# THE IMMEDIATE MANAGEMENT OF HEAD INJURIES
## A CLINICAL EXPERIENCE
## AT MUHIMBILI MEDICAL CENTRE

VICTOR VERCKYS GEORGE MWAFONGO (M.D.)

A DISSERTATION SUBMITTED IN PARTIAL
FULFILLMENT OF REQUIREMENT FOR
THE AWARD OF THE DEGREE OF MASTER
OF MEDICINE IN ANAESTHESIOLOGY
1984



The undersigned hereby declares that this dissertation has not been submitted for a degree in any other University.

DR. VICTOR V.G. MWAFONGO

Dr. V.V.G. Mwafongo Signature

Dr. N.J.A.M. Matekere Signature

Medical Stores Department
P. O. Box 3099
Dar es Salaam

ALL RIGHTS RESERVED. NO PART OF THIS DISSERTATION MAY BE REPRODUCED, STORED IN ANY RETRIEVAL SYSTEM, OR TRANSMITTED IN ANY FORM OR BY ANY MEANS ELECTRONIC, MECHANICAL, PHOTOCOPYING, RECORDING, OR OTHERWISE, WITHOUT THE PRIOR WRITTEN PERMISSION OF THE AUTHOR OR THE UNIVERSITY IN THAT BEHALF.

CONTENTS:


| | | |
|---|---|---|
| 1. | Acknowledgement | I |
| 2. | List of Abbreviations | II |
| 3. | Summary | III |
| 4. | Introduction and literature Survey | 1 |
| 5. | Patients and methods | 38 |
| 6. | Results | 40 |
| 7. | Discussion | 59 |
| 8. | Conclusion | 68 |
| 9. | References | 69 |

ACKNOWLEGEMENTS

I dedicate this dissertation to all members of the Department of Anaesthesiology.

I am most grateful to the Acting Head of the Department of Anaesthesiology Dr. N.J. Matekere, to Dr. Lweno and Dr. Egan of K.C.M.C. Hospital, for their tireless efforts in reading and correcting the manuscripts. I sincerely thank the Neurosurgeon Prof. Wu who helped me in the management of the patients.

Lastly I extend my gratitude to Miss Jesca Muhando who typed the final draft.

List of Abbreviations

CAT - Computerized Axial Tomography

ICP - Intracranial pressure

ICU - Intensive Care Unit

IPPV - Intermittent positive pressure Ventilation

## SUMMARY

Head injuries constitute a special and particularly tragic problem both in developing and developed countries: A number of lives are lost due to head injuries in all age groups commonest being below 40 years. The incidence being higher in the developed countries.

A survey was made to know what immediate treatment was given to patients with head injuries in Dar es Salaam in the whole chain from the accident site, on the way to Emergency Department at Muhimbili Medical Centre to the Wards, X-ray Department, ICU and Theatre.

There were 220 patients of head injuries admitted from 1st May 1982 to 30th May 1983. There were 45 deaths, of these 20 patients died or declared dead at the Emergency Department and 25 patients died after staying in the hospital for an average of 72 hours.

The commonest cause according to postmortem findings was due to intracranial haemorrhage which constituted 14 deaths, followed by subdural haemorrhage 11 patients.

The motor vehicle was the prime cause of head injury which constituted about 44.5%, the next commonest cause of fatal head injury being assault which recorded 16.3%.

A number of shortfalls were noted, these include absence of resuscitation to the injured at the site and on the way to the hospital. Inadequate resuscitation at the emergency department. Poor ambulance services, with uncoordinated communications between the accident site and the hospital. Less aggressive management of head injuries due to lack or frequent shortage of essential drugs and important monitoring equipments, for example equipment for intracranial pressure monitoring.

Among the suggested methods of improving the services include emphasy on resuscitation at the site of accident and on the way, decentralisation of ambulance services with adequate equipment for resuscitation, introduction of Radio call to the hospital and finally imparting the knowledge on the art of cardiopulmonary cerebral resuscitation to ambulance services personnel and the hospital.

# THE IMMEDIATE MANAGEMENT OF HEAD INJURIES

## Literature Survey

### Introduction:

Anaesthetists are being increasingly called upon to institute or advise on the management for various life threatening conditions in Casualty departments and elsewhere. Trauma is responsible for more deaths in all age groups under the age of 45 years than any single cause, and head injury is the most important factor in deaths due to trauma (Horton, 1975).

It is estimated that 100,000 patients with head injuries are admitted to hospital in Great Britain each year (Rowbothon, 1964). Of these some 20% are so serious that they require the attention of the anaesthetist. Horton, (1976), reported that in England and Wales there were 5116 deaths attributed to head injury; this figure comprising 16% of all fatalities in males between the ages of 15 and 20 years.

In the U.S.A. 2,000 people per 100,000 attend accident and emergency units every year due to head injury (Jennet and Macmillan, 1981).

In Tanzania Lweno (1979), found that among the deaths occuring in Muhimbili Hospital post trauma wards 75 - 90% were due to head injury. In 1975 there were 97 deaths, 85% due to head injury, in 1976 51 deaths 75% due to head injury, in 1977, 37 deaths, 90% due to head injury and finally in 1978 69 deaths, 88% due to head injury. Therefore head injuries constitute a special and particularly tragic problem and this is so in both developing and developed countries.

Assisting in the early management of these injuries constitutes one of the anaesthetist's most important duties (Hart, 1965).

Most patients with head injuries are treated initially by persons who are not specialists and who are relatively inexperienced in the management of neuro-surgical problems. Those who survive the initial period following injury may or may not receive skilled care later depending upon the availability of Specialist neurosurgeon. However, it is not generally recognized that the ultimate mortality and morbidity are highly dependent on the quality of the initial care. So anaesthetists who have to deal with head injuries should understand some of the pathological and physiological changes which take place and also the relationship between the respiratory system and the injured brain (Horton 1975 Before discussing that it would be of an advantage to understand the types of head injuries.

Types of head injuries

In brief, according to the mechanism, either the scalp; the brain; the skull; or intracerebral vessels may be injured separately or together (Rains et al., 1975). Motor vehicle accidents and falls account for the majority of the head injuries and their prevention is an international problem (Hart, 1965).

Some blows to the head fracture the skull; others may damage the brain without skull fracture. Assault is common in the African Society (Mwang'ombe, 1982). A skull fracture may occur without any effect on the

brain, when a blow produces a rotatory effect, there is a shearing acti upon blood vessels, not only in the coverings of the brain but at points of different consistency of tissue in the brain substance (Robbi 1974). From whatever cause the patient may have either:-

(i) Concussion

A concussion may be defined as the loss of consciousness and there is a brief temporary physiological paralysis of function without organic structural damage. This is usually followed by complete recovery.

(ii) Contusion

In cerebral contusion the stretching of tissue is severe. The architecture of the tissue is preserved but there is perivascular bleeding. There is rupture of fibres producing lesions at various levels of maximum stress. Unconsciousness is more prolonged, the initial recovery is slow or incomplete or death may result.

(iii) Laceration

A laceration is defined as traumatic lesion in which there is interruption of continuity of tissue.

(iv) Countre-coup lesion

A Countre-coup lesion is a lesion occuring opposite to the position of the blow. These lesions are common in the tips of the frontal and temporal lobes in falls on the back of the

head, and in the extremities of occipital lobes in falls on the front of the head the effect may be laceration of brain substance rather than contusion.

In some shearing lesions of the brain, intracerebral haemorrhage is produced. When laceration occurs, there is inevitable flooding of the subarachnoid space by blood, (Robbins, 1974; Rains et al; 1975).

Pathophysiology of Head Injuries

Following a severe head injury there is a widespread neuronal structural damage and widespread ischemia (Graham and Adams 1971).

This ischemic change will lead to metabolic acidosis due to an increased formation of lactic acid. The cerebral metabolic acidosis will lead to a fall in the pH of the cerebrospinal fluid (CSF), vasodilatation and increased cerebral blood flow (CBF) which modifies the formula below.

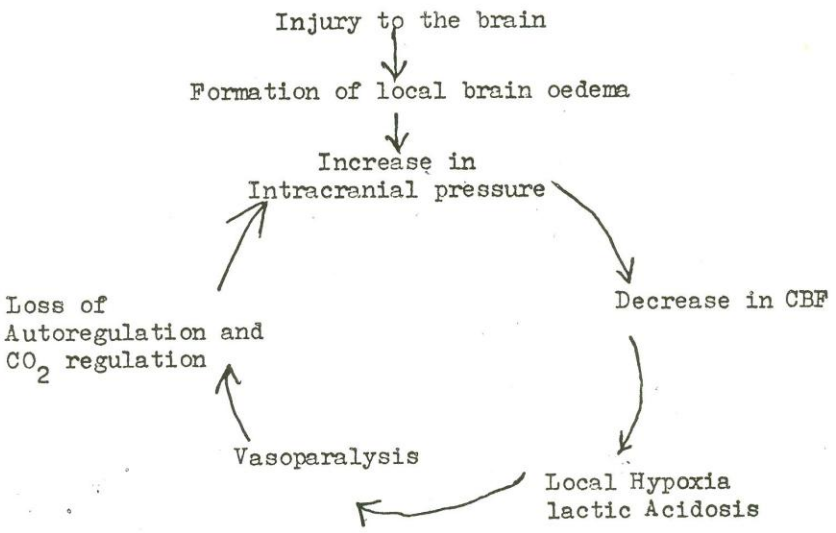
$$CBF = \frac{\text{Meansystolic arterial pressure - Mean intracranial pressure}}{\text{Cerebral Vascular resistance}}$$

(Horton, 1975; Horton, 1976)

OR

Accumulation of oedema fluid increases the tissue pressure and diminishes the regional blood flow. The reduction in perfusion of the oedematous areas may result in a critical hypoxia of the tissue and an increase in tissue and CSF, lactate. Tissue acidosis may impair local regulation of CBF and induce a state of vasoparalysis with loss of autoregulation and carbondioxide regulation (Reulen, 1978).

Diagramatically it can be presented as shown below:




Schematic representation of the pathophysiological sequence of events in brain oedema: (Reulen, Hadjidimos and Hase, 1973).

If the ICP rises to the level of systemic arterial pressure, cerebral blood flow ceases and cerebral death will take place if the process is not reversed or arrested. (Horton 1976, Fieschi et al, 1974).

Baseline assessment of head injuries

The purposes of the acute neurologic exam-ination in the head injured patient can be categorized as follows:

1. to permit the clinician to estimate the degree and location of cerebral injury.
2. to establish a baseline neurologic state to which all future examinations will be referred in order to identify improvement or deterioration.
3. to establish the presence or absence of mass lesion that might require surgical intervention and
4. to allow some estimate of prognosis for recovery to be made at the earliest possible time. This examination is intimately associated with the general systemic examination for evidence of other injuries' (Bruce, 1980).

In the management of acute head injury nurses are encouraged to write a succinct but detailed account of their observations, but this depends on a largely subjective interpretation of the responses elicited and results in inconsistencies.

Teasdale and Jennet (1974) produced a simple scoring system in which responsiveness is assessed as three types of behaviour motor responsiveness, verbal performance, and eye opening. Each of these main types of behaviour is subdivided into different levels of response. The observations made are plotted on a chart (see attached paper Fig. 2) of which the vertical axis consists of the

ASSESSMENT OF HEAD INJURIES

Fig:2

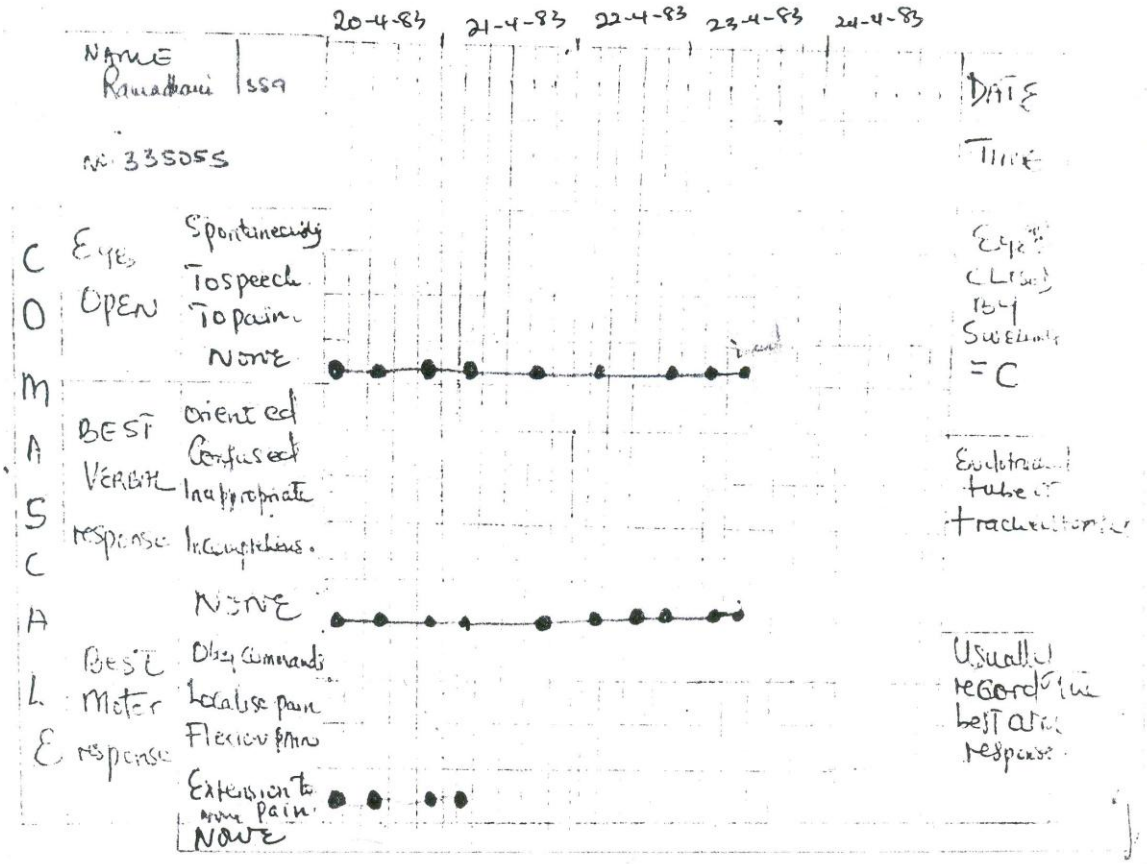



Chart illustrating used Glasgow Coma Scale.

data observed while the horizontal axis carries a suitable time scale (McDowall, 1976). Plotting the observed responses results in three lines which move upward with clinical improvement and downward with deterioration. In the scale the following can be incorporated, the recording of pulse rate, blood pressure, temperature, respiratory rate, and the pupil size.

The anaesthetist should also be aware of, and able to recognize, the signs of a rising intracranial pressure which are:

Deterioration in the level of consciousness.

Progressive slowing of the pulse rate.

A rising systolic blood pressure.

Increasing pulse pressure

A change in the pattern of respirations, which may become slow, irregular or periodic.

Dilating non reacting pupils which may be unequal (Horton, 1975).

Measurement of Intracranial pressure after head injury

The two sites commonly used for ICP measurements are the Ventricular System and the Extradural Space (Turner et al 1976).

Ventricular System:

The measurement of ICP is traditionally made from one or other lateral ventricle by the insertion of a catheter into the ventricle, either through a burr hole or through a twist drill hole in the skull.

The extradural Space:

Measurement of ICP from the extradural space is often technically easier to institute as it requires only the fixation of an appropriate device in a burr hole. The same good-quality recordings can be obtained from the extradural route as from the ventricular route, but, to achieve this, care must be taken to prevent any leakage of fluid from the site of the extradural pressure - measuring device.

Measurement of ICP is of value in the management of the acute stage of severe head injury because it gives early warning of oedema or intracranial bleeding Mcdowall, (1976). It cannot distinguish between the two, but it is possible by computer assisted tomography. It gives some light in the success or failure of regimens to decrease pressure (Teasdale, 1976).

The other major contribution of ICP monitoring is in the management of cerebral oedema, by such techniques as hyperventilation, dehydrating therapy, and steroids.

The last advantage is in the general care of the patient. ICP monitoring helps in determining the best pattern of mechanical ventilation, particularly when positive end expiratory pressure is employed or when careless handling can produce sudden increases in blood pressure and ICP (Mcdowall, 1976).

## Management of Head Injuries

The management of head injuries falls into four main categories.

1. Maintenance of patent airway
2. Correction of hypovolemia from associated injuries.
3. Reduction of brain swelling (Oedema) and
4. Finally evacuation of intracranial haemorrhage.

Before considering each category in turn it is better first to understand the concept of primary and secondary head injury.

The concept of primary and secondary head injury is important to those involved in the care of patients who have sustained head trauma (Bruce, 1980). The primary head injury results from the biomechanical effect of the forces applied to the skull and brain at the time of the traumatic insult and is manifested within milliseconds of the injury. A biomechanical change in its mildest form is represented as concussion, a transient but reversible loss of consciousness associated with pupillary dilatation, loss of corneal responses, bradycardia and bradypnoea (Bruce, 1980). In its most severe form,

physical disruption of the brain stem at the ponto medullary junction occurs with sudden death.

If the patient survives the initial head injury long enough to reach the hospital, this implies that the biomechanical injury has not been sufficiently severe to cause death.

If death occurs later definitely this must be due to a second series of events that have been triggered by the initial trauma but then has progressed from the time of injury to cause secondary damage and thus death (Bruce, 1980).

The secondary head injury can be avoided by medical care, and thus death can be prevented. But at present time, there is no treatment for the primary biomechanical brain damage and all therapy is directed to preventing secondary injury due to hypoxia, hypercarbia, hypotension or increased intracranial pressure (ICP) (Bruce, 1980). It is obvious that to be maximally effective, therapy to prevent secondary head injury should start immediately at the site of the accident.

## PRIORITIES

I. FIRST AID:

(a) The airway: Emergency therapy is begun at the site of the accident, if possible, in the ambulance, or certainly immediately upon admission to the emergency room. The commonest cause of deterioration after head injury is obstruction of the airway and consequent hypoxia, and this is both preventable and remediable (Horton, 1975). The injured patient is at risk from even partial airway

obstruction, either due to the tongue falling back or from aspiration of vomitus or of blood and secretions associated with oral pharyngeal injuries (including fractured base of the skull). The necessity for very urgent action to assure a clear airway and adequate respiration has been previously emphasized by several authors (Potter, 1965; Hart, 1965; Lewin, 1976; Horton 1976; Crockard 1981). The first priority in the management of head injury is the establishment and maintenance of a clear airway from the time that the patient is seen; as mentioned above which may be at the site of accident, (roadside), or in the Casualty department. As the first measure the patient should be turned to prone position and the mouth cleared by suction of either vomitus or blood.

(ii) Adequacy of ventilation:

If the patient is not breathing spontaneously and artificial ventilation is necessary, great care must be used not to hyperextend the neck, since cervical injury may also be present. Ten percent of patients with head injury have an associated cervical spine injury (Bruce, 1980). Failure to diagnose a cervical spine fracture may lead to serious medical and legal complications particularly if subsequent neurologic deficit develops or progresses. Head injured

patients should be considered to have cervical spine injury until proven otherwise. Tenderness or crepitus of the spinous processes of the cervical vertebrae may point to the diagnosis. Hyperextension of the neck during laryngoscopy and endotracheal intubation may displace an unstable fracture or dislocation leading to spinal cord compression. All diagnostic and therapeutic manouvres must be done extremely cautiously until the stability of the cervical spine is established." (Bruce, 1980).

Next in order is to empty the stomach of food, fluid, blood and air. A nasogastric tube should be passed, normally patients with head injury have a tendency of dilating the stomach with increased secretions and hence increased danger of aspiration (Hart, 1965).

(iii) Controlled hyperventilation

Lowering arterial $PCO_2$ by passive hyperventilation produces cerebral vasoconstriction, increasing cerebral vascular resistance and leading to a marked reduction in Cerebral Blood Flow (CBF) and in Cerebral Blood Volume (CBV). (Utting, 1980; Rottenberg et al 1980). Hyperventilation tends to increase arterial $PO_2$ and, thus, potentially improves cerebral oxygenation. Unfortunately, however, controlled hyperventilation is frequently ineffective in lowering ICP, and when effective is often only transiently so (Rottenberg, 1980).

Literature has shown that most unconscious patients with head injuries should be put on controlled or assisted ventilation (Gray 1980, Miller et al 1981).

There are several factors which support the contention that controlled hyperventilation is not only effective but also necessary treatment in patients with severe brain lesions (Gordon, 1975). It relieves the patient from a sometimes extremely exhausting and energy consuming muscular overwork. These seriously ill patients sooner or later progressively deteriorate with muscular fatigue and ventilation and perfusion inequality leading to hypoxemia and hypercarbia.

The extra work of breathing also puts a heavy burden on the heart which can lead to severe myocardial insufficiency and ultimately a cardiac standstill. Literature has also shown that marked elevation of intracranial pressure lead to significant degrees of left heart strain and pulmonary oedema (Ducker 1968; Beckman and Bean, 1969, 1970 Matjasko, 1980). Maire and Patton, 1956 postulate the existence of a hypothalamic edemagenic centre whose impulses arise from post chiasmatic hypothalamic structures. This oedemagenic centre is normally inhibited by structures in the preoptic area; destructive preoptic lesions release this inhibition, leading to pulmonary oedema. Brown (1956), suggest that neurogenic pulmonary oedema is mediated largely through the sympathetic system, specifically the alpha adrenergic system, and that sympathetic system response is centrally mediated because it is attenuated by central nervous system depressants. The immediate cause of neurogenic pulmonary oedema is a sudden increase in pulmonary capillary pressure and eventual capillary membrane disruption (Luisada 1967).

Some experts of controlled ventilation recommend that it should begin very soon after injury, which may be before a proper assessment of the severity of the brain damage has been possible sometimes very trivial. It is difficult in these circumstances to be sure that all patients receiving such treatment either needed it in the first place (Gordon, 1975).

Although controlled ventilation procludes the use of valuable signs in the assessment of the patient, there is little difficulty in deciding to use it when is obvious. Estimations of the venous and arterial blood gases are of considerable value in supplying this information (Hart, 1965).

In cases where blood gas analysis shows arterial hypoxaemia, the additional oxygen is an absolute necessity.

(iv) Tracheostomy

Although the importance of free airways and adequate ventilation in brain lesions was well known among neurosurgeons, there is hardly any worker on the literature published before 1950 who advocated the use of tracheostomy in head injured patients. In 1950 Bryce-Smith advocated the use of tracheostomy in the management of the patient with severe head injury (Bryce, Smith 1950). He stated that an endotracheal tube should be retained for no longer than 24 hours. Similar recommendations followed by Uliu and Rosomoff (1953), Maciver et al; (1958), Huang et al; (1963) and Rossanda et al; (1966) as they showed that this was the only way of

ensuring an absolutely clear airway for patients who remained unconscious during several days.

Although endotracheal tubes have been retained without harm for longer periods, 24 hours appear to be a reasonable acceptable upper limit when dealing with head injuries, Hart, (1965). By this time the patient's condition may have improved sufficiently to give rise to doubts above the necessity of doing a tracheostomy. Certainly a planned tracheostomy with an endotracheal tube in position is a much safer procedure than an emergency tracheostomy. Once a satisfactory airway has been established the tracheo bronchial suction and toilet may be instituted, to clear blood, mucus, vomitus or other debris (Gray et al; 1980).

II. SHOCK

This is usually not associated with head injuries. It usually occurs when other structures of the body have also been damaged for example lacerated spleen or liver, although laceration of the scalp can produce considerable loss of blood leading to hypotension and ultimately shock (Illingworth and Jennet, 1965).

The signs of hypovolemia should be sought and an assessment made of the degree of shock. The signs are pallor; tachycardia; hypotension; pale, cold, and sometimes cynosed extremeties; low central pressure; and oliguria. Hypovolemia may also cause confusion, restlessness, air hunger and coma. These

signs result from a loss of over 40 percent of blood volume in a previously healthy patient (Raj. Prithvi, P., Montgomery S.J., Bradley, V.H. 1976). The same signs may appear with smaller blood losses in geriatric or previously anaemic patients, in those who have received narcotics and tranquillizers or who have been badly transported. Normovolemic patients may simulate the signs of shock as a result of injury to the spinal cord, Cardiac tamponade or pneumothorax (Raj Prithvi, P. et al 1976).

Prolonged hypotension will almost certainly reduce the chances of survival of already devitalized brain, it may also lead to renal failure. (Illingworth and Jennet, 1965).

(ii) Pathophysiology of Haemorrhagic Shock

In brief untreated haemorrhage causes a low circulating volume which reduces the cardiac output. Mechanisms are set in motion to counteract this state. The venous system which normally accommodates 60 to 70 percent of the blood volume constricts in response to increased sympathetic tone, thus reducing the size of the vascular bed and compensating for losses of 10 percent of blood volume. This reflex compensation is mediated by low pressure receptors in the large veins (Prithvi Raj. et al 1976; Lillekei et al 1972).

When the blood loss exceeds 10 percent of the blood volume, baroreceptors in the carotid sinus and aortic arch are stimulated to provoke a further increase in sympathetic tone to discharge noradrenaline from the sympathetic nerve endings, and to cause

constriction. This is more marked in the circulation of the skin, splanchnic areas and kidneys but minimal in the circulation of the brain and myocardium. However after a loss of 20 to 30 percent of the blood volume, the blood pressure begins to fall. Venous pressure and stroke volume fall. Renal blood flow decreases further, urine output ceases, and tubular necrosis may occur. Coronary blood flow is reduced. There is a reflex hyperventilation stimulated by hypoxia and metabolic acidosis (Ganong, 1979). The physiologic dead space increases. The $PaCO_2$ may fall, aggravating the cerebral ischemia (Prithvi Raj. et al 1976).

(iii) Management

In the injured patient with shock, volume replacement is begun rapidly with 2 to 3 litres of balanced electrolyte solution through 2 or preferably 3 large I.V. Catheters. After the first 2 to 3 litres of crystalloids, at least $\frac{1}{4}$ to $\frac{1}{2}$ of the additional fluid given is colloid such as plasma or albumin (Wilson, 1979). Blood is used rather liberally in attempt to keep the haemoglobin at 12.5 to 14gm percent.

If the blood pressure in a recently injured patient does not return to normal following the rapid administration of 2 to 3 litres of a balanced electrolyte solution in 20 to 30 minutes the patient is losing blood and/or fluid quickly, so active haemorrhage is controlled by direct pressure to a wound or surgical exploration. Elevation of the foot of the bed is a quick and effective means of raising the blood

pressure and is a useful emergency measure (Ellis 1976, Wilson, 1979).

III. REDUCTION OF BRAIN OEDEMA

(i) The pathological nature of Cerebral Oedema

The simplest definition of cerebral oedema is an increase in volume of brain as a result of an increase in brain water content. The water content of the normal brain is 80% of the wet weight of the grey matter and 68 percent of the wet weight of the white matter. Typical values for the oedemat ous brain would be 82 percent for the grey matter and 77 percent for the white matter (Pappius and Gulati, 1963; Fishman, 1975).

In oedema due to head injury, the essential event is injury to cerebral vessels, resulting in an increased cerebrovascular permeability allowing escape of serum constituents into the surrounding extracellular space under the hydrostatic pressure of the systemic circulation (Reulen, 1978).

The extravasation of this fluid, whose composition is similar to plasma (Bakay and Haque, 1964) is confined to the area of the lesion (Bakay and Haque, 1964; Long 1970). Blood vessels outside the area of the lesion but within the oedema territory do not show increased permeability (Long, 1970; Bakay and Haque, 1964).

Disturbances of the blood-brain-barrier resulting in extravascular leakage of serum proteins and oedema fluid must be the result of either of physical destruction of the endothelium or of opening of endotheleal tight junctions (Hirano et al, 1970). The driving force for the extravasation of fluid seems to be the capillary transmural pressure gradient, that is the difference between the intravascular and the interstitial fluid pressure (Reulen, 1978).

In the majority of serious severe head injuries oedema plays a vital part in the associated increase in intracranial pressure. This usually develops slowly, with peak around 48 - 72 hours and regress slowly up to the 7th day (Northfield, 1973; Reulen, 1978). It may cause further deterioration of the patient with Uncal herniation which is manifested by deepening coma widely dilated pupils not reacting to light and finally respiratory arrest (Hunter, 1975).

(ii) Treatment and prevention

(a) The use of Hypertonic Solutions:

The most widely used agent is mannitol in a 20 percent solution. This has supplanted hypertonic glucose and urea in clinical use. The principle of all of these agents is the same. By abruptly increasing serum osmolarity water is drawn from the brain into the blood stream resulting in reduction of brain water content, brain tissue volume and ICP (Miller, 1981, Horton, 1975; Reulen et al 1972; Brown et al, 1979). Cerebral oedema is reduced both directly

and indirectly through the decrease in intracranial pressure (Miller, 1981, Northfield 1973 Rotternberg et al 1980). Literature has also shown that the hyperosmolar agents lower cerebro spinal fluid pressure by reducing the rate of cerebrospinal fluid formation (Rotternberg et al 1980). Mannitol was thought by Wise and Chater (1961) to be preferable to urea; it is inert and non-toxic, its penetration is mainly confined to the extracellular space and the rate of excretion is fairly rapid. A 20 percent solution in distilled water is used in doses of 1gm/kg body weight over approximately 10 minutes (Miller 1981, Northfield 1973). With the advent of continuous ICP monitoring it has been shown that in some cases doses of half or even a quarter of this amount may reduce ICP almost as effectively (Miller, 1981).

Intracranial pressure starts to fall within half an hour and persists for several hours. In dogs Rosomoff (1962) found that the lowest C.S.F. pressure was reached 20 - 30 minutes after infusion had ceased and that the original pressure was regained and surpassed within six hours, (Northfield, 1973).

When mannitol has to be given more often than once every 3 - 4 hours to control ICP the serum osmolarity must be followed closely. If this rises to more than 320 mosmol/litre and ICP remains elevated, administration of more mannitol is likely to lead to renal failure, metabolic acidosis, and death (Becker and Uries, 1972). If the oedema continues to expand during withdrawal

of water from elsewhere, there is a tendency for ICP to rise to above pretreatment levels when the effect of the mannitol wears off. This may explain the so called rebound effect, which has received considerable attention in the literature and its based mainly on experimental studies (Northfield, 1973; Miller, 1981). Rotternberg, (1980) stated that a rapid reduction in the volume of normal brain and CSF without a concomitant reduction in the volume of a co-existing intracranial mass lesion (such as an epidural or subdural hematoma) may allow for expansion of the mass lesion, which had previously been tamponaded by underlying brain. So mannitol is relatively contraindicated in these cases.

(b) Systemic diuretics

Several studies have advocated the use of frusemide and ethacrynic acid in the management of patients with intracranial hypertension (Reulen and Hadjidimos, 1973, Rotternberg, 1980). This systemic diuretic may reduce ICP in one of several ways. Firstly, diuresis per se will reduce the volume of the intracranial contents in proportion as the percentage amount of total body water decreases (Rottenberg, 1980). Cerebrospinal fluid production at the choroid plexus is apparently decreased by these drugs. Pollay, 1975 states that both drugs cause a reduction of cerebrospinal fluid production rate by 40-70%



without subsequent reduction of I.C.P. Frusemide 250mgs orally for 5 - 8 days has been found to reduce the brain oedema significantly (Reulen, 1978). At best its effect seems to be mild and in some studies it has been used in conjuction with an osmotic diuretic to prolong and intensify the effect on ICP (Cottrell et al, 1977). Finally it should be stressed that frusemide or ethercrynic acid can decrease intracranial pressure without producing marked elevations of serum osmolarity or serious electrolyte disturbances (Rotternberg, 1980).

(o) Steroid therapy

In spite of the fact that corticosteroids have been used to treat patients with cerebral oedema and increased ICP for almost 20 years the mechanism by which they reduce cerebral oedema have yet to be defined (Rotternberg et al, 1980). It is likely, however, that several actions of glucosteroids take part in the reduction of oedema and other damaged brain tissue. One factor is an increased cerebro vascular permeability, (Long 1966; Rovit and Hagen 1968). Direct effects upon the transport of sodium, potassium and fluid across cell membranes or the capillary-glial interface (Long, 1966), a stabilizing effect upon membranes or lysosomal activity and an antioxidizing effect have also been postulated. And finally the reduction of c.s.f. production (Pollay, 1975).

The most widely used agents are dexamethasone, betamethasone, and methylprednisolone.

The clinical effects in suitable patients are manifested in a few hours by improvement in the conscious level and neurological function. ICP does not become significantly reduced until after 24 hours of therapy, well past the clinical effect (Miller, 1981).

Dexamethasone is usually administered as an initial dose of 10mgs followed by 4mgs every 6 hours for 48 hours intramuscularly, reducing the dose over 5 days (Horton, 1975; Miller, 1981).

Dexamethasone is known to give a dramatic effect in the cerebral edema of tumours; it is less effective in head injuries, but it is worth trying (Horton, 1975; Hunter, 1975).

Steroid therapy is not without risk of gastric haemorrhage, wound dehiscence, and lowered resistance to infection - particularly in the high dosages that are now being used in neurosurgical practice (Miller, 1980).

(d) Hyperventilation

As stated before this technique is very useful in the reduction of ICP. Controlled ventilation is an effective method of reducing intracranial pressure, particularly when the arterial carbon dioxide tension ($PaCO_2$) is lowered between 25 and 30mmHg, providing that the resistance vessels of the brain are still responding to $CO_2$. (Horton, 1975; Miller et al 1977).

(e) C.S.F drainage

In patients who have an indwelling ventricular catheter, useful control of elevated ICP irrespective of the cause may be obtained by draining off CSF. External Ventricular drainage and Ventriculosystemic shunts reduce ICP by providing a low resistance pathway for CSF absorption (Rotternberg, 198C, Miller 1981). C.S.F. can be removed from lateral ventricle against a positive pressure of 15mmHg (20cm $H_2O$) where this is being used for continuous intracranial monitoring (Horton, 1975, Miller 1981). This positive pressure prevents the ventricles from collapsing around the catheter.

C.S.F. drainage by lumbar puncture is not indicated in cases of oedema or swelling because this procedure may promote tentorial or tonsilar herniation (Miller, 1981).

(f) Brain Resuscitation

Hypothermia

The role of hypothermia in brain resuscitation remains controversial. Generally speaking acute hypothermia will significantly prolong the brains tolerance for varying periods of ischemia depending upon the degree of hypothermia. Whether or not hypothermia induced after a period of complete brain ischemia offers any beneficial effects remains unknown (Samuels, 1980; Michenfelder, 1982).

Most of the trials of application of hypothermia have been done in animal models. (Michenfelder, 1982), has shown that when cats are cooled to 29°C for 48 hours and then rewarmed by external means there consistently develops a severe systemic acidosis followed by haemodynamic collapse and death. In dogs cooling to 29°C produces the expected immediate 50% reduction in cardiac output and whole body oxygen consumption. However with continued maintenance of hypothermia at 29°C there is a progressive reduction in both cardiac output and oxygen consumption such that after 24 hours the cardiac output is on the average less than 10% of control whereas oxygen consumption is reduced to about 30% of control. However, acid balance and vital signs appear normal. With rewarming cardiac output falls to increase significantly oxygen consumption increases only moderately and severe acidosis develops with hemodynamic collapse.

Whether or not those effects of chronic hypothermia are operative in man remains unknown. But there is possibility that such may occur.

(iii) Barbiturate therapy

The thought that barbiturate therapy may be effective was based upon the observation that deep thiopental anaesthesia results in a reduction of cerebral metabolic rate for oxygen ($CMRo_2$) of a magnitude similar to that induced by moderate hypothermia (30°) without any alteration in the brain's normal cerebral energy requirements (Michenfelder, 1982). Since acute hypothermia is a well accepted protective measure for prolonging the brain's tolerance to ischemia

as explained above, and since this is assumed to be based upon its metabolic depressive effects, it seemed logical to conclude that deep thiopental anaesthesia should induce a degree of protection comparable to that provided by hypothermia 30°C. But this does not apply following onset of complete cerebral ischemia. The other mechanism is that barbiturates may act as scavengers of membrane damaging free-radicals which might be performed during, or more likely after, an ischemic event. This hypothesis does not appear to be valid (Demopoulos et al, 1977).

Although barbiturates have been recently recommended for the treatment of head trauma patients there is no evidence that barbiturates provide any unusual protective or emeliorative effects over and above their capacity to reduce ICP.

In addition to barbiturates there are other drugs which have been suggested or found to be protective before or after global ischemia (Wolfson et al, 1977). Gammahydroxybutyrate has been reported to reduce metabolic rate by as low as 35% (in rats) (Michenfelder, 1982). Phenytoin has been reported in number of different animal models to provide a degree of cerebral protection. (Artruu et al, 1980). Midazolam a new water soluble benzodiazepine is a potent depressant of cerebral metabolic rate, similar to that observed with thiopental (Michenfelder 1982). Because of less systemic hemodynamic depression, midazolam may prove to be a useful agent for the purpose of brain protection (Michenfelder, 1982).

(iv) Treatment by Surgical Intervation:

(a) Investigations

With the advent of computerized axial tomography scan, valuable information is obtained very easily. If no computerized axial tomography scan is available, anteroposterior film of the skull may demonstrate a shift of the Pineal gland and help make the decision about arteriography (Bruce, 1980; Teasdale, 1976). Focal pathology such as acute epidurals, subdural, or intracranial haemorrhages and their relative size are easily identified (Bruce, 1980). Cerebral oedema and haemorrhages can readily be differentiated from true focal haematomas and unnecessary surgery avoided. The large number of patients without a surgically accessible or treatable lesion can readily be identified and not subjected to exploratory surgery. All patients who have significantly disturbed consciousness with evidence of a focal neurological abnormality, all patients who show signs of clinical deterioration and all patients who are in deep coma require to be closely observed and managed (Bruce, 1980).

(b) Management of Depressed Skull fracture

If the depressed fracture is closed, the rationale for surgical correction is, to evacuate such local mass if present as subdural, epidural or intracranial hematoma; and repair any local lacerations to prevent cerebral herniation through

the defect. All compound depressed skull fracture require surgical debridement (Northfield, 1973; Bruce, 1980).

Epidural hematomas

Epidural hematomas are the most readily correctable surgical lesions seen following trauma and it is to avoid missing an expanding epidural hematoma that the practice of hospitalising all patients with transcient unconsciousness is based (Bruce, 1980). It is extremely important to identify the epidural hematoma at the earliest possible time (i.e. when the headache and drowsiness are the only complaints)(Northfield, 1973). The best diagnostic test is CAT scan, if available. The advantage of the CAT scan is that it demonstrates not only the location of the clot but also defines any other lesions within the cranium, (Bruce, 1980). The treatment of an epidural hematoma is surgical evacuation. (Northfield, 1973; Rains et al, 1975; Bruce, 1980). Medical management to control rapidly progressive intracranial hypertension may be necessary to gain time to reach the operating room and evacuate the clot, (Bruce, 1980; McDowall, 1976).

Acute Subdural hematomas

Collections of blood in the subdural space that are identified within the first 72 hours following trauma are arbitrarily classified as acute subdural hematomas (Bruce, 1980).

Acute subdural hematomas may be classified into two main types. Simple-collections of blood or liquid in the subdural space but not apparently associated with underlying cerebral contusion or laceration and complicated liquid or clotted blood in the subdural space associated with obvious contusion or laceration of the surface of the brain (Jamieson, 1972).

The clinical presentation of patients with acute subdural hematomas is that of unconsciousness without a lucid interval with signs suggestive of a mass lesion, hemiparesis, unilateral decerebration, or pupillary enlargement.

If the major problem contributing to the patient's poor neurologic condition is the mass of the hematoma, then that should be removed. If the main reason for the patient's state is the underlying cerebral injury, then treatment should be directed against that. This suggests that patients with simple subdurals with little cerebral injury and a deteriorating or poor level of consciousness will benefit from removal of the mass.

Miller (1977) has shown that 58% of patients operated on for evacuation of acute subdural hematomas developed increased ICP greater than 20mmHg in the postoperative period and approximately half of these patients die because of uncontrolled intracranial hypertension. Thus, a major part of the treatment of acute subdural hematomas is the control of the brain swelling, edema, and elevated ICP whether or not surgical decompression is performed.

Intracerebral hematoma

The clinical picture may vary from no neurologic deficit to deep coma. Patients with slowly accumulating frontal or temporal lobe hematomas frequently demonstrate a rather slowly deteriorating level of consciousness with minimal focal signs over 24 to 48 hours. These secondary unusual clinical pattern are seen in patients with delayed hemorrhage. These patients show progressive recovery of level of consciousness over 10 to 14 days and then suddenly develop headache followed by rapid neurologic deterioration. According to Miller (1977), large solitary intracranial hematomas should be evacuated. The mortality following surgery for traumatic intracerebral hematoma has been reported to be 42% (Miller, 1977). The outcome depends on the level of consciousness at the time of surgery more than any other single factor.

(v) Gastrointestinal bleeding

The relationship between intracranial disease and gastro-intestinal bleeding has been known for years. Cushing ascribed the coincidence of peptic ulceration and intracranial pathology, therefore Cushing ulcer is the term usually applied to all gastro-intestinal ulcers co-existing with the brain disease. Eosophageal ulceration seems to be more common in the neurologic lesion while duodenal ulceration is more common in the non-neurological disorder (Bruce, 1980). There is no correlation between the type of neurosurgical

disorder (diffuse or discrete) or intracranial hypertension and gastroduodenal haemorrhage or ulceration. (Matjasko, 1980).

Cushing (1932), felt that a diencephalic parasympathetic centre stimulated vagal nuclei, inducing Vagal hyperactivity and subsequent acid hypersecretion.

Gastric mucosal barrier diruption may play a role in the pathogenesis of such ulcers. (Skillman, et al 1970).

Steroids do not alter gastric mucosal permeability but may enhance the effect of other agents that do for example ethanol and aspirin (Aubrey et al, 1972).

Watts and Clark (1969) have reported gastric hyperacidity in patients comatose from head injury. Daily gastric acidity was measured in 10 comatose patients, from the day following head injury. The acidity rose on the second day in all except in one patient who had no rise in acid secretion. Bleeding occurred in five of seven hypersecretors who were decerebrate with signs and symptoms of lower diencephalen or upper brain stem injury.

Signs and symptoms of gastro-intestinal ulceration and/or perforation may include pain, abdominal distention, ileus, anaemia, hypotension, hematemesis and melena, depending on the level of consciousness of the patient. Steroid induced bleeding into the Gastrointestinal tract may be painless. An antiacid therapy administered orally or via nasogastric tube may be of value in

preventing and treating postneurosurgical and steroid ulcers, (Matjasko; 1980). Bleeding is usually self limiting and may require nasogastric suction with cold saline lavage, fluid replacement and transfusion. $H_2$ receptor blockers (Metiamide, Cimetidine) reduce the bulk of all forms of gastric secretion. Clinical trials are not available; however this approach may prove to be effective in prophylaxis and therapy of a variety of stress ulcer syndromes. (McDonald, 1976; Mcdougall, 1977).

(vi) Nutrition:

In the adult there is no need to give fluid or food for the first 12 hours after injury unless there is evident dehydration from other cause, for instance vomiting (Northfield, 1973). After 12 hours in the unconscious patient fluids and food must be given through a gastric tube, until swallowing and cough reflexes have returned. The tube should be passed though the nose into the stomach. It should be removed for cleaning or replacement every 7 - 10 days. For the first 12 hours glucose and water are given in incremental volumes hourly starting with 30ml. If these are tolerated, milk is added and the intervals increased to 2 hours, until a total of about 2.5 litres is attained in the 24 hours. (Northfield, 1973). Carefully administered total parenteral nutrition, ideally with all three main nutrients (Carbohydrate, protein and fat), should be provided whenever tube feeding is impossible or contraindicated in patients who are unable to feed themselves (Ballinger, 1975).

BRAIN DEATH

The development of effective means of maintaining ventilation and supporting the circulation has led to the appearance of a syndrome of brain death. Harrison, (1976). One of the major difficulties in dealing with the subject of brain death has been imprecise usage of terminology, as in the synonymous use of words such as cerebral death, irreversible coma, cortical death, and brain death (Korein, 1980).

Death. The classic usage defining the term related to criteria used by a physician to ascertain the irreversible end - state of an individual. These criteria evaluate the cessation of vital functions. These vital functions have in the past included cessation of cardiac and respiratory activity and total unresponsiveness. Those classic clinical criteria may be supplemented by observations relating to rigor mortis or even putrification (Korein, 1980).

Harrison, (1976) states that the presence of unresponsive coma with apnoea and absent brainstem reflexes with isoelectric EEG records is tantamount to cerebral death. Drug overdosage and hypothermia must be excluded before the diagnosis is made and a repeat assessment in 12 hours is customary before withdrawing supportive measures.

Because of the limitations of knowledge surrounding the problem of death, various societies have developed their concepts and criteria for diagnosing death. Most often quoted are the Havard criteria, (Report of ad hoc committee of Harvard Medical School to examine definition of brain death); developed in 1968, which define cerebral death as irreversible coma (brain death) in terms of the following characteristics:

1. Absence of cerebral responsiveness
2. Absence of induced or spontaneous movement.
3. Absence of spontaneous respirations (requiring the use of a respirator).
4. Absence of cephalic and deep tendon reflexes.
5. Absence of drug intoxication or hypothermia.

Minnesota criteria (1971)

1. Basic prerequisite: diagnosis of irreperable cerebral lesion
2. No spontaneous movements.
3. No spontaneous respiration.
4. Absence of brain stem reflexes.
5. Persistence of condition unchanged for 12 hours.

Japanese criteria (1973) Uek, et al (1973).

1. Basic prerequisite: diagnosis of primary cerebral lesion.
2. Deep coma
3. Respiratory arrest

4. Bilateral dilated pupils and absent pupillary and corneal reflexes.

5. Flat EEG

6. Abrupt fall in blood pressure of 40mmHg with hypotension.

7. Persistence of condition for at least 6 hours.

A most important dysfunction of the brain stem is that of failure to maintain respiration. Occassionally a satisfactory test for apnea requires that the patient be off the respiratory for a prolonged period. (Korein, 1980).

Unnecessary anguish and distress to relatives and nurses may be caused by insensitive or thoughtless handling of brain dead patients. A correct approach should make it abundantly clear (both to relatives and to a wider public that in the act of disconnection, the doctor is not withdrawing treatment and allowing someone to die but ceasing to do something useless to someone who is already dead (Jennet and Hesset 1981). If a patient is thought to have a dead brain stem the doctor in charge of the case should have the diagnosis confirmed by appropriate colleagues using accepted criteria. The tests should be carried out by two medical practitioners who have skill in this (Pallis, 1983).

In Britain many units continued to ventilate most such patients to an inexorable systole. In the early 1970's practice began to change. In 1970 the Neurosurgical group at the Salpetriere, in Pa:

publised an important study showing that in nearly 70 patients with structural brain disease sustained apnoeic coma and absent stem reflexes always heralded asystole, despite the fullest possible ventilatory support. Publish evidence suggests that in most cases asystole develops within days, (Jennet et al 1981). Of the 63 patients diagnozed as brain dead in large Danish series (and maintained on the ventilator 29 developed asystole within 12 hours, 10 after 12 - 24 hours, 16 after 24 - 72 hours, and eight after 72 - 211 hours. Experience from Great Britain (Jennet, 1981) and elsewhere is in line with these observations.

Rehabilitation

Rehabilitation as regarded by Northfield is a controlled method of putting the brain back to work on trial, the work in this context comprising the varied and graded activities, mental and physical, which modern departments can provide in a pleasant and encouraging environment. The activities must cover wide range of specialities for example neurosurgery, psychiatry, psychology, orthopaedics, urology, speech, occupational and re-employment services. Rehabilitation may be focused on four main categories.

1. The general symptoms and disabilities

   Among these headache and various forms of giddiness are perhaps the most frequent; both tend to improve with time provided the activity is tailored to the individual. Confinement within the bed is avoided, provided the patient is physically fit he should come out of the bed.

2. Emotional disturbance

A charming and optimistic attitude should be displayed to the patient. He should feel free to discuss his illness. The patient should discuss affairs about his employment and home. A social worker is able to render greater assistance with regard to family and employment.

3. Neurologic defects

Physiotherapy is necessary for hemiperesis and occupational therapy if there is sensory loss as early as possible. Speech therapy is very necessary to dysphasics.

4. Return home and to work

Rehabilitation should be regarded as a continuing programme in the care of the patient until recovery is complete or until it has reached its maximum and the patient is settled in employment or in other activity on which he can make a living. But return to work requires an even greater exercise of judgement. The decision can be taken only with due regard to the domestic background; the severity of the injury; the type of sequale; and the type of treatment to be continued at home. (Northfield , 1973).

## A PERSONAL EXPERIENCE IN SOME HEAD INJURIES CASES:

### Introduction:

A survey was made to know what immediate treatment was given to patients with head injuries in Dar es Salaam at the site of accident on the way to Casualty and in the wards. From the results to note any shortfalls about the knowledge of personnel and management. With the support of the data to recommend some methods which I feel in the future will reduce the morbidity and mortality of head injuries at Muhimbili Medical Centre and the Country as a whole.

### 1.1 Patients:

All patients from the age of 13 and above with head injuries admitted through the emergency department from 1st May 1982 to June 1983 were followed from Casualty to Intensive Care Unit; Ward 9, 2 and the Operating theatre. History was obtained from the ambulance men, relatives or patients themselves.

### 1.2 Methods

Name, age, sex, circumstance of accident, accident and admission times, clinical states at admission using Collins and Glasgow Coma scale, types of resuscitation given to them at the site and Casualty, definitive treatment given to them in the wards, Intensive Care Unit and theatre were noted up to the seventh day of their stay in hospital.

For those patients who were in the wards, day to day management was recorded from the Casenotes, but for those who were admitted in the Intensive Care Unit, the daily management was done by myself, the resident anaesthetist in ICU with the help of the Neurosurgeon.

The following things were done to every patient on arrival in ICU.

- Airway was made clear by nasopharyngeal or oropharyngeal suction.
- Intubation was done to those with poor respiratory function.
- A good intravenous line was established.
- A nasogastric tube inserted.
- Urethral Catheter for monitoring Urinary output was mandatory.
- Temperature monitoring 2 hourly.

II Nursing Care

The following procedures were strictly adhered to, in all patients.

- Suctioning 1 hourly and when necessary depending upon the individual patient's rate of formation of secretions.
- Turning the patient 1 hourly
- Strict monitoring of vital signs that is pulse, blood pressure, respiration and temperature.
- Strict input and output chart.

- Application of talc powder to avoid pressure sores.
- Feeding through the nasogastric tube when the stomach function was established.

III Medications:

Some patients received 10mgs of dexamethasone as a start dose with 4mgs 6 hourly for the minimum of 5 days. Mannitol when available 20% 1 - 1.5g/kg was run fast within 10 - 15 minutes when the patient was scheduled for surgery.

50% glucose 20cc as a bolus dose was given to almost all pati ents.

Broad specturum antibiotics were given to all patients with open wounds.

Antitetanus serum 1,500 iu was given to all patients with open wounds irrespective whether they had any booster dose because of the unreliable history from the patients or relatives.

Results

There was a total of 220 head injuries in this particular period, 180 males (82%) and 40 females (18%). 45 deaths.

The most affected group was below 40 years with a peak at 2nd and 4th decade. Table I and Fig. I. demonstrate this phenomenon.

10.1 Showing No of patients against age groups of both sexes put together

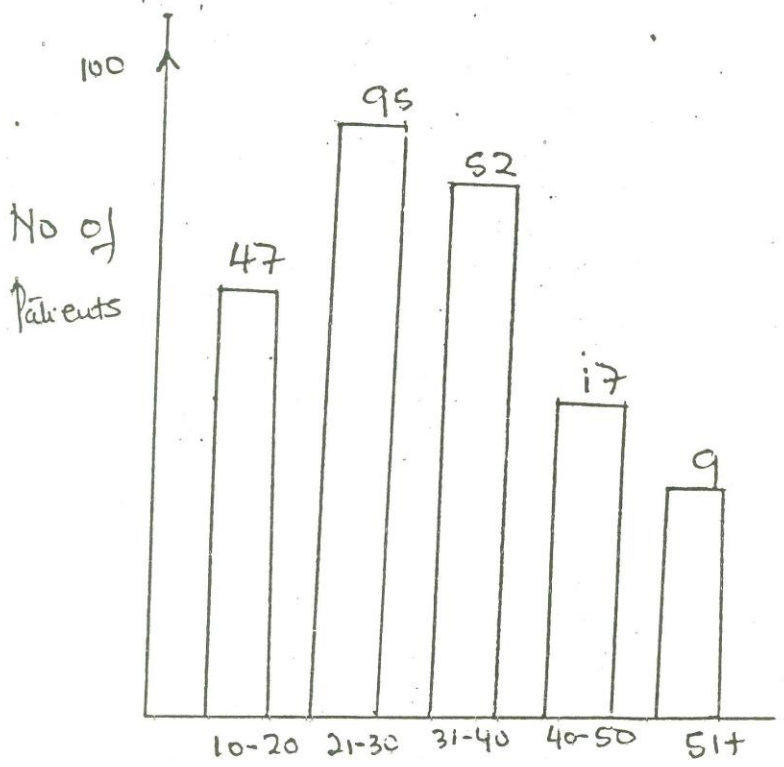


Fig 3.1 Showing No of patients against age groups of both sexes put together

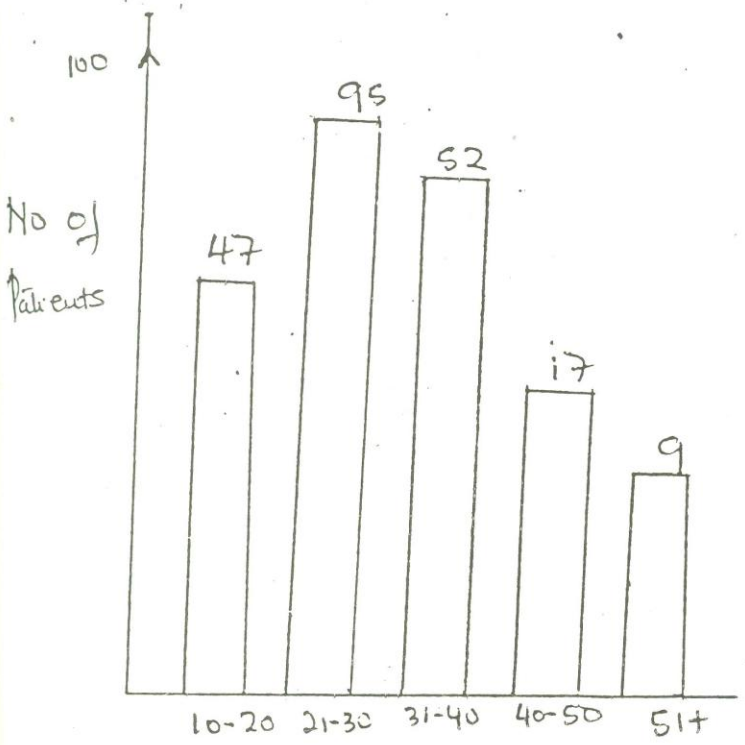


Table 1: Age and Sex distribution of Head Injuries

| Age Range | Male | Female | Both Male & Female | % |
|---|---|---|---|---|
| 13 - 20 | 40 | 7 | 47 | 21.4 |
| 21 - 30 | 80 | 15 | 95 | 43.2 |
| 31 - 40 | 45 | 7 | 52 | 23.6 |
| 41 - 50 | 10 | 7 | 17 | 7.7 |
| 51+ | 5 | 4 | 9 | 4.1 |
| Totals | 100 | 40 | 220 | 100 |

TABLE II: Circumstances of Accident

| | Female | Male | Both | % |
|---|---|---|---|---|
| Driver/Passenger | 18 | 80(40) | 98 | 44.5 |
| Pedestrian | 10(3) | 60(7) | 70 | 31.8 |
| Home | 2 | - | 2 | 0.9 |
| Work | - | 5 | 5 | 2.4 |
| Play | - | - | - | |
| Assault | 6 | 30 | 36 | 16.3 |
| Not known | 4 | 5 | 9 | 4.1 |
| Totals | 40 | 180 | 220 | 100 |

On circumstances of accident, road traffic accident was the commonest cause followed by assault.

In Road traffic accident 45% of patients had history of alcohol ingestion.

We had unknowns picked up on the road and some died without established history. This contributed approximately to 4.1% of the total, probably they were drunkards.

Number in brackets indicate history of alcohol intake.

Clinical State on admission is as shown in Table III below.

TABLE III: Clinical State on Admission

| | | |
|---|---|---|
| Conscious | 80 | (-) |
| Unconscious | 45 | (-) |
| Flaccid/decerebrate | 45 | (3) |
| 1 pupil dilated and fixed | 15 | (5) |
| Both pupils dilated and fixed | 10 | (10) |
| Hemiplegia/Hemiparesis | 15 | (3) |
| Severe associated injuries | 15 | (4) |
| Totals | 240 | (25) |

Number in brackets = Deaths

The total is more than 220 because some signs have been occurring together.

The majority of the patients were conscious 33% . 4.1% of the patients had dilated and fixed pupils and it is in this group where there was a high mortality.

Accident time and admission times were rarely noted except for the few patients I attended to and the 5 cases needing surgery whose average times which elapsed till surgery was performed were as shown below.

TABLE IV: Table showing time elapsed till surgery was performed

| Case No. | Approximate time in hours |
|---|---|
| 1 | 2 |
| 2 | 3 |
| 3 | 12 |
| 4 | 2 |
| 5 | 6 |

Comment: This table definitely shows that there was a delay in starting operative treatment when operation was indicated as in Case Nos. 3 & 5. One of the contributing factor was the delay from the Casualty to the X-ray department and again back to Casualty and then finally to the Ward. The other reason was that the Neurosurgeons usually stay far from the hospital, so when the resident doctor decided to call him there was always a lot of wasted time in waiting. And also the theatre was not always freely available for such emergencies.

Table V: Showing Survival time in Fatal Cases

| | No. of Patients |
|---|---|
| Death on scene of accident or on the way | 12 |
| Death at Casualty | 8 |
| Death within 48 hours | 20 |
| Death between 2nd and 4th day | 5 |
| Total | 45 |

12 patients died on the way to the hospital, 8 at the Casualty and 25 patients died in the hospital within 72 hours. That is 20.4% died within 72 hours. This could be due to poor first aid, associated complications or the severity of the head injury per se. Nothing conclusive can be drawn.

No resuscitation was done at the site - probably due to lack of staff, motivation, sympathy or fear of giving some information to the police when they arrive.

Table VII: Type of Resuscitation done at Casualty

| No. of Cases | Type of Resuscitation done |
|---|---|
| 70 | Clear pharynx by suction |
| 100 | Prone position |
| 50 | Oropharyngeal airway |
| 10 | Endotracheal intubation |
| 1 | Tracheostomy |
| 200 | Intravenous infusions |

N.B: Some patients were able to get two or three types of resuscitative measures at the same time, that is why the totals appear to be more than 220.

No resuscitation was done at the site or on the way. Some resuscitation was done at the Casualty. Positioning and suctioning being the commonest - see above. One tracheostomy was done at the Casualty which is rather unusual but this was necessitated by the critical condition of the patient who unfortunately died. Therefore facilities for tracheostomy should be available at the Casualty.

Table VIII: Types of Resuscitation done in WD 9

| No. of Cases | Type of Resuscitation |
|---|---|
| 50 | Clear pharynx by suction |
| 150 | Position patient |
| 5 | Oropharyngeal airway |
| - | Endotracheal intubation |
| - | Tracheostomy |
| 100 | Intravenous infusion |

TABLE IX: Types of Resuscitation done in Ward 2

| No. of Cases | Type of Resuscitation |
|---|---|
| 15 | Clear pharynx by suction |
| 25 | position patient |
| 2 | Oropharyngeal airway |
| - | Endotracheal intubation |
| - | Tracheostomy |
| 25 | Intravenous infusion |

40 patients were admitted in Ward 2 and 140 patients in Ward 9. Types of resuscitations rendered to them were similar in pattern except for the more work load experienced in Ward 9.

TABLE X: No. of Patients admitted in respective Wards

| Ward | No. of Patients |
|---|---|
| Intensive Care Unit | 40 |
| Ward 2 | 40 |
| Ward 9 | 140 |
| TOTAL | 220 |

5 patients required Intermittent Positive Pressure Ventilation (IPPV) level of consciousness; intracranial lesion and time of death are as shown in Table XI: and other measures done in the ICU are shown in Table XII.

TABLE XI: 5 Patients with non-reacting pupils dying on IPPV

| Age | Conscious level | Intracranial lesion | Death | $PaO_2$ | $PaCO_2$ |
|---|---|---|---|---|---|
| 55 | Coma | Subdural haematoma | 48 hrs | - | - |
| 34 | Coma | Primary brain stem | 36 hrs | 55 | 30 |
| 35 | Resp. to pain only | Large subdural | 4 hrs | - | - |
| 40 | Coma | Extradural haema-toma | 72 hrs | - | - |
| 22 | Semi Conscious | Subdural haematoma | 36 hrs | - | - |

TABLE XII: Types of Resuscitation done in I.C.U.

| No. of Cases | Type of Resuscitation done |
|---|---|
| 40 | Clear pharynx by suction |
| 40 | Position patient |
| 40 | Oropharyngeal airway |
| 25 | Endotracheal intubation |
| 1 | Tracheostomy |
| 40 | Intravenous Infusions |
| 10 | IPPV |

Comment: 1 patient had tracheostomy done after staying in the hospital for 4 days respiratory embarrasement being the indication. Finally he survived. He was changed to tracheostomy because of increased frequency of blocking the tube by secretions.

TABLE XVIII: Drugs used for reduction of Cerebral Oedema in some Patients

| Drug | Number of Patients |
|---|---|
| Mannitol | 50 |
| Dexamethasone | 80 |
| Methyl prednisolone | - |
| Betamethasone | - |
| 50% dextrose | 80 |
| Lasix | - |
| None | 70 |

Comment: Some patients missed mannitol and dexamethasone because they were not available. Lasix was not used in our patients routinely.

TABLE XIV: Table showing Types of Drugs Used in Controlling Brain Oedema and outcome

| Drugs | Recovered | Died/Vegetable | Totals |
|---|---|---|---|
| Dexamethasone and mannitol | 75 | 5 | 80 |
| Without dexamethasone ± mannitol | 42 | 8 | 50 |

Comment: Nothing specifically can be concluded from the above findings. Some patients missed the drugs just because there was an acute shortage of drugs during that period although it might appear that those patients who tended to get both drugs had greater chances of survival.

TABLE XV: Showing Operative findings and Outcome

| Normal brain | Cases | Outcome | |
|---|---|---|---|
| | | recovered | died |
| Normal brain | 5 | - | - |
| Significant Extradural ± contusion/swelling | 1 | 1 | |
| Significant subdural ± Contusion/Swelling | 2 | 1 | 1 |
| Contusions/lacerations/swelling | 2 | | 2 |
| Totals | 5 | 2 | 3 |

5 patients had craniotomies. 3 patients died and 2 recovered. Among the 3 who died 2 were delayed for operation. See Table IV. Deaths occurred between 12 - 36 hours after the operation.

TABLE XVI: Showing no. of patients Vs technique of Anaesthesia:

| No. of patients | Technique of Anaesthesia |
|---|---|
| 5 | Induction: preoxygenation, atropine, thiopentone just enough to keep the patient to sleep depending upon the level of consciousness, suxamethonium preceded by small dose of non-depolarizing neuromuscular agent pavlon 2mgs to reduce the fasciculations and hyperkalaemia. Then followed by gentle intubation.<br><br>Maintenance: $N_2O$, $O_2$, Pavlon 4-6mgs and halothane 0.5% Controlled hyperventilation |

Comment: It is difficult to conclude anything from such a small number of patients on the type of anaesthesia used and the outcome.

TABLE XVII: Conservative Treatment with Survival Rates

| TYPE OF INJURY | NO. |
|---|---|
| Brain Concussion | 102 |
| Brain Concussion + fracture of limbs | 40 |
| Brain concussion + thoracic injury | 3 |
| Brain concussion + maxilofacial injury | 10 |
| Brain contusion ± Subdural/Extra-dural | 40 |
| Total | 195 |

195 patients had conservative treatment including splinting of fractured limbs or mandibles. 5 patients had operative treatment. 20 patients died.

TABLE XVIII: Postmortem findings in some of the head injured Patients on whom postmortem could be performed

| Cause of death | No. of Patients |
|---|---|
| Intracranial haemorrhage | 14 |
| Subdural haemorrhage | 11 |
| Ruptured spleen & liver | 2 |
| Subarachnoid haemorrhage | 3 |
| Total | 30 |

Comment: Intracranial haemorrhage was the leading cause followed by subdural haemorrhage. There is need for aggressive management as far as evacuation was concerned.

## DISCUSSION

The results of this survey demonstrate that there is a lot to be done inorder to decrease morbidity and mortality of these young women and men who are in the productive age group and most needed by the society. As shown in Table I the commonest age group was below 40 years and males were more affected than females. The same pattern was found in Ibadan by Adeboye, (1976), and Mwang'ombe (1982) in Kisii district. This can be explained by the more adventurous pattern of life at this age group and history of ingestion of alcohol which was found in 50 cases and therefore partly responsible for the accident. Table II. The association of cerebral depression and head injury is extremely dangerous. The gravity of the head injury may be overlooked because of the intoxicated state. Although blood alcohol concentrates are not routinely performed but obvious alcohol consumption should be recorded.

Times of accident and admission were not recorded in most patients. Mockey, (1969) analysed the time of survival in fatal cases. He suggested that 43% of rider or vehicle occupants might have had a greater chance of survival if some medical treatment had been available at the scene of accident, within 10 minutes. If times were noted this would have given us a clue whether the 20 patients who died on the way, the cause was due to delay of medical help or severity of the injury.

This wouldn't be surprising because of poor communication which is presently existing in our country.

No resuscitation was done at the site for that matter little at the Casualty. More skilled resuscitation was given in the Intensive Care Unit followed by the general wards although it was inadequate. Experience has shown that however quickly the rescuers are alerted the first on the scene is usually a layperson who does not know anything about resuscitation. A high percentage of the people hurt in cars are pulled out by frantic rescuers who make the injuries worse or are even killed in the process. It pains to see also that ambulance men cannot handle the injured patient. So there is need for a training scheme to be drawn.

Road traffic accidents constituted an important source of head injuries. The vast majority of head injuries were minor in nature with at least 80% of victims surviving with or without sequale. Of those who died 75% did so within 24 - 72 hours of injury the mortality being directly related to the level of consciousness. The above findings are similar to the one of Lewik, (1959) when discussing on head injuries in Civil practice which gives 75% in 48 hours.

Since assessment of the level of consciousness is extremely objective depending on the experience of the observer, the site of application of the painful stimulus and the intensity of the

stimulus may differ from one observer to the other. As the management of the severely injured patients depends upon evaluation made by junior medical and by nursing staff, to be of practical value a system must be simple and based upon clearly defined criteria. Presently Muhimbili Medical Centre, Department of Orthopaedics and Trauma is using Collins level of Consciousness attached paper.

But experience has shown that Glasgow Coma Scale introduced by Teasdale and Jennet, 1974, (Teasdale, 1976), was more easier to understand and more useful in day to day monitoring. However, the final level of consciousness was the one assessed by the neurosurgeon in all cases so observational errors were minimised.

5 patients were put on the ventilator shortest survival was 4 hours and the longest survival was 72 hours. Now since these patients had apnoea and non-reacting pupils and were on deep coma especially the three cases table XI, probably they had imminent brain death. As we know Harrison, 1976 stated that brain death is pronounced when there is presence of unresponsive coma with apnoea and absent brain stem reflexes with an isoelectric EEG. But it must exclude drug overdosage and assessment should be repeated on two occasions at least 12 hours apart. If the patient qualified the above criteria there is no use keeping him or her on the ventilator (Harrison, 1976; McDowall, 1976). But our patients continued with IPPV till time of death. From sociological point of view it was a very big burden to the relatives who had to come to the hospital frequently to see them with some hope that the patients may recover.

Head Injury

= Scalp, Skull, brain

level of Consciousness.

1. Responds to spoken word, is alert cooperative and oriented.
2. Responds to spoken word, is confused and obeys simple commands.
3. Responds to spoken world only after receiving painful stimuli of supraorbital pressure; obeys commands only while receiving painful stimuli.
4. Does not respond to auditory stimuli or while receiving painful stimuli, but has purposeful, effective motor response to painful supraorbital or sternal pressure.
5. Responds to painful supraorbital or sternal pressure with purposeful but non-effective motor movements.
6. Responds with non-purposeful movements.
7. Responds to painful stimuli only with alteration in pulse and respiratory rate or with decerebrate posturing.
8. No response to painful stimuli but cough and gag reflexes are present.
9. No response to stimuli.
10. Lowest level of unconsciousness; the only next step below is death.

To the nurses was the reduced morale of having to change the beds or washing of mouths of electroenchegraphically dead patients.

From the economic point of view the half life of the ventilator was shortened. It is high time that at the Muhimbili Medical Centre a policy should be established when to stop the ventilator, probably when the above criteria are fulfilled.

Intracranial pressure monitoring was not done in all the patients. As we know the understanding of the dynamics of the ICP changes is important for many anaesthetists not only in specialised neurosurgical units but also in the care of unconsious patient with a head injury, either on the Accident and Emergency department or in the Wards and Operating theatres. In the acute stage of the management of head injury ICP measurements alerts the clinician at an early stage as to the occurrence of brain compression, caused either by oedema or haematoma formation. Turner and McDowall, (1978). In our situation this was not done resulting in the delay of decision to operate. Where there was imminent increase it could be reduced by hyperosmolar therapy, steroids, cerebrospinal fluid removal or hyperventilation and improvement could be detected by continuous ICP monitoring. It is unfortunate that facilities for the procedure are not available at the Muhimbili Medical Centre.

Brain oedema was routinely managed by using hyperosmolar solution, mannitol 20%, 50% dextrose followed by dexamethasone.

Their efficiency was difficult to prove hence the importance of ICP measurements to ensure an adequate dose. As shown earlier in the literature steroids, for example dexamethasone and betamethasone, are most effective in the relief of pressure state resulting from tumours, especially metastatic tumours or cerebral abscess. They appear to be less effective in oedema following head injury.

Of the 5 patients who underwent craniotomies 3 died giving the mortality rate of 60%. Nothing can be concluded from that keeping in mind that the sample size was small and the patients were very moribund

From the foregoing discussion we find that there are problems which need to be rectified. Problems of transport, problems of resuscitation at the site of injury and Casualty, and the training of Ambulance personnel. Ambulance services in Dar es Salaam are individualised. Each hospital or public institution has its own ambulance but as far as resuscitation is concerned they are all inadequate. The crew are not trained. It is high time for us to have an emergency communication system which links the public with the emergency services by radio call system. All ambulances should be in central pool, when a need arises a call should be made to the central system as being done for the needs of the police or fire. The response to such emergency calls is to mobilise an ambulance

from a base or sending available mobile unit nearest to the site of the accident.

One of the commonest problem for hospital service is the usual failure of communication within the hospitals. Communication fail because it is not available. Other emergency services - police, and fire even Usafiri Dar es Salaam (U DA) possess their own radio networks, the hospital should possess one as it is a big Medical Centre and National Referall hospital.

Because of financial constraints our ambulances should have at least minimum basic resuscitation equipments. These should include assorted endotracheal tubes and connections, Guedel airways, laryngoscope adult and child, assorted suction catheters, intravenous sets, large oxygen cylinder, suction apparatus, bag and mask. Spinal cord and cervical collars, and a few resuscitating drugs.

The main problems at the site of accident are of airway, again and again it has been stressed that hypoxia or anoxia is a killer. This can arise from obstruction by the tongue when the patient remains on the back after the accident or during transportation or can be caused by blood, vomitus or extended neck. So educating the police, ambulance crew and the public the correct position of the head injured patient is very essential. This position should keep the neck in the neutral position that is the semi prone. Efficient suction with the art of oropharyngeal intubation and the use of the

catheter are the basic needs of every ambulance man. These people should be trained by the cooperation of the departments of Emergency services and Anaesthesiology. The objectives should be to provide a comprehensive and skilled mobile resuscitation service within the area served by the hospital for all patients requiring resuscitation regardless of the underlying disease or injury. The duration of training should be at least 4 months in which the trainees are exposed to practical demonstration and practice in techniques of resuscitation and immediate care in the emergency department, the Intensive Care Units theatres and recovery rooms. If ambulance services are expensive then the trained passers-by may save the life by positioning the patient. This can be achieved only if there is a mass campaign especially when it concides with road safety week organized yearly by the traffic police. The traffics can issue placards and place them along the streets with just few words and drawings depecting how to position a patient.

The establishment of First Aid Care, depending on the cooperation between the Casualty department, police and ambulance services is very essential in dealing with these problems, Eastink (1977). A policy should be made on which the patient should be assessed and initially treated at the site and further observation to the hospital should be the next step. The implementation of this procedure is not simple but the system should be established as in other Centres for example United Kingdom Eastink, (1977), whereby every ambulance man when he sees the patient should be able to identify him, address,

locus of accident, time first seen, cause of accident, time and date examined. Any action taken as regards state of airway. And noting the condition of the patient on the way to the hospital whether level of consciousness is deteriorating or improving.

Since the greater number of hospital deaths occurred within few hours following admission, which are almost similar findings to ones seen by Hoffman (1976) whose deaths occured within 6 hours and more than half within 24 hours. Therefore the fundamental principles of maintenance of airway and adequate ventilation along with control and adequate replacement of blood loss should be adhered to vigorously.

Following admission of the patient to the emergency Casualty department, the resuscitation over all assessment, diagnosis and specific therapy should be done in a team work particularly the anaesthetist and accident surgeon. It is vital to exclude extra-cranial cause of coma especially with multiple injuries and history of ingestion of alcohol. (Lewik, 1976), demonstrated that three killing factors following severe head injury are intracranial haematoma, Cerebral hypoxia and increase in brain bulk. In Muhimbili cases intracranial haematoma was leading. But when the patient has a patent airway there is reduction of mortality.

Since the Muhimbili Casualty is not that vigorous there is a need for improvement. In future the department of Anaesthesiology

should provide one anaesthetist assistant who should be staying at the Casualty with all the necessary equipment for immediate resuscitation. This will include the resuscitation of other medical or surgical conditions. Or another alternative would be that all Casualty Officers should at least rotate in the department of anaesthesiology either in the main theatre or Intensive Care Unit and learn the art of resuscitation. At least they should be able to intubate and do cardiopulmonary resuscitation for the minimum of 3 months. This won't cost anything. What we need is cooperation between the two departments. Another point of consideration is that the Casualty should have a small X-ray unit in the vicinity of the Casualty so that if anything urgent arises there is no time delay to be X-rayed. For the time being any patient needing X-ray should be escorted by a trained personnel who can handle the problems of airway. Finally the neurosurgeon or for that matter all Consultants should stay within the hospital compound keeping in mind the existing poor communication.

CONCLUSION:

There is a need to improve general management of head injuries at Muhimbili Medical Centre.

REFERENCES

Adeloye, A., Olumide, A.A. (1976)
Pattern of Acute head injuries in Ibadan
Int. Surg. 61 Pg 478 - 80.

Artru, A.A., Michenfelder J.D.,
Cerebral protective, metabolic and vascular effects
of phenytoin.
Stroke 11: 377 - 382 (1980)

Aubrey, D.A., and Burns, G.P.:
Topically administered prednisolone and Antral
phase of gastric secretion.
Arch. Surg. 105: 448 - 453, 1972

Bakay, L. (1967).
Basic aspects of brain tumour localisation by
radioactive substances.
J. Neurosurg. 27, 239

Becker, D.P.; Vries, J.K. (1972)
Intracranial Pressure: Experimental and Clinical
Aspects.
(edited by Brock, M. Dietz, H.) Springer Verlag.
Berlin P. 309.

Ballinger, W.F.;
Manual of surgical Nutrition, Philadelphia, 1975.
W.B. Saunders Co.

Beckman, D.L. and Bean, J.W. (1975)
Pulmonary pressure volume changes attending head injury.
J. appl. physiol; 29/5, 631

Brown, F.D. Johns, L., Crockard, H.A., Mullan, S (1979)
In Neural Trauma (edited by Popp, A.J. et al) Raven Press:
New York pg. 281

Brown, F.K.:
Cardiovascular effects of acutely raised intracranial pressure
AM. J. physiol. 185: 510 - 514, (1956).

Brock, M. (1971)
Cerebral blood flow and intracranial pressure changes
associated with brain hypoxia. In Brain hypoxia.
ed. J.B. Brierley and B.S. Meldrum) p. 14: London:
William Heineman Medical Books Ltd.

Bruce, D.A. (1980).
In Management of severe head injury.
Edited by Cottrell, J.E; and Turndorf, H.)
The C.V. Mosby Company.
p. 191 - 208.

Bryce-Smith, R. (1950)
The management of head injuries from the Anaesthetist's
point of view.
Brit. Med. J., 2, 322.

Campbell, E.J.M., and Howell, J.B.L. (1960).
Simple and rapid methods of estimating arterial and
mixed venous $PCO_2$.
Brit. Med. J., 1, 458

Casualty Registry (1982 - 1983) Personal communication.

Cottrell, J.E.; Robustelli, A; P. Post, K; (1977)
Anaesthesiology, 47, 28

Crockard, H.A.
Early management of head injuries. (1982)
Brit. Jour. of Hosp. Med. Vol. 27/6 p. 635

Cushing, H:

Peptic ulcers and the interbrain,
Surg. Gynaecol. Obstet. 55: 1 - 34, 1932.

Demopoulos H.B. Flamm E. Seligman M.L. et al:

Molecular pathology of lipids in CNS membranes.
Oxygen and physiologic Function.
Edited by F.F. Jobsis. Dallas, Texas.
Professional Information Library, 1977, pp 491-508

Ducker, T.B. (1968)

Increased intracranial pressure and pulmonary oedema.
Clinical study of 11 cases.
J. Neurosurg., 28, 212.

Eastin, K. (1977)

Rescue emergency Care.
p. 75. London: William Heinemann Medical Books Ltd.

Ellis, H; Calne, R.Y. (1976)

In shock.
Lecture Notes on General Surgery.
Blackwell Scientific publications 4th Ed. (1976) pg. 3

Fieschi, C., Battistini, N., Beduschi, A. Rosanda, M. (1974)

Regional cerebral blood flow and intraventricular
pressure in acute head injuries.
Neurosurg. Psychiatr., 37, 1378

Fishman, R.A. (1975)

New England Journal of Medicine 293, 706

Ganong, W.F., (1979)

Review of Medical Physiology 9th Edition. pg 488.
Lange Medical publications.

Glenn, F., and Grafe, W.R.
Surgical Complications of adrenal steroid therapy
Ann. Surg. 165: 1023 - 1034, 1967.

Gordon, E. (1975).
A basis and practice of Neuroanaesthesia. On the management of acute head injuries, the non-operative approach
p. 249 - 253.
Amsterdam: Excerpta Medica.

Graham, D.I., and Adams, J.H. (1971)
Ischaemic damage in fatal head injuries.
Lancet, 1, 265.

Gray, C.G., Nunn, J.F. Utting, J.E. (1980)
4th Ed. in General Anaesthesia Vol. II.
Management of head injuries p.g. 1717
Butterworths and Co. Ltd.

Gutali, D.R.; (1963)
Acta neuropathologica 2, 451

Hart, S.M. (1965)
The anaesthetist and the early management of acute head injuries.
Brit. Med. J. 37, 189 - 190

Harrison, M.J.G., (1976).
The diagnosis of brain death
Brit. J. Hosp. Med; 10, 322 - 323.

Haque, I.U. (1964)
Morphological and Chemical studies in cerebral oedema,
J. Neuropathol. exp. Neurol; 23, 393.

Hirano, A., Backer, N.H., and Zimmerman, H.M. (1969).
Pathological alterations in the cerebral endothelial cell barrier to peroxidase.
Arch. Neurol; 20, 300

Hoffman, E. (1976).
Mortality and morbidity following road accidents.
Ann. R. Coll, Surg. Eng. 58, 233

Horton, J.M. (1975)
The immediate care of head injuries.
Anaesthesia Vol. 30, 212

Horton, J.M. (1976)
The anaesthetists contribution to the Care of head injuries.
Br. J. Anaesth. 48, 767.

Huang C.T. Cook, A.W. and Lyons, H.A. (1963)
Severe craniocerebral trauma and respiratory abnormalities.
Arch. Neurol (Clin.), 9, 545.

Hunter, A.R. (1964)
Neurological Anaesthesia.
Oxford Blackwell Scientific publications.

Illingworth, G., and Jennet, W.B. (1965)
The management of head injury.
Lancet 2, 511.

James E. Cottrell and Herman Turndoff in Anaesthesia and Neurosurgery.
Management of Head injury (edited by D.K.A. Bruce)
183 - 209. The C.V. Mosby Company 1980.

Jamieson, K.G., and Yelland, J.D.N.:
Surgically treated traumatic subdural hematomas,
J. Neurosurg. 37: 137, 1972

Jennet, B. and Corlin, J. (1978).

Preventable factors after recent head injury.
Injury 10, 31 - 39

Jennet, B., Hesset C., (1981).

Brain death in Britain as reflected in renal donors.
Br. Med. J. 283:359 - 62

Jennet B. and MacMillan, R. (1981)

in Management of Intracranial Diseases
Br. Med. J. 282, 101

Gleare J. Wilson P.

Brain death in three Neurosurgical Units.
Br. Med. J. 1981; 282: 533 - 9

Korein J., 1980.

In Brain death
Anaesthesia and Neurosurgery edited by Cottrell and Turdorf. pg. 282 - 292.
The C.V. Mosby Company.

Lewin, W.S. (1959.

Planning for head injuries
Br. Med. J. 1, 131

Lewik, W.S. (1976).

Changing attitudes to the management of severe head injuries.
Br. Med. J. 2, 1234

Lillehei R.C; Dietzman R.H., Motsay G.J., Schultz L.S., Romero L.H., Backman C.B. (1972).

Geriatrics, July (1972), p. 76

Long, D.M. (1970).
Capillary Ultrastructure and the blood brain barrier in human malignant brain tumours.
J. Neurosurg: 32, 127.

Luisanda, A.A.,
Mechanism of neurogenic pulmonary oedema,
Am. J. Cardiol. 20: 66 - 68, 1967

Lweno, H. (1969). (Personal communication, Paper presented during annual Scientific Conference of Anaesthesiologists of East Africa, in Nairobi, Kenya.

Maciver, I.N.; Frew, I.J., and Matheson, J.G. (1958).
The role of respiratory insufficiency in the mortality of head injuries.
Lancet, 390.

Mackay, G.M. (1969).
Some features of traffic accidents.
Br. Med. J., 4, 799

Macmillan, R. (1968).
In management of Intracranial Disasters.
Br. Med. J. 282 - 101

Maire, F.W; and Patton, H.D.
Neural Structures involved in the genesis of preoptic pulmonary oedema, gastric erosions and behaviour changes,
Am. J. Physiol. 184: 345 - 350, 1976

Matjasko M.J.
Peripheral Sequale of acute head injury.
Anaesthesia and Neurosurgery (edited by J.E. Cattrell and H. Turndorf).
The C.V. Mosby Company 1980 p. 217

McDonald, A.S., Steel, B.J. and Bottonley, M.G.:
Treatment of Stress induced upper gastrointestinal haemorrhage with Metiamide,
Lancet 1: 68 - 70, 1976.

Mcdougall, B.R.D., Bailey, R.J., and Williams, R.:
$H_2$ receptor antagonists and antiacids in the prevention of acute gastrointestinal haemorrhage in fulminant hepatic failure.
Lancet 1: 612 - 619, 1977.

McDowall, D.G. (1976).
Monitoring the Brain.
Anaesthesiology, 45, 1976 p. 130

Michenfelder, J.D. (1982)
Cerebral anoxia
Anaesthesiology Review Course lecture notes
113/1 - 113/9

Miller, J.D., Becker, D.P., Ward, J.D., Sullivan, H.G., Adams, W.E., and Rosner, M.J. (1977).
Significance of intracranial hypertension in severe head injury.
J. Neurosurg. 47, 503 - 516.

Northfield, D.W.C. (1973)
In the surgery of the Central Nervous System.
Head Injury p. 815 - 819.
Blackwell Scientific publications.

Mwang'ombe, W.J.M. (1982).
Head injuries at Kisii District Hospital
Proc Surg. East Africa 2: 5: 86.

Pallis, C., (1983).
ABC of Brain Stem Death Prognostic Significance of a dead brain stem.
Br. Med. J. Jan. 1983 6359, 123

Pappius, H.M., Dayes, L.A. (1965).
Archives of Neurology, 13, 395.

Pollay, M. (1975).
Formation of C.S.F. Relation of studies of isolated choroid plexus to the standing gradient hypothesis.
J. Neurosurg., 42, 665.

Potter, J.M. (1965).
Emergency management of head injuries.
Br. Med. J., 2, 1477.

Prithvi Raj. P; Montgomery S.J., Bradley V.H.
Agents and Techniques. Clinical Anaesthesia for the Surgery of Trauma.
Ed. Giesecke, A.H. Vol. 11/2 P.41 (1976)
by F.A. Davis Company.

Rains, H.A.J., Ritchie, H.D., (1975)
Bailey and Loves Short practice of Surgery
London, H.K. Lewis
p. 384 - 388

Ropoport, S.I. (1976).
Blood Brain Barrier physiology and Medicine
Raven Press New York.
Definition of irreversible coma: Report of ad hoc Committee (Chairman, H.K. Beecher) of Harvard Medical School to examine definition of brain death.
J.A.M.A. 205: 85 - 88, 1968.

Reulen, H.J. and Schiilmann, K. (Editors) 1972

In Steroids and Brain Edema.

Springer, Verlag, Berlin p. 203

Rossanda, M., Di Gingno, G., Corona, S., Bettinazzi, N. and Mangione, G. (1966)

Oxygen supply to the brain and respirator treatment in severe comatose states.

Acta Anaesth. Scand. 23/3 766.

Rosomoff, H.L. (1962).

J. Neurosurg. 19, 859.

Rottenberg D.A.; Posner J.B.,

Intracranial pressure control in Anaesthesia and

Neurosurgery edited by

J.E. Cottrell and H. Turndorf. p. 107

Mosby Company 1980

Rowbotham, G.F. (1965).

Acute injuries of the Head (4th Ed.)

Edinburgh: Livingstone.

Samuels, S.I. (1980).

Anaesthesia for Supratontorid tumour.

In Anaesthesia and Neurosurgery

(editors Cottrell, J.E., Turndorf, H.) p. 156

Reulen, H.J., Hadjidimos, A., and Hase, U. (1973)

Steroids in the treatment of brain Oedema;

in Advances in Neurosurgery I

(editors K. Schurmann, M. Brock, H.J. Reuben and

D. Voth). p. 92.

Berlin, Heidelberg, New York: Springer.

Robbins, (1974).

Pathologic basis of disease pg. 1525 - 1527

W.B. Saunders Company

Skillman, J.J., and Silen, W.
Acute gastroduodenal "stress" ulceration: barrier disruption of Varied pathogenesis?
Gastroenterology 59: 478 - 482, 1970.

Teasdale, G. (1976).
Assessment of head injuries
Brit. J. Anaesth. 48, 762 - 763

Uek, K., Takeudi, K., and Katsurada, K.
Clinical study of brain death; Presentation no. 286, Tokyo, 1973, Fifth International Congress of Neurological surgery.

Ulin, A.W. and Rosomoff, H.L. (1953).
Management of airway in acute head injury.
Arch. Surg. 67, 756.

Utting J.E. (1980).
Hypocapania, in General Anaesthesia,
4th ed, p. 461 London. Butterworths.

Watts, C.G., and Clan, K.,
Gastric acidity in the comatose patient,
J. Neurosurg. 30: 107 - 109, 1969.

Wilson, R.F.
Shock and Resuscitation following trauma in Emergency Medical Services and Traumatology.
Edited by R. Adams Cowley.
Papers presented at the U.S.A. Bicentennial Emergency Medical Services and Traumatology Conference and Exhibition May 9 - 12, 1976. Baltimore, Maryland.
p. 220 - 221.

Wise, B.L. and Chater, N. (1961).
Arch, Neurol. 4, 200

Wolfson, L; Sakurado, O; Sokoloff, L:
Effect of V- butyrolactone on local cerebral glucose utilization in the rat.
J. Neurochem. 29: 777 - 783, 1977