

THE PATTERN OF HEAD INJURIES AT THE KENYATTA  
NATIONAL HOSPITAL

A review of all the cases of head injuries admitted  
into the Kenyatta National Hospital between July 1st and  
December 31st 1979.

NIMROD JUNIAS MWANG'AMBEE, M.B.CH.B (NBI)

A thesis submitted as part fulfilment  
for the degree of Master of Medicine  
(Surgery) in the University of Nairobi,  
1980.

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

A handwritten signature in blue ink, appearing to read 'N.J. Mwang'ombe', with a stylized flourish at the end.

N.J. MWANG'OMBE  
CANDIDATE

This thesis has been submitted for examination with my approval.

Prof. Nelson Awori M.B.CH.B F.R.C.S.  
SUPERVISOR

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KENYATTA NATIONAL HOSPITAL

<u>CONTENTS.</u>	<u>PAGE.</u>
Acknowledgement	1
Summary	2
Historical introduction	3
Patients and methods	4
Age and sex distribution	5
The cause of injury	6
Types of instruments used in assault	7
Time interval between injury and admission	8
 <u>CLINICAL FINDINGS:</u>	
Level of consciousness, alcohol and epileptic seizures	10
Skull fractures	11
Common presentation in Patients with intracranial haematomas	12
Time interval between admission and surgery	13
Associated injuries	14
Clinical and operative findings	15
Mortality	16
Time interval between admission and death	17
Duration of hospital stay	18
Evaluation	19
Discussion	20
References	37
Appendix	39

Acknowledgement:

It is impossible in a work of this nature to single out everyone who has been instrumental to the successful completion of this thesis, but it is expected of me, as a matter of courtesy, to mention a few names. I would like to thank Ms. Margaret Cheptolo who did all the secretarial work for me with diligence, Mr. G. Sande for his suggestions in the Planning of the Study, Mr. J. Dar for accepting the heavy task of correcting the work and Prof. Ambrose Wasunna for his timely suggestions and advise.

Finally I would like to thank my supervisor Prof. Nelson Awori for his advise, constructive criticism and encouragement without which I would never have completed this thesis and for accepting the huge responsibility of endorsing this thesis. To everyone I say a big Ahsante sana.

## SUMMARY:

A review of the cases of head injuries admitted to the Kenyatta National Hospital over a period of six months between July 1st 1979 and December 31st 1979 is presented, and their patterns studied. During this period, 378 patients were admitted of which 330 were males and 48 were females. The youngest patient was two months old and the oldest, seventy nine years. 167 were involved in road traffic accidents, 146 were assaulted, 37 fell down, 5 were injured while at work and two were injured in sporting activities. Pedestrians paid a heavy toll in road traffic accidents accounting for one hundred and thirteen cases.

Seventy five patients were referred from other hospitals, provincial, District or within Nairobi.

Majority of the patients came to hospital within one day of sustaining the trauma, most of them within the first two hours. 133 patients (35.1%) were fully conscious when first seen in the ward 92 patients (24.3%) were drowsy, 56 (14.8%) confused, 42 (11.1%) stuporous, and 46 (14.7%) were comatose. 90 patients were strongly smelling of alcohol (23.3%), 66 patients (17%) had other associated injuries. The commonest associated injuries were fractures of the long bones, 37 (56%) cases, injuries to the chest, 10 (15.2%) cases, while 11 patients had more than two other associated injuries.

Two hundred and three cases had fractures of the skull out of which 149 (73.4%) were fractures of the vault, and 54 (26.6%) patients had fractures of the base. Cerebrospinal fluid otorrhea was present in 47 patients, and rhinorrhoea in 16 patients. Fourteen patients had epileptic seizures.

Thirty three patients had intra cranial haematomas, with deteriorating level of consciousness as the commonest presentation.

One hundred and eighteen patients underwent some form of surgery, twenty seven within two hours of admission, six between two and four hours, nineteen between four and six hours, twenty two between six and twelve hours, seventeen between twelve and twenty four hours and twenty seven after more than twenty four hours.

One hundred and eleven patients (30.8%) had soft tissue injuries, 207 (57.5%) had primary brain injury with no associated intracranial haematoma. Seventeen patients (4.8%) had subdural haematomas, and 16 extradural haematomas (4.6%).

Fifty nine patients died while in the wards, an overall mortality of 15.6%. most within the first twenty four hours of admission (49%). One hundred and eighteen patients underwent surgery and 14 died while in hospital during or after surgery, 11.9% operative mortality.

Most of the patients were out of hospital by the tenth day, only three patients stayed for more than one month.

The management offered was evaluated as satisfactory in most of the patients (295) and in a minority (75) the management could have been better.

In two hundred and ninety eight patients there was no contributing factor which could have worsened the head injury. In 34 the initiation of treatment was delayed and 20 patients had other injuries of significant severity.

It is hoped that the follow up of these patients will be the subject of a next study when the effects of the trauma will be assessed.

## HISTORICAL INTRODUCTION:

Guthrie (Hooper) in 1847 published his treatise on injuries of the head affecting the brain in which he remarked "Injuries of the head affecting the brain are difficult of distinction, treacherous in their course and for the most part fatal in their results". That was over a hundred years ago. The subject has changed immensely from that time, especially the understanding of the Pathophysiology of head injuries. This has had a strong influence in the managements of head injuries and the outlook is not as grim as it was during Guthrie's days.

To Guthrie and his contemporaries, sub arachnoid haemorrhage was called haemorrhagic meningitis and subdural haemorrhage, Dachs meningitis haemorrhagica interna. Management of head injuries was largely based upon the treatment accorded to apoplexy on the one hand and inflammation on the other, consisting mostly of bleeding, Purgation Cold packs to the head and blisters to the neck.

The major reason why surgical intervention was avoided because of the fear of infection, which usually resulted in a fatal outcome. With the advent of the ~~historical~~ era, Neurosurgery made a great step forward. In the 1880's Sir William Hecwen, was able to apply deductive thought combined with ~~histerian~~ principles of surgery and diagnose an intracranial clot and operate on it with complete recovery.

Harvey Cushing made the initial attempt to separate Neurosurgery from the rapidly expanding field of general surgery.

The period of World War 11 is mostly remembered for its great toll on human lives and the great sufferings it bestowed. It can also be seen as the period when the management of head injuries was ~~en~~ categorised into a separate sub discipline of Neurosurgery, largely through the efforts of Sir Hugh Cairns and Sir Charles Symonds.

After a lot of research on the frequency, causation and nature of head injuries the pattern is now more towards prevention, a good example is the evolution of the protective helmet and safety belts. Garset (1962) showed that the use of safety belts reduces the incidence of fatal and serious head injuries by 35%, although one should not forget the fact that with the advent of the safety belt came the seat belt syndrome (BJHM). All the same, injuries associated with the use of safety belts are usually mild in nature, serious injuries being found in only 0.7% of cases in the form of injuries to the lumbar spine, pelvic girdle and abdomen.

## PATIENTS AND METHODS:

Head injuries in Kenyatta National Hospital are managed in general surgical wards.

The patient who presents with a head injury problem is first seen at casualty by the admitting senior house officer (SHO). The SHO conducts a general examination of the patient and requests for a skull Xray, should he consider this to be necessary. He also institutes the initial management of this patient and makes a decision as to whether the patient needs admission or not. There's no observation ward for head injuries and admission of such cases into the casualty Recovery ward is usually discouraged because of the lack of adequate manpower to provide the necessary close observations. Therefore head injuries who need admission end up in the surgical wards, and due to shortage of beds, only those cases which warrant admission are usually admitted.

The management of un-complicated head injuries is carried out by the surgical firm under whose care the patient falls, and those head injuries which show complications are referred to the neuro-surgical unit. The senior house officer in the ward usually makes the necessary assessments and consultations dealing directly with his counterpart in the neurosurgical unit.

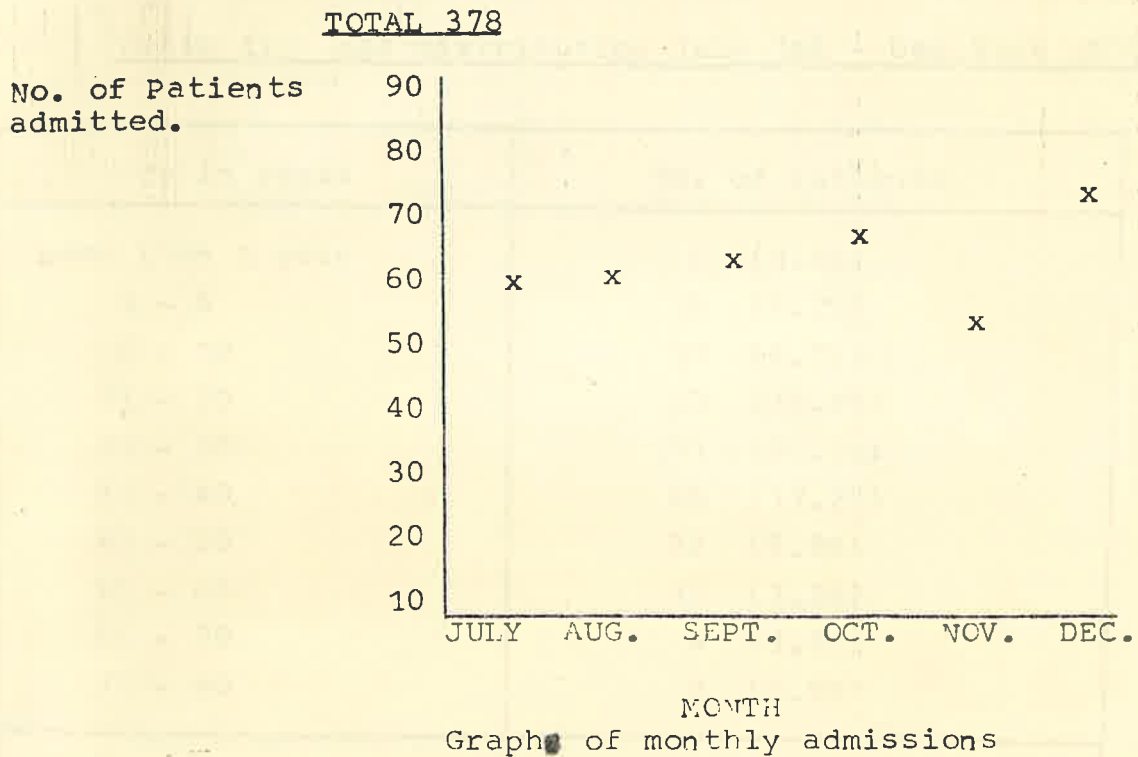
Patients who were admitted into the wards for head injury problems between July 1st 1979 and December 31st 1979 qualified to be included in the study. All the relevant information for the study was filled in a handout, part of the details of which are shown in the appendix.

The details looked for included personal details, the history of the injury, clinical findings, operative findings and a critical evaluation of the management. Other details were added if found necessary.

TOTAL ADMISSIONS:

Between July 1st 1979 and December 31st 1979, both dates inclusive, 378 cases of head injuries were admitted into the Kenyatta National Hospital. The pattern was very similar for each month except in the month of December where a peak was observed.

Fig. 1:





AGE AND SEX DISTRIBUTION:

The youngest age was two months and the oldest was seventy nine years. One hundred and ninety one patients (50.5%) were between twenty one and thirty years, six (1.6%) were less than one year and six were over sixty years, six (1.6%) were less than one year and six were over sixty years (see Table 1 and Fig.2). The peak of the age distribution was between twenty and thirty years, and the smallest number was seen at both extremes of age.

Table 1: Age distribution July 1st - Dec.31st 1979

Age in years	No. of Patients
Less than 1 year	6 (1.6%)
1 - 5	16 (4.2%)
6 - 10	17 (4.5%)
11 - 20	43 (11.4%)
21 - 30	191 (50.5%)
31 - 40	65 (17.2%)
41 - 50	22 (5.8%)
51 - 60	12 (3.2%)
61 - 70	4 (1.1%)
71 - 80	2 (0.5%)
<b>TOTAL</b>	<b>378</b>

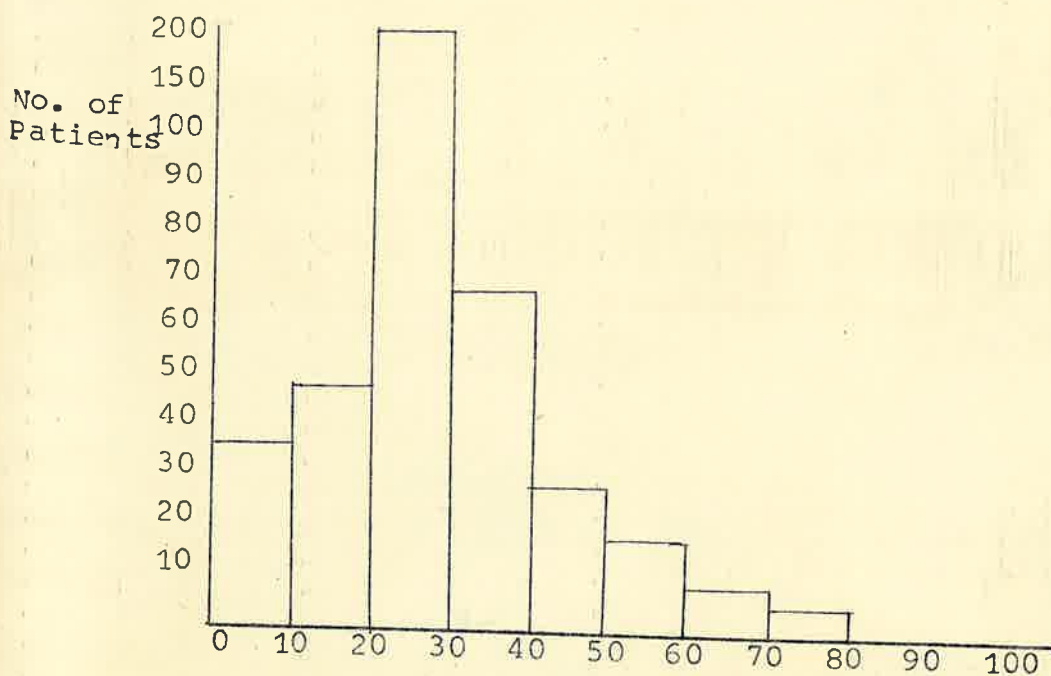


Fig.2 Graph of age distribution-  
July 1st - December 31st, 1979

Three hundred and thirty one patients were males (87.6%) and forty seven were females (12.4%).

Table 2: Sex distribution

MONTH	JULY	AUGUST	SEPT.	OCT.	NOV.	DEC.	TOTAL
Male Pts.	54(■)	57(■)	60	55(■)	39	66	331(87.56)
Female Pts.	8	5	6	11	10	7	47(12.44)
TOTAL	62	62	66	66	49	73	378

MALE 331 (87.6%)

FEMALE 47 (12.4%)

REFERALS: 75 (19.8%)

The Cause of Injury:

Most of the injuries were due to road traffic accidents, one hundred and sixty seven patients (46.3%), followed by assaults, 146 (40.4%), injuries sustained as a result of a fall from a height usually a tree or a tall building, 37 (10.3%), Industrial accidents, 5 (1.4%), sports, 2 (0.5%) and one interesting case of suicide by close range firing of a bullet into the head. These findings are shown below, Table 3.

TABLE 3

The cause of Injury:

NATURE OF INJURY.	NO. OF PATIENTS	PERCENTAGE
1. Road Traffic Accident	167	46.3
2. Assault	146	40.4
3. Fall	37	10.3
4. Industrial Accident	5	1.4
5. Sport	2	0.5
TOTAL	361	100%



Types of instruments used in assault:

Various types of instruments are used in assaults and at the Kenyatta National Hospital some of the most common were Rungus and Pangas and occasionally stones. One case was due to high velocity missile (bullet), a suicide case. These instruments were grouped into either sharp or blunt, as shown in Table 5.

TABLE 5

Types of instruments used in Assault:

Type of instrument	No. of Patients
Blunt, (Stones, Rungus, etc.)	95 (65.1%)
Sharp. (Panga, Knife, etc.)	32 (21.9%)
Unknown	19 (13.0%)
TOTAL	146 (100%)

Fig.3:



RUNGU

Fig.4:



PANGA

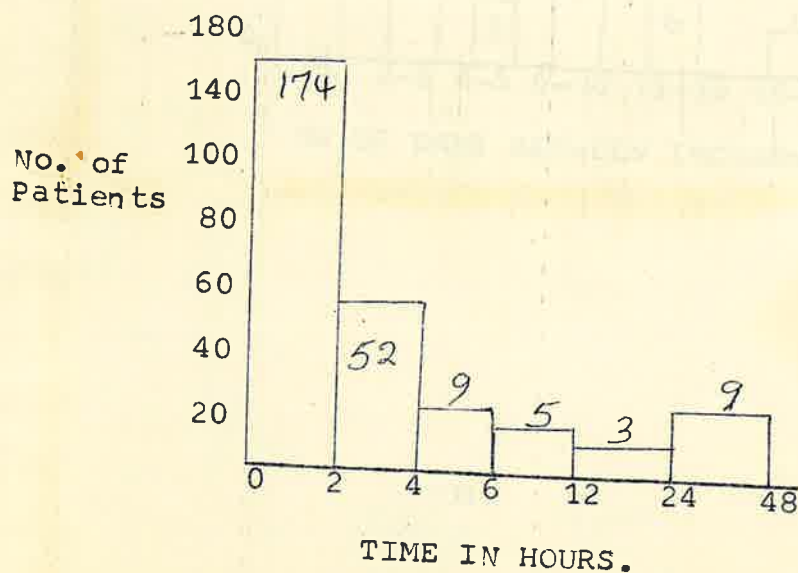
TIME INTERVAL BETWEEN INJURY AND ADMISSION:

In most of the patients the time when the event took place was recorded in the casualty card, as too was the time of admission. One hundred and seventy four patients came to hospital within two hours of having sustained the injury, 52 between two and six hours, 9 between four and six hours and 5 between six and twelve hours, 3 between twelve and twenty four hours and 9 between twenty four and forty eight hours. In other patients, it was not possible to work out the time in hours from when accident occurred to admission in terms of number of days.

Table 6: Time interval between injury and admission.

Time interval hours	No. of Patients
0 - 2	174
2 - 4	52
4 - 6	9
6 - 12	5
12 - 24	3
24 - 48	9
TOTAL	252

Fig. 5:

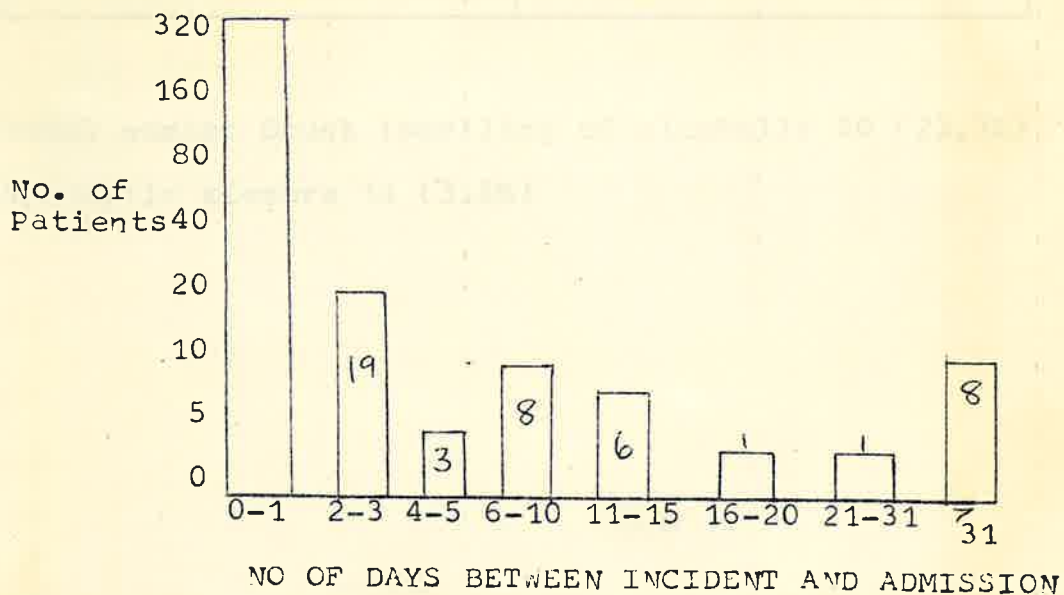


The information obtained on the number of days between accident and admission is shown in figure 6. As mentioned many of the patients in whom the date could not be obtained in terms of number of hours, 20 were seen within the first day of injury, 19 between second and third day and only 8 after one month after the incident.

Table 7: Time interval between injury and admission in days and number of Patients admitted.

Time interval, Days	No. of Patients
1st Day	320
2 - 3	19
4 - 5	3
6 - 10	8
11 - 15	6
16 - 20	1
21 - 31	1
More than 31	8
TOTAL	366

Fig. 6:



CLINICAL FINDINGS:

LEVEL OF CONSCIOUSNESS, ALCOHOL AND EPILEPTIC SEIZURES

One hundred and thirty three patients were fully conscious at the time of admission. Ninety two patients were drowsy, Fifty Six were confused and forty two were stuporous, while fifty five were comatose

Ninety patients were smelling of alcohol when they were seen first.

Fourteen patients had epileptic seizures of the grandmal type (3.6%).

Table 4: Level of consciousness on admission

Level of consciousness	No. of Patients	
Normal	133	35.2%
Drowsy	92	24.3%
Confused	56	14.8%
Stupor	42	11.1%
Coma	55	14.6%
TOTAL	378	(100%)

Total number Drunk (smelling of alcohol): 90 (23.3%)

Epileptic seizure 14 (3.6%)

## SKULL FRACTURES:

Fractures of the skull were diagnosed by radiography of the skull except where the base was involved, when clinical judgement was accepted. One hundred and forty nine patients had fractures of the base. Sixteen patients had cerebrospinal fluid rhinorrhoea and forty seven had cerebrospinal fluid otorrheah.

Table 8:

### Fractures of the skull

Type of fracture	No. of Patients	
Vault: Total	149	(73.4)
a) Compound	62	(41.6)
b) Simple	87	(58.4)
c) Depressed (simple & compound)	62	
Base: Total	54	(26.6)
a) CSF rhinorrheah	16	
b) CSF otorrheah	47	
TOTAL	203	

Carotid angiogram was done in thirty three patients and was normal in eighteen and abnormal in fifteen.



Common presentations in patients with Intra cranial haematomas.

Deteriorating level of consciousness was the commonest presentation. This was followed by weakness localised to one part of the limb or a half of the body. The results are shown in table 10. Bradycardia, systolic hypertension and vomiting were not very frequent findings. Eleven patients presented with a dilated pupil of significant localising value.

33 carotid angiograms were done and eighteen of these were normal

Table 9:

Presentation in patients with Intracranial Haematomas

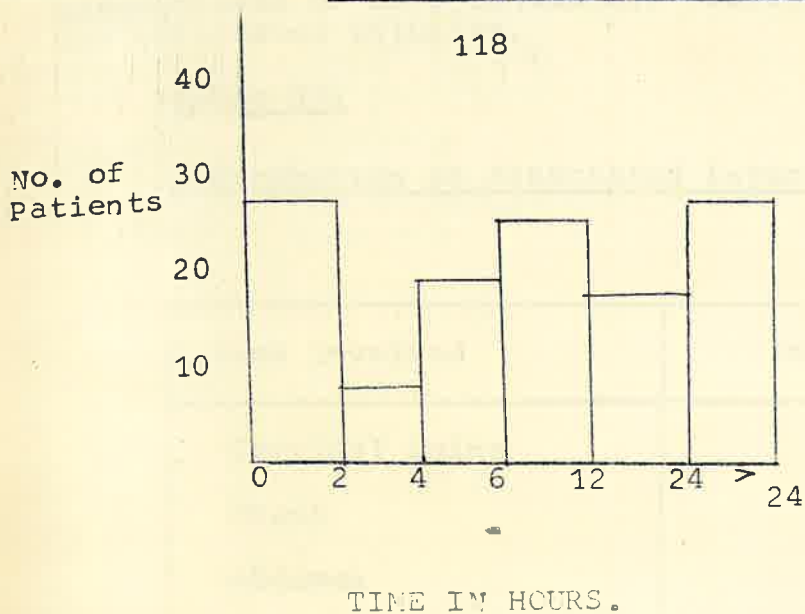
Symptom/ Sign	No. of Patients
Bradycardia	4
Systotic hypertension	2
Deterior. level of consciousness	23
Vomiting	4
Dilated pupil	11
Hemi (Mono) Paresis	17
Decerib, rigidity	4
Fits	1
CAROTID ANGIOGRAM ABNORMAL	15
NORMAL	18
TOTAL	33

TIME INTERVAL BETWEEN ADMISSION AND SURGERY:

This was done by recording the time the patient got admitted and the time he underwent surgery.

Thirty six patients were taken to theatre more than twenty four hours from the time they were admitted. This was the largest number. Twenty seven went to theatre within two hours, six between two and four hours, nineteen between four and six hours, twenty two between six and twelve hours, and seventeen between twelve and twenty four hours.

Fig. 7: No. of Patients operated vs. time in hours from admission to operation.



Associated Injuries:

The commonest associated injuries were fractures of the long bones, humerus, femur, tibia and fibula and radius and ulna.

66 patients were found to have other associated injuries and thirty seven had fractures of the long bones. Injuries to the chest were the next common associated injuries, ten patients having presented with this problem, which varied from undisplaced fracture of one rib to multiple rib fractures with pneumophorax or lung contusion. Eleven patients had more than one associated injuries.

Table 10:

Distribution of Associated Injuries.

System involved	No. of Patients
Cervical Spine	1 (1.5%)
Chest	10 (15.2%)
Abdomen	2 (3%)
Pelvis	3 (4.5%)
Long Bones	37 (56.1%)
Other Spine	2 (3%)
Multiple (>3)	11 (16.7%)
TOTAL	66 (100%)

CLINICAL AND OPERATIVE FINDINGS:

One hundred and eleven patients had simple soft tissue injuries which usually involved cuts on the scalp with no evidence of associated intracranial injury or damage. Two hundred and seven patients had primary brain damage. 17 patients had subdural haematomas and sixteen patients had extradural haematomas. One hundred and thirty three patients were grouped as mild head injuries, one hundred and forty five as moderately severe head injuries and 88 as severe head injuries.

Table 11:

Final clinical and operative findings

Diagnosis:	No. of Patients
Soft tissue injury	111 (31.6)
Primary Brain damage	207 (59.0)
Subdural Haematoma	17 (4.8)
Extradural Haematoma	16 (4.6)
TOTAL	351

Table 12:

Grading of the Head injury

Grade:	No. of Patients
Mild head injury	133 (36.3)
Moderate head injury	145 (39.6)
Severe head injury	88 (4.1)
TOTAL	366

There were 25 subdural haematomas from the initial clinical diagnosis (68%), while in the final operative diagnosis there were only 17 (4.9%).

Table 13:

Initial Clinical Diagnosis

Diagnosis:	No. of Patients	Percentage
Soft tissue injury	113	31
Primary Brain damage	210	57.5
Subdural haematoma	25	6.8
Extradural haematoma	15	4.1
Intracerebral haematoma	2	0.6
TOTAL	365	100%

MORTALITY:

378 patients were admitted between July and December. 59 of these died while in the ward, overall mortality of 15.6%. The largest number of patients were admitted during the month of December, and there were 10 deaths in this month. In September there were sixty six admissions and fourteen of these died. In July sixty two patients were admitted and 5 of these died.

118 patients underwent operations, 14 died, an operative mortality of 11.9%.

Table 14:

Number of patients admitted and number who died July - December, 1979

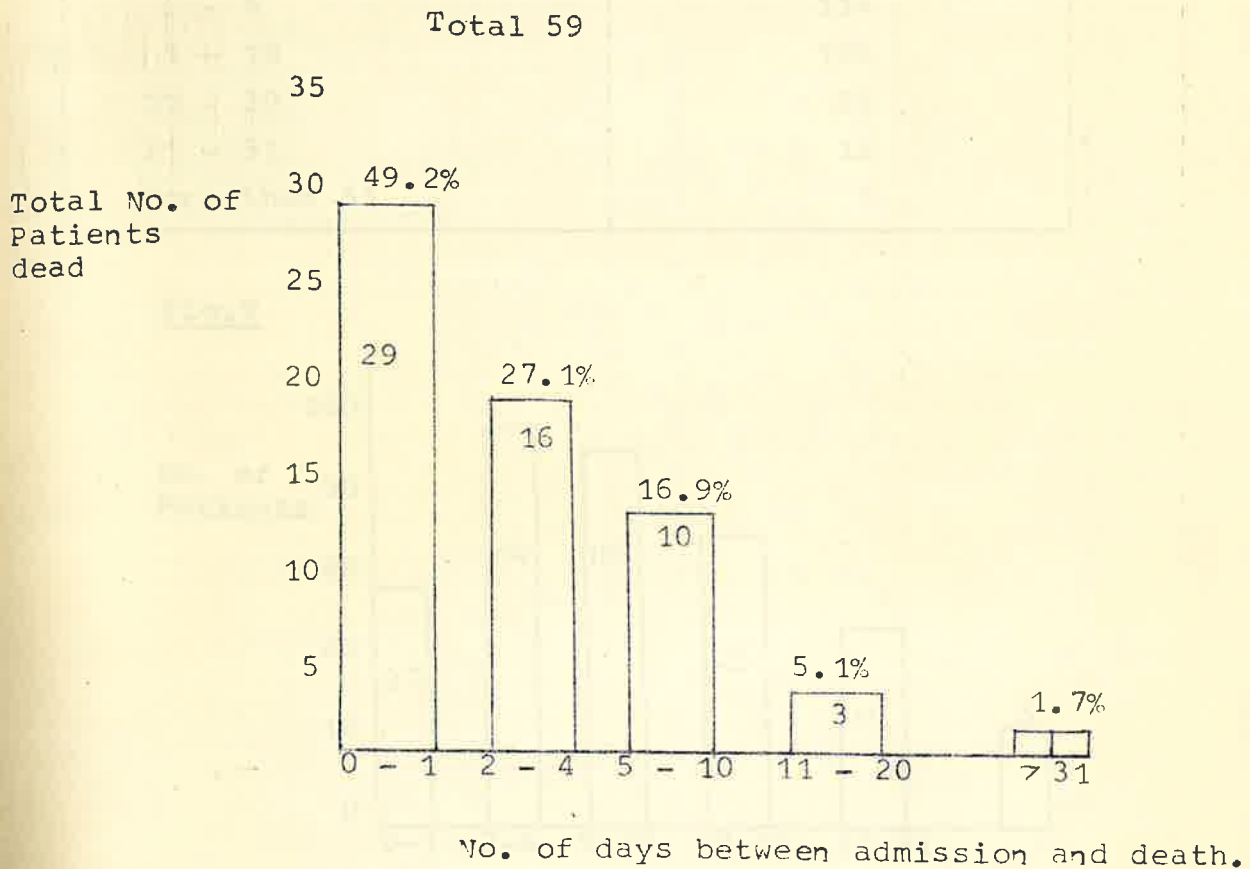
MONTH	JULY	AUGU.	SEP.	OCT.	NOV.	DEC.	TOTAL
No. admitted	62	62	66	66	49	73	378
No. died	5	10	14	11	9	10	59
Mortality	8.1	16.1	21.2	16.6	18.4	13.7	15.6

TIME INTERVAL BETWEEN ADMISSION AND DEATH:

Twenty nine patients died within twenty four hours of admission (49%). Only one patient died after more than one month of hospitalisation. More than 70% of the deaths occurred within the first forty eight hours. 16 patients died between two and four days, 10 between five and ten days and 3 between the 11th and 25th day. The number of the patients who died decreased with the duration of hospital stay. No patient was recorded with prolonged unconsciousness ( more than one month), an un common finding at the Kenyatta National Hospital.

Fig. 8

Time interval between admission and death



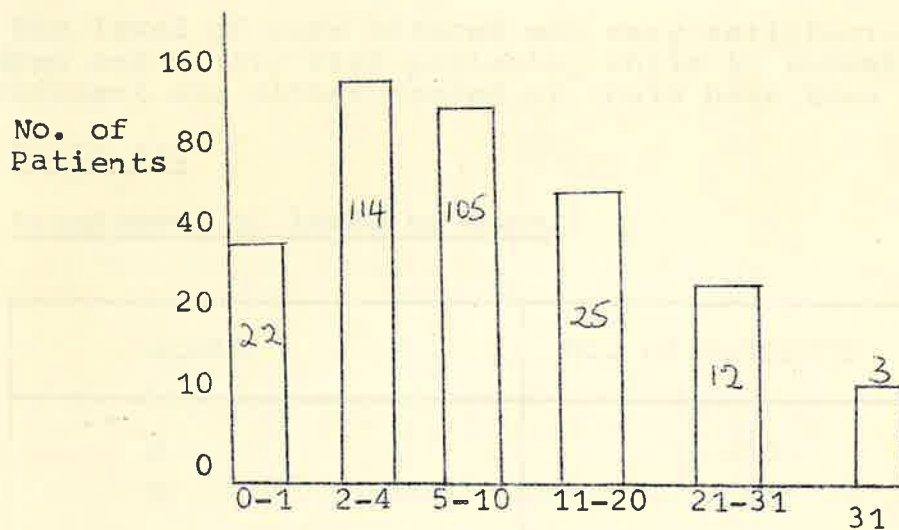
### DURATION OF HOSPITAL STAY:

Three patients were in hospital for more than one month. Two hundred and nineteen were hospitalised for a period varying from two to ten days, while twenty two patients were kept in for only two four hours. These findings are shown in figure 9 and Table 15. Prolonged hospitalisation, i.e. more than one month of hospital stay does not seem to be a common finding, such patients being on the minority side.

Table 15: Hospital stay in days.

Duration in days.	No. of Patients
0 - 1	22
2 - 4	114
5 - 10	105
11 - 20	25
21 - 31	12
More than 31	3

Fig.9



No. of Patients/In-Patients stay, of cases which were discharged.

EVALUATION:

On discharge or after death, an analysis was done to find out any factors which could have contributed to the severity of the head injury and the degree of the level of care offered to the patient.

In one hundred and fifty one patients the severity was due to a primary damage to the brain. Delayed treatment could be said to have contributed to the outcome in forty patients, while associated injuries aggravated the condition in twenty one patients. In eighteen patients multiple factors were involved. These results are shown in table 16.

Table 16:

Factors which could have contributed to the severity.

Contributing factor	No. of Patients
Primary brain damage	151
Delayed treatment	40
Other injuries	21
Multiple factors	18

The level of care offered was very satisfactory in two hundred and ninety five patients, while in seventy five patients improvement was either needed or could have been beneficial.

Table 17:

Assessment of level of care.

SCORE	NO. OF PATIENTS
A	295
B	45
C	30

A = Satisfactory

B = Fair

C = Unsatisfactory



## DISCUSSION:

The Kenyatta National Hospital is situated within the city of Nairobi which has a population of about 800,000. It is the only General Hospital in the City and it also functions as a National referral hospital for the whole country.

Three hundred and seventy eight cases of head injuries were treated in this hospital between July first and December thirty first, Nineteen seventy nine, 331 males (80%). Forty percent of these cases were assaults, partly explaining the high male predominance. Seventy five cases were referred. Kariobangi, Mathare Valley, Kawangware, Eastleigh and Majengo Pumwani are famous names in the city for crime and violence, as well as the brewing of illicit liquor. The Pattern of head injuries seen at the Kenyatta National Hospital has to be viewed against this background.

The month of December is full of festivities, such as Jamhuri Day and Christmas Season, and this could explain the peak in admissions seen during this month. Previous studies showed a relationship between day of the week and number of head injuries admitted, more cases being seen during weekends.

It would be ideal if all head injuries would be admitted for observation. This is usually not possible and the price paid for this is often high. In one instance, a serious head injury was missed. A young boy on his way to Nairobi Agricultural Show was knocked down by a car and brought to the Hospital. He had no obvious neurological deficits and general condition was alright. He was sent away. The next day he was brought back by his parents unconscious with a fixed dilated pupil and an obvious subgaleal haematoma in the temporal region. He died before he could undergo surgery.

Many cases of head injuries usually die in casualty before gaining admission, immediately on arrival. Such cases never enter into the hospital records (Barr, 1964).

In 1978 there were six thousand nine hundred and fifty six road accidents in Kenya, and in 1979 eight thousand four hundred and fifty (N.V. Nyagga). Nairobi Province led with 2581 road accidents in 1979 followed by Rift Valley 1617. North Eastern Province had the least road accidents, 135.

Table 18: Road accidents by Provinces.

Province	No. of accidents	
	1978	1979
Nairobi	1959 (28.2%)	2581 (30.5)
Rift Valley	1243 (17.9%)	1617 (19.1)
Coast	969 (13.9%)	1233 (14.6)
Central	1170 (16.8%)	1249 (14.8)
Eastern	840 (12.1%)	786 (9.3)
Western	379 (5.4%)	365 (4.3)
Nyanza	308 (5.4%)	484 (5.7)
North Eastern	88 (1.3%)	135 (1.7)
TOTAL	6956 (100%)	8450 (100%)

Fourteen thousand seven hundred and forty nine persons, were involved in road accidents in 1979 and twelve thousand four hundred and forty four in 1978. In 1978, 1588 persons were killed and 1979, 1661 killed. 41 percent of those killed in 1979 were pedestrians.

Table 19:

Accident statistics for 1978/1979

Type of person		No. of persons	
		1978	1979
Drivers:	killed	231 (14.5)	228 (13.7)
	seriously injured	806	829
	slightly injured	1073	1210
	TOTAL	2110 (17%)	2267 (15.4)
Motor Cyclists:	killed	21 (1.3%)	27 (1.6)
	seriously injured	145	145
	slightly injured	195	383
	TOTAL	361 (2.9%)	555 (3.8)
Pedal cyclists:	Killed	64 (4.0%)	80 (4.8)
	seriously injured	217	209
	slightly injured	384	426
	TOTAL	665 (5.3%)	715 (4.8)
Passengers:	killed	512 (32.2%)	634 (38.2)
	seriously injured	2184	2763
	slightly injured	3740	4353
	TOTAL	6436 (51.7%)	7750 (52.5)
Pedestrians:	killed	760 (48.0%)	692 (41.7)
	seriously injured	917	1136
	slightly injured	1195	1634
	TOTAL	2872 (23.1%)	3462 (23.5)
Total No. of persons:	killed	1588	1661
	seriously injured	4269	5082
	slightly injured	6587	8006
	TOTAL	12,444	14,749

In Kenya, hospital admissions are dominated by four major groups of diseases, infective and parasitic diseases, diseases of the respiratory system, normal deliveries and complications from delivery and accidents (Bonte).

Most of the cases seen in this series were between the ages of twenty and thirty years. This could be explained by the high number of cases of assaults which were seen (40.4%). There could

be a second reason related to urbanisation, immigration leading to an influx of persons between the ages of 15 and 44 into the big towns. According to the 1969 census the percentage of the underage population in the total population was just over 50%, but the Nairobi figure was 36.1%, much less than the national average. Such an influx could be related to the search for job opportunities in the big cities. (Ominde).

There were 331 males (87.6%) and 47 females (12.4%). The same explanation used for explaining the age pattern could be applied for the sex patterns. In immigration into the city would be dominated by males. It could also be said that the population at risk from head injuries from assault and being knocked down by cars has a male preponderance.

In 1972, road traffic accidents made up 0.65% of the 200,000 deaths which occurred in Kenya in that year. In 1979 over fourteen thousand people were involved in road traffic accidents and out of these, 1661 died. In the last six months of 1979, there were 378 head injuries, 46.3% due to road traffic accidents and 40.4% due to assaults. Assaults nearly equaled traffic accidents as a major cause of head injuries in this country. In Glasgow, assaults and fall as a result of drunkenness accounted for more than half of the head injuries while road traffic accidents accounted for only a quarter (Galbraith). The situation in Glasgow is slightly different from that found elsewhere in Britain where road traffic accidents compose more than 50% of the head injuries. Industrial accidents are a rare cause of head injuries in Kenya. Rungus and Pangas are weapons peculiar to this part of the world. Traditionally a rungu is carried as a sign of respect and importance. It has now become an important weapon and is in many instances misused. A panga, on the other hand is not a traditional instrument but is an innovation of the white man. Initially meant for use in the shamba, the indigenous African has not delayed in finding it other uses such as cracking people's skulls.

A delay in arriving to the hospital can mean life or death. Several factors could account for this delay. Perhaps the most important is distance. Patients who were referred from other hospitals always arrived after a delay of two to three days and more. Obviously these patients would first be admitted into the surgical ward of the peripheral hospital and then put under observations. If the patient's condition was noticed not to be improving then either of the following two things could happen. The patient could be taken to theatre if there is an obvious indication for doing so and primarily managed in that hospital or the patient could be transferred to Kenyatta National Hospital if the peripheral hospital lacks good theatre facilities or the doctor in charge does not feel competent enough to handle such a situation. One such patient was referred from Loitokitok hospital with an extradural haematoma of two days. He survived.

A patient could arrive late to the hospital because he did not feel particularly unwell after the trauma. A period of days might have passed and then his friends or relatives noticed a change in his behaviour and so brought him to hospital.

Most of the patients came to hospital within one day of the injury. This could be because most of the accidents and assaults occurred within Nairobi where good transport facilities are available and the public are very conscious of their health, not

hesitating to rush to hospital for the slightest trauma to the head. The fact that the health services offered are free is also important. It would therefore not be justifiable to correlate the outcome in most of these head injuries to a delay in arriving to the hospital. Majority of those patients who come from within Nairobi arrived at the hospital within two hours of sustaining the injury (174). Fifty two after an interval of between two and four hours, 9 between four and six hours, 3 between twelve and twenty four hours and 9 between twenty four and forty eight hours. This information could not be obtained in the remaining 126 cases. Transport facilities in the city are good, both public and private. The patients were usually brought to hospital by ambulance, a well organised facility provided by the City Council. Some times they were brought by fellow travellers or those who knocked them, for pedestrians this resulted in the early arrival to hospital for most of the patients.

Sevit (1968) in a review of fatal road accidents at Birmingham hospital also found that delay in arrival to hospital was not a contributing factor to the death of the patient, most of the patients arriving within two hours of the injury.

Evaluation of the state of consciousness is important in the determination of the pattern of head injury. The depth of the initial unconsciousness and the rate of recovery can be related to the severity of the original impact. In the uncomplicated injury where the patient may be expected to show progressive recovery the degree of unconsciousness is proportional to the severity of the injury and inversely proportional to the time which has elapsed since the accident. This may be important in prognosis, a patient seen to be quite stuporose two hours after injury having a better prognosis than one who reaches this stage of recovery after 24 hours (Hooper).

The progression of the conscious state may give an indication of the recovery state, and this is important in the management and care of the head injury (Jennet, 1974). Charts and records should be kept of the acute and subacute stages so that a continuous change of record is available for reference. The progress of the patient in the interval between the injury and the first recorded medical observation is most of the time not available, although its importance cannot be over emphasised (Sevit 1968). This information should usually be supplied by the person who was in charge of the patient from the time of accident upto the time he arrives at the hospital. About 15% of the patients admitted were found to be comatose and 35% had a normal level of consciousness, 24% were drowsy, 14% confused and 11% stuporous. Assessment of the level of consciousness is a most important aspect of the management of head injuries especially in predicting the ultimate outcome and development of complications, leading to a deterioration in the head injury. In assessing the cause of the deterioration one has to keep in mind such things as airway obstruction, convulsions, Thoracic or abdominal injuries, meningitis and renal failure which could cause the patients condition to deteriorate.

At the Kenyatta National Hospital observations on the patients level of consciousness and any other localising signs are recorded into a special chart (Fig. 10, 11).

.../4

# HEAD INJURY AND CRANIOTOMY CHART

This chart to be started in Casualty and continued in the ward.  
Underline the observations that are required

DATE	SURNAME	OTHER NAMES	REG. No.
± 1 or 2 HOURLY CHART*	<u>N O R E A M I A</u>	<u>ERESTIA</u>	
PULSE	<u>10 pm</u> <u>2 am</u> <u>6 am</u> <u>10 am</u> <u>2 p</u> <u>6 p</u> <u>10 pm</u> <u>2 pm</u> <u>6 pm</u>	<u>80</u> <u>80</u> <u>80</u> <u>80</u> <u>80</u> <u>80</u> <u>80</u> <u>80</u> <u>80</u>	<u>120</u> <u>120</u> <u>120</u> <u>120</u> <u>120</u> <u>120</u> <u>120</u> <u>120</u> <u>120</u>
BLOOD PRESSURE	<u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u>	<u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u>	<u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u> <u>130/70</u>
RESPIRATION	<u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u>	<u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u>	<u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u> <u>20/1-20/1</u>
TEMPERATURE	<u>37.3</u> <u>37.8</u> <u>37.4</u> <u>37.6</u> <u>37.9</u> <u>37.8</u> <u>37.6</u> <u>37.5</u> <u>37.2</u>	<u>37.3</u> <u>37.8</u> <u>37.4</u> <u>37.6</u> <u>37.9</u> <u>37.8</u> <u>37.6</u> <u>37.5</u> <u>37.2</u>	<u>37.3</u> <u>37.8</u> <u>37.4</u> <u>37.6</u> <u>37.9</u> <u>37.8</u> <u>37.6</u> <u>37.5</u> <u>37.2</u>
1. FULLY CONSCIOUS (answers questions intelligently)	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>
2. CONSCIOUS BUT CONFUSED (answers simple questions only)	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>
3. SEMI-CONSCIOUS (only responds to pain)	<u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u>	<u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u>	<u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u> <u>YES</u>
4. COMA (no response to pain)	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>	<u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u> <u>NO</u>
PUPILS (draw circles indicating relative size.)			
SIZE	R	L	
REACTION TO LIGHT	R	L	
FLITS	PRESENT	ABSENT	
SPONTANEOUS MOVEMENT (write "yes" or "no")			
PARALYSIS (if conscious write "yes" or "no"; if unconscious write "ext" or "flex" topain)	R. ARM	R. LEG	L. ARM
OTHER INJURIES			
CIRCLE IF INVOLVED			
CHEST	ARM	NECK	
ABDOMEN	LEG	SPINE	
DATE OF ADMISSION	TIME OF ADMISSION		

\*WHEN TIME FREQUENCY IS ALTERED A NEW CHART MUST BE STARTED

# HEAD INJURY AND CRANIOTOMY CHART

This chart to be started in Casualty and continued in the ward.

Underline the observations that are required

DATE: 25/12/79.	SURNAME	OTHER NAMES						REG. No.
1 or 2 HOURLY CHART*	Reese	26/12- 10 am	Nyaganyi	30/12	31/12	11/12	332225	
PULSE	80/min	94	96	96	94	88		
BLOOD PRESSURE	120/70	120/40						
RESPIRATION	20/min	20/min	20/min	20/min	20/min	20		
TEMPERATURE	38.5	38.5	38.5	38.5	38.5	38.5		
1. FULLY CONSCIOUS (answers questions intelligently)	NO	NO	NO	NO	NO	NO		
2. CONSCIOUS BUT CONFUSED (answers simple questions only)	YES	YES	YES	YES	YES	YES		
3. SEMI-CONSCIOUS (only responds to pain)	NO	NO	NO	NO	NO	NO		
4. COMA (no response to pain)	NO	NO	NO	NO	NO	NO		
PUPILS (draw circles indicating relative size)	SIZE	R 0	0	0	0	0		
	REACTION TO LIGHT	R +ve	+ve	+ve	+ve	+ve		
FITS	PRESENT	NO	NO	NO	NO	NO		
	ABSENT	YES	YES	YES	YES	YES		
SPONTANEOUS MOVEMENT (write "yes" or "no")	R. ARM	YES	YES	YES	YES	YES		
	R. LEG	YES	YES	YES	YES	YES		
	L. ARM	YES	YES	YES	YES	YES		
	L. LEG	YES	YES	YES	YES	YES		
PARALYSIS (if conscious write "yes" or "no"; if unconscious write "ext" or "flex" to pain)	R. ARM	NO	NO	NO	NO	NO		
	R. LEG	NO	NO	NO	NO	NO		
	L. ARM	NO	NO	NO	NO	NO		
	L. LEG	NO	NO	NO	NO	NO		
OTHER INJURIES	CHEST	NO	NO	NO	NO	NO		
	ABDOMEN	NO	NO	NO	NO	NO		
	ARM	NO	NO	NO	NO	NO		
	LEG	NO	NO	NO	NO	NO		
		NECK	NO	NO	NO	NO		
		SPINE	NO	NO	NO	NO		
		DATE OF ADMISSION						
		TIME OF ADMISSION						

\*WHEN TIME FREQUENCY IS ALTERED A NEW CHART MUST BE STARTED

These observations may be carried out half hourly, 1 or 2 hourly or even 4 hourly. They include pulse, blood pressure, respiration temperature, level of consciousness, the size of the pupils and their reaction to light, presence of fits, presence of spontaneous movements and evidence of paralysis if any. Also recorded are other associated injuries, specifically chest, abdomen, limbs, neck and spine. The charts shown in figs. 9 and 10 are those of N.T. Study 317, hospitalized for more than one month.

N.T.	I.P.352225	Study 317.
Female	25 years old.	
Admitted	03. 12. 79	
Injured	03. 12. 79	
Discharged	after 17.1.80.	

Was picked on the roadside by Police and brought to the casualty department of the Kenyatta National Hospital suspected to have been assaulted. Was found to have multiple cuts on scalp, comatose and no other signs. Radiography skull revealed no fracture. Had a convulsion in casualty but none was observed in the ward. Made a slow but gradual recovery and was able to go home. Diagnosis was primary brain damage."

Initially an attempt was made to measure quantitatively the amount of alcohol in the blood of those patients who were brought to be drunk. One of the certificates of analysis is reproduced as an example (fig.11).

REPUBLIC OF KENYA

GOVERNMENT CHEMIST'S DEPARTMENT

P.O. Box 20753

TEL. 23302/3

NAIROBI

CERTIFICATE OF ANALYSIS

Report Reference No.: P/HOSP/79/37

Lab. Sample No. 736

Sender's Reference:

Sender:

Dr. N.J. Mwangombe,  
Kenyatta N. Hospital,  
P.O. Box 20723,  
NAIROBI.

Date Received : 2-8-79.

Description of Sample:

One bottle containing blood labelled "MAINA IP. 332697."

Examination Required:

Alcohol

Analytical Report:

The sample of blood contained 186 mg. of alcohol (ethanol) per 100 ml. which is equivalent to a minimum intake of 4½ half litre bottles of beer or 10 whiskies.

Date: 3rd August, 1979

  
( P. M. KAMAU )

for: Government Chemist

PMK/RM.

GPK 1078-10m-6/77

FIG 91  
-29-



This became more difficult and a more crude estimation was eventually chosen, noting down whether the patient smelled of alcohol or not, 23% of the head injuries had evidence of having taken a significant amount of alcohol. In the Western infirmary Glasgow head injuries accounted for almost one third of acute male surgical admissions and there was a relationship between the intake of alcohol and the head injury (Galbraith 1976). Alcohol was found in the blood of nearly half of the patients admitted and the level was related to the mode of injury, higher levels being found where the injury was due to assault than road accidents or other injuries.

Assaults and fall as a result of being drunk accounted for more than half of the head injuries in Glasgow and road accidents made up only a quarter of cases compared with half in most other reports, and it was suggested that alcohol is an important factor contributing to the cause of head injury. The results in Kenyatta National Hospital are very similar to those found in Glasgow, a quarter of all the head injuries having been found to have taken some alcohol and nearly half of the head injuries having resulted from assaults.

A depressed level of consciousness in a patient who is drunk is often attributed to the effects of the alcohol. Constant evaluation is necessary to be able to obtain the true picture as the effect of alcohol wears off.

The cases of epilepsy recorded were those which were quite obvious. The less obvious, for example focal twitching were not recorded and had this been done, probably a higher figure than 3.6% would have been obtained. Epilepsy is less common after the blunt head injuries of civilian life than after the missile injuries of war time (about 5% compared with 45%). Trauma is gradually becoming an important and common cause of epilepsy of late onset, (Jennet, 1974). Epilepsy may be grouped into early epilepsy occurring in the first week after injury and that occurring later, late epilepsy (Jennet 1961)

Early epilepsy occurs in about 5% of injuries admitted to hospital, in a third the fit is within an hour of injury, in a third during the rest of the first day and in the remainder during the rest of the first week. 25% of patients with a fit in the first week go on to develop established late traumatic epilepsy, a greatly increased risk as compared with patients who come through the first week without a fit.

Late epilepsy can occur at any interval after injury but 60% occur within the first year. Usually this fit persists. There are some factors which tend to predispose to late epilepsy such as depressed fractures, intra cranial haematoma and of course, early epilepsy. It is thought that only those depressed fractures with a post traumatic amnesia of more than 24 hours are at a definite risk of developing late epilepsy. The ability to predict the risk of epilepsy enables the long term use of anticonvulsants to be more rationalised.

The thickness of the skull averages 2-6 mm in the adult. The vault is composed of an outer solid layer or table, a cancellous middle portion called the diploe and a solid inner table.

Skull fractures are more common after impact injury in which the inertia of the head supplies practically all of the reaction to the impacting object.

The conditions which contribute to the shape of the impact are the amount of kinetic energy, the relative masses of the colliding bodies, the elastic and viscoelastic properties of the bodies and energy absorbing materials between the bodies, the line of action with respect to the centres of mass and the area of impact, (Yeoman).

The impulse will deform or accelerate the head or both depending upon the shape of the impulse. Experimentally it has been shown that fracture will occur after impact of cadaver head with blunt bodies if the energy is sufficient and the exchange between the skull and the impacting body occurs in an extremely short period of time (Ommaya). A force of 900-1,700 lb in 0.001 sec. or less is needed to produce fracture.

Linear fractures constitute 80% of fracture of the skull, 50% occur in the midportion of the skull and extend toward the base of the middle fossa. Remaining half are equally divided between the frontal and occipital regions.

Linear fractures are the result of elastic deformation of the skull. The blow in the skull results in inbending at the site of impact and outbending of the surrounding area. The linear fracture begins in the area of outbending and extends towards the area of impact. A blow transmitting greater energy may cause a comminuted or multilinear fracture.

Fractures of the base commonly occur as a result of extension of fractures of the vault. They may occur separately as the result of stress concentrations building up among the many perforating foramina of the base. They may also result from indirect violence eg. fall from a height with the vertebral column being driven against occipital condyles.

Fractures of the base may also result from an impact on the lower jaw, the force being directed against the floor of the middle fossa by means of the condyloid process.

Depressed fractures result from an energy concentration sufficient to cause local failure of the bone. They may be perforating in type when the object is moving at a high velocity eg. bullet. Brain damage usually results from the shock waves. The depressed fracture may be of penetrating type where an object is moving at a moderately high velocity dissipating much of its kinetic energy at impact. The object penetrates bone driving small bone fragments inwards. The depressed fracture may have associated radial linear fractures: a blunt object e.g. a cricket ball or a brick moving at moderate velocity is usually the cause. The border of the depressed area presents as a curvilinear fracture and the bone will be fragmented by several radial fracture lines. A slow moving object will result in a depressed fracture with linear fractures elsewhere in skull. Other patterns of depressed fractures which may be met with in clinical practice are a depressed fracture pattern which conforms in contour to the shape of the object, a depressed comminuted fracture, or a depressed fracture with avulsion, the bone and intra cranial contents becoming avulsed as a result of continuing movement of the striking object.

Most patients with depressed fracture of the skull which has been properly treated recover rapidly and completely. Brain damage, if any is usually limited to the immediate vicinity of the fracture. Several serious complications may occur such as risk of intracranial infection, damage to dura and brain resulting in intracranial haematomas involvement of the dural venous sinuses resulting in torrential haemorrhage when elevation is attempted, epilepsy soon after the injury or following an interval. Some of these complications can be prevented by good management, based on awareness of the implications of this type of injury. Miller & Jennet (1968) reviewed 400 patients with depressed fracture of the skull treated at Glasgow between 1956 and 1967. They defined a depressed fracture as one involving depression of a bone fragment by at least the thickness of the skull. Infection occurred in 90% of the cases, and intracranial haematoma in 70%. The dural venous sinuses were involved in 11% of the patients. Any of the complications were associated with a significant increase in mortality, prolonged neurological deficit, and late post traumatic epilepsy (Jennet 1972).

At the Kenyatta National Hospital a unique type of depressed fracture was seen, the "Fork Jembe injury". Three cases were recorded between July and December, 1979. They were all referrals. The mother would usually go to the shamba to cultivate and would take the little one with her and probably keep her at a safe distance. As she proceeds to work the little one would unknowingly crawl below in between the mother's knees and be hit by the sharp ends of the Jembe.

A second explanation is that this could be some form of child abuse. Another possible explanation is that the Jembe could have been supported against a tree and it fell accidentally on to the head of the child when its support (base) was disturbed.

"K.W.	Study No. 095	Ward 14, Kenyatta National Hospital.
Sex	Female	Age 10 years
	Date of injury	09.08.79
	Date of admission	15.08.79
	Date of discharge	28.08.79

was referred to the Kenyatta National Hospital from Kiambu District Hospital six days after the injury. No attempt to do surgical toilet of the wound was made at Kiambu. The wound got infected and a decision was made to refer the patient. A history of having been injured by a forked jembe was volunteered by the parents. She had a scalp wound on the parietal region near the midline which was infected and oozed pus on compression. Radiological investigation revealed a depressed Parietal bone fragment but clinically general condition was good being in full possession of her senses with no neurological deficits. Diagnosis of intracerebral abscess fragment was removed, abscess cavity evacuated and antibiotics instilled. Post operative course was stormy but she finally came through it with good recovery.

It was noted that she had developed a localised swelling at the site of the scar which was pulsatile due to lack of a protective bony envelop and hydrocephalus as a complication was thought to be

a possibility. She went home thirteen days after admission".

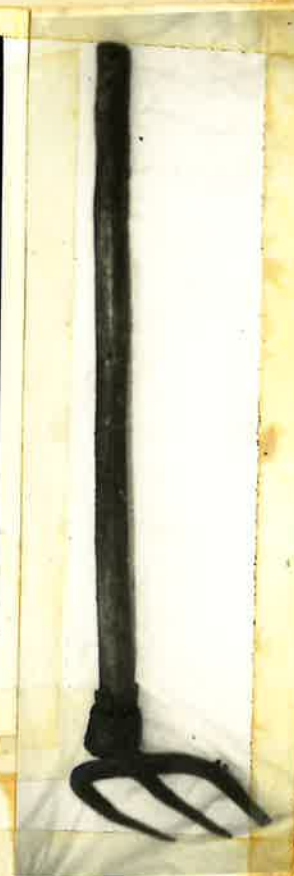
Fig. 22: -X-rays-

Fig. 13



FIG 12 313

Fork  
JEMBE



The distribution between fractures of the vault and fractures of the base was 73% fractures of the vault, and 27% of the base. Otorrhoea in fractures of the base was commoner than rhinorrhoea, but then one wonders how many cases of rhinorrhoea could have been missed, due to the relative difficulty in the detection of this sign (Mosley).

A deteriorating level of consciousness was the commonest presentation followed by hemi-mono-paralysis in intracranial haematomas. Thirty three carotid angiograms were done and eighteen were found to be normal.

Mass lesions in the supratentorial compartment of haematoma nature produce neurological symptoms by increasing the bulk contained within the rigid supratentorial compartment. The effects of the gradual increase in tissue mass is to push the brain downwards through the incisural notch causing herniation. Initially diencephalon then Pons and medulla are affected. Brainstem decompression caused by this mechanism may be divided into two syndromes, the central syndrome and the syndrome of uncus herniation and lateral brainstem compression. These syndromes represent a stage of decompression in the ability of intracranial contents to withstand further increase in mass. Papilloedema requires at least 18 to 24 hours to develop and is therefore uncommon in acute increased intracranial pressure. The most important sign of acutely increased intracranial pressure is a decrease in the state of consciousness. This sign may or may not be accompanied by bradycardia, increased pulse pressure, and slow and deep respirations.

The central syndrome is manifested by change in alertness or behaviour of the patient, the patient becoming more somnolent, Cheyne Stokes respiration, pupils are small and equal and react briskly to bright light, Doll's eye phenomenon, and the pupils will dilate when the skin of the ipsilateral side of the neck is pinched. As herniation progresses clinical signs of mid brain failure develop. The respiratory pattern changes from cheyne stokes respiration to sustained central hyperventilation, the pupils dilate moderately and become fixed at mid position, the ciliospinal reflex disappears, the Doll's eye phenomena becomes more difficult to elicit, coma progresses beyond the stage of purposeful response and is replaced by bilateral decerebrate rigidity in response to noxious stimulus, signs referable to lower pons and upper medulla gradually develop as the condition progresses. Hyperventilation is replaced by a more or less regular breathing pattern often with a rapid rate and shallow depth, pupils become dilated and fixed at midposition, there is negative dolls eye movements, response to noxious stimuli is flaccid and bilateral babinskis are elicitable. Terminally the patient enters the medullary stage where respiration is slow, irregular in rate and depth and interrupted by deep sighs or gasps. The pulse rate is variable and the blood pressure falls. Death is usually from respiratory failure.

In the syndrome of uncal herniation and lateral brainstem compression the expanding lesions developing in the lateral portion of the middle fossa or in the temporal lobe may be expected to shift the medial edge of the uncus and hippocampal gyrus toward the midline and through the incisural notch. Earliest sign is the unilateral dilating pupil. This is followed by an external third nerve paralysis, patient becomes stuporous and then deeply comatose. Progression results in symptoms similar to those seen in central diencephalic mid brain syndrome.

Stewart (1975) found deteriorating level of consciousness to be the commonest presentation in patients with intracranial space occupying haematomas. This was followed by upper motor neurone limb signs, a situation very similar to the one in the present series.

There may be no obvious indication for immediate surgery at the time of admission, this arising later. On the other hand, the indications may be there and the delay to take the patient to theatre may be due to avoidable or unavoidable circumstances. On many occasions at the Kenyatta Hospital the delay was avoidable. Such as obtaining theatre facilities, lack of blood, delay in obtaining neurosurgical opinion and delay in obtaining an angiogram. Such factors contributed to the delay in instituting early operative management. It would have been interesting to see if there's any relationship between delay in operation and operative mortality, but this was not done in the present study.

Seventeen percent of the head injuries had other associated injuries. Associated injuries can determine the outcome of a head injury just as a head injury can determine the outcome of the associated injury. The patient may be successfully nursed through the most difficult period only to find during attempted rehabilitation that the patient has an ignored posterior dislocation of the hip which would by then have passed the stage of reduction. Greater awareness of such salient points of associated injuries is necessary. Most of the associated injuries are more open and the problem to be solved is that of priorities. Probably chest injuries are the most dangerous of all the associated injuries as far as the head

injury is concerned.

Failure to appreciate the severity of the chest injury and institute appropriate measures may result in deterioration of the patients condition. The brain can tolerate very little periods of anoxia and any rise in blood carbon dioxide levels usually results in worsening of the already existing brain oedema (Lewin).

About 80% of the multiple injury patients have brain involvement (Sevitt). The Management of such patients requires setting out of priorities, if tangible results are to be obtained (Evans). The first stage should be at casualty and will usually be aimed at immediate life saving measures, such as clearing of the airway, stopping any external bleeding and attempts at diagnosing severe chest injuries such as open wound of the chest etc. The second stage is aimed at correction of blood volume. The third stage is to deal with any conditions which are directly responsible in the deteriorating condition of the patient, such as insertion of an intercostal tube, institution of intermittent positive pressure assisted ventilation, craniotomy for evacuation of a haematoma, laparotomy for internal bleeding, etc.

The fourth stage is the treatment of injuries causing severe loss of function if there is delay, such as injury to the spinal column, evidence of ischaemia in a limb, serious eye injuries, severe facial injuries, dislocations, fracture of a long bone interfering with nerve function and soft tissue tension in a limb. The fifth stage is the treatment of injuries not seriously compromised by delay.

Sevitt (1968) found that multiple injuries were very common after fatal road accidents, 70% had associated head injury and 45% had associated chest injury. Head injury is therefore the commonest injury in the patient with multiple injuries, and this with chest injuries contribute to the mortality. Sevitt also found that 50% of the victims of fatal road accidents were pedestrians, explaining the high frequency of Pelvic and lower limb fractures that was found. This could also be an explanation for the results at the Kenyatta National Hospital where fractures of the long bones formed the largest group of associated injury. Nearly 70 per cent of the subjects involved in road traffic accidents were pedestrians.

The importance of appreciating the presence of other injuries is very obvious especially from the prognosis point of view. Nearly 28 per cent of the multiple injured patients have associated head and chest injuries.

A diagnosis of soft tissue injury was made in those patients who had scalp injuries with no associated skull injury, who in most cases were discharged after not more than two days of hospitalisation. The diagnosis of primary brain injury was made in those cases of head injuries which were not subdural, intracerebral or extradural haematomas, but had associated skull fracture or a potentially severe head injury necessitating prolonged hospitalisation.

These cases were further classified into mild, moderate and severe head injuries. The severe head injuries were those with intracranial haematomas, those who died and those whose nature of injury resulted in prolonged hospitalisation in a state of depressed consciousness. The moderate head injuries were those with fractures

of the vault and a moderate period of hospitalation in a state of clouded sensorium, the mild head injuries were in most cases the patients with soft tissue injuries.

There were 16 cases of extradural haematomas (4.6%) and 17 with subdural haematomas (4.8%). In extradural haematoma, the clot is located between the skull and the dura. It usually results from injury to the extracerebral blood vessels, usually middle meningeal artery or veins or both, or dural sinuses or from the small vessels connecting the dura and the veins of the bony diploe. 90% of the cases are associated with linear skull fractures (Gurdjian 1965). Extradural haematomas occur as a complication in approximately 2% of head injuries requiring hospital care. It is usually caused by blunt low velocity blows to the head such as those suffered in falls or in fights. Usually there's a fracture crossing middle meningeal groove resulting in laceration of arteries and veins. Tear of the lateral sinus or of blood vessels entering the sinus caused by a linear or depressed fracture results in an extensive epidural haematoma in the posterior fossa. Tears of sagittal sinus are also implicated (Gurdjian, 1942).

In extradural haematoma of middle meningeal origin the lesion is usually unilateral. The classic picture of a short period of unconsciousness, followed by a lucid interval, followed in turn by unconsciousness and focal signs has been recognised for nearly a hundred years. The earliest sign is dilation of the ipsilateral pupil followed by a rapid deterioration of the state of consciousness.

A lucid interval is the period of alertness between the initial injury and secondary coma, and it represents the period between the unconsciousness of cerebral concussion occurring at the time of impact, and the beginning of diencephalic stupor and coma resulting from brain displacement and incisural herniation, suggestive of an intracranial haemorrhage. It is most commonly seen in extradural haematomas. The presence or absence of a lucid interval depends upon the depth and duration of the initial unconsciousness and the rapidity of expansion of the clot. Therefore the lucid interval may be absent or may range in duration from fifteen minutes to days or even months. If the source of the bleeding is arterial the interval will be short, 6-18 hours or less. Motor signs may be in the form of contralateral hemiparesis due to direct cerebral involvement by the expanding mass or ipsilateral hemiparesis when the expanding haematoma displaces the brain medially thus tending to press the contralateral cerebral peduncle against its neighbouring incisural border. Generalised convulsive seizures may be present and occasionally Jacksonian types are found. The cerebrospinal fluid is usually bloody with increased pressure. Where progression of the lesion is so rapid as to preclude angiography or the facilities for this are not available exploratory trephination is permissible. The use of osmotic dehydrating agents may lessen the acutely increased pressure and thus gain a brief respite in these rapidly progressive lesions and permit a satisfactory result. The time gained is a result of dehydration of the brain, not a lessening of haematoma size. Due to the danger of increase in the rate of mass formation accompanying the use of these agents, operative evacuation of the clot must promptly follow. 20% mannitol, 30% urea, and 50% glucose are all effective.

.../7

Gallagher (1968) reported 167 cases of extradural haematomas over a 25 year period, an average of about six patients per year.

At the Kenyatta National Hospital 16 patients were seen in six months, which is quite high. Gallagher reported a low incidence of extradural haematomas in blacks despite the fact that many of them had been admitted into the neurosurgical unit with cranial injuries and he concluded that the cranial vault in negroes was very thick making them less susceptible to fractures of the skull and therefore less susceptible to middle meningeal haemorrhage. This is in contrary to the findings at Kenyatta Hospital where extradural haematomas constituted 4.6 per cent of all head injuries. The high number of patients who sustained assaults in this series (40%) may be one of the reasons as extradurals are usually caused by blunt low velocity blows.

The strength of the dural attachments to the skull plays a major role in the development of the haematoma. A blow causes the dura to become separated from the skull directly beneath the site of impact; bleeding starts, the clot fills the extradural pocket and stripping of the dura occurs, resulting in further enlargement of the clot. In most age groups the dura can be separated from the skull rather easily. However, in infants and in the elderly the dura is so strongly adherent to the bone that it is difficult to separate the dura away from it and this explains why extradural haematoma is uncommon in the very young and in the aged. Gallagher reported a surgical mortality of 40% while Jamieson reports an overall mortality of 15% in 167 cases.

17 patients had subdural haematoma. Stewart in a retrospective study found 65 patients with subdural haematoma and 10 with extradural haematoma over a 13 month period at the Kenyatta National Hospital in 1975. He included chronic subdural haematoma patients without history of trauma seen in the medical wards, but these were specifically omitted in this study.

Subdural haematoma is a collection of blood beneath the dura and is a common complication of head injury. The condition may be classified into acute subacute and chronic depending upon its size, rapidity of growth and severity and rapidity of appearance of signs and symptoms that it produces. In acute subdural haematoma the bleeding is severe enough to produce signs and symptoms early after the injury (up to 24 hours). In subacute subdural haematoma the bleeding is less severe and is tolerated with significant symptoms for two to 10 days. In chronic subdural haematoma the initial haemorrhage produces no signs for several weeks or longer. Subdural haematoma occurs in about 5% of all head injuries (Feytag) (4.8% in the Kenyatta National Hospital).

Acute subdural haematoma has a higher mortality, 50-80%, subacute 25%, and chronic 15%. 20% of the subdural haematomas are usually bilateral. The haematomas may be located anywhere in the cranial cavity. The site of predilection is over the convexity in the frontal temporal parietal region. Posterior fossa is rarely involved.

Tear of the connecting veins between the cortical surface and the dura or cerebral contusions that tear venous or arterial channels are the commonest causes. Intracerebral bleeding may sometimes extend to the subdural space. Acute subdural haematoma may be difficult to diagnose because of the presence of associated



cerebral injuries. It commonly follows high speed impact, eg. automobile accidents. The impact may result in contusions and lacerations of the brain and brain stem and the clinical signs of generalised brain and brainstem damage will over shadow the signs of the expanding clot. It is the presence of these associated areas of damage that increases the mortality rate when compared to epidural haematoma. When the clot is the sole lesion the operative treatment is rewarding. The commonest clinical findings in a patient with acute subdural haematoma is an unconscious patient or a rapid deterioration of the state of consciousness, pupillary dilatation, loss of extra ocular movements, retinal haemorrhages, convulsions, and later decerebrate rigidity and midbrain pontine signs. The diagnosis largely depends on the history and physical findings supplemented with angiography where available, or exploratory trephination. The clot should be evacuated.

No patient had an intracerebral haematoma. Haemorrhages within the brain of upto 5mm in diameter are described as petechial or punctate lesions. Lesions larger than 5mm are designated as haematomas. In many of the operative findings the presence of petechial is confirmed and probably lack of defining the criteria between a haematoma and a petechia could lead to confusion and result in under diagnosis of the condition. Intracerebral haematoma occurs in less than 2% of patients with head injury. It is more common in the frontal and temporal regions due to stresses occurring in these areas at the time of mass movement from impact (Gurdjian 1968). Anatomical relationship of the frontal and temporal poles to the lesser wing of the spheroid and the roof of the orbit may be implicated. They are frequently found in association with penetrating and perforating wounds.

The clinical picture usually depends on the site. There is usually a lucid interval with increasing headaches. Contralateral paresis may be present as well as stupor or coma. One third of the patients have no associated skull fracture. In one third the fracture is contralateral to the haematoma. Diagnosis is confirmed by angiography either carotid or brachial for the cerebellar lesion. The management is operative although the results are poor, with a 55% mortality. For the Temporal lesion, a subtemporal craniectomy is ideal, the bulging discolored brain tissue being removed by suction to expose the clot, which can then be removed by suction.

For the frontal or parietal lesion, trephination is done or a small properly placed bone flap if the patients condition will permit. The intracerebellar haematoma is best removed by suboccipital exploration or bilateral suboccipital trephine.

Temporal lobe contusions may become surgical problems and deserve excision when clinical and angiographic indications are present, due to the danger of uncal herniation and brainstem damage in the untreated case (Ommaya 1966). Anterior portion of the temporal lobe may be excised, but on the left side one should stay in front of the 6 cm limit from the temporal tip if possible to avoid increasing speech disturbances. The following is a classical example:-

.../9

"M.W. Study No.066 Ward 21 Kenyatta National Hospital

Age	23 years	Sex	Male
Date of injury			29.07.79
Date of admission			01.08.79
Date of discharge			28.08.79

Patient was assaulted with an iron bar as he was heading home from work. He lost consciousness for a short while gradually regained his senses and proceeded home. He was quite alright for the next few days, after which he became irritable, was confused and at times would talk to himself. He was brought to the casualty department by his relatives. On examination in the ward, he was found to have a small bruise on the left side of the forehead and no other external injuries. He was confused but had no other neurological findings. Radiological examination of skull showed no fracture. He was put on head injury observations. On 15.8.79, a carotid angiogram was requested (Xrays: Fig.14).



On the basis of these findings, the patient was explored, first by burr holes which were not very rewarding, so a craniotomy was done. The dura was intact. There was no extradural clot. No subdural clot was found. The brain was oedematous. Needling was attempted using the conventional brain needle, but this was negative and the absence of a significant intracerebral clot was assumed. Diagnosis was temporal lobe contusion. Post operative was put on steroids and he recovered well enough to go home twelve days after the operation".

Probably one could argue that the operation could have had a decompressive effect on his condition and hence speeded up his recovery. Many ways have been suggested to surgically decompress a massively swollen brain, for example subtemporal decompression, Temporal or Frontal lobe resection, splitting of the tentorium or circumferential craniotomy (26 Jnl of Neuro 1968). The last of these has largely been abandoned as being too dangerous. It may lead to development of hydrocephalus due to an extra ventricular block or failure of the procedure to relieve bilateral uncal herniation with resultant aqueductal stenosis. The procedure has also been associated with relocation of the midbrain, and the hazards certainly outweigh the benefits.

In a six months period, 378 patients were admitted and 59 died, an overall mortality of 15.6 per cent. The interpretation of this figure depends upon many factors. What types of head injuries were admitted? Did they need admission? One should not forget out the possibility that cases could have been admitted which did not warrant admission and this could give a falsely encouraging picture. However, the pressure for beds at Kenyatta National Hospital and the fact that all these patients are managed in general surgical wards would make it very unlikely that patients who did not deserve beds would gain their way into the wards.

Nearly fifty per cent of the patients who died did so within 24 hours of admission. 14 of those 118 patients operated on died, an operative mortality of 13.4%.

These operations were for depressed skull fractures with or without an open wound, intracranial haematomas like extradural and subdurals.

Lewin (1976) reports on the gradual improvement of the mortality figures at Cambridge over a period of thirty years, from the initial 9 per cent to 3.7 per cent. The three major killing factors after head injury are intracranial haematoma, cerebral hypoxia and increase of brain bulk. Cerebral hypoxia could be prevented by dealing adequately with respiratory insufficiency.

Nearly fifty per cent of these patients who died did so within twenty four hours of admission. It has been shown that most of the serious head injuries die immediately on arrival and the cause of death is usually severe brain damage. This is the group of head injuries that one could have done very little to influence the ultimate outcome. Only one patient died after prolonged hospitalisation of more than one month.

Sevitt (1969) in a review of 250 subjects who died as a result of fatal road accidents found that 44% of those reaching hospital alive died within 24 hours of injury. He also found that early death after head injury was due to a direct contusion of the brain stem or third ventricular region, but many deaths during the first hours and subsequent days were from the complication of tentorial herniation of the midbrain, following intra cranial haemorrhage and the associated frontal or temporal coup or contre coup lesions.

The management of the head injury can be a great financial burden, and this is an often over looked factor. Proper care of an unconscious patient demands providence of enough nursing staff, physiotherapists, doctors and enough materials like linen, condom e.t.c. Half hourly observations are extremely taxing to the nursing staff and of course to be of any meaning they must be properly carried out. There may be two or three unconscious patients demanding such management at any one moment. Added to all this is the fact that the road to recovery is often a long and tedious one. At the Kenyatta Hospital the demand for beds is extremely high and many of the able patients are given an early discharge to convalescent at home and be followed up in the clinic. Three groups of patients can be identified. The first group is those who were fit enough to go home between the second and fourth day. There were one hundred and fourteen patients in this group, excluding twenty two patients who were discharged within twenty four hours of admission. This group can be taken to indicate the mild head injuries. The second group of patients were those who went home after five to ten days of hospitalisation and the third group of patients were those who stayed for more than ten days.

The mortality rates, both overall and operative are quite satisfactory, although in specialised units overall mortality figures reported are in the order of 5 per cent (Lewin 1967).

In one hundred and fifty patients the outcome was probably not preventable, being due to a primary damage to the brain. Delayed treatment contributed to the severity of forty patients. This was in the form of delay in arriving at the hospital, delay in taking the patient to theatre or a delay in obtaining neuro surgical opinion.

Associated injuries were thought to have had a direct influence to the severity of twenty one cases.

Seventy patients were thought to have been managed in a manner which could have been improved upon, but in majority of the patients the managment was thought to be quite satisfactory.

Head injury frequently produces a combination of physical and mental sequelae, which may become a great handicap (Walpole 1968). It is hoped that a follow up study will be planned in the clinic to study this aspect of head injury.

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APPENDIX 1

HEAD INJURIES ADMITTED INTO K.Y.H.

1. PERSONAL DATA:

NAME.....

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I.P. NO.

--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

STUDY NO.

--	--	--

SEX MALE 1 FEMALE 2

--

AGE IN YEARS

--	--	--

HISTORY OF INJURY.

2. DATE OF INJURY

--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

DATE OF ADMISSION

--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

DATE OF DISCHARGED

--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--	--

3. CAUSE OF INJURY:

- B.T.A. 1
- Assault 2
- Sport 3
- Work 4
- Fall 5
- Home accident 6
- Other 7

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4. TYPE OF INJURY

Blunt	1
Sharp	2
Compression	3

5. R.T.A.

Pedestrian	1
Cyclist	2
Driver	3
F.S.P.	4
B.S.P.	5
Other	6
Unknown	7

6. INJURY TO HOSPITAL TIME (HOURS)

- 2 hours	1
2 - 4 hours	2
4 - 6	3
6 - 12	4
12 - 24	5
24 - 48	6 REASON:
- 48	

OPERATIVE FINDINGS:

7. HOSPITAL TIME TO OPERATION TIME

- 2 hours	1
2 - 4	2
4 - 6	3
6 - 12	4 REASON:
12 - 24	5
- 24	6

8. REFFERAL:

Primary admission	1
From other hospital	2

9. PRIMARY ADMISSION:

Brought by:	Self	1
	Relatives	2
	Police	3
	Fellow traveler	4
	Other	5
	Unknown	6



APPENDIX 3

CLINICAL FINDINGS:

10. VAULT FRACTURE:

Present 1  
Absent 2

11. BASE OF SKULL FRACTURE:

Present 1  
Absent 2

12. DIRECTION OF THE BLOW (IMPACT)

Mid Frontal 1  
Lateral Frontal 2  
Lateral 3  
Postero Lateral 4  
Middle Posterior 5

13. C.S.F. LEAK:

C.S.F. Rhinorrhoea 1  
C.S.F. Otorrhoea 2  
Others 3

14. INJURIES: (OTHER)

Cervical Spine 1  
Chest 2  
Abdomen 3  
Pelvis 4  
Long bones 5  
Other spine 6  
Multiple 7

15. INITIAL DIAGNOSIS:

Primary brain damage 1  
Subdural clot 2  
Extradural clot 3  
Intra cerebral clot 4  
Intra ventricular clot 5

16. CONSCIOUS STATE:

Normally 1  
Drowsiness 2  
Confusion 3  
Stupor 4  
Coma 5

APPENDIX 4

17. FINAL DIAGNOSIS:

Primary brain damage	1
Subdural clot	2
Extra dural clot	3
Intra cerebral clot	4
Intra ventricular clot	5

18. BLOOD ALCOHOL mg/dl:

20 - 40	1
40 - 60	2
60 - 80	3
80 - 100	4
100 - 120	5
120 - 140	6
140 - 160	7
160 - 180	8
180	9

19. BLOOD SUGAR mg%:

20 - 40	1
40 - 60	2
60 - 80	3
80 - 100	4
100 - 120	5

20. EXPECTED OUTCOME:

Good (resume work)	1
Fair (work at lower level)	2
Bad (unable to resume work)	3
Residual mental deficit	4
Residual symptoms	5
Vegetable state	6
Dead	7

APPENDIX 5

21. ORIGINAL WORK LEVEL:

Manual	1
Skilled	2
Clerical	3
Professional/Managerial	4

CRITICAL EVALUATION OF THE MANAGEMENT.

22. MAJOR FACTOR IN SEVERITY:

Primary brain damage	1
Anoxia	2
Delayed treatment	3
Other injuries	4
Multiple factors	5
Lock of "Optimum" case	6

23. FUTURE MANAGEMENT:

Improvement essential	1
Desirable	2
Current satisfactory	3
Excellent	4