X^2 (1) = 2.763, $P < 0.05$ T =1.7

OUTCOME IN BOTH PROPRANOLOL AND CONTROL GROUPS

This is as shown in tables 20,21 and 22. Good outcome in propranolol group (complete recovery plus partial recovery) was 80 percent while it was 64.0 percent in the control group. This difference was not statistically significant ($P > 0.50$).

Normal angiographic findings patients were 7 in propranolol group. 4 had complete recovery, two had partial recovery and 1 was disabled. This gave good outcome as 86%. There were 5 normal angiographic findings patients in control. Three had complete recovery which was 60% and two died giving poor outcome as 40 percent.

There were 3 aneurysmal patients in propranolol group, 2 (66.7%) had complete recovery and 1 (33.3%) died. Control group had 6 patients with aneurysms. 4 patients (66.7%) had partial recovery, 1(16.7%) had complete recovery and one patient (16.7%) was disabled.

There were two patients with intracerebral haemorrhage in control and both died, none in propranolol.

One patient in control with arteriovenous malformation had complete recovery. The overall result is better recovery in propranolol than control even when individual abnormalities are compared.

TABLE 20 THE OUTCOME IN BOTH PROPRANOLOL AND CONTROL GROUPS.

GROUP	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL
NUMBER	4	1	4	5	14
CONTROL %	28.6	7.1	28.6	35.7	100
NUMBER	1	1	2	6	10
PROPRA-NOLOL %	10	10	20	60	100

TABLE 21 INTRACRANIAL VASCULAR ABNORMALITIES IN PROPRANOLOL AND CONTROL GROUPS

ABNORMALITIES	CONTROL GROUP		PROPRANOLOL GROUP	
	FREQUENCY	PER-CENT	FREQUENCY	PERCENT
ANEURYSM	6	43	3	30
INTRACEREBRAL HAEMORRHAGE	2	14	0	0
ARTERIOVENOUS MALFORMATION	1	7	0	0
NONE	5	36	7	70
TOTAL	14	100	10	100

TABLE 22 VARIOUS INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME IN BOTH PROPRANOLOL AND CONTROL GROUPS.

ABNORMALITIES	DEATH		DISABLED		PARTIAL RECOVERY		COMPLETE RECOVERY		TOTAL
	PROPRANOLOL	CONTROL	PROPRANOLOL	CONTROL	PROPRANOLOL	CONTROL	PROPRANOLOL	CONTROL	
ANEURYSMS	1	0	0	1	0	4	2	1	9
INTRACEREBRAL HAEMORRHAGE	0	2	0	0	0	0	0	0	2
ARTERIOVENOUS MALFORMATION	0	0	0	0	0	0	0	1	1
NONE	0	2	1	0	2	0	4	3	12
TOTAL	1	4	1	1	2	4	6	5	24

CHOLESTEROL LEVELS

Cholesterol levels were estimated in 26 patients. The range was from 2.24 to 7.5 mmol/litre and mean was 4.5 mmol/litre. They were all within normal limits except two and are shown in table 23.

17-ketosteroid, 17-Hydroxysteroid, vannilyl mandelic acid and catecholamines Levels in 24 hour urine specimen were all normal as shown in table 23.

I.V.U. (INTRAVENOUS UROGRAPHY) FINDINGS

20 patients had intravenous urography done on them including those with aneurysms. 19 patients had normal urography giving a percentage of normal as 95 percent. One patient's urography was reported as having bilateral hydroureters due to prostatic hypertrophy. Kidney function was fair although urea was raised. Thus none of these patients had polycytic kidneys, or coarctation of aorta.

POSTMORTEM FINDINGS

Seven patients had postmortem. Four of these patients were hypertensive and aged 45,46,50 and 90 years. They all had intracerebral haematoma with subarachnoid leakage and some degree of heart enlargement. Their lungs were normal.

TABLE 23 SHOWING SERUM CHOLESTEROL LEVELS AND 17-KETOSTEROID, 17-HYDROXYSTEROID, CATECHOLAMINES AND VANILLYL MANDELIC ACID (VMA) LEVELS IN 24 HOUR URINE SPECIMEN.

PATIENT	CHOLESTEROL IN MMOLS/L Normal (3.1-6.47)	17-KETOSTEROID UMOL/24 HR Normal (21-70)	17-HYDROXY-STEROID UMOLS/24HR Normal (21-87)	CATECHO-LAMINE UMOLS/ 24 HR. 0.05-0.9	VMA UMOLS/ 24 HR. Normal upto 36
P 1	4.5	27.0	51.6	0.9	NEGATIVE
2	4.15	18.2	42.5	0.9	NEGATIVE
3	-	-	-	-	NEGATIVE
4	4.8	26.6	47.5	0.9	NEGATIVE
5	6.3	35.0	48.0	0.9	NEGATIVE
6	7.1	34.0	45.0	0.9	NEGATIVE
7	4.95	18.0	16.0	0.9	NEGATIVE
8	3.9	29.0	72.0	0.9	NEGATIVE
9	3.9	41.0	74.0	0.9	NEGATIVE
10	4.3	40.0	81.0	0.9	NEGATIVE
11	3.5	D	D	0.9	NEGATIVE
12	4.3	-	-	-	-
13	-	-	-	-	-
14	5.2	29.0	44.0	0.9	NEGATIVE
15	4.7	25.0	36.0	0.9	NEGATIVE
16	3.6	38.0	62.0	0.9	NEGATIVE
17	5.4	23.0	66.0	0.9	NEGATIVE
18	3.76	37.0	52.0	0.9	NEGATIVE
19	2.24	35.0	60.0	0.9	NEGATIVE
20	7.5	16.0	50.0	0.9	NEGATIVE
21	3.47	29.0	65.0	0.9	NEGATIVE
22	-	-	-	-	-
23	4.53	3.0	6.0	0.9	NEGATIVE
24	4.75	21.0	24.0	0.9	NEGATIVE
25	3.47	32.0	70.0	0.9	NEGATIVE
26	3.4	19.0	37.0	0.9	NEGATIVE
27	3.8	--	-	0.9	NEGATIVE
28	5.2	22.0	62.0	0.9	NEGATIVE
29	4.4	16.0	12.90	0.9	NEGATIVE

KEY D-DISCARDED AS PATIENT HAD TAKEN DEXAMETHASONE

The other three were non hypertensive. One was a woman aged 60 years whose postmortem confirmed subarachnoid haemorrhage but no source was identified. The other was 28 year old man struck while taking his supper. He had right intracerebral haematoma. His heart and kidneys were normal. Last was 32 year old man alcoholic and heavy smoker who died of rebleeding on 26th day in hospital. Carotid angiography during life had shown left middle cerebral artery aneurysm near origin. Postmortem showed a clot around bifurcation of left internal carotid and subarachnoid blood more on left hemisphere.

Two patients who had postmortem aged 46 and 50 years old, had atheromatous plaques involving aorta and cerebral vessels. The one aged 50 had cholesterol level of 7.5 mmol/litre. The other one aged 46 cholesterol levels were not estimated.

Only one patient 90 year old man had isolated renal cyst in the cortex of left kidney otherwise both kidneys were of normal size.

RETROSPECTIVE STUDY

This study had a total of 80 patients. There were 57 males and 23 females giving male to female ratio of 2.5:1. This compares quite well with prospective study which showed ratio of 2.6:1. 20 patients out of 80 died giving a mortality of 25 percent. Females made 30 percent of the total death and male 70 percent. In prospective study 9 patients out of 29 patients died giving a mortality of 31 percent. Females made 22 percent of this and male 88 percent.

TRIBAL DISTRIBUTION

As in prospective study, Kikuyu tribe had largest number of patients and constituted 46.25 percent of the total number of patients. Kikuyu male constituted 40.4 percent of the total male and 29 percent of the total patients while the females constituted 61 percent of the total females and 17.5 percent of the total number of patients. The male to female ratio in this particular tribe was 1.6:1. The findings corresponds with those of prospective study. The other tribes are as shown in table 24.

AGE AND SEX DISTRIBUTION

There was only one patient below age of 20 comparing quite well with prospective study which had no patient in this age group. The peak

TABLE 24 THE TRIBAL DISTRIBUTION OF PATIENTS IN RETROSPECTIVE STUDY.

TRIBE	MALE	%	FEMALE	%	TOTAL	%
KIKUYU	23	28.75	14	17.5	37	47.25
KAMBA	8	10.0	2	2.5	10	12.5
LUO	8	10.0	0	0.0	8	10.0
LUHYA	4	5.0	2	2.5	6	7.5
SOMALI	4	5.0	0	0.0	4	5.0
MASAI	2	2.5	1	1.25	3	3.75
KALENJIN	1	1.25	1	1.25	2.	2.5
TAITA	2	2.5	0	0.0	2	2.5
NUBI	1	1.25	1	1.25	2	2.5
MERU	1	1.25	0	0.0	1	1.25
SAMBURU	1	1.25	0	0.0	1	1.25
KURIA	1	1.25	0	0.0	1	1.25
POKOMO	0	0.0	1	1.25	1	1.25
OTHER	2	2.5	1	1.25	3	3.75
TOTAL	57	72.25	23	27.75	80	100.0

TABLE 25 THE AGE AND SEX DISTRIBUTION OF PATIENTS IN RETROSPECTIVE STUDY.

AGE GROUP IN YRS	MALE	FEMALE	TOTAL	PERCENT
10 - 19	1	0	1	1.25
20 - 29	6	6	12	15.0
30 - 39	13	7	20	25.0
40 - 49	16	2	18	22.5
50 - 59	11	3	14	17.5
60 - 69	3	2	5	6.25
70 - 79	5	1	6	7.5
80 - 89	1	2	3	3.75
90 - 99	1	0	1	1.25
TOTAL	57	23	80	100.0%

M:F = 2.5:1
RANGE: Male = 15 - 90 years; Mean ($\bar{X}$) = 45.53;SD=15.81
Female= 20 - 80 years;Mean ($\bar{X}$) = 42.13;SD=19.06
Mean (M+F) = 44.55; SD=16.78

incidence was in the 4th decade, followed by 5th, 6th and then 3rd decades. Between age group 20 - 60 years was the 80 percent of population while between 20 - 50 years had 63 percent of the population studied. This also compares quite well with prospective study which had 66 percent of population in 20 - 50 years age group. The other age groups are as shown in table 25. Males had age range of 15 - 90 years and a mean age of 45.53 ± 15.81. Females had a mean age of 42.13 ± 19.06. Combined males and females had a mean age of 44.55 ± 16.78. Females had slightly lower mean age.

INTRACRANIAL VASCULAR ABNORMALITIES DISTRIBUTION ACCORDING TO ANATOMICAL SITES AND SEX

Abnormalities identification was by angiography and postmortem. 6 patients had bilateral carotid and brachial angiography all with normal findings. 30 patients had bilateral carotid angiography and 11 of these had positive findings (8 aneurysms, 2 intracerebral haematoma and 1 arteriovenous malformation). 9 patients had left carotid angiography only and 3 had positive findings (1 arteriovenous malformation, 1 aneurysm and 1 Moya Moya disease). 8 patients had right carotid angiography only with positive findings in two (2- aneurysms). 4 people

had postmortem done including one who had had bilateral carotid angiography with corresponding findings of left internal carotid aneurysm. Of the other three with no angiography during life, only one had positive findings of right intracerebral haematoma with bilateral chronic subdural haematoma.

11 patients had aneurysms. 4 were operated and all had partial recovery. Three of these were posterior communicating and the fourth one was carotid. The other 7 patients with aneurysms had conservative treatment. Three of these died, one with posterior communicating, one with anterior cerebral and one carotid. Two patients with anterior communicating artery aneurysms had complete recovery. Two patients one with posterior communicating and the other carotid aneurysm had partial recovery.

One of the two patients with arteriovenous malformation was operated and had partial recovery while the other had conservative treatment and was disabled. Three patients with intracerebral haematoma had conservative management and two had complete recovery while the other one was disabled. Patient with Moya Moya disease had conservative treatment with partial recovery.

39 of the 56 patients investigated had no abnormality found. 24 patients had neither

angiography nor postmortem in those who died and it is not known what they had.

Right posterior communicating artery aneurysm was the commonest site. For the overall aneurysms, posterior communicating aneurysm was commonest and formed 29.4 percent of the abnormalities found and 45.5 percent of the aneurysms. This compares very well with prospective study in which posterior communicating artery aneurysm formed 44.4 percent of the total number of aneurysms found. Right posterior communicating artery aneurysms formed 60 percent and were all in males while left posterior communicating aneurysm formed 40 percent and were all found in females. The other aneurysms and other abnormalities were as shown in tables 26 and 27.

In the whole group of investigated patients, normal angiographic findings formed the largest number of 70 percent. Aneurysms were second with 20 percent. Intracerebral haematomas contributed 5.4 percent and arteriovenous malformation 3.4 percent, while Moya Moya disease with the least number of patient contributed 1.8 percent. These findings are as shown in table 28. The pattern here is similar to that found in prospective study.

TABLE 26 DISTRIBUTION OF INTRACRANIAL VASCULAR ABNORMALITIES SITE ACCORDING TO SEX.

ABNORMALITY	MALE	FEMALE	TOTAL	PERCENT
ANTERIOR COMMUNICATING ARTERY ANEURYSM	2	0	2	11.8
RIGHT INTERNAL CAROTID ANEURYSM	0	1	1	5.9
LEFT INTERNAL CAROTID ANEURYSM	2	0	2	11.8
RIGHT POST-COMMUNICATING ARTERY ANEURYSM	3	0	3	17.6
LEFT POST-COMMUNICATING ARTERY ANEURYSM	0	2	2	11.8
RIGHT ANTERIOR CEREBRAL ARTERY ANEURYSM	1	0	1	5.9
RIGHT A.V.M.	1	0	1	5.9
LEFT A.V.M.	0	1	1	5.9
RIGHT INTRACEREBRAL HAEMORRHAGE	3	0	3	17.6
MOYA MOYA DISEASE (ANTERIOR CEREBRAL AND MIDDLE CEREBRAL ARTERY)	0	1	1	5.9
TOTAL	12	5	17	100

In this group M:F was 2.2:1 for those with aneurysm M:F was 2.3:1 for AVM group M:F was 1:1.

Key

POST - Posterior
AVM - Arteriovenous malformation.

TABLE 27 COMBINED RIGHT AND LEFT INTRACRANIAL VASCULAR ABNORMALITIES IN RETROSPECTIVE STUDY.

ABNORMALITY	FREQUENCY	PERCENT %
ANTERIOR CEREBRAL + ANTERIOR COMMUNICATING ARTERY ANEURYSM	3	17.6
POSTERIOR COMMUNICATING ARTERY ANEURYSM	5	29.4
INTERNAL CAROTID ANEURYSM	3	17.6
ARTERIOVENOUS MALFORMATION	2	11.8
INTRACEREBRAL HAEMORRHAGE	3	17.6
MOYA MOYA DISEASE	1	5.9
TOTAL	17	100.0

TABLE 28 INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME IN RETROSPECTIVE GROUP.

ABNORMALITY	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL	%
NONE	3	2	12	22	39	70.0
POSTERIOR COMM. ARTERY ANEURYSM	1	0	4	0	5	9.0
CAROTID ANEURYSM	1	0	2	0	3	5.4
ANTERIOR CEREBRAL AND ANTERIOR COMM. ARTERY ANEURYSM	1	0	0	2	3	5.4
ARTERIOVENOUS MALFORMATION (AVM)	0	1	1	0	2	3.4
INTRACEREBRAL BLEED (ICH)	1	0	0	2	3	5.4
MOYA MOYA DISEASE	0	0	1	0	1	1.8
TOTAL	7	3	20	26	56	100.4
PERCENT	12.5	5.4	35.7	4.64	100%	

KEY

COMM. - COMMUNICATING

INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME

These findings are as shown in table 28. Normal angiographic findings patients as well as being the largest group had best outcome. Complete recovery in this group was 56.4 percent and partial recovery 31 percent. Good outcome therefore (complete recovery plus partial recovery) was 87 percent. Mortality in this group was 8 percent and this made 43 percent of the total death.

For the aneurysm group, complete recovery was 18 percent, partial recovery 55 percent and therefore good outcome was 73 percent. This group had a mortality of 27.3 percent which made up 43 percent of the total death.

Intracerebral haemorrhage group had 66 percent complete recovery and mortality of 33 percent and contributed 16 percent of total death.

Arteriovenous malformation had 50 percent partial recovery and 50 percent disabled. The Moya Moya patient had partial recovery.

The pattern of outcome in normal angiographic findings, aneurysms and intracerebral haematoma is similar to that found in prospective study.

AGE AND OUTCOME

The age and outcome is shown in table 29. The results as in prospective study, indicated

TABLE 29 AGE AND OUTCOME IN RETROSPECTIVE STUDY

AGE GROUP	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL
10 - 19	1	0	0	0	1
20 - 29	3	0	5	4	12
30 - 39	3	2	3	12	20
40 - 49	2	1	4	11	18
50 - 59	5	1	5	3	14
60 - 69	1	1	1	2	5
70 - 79	2	0	3	1	6
80 - 89	2	0	1	0	3
90 - 99	1	0	0	0	1
TOTAL	20	5	22	33	80
MEAN AGE IN YEARS	52	47	48.2	41.97	46.5

that the younger the patient the better the outcome. The best outcome was in 3rd, 4th and 5th decades, where complete recovery plus partial recovery formed 75 percent of patients in these age groups. From 6th decade onwards, good outcome was 53 percent or less. There was also a great difference in mean age of those with complete recovery and those who died (42. and 52 years respectively). When age difference was tested using conditional chi square test, the result was found to be significant at 5 percent level. This also compares quite well with the findings in prospective study.

SEX AND OUTCOME

The findings are shown in table 30. Females with good outcome (complete recovery plus partial recovery) formed 65 percent, while poor outcome (disabled plus death) formed 35 percent. They had a mortality of 26 percent and made 30 percent of total death.

Males with good outcome formed 70 percent while those with poor outcome formed 30 percent. They had a mortality of 25 percent and made 70 percent of the total death.

Females and males had similar mortality 26 percent and 25 percent respectively. Survivors of both sex had similar good outcome 65 percent and 70 percent respectively.

COMBINED RESULTS OF PROSPECTIVE AND RETROSPECTIVE STUDIES.

These are outlined in tables 31 and 32.

TABLE 30 SEX AND OUTCOME IN RETROSPECTIVE STUDY

SEX	DEATH	DISABLED	PARTIAL RECOVERY	COMPLETE RECOVERY	TOTAL	PERCENT
FEMALE	6	2	6	9	23	29
MALE	14	3	16	24	57	71
TOTAL	20	5	22	33	80	100.0
PERCE-NT	25	6.25	27.5	41.25	100	

TABLE 31 FREQUENCY OF COMBINED INTRACRANIAL VASCULAR ABNORMALITIES IN PROSPECTIVE AND RETROSPECTIVE STUDIES

INTRACRANIAL VASCULAR ABNORMALITIES	FREQUENCY (NUMBER)	PERCENTAGE %
NONE	51	61
ANEURYSM	20	24
INTRACEREBRAL HAEMORRHAGE	8	10
ARTERIOVENOUS MALFORMATION	3	4
MOYA MOYA DISEASE	1	1
TOTAL	81	100%

TABLE 32 DISTRIBUTION OF ANEURYSMS IN THE COMBINED PROSPECTIVE AND RETROSPECTIVE STUDIES.

ANEURYSMAL SITE	FREQUENCY (NUMBER)	PERCENTAGE %
POSTERIOR COMMUNICATING ARTERY ANEURYSMS	9	45
ANTERIOR CEREBRAL & ANTERIOR COMMUNICATING ARTERY ANEURYSMS	5	25
CAROTID ARTERY ANEURYSMS	3	15
MIDDLE CEREBRAL ARTERY ANEURYSMS	2	10
BASILAR ARTERY ANEURYSMS	1	5
TOTAL	20	100%

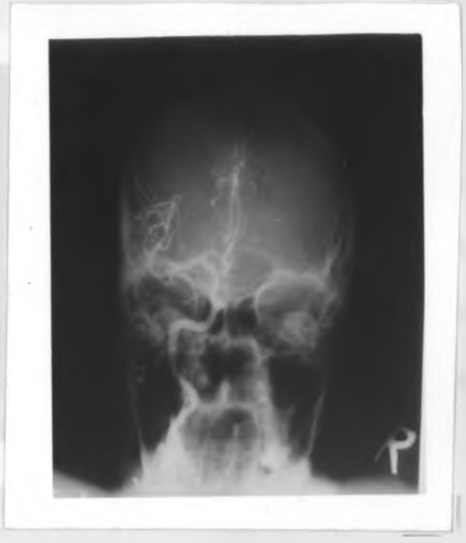

Fig 1a

Normal anteroposterior view of right carotid angiogram.

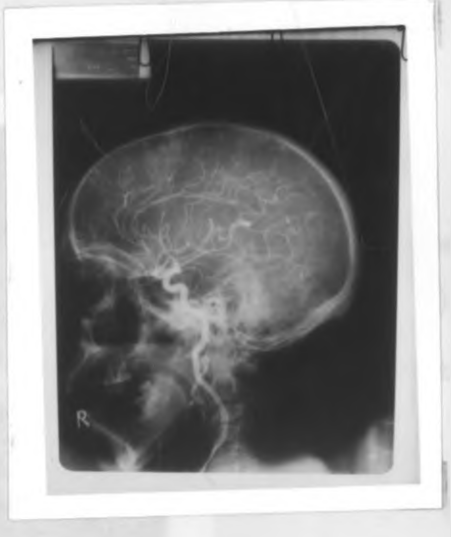

Fig 1b

Normal lateral view of right carotid angiogram

Fig 1a & b are angiograms of 70 year old man who was struck by subarachnoid haemorrhage when digging, had right sided hemiparesis and normotensive. He belonged to/propranolol group and had complete recovery.

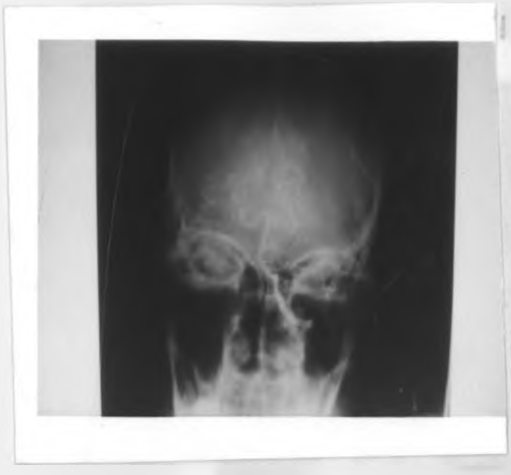

Fig 2a
Normal anteroposterior view of brachial (vertebral) angiogram showing vertebral, basilar and posterior cerebral arteries circulation.

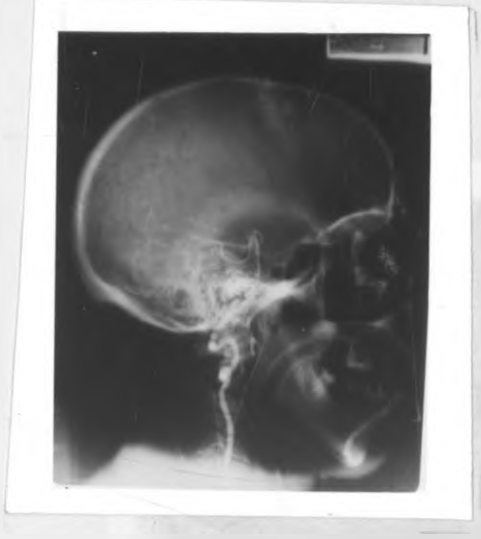

Fig 2b
Normal lateral view of brachial (vertebral) angiogram showing same circulation as 2a.

Fig 2a & b
are brachial vertebral angiogram of 70 year old man whose carotid angiograms are shown in fig 1a & b.

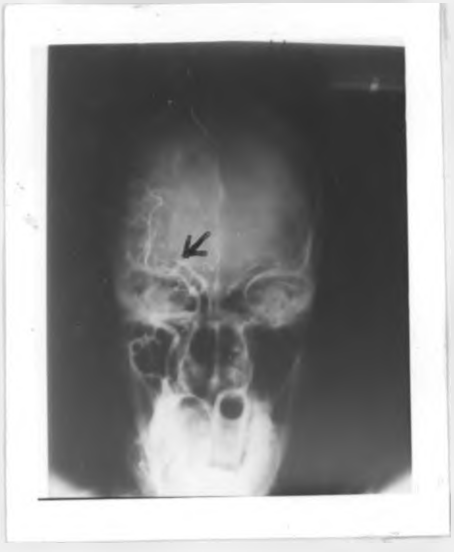

Fig 3a
showing antero-posterior view of right carotid angiogram with small aneurysm shown by arrow and very narrowed (spastic) anterior cerebral artery, but normal middle cerebral artery.

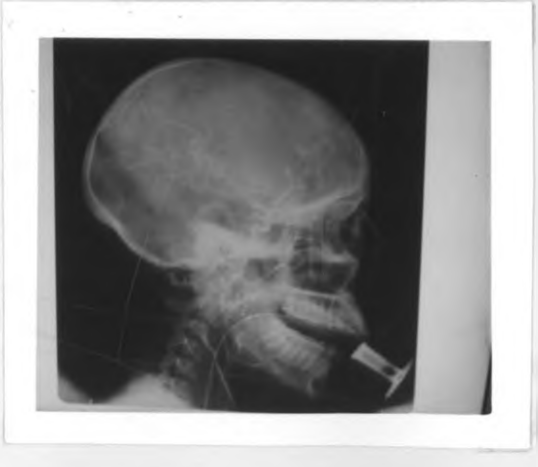

Fig 3b
Lateral view or right carotid angiogram showing very spastic anterior cerebral artery.

Fig 3a and 3b are carotid angiograms of 23 year old female who was struck by subarachnoid haemorrhage whilejogging.. She was in control group. She had left sided hemiplegia and was not hypertensive. She got disabled.

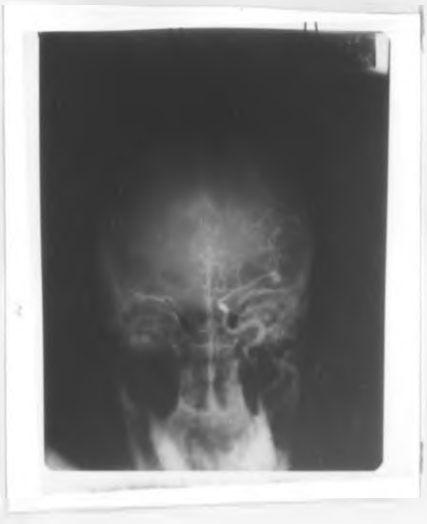

Fig 4a
shows anteroposterior view of left carotid angiogram with large aneurysm at middle cerebral artery at M_2-M_3

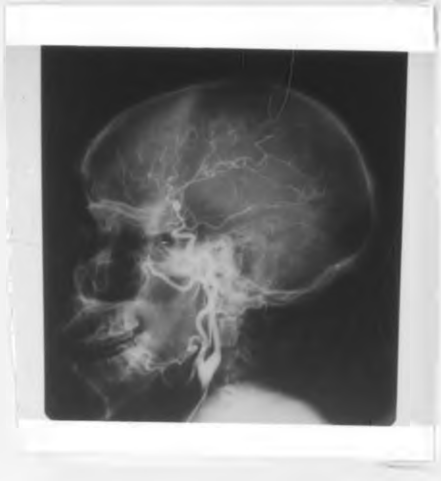

Fig 4 b
Lateral view of left carotid of same patient showing same aneurysm as 4a.

Fig 4a and 4b are carotid angiogram of 42 year old normotensive male who was struck by subarachnoid haemorrhage during coitus. He had right sided hemiparesis and aphasia. He was in control group. He had conservative management and had partial recovery (walking with support and slight confusion).

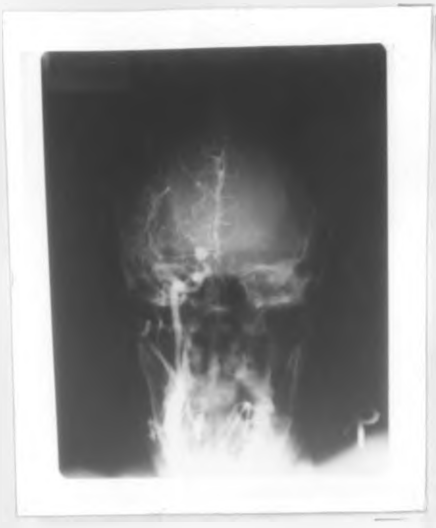

Fig 5 a

showing antero-posterior view of carotid angiogram with middle cerebral aneurysm near bifurcation.

Fig 5 b

showing lateral view of carotid angiogram with aneurysm on middle cerebral artery near origin.

Fig 5a and 5b show carotid angiogram (left) of 32 year old male heavy smoker and alcoholic. He was struck when drunk. He did not have lateralizing signs. He was in propranolol group but never gained full consciousness even after 3 weeks course. He died on 26th day in hospital due to rebleeding. Postmortem confirmed site of bleed to be at birfurcation of left internal carotid.

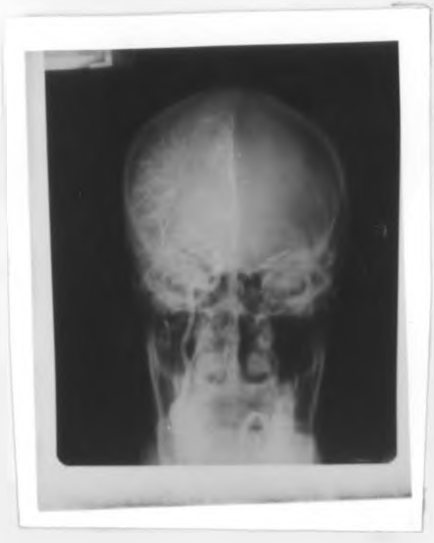

Fig 6a

showing antero-posterior view of left carotid angiogram with posterior communicating artery aneurysm.

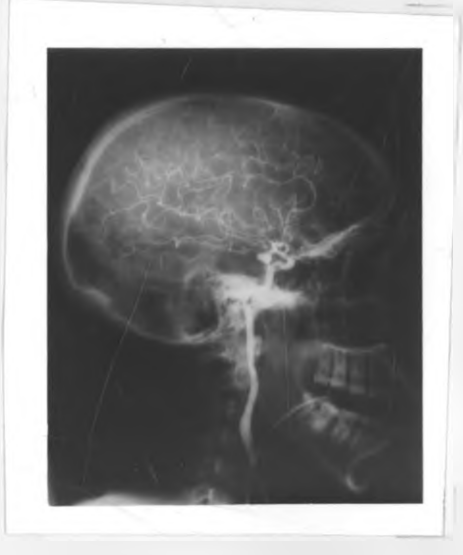

Fig 6b

showing lateral view of carotid angiogram with large aneurysm of left posterior communicating artery.

Fig 6a and b show left carotid angiogram of 27 year male heavy smoker and alcoholic struck when drunk and had left ptosis and diplopia. This aneurysm was clipped and patient was left with partial ptosis.

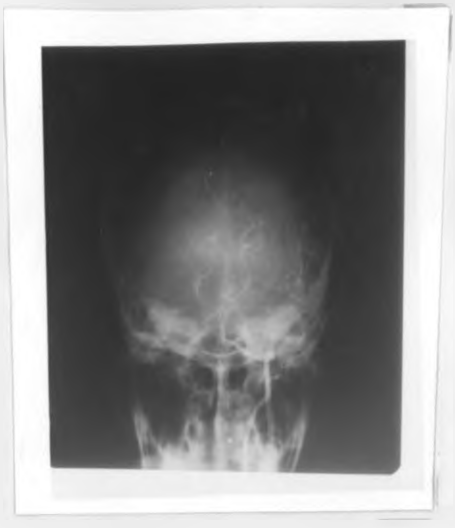

Fig 7a

shows anteroposterior view of brachial vertebral angiogram. Aneurysm not visible here.

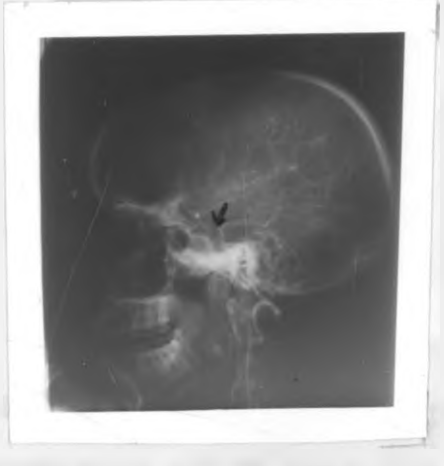

Fig 7b

shows lateral view of brachial-vertebral angiography showing small basilar aneurysm (Arrow).

Fig 7a and b show brachial-vertebral angiogram of 23 year old female heavy smoker and alcoholic struck when drinking alcohol. Had no lateralizing signs. Her carotid angiography showed spastic vessels but no aneurysms. She had complete recovery. She was normotensive and in propranolol group: She was managed conservatively.

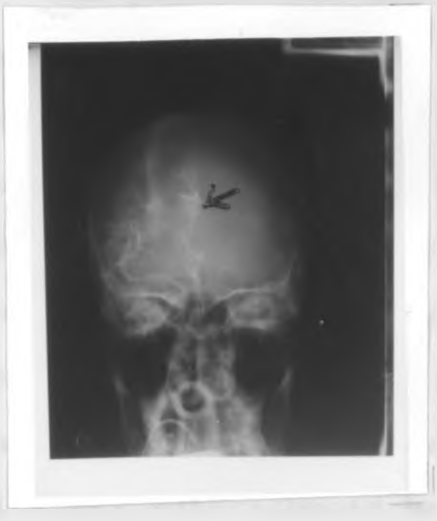

Fig 8a
Anteroposterior view of carotid angiogram showing arteriovenous malformation (arrow)

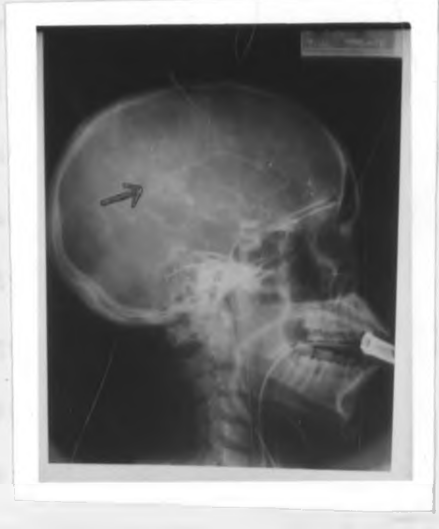

Fig 8b
Lateral view of carotid angiogram showing faintly arteriovenous malformation involving anterior and middle cerebral arteries (arrow)

8a & b show carotid angiograms with arteriovenous malformation of 30 year old male, heavy smoker, alcoholic and normotensive. He had no lateralizing signs and had complete recovery. He was managed conservatively.

DISCUSSION

INCIDENCE. The prospective study showed that subarachnoid haemorrhage formed 0.66 percent of patients in medical wards which gave the incidence as 6.6 per 1000. Aneurysmal subarachnoid haemorrhage formed 0.2 percent of all medical wards admission and an incidence of 2 per 1000 patients. Intracerebral haemorrhage formed 0.11 percent and incidence of 1.14 per 1000, arteriovenous malformation formed 0.02 percent and incidence of 0.23 per 1000 while normal angiographic findings subarachnoid haemorrhage formed 0.27 percent and incidence of 2.7 per 1000.

Subarachnoid haemorrhage formed 27.4 percent of all cerebral vascular diseases in medical wards. Aneurysms specifically formed 8.5 percent, intracerebral haemorrhage 4.7, and arteriovenous malformation about 1 percent. Patients with normal angiographic findings constituted 11.3 percent. Crowell et al (5) in review of literature quoted subarachnoid haemorrhage as representing about 8 percent of all cases of cerebrovascular disease. They also reported the overall prevalence of cerebral aneurysms in general population as 9.6 per 100 thousands. Bartlett (12) stated that a typical neurosurgical unit serving a population of 2 million will admit 120 patients with aneurysms and 12 with arteriovenous malformation each year.

MORTALITY 9 out of 29 patients with subarachnoid haemorrhage died giving case mortality of 31%. 8 patients (89%) died of the first bleed and one patient (11%) died of rebleed.

Mckissock et al (13) found mortality of 50 percent in their medically treated group of patients while they found mortality of 30 percent in their sugically treated patients. Logue et al (6) found a mortality of 44.4 percent in their medically treated patients. 6 percent of their death was due to initial haemorrhage. However mortality due to rebleeding in their study was 88 percent. Mckissock et al (13) mentioned that in very serious patients the natural death rate can be over 90 percent. They quoted surgical mortality as ranging from 50 percent to 3.6 percent and commented that if surgery were to reduce this natural death rate of 90 percent even to 50 percent mortality, it would be acceptable.

Levy et al (46) in review of literature mentioned that before surgical treatment was available 65 percent of the patients with raptured aneurysms died, 43 percent from initial haemorrhage and 22 percent from recurrent bleeding during following year.

The mortality in this study appear to be less than the generally reported mortality of the medically (conservatively) treated patients. The possible explanation could be, small number of patients studied, short follow up period, the large number of patients having normal angiographic findings who are known to have good prognosis and perhaps use of

propranolol in some of the patients who were observed to have better recovery than the control group.

Four of the patients (44.4 percent) died in the first 72 hours, seven patients (78 percent) were dead in the first week and two patients (22 percent) died after second week but before end of four weeks. This shows that the highest mortality of patients with subarachnoid haemorrhage is in the first week. This agrees with Mckissock's findings (13).

RECURRENT BLEEDING

Two patients had recurrent bleeding proved by Lumbar puncture in one and the other at autopsy. One of them had left middle cerebral artery aneurysm and the other right posterior communicating artery aneurysm. One of them died as a result of rebleed while the other was disabled. This gave percentage of rebleeding as 7 percent of the total patients with subarachnoid haemorrhage. For the group with aneurysms, rebleeding was 22 percent. The rebleeding occurred on 26th and 28th day after the first bleed in the two patients. Recurrent bleeding is said to reach its peak in the second week (6,13). A smaller risk is found in the third week, with a secondary peak in the sixth week and a recurrence of up to 80 percent has been reported at the end of eighth week (6,13). In patients with multiple aneurysms, the risk of rupture of second aneurysm has been reported as 80 percent in one series (14).

Rebleeding has also been found to occur up to 21 years after initial subarachnoid haemorrhage (47). A mortality of up to 90% has been reported following rebleeding while survivors are found to die at a rate of 3.3 percent per year.

Rebleeding in patients with arteriovenous malformations has been reported to be between 3.7 and 6.25 percent per year. The mortality due to rebleeding has been given as between 0.9 and 1.6 percent per year (54).

Patients with normal angiographic findings have relatively low rebleeding rate. It has been given as 7 percent and mortality due to rebleeding 1.5 percent in one series (1).

The trend of results in this study seem to differ from the common observation but the number of patients is too small and follow up period too short for any scientific conclusion to be drawn.

TRIBAL DISTRIBUTION

As is common with most studies in this hospital, Kikuyu tribe had the largest number of patients in both prospective and retrospective studies. This has been attributed by several people to the closeness of Kenyatta National Hospital to the geographical residence of the Kikuyu tribe. When the tribal distribution in this study based on Kenyatta National Hospital admission rates was tested using chi-square test (χ^2 (6) =9.15 , $P>0.1$), the difference was not statistically significant.

AGE AND SEX DISTRIBUTION

There was only one patient below age of 20 years in the combined prospective and retrospective studies while one patient was seen in each study in 90 - 99 age group. The majority of the patients were found in 20-50 age group which formed over 60 percent of patients with subarachnoid haemorrhage in both prospective and retrospective studies. The mean age for males was about 48 years and females 37 years in the prospective study. This showed a slight tendency for men to be older ($P = .13$) but not significantly so. Magee et al (3) found that incidence of subarachnoid haemorrhage was low under age of 20 years but thereafter it rises. Crowell et al (5) stated that the prevalence of aneurysms is highly correlated with age with peak incidence in the sixth decade of life. They also stated that aneurysms are found in all decades after the second but were rare in childhood and adolescence. Shephard et al (1) in their series of 1180 patients with spontaneous subarachnoid haemorrhage found mean age to be 47 years.

Males were more than females in this study giving male to female ratio as 2.6:1(21:8). The retrospective study male to female ratio was 2.5:1 (57:23). During the period of prospective study, 2528 males and 1865 females were admitted and treated in adult medical wards

giving male to female ratio as 1.4:1. This shows that the largest number of the affected males cannot be explained by higher male admission rate. When male-female difference was tested using conditional chi-square test (χ^2, =2.61 and P=.11) the difference was not significant but this could be due to the small number of patients in this study. The trend of more male involvement than females in this study differs from what is reported by several authors from Western countries. Crowell et al (5) indicated that a slight tendency towards female preponderance is characteristic. Bull et al (4) reported work by Mckissock of 1769 ruptured intracranial aneurysms as showing female-male ratio of 3:2. Walter et al (31) in their work on beneficial effects of adrenergic blockade in patients with subarachnoid haemorrhage had male to female ratio of 1:1.7 in whole group of their patients and 1:2.1 ratio in the group with aneurysms. Taha et al (20) in their study on relation of smoking and subarachnoid haemorrhage had male: female ratio of 1:2. In present study the more male involvement is reflected in various categoriesof subarachnoid haemorrhage. In normal angiographic finding subarachnoid haemorrhage male to female ratio was 2:1 (8:4) and the aneurysmal subarachnoid haemorrhage male to female ratio also 2:1 (6:3). Intracerebral haemorrhage giving subarachnoid leakage was found in 5 men but no females while arteriovenous malformation causing subarachnoid haemorrhage was found in only one male but no female. The same trend is also

shown in retrospective study. However the number in this study is too small to compare with some of the quoted numbers by the above authors. Although the number in this study is small, perhaps the difference shown especially in sex distribution may be attributed to the difference in exposure of some of the commonly quoted risk factors. Oral contraceptives and cigarette smoking appear to have synergistic effect in subarachnoid haemorrhage. As indicated by Petitti et al (18) females who had used oral contraceptive and were smokers had risk of developing subarachnoid haemorrhage of 22 times that of females who neither smoked nor used contraceptives while smokers alone had a risk of 5.7 times and oral contraceptive users a risk of 6.5 times. It is easy to imagine and perhaps this may be true that in western countries where a lot of literature about this subject comes from have more females using oral contraceptives, smoking and consuming alcohol than our country hence more females with subarachnoid haemorrhage than males. In the prospective part of this study there was not a single female who had used oral contraceptives but there was one female a heavy smoker and alcoholic who had aneurysmal subarachnoid haemorrhage. In retrospective study there was only one female who had used contraceptive pill for about 10 years and had normal angiographic subarachnoid haemorrhage.

Contrary to western countries, in our set up, more men than women smoke, consume alcohol and are exposed to trauma hence this may explain why more men present with subarachnoid haemorrhage than women. In this study, alcohol was the leading associated factor, followed by hypertension then smoking. Alcohol and smoking combined in the same patient were associated with largest number of abnormalities (three aneurysms and one arteriovenous malformation).

OCCUPATION DISTRIBUTION

The largest number of patients which formed 24 percent were unemployed. This was followed by business as the leading occupation and then house wifely. The unemployed group being the largest may be a reflection of the large number of unemployed people in the society. On the other hand, the three groups of people may have high incidence of psychological stress. Psychological stress can cause temporary or sustained rise in blood pressure and rupture as well as growth of aneurysm is dependent on water hammer effect. In this study, two patients developed subarachnoid haemorrhage while emotionally upset. One was annoyed by friends while the other one had just returned from a funeral. Magee et al (3) found that 90 percent of his patients did not have any physical stress and were going on with their normal duties when struck. 43 of

his patients were either in bed, rising from bed or at home or office. 10 of his patients were either walking or dancing. These observation lead to his statement that between rest and effort, the former (rest) deserves the greater share of blame. While most of patients may be at physical rest, they may not be at mental rest and may be infact be very much stressed. In this study four patients who were unemployed had aneurysms, and a 5th one had intracerebral bleed and the remaining two had normal angiographic findings. Although psychological stress has not been emphasized as physical strain it may be contributing in a good number of patients. This is however very difficult to measure.

DISTRIBUTION OF ASSOCIATED FACTORS

Alcohol was the leading factor of association. It was found in 10 of the 29 patients which was 35% of the total number of patients. Nine patients (90 percent) of those with alcohol association were males and only one patient (10 percent) was a female. This may be a reflection of proportion of alcohol drinking male/and females in our surrounding. Alcohol has been associated with haemorrhagic stroke and recently it has been associated with development of spontaneous subarachnoid haemorrhage upto 24 hours after acute alcoholic intoxication. Alcohol causes thrombocytopenia, hypertension, irreversible vasospasm and even vessel rupture (21,22). Although alcohol has not featured prominently in the past as an important association with subarachnoid haemorrhage the

observation in this study suggest some blame on alcohol. These patients in the study associated with alcohol were struck while drinking, while in bed or when rising from bed within 24 hours after intoxication and most of them consumed over 5 bottles of beer daily. However the number is too small for any conclusion to be drawn.

Hypertension was second in the list of association with seven patients (24 percent). This is a well known risk factor for stroke. In subarachnoid haemorrhage, it causes intracerebral bleed with subarachnoid space leakage of blood in about 75 percent of cases (16). Davis (55) has stated that two thirds of ruptured aneurysms occur in females and half of these are hypertensive. In this study five of the seven patients were males four of whom died and had intracerebral bleed on postmortem. Hypertension causes arterial fibrinoid necrosis and arteries so damaged form miliary aneurysms which are ruptured by raised intracerebral pressure (16,17).

Smoking was the third single factor of association with 5 patients out of 29, giving 17 percent. Four of these patients (80 percent) were males and one (20 percent) was a female. Smoking has been stressed by a number of authors as a risk factor associated with subarachnoid haemorrhage (18,20).

Emotional upset was associated with two patients. Physical strain such as coitus, defecating, digging and jogging were associated with one patient. each giving a total of 14 percent. This suggests the importance of physical strain as a risk factor in subarachnoid haemorrhage as is usually stressed in books. The observations here also suggest that psychological stress may have some part to play as a risk factor in subarachnoid haemorrhage. Magee et al (3) suggest that rest may be more to blame than physical strain in causing subarachnoid haemorrhage.

ASSOCIATIONS AND INTRACRANIAL VASCULAR ABNORMALITIES

Alcohol and smoking combined were associated with the largest number of abnormalities than any other single or combined factors. They were associated with 26.7 percent of the abnormalities (4 out of 15). Twenty percent of these abnormalities were made of aneurysms and 6.7 percent made of arteriovenous malformation. The two factors were associated with 33 percent of total number of aneurysms. Perhaps the two factors may have synergistic contribution towards spontaneous subarachnoid haemorrhage.

Alcohol alone was associated with 33.3 percent of the total abnormalities found (5 out of 15).

Twenty percent of these was made up of aneurysms while 13.3 percent was made up of intracerebral haematoma. These observations still emphasize the importance of these associated factors although mere chance cannot be ruled out.

Hypertension was associated with 26.7 percent of the total abnormalities all of which were intracerebral haematomas. Of all intracerebral haematomas 80 percent (4 out of 5) were associated with hypertension and all died. This is in keeping with what is commonly known about hypertension that it causes intracerebral bleed which is associated with poor prognosis.

The other abnormalities consisted of six aneurysms and one intracerebral haematoma. Two of the aneurysms which formed 13 percent of total abnormalities found and 22 percent of total aneurysms found, ruptured when the patients were emotionally upset. Four remaining aneurysms 26.7 percent of total abnormality and 44.4 percent of aneurysms ruptured while patients were having some physical strainnamely coitus, wiping the floor, jogging and walking. This points to the association of aneurysm rupture and physical strain.

DISTRIBUTION OF INTRACRANIAL VASCULAR ABNORMALITIES IN ANATOMICAL SITES AND SEX.

In the prospective study abnormalities were found in fifteen patients of the twenty seven patients investigated. These consisted of nine aneurysms, five intracerebral haematomas and one arteriovenous malforma-

tion. In retrospective study, seventeen abnormalities were detected out of fifty six patients investigated consisting of eleven aneurysms, three intracerebral haematomas, two arteriovenous malformation and one Moya-Moya disease.

All intracerebral haematomas in both prospective and retrospective studies were in males. For aneurysms, male to female ratio was 2:1 and for arteriovenous malformation in both studies combined male to female ratio was also 2:1.

In the prospective study, majority of the abnormalities were on the left side. Five (62.5%) of the aneurysm were on the left side and three (37.5%) on the right side. Four (80%) of intracerebral bleed were on the left side and one (20%) was on the right side. Magee et al (3) stated that the incidence of aneurysms on the right side of circle of Willis is twice as much as on the left side. They also stated that this incidence is the reverse of the common experience in intracerebral haemorrhage of other varieties. The results in this study differ from the findings of Magee and his associates.

Posterior communicating artery aneurysms was the commonest. There were 4 (44%) out of 9 aneurysms in prospective study and 5(45%) out of 11 aneurysms

in retrospective study. These results differ from what is reported by several authors elsewhere as they give anterior cerebral and anterior communicating arteries combined as the leading site of aneurysms. Bull et al (2) gave 30 percent for anterior cerebral and anterior communicating artery aneurysms and 25 percent for posterior communicating artery aneurysms. Magee et al (3), Mckissock et al (13), Adams et al (47) and Barton et al (53) have all given anterior cerebral and anterior communicating artery aneurysms as the leading site and have found about 34.9%, 28.3%, 24% and 26.9% respectively. The present study results are compared with those of Magee, Mckissock, Adam and Barton in tables 8 and 9 . The statistical result show no correlation between results in this study and those of Magee and Barton but show satisfactory correlation with the findings of Mckissock and Adam.

INTRACRANIAL VASCULAR ABNORMALITIES AND OUTCOME

The results of both prospective and retrospective studies showed similar trend, with best outcome in normal angiographic finding, followed by ruptured aneurysm and finally intracerebral haemorrhage.

In prospective study, normal angiographic findings good outcome (complete recovery plus partial recovery) was found in 83 percent of patients in this group and poor outcome (disabled plus death) 17 percent. In aneurysmal group, good outcome was found in 67

percent and poor outcome in 33 percent. The group with intracerebral haemorrhage had 100 percent mortality. The only one patient with arteriovenous malformation had complete recovery.

In retrospective study, normal angiographic findings subarachnoid haemorrhage good outcome was 87 percent and poor outcome 13 percent. Aneurysmal group of subarachnoid haemorrhage good outcome was 73 percent and poor outcome 27 percent. The group with intracerebral haemorrhage good outcome was 67 percent and poor outcome was 33 percent. While the trend was maintained, the outcome in the intracerebral haemorrhage was better in retrospective than prospective. Two patients with arteriovenous malformation one was disabled while the other had partial recovery which gave 50 percent good outcome and 50 percent poor outcome. The only one patient with Moya Moya disease had partial recovery.

Normal angiographic findings subarachnoid haemorrhage is generally said to have best prognosis and Shephard et al (1) have recently confirmed this in the large series of 1180 patients followed over 22 years. Intracerebral haemorrhage on the other hand is said to have worst prognosis among the three lesions sometimes warranting emergency evacuation of the haematoma in trying to improve the prognosis (5,56). The results in this study therefore agree with general finding

but the number here is very small and follow up period short.

Arteriovenous malformation/s are said to have better prognosis than aneurysms. However in the present study there were only three arteriovenous malformations in both prospective and retrospective group. One had complete recovery, the other had partial recovery and one was disabled. This gave good outcome as 67 percent and poor outcome as 33 percent. On the other hand there were a total of 20 aneurysms in both groups. Four died, two were disabled, nine had partial recovery while five had complete recovery. This gave good outcome as 70 percent and poor outcome as 30 percent. The number of arteriovenous malformations in this study is too small compared with the number of aneurysms, that it is difficult to say whether this general observation about the two lesions is the same here or not.

When the results in prospective study as shown in table 13 were tested using conditional chi-square test (X^2 3=13.76 and $P < .005$), intracerebral haemorrhage group of abnormality had significantly poorer outcome compared with others. The others did not differ statistically. Retrospective study results as shown in table 28, were tested using chi-square test (X^2 6=6.15 and $P = .50$). There was no statistical difference observed

between abnormalities as regards the outcome. However when normal angiographic findings were compared with others and tested with ($P < 0.05$ and $X^2_1 = 5$) there was a significant good outcome. The recovery rate in normal angiographic findings was 79% and in others combined 57%.

AGE AND OUTCOME

The results in both prospective and retrospective group in tables 14 and 29 respectively showed a trend that the younger the age group the better the outcome. In prospective study, the age groups 20-39 years had good outcome (partial and complete recovery) in 75% of the patients and poor outcome (death and disabled) in 25% of the patients. The age groups above 40 years had good outcome in less than 50% of the patients. The mean age for those with complete recovery was lower than those who died (42 and 52 respectively). When those who lived were compared with those who died and tested using conditional chi-square test ($X^2_1 = 1.755$ and $P > 0.1$) this was not statistically significant.

In retrospective study, the age groups 20 - 49 years had good outcome in 75% and poor outcome in 25%. The age groups above 50 years had good outcome in 53% and below. The greater chances for recovery were in the age group 30 - 49 years on statistical

testing (X^2_3 = 9.81 and P=.02). The other age groups did not show significant differences. The mean ages for those who had complete recovery and those who died were 42 and 52 years respectively. When those who lived were compared with those who died and tested using chi-square test (X^2_1 =4.534 and $P < 0.05$), this was found to be significant at 5 percent level. Magee et al (3) found that there was age influence on recurrence of bleeding, death, good functional recovery and recovery with such complications as hemiplegia, vertigo, fits and diplopia. Best recovery was in 21 - 30 age group in his series and above 40 years good recovery was rare.

SEX AND OUTCOME

The results in both prospective and retrospective studies showed there was no difference in outcome over the sexes.

ELECTRO-CARDIOGRAPHIC CHANGES IN SUBARACHNOID HAEMORRHAGE

Electrocardiographic abnormal findings were observed in 73 percent of all the patients where this investigation was carried. T wave changes were the leading abnormality and included flat T wave which was the most frequent abnormality followed by tall T wave and then inverted T wave. Depressed ST segment

and pathological Q wave followed the T wave changes in the same frequency, while ventricular hypertrophy, arrhythmia, prolonged P-R and shorten P-R interval ranked third and occurred with same frequency. Electrocardiographic abnormalities have been reported in literature as ranging from 50 to 80 percent and the abnormalities found here are within the reported range (5,8-11).

The changes occurred in 69 percent of the control group and 78 percent of the propranolol group. The changes were still present after 10 days of treatment in both groups. Findings in this study indicate that ECG changes occur quite early and persist for sometime. This agrees with what has been reported in the literature that the ECG changes as well as myocardial necrosis have occurred as early as 17 hours after subarachnoid haemorrhage and they can persist upto 6 weeks. The occurrence and persistent ECG changes in patients on propranolol suggests that propranolol may not have been effective. Abnormal hypothalamic response with increased catecholamine production via symphathetic system and increased circulating steroid level via the pituitary - adrenal axis has been suggested as the cause of ECG changes as well as myocardial damage by those who have shown propranolol to be protective (30,31

All patients in this study had normal catecholamine and steroid levels as shown by estimation of urinary catecholamine, vanillyl mandelic acid, 17-ketosteroid and 17-hydroxysteroid in 24 hour urine specimen. This suggests why propranolol may not have worked in these patients. Propranolol is said to be less effective in hypertensive African patients (57). It may not be surprising then if it did not work in this study as all patients were Africans.

Other mechanisms causing ECG changes and myocardial injury in which propranolol has no control may have been functioning in these patients. Low serum potassium is one of the suggested mechanisms but all these patients had normal potassium, sodium, calcium and phosphate as well as urea. This leaves locally released norepinephrine metabolic effect on myocardium and irritation of area 13 on the orbital surfaces of the frontal lobes which is thought to be the cortical representation of vagus nerve as possible mechanism which might have caused ECG changes in these patients (5,8 - 11). In addition to these postulated mechanisms, perhaps other mechanisms which have not come to light may also be acting.

RECOVERY PERIOD AND OUTCOME IN BOTH PROPRANOLOL AND CONTROL GROUPS.

The mean recovery period for the control group

was 13 days and 8 days for the propranolol. When the results were tested using conditional chi-square test (X_1^2=2.763 and P value<0.05) the difference in the recovery period was significant at 5 percent level.

Good outcome (complete recovery plus partial recovery) was found in 80 percent of patients in propranolol group while it was 64 percent in the control group. Poor outcome (Death plus disabled) was 20 percent in propranolol group and 36 percent in control group.

For the normal angiographic group of patients, good outcome was 86 percent in propranolol and 60 percent in control. Poor outcome was 14 percent in propranolol and 40 percent in control. This group of patients has however best outcome whether on propranolol or not.

For the aneurysmal group of patients, three were in propranolol and 2 (66.7%) had complete recovery and 1 (33.3%) died. Six patients were in control and 4(66.7%) had partial recovery, 1(16.7%) had complete recovery and 1(16.7%) was disabled.

There were two patients with intracerebral haemorrhage in control and both died, none in propranolol. There was one patient with arteriovenous malformation in control and had complete recovery.

The overall result show a trend of better outcome in propranolol group even when individual lesions in the two groups are compared. This however is not statistically significant ($P > 0.5$).

CHOLESTEROL LEVELS IN SUBARACHNOID HAEMORRHAGE

Cholesterol levels were done in 26 patients in an attempt to find if there is any correlation between serum cholesterol levels and subarachnoid haemorrhage. This was done having in mind the fact that atherosclerotic aneurysms rank second to berry aneurysms and that 80% of subarachnoid haemorrhage is due to aneurysms (4,6). The cholesterol levels ranged from 2.24 to 7.5 mmols per litre and mean was 4.5 mmols per litre. Most of these agree with levels given by Ojwang et al (58). Three patients had levels on the higher side. One patient a 70 year old man with normal angiographic subarachnoid haemorrhage had serum cholesterol of 6.3 mmols. The second patient was a 32 year old man who had left posterior communicating artery aneurysm, had cholesterol level of 7.1 mmols. This level was above the normal range of 3.1 to 6.47 mmols/litre. The highest level of 7.5 mmols/litre was in 50 year old business man, alcoholic and hypertensive who on postmortem was found to have atheromatous plaques involving aorta and cerebral vessels starting from circle of willis. Another patient whose cholesterol levels were not done was a 46 year old man hypertensive who on postmortem had

atheromatous plaque on aorta and cerebral vessels and left circumflex artery thrombus. Both these patients with atheroma had left intracerebral haemorrhage with subarachnoid leakage and died within 48 hours. The number of patients with high level cholesterol and subarachnpid are very few and there was only one with subarachnoid haemorrhage, high cholesterol level and atheromatous plaque on postmortem. It is difficult to say from these results whether there is any correlation between cholesterol and subarachnoid haemorrhage. Even if there were any correlation of cholesterol levels and atherosclerotic aneurysms they are quite rare, and 50 times less when compared with berry aneurysm (4).

17-KETOSTEROID, 17-HYDROXYSTEROID, VANILLYL MANDELIC ACID AND CATECHOLAMINES IN SUBARACHNOID HAEMORRHAGE.

High levels of catecholamines and adrenergic steroids are reported by some authors to follow subarachnoid haemorrhage. Catecholamines have been associated with cerebral vasospasm and hence neurological deterioration while catecholamines together with steroids have been blamed on myocardial injury and ECG changes (8). This understanding have led some authors to use propranolol to combat the catecholamine effects and reports of success in this have been published (30,31).

The levels in this study were normal unlike what is in literature. The number of patients in this study (24 patients) is too small and perhaps one needs a large number to verify the truth about this. Alternatively, the African patients may be behaving differently compared to caucasians whose findings have been reported.

SUBDURAL HAEMATOMA IN ASSOCIATION WITH SUBARACHNOID HAEMORRHAGE

Subdural haematoma in association with subarachnoid haemorrhage especially following intracranial aneurysm has been reported (53). In this study both prospective and retrospective combined, there was only one patient in retrospective study who was found at postmortem to have right intracerebral haemorrhage and bilateral subdural haematoma. Considering the total number of investigated patients in both prospective and retrospective groups were 83, this finding gave proportion of 1.2 percent. Barton et al (53) found eleven cases of subdural haematoma in association with intracranial aneurysms in a series of 839 angiogram over 20 years. This they said represented

an incidence of 1.3%. The finding in this study agrees with the finding of Barton and associates. In literature review they quoted the incidence of this association as ranging from 0.5% to 8%. According to their review, the association is most common with internal carotid artery, then middle cerebral artery, then anterior communicating and very rarely vertebrobasilar artery aneurysms.

EXTRACRANIAL ASSOCIATIONS OF ANEURYSMAL SUBARACHNOID HAEMORRHAGE.

Intracranial aneurysms are associated with various abnormalities. Polycystic kidney association has been reported to range from 6 - 30 percent, coarctation of aorta 20 percent and fibromascular hyperplasia of renal arteries in 50 percent in one series (2,46). In prospective study intravenous urography in 20 patients did not reveal any of these abnormalities. For those who had postmortem done on them, only one patient 90 year old man had an isolated renal cyst in the cortex of the left kidney but the kidneys were of normal size. The patient had left intracerebral haemorrhage. The results suggest that these associations are rare. If this single cyst is taken as an association other than an incidental finding it gives incidence of 3.7 percent.

MANAGEMENT

The prospective group of this study had 9 patients with aneurysms, 5 patients with intracerebral haematoma, 1 patient with arteriovenous malformation, 12 patients with normal angiographic findings and two patients who were not investigated. Only one patient in this group was operated. This was a 27 year old man who was an alcoholic, heavy smoker and normotensive whose angiography showed left posterior communicating artery aneurysm. The aneurysm was clipped on 36th day after bleed. He recovered well and was discharged walking without support, fully conscious and well orientated but with ptosis on the left eye and reduced vision on the same eye. This patient also received medical management also but not propranolol. The other 28 patients received medical treatment only, which consisted of bedrest, diazepam 5mg twice a day, laxatives, dexamethazone 4mg 8 hourly, antihypertensives where necessary and physiotherapy. Propranolol was given to some patients.

The retrospective group had 11 patients with aneurysms, 3 with intracerebral haematoma, 2 with arteriovenous malformations, 1 with Moya Moya disease, 39 with normal angiographic findings and 24 patients were not investigated. Only 5 patients in this group were operated. Three of these patients had posterior communicating artery aneurysms which were clipped,

one had internal carotid artery aneurysm and had internal carotid ligation while the fifth patient had arteriovenous malformation and was cauterized. All these patients had partial recovery. Good outcome in this operated group was 100 percent. Seven patients with aneurysms received medical management only. Three patients (one with posterior communicating artery aneurysm, anterior cerebral artery aneurysm and the other carotid artery aneurysm) died. This gave a mortality of 43 percent. Two patients with anterior communicating artery aneurysms had complete recovery and two others, one with posterior communicating artery aneurysm and the other with carotid artery aneurysm had partial recovery. This gave good outcome in this medically treated group as 47 percent. The other patients in retrospective group had medical treatment only.

Patients with aneurysms and arteriovenous malformation require surgery as their definitive management. The patients with intracerebral haematoma sometimes requires emergency operation and evacuation of the haematoma when the clinical state shows deterioration (5). The surgical approach for aneurysms include microsurgical obliteration of aneurysms using various clips, and stereotactic obliteration of aneurysms by introducing fine thrombogenic wire or ferromagnetic particles into aneurysms with local magnetic attraction to hold the

thrombogenic particles within aneurysm Lumen (5). Arteriovenous malformation requires total surgical excision or embolization. Where the patient is not fit for the conventional therapy, stereotactic bragg-peak proton beam is used (12,42,43).

Patients with normal angiographic findings do not require surgery. These are given medical treatment in the acute state and have best prognosis.

Comparison of both medical and surgical management of patients with subarachnoid haemorrhage have shown that surgical management offers a definite lowering of mortality by preventing recurrent haemorrhage (6, 13,45).

In the present study, there were 23 patients (20 patients with aneurysms and 3 with arteriovenous malformation)with definite indications for surgery and 8 patients (with intracerebral haematoma) with relative indication. Out of these, only 6 patients were operated. The aneurysm clips as well as clip holding forceps were not available during the prospective part of this study and therefore, operation was not possible.

CONCLUSIONS.

1. Subarachnoid haemorrhage peak incidence is between 20 and 50 years. There is slight tendency for males to be slightly older. The younger the patient the better the outcome.

2. More males were involved than women at a rate that cannot be explained by just higher male admission rate. This is unlike the reports from western countries where females are said to be more involved than males. There was no difference in outcome between sexes.

3. Alcohol was the leading associated factor followed by hypertension and then smoking. Alcohol and smoking combined were associated with the largest number of intracranial vascular abnormalities found, in particular the aneurysms.

4. The largest number of patients with subarachnoid haemorrhage was associated with normal angiographic findings. This formed 44 percent in prospective group, 70 percent in retrospective group and 61 percent when the two groups are combined. Second largest group had ruptured intracranial aneurysms. This accounted for 33 percent in prospective study, 20 percent in retrospective and 24 percent when the two were combined. Intracerebral haemorrhage

was third and accounted for 19 percent in prospective, 5 percent in retrospective and 10 percent when combined. Arteriovenous malformation came last and formed 4 percent in prospective, 3 percent in retrospective and 4 percent when combined.

Normal angiographic findings had best prognosis followed by aneurysms group. Intracerebral haemorrhage had worst prognosis compared with others.

5. The commonest site of the aneurysms at Kenyatta National Hospital is posterior communicating artery (45%), followed by anterior cerebral and anterior communicating artery (25%) then carotid artery (15%), middle cerebral artery (10%) and finally basilar artery (5%).

6. Electrocardiographic changes were observed in 73% patients with subarachnoid haemorrhage.

7. Patients on propranolol showed a trend of shorter recovery period compared with control. There was also a trend of better outcome in propranolol group compared with control.

8. 17-ketosteroid, 17-hydroxysteroid, vanillyl mandelic acid and catecholamines were normal in all the patients.

9. Renal abnormalities associated with subarachnoid haemorrhage and in particular intracranial aneurysms are rare unlike what is reported by some authors.

10. Association of subdural haematoma with subarachnoid haemorrhage is extremely rare. This is in keeping with what is reported in literature.

RECOMMENDATIONS

1. Follow up study of these patients to find out the natural history of subarachnoid haemorrhage in our set up.

2. Further trial of propranolol preferably on a large group.

3. 28 patients in prospective and 75 in retrospective studies were treated conservatively. I would therefore recommend that various drugs which can offer some help in these patients such as anti-fibrinolytics, and calcium antagonists be looked into to establish how useful they would be in our set up.

ACKNOWLEDGEMENTS

I wish to express my heartfelt gratitude to the following without whose help, this Thesis would not have been possible.

1. Dr. G.R. GANAPATHY my supervisor for his tireless guidance and advice throughout the course of this Thesis.
2. DR. KITONYI of Department of radiology for performing as well as reporting on the angiograms on patients in prospective study.
3. Consultants, senior house officers and House Officers, for allowing me to have access to patients in their wards.
4. All Doctors who participated in neuroradiological meetings during which the angiograms in prospective study were discussed.
5. Nurses in medical wards for their co-operation especially during the carotid and branchial angiography as well as during 24 hour urine specimen collection.
6. Mr. Nderitu of department of Medicine for Laboratory analysis.
7. Medical records staff for their assistance with files in retrospective study and calculating the total admissions in medical wards.

8. Mr. Gemert and Mr. Lawrence Ndungu both of Kenya Medical Research Institute for statistical analysis.

9. Mr. Caleb Oloo of Radiology department for excellent photography.

10. Mrs. Margaret Kamau of department of Medicine for typing this work.

11. My Sponsor Ministry of Health.

12. Finally I would like to thank my family and especially my wife Christine Njeri for her tireless encouragement and great understanding despite my absence from home.

APPENDIX I

SPONTANEOUS SUB-ARACHNOID HAEMORRHAGE STUDY

PROFORMA FORM

NAME ---------- AGE ---------- SEX ------

ADDRESS ---------- CHIEF ---------- TEL ------

IP NO ---------- WARD ---------- BED ------

OCCUPATION ---------- TRIBE ----------

MARRIED ---------- SINGLE ---------- DIVORCED ------

WIDOW ---------- WIDOWER ---------

ADMISSION ---------- DISCHARGE --------

HISTORY

1. Present complaint

2. HISTORY OF PRESENT ILLNESS

3. PAST MEDICAL HISTORY

APPENDIX 2

4. OBSTETRIC AND GYNAECOLOGICAL HISTORY

5. FAMILY HISTORY

6. SOCIAL HISTORY	Duration	Frequency	Quantity
(a) Alcohol Drinking	--------	---------	---------
(b) Smoking	--------	---------	---------

7. SYSTEMIC ENQUIRY

APPENDIX 3

SPECIFIC INFORMATION TO LOOK FOR IN ENQUIRY

1. Abrupt onset of severe Headache
2. Nausea and vomiting
3. Neck pain and Backache
4. Convulsions
5. Unconsciousness
6. Vertigo
7. Diplopia
8. Ptosis
9. Disturbed vision
10. Dysarthria
11. Ataxia
12. Inability to talk
13. Inability to walk
14. Left sided weakness and disturbed sensation
15. Right sided weakness and disturbed sensation
16. Weakness of one limb indicate
17. Any other to be specified.

ACTIVITY PRECEDING ONSET OF ILLNESS

1. Strenous exercise specify
2. Rest
3. Emotional disturbance - Happiness
   - Sadness.
4. Strenous work - specify
5. Smoking
6. Alcohol Ingestion

APPENDIX 4

7. Epileptic fit
8. Any other.

PRESENT ASSOCIATED DISEASES.

Disease	Duration	Treatment if any
1. Diabetes		
2. Hypertension		
3. Epilepsy		
4. Bleeding disorder		
5. Pregnancy		
6. Hormonal contraceptives		
7. Any other.		

HISTORY OF DRUGS

Drugs	Duration	Amount per day
1. Anticoagulants		
2. Steroids		
3. Hormonal contraceptives		
4. Alcohol		
5. Smoking		
6. Salicy lates		
7. Any other.		

PAST MEDICAL HISTORY

1. Migraine Headache with vomiting
2. Trauma on the Head

APPENDIX 5

3. Hospitalisation
4. Operations
5. Any other.

FAMILY HISTORY

This will refer to parents, grandparents, brothers, sisters, uncles and aunts.

1. Similar illness
2. Migraine headache
3. Diabetes
4. Hypertension
5. Bleeding disorder
6. Any other

PHYSICAL EXAMINATION

GENERAL

1. Pallor
2. Cyanosis
3. Oedema
4. Jaundice
5. Lymphadenopathy
6. Temperature
7. Pulse
8. Bp
9. Peripheral pulses
   a. Radial
   b. Brachial
   c. Carotid

APPENDIX 6

9. d) Femoral
   e) Popliteal
   f) Posterior tibial
   g) Dorsalis pedis.

10. Bruit
   a) Carotids
   b) Orbits.

CENTRAL NERVOUS SYSTEM

1. SKULL

(i) Size
(ii) Symmetry
(iii) Evidence of trauma
(iv) Deformity if any
(v) Bruit (a) Orbits
(b) Temporal
(c) Parietal

2. SPINE

(i) Kyphosis
(ii) Scoliosis
(iii) Lordosis
(iv) Any other deformity.

3. MENTAL FUNCTION

(a) Conscious level
(i) Fully conscious
(ii) Orientation

APPENDIX 7

3. (iii) Intelligence
(iv) Memory
(v) Speech

(b) Conscious but drowsy
(c) Unconsciousness.

4. CRANIAL NERVOUS LESSIONS	Right side	Left side
I. Sence of smell	----------	----------
II. (a) Visual Acuity	----------	----------
(b) Field defect	----------	----------
(c) Colour vision	----------	----------
(d) Fundal examination	----------	----------
III, IV, VI,		
(a) Ptosis	----------	----------
(b) Diplopia	----------	----------
(c) Pupils	----------	----------
V, - Sensation on the face	----------	----------
- Masseter muscles	----------	----------
VII Facial Expression	----------	----------
VIII Hearing - Weber's test	----------	----------
- Rinne's test	----------	----------

IX and X

(a) - Gag reflex	--------	--------
- Soft palate movements	--------	--------
- uvular movements	--------	--------
- sternomastoid and trapezins muscle	---	--------

APPENDIX 8

XII (a) Tongue movement ---------- ----------

(b) Tongue wasting ---------- ----------

5. Signs of maningeal irriration

(i) Neck stiffness ---------- ----------

(ii) Kernig's sign ---------- ----------

(iii) Straight leg raising ---------- ----------

6. MOTOR FUNCTIONS	Right side	Left side
(i) Bulk of muscle	----------	----------
(a) Normal	----------	----------
(b) Wasted	----------	----------

II) Tone of muscle.

(a) Normal	----------	----------
(b) Reduced	----------	----------
(c) Increased	----------	----------

III) POWER

GRADE 0

1

2

3

4

5

IV) COORDINATION

(a) Present	----------	----------
(b) Lost	----------	----------

V) REFLEXES

APPENDIX

	BJ	SJ	TJ	KJ	AJ	PR	ABDR
RIGHT							
LEFT							

Key

- absence
+ reduced
++ Normal
+++ increased reflexes
++++ Increased reflex and clonus.

VI Romberg's sign - where possible

VII Gait - where possible

VIII Involuntary movement

(a) Present

(b) Absent.

7. SENSORY FUNCTION

(a) Light touch

(b) Pain

(c) Temperature

(d) Position

(e) Vibration.

8. Cerebellar signs

(a) Present

(b) Absent.

RESPIRATORY SYSTEM

Examination of the chest to rule out any abnormality, dyspnea, and orthopnea.

APPENDIX 10

CARDIOVASCULAR SYSTEM

1. Radial pulse character
2. Peripheral pulses
   (a) Radial
   (b) Brachial
   (c) Carotid
   (d) Femoral
   (e) Posterior Tibial
   (f) Popliteal
   (g) Dorsalis pedis pulses.
3. BRUIT ON
   (a) Carotid
   (b) Over eye balls
   (c) Over skull

Daily Bp

Precordium

(a) Apex Beat location and character
(b) Presence of Thrill
(c) Murmur if present.

ABDOMEN

1. Tenderness
2. Ascites
3. Hepatomegaly
4. Splenomegaly
5. Palpable kidneys
6. Renal Bruit
7. Any other.

APPENDIX 11

INVESTIGATION

All the patients will be subjected to the following investigations.

1. BLOOD TESTS

(a) Complete hemogram - Hb, WBC, Platelets and ESR

(b) Coagulation screen

(c) Serum cholesterol

(d) Urea and electrolytes

(e) Serum creatinine

(f) Serum catecholamine

(h) LFT's

(i) Blood sugar fasting and postprandial.

2. Lumbar puncture and CSF for

(a) Macroscopic examination

colour - bloody

- xanthochromic

(b) Sugar and protein

(c) VDRL and kahn

(d) AFB

3. URINE

(a) Urinalysis - especially sugar and protein

(b) 24 hour urine for catecholamines and 17-ketosteroid.

4. EEG

5. ECG

6. MANTOUX test

7. Radiological tests.

APPENDIX 12

a) Skull x-ray AP, lateral

b) chest x-ray PA where possible

c) Four vessel cerebral angiography

d) IVP to rule out kidney malformation especially polycystic type.

8. POSTMORTEM

In case of death of patients and where consent from relatives can be obtained.

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