ROLE OF MEAN ARTERIAL PRESSURE IN MONITORING SEVERE HEAD INJURY PATIENTS AT A TERTIARY HOSPITAL

A PROSPECTIVE DISSERTATION STUDY TO BE SUBMITTED IN PART FULFILMENT FOR THE DEGREE OF MASTERS OF MEDICINE IN SURGERY UNIVERSITY OF NAIROBI.

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DEDICATION

I dedicate this book to my family especially to my wife Pamela, my daughters Angela and Hope, and to my sons Christian and Geoffrey. Without their love, support, understanding, and encouragement, completion of this work would not have been possible.

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LIST OF ABBREVIATIONS

ATLS.....Advanced Trauma Life Support

BBB.....Blood Brain Barrier

BP.....Blood Pressure

CBF.....Cerebral Blood Flow

CPP.....Cerebral Perfusion Pressure

CSF.....Cerebrospinal Fluid

CT.....Computerized Tomography

GCS.....Glasgow Coma Scale

GOS.....Glasgow Outcome Score

ICP .....Intracranial Pressure

ICU.....Intensive Care Unit

KNH.....Kenyatta National Hospital

MAP.....Mean Arterial Pressure

MRI.....Magnetic Resonance Imaging

PVI.....Pressure Volume Index

RTA.....Road Traffic Accident

SOL.....Space Occupying Lesion

USA.....United States of America

AANS.....American Association of Neurological Surgeons

ISS.....Injury Severity Score

AIS.....Abbreviated Injury Scale

SPSS.....Statistical Package for Social Sciences

APACHE.....Acute Physiology and Chronic Health Evaluation

TRISS.....Trauma and Injury Severity Score

SD.....Standard deviation

ANOVA.....Analysis of variance

A & E.....Accident and Emergency

ABSTRACT

BACKGROUND:

Maintenance of cerebral blood flow (CBF) depends on a balance between intracranial pressure (ICP) and mean arterial pressure (MAP). In traumatic head injury, ICP is raised interfering with normal CBF. Invasive monitoring of ICP and MAP is the ideal tool for determining cerebral perfusion pressure (CPP) in severe traumatic head injury. This study aimed at establishing how noninvasive MAP measurement associated with outcome after severe traumatic brain injury and its possible role in monitoring.

METHODS:

This was a prospective analytical study carried out over ten month period (November 2007 to August 2008). It involved 73 patients admitted at Kenyatta National Hospital (KNH) with severe traumatic head injury (Glasgow Coma Scale scores 3-8).Their initial Blood Pressure, MAP, Injury Severity Score (ISS), plus physical and radiological findings were recorded as were the interventions involved. They were followed up through the resuscitation phase in intensive care for 3 weeks, then wards and clinics for a maximum of 6 months. Their Glasgow Outcome Scores (GOS) were determined and associations with their MAPs determined.

RESULTS:

There were 6 females and 67 males all totaling to 73. Majority, 80.3% were in the age bracket 20-45 years. Most of the injuries were due to assaults followed by Road Traffic Accidents.

There was no significant association between admission MAP with age, gender, time from injury to admission, intracranial hematomas nor alcohol consumption.

A low MAP (<90mmHg), low Glasgow Coma Score (3-4), and high ISS (>34 ± 2) associated with poor outcome (Severe Disability or death).

Mean arterial pressure at admission did not have an association with outcome at end of follow up but there was a significant negative correlation between Injury Severity Score (ISS) and MAP.

CONCLUSIONS:

- Majority of our patients (over 60%) presented with low MAP (<90mmHg) hence need for intervention. Admission Glasgow coma score (GCS) and ISS were better predictors of outcome than MAP.

- The longer the time to admission, the greater the likelihood to capture patients with a low MAP.

- Improving GCS and GOS associated positively with increasing MAP although no threshold effect was observed.

The role of non invasive MAP in monitoring severe head injury is evident when combined with ISS and GCS in our set up.

1.0: INTRODUCTION

Invasive ICP measurement has been the gold standard in severe head injury monitoring. GCS and MAP can give a reasonable estimate of ICP since intracranial pressure is a function of MAP[1, 2, 3].

Transcranial Doppler ultrasound is a non invasive technique for ICP monitoring which studies the flow velocity waveform in the middle cerebral artery. The importance of Transcranial Doppler ultrasound in head injured patients is that, the flow velocity pulsatility correlates with cerebral arteriovenous oxygen difference as cerebral perfusion pressure falls[4]. Transcranial Doppler studies may not be easily accessible in our set up due to costs of equipment and lack of trained standby staff. This renders it not cost effective for our hospitals. In a study by Trabold *et al.*, the prognostic value of Transcranial Doppler ultrasound was demonstrated in children but this may be difficult to reproduce in adults due to their closed fontannels[5].

Glasgow coma scale has been used for monitoring on head injury charts and to predict likely outcomes after severe traumatic head injury. It has several limitations including observer errors. It cannot be effectively assessed on intubated and sedated patients. Repetitive administration of the score as a tool for monitoring is cumbersome[6].

In studies on predictive ability of GCS using GOS as outcome, only a few studies have done a correlational analysis between GCS and GOS[7]. Most studies have used GCS to prognosticate and not as a tool for monitoring. In the study on outcome and prognostic factors for traumatic brain injury by Demetrios *et al*,. only GCS of 3,hypotension, a high

abbreviated injury scale , age over 55 and penetrating head trauma were considered poor prognostic factors[8]. In the same study, no other GOS outcome category other than death is mentioned. Since it was a retrospective study, many factors could have been omitted.

Secondary brain injury, believed to be ischaemic in origin, is associated with post injury hypotension, hypoxemia, and intracranial hypertension, while hypoxia and hypotension are known to be associated with high mortality and neurologic morbidity[9]. To minimize morbidity and mortality, head injured patients require prevention and prompt treatment of hypotension[10].

Intra-arterial catheterization and monitoring of arterial pressure is ideal for determination of MAP. At KNH, we are not able to routinely do invasive monitoring of ICP and MAP; hence other modalities need to be studied to help improve the management of these patients.

Non invasive MAP determination includes hybrids of auscultatory and oscillatory devices. The MAP can be calculated from sphygmomanometrically determined pressure or continuously monitoring devices that also employ memory. These indirect methods are significantly accurate for diagnostic and therapeutic studies[11, 12, 13, 14] .

Mean arterial pressures of less than 90mmHg have been found to correlate poorly in patients with severe brain injury [4, 15]. This study will look at how non-invasively determined MAP may contribute in the monitoring of these patients in our set up. While

studying the prognostic value of admission blood pressure in traumatic brain injury, Isabella *et al.,* observed a smooth U shaped relationship between systolic BP and MAP[16]. While appreciating the influence of hypotension on outcomes, there was no evidence of an abrupt threshold effect. Good outcomes were observed with systolic BP of the order 135mmHg and MAP of 90mmHg.This was a retrospective analytical study and both extremes of systolic BP and MAP that they correlated with poor outcome were not detailed[16].

Manley G. *et al.*, while studying hypotension, hypoxia and head injury concluded that, hypotension and not hypoxia in initial phase of resuscitation, is associated with poor outcome[17]. Only CT scans with intracranial pathology were used to recruit the patients and no other outcome measure other than death was considered[17]. Czosnyka *et al.*, study incorporated many measured parameters and several statistical methods making it difficult to interpret results[4]. Although they used modern machines, the study and its application may not be applicable in our set up due to the costs involved.

Non invasive continuous mean arterial pressure monitoring will provide an opportunity for us to detect hypotension. It offers the patient, doctors, hospitals and government a cheap, reliable and reproducible tool for monitoring severe head injured patients.

2.0 LITERATURE REVIEW

Earliest reported series on head injury and its management appear around 1700 BC, when four depressed skull fractures were treated by Egyptians by leaving the wounds unbandaged after draining intracranial cavity and anointing the scalp wounds with grease[18]. Mid 19th century saw the advent of antibiotics; Pasteur, Robert Koch in bacteriology, Joseph Lister in asepsis, which dramatically decreased the incidence of local, systemic infections and mortality[19]. In Kenya, the practice of traditional craniotomy by the "ababari emetwe" (craniotomists) of the Kisii tribe of Kenya has probably been on for centuries[20].

Annual incidence of traumatic brain injury in the United States is 180 – 220/100,000 within a total population of 300 Million. A fatality rate of over 10% with over 550,000 patients hospitalized annually has been reported. The male to female ratio is 2:1, and the majorities are below 35 years of age. Over 50% of 100,000 trauma related deaths are secondary to head injuries[21, 22]. Road traffic accidents (RTAs), contribute to over half of the injuries. Other causes include falls, assault, sports related injuries and penetrating trauma. Mwangombe reported 378 admissions with head injury at KNH over a six-month period. Of the 378 admissions, RTAs, assaults and falls contributed the most in that order. The male to female ratio was 4:1 with the age range 25-35 years most affected[23]. Kiboi in reviewing the outcome of severe head injuries at KNH showed that, 10.3% of head injuries were severe out of which 5.3% suffered hypotension. Of the admissions, 63.2% died within 48 hours of admission. Most of these patients' ages were between 26-45 years[24]. Opondo observed that 14.3% of intensive care admissions are secondary to

severe head injury with an attendant mortality of 54%. Glasgow Coma Score less than 5, diffuse axonal injury, intracranial space occupying lesions and blood sugar less than 5 were poor prognostic factors[25].

Since resuscitation and intensive care started to save lives of many severely head injured patients post World War II, neurosurgeons were encouraged since much of the mortality and disability was reduced[26].

Chestnut *et al.* on role of secondary brain injury in determining outcome from severe head injury demonstrated that hypoxia contributed to 6% mortality. Hypotension contributed to 33% while hypoxia and hypotension combined contributed to 48% mortality[27].

In a study on the burdens of secondary insults in head injury by Jones *et al.* hyperglycemia is noted as one of the burden but is not included in subsequent analysis[28,29] .

2.1 PATHOPHYSIOLOGY

Traumatic brain injury may be classified in many ways;

a) Severity as mild, moderate or severe.

b) Resulting pathology as focal or diffuse although mostly mixed.

c) Mechanism of injury as either closed or penetrating.

d) Aetiology as either primary or secondary.

Secondary brain injury refers to any subsequent injury to the brain after initial insult. This results from intracranial hematomas, systemic hypotension, hypoxia, and elevated intracranial pressure or biochemical and metabolic imbalances. Free Oxygen radical production in head trauma has been linked to further cell damage. Cerebral contusions make up to 45% of primary intra-axial traumatic lesions. Intracerebral haematomas make 10%. The rest are epidural, subdural, subarachnoid and intraventricular hemorrhages.

The acute phase response in head injured patients lasts approximately twenty-one days post injury; therefore, physiologic modulation to minimize secondary injury is required through this period[6, 30].

2.1.1 Intracranial pressure and volume relationships

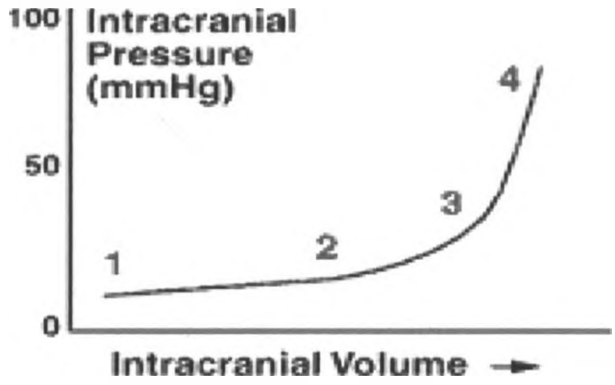
The brain is contained within the skull; a rigid, fixed compartment, hence only small increases in volume can be tolerated before the pressure increases dramatically as defined in the Monroe-Kellie doctrine. The pressure changes within the skull are drawn in a classical curve as shown in Figure 1, showing an increase in volume with little change in pressure until a certain point is reached when further small change in volume results in a large increase in pressure[1].

The intracranial volume (Vi/c) is equal to the sum of its components, as follows;

$$Vi/c = V\ (brain) + V\ (CSF) + V\ (blood)$$

In adults, the total intracranial volume is approximately 1500mls. The brain, accounts for 85-90%, intravascular cerebral blood 10% and CSF <3%. The normal CSF volume is about 75mls with a production rate of about 14 - 36mls/hour.

Figure 1: Showing the relationship between intracranial pressure and intracranial volume.



1-2 compensation phase,

3-4 decompensation phase.

In significant head injury, cerebral edema develops increasing the relative volume of the brain. The ICP will rise unless some compensatory action occurs such as the decrease in the volume of one of the other intracranial components hence the concept of intracerebral compliance, which refers to the change in pressure due to changes in volume.

Compliance = change in volume/change in pressure.

Compliance is based on the pressure volume index (PVI), which describes change in intracranial pressure that occurs when a small amount of fluid is added to or withdrawn from the intracranial compartment. Simply stated, therefore, the brain has very limited

compliance and cannot tolerate significant increases in volume. Cerebral Perfusion pressure (CPP) is the difference between the MAP and the ICP.

$$CPP = MAP - ICP$$

The MAP is diastolic pressure plus one third of the pulse pressure (difference between the systolic and diastolic). Mean arterial pressure is therefore nearer diastolic, and is the best value to estimate the "head of pressure" perfusing in the brain:

$$MAP = \text{Diastolic BP} + 1/3 \text{ Pulse Pressure}$$

CPP is the net pressure of blood delivery to the brain. Normal CPP is 80mmHg.

2.1.2 Cerebral blood flow

When CPP is reduced to 50mmHg, there is metabolic evidence of ischemia and reduced electrical activity[31]. In normal adult CBF is constant in the range of MAPs of 50-150mmHg. Normal CBF is 45-50ml/100gm/minute. Physiologic determinants of CBF are; CPP, arterial Oxygen tension (PaO_2), arterial carbon dioxide tension (pCO_2) and cerebral metabolic rate. It is a result of auto regulation where arterioles constrict or dilate within a specific range of blood pressure to maintain a constant amount of blood flow to the brain[28, 32].

Auto regulation is poorly understood[30, 33]. It is thought to be due to the interplay between myogenic and metabolic mechanisms that influence perivascular nerves plus the vascular endothelium. There are several forms of auto regulation that have been postulated hence; pressure controlled, viscosity dependant and metabolic auto-regulation. Pressure auto regulation holds that between the systolic BP of 50 and 160mmHg, the diameter of

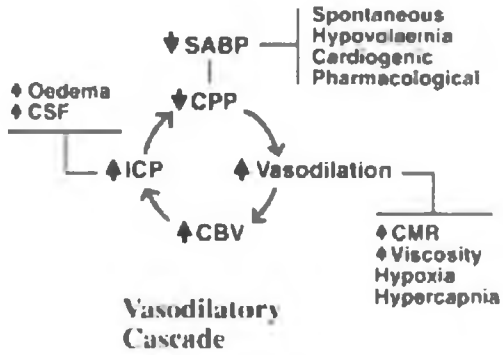
cerebral arterioles is altered to maintain a constant blood supply to the brain. Viscosity auto regulation alters blood vessel caliber therefore the blood flow to the brain in response to changes in blood viscosity. Metabolic auto regulation expounds the responsiveness of cerebral vasculature to metabolites[31, 34]. In normal brain, the blood brain barrier regulates entry of endogenous and exogenous substances into parenchyma based on size, charge and lipid solubility.

Electrolyte concentration is maintained by selective permeability and continuous action of ionic pumps throughout the brain. Auto regulation therefore, controls the arterial caliber depending on physiological changes (Fig 2a, 2b). However, in the traumatized brain CBF may become blood pressure dependent. Hence as the arterial pressure rises it causes an increase in cerebral volume. Similarly as the pressure falls, so will CBF, therefore, reducing ICP but also inducing an uncontrolled reduction in CBF[35, 69].

In this situation if the CPP falls below the critical value of 70 mmHg, the patient will have inadequate cerebral perfusion. Auto regulation will cause cerebral vasodilatation leading to a rise in brain volume. This in turn will lead to a further rise in ICP and induce the vicious circle described by the vasodilatation cascade (Fig 2a), that results in cerebral ischemia.

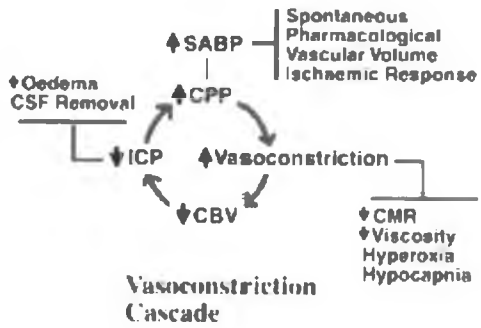
In the figures 2a and 2b, CMR = Cerebral metabolic rate; SABP = systemic arterial blood pressure and CBV = cerebral blood volume.

Figure 2a: Showing the vasodilatation cascade.



This process can only be broken by increasing the blood pressure to raise CPP, inducing the vasoconstriction cascade (Fig 2b). This explains why the maintenance of arterial blood pressure at adequate levels by careful monitoring and rapid correction if it falls is so important[36, 37, 38]. Normal ICP in adults is 5-15 mmHg. Intracranial pressure greater than 20mmHg signifies intracranial hypertension[66,67,68].

Figure 2 b: Showing vasoconstriction cascade.



2.1.3 Measuring intracranial pressure.

At autopsy in the Natal study, failure to recognize raised ICP was found to have significantly contributed to mortality[39].

Jugular bulb catheter monitoring may be useful in severely ill patients. Oxygen extraction exceeding 60% shows minimal oxygen delivery (Normal oxygen extraction is 30-40%). Due to the cost and disadvantages associated with these invasive techniques (table 1), a reasonable inference on the ICP can be deduced. This is achieved by monitoring the MAP and GCS then making a reasonable inference:

Drowsy and confused; (GCS 13-15), ICP = 20mmHg while severe brain swelling; (GCS<8), ICP = 30mmHg[1, 2, 64]. Transcranial Doppler is a promising non-invasive technique for monitoring cerebral auto regulation in head injured patients. Flow velocity pulse waveforms within the middle cerebral arteries in head injured patient's correlates with the cerebral arteriovenous oxygen difference as CPP falls, which has clinical significance[40].

The gold standard for measuring of MAP is direct intra-arterial measurement with a catheter. Clinically, it is determined by measuring the systolic and diastolic blood pressures then calculated using the formula;

MAP = Diastolic BP + 1/3 Pulse Pressure.

Table 1: ICP monitoring techniques.

DEVICE	ADVANTAGE	DISADVANTAGE
Intraventricular catheter	Potentially most accurate	Risks bleeding, infection, tissue damage
Subarachnoid bolt	Ease of placement. Decreased potential for tissue damage. Low infection rate	Potential for loss of pressure wave due to micro leaks. Easily obstructed No removal of CSF Reading may not reflect global ICP
Counter pressure systems (Ladd ICP monitor)	Low infection rate	No observable wave form Difficult to zero and calibrate in vivo
Fibreoptic ICP device (Camino monitor)	Continuous waveform available. Records pressures from any intracranial location. In vivo calibration possible.	In vivo zeroing out not possible. Monitor failure occurs with damage to optic fibers

2.2 BLOOD PRESSURE AND MAP MONITORING.

In 1733, Reverend Stephen Hales inserted a long glass tube upright into an incision in a horse's artery, with arterial pressure causing blood level to rise in the tube[36].

The automated devices of today apply sound based algorithms to estimate BP although the machines lack validation ability. The devices may not adequately compensate for patient conditions such as hypotension (low BP) where Korotkoff sounds may be muted.

To make automated measurement more reliable, oscillometric devices were created. The term "oscillometric" refers to any measurement of the oscillations caused by the arterial pulse.

Unlike auscultatory techniques, which measure systolic and diastolic but estimate MAP, oscillometric techniques measure MAP but estimate systolic and diastolic pressures. Alternative blood pressure and MAP determination include hybrids of both auscultatory and oscillatory devices. Others are infrasound, ultrasound, impedance plethysmography and arterial tonometry. The direct and indirect methods yield similar measurements, but these are rarely identical since the direct method measures pressure while the indirect is more indicative of flow. The indirect method is sufficiently accurate for diagnostic and therapeutic studies[11, 12, 13, 14]. It is simple, practical, low in cost and noninvasive.

Blood pressure fluctuates from day to night over long periods of time. A variety of other factors do influence blood pressure hence; trauma, exercise, sleep, temperature, emotional and psychological factors. The British Hypertension Society has recommended the use of one large cuff (21.5 by 35cm) for all adults with an arm circumference up to 42cm[14].

2.3 THE GLASGOW COMA AND OUTCOME SCALES.

The GCS described by Jennet and Teasdale in 1974 describes the level of consciousness and broad categories of head injury[41, 42].

Hence;

Mild head injury 13-15

Moderate head injury 9-12

Severe head injury <8

For intubated patients maximal score is 10T and poorest 2T [32]. Overall score lies between 3 and 15.

2.3.1 Indications for use of the Glasgow coma scale.

The GCS findings form the basis for clinical management decisions, such as necessity for computed tomography, surgical intervention and drug modalities. The GCS, along with cardiovascular and respiratory status, are the major components of the Revised Trauma Score, a predictive tool for survivability of trauma patients. The GCS is universal, easy to compute and communicate to others.

2.3.2 Limitations of the GCS.

Premedication of trauma patients during pre hospital care with paralytic, anxiolytic, and analgesic drugs for the purposes of airway management, tracheal intubation is common. In high complete spinal cord injury together with head injury, the patient will typically be intubated, unable to move all extremities and may not be able to open his or her eyes. The GCS is used to assess adolescent and adult patients but not paediatric patients less than three years since it lacks the developmental considerations.

2.3.3 The Glasgow outcome scale.

This describes the overall social function rather than neurological deficits, and is useful in monitoring recovery. The outcomes so described at six months after injury correlate well

with early coma scale scores, which are therefore useful predictors of likely outcome. The GOS is composed of five exclusive categories with corresponding scores, good recovery (GOS 5) to death (GOS 1) [43]. These summarize the social capacity of the patient rather than listing specific disabilities thus;

Good Recovery (5)

Resumption of normal life, with capacity to work even if pre-injury status has not been achieved. Some patients have neurological or psychological deficits.

Moderate Disability (4)

Have some disability like dysphasia, hemi paresis, epilepsy, and memory or personality deficits but are able to look after themselves.

Severe Disability (3)

A conscious patient who is dependent for daily support from another person by reason of mental or physical disability or both.

Persistent vegetative state (2)

Unconscious patient who is wholly dependant on other people.

Death (1)

Applies only when the death occurs either in the initial period of hospital or a specified time thereafter.

2.4 INJURY SEVERITY SCORE.

Head injuries do not occur alone but in association with other injuries which could determine morbidity and mortality [5, 44].

The ISS is defined as the sum of squares of the highest Abbreviated Injury Scale (AIS) grade in the three most severely injured body regions[45]. The AIS is an anatomically

based system of grading injuries ranging from 1(minor injury) to 6(lethal injury). In the ISS, six body regions are defined hence, the thorax, abdomen and visceral pelvis, head and neck, face, bony pelvis and extremities, and external structures. Only one injury per body region is allowed. The ISS ranges from 1-75. An ISS of 75 is assigned to any patient with AIS of 6.

2.5 MANAGEMENT OF SEVERE HEAD INJURY.

Treatment of these injuries begins with Advanced Trauma and Life Support guidelines to prevent hypoxia and hypotension[46, 65]. After stabilization and determination of GCS, a neurologic examination should be performed then a CT scan obtained. Less than 10% of patients with traumatic brain injury have an initial surgical lesion. Other interventions on the stabilized patient include prophylaxis against Cushings ulcers using proton pump inhibitors, H_2 antagonists and sucralfate. Coagulopathy like disseminated intravascular coagulation should be watched for and anticonvulsants prescribed on merit to prevent seizures.

Indications of surgery include;

- Extra axial haematoma with midline shift greater than 5mm.
- Intra axial haematoma with volume greater than 30mls.
- Open skull fracture.
- Significant depressed skull fracture (more than 1 cm inward displacement).
- Temporal or cerebellar haematoma greater than 3cm in diameter.

Intravenous fluids are administered to maintain the patient in a state of euvolemia or mild hypervolemia. Fluid restriction decreases intravascular volume and therefore cardiac

output. A decrease in cardiac output often results in decrease in cerebral flow hence decreased CPP and may lead to increase in cerebral edema and ICP (Fig 2a). While correlating different levels of ICP and CPP, Niels Juul. *et al.,* found that ICP of over 20mmHg was a powerful predictor of neurologic worsening. They were not able to demonstrate any significant benefit of CPP greater than 60mmHg[47]. Measures should be instituted to keep ICP less than 20mmHg.

The volume of one of the intracranial components must be reduced to improve intracranial pressure.

Blood.

Head elevation by 20-30 degrees increases venous outflow and decreases volume of venous blood in the brain. This has only a modest effect on ICP[48].

Arterial blood volume.

Mild to moderate hyperventilation in which PCO_2 is reduced to 30-35mmHg causes arterial vasoconstriction decreasing the blood volume enough to reduce ICP and this effect lasts 48-72 hours.

Cerebrospinal fluid (CSF).

CSF production in adults approximates 20mls/hour or 500mls/day. External ventricular drain with removal of small amounts of CSF hourly can greatly improve ICP. This offers temporary relief with a high risk of infection.

Brain tissue component.

When edema is present, this is the component whose volume increases. Treatments of raised ICP that reduce total brain volume include,

- Volume targeted strategies (Lund Concept)[35] .
- Perfusion augmentation strategies (CPP strategies).
- Metabolic suppression.
- Decompressive procedures.

Mannitol is an osmotic diuretic commonly used and has a rapid onset of action and duration of action of 2-8 hours. It is given as a bolus at 0.25-1g/kg every 4-6 hours. It is more effective if given as intermittent boluses than infusion. Careful hydration has to be administered to maintain euvolemia. Maximal daily dose is 4g/kg/day. Daily doses higher than this can lead to renal toxicity. Mannitol should not be given to patients whose serum sodium Osmolality is greater than 315 mOsm or sodium level greater than 145meq/L. It is preferred to frusemide as it causes less severe electrolyte imbalance compared to loop diuretics. Mannitol and frusemide combined have a synergistic action but with more severe electrolyte imbalances[49]. Urea and glycerol have been used as osmotic diuretics but have smaller molecules and tend to equilibrate in the brain faster than Mannitol. Accidental skin infiltration with urea causes sloughing.

CPP management strategies involve artificially elevating BP to increase MAP and the CPP. These are based on the understanding that auto regulation is impaired in the injured brain and pressure passive flow develops in those areas leading to tissue acidosis and lactate accumulation. Vasodilatation occurs with increase in cerebral edema and ICP.

Raising CPP to greater than 65-70 mmHg lowers ICP as blood flow to injured areas increases hence the acidosis decreases. Hypertonic saline has been recommended by some workers as increase in serum sodium correlates with lower ICP but a higher CPP[50].

Metabolic therapies are designed to decrease the cerebral metabolic rate, which decrease ICP. It can be achieved via drugs or induced hypothermia. These therapies are reserved for situations where other interventions have failed as they have severe adverse effects including; hypotension, immunosuppression, coagulopathies, arrhythmias and myocardial suppression. Barbiturate coma is induced with pentobarbital at 10mg/kg over 30 minutes then 5 mg/kg for 3 hours and maintained at 1-2 mg/kg/hour. This is maintained up to 48 hours.

Mild hypothermia involves decreasing core temperature to 34-35 degrees centigrade for 24- 48 hours then slowly rewarming the patient over 2-3 days. Hypothermic patients risk hypotension and systemic infections[51].Decompressive craniotomy is used in patients with traumatic head injury with refractory ICP elevation. A large section of the skull is removed and the dura is expanded.

3.0 STUDY RATIONALE AND JUSTIFICATION.

Management of severe traumatic brain injury at KNH is focused on raised intracranial pressure based on identifiable intracranial space occupying lesions and CT scan findings of edema or compound fractures. Only a limited number of patients benefit from intraventricular catheterization and ICP monitoring. Likewise, only a few have central venous pressure measurements. Unfortunately in our setup, the cost of invasive monitoring of ICP as recommended, including space, equipment and manpower are prohibitive.

Limited bed space in the intensive care unit on many occasions leads to severely injured patients being admitted to general surgical wards where monitoring is minimal and probably reflects on outcome. Patients (less than 72 hours post trauma) are managed in the general surgical wards unless they are intubated and need mechanical ventilation which is only possible in ICU, Neurointensive unit and A&E. This calls for cost effective techniques in monitoring and managing these patients. Previous studies at KNH have identified factors contributing to secondary brain injury after trauma but specific factors like hypotension, hypoxia and raised ICP have not been addressed in depth.

This is a prospective study that specifically seeks to isolate independent factors for analysis unlike previous retrospective studies that omitted some factors due to inadequate recorded data. Most studies on MAP and outcome are invasive and carried out in other centres which may not reflect our picture.

This study hopes to add knowledge on threshold levels of MAP that should prompt intervention in our set up. The study seeks to offer cheaper tools for monitoring other than the invasive techniques.

3.1 OBJECTIVES.

3.1.1 Main objective.

To determine the role of non-invasive mean arterial pressure measurement in monitoring patients with severe head injury in our set up.

3.1.2 Specific Objectives.

1. To determine the mean arterial pressures at admission in patients with severe injury.
2. To determine the association between admission mean arterial pressure and various independent factors that could influence it.
3. To determine the association between initial mean arterial pressure and Glasgow Coma Scale at admission.
4. To determine how the initial mean arterial pressure at admission associated with outcome of patients with severe head injury.

3.2 STUDY METHODOLOGY.

3.2.1 Study design.

This analytical prospective study was carried out following approval by KNH ethics and research committee. It revolved around the Accident and Emergency department (A/E), Intensive Care Unit (ICU), High Dependency Unit (HDU), neurosurgical unit, general surgical and Orthopaedic wards.

3.2.2 Study area.

This study was carried out at KNH. The hospital is a national and regional referral facility with 50 wards and over 1800 beds situated in the capital city, Nairobi Kenya.

3.2.3 Study population.

Patients aged 12 and above admitted with severe traumatic head injury (GCS 3-8).

3.2.4 Sample size

Calculated using the formula by Kish and Leslie;

$\mathbf{N = Z^2 p (1 - p)/D^2}$

N = Sample size

Z = Standard error from mean corresponding to 95% level of confidence (1.96)

P = p-value at 0.05

D = Absolute precision (0.053)[24].

Taking **P** to be 0.05 and **D** to be 0.053, then;

$$N = \frac{(1.96 \times 1.96) \times 0.05(1-0.05)}{(0.053 \times 0.053)}$$

$= 73.$

3.2.5 Sampling method.

A trial run of the questionnaire was carried out on 10 randomly selected patients at A/E. A portable mercury blood pressure machine with 21.5 by 35 cm portable sphygmomanometer cuff and the bell of a class 11 Littman's stethoscope were used. The focus here was to correlate the portable BP machine readings with those of the newly installed Oscillometric Phillips monitors in A/E and ICU that are still within the calibrated one year warranty.

Blood pressures taken using the mercury BP machine were then compared with those recorded by the Phillips monitors in the resuscitation room at A/E department on the same patients. The mean difference between the two measurements was found to be ± 3 mmHg. Successive severe head injury patients aged 12 years and above were then screened for inclusion into the actual study until the sample size of seventy three was achieved.

3.2.6 Inclusion criteria.

All acute severe head injury patients (GCS 3 – 8) aged 12 years and above presenting at the Accident and Emergency department, admitted and consent obtained from either the guardian, A/E or ICU consultants.

3.2.7 Exclusion criteria.

1. Dead on arrival.

2. Patients who did not consent.

3. Referrals 3 weeks after injury.

4. No initial blood pressure recordings.

3.2.8 Data collection procedure.

All patients had a Glasgow Coma Score of between 3 and 8. In addition to patient demographics, brief history on place, time; mechanism of injury, alcohol ingestion prior to injury, comorbity and initial intervention was obtained in each case followed by examination.

The initial blood pressure (± 3mmHg), Glasgow coma score (3-8), degree of injury were recorded amongst other parameters as per questionnaire. The injury severity scores (expressed as sum of squares of the abbreviated injury scores in 3 most injured body regions; range 3-75) and mean arterial pressures (mmHg) were then computed and recorded.

Once admitted, the patients were followed up and information about intubation/type of ventilation, CT scan findings, surgical procedures and outcomes were recorded. Branded Phillips monitors capable of a 24 hour continuous blood pressure, mean arterial pressure and Oxygen saturation monitoring memory were employed at A/E, ICU and HDU but the portable BP machine for the general surgical wards and clinics.

All patients were then followed up through the hospital process in ICU, wards and clinics on discharge. All the requisite parameters were recorded at set times much as the overall status was retrieved from the monitor memories.

3.2.7 Ethical consideration.

The study commenced on 21st of November 2007 after authority was granted by the KNH Ethical and Research committee upon perusal and approval of my proposal.

I maintained confidentiality of the patient and my findings throughout the study and undertook to publish the results on completion of the study.

Those who qualified for inclusion but declined to consent were not denied of services.

Informed consent was obtained from the next of kin, A/E or ICU consultants (my supervisors did not consent for any patient at any time).

3.2.8 Data Management.

Data from the questionnaire was entered into Epi data software and then exported to SPSS 11.5 for analysis. Data editing and reconciliation which included coding and crosstabulation was undertaken before analysis was done.The analysis was supplemented by other packages like Microsoft excel.

Categorical data like GCS and GOS was expressed as subgroups and Spearman's correlation done. Non parametric statistical methods were also used since these variables did not have normal distribution. Rank correlation coefficients were calculated between

grade of initial Glasgow Coma Score and Glasgow Outcome Score at 3 months and six months, patients' age and studied parameters.

Pearson correlation was used for scale variables hence; MAP and time from injury to admission in hospital.

Analysis of variance (ANOVA) was used to present differences in selected indices (means of mean arterial pressure) in separate Glasgow outcome groups. P value of <0.05 was considered significant.

3.2.9 Study Limitations.

1. This was an analytical study hence increased possibility of observer error.
2. The machines utilized had manufacturer calibration and were assumed to be still within international calibration standards.
3. The study duration was short and sample size small hence large standard error.

4.0 RESULTS

A total of 73 patients were recruited. Seven (9.6%) of the recruited patients were excluded from the final analysis. Of the seven who were excluded, two (2.7%) of the patients had absconded from the hospital. The remaining 5 (6.8%) patients were lost during follow up. Sixty six (90.4%) patients of the recruited 73 were included in this final analysis.

Majority of the patients were in the 20 to 45 year age bracket. The male to female ratio was 10:1(60 men and 6 females). Eight (12.1%) patients were referrals from outside the city.

Assaults contributed to most of the injuries followed by road traffic accidents at 30(45.5%) patients and 26(39.4%) patients respectively. Falls contributed to 6(9.1%) severe head injuries, two (3%) patients had missile injuries and 2 (3.0%) patients were unclassified. Twenty two (33.3%) patients took 6 hours to reach the Accident and Emergency room from time of injury while 10(15.2%) took over 72 hours.

Fifty three patients (80%) had a history of alcohol ingestion prior to injury or smelt of it at admission but determination of blood alcohol levels was not carried out. Forty one patients (62.1%) had haematomas followed by diffuse axonal injury 10(15.2%), contusions and edema in 6(9.1%) patients and concussion in 2(3.0%) patients. Fifteen (22.7%) patients underwent surgery while 51(77.3%) patients were managed conservatively. Thirty four (51.5%) patients had mean arterial pressures above 90mmHg.

The mean of mean arterial pressures for patients who died was 79mmHg (± 2.93) while those who survived was 93mmHg (± 3.01).While GCS was categorized as 3-4, 5-6 and 7-8 ,the mean of the MAP versus ISS respectively were 78,90,91 and 38,38,32.

All patients admitted received intravenous phenytoin prophylaxis. Post traumatic epilepsy was reported in 6 patients with Severe Disability (SD) and Persistent Vegetative State (PVS) in spite of phenytoin. Two (0.3%) patients were categorized as PVS. The mortality in this study at 6 months was 27 (40.9%) patients.

Mean arterial pressure was divided into subgroups hence >50mmHg, 51-90mmHg and over 90mmHg then subsequently treated as categorical data in the analysis and not continuous data against the independent variables. However, means of MAP were treated as continuous data and correlation carried out with means of ISS.

4.1 SUMMARY OF RESULTS

Table 2: Showing a summary of the results.

ITEM	VALUE	%/RATIO/UNIT
Age group(years)	20-45	80.3%
Sex (M:F)	60:6	10:1
Aetiology; Assaults	30	45.5%
RTA	26	39.4%
Falls	6	9.1%
Missiles	2	3.0%
Others	2	3.0%
MAP(Mean) Survivors	93 (± 3.01)	mmHg
Dead	79 (± 2.93)	mmHg
Hematomas	41	62.1%
Use of mechanical ventilation	41	62.1%
Mortality	27	40.9%
GCS(Category) Versus MAP (Mean)		
3-4	78 (± 2.84)	mmHg
5-6	90 (± 2.47)	mmHg
7-8	91 (± 3.01)	mmHg
GCS,MAP(Mean) Versus ISS(Mean)		
3-4	78mmHg	38 (± 1.23)
5-6	90mmHg	38 (± 1.64)
7-8	91mmHg	32 (± 2.34)

4.1.1 Distribution of Patients by MAP at admission versus Age

Table 3: Showing the distribution of MAP amongst Age groups.

MAP	Age					Total
	12-20 years	20-45 years	45-55 years	55-60 years	>60 years	
<50	0	6(85.7%)	0	1(14.3%)	0	7(100%)
51-90	3(8.1%)	31(83.8%)	1(2.7%)	1(2.7%)	1(2.7%)	37(100%)
>90	1(4.5%)	16(72.7%)	4(18.2%)	0	1(4.5%)	22(100%)
TOTAL	4(6.1%)	53(80.3%)	5(7.6%)	2(3.0%)	2(3.0%)	66(100%)

The table 3 above shows the distribution of patients by MAP and age. Fifty three (80.3%) patients were from the age bracket of between 20 and 45 years. Fourty four (66.7%) patients had MAPs below 90mmHg. Patients between the ages of 45 and 55 years were 5(7.6%) followed by those in the age bracket of 12 and 20 years who were 4(6.1%). The number of patients above the age of 55 years was 4(6.1% of total population). This indicated that extremes of age had few patients involved. There was no significant association between MAP and age ($\chi^2 = 8.98$; $p = 0.062$).

4.1.2 Distribution of MAP by Gender

Table 4 below shows the distribution of patients' MAP by gender. There were more male patients than females who were investigated within all MAP groups with 39 males (65.0% of the male patients) having MAPs below 90mmHg. Five (83.3%) out of the 6 females had MAPs below 90mmHg. Chi-square was used to determine whether there was

any significant relationship between the MAP and gender of the patients and none was found ($\chi^2 = 2.221$, $p = 0.329$).

Table 4: Showing the distribution of MAP by Gender.

MAP	Gender		Total
	Male	Female	
<50	7(100.0%)	0	7(100%)
51-90	32(86.5%)	5(13.5%)	37(100%)
>90	21(95.5%)	1(4.5%)	22(100%)
Total	60(90.9%)	6(9.1%)	66(100%)

4.1.3 MAP and Cause of Injury

Table 5: Showing relationship between MAP and cause of injury

	Cause of injury					
MAP	RTA	Fall	Assault	Missile	Others	Total
<50	2(33.3%)	1(16.7%)	2(33.3%)	1(16.7%)	1(1.5%)	7(100%)
51-90	15(40.5%)	2(5.4%)	19(51.4%)	1(2.7%)	0	37(100%)
>90	9(40.9%)	3(13.6%)	9(40.9%)	0	1(4.5%)	22(100%)
Total	26(39.4%)	6(9.1%)	30(45.5%)	2(3.0%)	2(3.0%)	66(100%)

Table 5 above shows the distribution of the patients according to MAP groups and causes of injury. Assaults resulted in 30 (45.5%) severe head injuries followed by RTAs 26 (39.4%) and falls 6 (9.1%) patients. Twenty one of the 44 (47.8%) severe head injuries with low MAP were due to assaults. There was no significant relationship between MAP and cause of injury ($\chi^2 = 7.735$ $p = 0.460$).

4.1.4 MAP and Time between Injury and Hospitalization

Figure 3: Showing Percentages of population and Time from injury to hospitalization

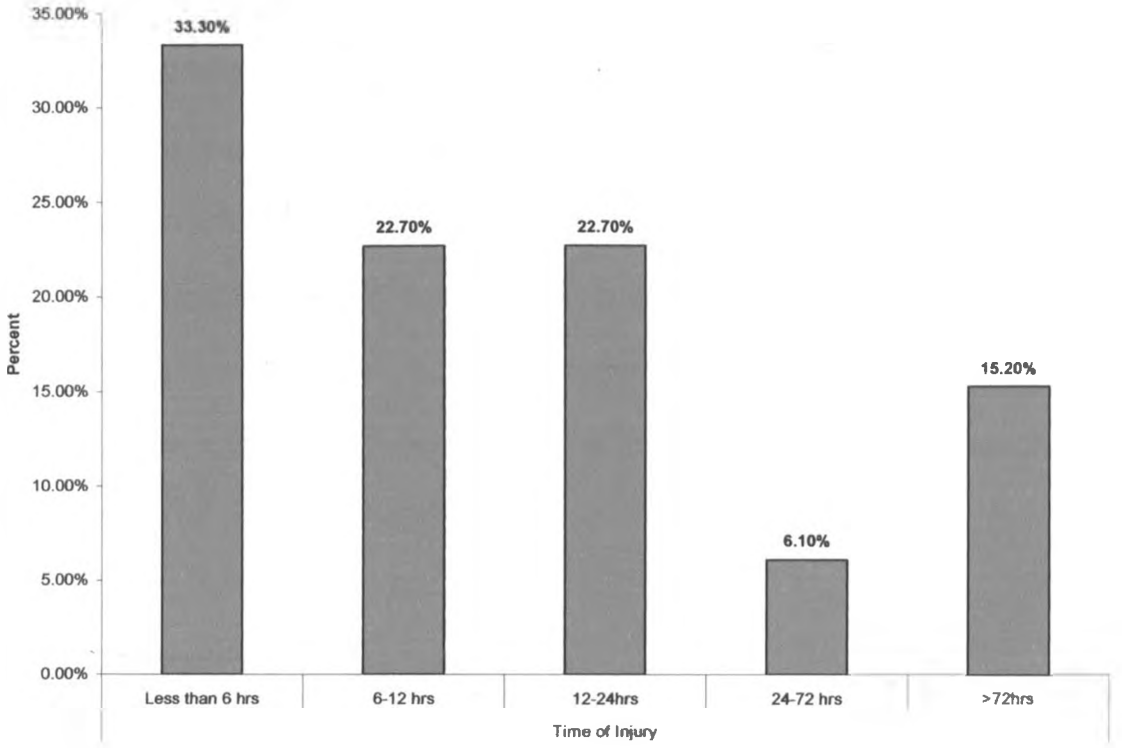


Figure 3 above show the duration between the time of injury and time of admission. Twenty two (33.3%) patients representing the majority took 6 hours to get to the A/E department and subsequently got admitted. Fifteen (22.7%) patients took between 6 to 12 hours and a similar number between 12 to 24 hours from time of injury to admission. Thirty (45.5%) patients therefore took between 6 and 24 hours to admission. Four (6.1%) patients who formed the minority took between 24 and 72 hours from injury to admission. The remaining 10(15.2%) patients arrived after 72 hours. The number of patients therefore tended to diminish by time with most (33.3%) arriving in first 6 hours and minority (6.1%) within 24 to 24 hours. The peak after 72 hours (15.2%) represented

eight referrals (12.1%) from other health facilities outside the city and only 2(3.1%) straight admissions from within Nairobi.

Fourteen (63.6%) of the 22 patients who arrived in first 6 hours, 9 (60.0%) of the 15 patients who presented within 6 to 12 hours and 10 (66.7%) of the 15 who were admitted between 12 to 24 hours upon injury had MAPs below 90mmHg.Three of the 4 (75%) patients admitted between 24 and 72 hours had a MAP below 90mmHg.Eight (80%) of the 10 patients admitted after 72 hours had MAPs below 90mmHg.

Over 60% of the admissions in all the intervals of admission had a MAP below 90mmHg.There was no significant relationship between MAP and time from injury to hospitalization ($\chi^2 = 7.402$, $p = 0.494$)

4.1.5 Alcohol Intoxication and MAP

Table 6: Showing the association of MAP to Alcohol consumption

MAP	Yes	No	Yes (%)	No (%)
<50	7	0	13.2	0.0
51-90	26	11	49.1	84.6
>90	20	2	37.7	15.4
Total	53	13	100.0	100.0

Table 6 above shows the state of alcohol intoxication at the time of their injury. Fifty three (80.0%) of the patients were supposedly intoxicated with alcohol at the time they arrived at the hospital, only 13 (20.0%) were not. Thirty three of the 53(62.3%) patients who were positively associated with alcohol ingestion prior to injury had MAP below 90mmHg. For the non-intoxicated; eleven (84.6%) patients had MAPs below 90mmHg and 2(15.4%) patients above 90mmHg.

There was no significant association between MAP and alcohol consumption (χ^2 =0.625 p=0.732).

4.1.6 MAP versus CT scan Findings

Table 7: Distributions of MAP versus CT scan Findings.

		CT scan findings						Total
		Concussion	Contusion	Laceration	Haematomas	DAI	Oedema	
MAP Group	<50	0	1	0	6	0	0	7
	51-90	1	4	0	19	9	4	37
	>90	1	1	1	16	1	2	22
Total		2	6	1	41	10	6	66

The diagnosis reached at based on CT scan findings is presented in the table 7 above. Most of the patients had haematomas 41(62.1%) followed by Diffuse axonal injury (DAI) 10(15.2%). Contusion and edema had 6(9.1%) patients each while concussions were 2(3.0%). Chi square test was carried out between MAP versus CT scan findings (diagnosis) and no significant relationship was established in either case (χ^2 =5.885 p = 0.660).

4.1.7 Use of Mechanical Ventilation

Table 8: MAP Vs Use of mechanical ventilation

	Used Mechanical ventilation		
MAP	Yes	No	Total
<50	4(57.1 %)	3(42.9%)	7(100%)
51-90	23(62.2%)	14(37.8%)	37(100%)
>90	14(63.6%)	8(36.4%)	22(100%)
TOTAL	41(62.1%)	25(37.9%)	66(100%)

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Table 8 shows that 41(62.1%) were mechanically ventilated while 25(37.9%) were not. Of the patients who were not ventilated mechanically, only one was designated 'do not resuscitate' as the injuries were incompatible with life. Seventeen (25.8%) patients who had MAPs less than 90mmHg were not mechanically ventilated although generally more patients were ventilated in every MAP group than those who were not. No significant relationship was found between MAP and mechanical ventilation ($\chi^2 = 1.445$, $p = 0.486$).

4.1.8 Surgical Intervention

Table 9 below shows the distribution of patients who required surgical intervention after injury versus their MAPs. Fifteen (22.7%) of the 66 patients underwent surgery while the rest (77.3%) did not. Eight of the 15 (53.3%) patients who underwent surgery had MAPs below 90mmHg. The most frequent operations were craniotomy and surgical toilets and the two were undertaken at equal rates. A larger population of patients (77.3%) required conservative management. No significant relationship existed between the two variables ($\chi^2 = 8.036$, $p = 0.436$).

Table 9: Distribution of patients by MAP and Surgical Intervention.

MAP	Type of Operation					Total
	Burr Holes	Craniotomy	Surgical Toilet	Orthopedic surgery	Other	
<50	1(50.0%)	0	1(50.0%)	0	0	2(100%)
51-90	0	2(33.3%)	2(33.3%)	0	2(33.3%)	6(100%)
>90	1(14.3%)	3(42.9%)	2(28.6%)	1(14.3%)	0	7(100%)
Total	2(13.3%)	5(33.3%)	5(33.3%)	1(6.7%)	2(13.3%)	15(100%)

4.1.9 MAP versus ISS

Table 10: Correlation between MAP and ISS in specific age groups

		Mean	±
12-20yrs	MAP	81.3	2.61
	ISS	23.0	3.98
20-45yrs	MAP	86.6	3.20
	ISS	37.1	1.88
45-55yrs	MAP	107.0	2.92
	ISS	29.4	5.73
55-60yrs	MAP	62.0	3.34
	ISS	35.0	2.42
>60yrs	MAP	100.0	1.38
	ISS	30.5	3.67

To determine the pattern of MAP within the defined age groups, and to correlate the MAPs with the severity of injury table 10 above was designed. Pearson correlation was carried out on the means of the patients' MAP and ISS. There was a significant negative correlation between ISS and MAP ($r = -0.382$, $p=0.001$). This indicated that a unit increase in ISS lead to a corresponding decrease in MAP.

4.1.10 MAP versus GCS at Admission

Table 11: MAP Vs GCS

	GCS			Total
MAP	3-4	5-6	7-8	
<50	3(42.8%)	2(28.6%)	2(28.6%)	7(100%)
51-90	10(27.0%)	13(35.1%)	14(37.8%)	37(100%)
>90	5(22.7%)	7(31.8%)	10(45.5%)	22(100%)
Total	18(27.3%)	22(33.3%)	26(39.4%)	66(100%)

The GCS groups subcategorized as 3-4 to imply poor GCS score, 5-6 as moderate and 7-8 as favourable score. The table above shows that with increasing MAP the GCS score tended more towards the favourable range although no significant association was established between the two variables ($\chi^2 = 4.702$, $p = 0.319$).

4.1.11 GCS and GOS

Table 12 .Correlation between GCS and GOS

		Glasgow outcome Scale					Total
		Good recovery	Moderate disability	Severe Disability	PVS	Death	
GCS	3-4	3(16.7%)	2(11.1%)	1(5.3%)	2(10.5%)	10(55.6%)	18(100%)
	5-6	2(9.1%)	5(22.7%)	5(22.7%)	0	10(45.5%)	22(100%)
	7-8	8(30.8%)	6(23.1%)	5(19.2%)	0	7(26.9%)	26(100%)
Total		13(19.7%)	13(19.7%)	11(15.7%)	2(2.9%)	27(40.9%)	66(100%)

The GCS predicted outcome fairly accurately in all categories of GOS. Chi square test was done on the variables hence; ($\chi^2 = 20.782$, $p = 0.008$).

Therefore, a lower GCS corresponded with a poorer GOS (Table 12). Spearman's correlation was carried out between the admission GCS and ISS (Table 13) with a significant negative correlation arrived at ($r = -0.131$ $p=0.01$).There was a negative correlation between ISS and GCS. This meant that a unit increase in injury severity score was reflected in a lower GCS score.

4.1.12: GCS and ISS

Table 13: Spearman's correlation between GCS and ISS

			ISS	GCS
Spearman's rho	ISS	Correlation Coefficient	1.000	-.131
		Sig. (2-tailed)	.	.281
		N	66	66
	GCS	Correlation Coefficient	-.131	1.000
		Sig. (2-tailed)	.281	.
		N	66	66

4.1.13 MAP versus GOS

Table 14: MAP versus GOS

	Glasgow outcome Scale					Total
	Good recovery	Moderate disability	Severe Disability	PVS	Death	
<50	0	1(14.3%)	0	0	6(85.7%)	7(100%)
51-90	7(18.9%)	8(21.6%)	9(24.3%)	1(2.7%)	12(32.4%)	37(100%)
>90	6(27.2%)	4(18.2%)	2(9.1%)	1(4.5%)	9(40.9%)	22(100%)
Total	13(19.1%)	13(20.6%)	11(16.2%)	2(2.9%)	27(40.9%)	66(100%)

Figure 4: Distribution of patients MAP versus GOS

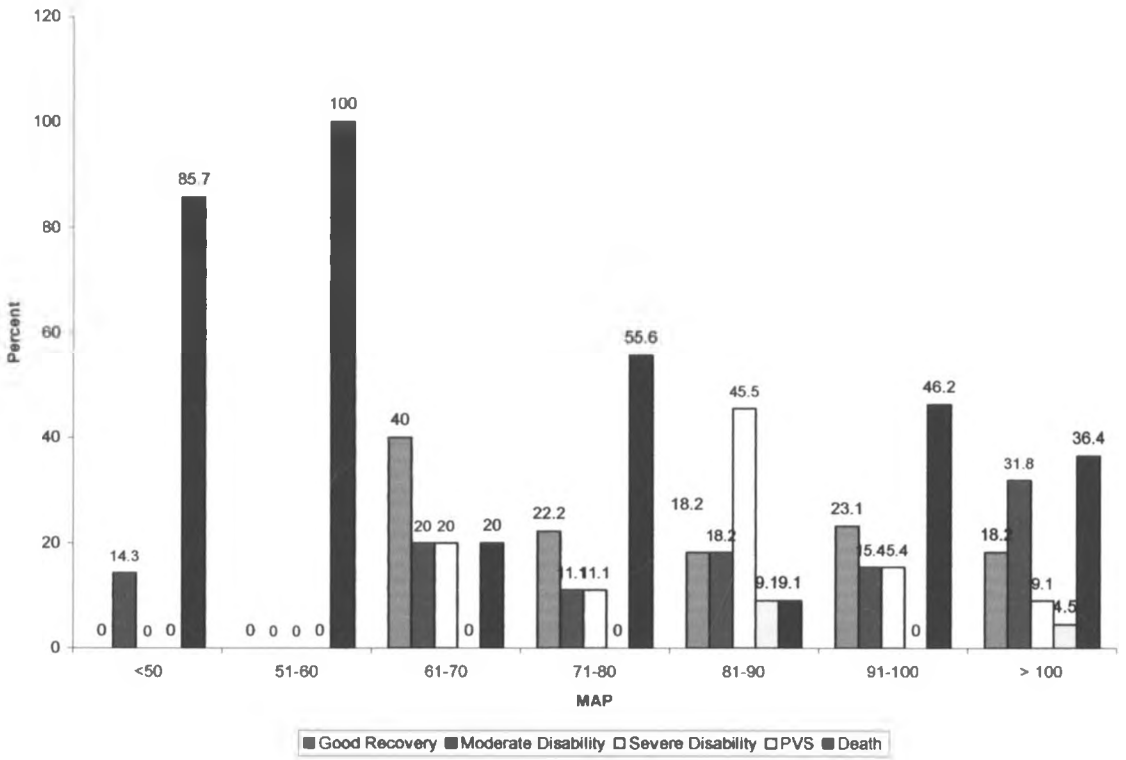
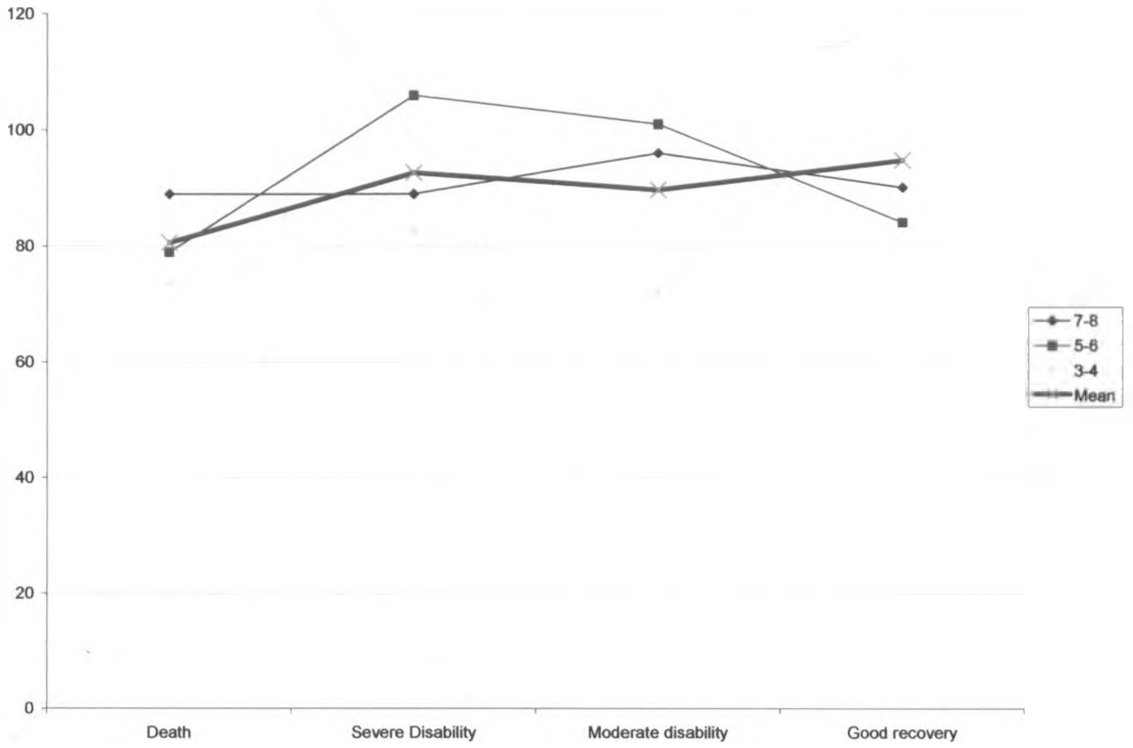


Figure 4 and table 14 depict the distribution of the patients' MAP and GOS. Eighteen of the patients who died representing 66.7% of the deaths had MAPs below 90mmHg.From figure 5, the number of patients who died tended to decrease with increasing MAP.

Using ANOVA analysis for means of MAP at admission and GOS groups at end of the follow up, MAP at admission did not have a significant association with GOS outcomes ($p = 0.156$).

4.2 ASSOCIATION BETWEEN MAP, GCS, GOS

Figure 5: Association between MAP, GCS and GOS



In this analysis, GCS groups (3-4, 5-6, 7-8), were analyzed against GOS and the means of the MAP.A subtle gradient for the mean of MAP indicated by the thick red line showed a linear relationship between MAP, GCS and GOS. This indicated that at a lower MAP, poor GCS led to poor outcome and vice versa.

5.0 DISCUSSION

Over 50% of 100,000 trauma related deaths in US are secondary to head injuries[21, 22]. The mortality rate by the Traumatic Data Bank 1984-1987 was 35% in the best of centres worldwide.

Locally, Kiboi found 63.2 percent of the deaths in 48 hours of admission. In Opondo's study, the mortality rate was 54%. In Mwangombe's study, patients with a pulse rate of less than 60 per minute at admission had a mortality of 100% but 69% for those with over 120 beats per minute.

In this study, the overall mortality was 40.9% with 74.1% of the deaths occurring in the first 48 hours and this was an increase compared with Kiboi's study. Eighteen (66.7%) of the 27 patients who died had MAPs below 90mmHg at admission. In a study by Jones *et al.* hypotensive episodes which were defined as MAP below 70mmHg were identified in 73% of the patients[28]. Chestnut *et al.* in a study on influence of early (admission) or late (ICU) hypotension on outcome, reported that hypotension (systolic BP <90mmHg) after severe TBI dramatically reduced the likelihood of favorable outcome[46].

Czosnyka *et al.* demonstrated that cerebral pressure auto regulation is significantly disturbed in the first 2 days after head injury in patients with poor outcome[4].Byron Young *et al.* also showed that the acute phase response period after severe trauma to the head lasted 21 days with the first 72 hours most intense[6].

Mean arterial pressure is an important component in calculating CPP since CPP is not easily determined by direct measurement, hence;

CPP=MAP-ICP[47].MAP has been used as a screening tool for pre-eclampsia. Leona *et al.*demonstrated that the detection rate of pre-eclampsia by log multiple of the median MAP and maternal variables was 62.5% for a false positive rate of 10%[52]. Domanski et al. carried out a study on independent prognostic information provided by sphygmomanometrically determined pulse pressure and MAP in patients with left ventricular dysfunction. Pulse pressure and MAP related positively with each other. MAP was inversely related to total and cardiovascular mortality ($p<0.0001$) while pulse pressure was shown to be an independent predictor of total and cardiovascular mortality ($p<0.02$)[54].

Avanzini F. *et al.* on pulse pressure and MAP as predictors of death after myocardial infarction showed that patients with an MAP of 80mmHg or less had a 48% risk of cardiovascular death(95% confidence interval, $p = 0.001$). Those with a pulse pressure above had a 35% higher risk (95% confidence, $p = 0.007$)[62].

Worldwide figures show that most head injury patients are less than 35 years of age. Table 3 showed the distribution of patients by MAP and age. Fifty three (80.3%) patients were from the age bracket of between 20 and 45 years. This represented the highest population in this study that also had highest number of assaults (93.7%), ISS (37.1±1.88) and use of alcohol (92%). Thirty seven (69.8%) patients from that age bracket had MAPs below 90mmHg. Extremes of age had few patients involved. There

was no statistical significance on correlating MAP and age (p=0.062). Robert *et al.,* in their study showed that pulse pressure correlated strongly with systolic pressure (p=0.001) and therefore death in the elderly, while diastolic pressure and MAP had no significant association with mortality (p=0.11)[53].

Epidemiological studies of gender differences in outcome are limited. According to traumatic data bank figures on head injury, the worldwide male to female ratio is 2:1. Mwangombe's study revealed a ratio of 4:1. Table 4 showed the distribution of patients' MAP by gender. More male patients than females (ratio 10:1) were investigated within all MAP groups with majority of the patients having MAPs below 90mmHg for both sexes in our study. In the study by Domanski *et al.*, non invasive BP measurement was found to provide two independent prognostic factors for survival. By adjusting for other modifying factors, high pulse pressure was shown in the females but no significance was shown on MAP between the sexes[54]. This contradicted findings by Jess *et al.,* who had shown in their study that gender is an independent predictor of outcome[55]. In their study, women were 1.75 times more likely to die and 1.57 times more likely to experience poor outcome than men. Our study was carried out during electioneering hence more men were involved in campaigns and exposed to injury. There was no association between MAP and gender.

Most traumatic brain injury world wide is due to road traffic accidents, falls, assaults, sports and missiles in that order. Locally, in Mwangombe's study, road traffic accidents,

assaults and falls contributed in the same order like US figures. The causation and age ranges compare well with another study locally by Kiboi as 25-35 years[23].

In this study, assaults (45.5%), Road Traffic Accidents (40.9%) and falls (9.1%) were identified in that order. This change in ranking can be attributed to the period of study which was run up to the general election and subsequent post election violence. More males were involved in the campaigns, alcohol taking and the violence that exposed them. The introduction of speed governors on our roads and use of safety belts could have reduced the RTA related accidents.

Assaults here resulted in more head injuries (45.5%) than RTAs (40.9%) and had 21(70%) compared with 17(65.4%) patients whose MAPs were below 90mmHg. No association was found between MAP and cause of injury ($p = 0.460$).

Figure 3 showed the duration between the time of injury and arrival at the hospital (Time of admission). The number of patients tended to diminish by time with most (33.3%) arriving in first 6 hours and minority (6.1%) within 24 to 72 hours. The severity of their injuries that related to low MAP could have necessitated faster transfer to hospital as evidenced with one of the patients with a very low MAP(<50mmHg) who was labelled; do not resuscitate (DNR) in spite of early arrival..

Above 60% of the admissions in all the intervals of admission (except the 24-72 hrs interval who had similar treatment) had an MAP below 90mmHg.There was no statistical significance between MAP and time from injury to hospitalization ($p = 0.494$).Manley G.

et al. studied the frequency, duration and consequences of hypotension, hypoxia on outcome in head injury. They demonstrated that hypotension, but not hypoxia, occurring in the initial phase of resuscitation is significantly ($p = 0.009$) associated with increased mortality, even when relatively short[17]. Stocchetti *et al.,* while quantifying the occurrence of arterial hypotension, and oxygen desaturation at the scene of accident, in helicopter evacuated head injury patients, found both as frequent occurrences. Hypoxemia was easily detected and managed but the contrary happened with hypotension[63].

Only one of the 4 patients (25%) admitted between 24 and 72 hours in our study had an MAP above 90mmHg while 75% were below. This could mean that these patients who had low MAPs and had no resuscitation by 24 hours died. The referrals were mainly from district and some from provincial hospitals which lack specialists, CT scan and resuscitation facilities. Eight (80 %) of the 10 patients admitted after 72 hours had MAPs below 90mmHg.These represented mainly the group of patients who had received some form of support prior to referral, therefore the longer the time to admission the greater the likelihood to capture patients with a low MAP unless some resuscitation had been done. This seems to agree with findings and recommendations by Marmarou *et al.* In their study, they recommended that monitoring be instituted not later than 18 hours post injury and to continue at least up to 60 hours from injury[3].

A study in Glasgow established that alcohol was a major associated factor in causation of severe head injury with 62% males and 27% females having detectable levels in blood (>5mg/100mls)[56].Alcohol levels were significantly higher in patients who had 'a

fall under influence', or were victims of assault than those from RTAs or other accidents. Brian *et al.,* showed that alcohol intoxicated young people with central nervous system injuries were over twice as likely to have decreased time to death (odds ratio 2.04 with 95% C.I.)[57]. Kraus *et al.*, found out in their study that, high blood alcohol levels were most frequent among brain injured subjects aged 25-44 years and among subjects from RTAs and assaults. Controlling for other predictors, ISS and hospital mortality were inversely related to alcohol levels[58].

Table 6 showed the perceived state of alcohol intoxication at the time of their injury. Fifty three (80.0%) of the patients were supposedly intoxicated with alcohol at the time they arrived at the hospital. Thirty three of the 53(62.3 %) patients who were positively associated with alcohol ingestion prior to injury had MAP below 90mmHg while 11 of the 13 non alcohol associated injuries were below 90mmHg. There was no significant relationship between MAP and alcohol consumption ($p = 0.732$).

Cerebral autoregulation is significantly affected in the first 2 days after head injury in patients with unfavorable outcome. This is particularly in areas surrounding intracranial mass lesions[4]. Most (62.1%) of our patients had identifiable lesions on CT scan.

Magnetic resonance imaging is superior to CT scan in defining diffuse axonal injury. Marshall *et al.*, published a scheme that classifies head injury based on CT scan findings[59]. The diagnosis reached at based on CT scan findings is presented in the table 7.Chi square test was carried out between MAP versus CT scan findings (diagnosis) and

no significant association was established in either case (p = 0.095).This implies that MAP did not reflect the CT scan finding or lesion post head injury hence, patients need adequate resuscitation prior to CT scan.

Table 8 showed the relationship between MAP and use of mechanical ventilation. No association was found between MAP and mechanical ventilation (p = 0.486).Bullock M.R. *et al* like other studies showed that, hypoxic ischaemic brain damage was contributory cause of death in 88% of patients thus recommending early intubation and mechanical ventilation[30,31,39]. The timing of intubation in our study was not done but a majority (62.1%) of patients were intubated and ventilated. The main reason for failure to intubate and ventilate some patients was lack of mechanical ventilators and ICU beds. McGuire *et al.*, investigated the cerebral perfusion pressure changes on varying the levels of positive end-expiratory pressure in 18 severely head injured patients. They found out that higher levels of positive end-expiratory pressure did not change intracranial pressure or cerebral perfusion pressure in the patients as the CPP remained >60mmHg (p =0.05)[60].

The most frequent operations in our study were craniotomy and surgical toilets and the two were undertaken at equal rates (Table 9). A larger population of patients (77.3%) required conservative management. Of the 51 conservatively managed patients, thirty (58.8%) had good outcome. No association existed between the two variables (p = 0.436). There is therefore need for specialized ICU to cater for severe head injury patients.

Table 10 showed the relationship of MAP to ISS. Sarrafzaddah *et al* showed that over 50% of severe head injuries have other injuries like bone, facial injuries and chest injuries. They reported that the outcome is worse when patients had other injuries in addition[44]. In the study by Trabold *et al,* in assessing the potency of Transcranial Doppler Ultrasound (TCD), a higher mean ISS (32 ± 8) was associated with poor prognosis (GOS 3-5) while a lower ISS (19±11) was associated with good prognosis (GOS 1-2, $p<0.05$)[5]. Demetrios *et al* demonstrated in their study that penetrating trauma, head ISS (24±11; $p=0.65$), hypotension at admission (systolic BP<90mmHg) and age above 55 years at admission play a critical role on outcome[8].Pearson correlation was carried out on the means of the patients' MAP and ISS in our study. There was a significant negative correlation between ISS and MAP ($r = -0.382$, $p=0.001$). This indicated that a unit increase in ISS led to a corresponding decrease in MAP therefore poor outcome with decreasing MAP.

Table 11 showed that with increasing MAP, the GCS score tended more towards the favourable range although no significant association was established between the two variables ($p = 0.319$).

Outcomes in severe traumatic head injury are best determined using GOS. GOS is a simple score to determine and is easily interpreted by many including paramedics. This score can be determined at 3 months, six months and twelve months[43].

While comparing outcomes after severe traumatic head injuries using cerebral blood flow, targeted strategy and intracranial pressure targeted strategies, Rosner *et al.*, found CPP>70mmHg was associated with comparable excellent overall results both at 3 and 6 months[61].Robertson *et al.*, on CBF strategy found no difference in outcomes at 3 and six months. In our study GOS was determined at 3 months and six months. The outcome in surviving patients at 3 months compared well with outcomes at six months.

From figure 4, outcomes tended to improve with increasing MAP but there was no association between MAP and outcomes in our study (Table 14.ANOVA p = 0.156). However with MAP analyzed against GCS and GOS on a graph (Figure 5), good outcome associated positively with increasing MAP. Figure 4 showed that the number of patients who died decreased with increasing MAP. Isabella B., *et al* studied the relationship between admission blood pressure and MAP on GOS at 6 months. A smooth U-shaped graph was obtained from the analysis between systolic BP and MAP and GOS. Both lower and higher levels of systolic BP and MAP were associated with poor outcome. Best outcomes were associated with systolic blood pressures of the order 135mmHg and MAP of 90mmHg[16].In our study, the means of MAP differed significantly among the groups of GOS.

Several studies have used GOS as the primary outcome measure (Bishara *et al.*,1992;Changaris *et al.*,1987;Choi *et al.*,1988;Choi *et al.*,1991;Lokkeberg *et al.*,1984;Pal *et al.*,1989)[7].Of the reviewed studies that used GOS as the primary outcome measure, only two reported correlational analysis between GCS and GOS in attempting to predict outcome in head injured patients.

Bishara and associates (1992) reported statistically significant relationships ($p<0.0001$) between admission GCS scores and GOS at 6 and 12 months, while Changaris *et al.*, reported significant ($p<0.001$) positive correlations between GCS scores and GOS categories.

Table 12 showed the GCS and GOS findings in this study. The GCS groups were subcategorized as 3-4 to imply poor GCS score, 5-6 as moderate and 7-8 as favourable score. The GOS groups were categorized as follows; good, moderate disability as favourable outcomes, while severe disability, persistent vegetative state and death as poor outcomes.

From table 12, it was evident that as GCS improved, the outcome within the GOS groups improved. A significant association was found between GCS and GOS ($p=0.008$), hence a lower GCS corresponded with a poorer GOS .The GCS predicted outcome fairly accurately in all groups. This was in agreement with findings by Diringer and Edwards that GCS successfully classified functional outcome in 71% of patients. In their study, a higher GCS e.g. 6-8 meant patient likely to recover fully while 3-4 meant poor outcome.

In our study, 18 patients had a GCS of 3-4 with a mortality of 12(68.4%) patients. The 22 patients who had GCS of 5-6 had a mortality of 10(43.5%) patients. Of the 26 patients whose GCS was 7-8, seven of the patients died (25% mortality).

Figure 5 indicated that at a lower MAP, poor GCS led to poor outcome and vice versa. It shows a linear relationship between mean of MAP, GCS and GOS. The lower the mean of MAP, the likelihood of lower GCS and poor GOS is eminent. Therefore we can deduct that the lower the MAP (with critical level of 90mmHg) the poor the GOS score.

5.2 CONCLUSION

- Majority (66.7%) of our patients presented with a low MAP (<90mmHg).Only a few patients (10.6%) with MAP below 50mmHg reached the hospital with (6/7) 85.7% mortality. An MAP of ≤ 50mmHg should signal poor outcome while MAP ≥ 90mmHg signals good outcome (mean MAP for survivors 93 ± 3.01 but 79 ± 2.93mmHg for the dead).

- Up to 80% of the patients with majority in 20-45 year bracket were associated with alcohol prior to injury. This age group also recorded the highest number of assaults (93.3% of total assaults), and a high mean ISS (37.1 ± 1.88) unlike other groups. The 20-45 year age group has the highest risk for severe head injury.

- Not all patients in our hospital requiring intubation and ventilation receive it. All patients in this study qualified for it but only 41(62.1%) patients benefited. This could have an effect on outcome.

- Most (77.3%) patients were conservatively managed with most (8/15 or 53.3%) of patients who needed surgery having MAP <90mmHg. Therefore MAP <90mmHg in our hospital is significant.

- The late arrival to hospital after 72 hours post injury represented referrals from other hospitals. Eight of the 10 patients (80%) had MAP below 90mmHg.There is a very high possibility that patients arriving after 72 hours are likely to have poor

outcome. The longer the time to admission, the more the likelihood to capture patients with a low MAP, unless some adequate resuscitation had been done.

- There was no statistical significant association between MAP and age, gender, use of mechanical ventilation, time to admission, CT scan findings and surgical intervention probably because the sample size was small. Improving GCS and GOS associated positively with increasing MAP although no threshold effect was observed. Admission GCS and ISS were better predictors of outcome than MAP.

- Mean arterial pressure determination in our severe head injury patients is useful when used in combination with ISS and GCS. The mortality in this study having been 40.9% is still far above the 35% by traumatic data bank and therefore need for better monitoring and intervention.

5.3 RECOMMENDATIONS

- That KNH needs to carry out a study on levels of alcohol in our head trauma patients and the association with outcome. The government needs to help hospitals to acquire alcohol level determining facilities, to identify the patient's level of intoxication. The government should institute regulation of alcohol consumption to protect the 20- 45 year age group which is the most affected yet the most productive.
- A study be done by KNH to evaluate the effect of delayed and failure to ventilate on outcome. There is need for more ventilators to be acquired to accommodate the high number of head injuries. KNH should institute the mandatory requirement that all patients with GCS <8 need prompt intubation and ventilation. The government should provide more ambulances to improve on their speedy transfer since only 33.3% of patients arrived in 6 hours.
- There is need for the government to introduce telemedicine and CT scan machines in referral origin centres, as this would facilitate consultation between doctors, and improve on outcomes by optimizing early care before transfer to specialized centres.
- That MAPs below 90mmHg threshold should signal need for intervention and ISS should be determined alongside GCS and MAP in severe traumatic brain injury.
- A study to establish the effectiveness of phenytoin in seizure prophylaxis should be carried out since breakthrough convulsions were reported in six (10%) patients in spite of treatment.

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APPENDIX I A

CONSENT FOR PARTICIPATION IN THE STUDY

Consent explanation

I am Dr. Ondede Kennedy, a postgraduate student at University of Nairobi. I am doing a study on the role of mean arterial pressure in the monitoring of severe head injury.

Intracranial pressure is important in head injured patients as it has a big role in the outcome of the patients. I will not influence the management of the patient but would like to monitor the mean arterial pressure, and the progress of the patient.

I will ask you a few questions and also examine the patient. I will subsequently follow up the patient through the hospital process. I will visit the patient at 72 hours then end of 1st, 3rd and 6th months. I will examine the patient at every visit and record my findings. All information will be treated with strict confidentiality. The results of this study will be useful in care of patients in future. Participation is completely voluntary and you are free to withdraw your patient from the study at any point and that would not affect treatment in any way.

Consent

I..... has been explained to the purpose and conditions of my patient's involvement in this study. I agree to the above and give consent on his/her behalf to be included in the study or for a minor I agree to the above and give consent for. ..... Registration number..... to be included in the study.

Name of guardian/Consultant .....Witness.....

Sign.....Date.....Sign.....Date.....

APPENDIX 1B

IDHINI YA KUSHIRIKI KATIKA UTAFITI

MAELEZO

Mimi ni Daktari Ondede Kennedy, mwanafunzi wa shahada ya pili katika chuo kikuu cha Nairobi. Ninafanya utafiti kwa wagonjwa walio jeruhiwa vichwani.

Nguvu za damu ni muhimu kwa walio jeruhiwa vichwani na matokea ya tiba hutegemea jinsi tiba hii imetekelezwa.Sita shawishi tiba ya mgonjwa bali nitachunguza atakavio tibiwa na matokeo yake. Matokeo ya utafiti huu yatasaidia katika matibabu ya baadaye ya wagonjwa.

Nitakuuliza maswali machache kisha nimupime.Nitaendelea kumutembelea hospitalini, kisha mwisho wa mwezi wa kwanza, wa tatu na wa sita.Nitampima kila ninapomtembelea na kunukuu nitakayo yapata. Habari yoyote nitakayo pata itakuwa siri.Kushiriki kwenu katika utafiti huu ni kwa hiari yenu, na uko huru kumwondoa wakati wowote bila kudhuru utaratibu wa kimatibabu kwa vyovyote vile.

IDHINI

Mimi..... nimeelezewa

Kiini na mashariti ya kushiriki kwa mgonjwa wangu kwenye utafiti huu, na nimemruhusu daktari kufanya utafiti huu kwake ama Kwa mtoto wangu.....

mwenye nambari ya usajili.....

Jina.....

Sahihi.....Tarehe.....

Sahihi Kwa mtoto chini ya miaka 18 .....Tarehe.....

Shahidi.....Sahihi.....Tarehe.....

APPENDIX 11

DATA SHEET

1. Patient's personal details

- Initials ☐
- Age 1. <13 yrs ☐
  - 2. 12-20 yrs ☐
  - 3. 20-45 yrs ☐
  - 4. 45-55 yrs ☐
  - 5. 55-60 yrs ☐
  - 6. >60 yrs ☐
- Sex Male ☐
  - Female ☐
- Time from injury 1. 6 hrs ☐
  - 2. 6-12 hrs ☐
  - 3.12-24 hrs ☐
  - 4. 24-72 hrs ☐
  - 5. >72 hrs ☐
- Cause of injury 1. RTA ☐
  - 2. Fall ☐
  - 3. Assault ☐
  - 4. Missile ☐
  - 5. Sports ☐
  - 6. Others ☐

- Alcohol intoxication Yes ☐

  No ☐

- Date of admission ☐
- Date of discharge ☐
- Death ☐

2. Diagnosis

   ☐ Concussion

   ☐ Contusion

   ☐ Laceration

   ☐ Haematoma

   ☐ Diffuse axonal injury

   ☐ Other injuries

   ☐ Premorbid conditions

3. Surgical intervention. ☐

4 Date of operation ☐

5 Use of mechanical ventilation ☐

6 Type of operation

   ☐ Burr holes

   ☐ Craniotomy

   ☐ Surgical toilet

   ☐ Abdominal surgery

☐ Orthopedic surgery

☐ Others

Blood loss.....mls

Total input.....mls

Total output.....mls

Balance +ve.....-ve.....

7 Length of surgery in. .....hours.....minutes.

8 Complications post operatively

9 Overall patient assesment

VARIABLES OF ASSESSMENT	DURATION POST TRAUMA(Hours0,72.end of 1,2,3 & 6 months					
	0 hrs/At admission	72 hours	1 month	2 month	3 month	6 month
GLASGOW COMA SCALE						
E V M						
VITAL SIGNS						
-RESPIRATORY RATE						
-PULSE RATE						
-TEMPERATURE						
-BLOOD PRESSURE-						
SYSTOLIC BP MMHG						
1. >160						
2. 140-160						
3. 120-140						
4. 100-120						
5. 90-100						
6. 60-90						
7. <60						
-DIASTOLIC BP MMHG						
1. >140						
2. 120-140						
3. 100-120						
4. 90-100						

5. 80-90

6. 60-80

7. <60

MAP=DIASTOLIC BP

+1/3 PULSE

PRESSURE

GLASGOW OUTCOME SCALE

1.GOOD RECOVERY 2.MODERATE DISABILITY 3.SEVERE DISABILITY 4.PVS

5.DEATH

INJURY SEVERITY SCORE CALCULATION IN MULTIPLE INJURIES

REGION	Injury	AIS	**AIS^2**
HEAD/NECK			
FACE			
CHEST			
ABDOMEN			
EXTREMITY			
EXTERNAL			
TOTAL INJURY SEVERITY SCORE(ISS)			

10. MEAN SATURATION OF OXYGEN.....CARBON DIOXIDE.....

11. DRUGS ADMINISTERED

- MANNITOL
- LOOP DIURETICS

☐ FRUSEMIDE

☐ ETHACRYNIC ACID

☐ ACETAZOLAMIDE

- OTHERS;

☐ STEROIDS,

12. AT END OF 3 MONTHS OR DISCHARGE PRIOR TO EXPIRY OF 6 MONTHS

HOSPITAL STAY IN DAYS .....

MEAN ARTERIAL PRESSURE.....

GLASGOW COMA SCALE .....

GLASGOW OUTCOME SCALE.....

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Email: KNHplan@Ken.Healthnet.org

Ref: KNH-ERC/ 01/ 4962

22nd November 2007

Dr. Ondede K.
Dept. of Surgery
School of Medicine
University of Nairobi

Dear Dr. Ondede

RESEARCH PROPOSAL: "ROLE OF MEAN ARTERIAL PRESSURE IN MONITORING SEVERE HEAD INJURY PATIENTS AT A TERTIARY HOSPITAL." (P122/5/2007)

This is to inform you that the Kenyatta National Hospital Ethics and Research Committee has reviewed and **approved** your revised research proposal for the period 22nd November 2007 – 21st November 2008.

You will be required to request for a renewal of the approval if you intend to continue with the study beyond the deadline given. Clearance for export of biological specimen must also be obtained from KNH-ERC for each batch.

On behalf of the Committee, I wish you fruitful research and look forward to receiving a summary of the research findings upon completion of the study.

This information will form part of database that will be consulted in future when processing related research study so as to minimize chances of study duplication.

Yours sincerely

PROF A N GUANTAI
SECRETARY, KNH-ERC

c.c. Prof. K.M. Bhatt, Chairperson, KNH-ERC
The Deputy Director CS, KNH
The Dean, School of Medicine, UON
The Chairman, Dept. of Surgery, UON
Supervisor: Mr. Julius G. Kiboi, Dept. of Surgery, UON
Mr. Khainga S.O. Dept. of Surgery, UON