THE ASSOCIATION OF SKULL FRACTURES WITH INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY

A DISSERTATION SUBMITTED AS PART FULFILMENT FOR THE AWARD OF DEGREE OF MASTER OF MEDICINE IN SURGERY AT THE UNIVERSITY OF NAIROBI

PRINCIPAL RESEARCHER

DR SAMSON MISANGO, MBChB (Univ. of Nbi)
RESIDENT MMED STUDENT,
UNIVERSITY OF NAIROBI
DEPARTMENT OF SURGERY

SUPERVISOR:

DR KIBOI J.G.

DECLARATIONS

This dissertation is my original work that has not, to my informed knowledge, been presented for a degree in any other university.

Signed ..... Date 25/3/08.

DR. SAMSON MISANGO
MBChB (NBI)
Reg. No. H58/7630/04

This dissertation has been submitted with my approval as university supervisor

Signed ..... Date 25/3/08.

MR. KIBOI J.G
MBChB, M.Med (Surgery) U.O.N

CONSULTANT NEUROSURGEON/LECTURER
UNIVERSITY OF NAIROBI
DEPARTMENT OF SURGERY
NAIROBI

DEDICATION

This dissertation is dedicated to my late sister, Anne Khasoa Misango, who looked up to me and gave me a sense of importance and self belief, way back in my teenage and early college life. May she rest blissfully as I fulfil all her expectations of me.

AKNOWLEDGEMENT

I wish to thank my supervisor, Dr. Kiboi J.G. for his constant availability in guiding me throughout this project, and his encouraging comments at the appropriate moments.

I acknowledge my wife and daughter for their understanding during my long absences from the house as I endured the long hours to balance my work and studies as I worked on my project. Acknowledgements also go to all my other family members and friends for being patient with me as a student.

Sincere gratitude to Janet, the pleasant statistician, without whom all the data in my possession would have remained meaningless.

Last but not least, my sincere appreciation to all the University of Nairobi department of surgery members and colleagues who gave me valuable input during the project, and also the Kenyatta National Hospital ethics and research committee for their guidance. I cannot forget all my research assistants and all those who contributed their time and resources to assist me in my study.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

APTT	Activated Partial Thromboplastin Time
ATLS	Advanced Trauma Life Support
AVPU	Alert, Verbal, Pain, Unresponsiveness
BI	Brain Injury
CAT	Computed Axial Tomography
CBF	Cerebral Blood Flow
CBV	Cerebral Blood Volume
CSF	Cerebral Spinal Fluid
CT	Computed Tomography
DIC	Disseminated Intravascular Coagulopathy
ECG	Electrocardiography
EDH	Extradural Hemorrhage/Hematoma
GCS	Glasgow Coma Scale
HT	Head Trauma
ICB	Intracranial Bleeding
ICH	Intracranial Hemorrhage/Hematoma
ICU	Intensive Care Unit
KNH	Kenyatta National Hospital
MRI	Magnetic Resonance Imaging
PT	Prothrombin Time
TBI	Traumatic Brain Injury

SUMMARY

Six hundred and seventy five patients were admitted at the Kenyatta National Hospital with blunt head injury in the period between June 1st 2007 and October 12th 2007. Out of this number, three hundred patients with skull fractures were recruited into the study having been admitted into the respective units and fulfilling the inclusion criteria. This prospective study was conducted in the period, with approval from the Ethics and Research Committee of the Kenyatta National Hospital.

In the study population, 274 patients (91.3%) were males and 26 patients (8.7%) were females. Out of the total population, 124 patients (41.3%) were unemployed while 84 were self employed (28%), casual labourers were 54 (18%), 20 (6.7%) were students and 16 were formally employed 18 (6%).

The causes of injury resulting in the skull fracture were found to be assault (49.7%), RTA (31.7%), falling objects (16%), sporting injuries (1.7%) and others (1%). At presentation, 74.7% of the patients had mild head injury, 15% had moderate head injury, 7.7% had severe head injury and 2.7% had critical head injury. Pupilary state was abnormal in 14.7% of the patients, neurological deficits were observed in 17.3%, and signs of basal skull fracture were evident in 20.7% of the patients.

Intracranial bleeding was present in 24.3% of the patients, presenting as intracerebral bleed in 10.7% of the total population, extradural in 7%, subdural as 3.7%, subarachnoid in 0.3% and combination of bleeds in 2.7%. The location of the bleed was parietal in 18.7%, temporal in 3%, and combination in 2%. Most commonly fractured skull bone was the parietal bone (43%) then basal bones 26%, frontal 21.3%, occipital 5.3%, temporal 2.3% and multiple bones in

2%. The fracture was simple in 57% of the cases, compound linear in 25.3%, compound depressed in 10.7% and simple depressed in 6.3%.

Majority of the patients were treated non-surgically (61.3%) while the rest (38.7%) were operated upon. By the end of one week of admission, 87.7% of the patients had been discharged, 10.7% had died and 1.7% were still receiving inpatient care.

Upon statistical analysis of the results, there was a positive correlation in the presence of neurological symptoms in patients with skull fractures and the presence of intracranial bleeding. The results were in conformity with earlier studies done internationally. The site of the bleeding could however not be reliably determined by the location or type of fracture.

INTRODUCTION

Skull fractures have been associated with head trauma from biblical days. The earliest reference to head trauma is best illustrated by Albrecht Durer's depiction of the biblical slaying of Abel by Cain. Had Cain desisted at this point, as we are told he did not, Abel might have required the control of arterial bleeding, a wound toilet, the care of an open head wound, and treatment for a fracture of the vault of his skull (1).

The association of head trauma, skull fractures and intracranial bleeding has been studied by various authors over time. This dissertation explores the contradictory conclusions that have been made during that period, and the unresolved issues relating to the occurrence, diagnosis and management of these injuries.

There is no standard definition for Head Injury, but it can be described as head trauma with occurrence of neurological disturbances. The Neurological disturbances may be transient, long term or permanent. Synonyms for head injury used in this text include head trauma (HT), traumatic brain injury (TBI), brain injury (BI).

The study will utilize the Glasgow Coma Scale (GCS) in assessing severity of injury. It has consistently been a good prognostic indicator in most studies done so far, and is universally accepted as an objective assessment of degree of injury (2).

The pathophysiology associated with head injury is broadly divided into primary and secondary injuries. This dissertation will be looking at the association of skull fractures with intracranial bleeding, which are both primary injuries in the head injured patient.

The major focus in the management of acute closed head injury is the prevention of secondary injuries and the preservation of neurological functions not damaged by the primary injury. This is achieved by adhering to the accepted protocols of head injury management (3).

A skull fracture is defined as an abnormal break in the continuity of the bony covering of the brain. The biomechanics involved negate against the spontaneous occurrence of a skull fracture, except in the setting of a pathological fracture. A non linear association exists between the force of impact and the occurrence of skull fractures. The fracture may be insignificant, but when associated with an altered level of consciousness, or a focal neurological deficit, it may be an indicator of an intracranial hematoma or cortical contusion (4).

Skull fractures are classified as either linear or depressed. Linear fractures can either be skull vault or skull base fractures. Vault fractures and depressed fractures can either be closed (simple) or open (dirty/contaminated/compound). Intracranial Bleeding (ICB), also called Intracranial Hematoma (ICH) or Intracranial Hemorhage (ICH), is defined as the pathological accumulation of blood within the intracranial compartment. The bleeding may be intra axial i.e within the brain tissue e.g. intracerebral bleed, or extra axial i.e. outside the brain tissue e.g. EDH, subdural bleed, subarachnoid bleed, intraventriular bleed. When associated with trauma, most of the patients with intracranial hemorrhage also have associated skull fractures, predominantly a linear type. 80 - 85 % of acute extradural bleeding have an associated linear skull fracture. The CAT scan is the definitive tool for accurate diagnosis of the extradural bleeding and other types of intracranial heamorhage (5).

Before the ready availability of CAT scan as a diagnostic tool in head injury, the clinical assessment combined with the XRay findings were an important

indicator of intracranial pathology. This was aptly demonstrated by Miller et al and later disputed by Holmes et al (6,7). However, many centres exist where the CAT scan is still an elite investigative tool, and relying on it to diagnose intracranial pathology in those setups will result in unacceptable delays in surgical interventions where appropriate. Moreover, even in the setup of KNH with available CAT scan, a significant number of patients requiring the investigation encounter several logistical problems prior to having it performed, probably resulting in delays in diagnosing and subsequently affecting their management.

The presentation of head injured patients will depend on the mechanisms of njury and the presence of intracranial pathology.

Management of head injured patients at KNH is complicated by the absence of tandardized admission protocols, especially in regard to what is described as nild head injury. In this category of patients, the criteria for admission is fairly ubjective, leading to inability to objectively assess the management outcomes f different patients with head injury since their treatment modalities will be nfluenced by a variety of factors. The patients to be observed and the patients) be subjected to serial CAT scans remains a subject of research with no niform across board recommendation (5,6).

LITERATURE REVIEW

Historic Background

The first description of a skull fracture is in Edwin Smith's papyrus, the oldest known surgical journal where a conservative and expectant approach towards the management is described. The results of this approach are better compared to the more aggressive and less favorable one described in Hippocratic medicine (1).

Occipital condylar fracture was first described by Charles Bell in 1817, based on autopsy findings (1). It was not until 1962 that the same fracture was described by Ahlgren as an X ray finding and only in 1983 was it described as a CAT scan finding by Peeters (5).

Development – skull vault forms from membranous bone, with intramembranous ossification commencing from the 6th intrauterine week and syndesmotic sutural joints fusing in the 5th decade. Skull base bones form by endochondral ossification of the cartliginous base of the developing cranium. Skull vault growth is driven by growth of the underlying bone, being maximal in the first 12 months after birth, when the brain mass and volume doubles, and fusing complete by the 10th to the 12th year of life (8). The importance of this development is that different types of fractures occur at different age groups with resultant intracranial sequelae being different in the different age groups.

Anatomic Considerations and pathophysiology

The skull is thickened at certain anatomic sites i.e. the glabella, the external occipital protuberance, mastoid process and the external angular process and is reinforced by three arches on either side. The skull vault is composed of cancellous bone (diploe) sandwiched between two tables/layers of lamina bone, the

external lamina (1.5mm thick) and the internal lamina (0.5mm thick). The diploe does not form where the skull vault is covered by muscles, leaving the vault weak at these areas and prone to fractures (4).

The skull is prone to fractures at certain anatomic sites, i.e. the thin squamous temporal and parietal bones over the temples, the sphenoid sinus, the foramen magna, the petrous temporal ridge, the inner surface of the sphenoid wing at the skull base. The middle cranial fossa is the weakest with thin bones and multiple foramina. Other weak areas prone to fracture include the cribriform plate and the orbital roof in the anterior cranial fossa, and the area between the mastoid and dural sinuses in the posterior cranial fossa (1,4). Skull fractures occur in 85-95% of adult cases with epidural hematoma, but they are much less common in children because of the plasticity of the immature calvaria (5).

Intracranial bleeding results from disrupted vessels in the vicinity of the fracture, or remote from the fracture site. The vessels may be arteries (cerebral or meningeal), veins (cerebral or bridging) or sinuses. Epidural hematoma usually results from a brief linear contact force to the calvaria that causes separation of the periosteal dura from bone and disruption of interposed vessels due to shearing stress. Arterial or venous structures may be compromised, causing rapid expansion of the hematoma; however, chronic or delayed manifestations may occur when venous sources are involved. Extension of the hematoma usually is limited by suture lines owing to the tight attachment of the dura at these locations (1).

Unlike the other intracerebral bleeds, EDH is not generated secondary to head motion or acceleration. EDH is mainly caused by structural disruption of the dural and skull vessels commonly associated with calvarial fractures. Laceration of the middle meningeal artery is the most common etiology.

In the posterior fossa, disruption of dural venous sinuses (eg, transverse or sigmoid sinus) by fracture may lead to EDH. Disruption of the superior sagittal sinus may cause vertex EDH (4).

Association of skull fractures with Subdural bleeding is not well studied. However, it is commonly associated with other intracranial events and is thus an indication of severe head injury in the acute setup. Deceleration injuries are often the cause of subdural bleeding from rupturing of veins via a shearing mechanism. Other entities, such as child abuse and ventricular decompression, also can result in subdural bleeding, and spontaneous hemorrhages may occur in patients receiving anticoagulants or patients with a coagulopathy condition. Compression of a dural sinus does not directly cause a subdural hematoma, although compression may result in a venous infarction (4,7).

Cerebral contusions and intracerebral bleed commonly seen in the frontal and temporal lobes may accompany a skull fracture, the so called fracture contusion. However, these are not well studied and no data exists on the association. There is also no data showing skull fracture association with subarachnoid or intraventricular bleed.

Incidences

Universally, simple linear fractures are commonest, (make up 75%) especially in children under 5 yrs. Most involve the temporal bone in 48% and basilar skull fracture in 19-21% of all skull fractures (4,10).

85 – 95% of adult epidural hematomas will have an associated skull fracture (11). The temporoparietal region and the middle meningeal artery are involved most commonly (66%), although the anterior ethmoidal artery may be involved in frontal injuries, the transverse or sigmoid sinus in occipital injuries, and the superior sagittal sinus in trauma to the vertex (10). Bilateral epidural hematomas

account for 2-10% of all acute epidural hematomas in adults but are exceedingly rare in children (5). Posterior fossa epidural hematomas represent 5% of all cases of epidural hematomas. They may be associated with posterior fossa basal skull fracture, with a poor prognosis due to the limited intracranial space in this compartment (12).

Depressed fractures are usually frontoparietal (75%) temporal 10%, occipital 5% and others 10% (13,14). 75–90% of depressed skull fractures are open/compound (15,16). Compound depressed fractures make up 25% of all skull fractures and simple depressed about 6% (16,17). Intracranial bleeding is usually localized to the contused brain parenchyma, the so called fracture contusion.

Biomechanics of fracture:

A fracture depends on a direct force being applied to the skull, with many variables in play including surface area of the force, velocity of the force, point of impact, the age of the patient. There is no direct association of these variables and thus no specific formula to determine the fracture occurrence.

Similar impact loads delivered at similar skull sites in cadaveric skulls have demonstrated a non linear association of fractures with trauma. This complicates our understanding of skull fractures (4).

In experimental studies, it has been shown that a force of 400 – 700kg must be applied to the skull for less than 0.001s to cause a fracture. If the duration is longer than this, the skull accelerates. In adults and older children, the skull has great resistance against compression but weak tensile strength (reversible stretch ability), therefore if a direct force is applied to the skull, it deforms instantaneously, with the weaker tensile strength resulting in the inner table fracturing initially. If the force is significant, the outer table will also fracture. In

children, the skull still has elastic property and can therefore be deformed without fracturing (high tensile strength). The propagation of the fracture depends on the force applied and the local anatomy i.e. the thickness of the bone and the presence of bony ridges (4).

Sutural diastasis is the occurrence of a skull fracture across a suture line. In children, it is not alarming. In adults with fused sutures, it is an indication of significant trauma/force, and intracranial complications may be associated (4).
Remote effects refer to the occurrence of a fracture at a site distant to the point of force application due to the transmission of energy forces through the facial bones or the occurrence of release fracture i.e. blow out fracture. There results a reversal of the energy forces, with compressive forces/strain on the inner table and tensile forces/strain on the outer table (4).

The association of skull fractures with intracranial bleed has been extensively studied. It is generally accepted now that one in four head injury patients with a skull fracture and neurological symptoms will have an intracranial event, while in the absence of neurological symptoms, the risk is 1 in 6000 (18,19,20).

Linear Skull Fractures result as a consequence of low energy blunt trauma applied over a wide surface area of the skull. It runs through the entire skull thickness i.e outer table, diploe and inner table. It is by itself of no clinical relevance except in situations where it runs through vascular channels, venous sinus groove or a suture line where it may cause epidural hematoma, dural sinus thrombosis/epidural bleed or sutural diastasis respectively. One in four patients presenting with a linear skull fracture and associated neurological symptoms will be found to have an intracranial bleed (18,19,).

A linear skull fracture line must be distinguished from a suture line as seen on a plain radiograph.

Differences Between Skull Fractures and Sutures

Fractures	Sutures
• Greater than 3 mm in width • Widest at the center and narrow at the ends • Runs through both the outer and the inner lamina of bone, hence appears darker • Usually over temporoparietal area • Usually runs in a straight line • Angular turns	• Less than 2 mm in width • Same width throughout • Lighter on x-rays compared with fracture lines • At specific anatomic sites • Does not run in a straight line • Curvaceous

A Basilar Skull fracture is in essence a linear skull fracture involving the base of the skull. It is usually associated with a dural tear and is found at specific points of the skull base. The close association of the paranasal and mastoid air sinuses with the dura/skull base usually renders these fractures compound/open. Basilar skull fractures are associated with significant head injury and are an indicator of intracranial sequelae, especially if associated with neurological symptoms (12, 21). Literature is sparse in regard to the incidence of these fractures and the association with the intracranial bleed that results.

Basilar skull fractures can be divided anatomically according to the fossae location, i.e. anterior, middle or posterior fossa. The probability of intracranial bleeds occurring in each of these situations is not well studied, as the diagnosis of the respective fractures is mostly clinical, relying on the specific cranial nerve lesions for localizing the injury. What is not in doubt is the implication of severe injury whenever a skull base fracture is diagnosed, and hence the greater the probability of intracranial bleeds. Posterior fossa fractures are a rare

occurrence but when associated with an intracranial bleed it can be rapidly fatal due to the relatively small and crowded intracranial compartment and the vital brain stem structures in close proximity (12). Cranial nerves 6^{th} – 12^{th} are at risk of injury in these fractures, and there is an association with cervical spine injuries, which must be looked for (12,22).

Due to the insensitivity of radiological scans in diagnosing these fractures, this study will rely on the recognized clinical symptoms to diagnose skull base fracture, whether radiologically evident or not (12).

Depressed Fractures may be simple or compound. Simple depressed fracture are common in children, resulting from birth injuries and fall from a height. A depression of > 5mm (the skull width) is likely to have injured the dura. In a pond fracture (ping pong fracture), inner table and dural are intact. There is no qualified association with clinically significant intracranial events (22).

Compound depressed fractures are a surgical emergency to prevent infective sequelae. Intracranial sequelae tends to be more localized in this injury as opposed to linear factures (20). The intracerebral bleeding is the so called fracture contusion.

CLINICAL PRESENTATION

A patient with head injury is easily recognized in any accident and emergency unit. Arieta et al in 1997 presented a practical protocol of head injury management in the emergency department (3). They listed the important features in the history as age, sex, mechanism of injury, altered mentation, loss of consciousness, loss of voluntary movements, vomiting, convulsions,

headache and photophobia. Awareness of the above features in each head injury patient will triage the patients and individualize their management.

Children are at an increased risk of developing intracranial bleeding with relatively minor trauma due to their relatively thin protective skull (9). Further, due to the elastic nature of their skull, extensive brain parenchyma injury and bleeding may occur in the absence of a fracture (4). Males are more at risk of sustaining severe trauma as compared to females and the presence of a skull fracture in a male suggests a severe force was involved since the male skull is thicker and stronger than the female skull (4,14,17). The mechanism of injury has a direct bearing on the severity of the injurious force and also influences the possibility of associated injuries in other body systems. The loss of consciousness and voluntary movements, headaches, vomiting, convulsions and photophobia are all suggestive of an intracranial event and will influence further evaluation (6).

Simple linear fractures may only present with a boggy scalp swelling, with a range of neurological signs and varying levels of consciousness depending on the site and extent of the intracranial sequelae while compound fractures may have associated evidence of a dural laceration with CSF leak or brain herniating through the wound. Depressed skull fractures may present with features of focal contusion effects over and above the head injury presentation while skull base fractures will present with a myriad of symptoms, depending on the location of the fracture within the cranial fossa and the associated intracranial sequelae. Other symptoms suggestive of skull base fracture will include rhinorhoea, otorrhoea, panda sign, and battle sign (23). Skull base fractures in the posterior cranial fossa, when associated with intracranial bleeding, have a poor prognosis due to the limited space in the fossa and the close proximity with vital brain stem structures (12).

Patients with skull fractures and accompanying epidural hematoma (E
have external evidence of head injuries and systemic injuries may
present. Depending on the force of impact, patients may present with
consciousness, brief loss of consciousness, or prolonged loss of consc
The classic lucid interval will occur in 20-50% of patients with EDH (
severe intracranial hypertension, a Cushing response may occur. This
systemic hypertension, bradycardia, and respiratory depression. This
usually occurs when cerebral perfusion is compromised by
intracranial pressure (19,20).

Subdural hematomas may be clinically silent when small and discover
when imaging of the brain is performed as part of the workup for traum
patients may complain of headache or dizziness when an isolated
hematoma is present. When the hematoma is larger, symptoms usuall
from mass effect on the brain tissue or from adjacent parenchymal
Decreased mental status, unsteady gait, headache, deviated gaze, and res
depression may be presenting symptoms (19).

Common symptoms of subarachnoid bleeding include headache,
vomiting, restlessness, fever and nuchal rigidity caused by blood
subarachnoid space. Intraventricular bleeding will present with non
symptoms and is an indicator of more severe head injury, while paren
bleeds will give local space occupying effects and neurological distu
depending on the position of the bleed (20).

EVALUATION

Head trauma patients often have multiple organ injuries. Irrespective
pattern of skull fracture or the presence or absence of intracranial
assessment of patients with severe head injuries involves

Patients with skull fractures and accompanying epidural hematoma (EDH) may have external evidence of head injuries and systemic injuries may also be present. Depending on the force of impact, patients may present with no loss of consciousness, brief loss of consciousness, or prolonged loss of consciousness. The classic lucid interval will occur in 20-50% of patients with EDH (11). With severe intracranial hypertension, a Cushing response may occur. This involves systemic hypertension, bradycardia, and respiratory depression. This response usually occurs when cerebral perfusion is compromised by increased intracranial pressure (19,20).

Subdural hematomas may be clinically silent when small and discovered only when imaging of the brain is performed as part of the workup for trauma. Some patients may complain of headache or dizziness when an isolated subdural hematoma is present. When the hematoma is larger, symptoms usually result from mass effect on the brain tissue or from adjacent parenchymal injuries. Decreased mental status, unsteady gait, headache, deviated gaze, and respiratory depression may be presenting symptoms (19).

Common symptoms of subarachnoid bleeding include headache, nausea, vomiting, restlessness, fever and nuchal rigidity caused by blood in the subarachnoid space. Intraventricular bleeding will present with non specific symptoms and is an indicator of more severe head injury, while parenchymal bleeds will give local space occupying effects and neurological disturbances, depending on the position of the bleed (20).

EVALUATION

Head trauma patients often have multiple organ injuries. Irrespective of the pattern of skull fracture or the presence or absence of intracranial bleeding, assessment of patients with severe head injuries involves a primary and a

secondary survey according to the Advanced Trauma Life Support (ATLS) protocols (24). The primary survey is a focused physical examination directed at identifying and treating life-threatening conditions present in a trauma patient and thereby preventing secondary brain injury. The primary survey involves assessing the following:

A – Airway

B – Breathing

C – Circulation. Hypertension with bradycardia and respiratory changes indicate a cushing response. Hypotension in a head injured patient may indicate an associated spinal cord injury.

D – Neurologic Examination. Responsiveness is assessed by the alert, verbal, pain, unresponsiveness (AVPU) system or the Glasgow coma scale (GCS) (appendix 2). Pupilary response and motor ability assess possible intracranial events. While being a good indicator of intracranial bleeding, the abnormal pupil may be misleading in anterior fossa fractures that may directly damage the optic nerve (23).

The secondary survey of patients with head trauma is a detailed examination and assessment of the systems with the goal of identifying all traumatic injuries and directing further treatment (24)

INVESTIGATIONS

Investigations are directed at identifying associated intracranial sequelae in order to plan for definitive management (3). In skull fractures with intracranial bleeding, the diagnostic investigation of choice is a CAT Scan (22).

Radiology:

Skull X-rays

These have been the standard radiological investigation in head injuries, and still have their place, even with the introduction of CAT scans. The advantages

of skull x-rays include the relative smaller costs involved. A skull xray will pick out the majority of linear fractures. Air fluid levels are well shown within the para-nasal sinuses and cranium, and the cranio-cervical junction is well delineated. On a skull x-rays, the majority of adult patients have a calcified pineal gland and therefore in departments with no access to CAT scans, a skull x-ray may reveal midline shift due to a mass lesion. Previously, in the absence of a CAT scan, management plans would be made on the result of the skull x-ray (3). However, most authors are in agreement that evaluation of a head injured patient is incomplete without a CAT scan and thus condemn the practice of basing management decisions on a skull Xray (5,7,25). The diagnosis of a base of skull fracture remains clinical and may not be shown on skull x-rays but the associated radiological signs of pneumocephaly and air-fluid level in the frontal or sphenoid sinuses suggest the presence of such a fracture. The diagnosis of a skull fracture in the absence of neurological symptoms is of no added value in the management of head injured patients (3,25). A skull fracture in the presence of neurological symptoms is suggestive of severe head injury and intracranial sequelae will need to be looked for. This can only be picked out by a CAT scan, which puts into question the value of a skull X-ray.

Patients who require a CAT scan do not require a skull x-ray. (3)

CAT *Scanning*

This has revolutionized the management of trauma, in particularly head injuries, with good resolution of the cranial vault on axial bone windows' and the intracranial contents on 'soft tissue windows', but there are limitations with imaging the posterior fossa, while for base of skull imaging, coronal scans must be done. Two-dimensional CAT scanning in trauma patients is sufficient in the radiological assessment of skull fractures, with no further information gained from three-dimensional scanning. It is the investigative tool of choice to detect

surgically significant intracranial hemorrhage (3). The CAT scan is also used to monitor the progression of an intracranial bleed and thereby dictating the time of surgical intervention (25).

Magnetic resonance imaging

This is not superior to CAT scanning in the acute assessment of head injured patients, due to the length of time taken for each scan, the need to use non ferro-magnetic anesthetic equipment and the poor resolution of the bone. It is of ancillary value for suspected ligamentous and vascular injuries (25).

OTHER INVESTIGATIONS

Biochemical confirmation of CSF leak, baseline blood investigations especially in the presence of bleeding, blood biochemistry for amylase and/or lipase, coagulation profile, grouping and cross matching blood, arterial blood gas analysis, blood and urine toxicology screens and wound cultures are all investigations that will need to be individualized for the head injured patient (21,25). ECG is required to pick re entry dysrythmias, and Xenox scanning to monitor cerebral perfusion are useful in the ICU setup but the latter is not easily available and the costs are prohibitive (26,27,28).

Monitoring of Intracranial Pressure (ICP)

ICP monitoring has been accepted as a vital ingredient of severe head injury management, infarct, most literature supports it as a standard of care in severe head injury though there has been no conclusive prospective, randomized trial showing benefits of this monitoring (26). Various modes of monitoring are in use including external intraventricular drains, subarachnoid and epidural monitors, intraparenchymal monitors and lumbar drains (27,28).

TREATMENT

May be surgical or non surgical. A neurosurgical consultation is mandatory in all head injury patients

Non surgical Care (Medical Care):

The goal of medical care of patients with head trauma is to recognize and treat life-threatening conditions and to eliminate or minimize the role of secondary injury. Patients with severe head trauma are at increased risk of developing cerebral edema, respiratory failure, and herniation secondary to the increased ICP. Frequent serial assessments of the neurologic status must therefore be performed (3,29). They should be nursed with the head elevated to 30 degrees to improve venous drainage and therefore decrease the ICP without affecting the CBF.

The Brain Trauma Foundation has developed guidelines regarding the medical management of patients with severe head injury. These guidelines suggest that cardiopulmonary resuscitation should be the foundation upon which treatment of intracranial hypertension must be based. They also state that, in the absence of any obvious signs of increased ICP, no prophylactic treatment should be initiated because this may directly interfere with the optimal resuscitation process. In the guidelines, A = airway management, B = breathing, C = circulation/cardiovascular management/C spine stabilization, D = increased ICP and cerebral perfusion management, D.I.C management, Seizure management (28,29). Other considerations of importance will include nutritional support, physiotherapy and rehabilitation.

Surgical Care: Two decades ago, the prompt surgical evacuation of subdural hematomas in less than 4 hours was believed to be a major determinant of an optimal outcome (30,31). Subsequent studies have found that the extent of the original intracranial injury and the generated intracranial pressures are more important than the timing of surgery (11).

Surgical drainage of subdural hematoma is not required in most cases. However, surgical decompression is required in the presence of a rapidly expanding epidural or subdural hematoma that causes an increase in ICP and focal compression (29). Patients with subdural hematoma with midline shift or altered mental status should have the hematoma emergently drained.

The craniotomy and surgical drainage of an epidural hematoma and repair of vessels should be done immediately if signs of increased ICP (altered mentation, focal neurologic signs, pupillary changes, or a midline shift) are present.

Conservative management with close monitoring in an I.C.U or high dependency unit is acceptable if there are no focal neurologic signs, altered mentation, or pressure effects with a midline shift and the hematoma is less than 2cm at its greatest diameter (31).

All patients with compound skull fractures require surgical debridement/toilet and most will require evacuation of the hematoma and receive prophylactic antibiotics. Anticonvulsants have been found to be useful (31,32).

Compound Depressed skull fractures require surgical elevation if the depth of the depression is thicker than the calvaria or the depression is greater than 1 cm, and if bony fragments are causing the compression against the brain tissue. It is a surgical emergency to prevent infective sequelae. Intracranial sequelae tends to be more localized in this injury as opposed to linear factures (22).

Surgery consists of establishing initial wound closure to achieve hemostasis and prevent infection. Definitive surgery must be performed as soon as possible if

there is suspected dural tear, presence of intracranial hematoma or presence of moderate to severe wound contamination (3,29).

The close association of the paranasal and mastoid air sinuses with the dura/skull base usually renders skull base fractures compound/open. There has been no adequate controlled study to assess the benefit of prophylactic antimicrobial therapy in these fractures to prevent meningitis. The working party of the British Society for Antimicrobial Chemotherapy recommends close monitoring of patients with these fractures to diagnose meningitis early and treat appropriately.

Some authors have advocated surgical decompressive craniectomies for patients with increased intracranial pressure refractory to conventional medical treatment. Of 57 patients with head injuries undergoing this procedure, 58% reportedly attained a good outcome (28).

The operative and nonoperative management of intracranial injuries is an ever-evolving area of study and, at present, more a matter of neurosurgical judgment than hard and fast decision rules.

PROGNOSIS

This discussion has delineated a myriad of prognostic factors. Head injuries may result in death, a vegetative state, partial recovery, or full return to work. Each patient presents with a unique baseline neurological make up, mechanisms of injury, secondary complications, and postinjury adjustment and support system.

The most important prognostic factors are probably age, mechanism of injury, postresuscitation GCS score, postresuscitation pupillary reactivity, postresuscitation blood pressures, intracranial pressures, duration of posttraumatic amnesia or confusion, sitting balance, and intracranial pathology identified on neuroimaging (2,18).

The mortality rate of severe head injuries ranges from 25-36% in adults within the first 6 months after injury. Most deaths occur within the first 2 weeks (2).

One study reported that at least 53% of 300 survivors of a severe head injury were either severely disabled or in a vegetative state at the time of hospital discharge (32).

A more recent study found that 29% of survivors were either vegetative or severely disabled after 6 months of follow-up care (2).

Conversely, 82% of 67 patients with mild or moderate head injury experienced a good 1-year outcome, and 73% were able to return to work. However, subjective complaints persisted in a large minority, with more than one third of patients reporting drowsiness, fatigue, forgetfulness, poor concentration, and irritability. Other studies have identified dizziness along with analgesic and psychotropic medication use as predictors of failure to return to work after mild and moderate head injuries (32).

In a study of patients with head injuries in 1987, Frankel, J.E. et al found that all 59 patients who were aged 65 years or older and scored less than 11 on the post - resuscitation GCS either died or were left with severe disability (32). Future studies are needed to determine whether this grim prognostic indicator has more universal validity.

RATIONALE/STUDY JUSTIFICATION

The management of head injury patients at the kenyatta national hospital is never complete without a base line skull xray taken to determine the presense or absence of skull fractures as an indicator of the severity/prognosis of the injury. During the past 6 months (Aug 2006 to Jan 2007), patients admitted to KNH were 82, 76, 76, 63, 80, 55, total 432. Out of this number, skull xray was requested and was performed on all the patients, while the CAT scan was requested in all patients who exhibited neurological disturbances on admission or subsequently in the ward. Of the requested CAT scans, less than 50% were actually done, thereby having an impact on the patient management. Presence of skull fractures have been linked to the occurrence of intracranial bleeding in most studies done elsewhere. However, the studies have been limited to the association of skull fractures with epidural hematoma and no study exists in the literature showing the association, if any, between skull fractures and the other forms of intracranial bleeds. In the developed world, the skull xray investigation has been described as outdated and a waste of resources where "superior" imaging modalities are available as a first line radiological investigative tool (20,22). Despite abundance literature from the west that supports this trend, it has been impossible to translate this practice at KNH, and by extension the rest of the public hospitals in this part of the world. There is no study that has been done locally to determine whether the skull xray should indeed be done away with or whether there is any justification to its continued use in head injury patients. Similarly, there is no local study that shows the association of skull fractures with intracranial bleeding.

This study set out to look at the patterns of skull fractures (as evidenced from a plain skull xray, which was used to diagnose this lesion) in blunt head injury patients, and their relationship with intracranial bleeding. The bleeding studied

was all forms of intracranial bleeding i.e. epidural, subdural, parenchymal, subarachnoid and intraventricular bleeding.

It is hoped that the results of the study will determine whether the presence of a skull fracture on X-Ray in head injury patients admitted at the Kenyatta National Hospital can be used as a pointer of intracranial bleeding in our population , and as a prognostic measure in the management of head injury patients. By extension, the study also set out to assess the usefulness of the plain skull X-Ray as an investigative tool.

STUDY QUESTION

Do skull fractures in blunt head injury have a direct bearing on the incidence of intracranial bleeding in our population?

STUDY OBJECTIVES

MAIN (BROAD) - to determine the association between skull fractures and intracranial bleeding in symptomatic blunt head injury patients

SPECIFIC

1. To demonstrate the association of skull fractures with intracranial bleeding in blunt head injury.
2. To determine the patterns (causes, incidence and types) of skull fractures in patients admitted with blunt head injury
3. To determine the patterns of intracranial bleeding in patients admitted with blunt head injury and skull fractures
4. To determine the effectiveness of the skull xray and clinical indicators in evaluating patients with head injury having intracranial bleeding.
5. To determine current management modes and the duration of hospitalization of patients admitted at the KNH with skull fractures secondary to blunt head injury.

DY MATERIALS AND METHODS

DY DESIGN

was a prospective cohort study

ry with skull fracture, the variable

eding

UDY SETTING

is study was carried out in the

ult general surgical wards and

TUDY POPULATION

he study population consisted

ational hospital with a diagnosis

nd fulfilling the inclusion

urrently presenting at the

Xray imaging as a baseline

skull fracture is recommended

equested for those with

NCLUSION CRITERIA

Patients aged >12yrs

Patients admitted

Patients with a skull

Xray

Patients who

Patients with

STUDY MATERIALS AND METHODS

STUDY DESIGN

This was a prospective cohort study, the common causal factor being blunt head injury with skull fracture, the variable being presence or absence of intracranial bleeding

STUDY SETTING

This study was carried out at the kenyatta national hospital, specifically in the adult general surgical wards and the Intensive Care Unit (I.C.U)

STUDY POPULATION

The study population consisted of all adult patients admitted at the Kenyatta National hospital with a diagnosis of a skull fracture following blunt head injury and fulfilling the inclusion criteria for the study. All head injured patients currently presenting at the Kenyatta National Hospital are subjected to a skull Xray imaging as a baseline study, and any of these patients diagnosed with a skull fracture is recommended hospitalization for observation. A CAT scan is requested for those with neurological symptoms.

INCLUSION CRITERIA

- Patients aged >13ys
- Patients admitted within the study period
- Patients with a blunt head injury and a skull fracture demonstrated on the Skull Xray
- Patients who have given a signed informed consent to be included in the study
- Patients with a CT scan

EXCLUSION CRITERIA

- Patients outside the above age group
- Non consenting patients
- Patients with no skull xray
- Penetrating head injuries

SAMPLE SIZE

This was determined by the Fischer`s formula

$N = Z^2P(1-P)/d^2$

where;

N= sample size to be determined.

Z= confidence limit, corresponding to 95% (1.96)

d = absolute precision of the estimate (0.05)

P = prevalence of intracranial bleeding in blunt head injury with skull fracture, estimated at 25%

N = (1.96 x 1.96) x 0.25 (1 – 0.25) / (0.05 x 0.05)

= 288. To be rounded off to 300 patients

STUDY LIMITATIONS

1. Study was confined to the Kenyatta National Hospital. Although it is a national referral hospital, the findings may not be representative of the entire Kenyan population and may not be reproducible in the smaller centres.
2. The inconsistent availability of the CT Scan facility at the Kenyatta National Hospital caused some delay the data collection

DATA ANALYSIS

The collected data in the questionnaires was recorded in a data sheet using the statistical package for social sciences soft ware (SPSS) and analyzed. This was done with the assistance of a statistician. The data was presented visually in tables, pie charts, and bar graphs. Where indicated, test of significance was applied using either Chi-Square (x square) analysis or Fisher's exact test and statistical significance defined as a p-value less than 0.05.

ETHICAL CONSIDERATIONS

In this prospective study, approval was obtained from the Ethics and Research Committee of the KNH to conduct the study. Several measures were undertaken to ensure absolute confidentiality of the information in these records. These included;

a) All patients were required to give written consent to be recruited in the study. Parental/relative's/guardian's consent was required for the minors and the patients with neurological/ higher centre disturbances.
b) Limiting access of the research data to the principal investigator only during the study
c) Individual names were kept secret and the patients only coded for in case numbers for the study

The published results of this study will be available for use by members of the medical fraternity and access will be through the University of Nairobi, Department of Surgery.

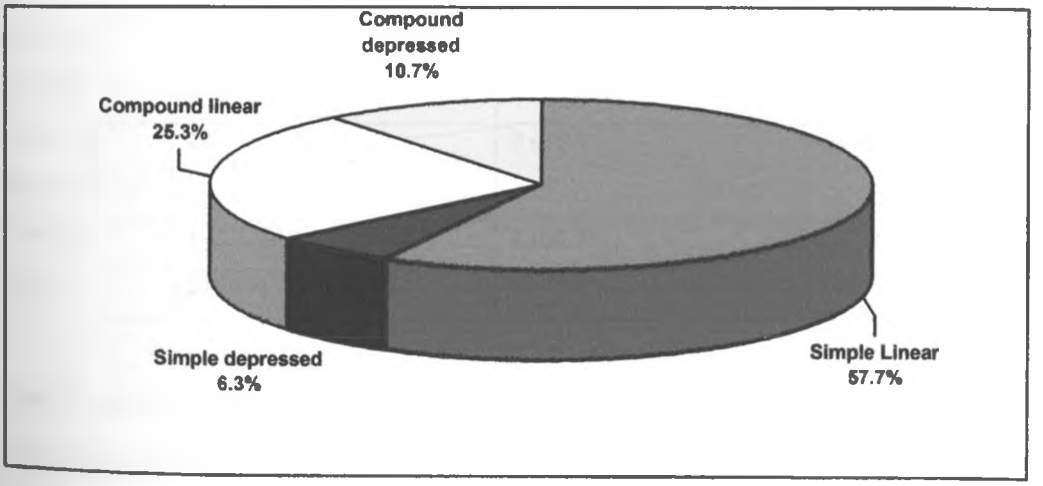
RESULTS

From a total six hundred and seventy five patients hospitalized at the Kenyatta National Hospital with blunt head trauma in the period between June 1st 2007 and October 12th 2007, three hundred patients (44%) had skull fractures and were recruited into the study (table 1). Majority of the fractures were simple linear (57.7%), followed by compound linear (25.3%) then compound depressed (10.7%) and simple depressed (6.3%) (Fig.1). There was a significant male preponderance with 91.3% being males (Fig 2). Most afflicted age group was the young adults (ages 20 – 35yrs) with 215 cases, followed by the middle aged population (ages 36 – 50yrs) (table 3, Fig 4). The incidence of intracranial bleeding in the total population was 24.3% (table 8, Fig 5).

TABLE 1: INCIDENCE OF SKULL FRACTURES IN PATIENTS WITH BLUNT HEAD INJURY.

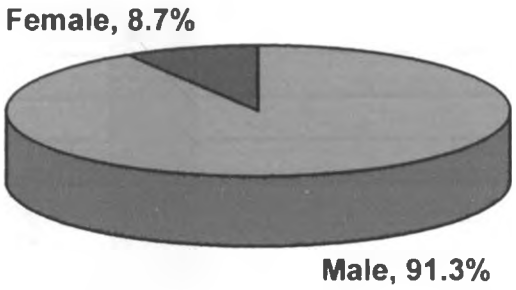
	PRESENT	ABSENT	TOTAL
NUMBER	300	375	675
%	44.44%	55.56%	100%

FIGURE 1: TYPES OF SKULL FRACTURES IN BLUNT HEAD INJURY.



57.7% of the fractures were simple linear and 25.3% compound linear. Compound depressed fractures constituted 10.7% and simple depressed 6.3%. (Fig 1)

FIGURE 2: SEX DISTRIBUTION OF BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURES.



Males comprised 91.3% of the study group and females 8.7%. (Fig 2)

There was a significantly higher risk of males being victims of head injury with skull fractures than females ($p = 0.0015$, confidence interval 95%).

TABLE 2: SEX DISTRIBUTION AND CAUSES OF SKULL FRACTURES IN BLUNT HEAD INJURY.

CAUSE	MALE	FEMALE	TOTAL
	NUMBER (%)	NUMBER (%)	NUMBER (%)
RTA	75 (78.9%)	20 (21.1%)	95 (31.6%)
ASSAULT	146 (98.0%)	3 (2.0%)	149 (49.7%)
SPORTS	4 (80.0%)	1 (20.0%)	5 (1.7%)
FALLING OBJECTS	48 (100.0%)	0 (0%)	48 (16.0%)
OTHERS	1 (33.3%)	2 (66.7%)	3 (1.0%)
TOTAL	274 (91.3%)	26 (8.7%)	300 (100%)

Assault accounted for most of the causes of the head injured patients constituting 49.7%, followed by RTA, falling objects, and sporting injuries comprising respectively 31.7%, 16% and 1.7%. Among the causes captured as others, one female patient sustained a skull fracture during an attempted suicide incident as she victim jumped off a building, one male and one female fell from

FIGURE 2: SEX DISTRIBUTION OF BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURES.

Males comprised 91.3% of the study group and females 8.7%. (Fig 2)

There was a significantly higher risk of males being victims of head injury with skull fractures than females ($p = 0.0015$, confidence interval 95%).

TABLE 2: SEX DISTRIBUTION AND CAUSES OF SKULL FRACTURES IN BLUNT HEAD INJURY.

CAUSE	MALE	FEMALE	TOTAL
	NUMBER (%)	NUMBER (%)	NUMBER (%)
RTA	75 (78.9%)	20 (21.1%)	95 (31.6%)
ASSAULT	146 (98.0%)	3 (2.0%)	149 (49.7%)
SPORTS	4 (80.0%)	1 (20.0%)	5 (1.7%)
FALLING OBJECTS	48 (100.0%)	0 (0%)	48 (16.0%)
OTHERS	1 (33.3%)	2 (66.7%)	3 (1.0%)
TOTAL	274 (91.3%)	26 (8.7%)	300 (100%)

Assault accounted for most of the causes of the head injured patients constituting 49.7%, followed by RTA, falling objects, and sporting injuries comprising respectively 31.7%, 16% and 1.7%. Among the causes captured as others, one female patient sustained a skull fracture during an attempted suicide incident as she victim jumped off a building, one male and one female fell from

a standing position after slipping. Apart from this category of causes, the males dominated in all the other categories as the more likely victims (table 2, Fig 3).

FIGURE 3: CAUSES OF SKULL FRACTURES IN BLUNT HEAD INJURY.

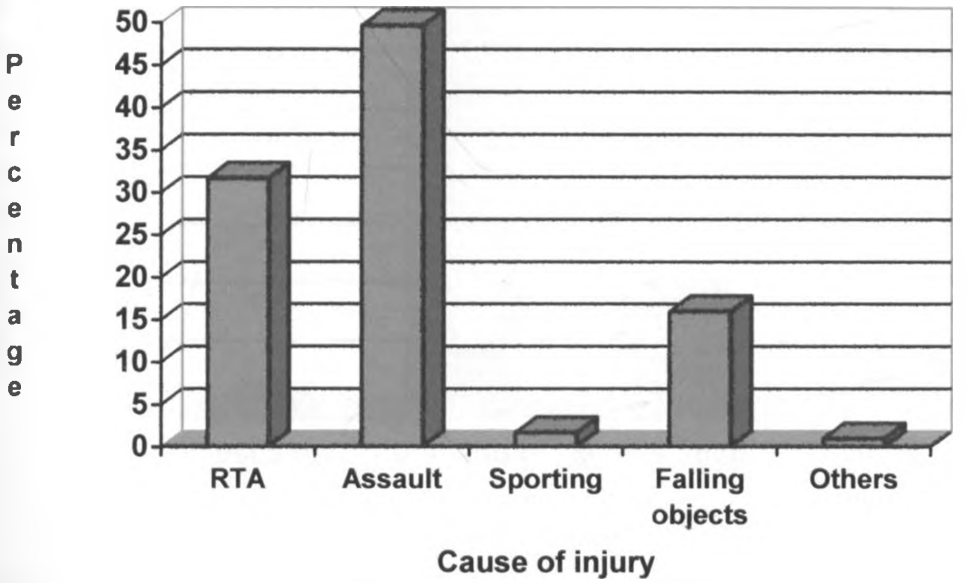


TABLE 3: AGE DISTRIBUTION IN THE INCIDENCE OF SKULL FRACTURES IN BLUNT HEAD INJURY PATIENTS.

AGE (YRS)	INCIDENCE	%
< 20 (TEENAGERS)	2	0.7%
20 – 35 (YOUNG ADULTS)	215	71.7%
36 – 50 (MIDDLE AGED)	62	20.6%
> 50 (ELDERLY)	21	7.0%
TOTALS	300	100%

Most of the patients in the study were young adults, i.e. age group 20 – 35 yrs, constituting 71.7 percent. The youngest individual in the study population was 16yrs while the oldest was 62yrs. There was a significantly higher chance of a young adult presenting with a skull fracture than a middle aged individual ($p = 0.0015$, confidence interval 95%) or a teenager ($p = 0.000$, 95% confidence interval) (table 3, Fig 4).

TABLE 5: CAUSES OF BLUNT HEAD INJURY IN RELATION TO THE TYPE OF SKULL FRACTURE.

TYPE	RTA (%)	ASSAULT (%)	SPORTS (%)	FALLING OBJECTS (%)	OTHERS (%)	TOTAL (%)
SIMPLE LINEAR	79 (83.2%)	40 (26.8%)	4 (80.8%)	47 (97.9%)	3 (100%)	173 (57.7%)
SIMPLE DEPRESSED	9 (9.5%)	9 (6.0%)	1 (20.2%)	0 (0%)	0 (0%)	19 (6.3%)
COMPOUND LINEAR	7(7.4%)	68 (45.6%)	0 (0%)	1 (2.1%)	0 (0%)	76 (25.3%)
COMPOUND DEPRESSED	0 (0%)	32 (21.5%)	0 (0%)	0 (0%)	0 (0%)	32 (10.7%)
TOTAL	95 (31.7%)	149 (49.7%)	5 (1.6%)	48 (16%)	3 (1.0%)	300 (100%)

The majority of fractures were simple linear and most were due to RTA (83.2%), but assault was the dominant cause of compound linear fractures, simple linear fractures and compound linear fractures constituting respectively 45.6%, 6% and 31.5% (table 5).

TABLE 6: LOCATION OF SKULL FRACTURE IN BLUNT HEAD INJURY.

LOCATION	NUMBER	%
FRONTAL	64	21.3%
PARIENTAL	129	43.0%
OCCIPITAL	16	5.4%
TEMPORAL	7	2.3%
BASAL	78	26.0%
MULTIPLE	6	2.0%
TOTAL	300	100%

The bone commonly fractured in skull fractures was found to be the parietal bone at 43%, followed by the basal skull bones at 26%, frontal bone 21.3%, occipital 5.3%, temporal bone 2.3% and multiple sites at 2% (table 6).

TABLE 7: TYPES OF MUTIPLE FRACTURES IN BLUNT HEAD INJURY.

TYPE	NUMBER	%
PARIETAL/TEMPORAL	1	16.7%
OCCIPITAL/TEMPORAL	1	16.7%
PARIETAL/OCCIPITAL	1	16.7%
FRONTAL/PARIETAL	3	50.0%
TOTAL	6	100%

Of the cases with multiple fractures, 3 (50%) sustained fractures of the frontal/parietal bones, while there was one patient in each of the other categories of patents with multiple fractures (table 7).

TABLE 8: INCIDENCE OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURED PATIENTS WITH SKULL FRACTURE.

	PRESENT	ABSENT	%
NUMBER	73	227	300
%	24.30%	75.70%	100%

24.3% of the study population with skull fractures had evidence of intracranial bleeding, while 75.7% had no evidence of intracranial bleeding (table 8, Fig 5).

FIGURE 5: CAT EVIDENCE OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURE.

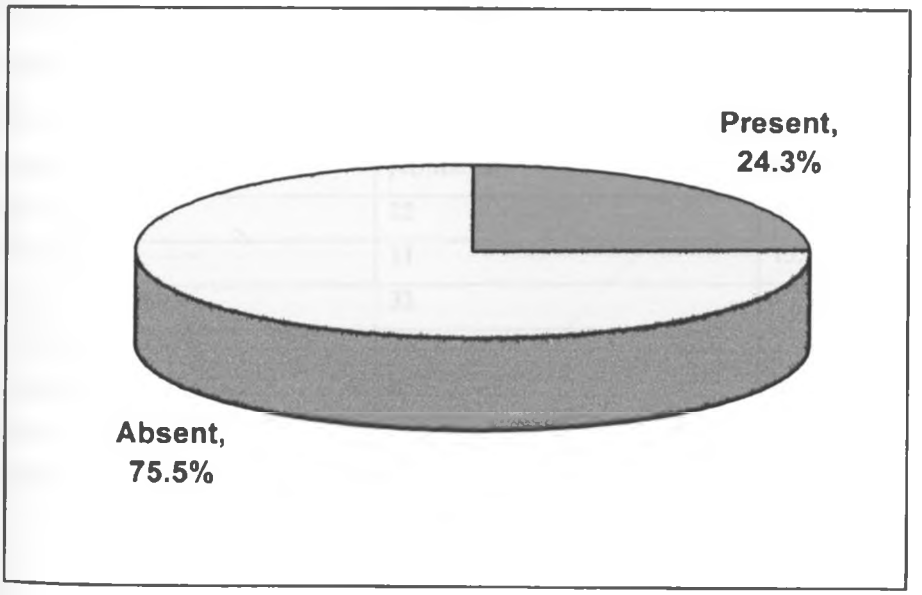
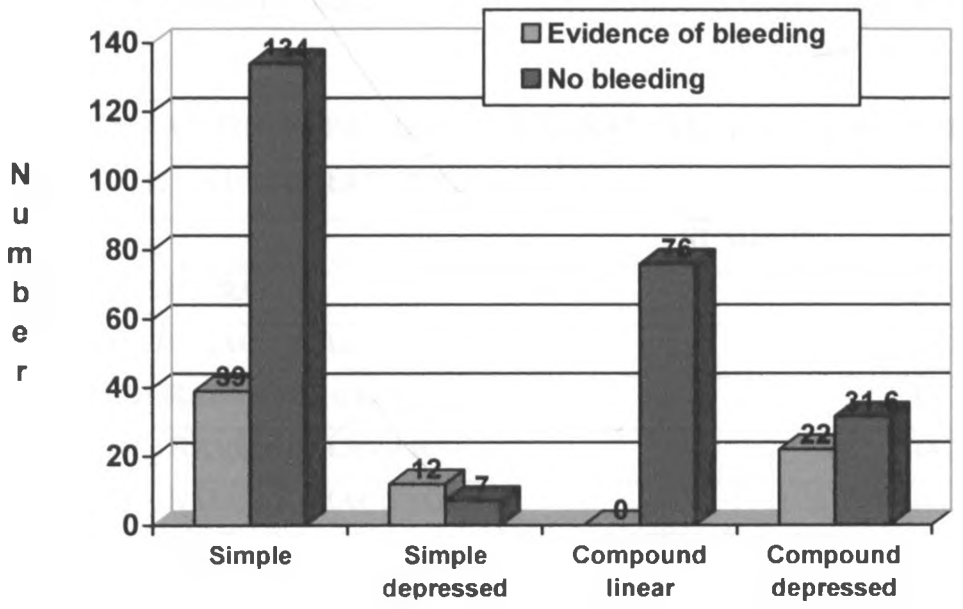


FIGURE 6: ASSOCIATION OF TYPE OF SKULL FRACTURE WITH CAT EVIDENCE OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY PATIENTS.



Among the various fracture types, there was a higher proportion of intracranial bleeding in the simple depressed fractures and least in the compound linear fracture. However, on statistical analysis, no statistically significant association was demonstrated between the type of skull fracture and the presence of intracranial bleeding (p value > 0.005) (Fig 6).

TABLE 9: TYPES OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

TYPE	NUMBER	%
EXTRADURAL	22	30.1%
SUBDURAL	11	15.1%
INTRACEREBRAL	32	43.8%
SUBARACHNOID	1	1.4%
INTRAVENTRICULAR	0	0%
COMBINATION	7	9.60%
TOTAL	73	100%

Intracerebral bleeding occurred in 43.8% of the population followed by extradural bleeding in 30.1% and subdural bleeding in 15.1%. In 9.6%, there was a pattern of intracranial bleeding that encompassed more than one type (Table 9).

TABLE 10: COMBINATION TYPES OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

COMBINATION	NUMBER	
SUBDURAL/INTRACEREBRAL	2	28.6%
INTRACEREBRAL/SUBARACHNOID	1	14.3%
EXTRADURAL/SUBDURAL/INTRACEBRAL	1	14.3%
INTRACEREBRAL/INTRAVENTRICULAR	1	14.3%
EXTRADURAL/SUBDURAL/SUBARACHNOID	1	14.3%
EXTRADURAL/SUBDURAL	1	14.3%
TOTALS	7	100%

There were seven patients with multiple types of intracranial bleeding. Among this category of patient, there were two patients with subdural and intracerebral bleeding, and one each with the combinations as outlined in the table (table 10).

TABLE 11: LOCATION OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

LOCATION	NUMBER	%
PARIETAL	56	76.7%
OCCIPITAL	2	2.7%
TEMPORAL	9	12.3%
FRONTAL	0	0%
COMBINATION	6	8.2%
TOTAL	76	100%

Bleeding occurred commonly in the parietal area (76.7%) and the temporal area 12.3%. There was no frontal bleeding in the study group while bleeding occurred in multiple locations in 8.2% (table 11).

TABLE 12: COMBINATION LOCATIONS OF INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

	NUMBER	%
PARIETAL/OCCIPITAL	3	50.0%
FRONTAL/PARIETAL	1	16.7%
PARIETAL/TEMPORAL	1	16.7%
FRONTAL/OCCIPITAL	1	16.7%
TOTAL	6	100%

There were six patients with multiple locations of intracerebral bleeding. Among this category, three patients had parietal and occipital bleeds, while the rest had multiple sites as outlined in the table (table 12).

TABLE 13: PUPILLARY STATE IN RELATION TO INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

PUPILARY STATE	INTRACRANIAL BLEEDING		TOTALS
	PRESENT	ABSENT	
	NUMBER (%)	NUMBER (%)	NUMBER (%)
NORMAL	42 (16.4%)	212 (83.6%)	256 (85.3%)
ABNORMAL	31 (70.5%)	13 (29.5%)	44 (14.7%)
TOTALS	73 (24.3%)	227 (75.7%)	300 (100%)

44 patients (14.7%) had abnormal pupils on admission while 256 (85.3%) had normal pupils.

In 70.5% of the patients with abnormal pupils on admission, there was radiological evidence of intracranial bleeding while among the patients with normal pupils on admission, only 16.4% had intracranial bleeding. In a two by two analysis, the association of pupilary state with presence of intracranial bleeding was statistically significant (p 0.000, confidence interval 95% using Pearson Chi-Square) (table 13).

TABLE 14: NEUROLOGICAL DEFICIT IN RELATION TO INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

NEUROLOGIC DEFICIT	INTRACRANIAL BLEEDING		TOTALS
	PRESENT	ABSENT	
	NUMBER (%)	NUMBER (%)	NUMBER (%)
PRESENT	39 (75.0%)	13 (25.0%)	52 (17.3%)
ABSENT	34 (1.7%)	214 (86.3%)	248 (82.7%)
TOTALS	73 (24.3%)	227 (75.7%)	300 (100%)

53 (17.3%) patients had neurological deficits on admission while 248 (82.7%) patients had none.

Of the patients admitted with neurological deficits, 75% had intracranial bleeding and in the patients without neurological deficits, this number was 1.7%. The association of neurological deficit with intracranial bleeding was statistically significant using Chi-Square Testing (p value < 0.05) (table 14).

TABLE 15: GCS CONSCIOUS LEVEL IN RELATION TO INTRACRANIAL BLEEDING IN BLUNT HEAD INJURY WITH SKULL FRACTURE.

GCS	INTRACRANIAL BLEEDING		TOTALS
	PRESENT	ABSENT	
	NUMBER (%)	NUMBER (%)	NUMBER (%)
14 - 15	13 (5.8%)	211 (94.2%)	224 (74.7%)
9 - 13	36 (80.0%)	9 (20.0%)	45 (15.0%)
6 - 8	18 (78.0%)	5 (21.7%)	23 (7.7%)
< 6	6 (75.0%)	2 (25.0%)	8 (2.7%)
TOTALS	73 (24.3%)	227 (75.7%)	300 (100%)

Majority of the head injured patients (74.5%) had a GCS of 14 – 15 (mild head injury), 15% had GCS of 9 – 13 (moderate head injury), 7.7% had GCS of 6 – 8 (severe head injury) and 2.7% had GCS of less than 6 (critical head injury). Intracranial bleed was present in 80% of patients with moderate head injury and 5.8% of patients with mild head injury. The association of GCS with incidence

of intracranial bleeding was not statistically significant (p value >0.05 with a confidence interval of 95% using Chi-Square Testing) (table 15).

TABLE 16: TREATMENT MODALITIES IN BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURE.

	NUMBER	%
SURGICAL	116	38.7%
NON SURGICAL	184	61.3%
TOTAL	300	100.0%

116 (38.7%) were treated surgically and 185 (61.3%) non surgically (table 16).

TABLE 17: AGE IN RELATION TO OUTCOME AT 1 WEEK IN BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURE.

AGE (YRS)	DISCHARGED (%)	DEAD (%)	STILL HOSPITALIZED (%)	TOTALS (%)
< 20	1 (0.4%)	1 (3.1%)	0 (0%)	2 (0.7%)
20-35	194 (73.8%)	19 (59.4%)	2 (40%)	215 (71.7%)
36-50	54 (20.5%)	7 (21.9%)	1 (20%)	62 (20.7%)
> 50	14 (5.3%)	5 (15.6%)	2 (40%)	21 (7.0%)
TOTALS	263 (87.5%)	32 (10.7%)	5 (1.7%)	300 (100%)

While the young adults comprised the majority of the patients,. this group also had the best and worst prognosis. 73.8% had been discharged by the end of one week while 59.4% had died (Table 17).

TABLE 18: PRESENCE OF INTRACRANIAL BLEEDING IN RELATION TO OUTCOME AT ONE WEEK IN BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURE.

BLEEDING	DISCHARGED (%)	DEAD (%)	STILL HOSPITALIZED (%)	TOTALS (%)
PRESENT	54 (73.9%)	15 (20.5%)	4 (5.5%)	73 (24.3%)
ABSENT	209 (92.1%)	17 (7.5%)	1 (0.4%)	227 (75.7%)
TOTALS	263 (87.7%)	32 (10.7%)	5 (1.7%)	300 (100%)

263 patients (87.5%) were discharged by the end of one week in hospital, while 32 (10.7%) had died by the same period. Five patients (1.7%) were still hospitalized by the end of 1 week (tables 17 and 18).

Of the patients still in hospital at 1 week, 2 (40%) were aged >50yrs while none was less than 20 yrs of age. Among the patients who died by the end of the week, 59.4% were young adults while only 1 (3.1%) were under 20 yrs of age (table 17).

Among the 73 patients with intracranial bleeding, 20.5% were dead by the end of 1 week post admission while, 73.9% had been discharged and 5.5% were still hospitalized as compared to the 227 patients with no intracranial bleeding where 7.5% were dead as at 1 week post admission, 92.1% had been discharged and 0.4 were still hospitalized by the end of this period (table 18).

TABLE 19: ASSOCIATION OF TYPE OF FRACTURE WITH INTRACRANIAL BLEEDING IN MILD HEAD INJURY (GCS 14-15) PATIENTS WITH NEUROLOGIC SIGNS (PUPILARY ABNORMALITIES AND NEUROLOGICAL DEFICITS).

NEUROLOGIC SIGNS	FRACTURE TYPE	INTRACRANIAL BLEEDING		TOTAL
ABSENT		PRESENT	ABSENT	
		N (%)	N (%)	N (%)
	SIMPLE	3 (2.5%)	119 (97.5%)	122 (54.5%)
	SIMPLE DEPRESSED	10 (58.5%)	7 (41.2%)	17 (7.6%)
	COMPOUND LINEAR	0 (0%)	75 (100%)	75 (33.5%)
	COMPOUND DEPRESSED	0 (0%)	10 (100%)	10 (4.5%)
	TOTAL	13 (5.8%)	211 (84.2%)	224 (100%)
PRESENT	N/A	N/A (N/A)	N/A (N/A)	0 (0%)

None of the patients with mild head injury had neurological signs (table 19) (both an abnormal pupillary reaction and neurological deficits). Only 5.8% of these mildly injured patients had CT evidence of intracranial bleeding. There was a statistically insignificant association between the type of fracture and presence of intracranial bleeding in this population (p 0.689 using linear-by-linear association on the Chi-Square testing) (table 19).

In the absence of patients in this population with neurologic signs, no association could be computed linking this presentation with the occurrence of intracranial bleeding.

TABLE 20: ASSOCIATION OF TYPE OF FRACTURE WITH INTRACRANIAL BLEEDING IN MODERATE HEAD INJURY (GCS 9-13) PATIENTS WITH NEUROLOGIC SIGNS (PUPILARY ABNORMALITIES AND NEUROLOGICAL DEFICITS).

NEUROLOGIC SIGNS	FRACTURE TYPE	INTRACRANIAL BLEEDING		TOTAL
		PRESENT	ABSENT	
		N (%)	N (%)	N (%)
PRESENT	SIMPLE	21 (75%)	7 (25%)	28 (96.6%)
	SIMPLE DEPRESSED	1 (100%)	0 (0%)	1 (3.4%)
	COMPOUND LINEAR	0 (0%)	0 (0%)	0 (0%)
	COMPOUND DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	TOTAL	22 (75.9%)	7 (24.1%)	29 (100%)
ABSENT	SIMPLE	7 (87.5%)	1 (12.5%)	8 (50%)
	SIMPLE DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	COMPOUND LINEAR	0 (0%)	1 (100%)	1 (6.3%)
	COMPOUND DEPRESSED	7 (100%)	0 (0%)	7 (43.8%)
	TOTAL	14 (87.5%)	2 (12.5%)	16 (100%)

In this population of patients with moderate head injury, clinical symptoms, comprising both an abnormal pupil and neurological deficits, were present in 29

patients out of a total of 45. Using chi-square testing, the association of the type of fracture with the presence of bleeding was not statistically significant in the population (p value 1.566 using the Pearson chi-square).

There was similarly no significant association between these variables in the patients with no clinical symptoms (p value 1.018 using Pearson chi-square) (table 20).

TABLE 21: ASSOCIATION OF TYPE OF FRACTURE WITH INTRACRANIAL BLEEDING IN SEVERE HEAD INJURY (GCS 6-8) PATIENTS WITH NEUROLOGIC SIGNS (PUPILARY ABNORMALITIES AND NEUROLOGICAL DEFICITS).

NEUROLOGIC SIGNS	FRACTURE TYPE	INTRACRANIAL BLEEDING		TOTAL
		PRESENT	ABSENT	
		N (%)	N (%)	N (%)
PRESENT	SIMPLE	2 (28.6%)	5 (71.4%)	7 (87.5%)
	SIMPLE DEPRESSED	1 (100%)	0 (0%)	1 (12.5%)
	COMPOUND LINEAR	0 (0%)	0 (0%)	0 (0%)
	COMPOUND DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	TOTAL	3 (37.5%)	5 (62.5%)	8 (100%)
ABSENT	SIMPLE	1 (100%)	0 (0%)	1 (6.7%)
	SIMPLE DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	COMPOUND LINEAR	0 (0%)	0 (0)	0 (0%)
	COMPOUND DEPRESSED	14 (100%)	0 (0%)	14 (93.3%)
	TOTAL	15 (100%)	0 (0%)	15 (100%)

Ch-square testing of this population shows no statistically significant association between the types of fracture and the presence of intracranial bleeding in the severely injured patients with clinical symptoms (p value 1.375 using Fischer's exact test) (table 21).

TABLE 22: ASSOCIATION OF TYPE OF FRACTURE WITH INTRACRANIAL BLEEDING IN CRITICAL HEAD INJURY (GCS <6) PATIENTS WITH NEUROLOGIC SIGNS (PUPILARY ABNORMALITIES AND NEUROLOGICAL DEFICITS).

SYMPTOMS	FRACTURE TYPE	INTRACRANIAL BLEEDING		TOTAL
		PRESENT	ABSENT	
		N (%)	N (%)	N (%)
PRESENT	SIMPLE	5 (83.3%)	1 (16.7%)	6 (85.7%)
	SIMPLE DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	COMPOUND LINEAR	0 (0%)	0 (0%)	0 (0%)
	COMPOUND DEPRESSED	1 (85.7%)	0 (0%)	1 (14.3%)
	TOTAL	6 (85.7%)	1 (14.3%)	7 (100%)
ABSENT	SIMPLE	0 (0%)	1 (100%)	1 (100%)
	SIMPLE DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	COMPOUND LINEAR	0 (0%)	0 (0%)	0 (0%)
	COMPOUND DEPRESSED	0 (0%)	0 (0%)	0 (0%)
	TOTAL	0 (0%)	0 (0%)	1 (100%)

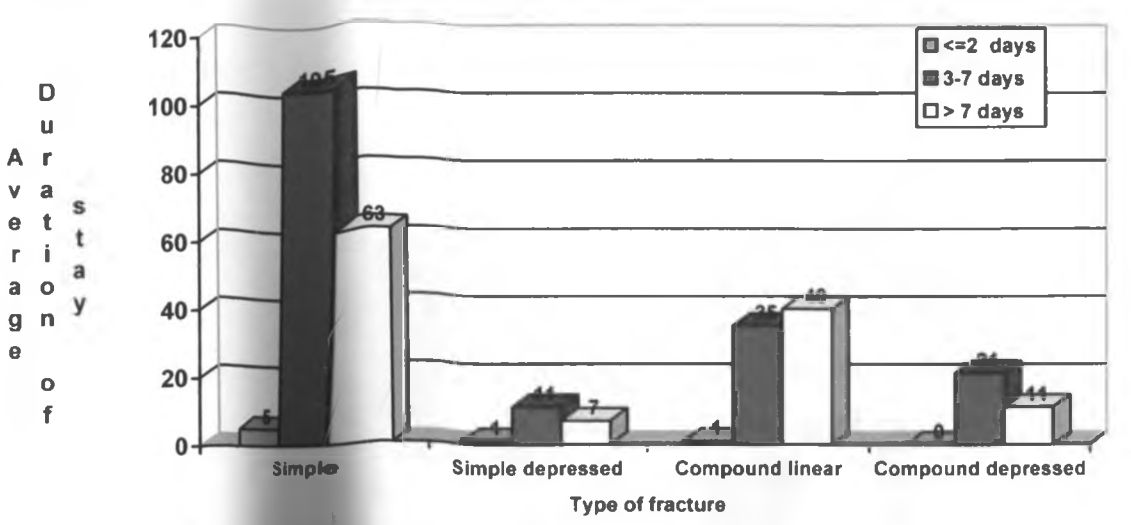
In the population of critically injured patients with clinical symptoms, there was no statistically significant association between the type of fracture and presence of intracranial bleeding (p value 0.857 using Fisher's exact test) (table 22).

TABLE 23: ASSOCIATIONS OF TYPE OF FRACTURE, PRESENCE OF INTRACRANIAL BLEEDING AND THE DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY.

TYPE OF FRACTURE	INTRACRANIAL BLEEDING	NUMBER (N) OF PATIENTS	MEAN DURATION OF HOSPITAL STAY
SIMPLE LINEAR	PRESENT	39	8.09
	ABSENT	134	6.34
SIMPLE DEPRESSED	PRESENT	12	8.71
	ABSENT	7	6.58
COMPOUND LINEAR	PRESENT	0	0
	ABSENT	76	7.25
COMPOUND DEPRESSED	PRESENT	22	10.40
	ABSENT	10	7.77

Irrespective of the fracture types, patients with intracranial bleeding had on average a longer period of hospital stay compared with the ones with no intracranial bleeding (p value 0.0006 by independent samples test) (table 23).

FIGURE 7: ASSOCIATION OF TYPE OF FRACTURE WITH THE DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY.



There was a random distribution in the duration of hospital stay within the various fracture types (Fig 7).

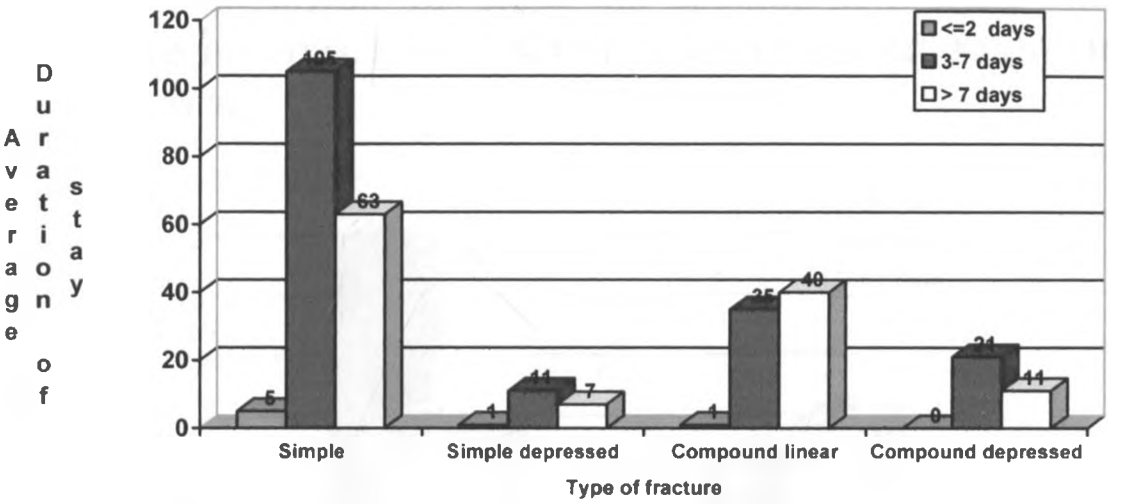
Analysis of the results by the independent samples test showed no statistically significant association between the duration of hospital stay and the various types of skull fracture i.e simple, simple depressed, compound linear, compound depressed (respectively p values 0.323, 0.792, N/A, 0.682).

TABLE 24: ASSOCIATION OF TYPE OF FRACTURE, TREATMENT MODALITY AND DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY PATIENTS.

TYPE OF FRACTURE	TREATMENT MODALITY	NUMBER (N) OF PATIENTS	AVERAGE DURATION OF HOSPITAL STAY
SIMPLE LINEAR	SURGICAL	155	6.66
	NON-SURGICAL	18	7.33
SIMPLE DEPRESSED	SURGICAL	2	4.00
	NON-SURGICAL	17	7.76
COMPOUND LINEAR	SURGICAL	7	5.00
	NON-SURGICAL	69	7.48
COMPOUND DEPRESSED	SURGICAL	21	7.95
	NON-SURGICAL	11	9.82

Irrespective of the fracture types, patients with intracranial bleeding had on average a longer period of hospital stay compared with the ones with no intracranial bleeding (p value 0.0006 by independent samples test) (table 23).

FIGURE 7: ASSOCIATION OF TYPE OF FRACTURE WITH THE DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY.



There was a random distribution in the duration of hospital stay within the various fracture types (Fig 7).

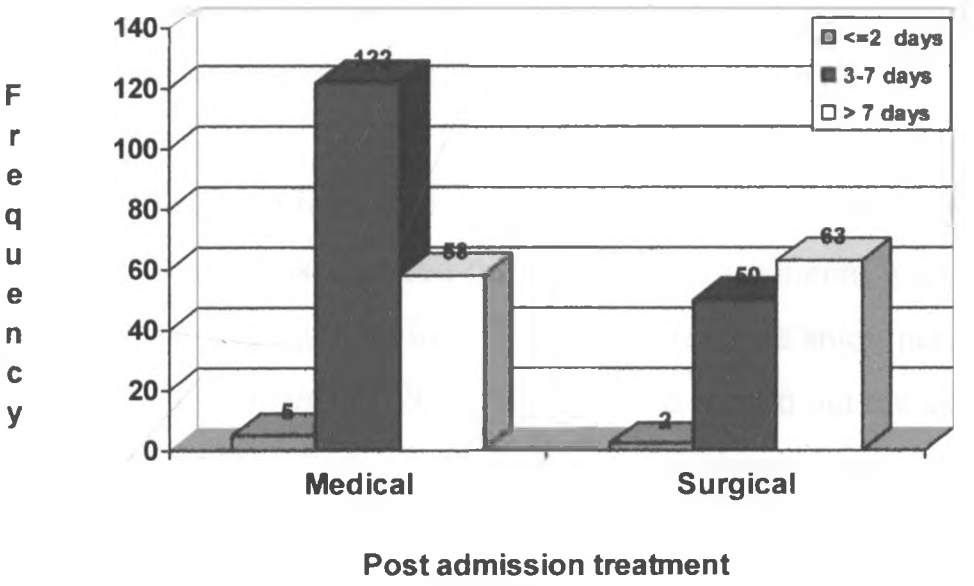
Analysis of the results by the independent samples test showed no statistically significant association between the duration of hospital stay and the various types of skull fracture i.e simple, simple depressed, compound linear, compound depressed (respectively p values 0.323, 0.792, N/A, 0.682).

TABLE 24: ASSOCAITION OF TYPE OF FRACTURE, TREATMENT MODALITY AND DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY PATIENTS.

TYPE OF FRACTURE	TREATMENT MODALITY	NUMBER (N) OF PATIENTS	AVERAGE DURATION OF HOSPITAL STAY
SIMPLE LINEAR	NON SURGICAL	155	6.66
	SURGERY	18	7.33
SIMPLE DEPRESSED	NON SURGICAL	2	4.00
	SURGERY	17	7.76
COMPOUND LINEAR	NON SURGICAL	7	5.00
	SURGERY	69	7.48
COMPOUND DEPRESSED	NON SURGICAL	21	7.95
	SURGERY	11	9.82

On average, patients managed non surgically had a shorter period of hospitalization. However, utilizing the independent samples testing, the association of the treatment modality with the hospitalization stay was only found to be statistically significant in the patients with compound linear fracture (p value <0.005) (table 24, Fig 8).

FIGURE 8: ASSOCIATION OF MODE OF MANAGEMENT WITH THE DURATION OF HOSPITAL STAY IN BLUNT HEAD INJURY PATIENTS WITH SKULL FRACTURE.



DISCUSSION

The association of head trauma, skull fractures and intracranial bleeding has been studied by various authors over time. Contradictory conclusions have been made in that period, and there are still a lot of unresolved issues relating to the occurrence, diagnosis and management of these injuries. This study looked at the association of the various types of fractures in blunt head injury with the occurrence of the various forms of intracranial bleeding

Three hundred (300) patients with skull fractures were recruited into the study from a population of six hundred and seventy five patients with blunt head injury admitted at Kenyatta National Hospital during the study period between June 1st 2007 and October 12th 2007. The aim was to find out the association of skull fractures in blunt head injured patients with the presence of intracranial bleeding.

Out of all the patients admitted with blunt head injury, there hundred (44.4%) were found to have skull fractures radiologicaly (skull Xray) and this served as the study group to determine the association with intracranial bleeding. The study revealed that a large majority of the recruited patients were males constituting 91.3% of the population (table 3 & Fig2). All the available studies, both local and global, analysing the occurrence of head injuries in the population show that males are statistically significantly more likely to be victims of head injuries as compared to the females (11,14,17). Said, in his study on the outcome of the management of extradural hematomas at KNH in Kenya, found out that 96.7% of his study population were males (11). The young adults (age group 20 – 35yr) were ones mostly at risk of being victims of head injuries within the population, corresponding to the most active

unemployed youth are a vulnerable population in regard to risk exposure to situations predisposing to head injuries, either as aggressors or victims in assault cases.

The elderly head injury patients (age >50yrs) in this study formed only 7% of the population. This group is relatively sedentary and not aggressive therefore not subjected to the risk factors of the young adults. This is similar to the trend observed in Scandinavian countries and the US (13,17).

As regards the types of skull fractures, this study revealed that simple linear fractures are the commonest in occurrence, making up 57.7% of all fractures in the population (table 5). Universally, available literature estimates the occurrence of simple linear fractures as up to 75%, therefore the findings roughly concur (4,10). Simple depressed fractures made up 6.3% of the study population which compared well with the literature figure of 6% (16,17). Compound linear fractures comprised 25.3% of all fractures which can not be compared with the universal figures since literature is not available. However, compound depressed fractures made up 10.7% of all fractures studied compared with 25% reported in the literature (16). This difference appears large but may in part be explained by the fact that most studies included penetrating head injuries among the causes while in this study, the group excluded. Moreover, since most of the fractures were caused by assault directed at the relatively stronger frontal bone with a thick diploe, the sharp objects used (e.g. knives) would not generate enough force to fracture and depress this skull as readily as a huge blunt force that occurs in the setting of RTA or falling objects (4).

The most commonly fractured bone was found to be the parietal bone in 43% of all skull fractures (table 6). In the literature, the temporal bone is the most commonly fractured bone in 48% of skull fractures, which was not in keeping

population group in terms of travelling, adventuring, exploring and aggressiveness (table 2 & Fig 1). This group comprised 71.7% of the study population, and could be a major focus group in terms of implementing preventive measures to reduce the occurrence of head injuries in the general population. This mirrors similar demographic characteristics observed in Scandinavian and other European countries that show a peak incidence of head injuries in the young adults (14,15).

However, there has been a progressive shift in the causes of head injuries since the 1980s when the dominant cause was RTA as shown by a study by Mwangombe in1980 (33). At that time, RTA accounted for 46% of head injuries and assault 40.6%. Omondi in 1990 found assault cases to be higher at 30% compared to RTA at 27% while Said in 2002 found 61% of head injuries to be due to assault and 19% RTA (34,11).
This study has revealed that assault cases currently account for 49.7% and RTA 31.7% of the blunt head injury patients with skull fractures. This shows a gradual decrease in the proportion but not the absolute number of patients hospitalized with head injuries due to RTA, while there was a corresponding increase in the proportion of patients hospitalized with head injuries due to assault.

The reasons for increasing assault incidences relative to RTA could be due to the increased awareness of the population on travelling safety guidelines and possibly strict enforcement of traffic rules, while rising unemployment and increased poverty levels over time may have led to increased incidences of muggings, domestic violence and general violent crimes. Moreover, across the different age groups, the unemployed constituted the majority (41.3%) of the population while the least affected were the formally employed individuals comprising 18% of the population. The implications for this is that the

with this study findings (4,10). Temporal fractures accounted for a meagre 2.3% of the fractures in this study. The reasons for these differences are not immediately clear but it could be due to observer error in misrepresenting temporal fractures as parietal fractures or it could be an indication of the unique biomechanics and patterns of skull fractures in our setup. More studies will be required to analyse the patterns to be able to lay any significance in the apparent variance.

The incidence of basilar skull fracture was 26% in this study as compared to 19 – 21% in the literature (4,10). The difference here is attributed to the inter observer interpretation since basilar fractures are clinically diagnosed with no objective imaging modalities in routine use to diagnose the fractures. It is possible that some epistaxis could have been misinterpreted as rhinorrhoea since biochemical studies were not performed on the fluid and the results were based on the interpretation of the investigator or research assistant. The otorrhoea and/or rhinorrhoea was often of short duration and had invariably stopped during the review in the units.

Worth noting is the occurrence of fractures at multiple locations in 2% of all the fractures. This pattern of skull fractures is not well studied as evidenced by the absence of data in the literature regarding it.

The incidence of intracranial bleeding in blunt head injury with skull fracture was found to be 24.3% (table 8), which compares well with the published data of 25%. This considers all the three hundred patients hospitalized, whether or not they had neurological symptoms at the time of evaluation. It is generally accepted that 1:4 head injury patients with a skull fracture and neurological symptoms will have an intracranial event/bleed while in the absence of neurological symptoms, the risk is 1:6000 ((18,19,20).

In this study, 75% of the population with a skull fracture and neurological signs had intracranial bleeding, while among those with no neurological signs, the incidence was 1.7% (table 14). The difference is attributed to the non specific criteria of neurological symptoms, which in some studies has included the history of concussion, vomiting and headaches while in other studies only the more serious symptoms of convulsions and prolonged loss of consciousness are included. In this study, specific symptoms were not included in the questionnaire. Evaluation was subjective and confined to the ones present at evaluation, with neurological signs used to assess the presence or absence of neurological deficits. There is a need to standardize the neurological criteria in assessment of head injury in order to produce findings that are reproducible. The study by Miller and Holmes looked at the usefulness of the clinical indicators in head injured patients to predict the incidence of intracranial bleeding and hence reduce the ordering of CT scans for mild head injury (6). Although the aim of the Miller study was to minimize unnecessary scans, the findings about the relationship of signs and bleeding co relates with the results in this study.

The most commonly occurring intracranial bleeding in this study was intracerebral, occurring in 43.8% of the bleeds (table 9). Epidural bleeding was present in 30.1% and subdural bleeding in 15.1%. The subarachinoid bleed was rare at 1.4% while no single case of isolated intraventricular bleed was noted. Previous studies have concentrated on the occurrence of epidural bleed and its association with skull fractures indicating that 85 – 95% of epidural bleeds will have an associated skull fracture and 25% of temporal skull linear fractures will have an epidural bleed (11). Literature is scant on the association of skull fractures with the various other patterns of intracranial bleeding, therefore more complimentary studies are needed on this subject. What is noteworthy is the occurrence of intraventricular bleeding in combination with intracerebral

bleeding, indicating a possible causal association with severe head trauma (table 10).

Bleeding, when it occurred, was commonly in the parietal cortex, corresponding to the commonly fractured skull bone (tables 6&11). This association points to the possibility of coup injury being dominant, as opposed to contra coup injury. The bleeding was parietal in 76.7%, temporal in 12.3%, occipital in 2.7% and none in the frontal cortex. There are no studies available in literature showing the association of the location of intracranial bleed with the site of fracture.

There was a noticeable relation between the clinical indicators of severe head injury and the occurrence of intracranial bleeding. When the state of the pupils, the GCS and neurological deficits were each analysed separately, they were all found to have a statistically significant association with the occurrence of intracranial bleeding (tables 12,13,14).

Said demonstrated the importance of the pupil assessment in head injury to predict the presence of an extradural bleeding (11). The clinical parameters are included in all neurological assessments in the head injured patient and are an important indicator of intracranial events and consequently the outcome. In this study, pupilary changes were noted in 14.7% of the patients and of this number, bleeding was present in 70.5% of them (table 13). Neurological deficits were present in 17.3% of the study population and 75% of them had intracranial bleeding (table 14).

An intracranial bleed is a space occupying lesion which will have pressure effects on adjacent structures within the brain. As the blood collects, the increased compression on the motor and sensory cortices will result in respectively accompanying motor and sensory deficits. Increased pressure will

eventually displace the temporal lobe, stretching the occulomotor nerve which runs just above the edge of the tentorium, resulting in the pupilary changes that may accompany the head injury. Pupilary changes may also occur in direct injury to the optic nerve as a result of anterior cranial fossa fractures and are not pathognomonic of intracranial bleeding (23). In this study, head injury patients with a skull fracture and pupilary changes had a 70% chance of having an intracranial bleed, while head injured patients with neurological deficits had a 75% chance of having an intracranial bleed.

A slightly different trend was noted in regards to the GCS state as a predictor of intracranial bleeding. Mild head injury (GCS 14 – 15) comprised the majority of the injured at 74.7%, while moderate head injury (GCS 9 – 13) had 15%, severe head injury (GCS 6 – 8) 7.7% and critical head injury (GCS < 6) 2.7%. There was a higher probability of intracranial bleeding in the moderately injured patients at 80%, followed by the severely injured at 78.3% and the critically injured at 2.7%. The mildly injured had a 5.8% probability of having a bleed. However, when subjected to statistical testing, the association of GCS with the the occurrence of intracranial bleeding was not found to be statistically significant ($p < 0.005$). It should be noted that in the severely injured persons with massive intracranial bleeds, the condition is rapidly fatal, especially if the bleeding is in the posterior fossa. This is due to the small and crowded compartment and the vital brain stem structures in close proximity together with the association with cervical spine injuries (12,22). It is thus probable that the relatively less proportion of intracranial bleeding recorded in the critically injured patients could be attributed to higher mortality rate prior to admission. This study would be complimented by a similar study looking at the association of fractures with intracranial bleeding in the post mortem cases, since the findings would show the rapidly fatal types of bleeding that would otherwise be missed in this current study.

On analysis of the association of the types of fractures and the presence of intracranial bleeding in patients with clinical signs of both an abnormal pupil and neurological deficits, this study found that the fracture type had no predictive value on the occurrence of a bleed. This suggests that the presence, as opposed to the pattern, of skull fracture in association with clinical symptoms will determine the presence or absence of an intracranial bleed. There is no comparative study in the literature analysing this association.

Out of all the patients in the study, 61.3% were treated non surgically (table 15). The aim in management of head injuries is to prevent the progression of the secondary events and this is mainly achieved non surgically (3). Surgery was indicated in the patients with intracranial bleeding with evidence of raised intracranial pressure, patients with compound fractures and patients with significantly depressed fractures. Determining the mode of treatment was dependent on the clinical presentation and the radiological findings. Significantly depressed fracture with lateralizing symptoms even in the absence of scalp wound necessitated operative treatment. Obvious indications for surgery included surgical toilet for compound fractures to reduce the chances of infective sequelae (brain abscesses and meningitis) and evacuation for significant extradural and subdural hematomas with lateralizing symptoms. In the borderline, were patients with clinically elevated intracranial pressures but with no localized hematomas on imaging. These cases were assumed to be due to cerebral oedema and in well equipped centres, intracranial pressure monitoring and CSF drainage devices would have been useful in improving the outcome (26,27). Contrary to the perception within the hospital, these devices are not expensive, and some modified versions have been in use in some remote hospitals in India, where the social economic conditions are similar and at times even worse that ours (27). This illustrates that as much as we might be

unfortunate in terms of resources, we might also be lacking the administrative will to address the issue of the availability of some facilities.
However, not all patients requiring surgery actually underwent the procedure (table 24). The reasons included delays in getting theatre leading to spontaneous healing, abscondment, inappropriateness of surgery due co morbidities, and deaths occurring prior to surgery.

The presence of a skull fracture and clinical symptoms had a direct association with the presence of an intracranial bleeding with the majority of the patients benefiting from non surgical treatment. However, the pattern of fracture had no predictive value in determining the presence or pattern of bleeding (tables 19,20&21). Demonstration of a skull fracture by itself has thus no bearing on the further management of a head injured patient, especially in respect to an intracranial event, lending weight to the critics of routine Xray imaging of the skull in head injured patients. Evidence of intracranial bleeding was confirmed by CAT scan, destroying the myth that this is an elite investigation only reserved for the upper end class in society. Indeed, when the KNH CT scan machine was not operational, the hospital and in many instances the patients were able to make arrangements to have the images taken elsewhere. There is a need for tertiary health institutions to partner with other institutions in order to provide a continuum of services in the event of equipment failure which is wont to occur. It however doesn't obviate the need to ensure that tertiary institutions like KNH purchase cost effective equipments which are accompanied by a good service backup to provide a self sufficient working environment that is not dependent on the goodwill of perceived competitors.
The CT scan is a mandatory investigative tool in head injured patients and the plain X Ray plays no role other than demonstrating the presence or absence of a fracture, which in most cases will clinically be detected (5,9,23). The routine

use of XRays in head injured patients should therefore be condemned as a waste of resources, especially in a third world institution.

The outcome at the end of 1 week was assessed and 87% of all the patients admitted had been discharged by that time (tables 16,17). Most of them remained in the ward for various non medical reasons. There was a mortality of 10.7% and 1.7% were still hospitalized at the end of 1 week. There was a general trend in this study indicating that patients managed non surgically had a shorter hospital stay compared with the ones managed surgically, but this did not factor in the presence or absence of intracranial bleed (table 24). Moreover, a vast majority of patients received non surgical as opposed to surgical management. This indicates that the majority of acute head injured patients will respond to non surgical management and will have relatively short hospital stay. There was a statistically significant difference in the mean hospitalization duration between the patients managed non surgically and surgically among the patients who had compound depressed fractures. This difference was not statistically significant in the other fracture types. The apparently better short term outcome of the patients managed non surgicaly has little to do with the management mode but rather by the relatively better clinical state or severity of injury at presentation. The study did not follow up the patients to assess the prognosis after discharge but according to the available data, mortality in head injured patients range from 25 – 36% in adults within the first 6 months of injury with most deaths occurring within the first 2 weeks even in the most advanced centres (2). An Australian study of patients with head injuries incurred in the period 1984 – 1991 found that all 59 patients who were aged 65 years or older and scored less than 11 on the post resuscitation GCS either died or were left with severe disability (18,32).

CONCLUSION

Head injury continues to be an important cause of morbidity and mortality in the world, acquiring even more significance in a third world setup with limited infrastructure and equipments as was the case in this study. Studies of this nature help to create and cement the awareness of this condition and the impact it has on the society. It is a serious drain on resources in terms of morbidity and mortality and hence requires to have a special place in terms of budgetary allocation and health planning in any institution. Tertiary health institutions need to be equipped with dedicated and committed units specifically dealing with head injuries so as to lessen the negative impact of the condition through optimized care.

Three hundred patients with skull fractures were recruited from a population of six hundred and seventy five patients with blunt head injury. This comprised 55.56% of all patients with blunt head injury. Majority of the patients with skull fractures were males (91.3%) in the age group 20 to 35yrs (young adults), possibly due to the aggressive nature of this population. The commonest cause of injury was assault. Assault has superseded RTA as the commonest cause of head injury in our population, and with the current trend, we are likely to witness even more cases of assault being reported as unemployment rises and insecurity increases. As well as educating the public on self security measures, there is a huge responsibility burden that the government authorities need to take up to ensure that we live in a secure environment devoid of muggings and attacks. This may be easier said than done, given that security is a sensitive matter that often acquires political connotations from time to time. Any measures targeting this age group would achieve more in terms of prevention measures other than blanket measures that sweep across board which tend to be

expensive and not sustainable especially in the setup of limited resources. Health education in schools and other institutions, on the risk factors associated with the causes of head injuries and tips on first aid to manage the early complications will assist in reducing the morbidity and mortality.

Simple skull fractures were the commonest type of fractures, comprising 57.7% of the population, followed by compound linear fractures, compound depressed fractures and simple depressed fractures comprising respectively 25.3%, 10.7% and 6.3%. Most fractures involved the parietal bone (43%), followed by basal bone (26%), frontal bone (21.3%), occipital bone (5.3%) and temporal bone (2.3%). The skull was fractured in multiple sites in 2.0% of the population. The fractures were demonstrated in all cases except basal fractures by Xray and later confirmed by CAT scan. Basal fractures were diagnosed clinically.

The incidence of intracranial bleeding in the patients with skull fractures was 24.3% in this study, in keeping with the universal figures. The majority of this bleeding was intracerebral (43.8%), with extradural bleeding accounting for 30.3%, subdural bleeding 15.1% and subarachnoid bleeding 1.4%. There were no cases of isolated intraventricular bleeding while the type of bleeding was combined in 9.6%. The commonest site of intracranial bleeding was in the parietal region (76.7%), conforming with a coup type of injury from a parietal fracture. Bleeding patterns were demonstrated in all cases by CAT scan.

The presence of a skull fracture with pupillary changes was 70% predictive of intracranial bleeding while the presence of a skull fracture with neurological deficits was 75% predictive of an intracranial bleed. This study illustrated that, in the blunt head injury patient with either pupilary changes or neurological deficits, there is no statistically significant association between the presence of a skull fracture, irrespective of the type, with the pattern of intracranial bleeding

(tables 19,20&21, p value > 0.05). The routine use of the skull Xray in all head injured patients should be condemned since it does not add much value in the subsequent management of the patient. Moreover, most clinically relevant skull fractures can be diagnosed clinically without imaging (5,9,23). The use of the Xray was found useful in the blunt head injured patient with either pupilary changes or neurological deficits, since the presence of a skull fracture in these cases was predictive of an intracranial bleed. The CAT scan was available to demonstrate the presence and pattern of intracranial bleeding, contrary to the perception that this is an elite and therefore unavailable investigative tool. Where the CAT scan is available, the skull Xray is an obsolete tool in the blunt head injury patient.

The pattern of skull fracture had no statistically significant association with the pattern of intracranial bleeding. There was, however, a significant association between the pattern of fracture and the mode of management, which influenced the duration of hospital stay. Most of the blunt head injured patients with linear skull fractures at the KNH were managed non surgically, while most of the compound and depressed skull fractures were managed surgically.

The average duration of hospitalization stay was longer in the population managed surgically compared with the ones managed non surgically. However, on independent sample testing, this association was proved not to be statistically significant in the various types of fractures, except in the patients with compound linear fractures. The average duration of hospital stay in blunt head injured patients with skull fractures ranged from 4 days in the simple depressed fractures managed non surgically, to 9.82 days in the compound depressed fractures managed surgically.

Prognosis was difficult to assess in the absence of an adequate follow up period but in the 1 week post admission, the mortality was over 10% which spells a grim figure in the outlook of head injured patients. The clinical condition of the patient on admission had a bearing on the short term outcome of the hospitalization. A disturbing aspect in the management of the patients in the study was the management of patients in the general surgical units. All acute head injured patients were admitted into the general surgery units and seen from time to time by the neurosurgical team upon consultation by the general surgeons. This resulted in undue delays since the consultations were not regular and even when they were, the visits were erratic. It is probable that some crucial management decisions, if made and followed up keenly by the neurosurgical teams in their units, could have improved the outcome of a majority of the patients. Since the management of the secondary brain injuries will determine the final outcome in most of the cases, it is imperative that institutions that are committed to managing head injuries be equipped with monitors of intracranial pressure and adequate drugs to control raised intracranial pressure. Training of personnel will complement the available equipments and this should be an ongoing process to keep pace with new advances in management modes as they unfold worldwide.

RECOMMENDATIONS

Tertiary health institutions need to be equipped with dedicated and committed units specifically dealing with head injuries so as to lessen the negative impact of the condition that results from well intentioned but insufficient general care.

Health education in schools and other institutions, targeting the population most at risk, on the risk factors associated with the causes of head injuries and tips on first aid to manage the early complications will assist in reducing the morbidity and mortality.

Improving security will translate to a direct reduction of assault cases which is the leading cause of head injury. This is a direct challenge to the law enforcing authorities.

Routine use of the skull xray in blunt head injury patients should be discouraged as it does not add value to the management. Where CAT scan facilities exist, this should be the investigation of choice. Where CAT scan is unavailable, the skull Xray should only be used in the blunt head injury patient with either pupilary changes or neurological deficits to demonstrate and document the presence of a skull fracture. This should then serve as an indicator of a possible intracranial bleed, leading to referral to an institution with CAT scan facility to document the presence or absence of a bleed and further management

There is a need for tertiary health institutions to partner with other institutions in order to provide a continuum of services in the event of equipment failure which commonly occurs. It however doesn't obviate the need to ensure that tertiary

institutions like KNH purchase cost effective equipments which are accompanied by a good service backup to provide a self sufficient working environment that is not dependent on other institutions.

Continuous training of personnel will complement the available equipments and this should be an ongoing process to keep pace with new advances in management modes as they unfold worldwide.

Last but not least, KNH should have dedicated units dealing with acute head injuries and not lump them in other units where they end up having their management delayed at best, or neglected completely at worst.

REFERENCES

1. King, M., Bewes, P C., Awori, N. et al. (eds). Trauma: primary surgery; v. 2. Oxford University Press, New York. 1987; p ii

2. Nissen, J J., Jones, P A., Signori, D.F et al. Glasgow head injury outcome prediction programme: an independent assessment. J Neurol Neurosurg Psychiatry 1999 ; **67:**796-9

3. Arieta, C., Caroli, M. and Balbi, S. Management of head-injured patients in the emergency department: a practical protocol. Surg Neurol. 1997; **48:**213-9

4. Yoganandan, N., Pinta, F A., Sances, A. Jr. et al. Biomechanics of skull fracture. J Neurotrauma. 1995; **12:**659-68

5. Halley, M.K., Silva, P D , Foley, J. et al. Loss of consciousness: when to perform computed tomography? Pediatric Crit Care Med. 2004; **5:**230-3

6. Miller, E.C., Holmes,J.F. and Derlet, R.W. Utilizing clinical factors to reduce head CT scan ordering for minor head trauma patients. J Emerg Med. 1997; **15:**453-7

7. Holmes, J..F, Bajer, M.E. and Derlet, R.W. Failure of the Miller criteria to predict significant intracranial injury in patients with a Glasgow Coma Scale of 14 after minor head trauma. Acad Emerg Med. 1997; **4:**788-92

8. Moore, K.L., Persaud, T.V (eds). The Developing Human: clinically oriented embryology, 7th ed. Saunders: Elsevier Science, Pennsylvania. 2003; p. 439

9. Munoz-Sanchez, M.A., Murillo-Caberaz,F., Cavuela, A. et al. The significance of skull fracture in mild head trauma differs between children and adults. Childs Nerv Syst. 2005; **21:**128-32

10. Engberg, A.W. andTeasdale, T.W. Traumatic brain injury in Denmark 1979-1996. A national study of incidence and mortality. Eur J Epidemiolol. 2001;**17**:437-42

11. Said, S.O. The outcome of management of extradural hematomas at Kenyatta National Hospital. M.Med Dissertation, Univ. of Nbi. 2002.

12. Sripairoikul, B., Saeheng, S., Ratanalert, S. et al. Traumatic hematomas of the posterior cranial fossa. J Med Assoc Thai. 1998; **81:**153-9

13. Adekoya, N., Thurman, D.J., White, D.D. et al. Surveillance for traumatic brain injury deaths-United States, 1989-1998. MMWR Surveill Summ. 2002; **51:**1-14

14. Steudel, W.I., Cortbus, F. and Schwerdtfeger, K. Epidemiology and prevention of fatal head injuries in Germany-trends and imparc of the reunification. Acta Neurochir (Wien). 2005; **147:**231-42

15. Centre for Disease Control and Prevention (CDC). Incidence rates of hospitalization related to traumatic brian injury-12 states. MMWR Morb Mortal Wkly Rep. 2006 3; **55:**201-4

16. Rutland-Brown, W., Langlois, J.A., Thomas, K.E. et al. Incidence of traumatic brain injury in the United States, 2003. J Head Trauma Rehabil. 2006; **21:**544-8

17. Engberg, A.W. and Teasdale, T.W. Epidemiology and treatment of head injuries in Denmark 1994-2002, illustrated with hospital statistics. Ugeskr Laeger. 2007; **169:**199-203

18. Mellick, D., Gerhart, K.A. and Whiteneck, G.G. Understanding outcomes based on the post-acute hospitalization pathways followed by persons with traumatic brain injury. Brain Inj. 2003; **17:**55-71

19 Teasdale, G.M., Murray, G. and Anderson, E. Risks of acute traumatic intracranial hematoma in children and adults: implications for managing head injuries. BMJ 1990; **300:**363-7

20. Mendelow, A.D., Teasdale, G., Jennett, B. et al. Risks of intracranial hematoma in head injured adults. BMJ. 1983; **27:**287

21. Murshid, W.R. Management of minor head injuries: admission criteria, radiological evaluation and treatment of complications. Acta Neurochir (Wien). 1998; **140:**56-64

22. Macpherson, B.C., Macpherson, P. and Jennett, B. CT evidence of intracranial contusion and hematoma in relation to the presence, site and type of skull fracture. Clin radiol. 1990; **42:**321-6

23. Pretto, F.L., De Almeida, C.S. and Casulari, L.A. Positive predictive values of selected clinical signs associated with skull base fractures. J Neurosurg Sci. 2000; **44:**77-82.

24. American College of Surgeons Committee on Trauma ; Advanced Trauma Life Support (ATLS) : a framework for the management of the injured patient, 1980-1997

25. Murshid, WR. Management of minor head injuries: admission criteria, radiological evaluation and treatment of complications. Acta Neurochir (Wien). 1998; **40:**56-64

26. Lundberg, N. Intracranial pressure Monitoring. Proc. Roy. Soc. Med 1971; **65:**6-8

27. Joseph, M. Intracranial pressure monitoring in a resourse-constrained environment: a technical note. Neurol India 2003; **51:**333-335

28. Society of critical care medicine and world federation of paediatric intensive and critical care societies. Pediatric critical care medicine 2003; **4**

29. Arieta, C., Caroli, M. and Balbi, S. Management of head injury patients in the emergency department: a practical protocol. Surg Neurol. 1997; **48:**213-9

30. Teasdale, G. and Galbraith S. Extra dural hemorrhage: effects of delayed treatment. Br Med J 1979; **12:**1793

31. Mendelow, A., Karmi, M.Z., Paul, K.S. et al. Extradural hematoma: effect of delayed treatment. Br Med J. 1979; **12:**1240-2.

32. Frankel, J.E., Marwitz, J.H., Cifu, D.X. et al. A follow up study of older adults with traumatic brian injury: taking into account decreasing length of stay. Arch Phys Med Rehab. 2006; **87:**57-62

33. Mwangombe, N.J. The pattern of head injuries at Kenyatta National Hospital. M.Med Dissertation, U.O.N. 1980

34. Omondi, J.M. Intracranial haematomas at Kenyatta National Hospital. M.Med Dissertation, U.O.N. 1990

APPENDIX 1 – QUESTIONNAIRE

Date of admission ..... Date of Data Collection .....

A) Patient's Name.....

Inpatient Number.....

Study Number .....

Residence .....

B) Age in yrs .....

a. 13-19
b. 20-39
c. 40-55
d. Over 55

C) Occupation

a. Student
b. Casual
c. Permanent
d. Self employed
e. Others

D) Sex

a. Male
b. female

E) Cause of head injury

a. Road traffic accident
b. Assault
c. Sporting
d. Falling object
e. Others

F) GCS Consciousness level on admission .....

a. 14-15
b. 9-13
c. 6-8
d. Below 6

G) Pupilary state at admission

a. normal
b. abnormal

H) Neurological deficits at admission

a. Present
b. Absent

I) Clinical evidence of basal skull fracture

a. Present
b. Absent

J) CT scan done

a. Yes
b. No

K) CT evidence of intracranial bleeding

a. Present
b. Absent

L) Location of Fracture

a. Frontal
b. Parietal
c. Occipital
d. Temporal
e. Basal
f. Multiple

M) Type of fracture

a. Simple Linear
b. Simple depressed
c. Compound linear
d. Compound depressed

N) Type of intracranial bleeding

a. Extradural
b. Subdural
c. Intracerebral
d. Subarachnoid
e. Combination

O) Location of intracranial bleed

a. Frontal
b. Parietal
c. Occipital
d. Temporal
e. Combination

P) Post admission treatment

a. Medical
b. Surgical

Q) Duration of hospitalization in days .....

a. < 2 days
b. 2 days to 7 days
c. > 7 days

APPENDIX 2 - GLASGOW COMA SCALE (GCS)

Eye Opening

Score	≥1 Year	0-1 Year
4	Opens eyes spontaneously	Opens eyes spontaneously
3	Opens eyes to a verbal command	Opens eyes to a shout
2	Opens eyes in response to pain	Opens eyes in response to pain
1	No response	No response

Best Motor Response

Score	≥1 Year	0-1 Year
6	Obeys command	N/A
5	Localizes pain	Localizes pain
4	Flexion withdrawal	Flexion withdrawal
3	Flexion abnormal (decorticate)	Flexion abnormal (decorticate)
2	Extension (decerebrate)	Extension (decerebrate)
1	No response	No response

Best Verbal Response

Score	>5 Years	2-5 Years	0-2 Years
5	Oriented and able to converse	Uses appropriate words	Cries appropriately
4	Disoriented and able to converse	Uses inappropriate words	Cries
3	Uses inappropriate words	Cries and/or screams	Cries and/or screams inappropriately
2	Makes incomprehensible sounds	Grunts	Grunts
1	No response	No response	No response