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Munthree, Nash

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Analysis of the neuro-physical benefits of surgically elevating depressed skull fractures in patients who have sustained non-missile traumatic head injuries at the Chris Hani Baragwanath Academic Hospital.

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A research report submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in fulfilment of the requirements for the degree of Master of Medicine in Neurosurgery

August 2018

Johannesburg

Declaration

I, Nash Munthree, declare that this dissertation is my own work. It is being submitted for the Degree of Master of Medicine in the branch of Neurosurgery. It is being submitted at the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at this or any other University.

Signature: …….

……..……day of……..…..…20……in……..……

Dedication

Dedicated to God.

Dedicated to my family Rajen, Maga, Leo and Nadhiya.

Acknowledgements

Neurosurgical consultants Dr J Ouma and Dr S Gowan for their advice and guidance.

The past and present staff from the Departments of Neurosurgery and Anaesthesia at the Chris Hani Baragwanath Academic Hospital

Abstract

This study reports on 30 patients who presented with traumatic (non missile) depressed skull fractures with resultant neuro-physical deficits admitted to the neurosurgical unit at the Chris Hani Baragwanath Academic Hospital for treatment. The 30 patients included in this study underwent formal surgical elevation and debridement of their fractures within 48 hours of injury at the Chris Hani Baragwanath Academic Hospital theatre. The surgical procedures were undertaken by five neurosurgical registrars all with similar neurosurgical experience within our department.

The total number of 30 patients included 2 subsets of patients. Group A (n=11) being patients with isolated depressed skull fractures and no other intracranial injuries. Group B (n=19) included patients with depressed skull fractures and concomitant intracranial injuries.

The mechanism of injury varied amongst patients. Twenty-two patients were assaulted. Six patients were involved in motor vehicle accidents (MVA). Two patients were involved in pedestrian vehicle accidents (PVA).

Clinically, 11 patients had compound injuries and 19 patients had closed injuries. Seven patients had dural tears noticed at the time of operation. All patients presented with neuro-physical deficits consistent with the site of injury on the skull vault.

All patients presented with the depth of depression of the fracture greater than the width of the skull table on radiological imaging. Nineteen patients presented with associated bleeds namely, extradural hematomas (n=7), subdural hematomas (n=4), cerebral contusions (n=5) and intracerebral haemorrhages (n=4).

A pre- and post-operative comparison of the neuro-physical deficits of the enrolled patients was made. Radiological and clinical factors associated with the injury have also been assessed for a possible causal relationship to clinical outcomes.

Analyses of the data revealed that post-operatively, patients with isolated skull fractures were more likely to have improvements in motor function than patients with depressed skull fractures with an associated haemorrhage (p value = 0.21). The degree of improvement in motor function based objectively on the Medical Research Council (MRC) motor scale was also higher in the group of patients with isolated skull fracture as compared to the group of patients with depressed skull fractures with an associated haemorrhage (p value = 0.30).

Younger age (p value = 0.12), shorter time to surgery (p value = 0.66) and open injuries (p value = 0.45) were regarded as good prognostic markers for post-operative improvements in motor function.

The results indicated a predilection for post-operative functional motor improvements in the upper limb as opposed to the lower limb. The average degree of motor neurological improvement as measured by the MRC motor scale was slightly greater in the upper limb by 0.86 points as compared to the lower limb group (p value = 0.07).

List of Figures Page

1) Coronal section through the scalp, skull and brain illustrating the relationship of the different structures at the superior sagittal sinus	2
2) Series of events occurring during signal transmission at the level of the synaptic cleft	10
3) Indications/criteria for CT in patients presenting to the emergency department following minor head injuries	19
4) Return to play guidelines in patients who have sustained a concussion	21
5) Box Plot graph illustrating age distribution in the groups of patients with isolated skull fractures and those with a skull fracture with an associated intracranial haemorrhage.	43

List of Tables Page

1) Marshall classification	6
2) The Rotterdam CT scoring system	7
3) Mortality at 6 months post injury using the Rotterdam CT scoring system	7
4) Evaluation of demographic, radiological and clinical data in relation to the improvement in motor neurological function of patients after 6 months	39
5) Group A including patients with post traumatic isolated depressed skull fractures	40
6) Group B including patients with post traumatic depressed skull fractures with an associated underlying haematoma	41
7) The mean age distribution and standard deviation between patients with skull fractures and those with associated haematoma	43
8) The age distribution between those patients with improvement in motor function vs patients with no improvement in motor function	43
9) The frequency of gender within the total sample group	44
10) The frequency of gender distribution between the skull fracture and skull fracture with associated haematoma groups	44
11) The frequency of open versus closed injuries in the study sample	45
12) The distribution of patients with isolated skull fractures stratified by classification of skull fracture versus presence/absence of an improvement in motor function	45

List of Tables: Continued Page

13) The distribution of patients with skull fractures and associated haematomas stratified by classification of skull fracture versus presence/absence of an improvement in motor function	46
14) The distribution of time periods between time of injury and time of surgery	47
15) The comparison between patients with skull fractures only and those with skull fractures with associated haematomas versus time to surgery	47
16) The relationship between the improvement in patients motor function and time to surgery	48
17) The frequency of patients who presented with traumatic dural defects	49
18) The relationship between the frequency of traumatic dural defects and an improvement in motor neurological function, in the group of patients with skull fractures with an associated intracranial haematoma	49
19) The relationship between the frequency of traumatic dural defects and an improvement in motor neurological function, in the group of patients with isolated skull fractures	50
20) Mechanism of injury in the total patient sample	50
21 The relationship between mechanism of injury and improvements in motor function in the total sample of patients	51

List of Tables: Continued Page

22) The distribution of patients stratified by the presence of an isolated skull fracture vs a skull fracture with an associated intracranial haematoma, relative to improvements in their respective motor scores.	52
23) The comparison of mean number of points improved on the Medical Research Council (MRC) scale between the isolated skull fracture group and the skull fracture with associated haematoma group	53
24) The distribution of patients relative to the respective improvement in points on the MRC scale in the lower limb	54
25) The distribution of patients relative to the respective points improved on the MRC scale in the upper limb	55
26) Comparison between the average post-operative (6 months) MRC scale motor scores of the upper and lower limbs	56

List of Abbreviations

1) American College of Emergency Physicians (ACEP)
2) Adenosine diphosphate (ADP)
3) Adenosine triphosphatase (ATPase)
4) Adenosine triphosphate (ATP)
5) Blood brain barrier (BBB)
6) Central nervous system (CNS)
7) Cerebrospinal fluid (CSF)
8) Chris Hani Baragwanath Academic Hospital (CHBAH)
9) Computed tomography (CT)
10) Deoxyribose nucleic acid (DNA)
11) Diffuse axonal injuries (DAI)
12) Glasgow coma score (GCS)
13) Motor vehicle accident (MVA)
14) Medical research council (MRC)
15) Magnetic resonance imaging (MRI)
16) National Institute for Health and Care Excellence (NICE)
17) Nicotinamide adenine dinucleotide (NAD)
18) Pedestrian vehicle accident (PVA)
19) Post traumatic seizures (PTS)
20) Skull radiograph (SXR)
21) Traumatic brain injury (TBI)
22) Traumatic head injury (THI)
23) Versus (vs)

Table of contents Page

Declaration	ii
Dedication	iii
Acknowledgement	iv
Abstract	v
List of figures	vii
List of tables	viii
List of abbreviations	xi
Table of contents	xii
1) Literature Review	
1.1) Introduction	1
1.2) Relevant anatomy	2
1.3) Epidemiology	4
1.4) Classification	5
1.5) Biomechanical basis for THI	8
1.6) Pathology	10
1.6.1) Neurochemical Changes in THI	10
1.6.2) Alteration in cerebral glucose metabolism in THI	12
1.6.3) The role of free radicals in THI	13
1.6.4) The role of mitochondria in THI	14
1.7) Complications	14

Table of contents: continued Page

1.8) Management	17
1.8.1) Investigations	17
1.8.2) Prevention	20
1.8.3) Medical therapy	22
1.8.3.1) Antibiotics	22
1.8.3.2) Antiepileptics	24
1.8.4) Surgical indications	25
1.8.5) Surgical technique	26
1.8.6) Relevant literature in the South African landscape	30
1.8.7) Rehabilitation	31
1.9) Hypothesis	32
1.10) Aims	32
1.11) Objectives	32
2. Methods	
2.1) Design	33
2.2) Site of study	33
2.3) Study population	33
2.4) Sampling	33
2.4.1) Sampling size including statistical rationale	33

Table of contents: continued Page

2.4.2) Inclusion criteria	34
2.4.3) Exclusion criteria	34
2.5) Measuring tool or Instrument	34
2.6) Data Collection	35
2.7) Sources of Bias	36
2.8) Confounder	36
2.9) Variables	37
2.10) Data analysis	37
2.11) Ethics	38
3. Results	
3.1) Records reviewed	38
3.2) Age	42
3.3) Gender	44
3.4) Open versus closed injuries	45
3.5) Time of injury to time of surgery	46
3.6) Dural tear	48
3.7) Mechanism of injury	50
3.8) Isolated skull fracture versus skull fracture with associated bleed	52
3.9) Upper limb versus lower limb	54

Table of contents: continued Page

4. Discussion	
4.1) Study sample	57
4.2) Age	58
4.3) Gender	59
4.4) Open versus closed injury	59
4.5) Time of injury to time of surgery	60
4.6) Dural tear	61
4.7) Mechanism of injury	62
4.8) Isolated skull fracture versus skull fracture with associated bleed	62
4.9) Upper limb versus lower limb	63
4.10) Limitations	63
4.11) Conclusion	64
5) References	66
6) Appendix	72

1.1) Introduction

The term Traumatic Brain Injury (TBI) has been used as the preferred nomenclature in the literature to describe post traumatic injuries of the brain. Conformity in the literature with regards to injuries of the surrounding structures of the brain is however ambiguous. In view of this fact, for the purposes of this study the term Traumatic Head Injury (THI) has been adopted as it encompasses post traumatic injuries to the brain and the relevant surrounding structures such as the skull and scalp. THI have traditionally accounted for a significant proportion of mortality and morbidity in South Africa (1). The clinical deficits incurred by patients after such injuries may include neuro-cognitive, neuropsychiatric and neuro-physical deficits. Patients who have sustained depressed skull fractures form just one subset of the spectrum of pathology included under THI. Chris Hani Baragwanath Academic Hospital (CHBAH) situated in the south of Johannesburg shares similar characteristics with national statistics in that our neurosurgical unit is tasked with the management of several patients with THI on a daily basis. It is often a challenge to expedite operations on patients with depressed skull fractures due to a large backlog of caseloads and the lack of resources available in the public sector. As a result, patients with depressed skull fractures are often managed conservatively, deferring elevation of the fracture formally in theatre. This study focuses on patients who have sustained depressed skull fractures with resultant neuro-physical deficits and aims to further guide management decisions.

1.2) Relevant anatomy

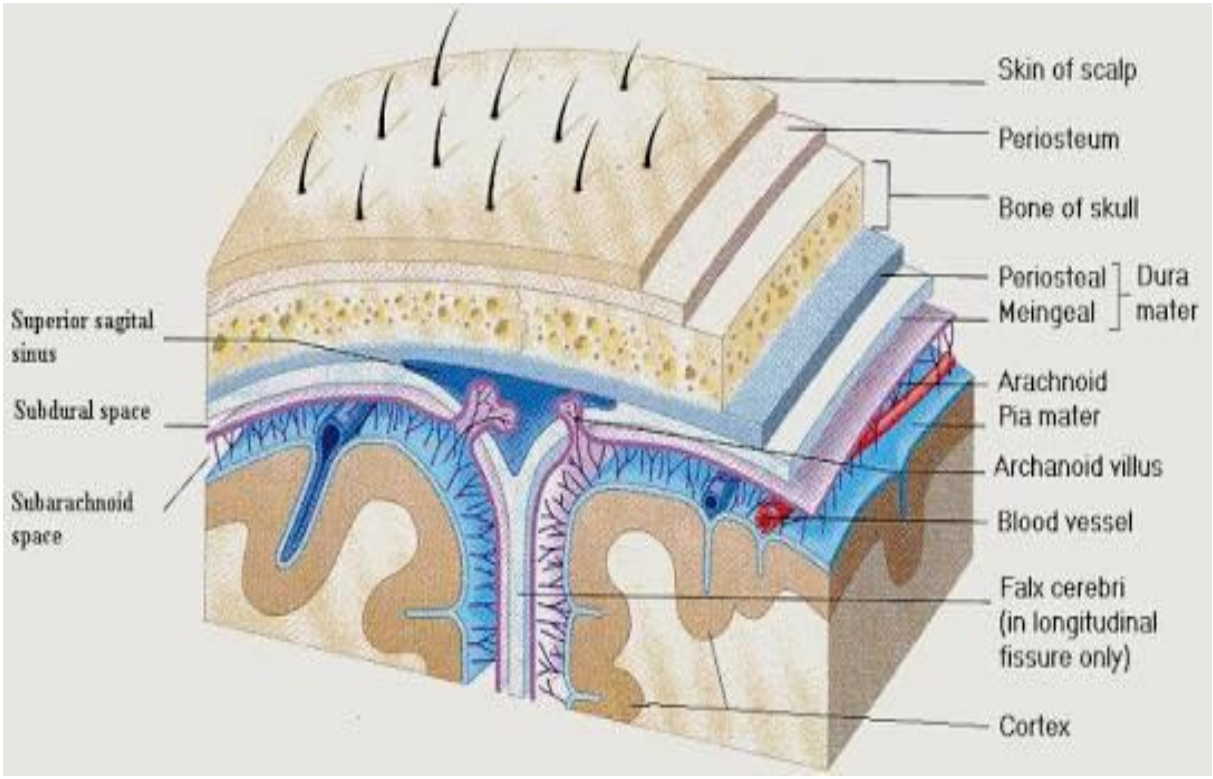


Figure 1: Coronal section through the scalp, skull and brain illustrating the relationship of the different structures at the superior sagittal sinus (2).

Figure 1 illustrates the adjacent relationship of the skull to various intracranial and extracranial structures. The skull is surrounded by the scalp extracranially, which is made up of five different layers. The layers from outermost to innermost include the skin, connective tissue, aponeurosis, loose areola tissue and periosteum respectively (3).

The skull is an osseous structure which serves to enclose and protect the brain as its primary function. It is made up of several membranous bones which, with age, fuse along suture lines to form a single rigid structure. In adulthood the skull is comprised of an outer and inner table composed of compact bone. Between these layers of compact bone is the diploe composed of cancellous bone with red bone marrow (4).

The skull contours around the brain and is separated by the meninges from the brain. There are three meningeal layers. The dura is the outermost layer composed of an outer periosteal layer and the inner meningeal layer. Below the dura is the thin translucent arachnoid layer which is a supporting layer enclosing vascular structures and cerebrospinal fluid. The inner most layer is the pia which is adherent to the brain (4).

Physiologically the nervous system can be divided into the somatic nervous system and the autonomic nervous system (4). The somatic nervous system can be further divided into the motor and sensory system. The purpose of this study is mainly to focus on the neuro-physical deficits patients incur as a result of their injury. The relevant structures that combine to form this part of the motor system are called the corticospinal/pyramidal tract.

The pyramidal tract is a tract that originates in the pre-central gyrus of the frontal lobe bilaterally. The motor impulses originate from the giant pyramidal cells or Betz cells located in the pre-central gyrus. There is an anatomic representation of the various motor structures supplied in the pre-central gyrus which is referred to as the motor homunculus. The upper motor neuron then conducts the impulse via its axons through the corona radiata and posterior limb of the internal capsule. The axons thereafter descend passing through the midbrain, pons and medulla.

The lower medulla is the region where approximately eighty-five percent of the axons decussate and then continue to descend in the white matter of the lateral funiculus of the spinal cord on the contralateral side. Approximately fifteen percent of axons do not decussate and continue to descend on the ipsilateral side of the spinal cord (4).

The descending axons within the corticospinal tract terminate at different levels within the spinal cord depending on the area of supply. The axons terminate in the anterior horn of the grey matter of the spinal cord (4). These axons then synapse with anterior horn cells of the lower motor neuron. The lower motor neuron then goes on to supply the motor end plate within the respective muscle groups.

1.3) Epidemiology

Statistics for THI in South Africa are currently lacking. A proposal for a THI data bank was made several years prior by the then national Minister of Health but as yet, nothing has come to pass. As a result, statistics acquired for THI in South Africa are often based on unreliable and incomplete medical records from state hospitals (1).

A study by Nell had been conducted in the Johannesburg area. The study found the incidence of THI to be 316/100 000 annually with a male to female predisposition of 4:1 (5).

In the United States the incidence of THI is 506.4 per 100 000. This equates to 1.5 million people suffering from traumatic brain injuries each year (1).

Naidoo, a South African author noted that the major risk factors for a THI in South Africa were low socioeconomic status, extremes of age and male gender. Naidoo also noted that the mechanism of injury that accounted most for THI in South Africa was interpersonal violence (1).

1.4) Classification

Skull fractures are generally divided into fractures of the skull vault and fractures of the skull base. The focus of this study was aimed at fractures of the skull vault. A skull fracture of the skull vault may be described as either open or closed depending on whether the integrity of the scalp overlying the skull fracture has been maintained (6). Fractures of the skull vault are then further described based on their appearance as linear, depressed or comminuted (7).

A skull fracture in the setting of trauma should be regarded as a risk factor for underlying intracranial lesions such as brain contusions and hematomas (6, 8, 9). Very rarely do patients sustain skull fractures in isolation. Thus an understanding of the spectrum of disease within the broader umbrella of THI is essential.

A number of different methods have been described to classify THI. One such method is a description of the mechanism of injury i.e. closed, penetrating, crush and blast injuries (2). In recent literature, blast injuries have been regarded as separate entities due to the different neuro-pathological mechanisms involved. Clinically THI may be classified into five categories: skull fractures, focal injuries, diffuse brain injuries, penetrating injuries and blast injuries (2). Clinical scoring systems have been used to assess the severity of injury. One such score is the Glasgow Coma Score (GCS) (10). There are three components to the score including a motor, verbal and eye response. The score ranges from 3 to 15. Teasdale proposed a score of 13-15 as a mild THI, 9-13 as a moderate THI, 3-8 as a severe THI (10).

There have also been severity scoring systems described based on radiological criteria for THI. One such score was proposed by Marshall and is highlighted in Table 1 (11). Patients were classified into one of six categories based on increasing severity. Two major radiological criteria were used namely the severity of brain swelling and the presence and size of mass lesions.

Table 1: Marshall Classification of radiological predictors in patients with THI (11)

Diffuse injury I	No visible intracranial pathology on CT scan
Diffuse injury II	Cisterns are present with midline shift of 0-5mm and/or lesions densities present. No high or mixed density lesion >25cm^3 may include bone fragments and foreign bodies
Diffuse injury III (swelling)	Cisterns compressed or absent with midline shift of 0-5mm, no high or mixed density lesion >25cm^3
Diffuse injury IV (shift)	Midline shift >5mm, no high or mixed density lesion >25cm^3
Evacuated mass lesion	Any surgically evacuated lesion
Non evacuated mass lesion	High or mixed density lesion >25cm^3 not surgically evacuated

A more recent radiological scoring system mentioned in the literature is the Rotterdam CT Score as highlighted in Table 2 (12). Published in 2006, it appears to be the more favoured of the two scoring systems. Table 3 serves as an objective and useful tool in prognosticating mortality in patients who have sustained THI (12).

Table 2: The Rotterdam CT scoring system for THI (12).

Criterion	**Points**
Basal cisterns	
Normal	0
Compressed	1
Absent	2
Midline Shift	
No shift or <5mm	0
Shift >5mm	1
Epidural Mass Lesion	
Absent	0
Present	1
Intraventricular haemorrhage or traumatic subarachnoid haemorrhage	
Absent	0
Present	1

Table 3: Mortality at 6 months post injury using the Rotterdam CT scoring system (12)

Score	Mortality (%)
1	0 %
2	7 %
3	16 %
4	26 %
5	53 %
6	61 %

1.5) Biomechanical basis for THI

A THI is the culmination of the response of the brain and surrounding tissues to the external forces applied to it (13).

Traumatic loads can be applied to the skull in either a static or dynamic fashion. Static loading of a force to the skull is an uncommon occurrence and is used to describe a load which is applied to the skull over a long period of time. Examples of these injuries include squeezing or crushing of the skull as seen in building collapses or machinery accidents. Common injuries incurred after such static forces have been applied include skull vault/base fractures (14, 15).

Dynamic loading is the more commonly encountered loading to the head that is seen in clinical practice. Dynamic loads refer to a force or forces applied to the head rapidly over a short period of time. Dynamic loads are further divided into two types namely, impulsive or impact loads (14, 15). Impulsive loads refer to forces that indirectly set the head into motion by applying a force to other parts of the body. An example of this type of injury is seen in motor vehicle accidents (MVA) when a passenger with a restrained torso and unrestrained head is set violently into motion. The resulting inertial forces cause the brain to move within the skull. The interaction of the brain with the inner structures of the skull during this inertial movement can result in gliding contusions at the skull base as well as coup and contra coup injuries at the brain surface (14, 15).

Impact loading is slightly more complex in that injuries incurred may involve a combination of contact forces and inertial forces. The degree of injury produced by the contact force is based on the magnitude of the force applied, the size of the impact area over which this force is applied and the direction at which this force is applied. The impact loads may cause local tissue injury at the site of contact as well as injury at a distant site due to shockwave energy transfer through the underlying tissue (14, 15).

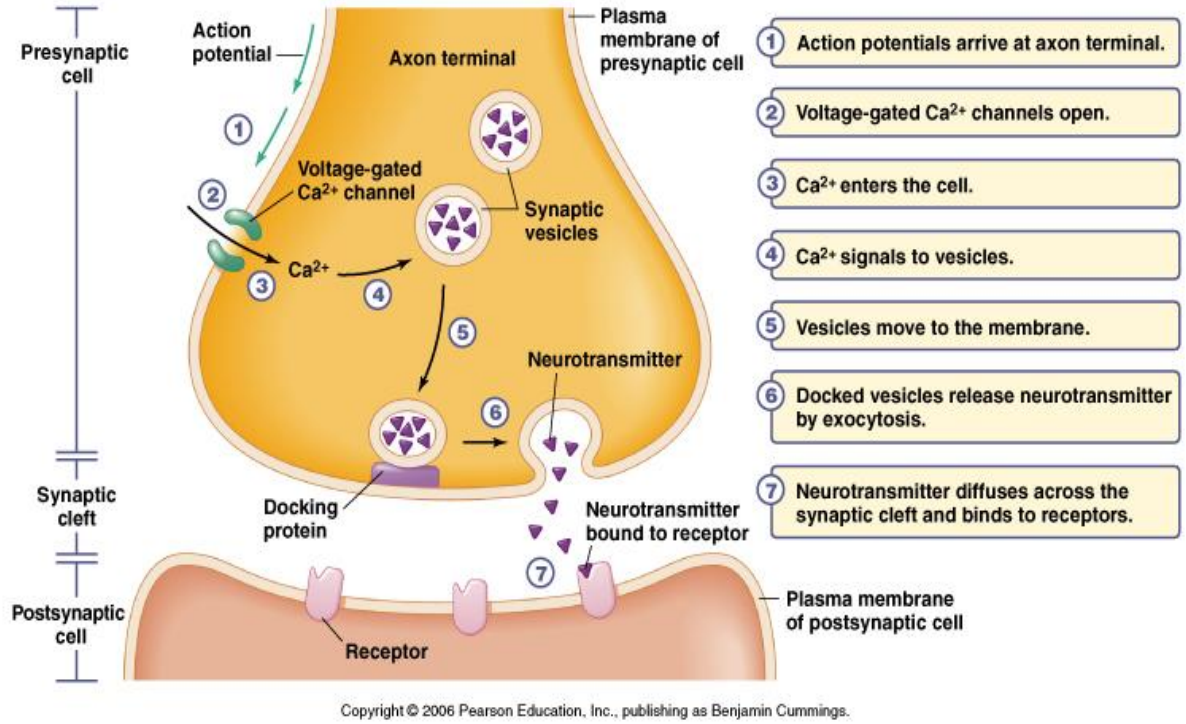
Based on the biomechanical stresses applied to the brain and surrounding structures, the above discussion helps to objectively describe injuries and postulate the nature of injuries incurred.

Ultimately however the true nature and degree of injury depends on the tissue involved and its ability to resist the strain and distortion from the force applied. Tissue injury results if the force overcomes the ability of said tissues to resist this strain and distortion. Various tissues demonstrate different properties and responses to such strain. Bone for example has a higher strain threshold than brain parenchyma. Tissue strain may be applied in different fashions namely compressive, tensile, shear and dilatational (16, 17).

1.6) Pathology

1.6.1) Neurochemical Changes in THI

The successful transmission of a signal from the presynaptic neuron to the postsynaptic neuron requires a series of specific steps to occur within controlled homeostatic conditions. These steps are highlighted in Figure 2 (18). Ionic changes across the bilipid membrane are meticulously regulated by energy-dependent sodium-potassium (Na^+-K^+) adenosine triphosphatase (ATPase) pumps, which maintain the membrane potential between −40 and −70 mV (19). Depolarisation occurs when an action potential surpasses this threshold within the presynaptic membrane. Neurotransmitters are then released from the presynaptic membrane into the synaptic cleft to be detected by the post synaptic membrane. The neurotransmitter may then influence the postsynaptic membrane in an excitatory or inhibitory manner.



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Figure 2: Series of events occurring during signal transmission at the level of the synaptic cleft (18).

THI may induce transient cell membrane disruptions which causes a redistribution of ions and neurotransmitters. This re-distribution alters the membrane potential of the presynaptic and post synaptic membrane. In the hyperacute setting (<1 hour) after a THI, massive amounts of glutamate are released from the presynaptic terminal into the synaptic cleft. The glutamate then causes an alteration in ionic equilibrium in the post synaptic membrane leading to loss of neuronal function (20).

Potassium release by neurons after a THI was also noted as a key determinate in the severity of cell dysfunction. The rise in extracellular potassium was directly proportional to the severity of injury incurred. Kawamata et al (21) noted during experiments with microdialysis of the brain that extracellular potassium levels had risen by 1.4 to 5.9 times the normal value depending on the severity of injury. Tetrodotoxin is a neurotoxin that prevents neuronal cell firing. Local administration of tetrodotoxin subsequently reduced the potassium surge after a THI suggesting that the rise in potassium was related to neuronal cell firing. Kynurenic acid, an antagonist of excitatory amino acids was also noted to attenuate the potassium surge after a THI. This suggests that the potassium surge is dependent on excitatory neurotransmitters. In order for neuronal function to return, ionic equilibrium needed to be restored. Re-establishment of ionic equilibrium is an energy dependant process which requires adenosine triphosphate (ATP) (21).

1.6.2) Alteration in cerebral glucose metabolism in THI

Glucose is the primary energy source metabolized by the adult brain. Glucose is used during the process of glycolysis and oxidative phophorylation to form ATP, water and carbon dioxide. ATP is key to cell function and assists in maintaining cell membrane integrity, ionic homeostasis through the ATPase membrane pump system as well as being the energy source involved in nerve impulse transmission (22).

Fludeoxyglucose and [14C] 2-deoxyglucose are chemical isotopes used in radiological imaging to measure glucose uptake in the brain. These isotopes have been used to show an increase in glucose uptake followed by a prolonged period of glucose metabolic depression in patients with THI (23). Hovda et al noted during studies in rats that there was a transient increase in the cerebral metabolism of glucose. This occurred in the first 30 minutes following a THI (24). It is hypothesized that this initial increase in cerebral glucose metabolism is due to the increased energy demand by the cells to restore ionic balance and neuronal membrane potential (24, 25). Bergsneider noted that this phenomenon was not limited to the first 30 minutes following injury but lasted up to 8 days after the THI (26).

Cerebral glucose metabolic rates within the thalamus, brain stem and cerebellum correlated positively with the level of consciousness as measured by the GCS (27). Thomas et al noted that there was a definite age related correlation in the duration of the cerebral glucose metabolic depression (28). Metabolic rates in juvenile rats returned to normal within 3 days as opposed to adult rats which returned to normal within 10 days (28). The rate of cerebral glucose metabolism in subcortical structures returned to normal within 3 days on average whereas those of cortical structures were 5 days on average (29).

1.6.3) The role of free radicals in THI

A rise in free radical formation has been noticed in patients with THI. Free radicals are molecules with unpaired electrons and are highly reactive. Free radicals are a contributing factor to the metabolic crisis experienced by patients with THI. In an attempt to gain electrons from surrounding molecules within local tissues, structures such as cell membranes, proteins and deoxyribose nucleic acid (DNA) may be damaged (22).

There are two types of free radicals. The first type is the reactive oxygen species and the second type is the reactive nitrogen species. During normal levels of cerebral metabolism antioxidant and scavenging systems are able to cope with the rate of free radical production (22). However, in patients with THI, production of reactive oxygen species overwhelms aforementioned systems and results in oxidative damage (30-32). Intracellular accumulation of calcium noted in patients with THI increases the production of a number of enzymes. These enzymes include xanthine dehydrogenase, phospholipase A2 and nitric oxide synthase. The aforementioned enzymes are associated through their respective actions, with increased superoxide and nitrosonium production (33).

Free radical damage is also known to reduce ATP production through its activation of DNA repair enzymes (22). Poly-adenosine diphosphate (ADP) ribose polymerase is one such enzyme and is noted to rise during THI. Poly-ADP ribose polymerase consumes NAD^+ in the presence of DNA strand breaks, inhibiting glycolytic processes and reduces ATP production (34, 35).

1.6.4) The role of mitochondria in THI

The mitochondria of neuronal cells play a key role in the energy crisis following a THI. Intracellular accumulation of calcium and reactive oxygen species contributes significantly to mitochondrial dysfunction (22). Calcium influx overwhelms the mitochondrial buffer systems and leads to the collapse of the mitochondrial membrane potential (36).

One hypothesis by Brennan et al suggests that excessive glutamine stimulation activates poly-ADP ribose polymerase, causing depletion in NAD^+ stores which ultimately leads to cell death through the inactivation of normal metabolic processes (37). Another hypothesis suggests that excessive mitochondrial accumulation of calcium results in membrane potential compromise. This then initiates a series of events leading to increased free radical production, reduced NAD^+ levels and ultimately cell death (38, 39).

The exact order of events leading up to mitochondrial dysfunction in patients with THI remains controversial. There is however enough evidence to suggest that the end result following a THI is impaired mitochondrial function, reduced energy production and the potential for cell death in experimental models (40).

1.7) Complications

The nature and severity of complications associated with a depressed skull fracture are often related to the site of injury, its relationship to underlying structures as well as the nature of the injury i.e. open or closed. Depressed skull fractures may complicate up to 6% of THI and thus contributes significantly to the morbidity and mortality of patients (41).

The presence of a traumatic skull fracture is an important risk factor for intracranial lesions such as hematomas or contusions, unfavourable outcomes and death (7-9). Macpherson et al found that 603 of 850 patients with a cranial fracture had an associated intracranial lesion (contusion, hematoma) compared with 245 of 533 patients who did not have an associated intracranial lesion (42).

Cerebrospinal fluid (CSF) leak can be expected in 1-3% of all THI (43-45). CSF leaks may present acutely or in a delayed fashion. The sources for delayed presentation may include raised intracranial pressure acutely, cerebral oedema or a hematoma that may be obstructing flow of CSF through the traumatic fistula (46, 47). Ziu et al noted in their meta analysis of patients with CSF leaks that 50% of patients presented within 2 days of injury, 70% of patients presented within 1 week of injury and 99 % of patients presented within 3 months post injury (48).

Late onset epilepsy is another recognised complication of depressed skull fractures. Steinbok et al (49) noted an incidence of 15% for late onset epilepsy in their series of 111 patients.

Open skull fractures accounted for up to 90% of total skull fractures in some series (50). Open skull fractures are associated with infection rates of between 1.9% and 10.6% (51-54). Neurological sequela rates of up to 11% have been noted in patients with open skull fractures and secondary infective complications (55). Mortality rates from the subsequent infective sequela range have been reported from 1, 4% to as high as 19% in some series (41, 50, 54-56). This highlights the notion that open skull fractures should be treated with utmost priority as the potential for devastating complications are ever present.

Depressed skull fractures of the frontal region of the skull represent another subset of head injuries with particular complications to that region. There are particularly unique complications experienced by this subset of patients due to injury of the surrounding structures. These surrounding structures include the frontal air sinuses, anterior skull base, olfactory apparatus and frontal lobes. Depressed skull fractures involving the inner table of the frontal bones can lead to central nervous system (CNS) infections (meningitis, brain abscess, subdural empyema) in up to 15-30% of patients, mucocele cysts, chronic sinusitis, CSF leakage, late onset post traumatic epilepsy as well as disturbances of smell and taste (7, 41, 52, 54, 56-59).

1.8) Management

1.8.1) Investigations

Radiological evaluation of patients following a THI is an essential tool in evaluating both the type and extent of cranial injuries experienced (60). A fracture of the skull following trauma to the head was first described using a skull radiograph (SXR) in 1962. Later, a skull fracture was first described using computed tomography (CT) in 1983 (61). The preliminary examination of THI patients with a SXR has now been largely superseded by the use of CT (62).

In a study by Chawla et al of 42 patients with post traumatic skull fractures, autopsy examination found skull vault fractures in 28 patients (63). Retrospective review of radiological images for the same set of patients revealed that SXR detected 20 cases of skull vault fractures while CT had a higher yield of 25 cases with skull vault fractures (63). Looking at the above data in inverse, one may conclude that the missed diagnosis rate for skull fractures with SXR was 19, 1% and with CT, 7.1%.

Non contrast CT has become the gold standard for investigating patients in the acute stage following closed head injuries (64). Although magnetic resonance imaging (MRI) has been shown to be superior in detecting cerebral pathology e.g. in the cases of diffuse axonal injuries (DAI), CT is the preferred investigation in the management of acute closed head injuries owing to its cost effectiveness (64).

The criteria for imaging patients following a THI vary amongst institutions. There has been a number of algorithms developed to help guide clinicians in their decision to image patients who have sustained a THI. The algorithms that have come in to vogue recently and which are used to guide our practice at CHBAH has been the National Institute for Health and Care Excellence (NICE) algorithm, The New Orleans Criteria, Canadian CT Head Rules and the American College of Emergency Physicians (ACEP) Clinical Policy Recommendations (65). Figure 3 offers some detail into each of the above mentioned criteria (65).

Indications for Computed Tomography (CT) in Patients Presenting to the Emergency Department with Minor Head Injuries from the Canadian CT Head Rule, ACEP Recommendations, National Institute for Health and Clinical Excellence (NICE) Recommendations, and New Orleans Criteria

Canadian CT Head Rule

CT head scan is only required for patients with minor head injuries with any one of the following:

High risk (for neurological intervention)

- GCS score <15 at 2 h after injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture (hemotympanum, "raccoon" eyes, cerebrospinal fluid otorrhoea/ rhinorrhoea, Battle's sign)
- Vomiting ≥ 2 episodes
- Age ≥ 65 years

Medium risk (for brain injury on CT)

- Amnesia before impact > 30 min
- Dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 3 feet or 5 stairs)

Minor head injury is defined as witnessed loss of consciousness, definite amnesia, or witnessed disorientation in patients with a GCS score of 13–15. Not applicable if warfarin use or bleeding disorder or patient suffered a seizure before arrival to the ED.

ACEP Clinical Policy Recommendations

Which patients with mild TBI should have a noncontrast head CT scan in the ED?

Level A recommendations. A noncontrast head CT is indicated in head trauma patients with loss of consciousness or posttraumatic amnesia only if one or more of the following is present: headache, vomiting, age greater than 60 years, drug or alcohol intoxication, deficits in short-term memory, physical evidence of trauma above the clavicle, posttraumatic seizure, GCS score less than 15, focal neurologic deficit, or coagulopathy.

Level B recommendations. A noncontrast head CT should be considered in head trauma patients with no loss of consciousness or posttraumatic amnesia if there is a focal neurologic deficit, vomiting, severe headache, age 65 years or greater, physical signs of a basilar skull fracture, GCS score less than 15, coagulopathy, or a dangerous mechanism of injury.*

* Dangerous mechanism of injury includes ejection from a motor vehicle, a pedestrian struck, and a fall from a height of more than 3 feet or 5 stairs.

NICE Head Injury Guideline Recommendations

Request CT scan immediately if any of the following are present:

- GCS score < 15 at 2 h after injury
- Suspected open or depressed skull fracture
- Sign of fracture at skull base (hemotympanum, "panda" eyes, cerebrospinal fluid from ears or nose, Battle's sign)
- Posttraumatic seizure
- Focal neurological deficit
- > 1 episode of vomiting
- Amnesia of events > 30 minutes before impact

Request CT scan immediately if loss of consciousness or any amnesia and any of the following are present:

- Age ≥ 65 years
- Coagulopathy (history of bleeding, clotting disorder, current treatment with warfarin)
- Dangerous mechanism of injury (pedestrian or cyclist struck by motor vehicle, occupant ejected from motor vehicle, fall from height > 1 m or 5 stairs)

New Orleans Criteria

In patients with GCS of 15, loss of consciousness, and normal findings on a brief neurologic exam, indications for CT scan are as follows:

- Headache
- Vomiting
- Seizure
- Intoxication
- Short-term memory deficit
- Age > 60 years
- Injury above the clavicles

Figure 3: Indications/criteria for CT in patients presenting to the emergency department following minor head injuries (65).

1.8.2) Prevention

A THI can result in a significant psychological, physical and financial burden to the patient, the patient's family and a country's healthcare sector. In patients with severe THI, not only does the impact of acute management of the injury serve as a significant challenge, but most of these patients will require lifelong physical and cognitive therapy. Patients with mild THI also suffer considerable cognitive and somatic symptoms for a significant period of time. Caring for these patients thus requires a considerable work force and allocation of resources. This fact ultimately impacts negatively on national budgets and the work force in general. THI should be regarded as preventable conditions and the need to introduce preventative measures at a primary health care level should be of utmost importance (66).

Road traffic accidents account for a significant proportion of THI worldwide. Road traffic safety has thus become the primary focus for improvement in many countries. Safety campaigns highlighting the use of safety belts, roadworthiness of vehicles, use of helmets by motor cyclists and the avoidance of drinking and driving are just some of the key issues that have received focus during these campaigns (67).

Interpersonal violence is another factor responsible for many patients who sustain THI. Studies in third world countries have shown a direct relationship between these high levels of social violence and suboptimal education systems, unemployment and poor socioeconomic status (1). It stands to reason that improvements in education systems and empowering people through skills development will go a long way in dramatically reducing interpersonal violence.

Sports related THI has also received greater attention in recent times. This has been in part due to the release of an increasing literature bank regarding sports related THI. There is a growing concern for the long term effects of such injuries. Concussion, Post Concussive Syndrome and Chronic Traumatic Encephalopathy are just some of the disease entities receiving attention at the present moment. Parent education regarding contact sports and its possible effects are becoming standards of care in schools in first world countries. The use of protective gear such as helmets and padded head wear is now made mandatory in sports such as rugby, American football and boxing. Coaches and medical attendees at these sports events also receive training on how to assess these patients post injury and have guidelines on how to take action upon their findings. Figure 4 illustrates one such guideline which is the Return to Play Guidelines released by the American Academy of Paediatrics in managing patients who have sustained Concussions (68).



Figure 4: Return to play guidelines in patients who have sustained a Concussion (68).

1.8.3) Medical therapy

1.8.3.1) Antibiotics

The use of antibiotics in the setting of traumatic skull fractures has been researched previously. The decision to institute prophylactic vs therapeutic antibiotics, the choice of antibiotic, route of delivery and dosage schedule are just some of the concerns that need to be addressed when commencing a course of antibiotics. However many clinicians commence patients on antibiotics without adequate indications and in an incorrect manner. This reflex practice of inappropriate antibiotic use in patients with traumatic skull fractures can result in serious long term complications. One such complication is antibiotic resistance which is an increasing concern amongst health care institutions worldwide. The number of antibiotics within our armamentarium is slowly diminishing as the number of antibiotic resistant organisms increases. The need for stricter antibiotic stewardship is thus of paramount importance.

Surgical site infection has been classified by the Centres for Disease Control and Prevention. Wounds may be classified into clean, clean-contaminated, contaminated and dirty based on the location of the wound and presence of inflammation/infection (69). Depressed skull fractures with an overlying laceration have been classified as contaminated and an open depressed skull fracture with in-driven foreign material has been classified as dirty (69). Antibiotic use in contaminated and dirty wounds should be considered therapeutic (2). Patients with classified clean wounds have a less than <1% infection rate with the use of prophylactic antibiotics (70). The rate of infection increases to as high as 6-10% in patients with dirty wounds despite receiving therapeutic antibiotics (70).

Antibiotic use in clean and clean contaminated wounds may be considered prophylactic (2). There are a number of principles that one should adhere to when administrating prophylactic antibiotics to avoid antibiotic resistance. The choice of antibiotic should be targeted at the common pathogenic organisms occurring in the area of the wound. The antibiotic should be administered at a suitable time prior to surgery so as to achieve an adequate concentration at the tissue level. Antibiotics should be calculated according to the weight of the patient and may need a repeat administration in prolonged surgery to achieve adequate therapeutic tissue concentrations. Antibiotics should not be continued for more than a few hours post surgery (69).

Patients with skull fractures often present with septic complications which are exclusively extradural. However, this is not always the case and at times the infection extends below the dura to manifest as meningitis, empyema or a brain abscess. The overall goal of antibiotic treatment is to have an adequate delivery in terms of concentration of an antibiotic to the correct compartment (2).

It is at this point that a thorough understanding of the blood brain barrier (BBB) becomes essential. The BBB is predominantly formed by the tight junctions between the endothelial cells of cerebral vasculature. It serves as a barrier for protecting the CNS from harmful substances circulating through the blood and helps facilitate the transfer of important nutrients to the CNS (2). The ability of a substance to pass through the BBB depends on its molecular weight, ionisation, lipophilicity, pH, presence of inflammation, plasma protein binding and transport mechanisms (71). Increased lipophilicity, influx transporters and the presence of inflammation can increase the permeability of the BBB to antibiotics (72).

A number of methods may be employed to achieve the goal of adequate antibiotic delivery. The dose of antibiotic may be increased provided that the toxicity threshold is not exceeded. The choice of antibiotic may be adjusted to antibiotics which have better CNS penetration properties such as quinolones (73). The antibiotics may be delivered directly across the BBB by indwelling catheters such as lumbar drains or ventricular catheters (2).

1.8.3.2) Antiepileptics

An epileptic seizure is a transient occurrence of signs and symptoms due to abnormal excessive or asynchronous neuronal activity in the brain (74). In patients with THI, 2% with mild injuries will develop post traumatic seizures (PTS) and in patients with moderate to severe THI the risk may increase to between 8-50% (75, 76). Risk factors for developing PTS include alcoholism, younger age, penetrating injuries, intracranial haematoma, severity of injury, duration of posttraumatic amnesia, loss of consciousness, depressed skull fracture, lesion location and focal neurological deficits (76, 77). PTS may be classified as early PTS, with seizures occurring within 7 days of the injury or late PTS if seizures occur after seven days of the injury (78).

Early retrospective trials have shown that the use of phenytoin was effective in preventing early PTS (79, 80). The majority of studies do not support the use of prophylactic anticonvulsants for the prevention of late PTS (81). Routine seizure prophylaxis later than one week following a THI is therefore not recommended (82). If late PTS occurs patients should be managed at that point in accordance with standard measures for new onset seizures (82).

1.8.4) Surgical Indications

The topic of management of depressed skull fractures has been researched previously. There are however differing opinions in the literature regarding the optimal management of such patients. The traditional approach of surgically elevating fractures using certain radiological and clinical guidelines is well established.

The indication for surgical management following a traumatic depressed skull fracture is clear in the literature. The indications include evidence from radiological and clinical examination to suggest a:

- Cosmetic defect
- The depth of depression of the bone fragment greater than that of the skull table
- Severely contaminated wound
- Dural tear
- Underlying haemorrhage with significant mass effect
- Depressed fracture causing a significant defect of the inner table of the frontal sinuses
- Depressed fracture overlying a dural venous sinus, causing occlusion of the sinus (relative indication).

This has been the teaching traditionally (7).

1.8.5) Surgical Technique

Renghachary in his book, Neurosurgical Operative Atlas has succinctly and eloquently described the procedure of elevating post traumatic depressed skull fractures. These are the surgical principles which guide our units practice in managing such patients (83).

Prior to surgery the appropriate medical therapy should be administered to the patient, Prophylactic antibiotics as per local guidelines should be administered 30 minutes before the skin incision. In severely contaminated wounds relevant samples are taken during the operation and only when confirmed infection has occurred after the procedure should therapeutic antibiotics be started (83). Prophylactic antiepileptic drugs should be administered to achieve a therapeutic blood level range prior to surgery in patients at high risk for PTS.

The head of the patient is first secured using a Mayfield 3-point head holder. Care should be taken to avoid insertion of the clamp pins over the area associated with the depressed skull fracture. Fractures located in the frontal region favour patients being positioned in the supine position, while fractures located in the occipital area favour the patient being in the prone position and patients with fractures over the temporal or parietal region favour a lateral position while being operated. Draping over the involved area should accommodate for an extension of the incision if additional exposure is required during the exploration.

In the elevation of closed depressed fractures a suitable skin incision behind the hairline may be used to expose at least 2cm circumferentially of normal bone around the fracture site. A general understanding of the vascular supply of the scalp is important to plan your scalp flap. Accordingly, a balance should be maintained in order to prevent flap ischemia, while still having adequate exposure. A linear, curvilinear S incision or a scalp flap are some of the common scalp incisions made. In special cases where there is a large intracranial lesion that needs addressing as well e.g. hematomas, a large frontotemporal-parietal scalp flap is preferred.

In cases where there is a compound wound, wound edges are debrided until normal tissue is encountered. Incorporation of the compound wound into the final surgical incision is acceptable. One should be mindful of the amount of scalp tissue debrided throughout the procedure, with a view to closure of the scalp wound. Foreign material should be removed. The wound should be thoroughly irrigated with copious amounts of antibiotic infused saline. The overlying pericranium is then assessed from below the wound edges and below the scalp flap. If the pericranium is shredded or contaminated it should be debrided. If the pericranium is healthy and viable it should be harvested as a dural substitute in cases of dural defects. The pericranium assists in creating a water tight closure of the dura and acts as another layer for prevention of sepsis extending below the dura.

The skull fracture is then assessed after a 2cm circumferential margin of soft tissue has been cleared. Often because of the anatomy of the skull being composed of the cortical outer table, the diploe and the cortical inner table, fragments of the skull fracture may be loose or interlocked between other fragments of bone. The loose fragments are removed gently without injury to underlying structures. In cases where the bone fragments are interlocked, a burr hole is created away from and adjacent to the fracture site so that normal dura can be visualized. Care should be taken to avoid placing the burr hole overlying fragments of bone that may be angulated underneath away from the fracture site. The dura is then separated from the underlying bone around the burr hole using a Penfield No. 3 or No. 4 dissector. A slot craniectomy is then created around the fracture site using a 2 or 3mm Kerrison punch. Care should be taken to not insert the foot of the Kerrison punch below the dura in cases where the dura is torn. The bone fragments should become loose at which point they may be removed gently in the least traumatic direction to underlying vital structures. In cases of severe contamination or the fragments are too small for reconfiguration, they are discarded. The patient will then be brought back for an elective cranioplasty procedure. In other cases, the bone fragments and its relationship to each other should be noted for later reconfiguration. They should be placed in a 10% povidine iodine solution.

The dura is then inspected. Dural tack up sutures are inserted using 3-0 silk at the edges of the bone defect. If the dura is intact, do not open the dura unless there is an underlying lesion e.g. hematoma that needs evacuation. A simple dural laceration may be extended and underlying debris/hematomas flushed out with antibiotic infused saline. If the dura is shredded and not viable it should be debrided until viable dura is encountered.

Brain parenchyma is assessed. If pulped brain is visible it should be gently suctioned as it is no longer viable. A soft tipped No 18 angiocatheter may be used in the depths of cortical lacerations to irrigate and remove small bone fragments and debris minimizing cortical resection.

The dura is then sutured using 3-0 silk in a water tight fashion. Dural defects may be covered with pericranium and sutured in place. The decision to replace the bone fragments is based on the contamination of the wound and the size/comminution of the bone fragments. In cases with severe contamination and small comminuted fragments, provision should be made for a delayed cranioplasty procedure. Alternatively, the bone fragments may be wired together or, using a suitable plating system replaced at the primary setting.

Akram et al in a study of 51 cases found that the removal of bone fragments in compound skull fractures irrespective of the degree of contamination was not essential and that the replacement of the fragments at the primary operation was a suitable alternative (84).

A subgaleal Pencil drain may be used for 24 hours in cases where absolute hemostasis could not be achieved as in cases with bleeding fracture lines or contused scalp. After the debridement, the scalp should be closed in two layers. The galea is closed with absorbable suture. The cutaneous layer is closed with either skin clips or non-absorbable suture such as nylon. A delayed contrast CT scan with soft tissue and bony windows of the brain is performed after 4 to 5 days to look for evidence of delayed sepsis.

1.8.6) Relevant literature in the South African landscape

Studies have also been published supporting evidence for an alternative approach. One such recognised study was undertaken in the neurosurgical unit in Bloemfontein and is often used to guide management principals locally today. This study by CM van den Heever looked at 284 patients with traumatic non missile injuries and found that the group of patients managed conservatively (160 patients) with simple debridement methods and suturing in casualty compared more favourably than the group of patients (124 patients) that were managed with formal surgical elevation and debridement in theatre (51). Objective markers used to compare groups after follow up were: septic complications, CSF leaks and fatal outcomes. Criticisms of the aforementioned study include the absence of neurological deficits as an objective measure to compare groups of patients and the lack of differentiation between patients with isolated skull fractures and those with combined skull fractures and underlying pathology related to the trauma.

The literature focus thus far has been on treating the complications of depressed skull fractures namely cosmetic deformity, avoiding septic complications, preventing CSF leaks and evacuating underlying space occupying lesions following traumatic skull fractures (85). In so doing the pool of scientific literature serves to compare both modalities of management i.e. conservative vs surgical management and their respective outcomes.

This proposed study however focuses on a specific group of patients. The study includes patients with neuro-physical deficits as a result of the depressed skull fracture. The study aims to investigate the hypothesis that elevation of the isolated depressed skull fracture does in fact improve neurological deficits when the skull fracture causes mass effect on underlying neuronal structures.

A potential confounding factor in this study, as has been noted through anecdotal experience at our facility, is that in cases where a depressed skull fracture exists with underlying intracranial haemorrhage, clinical improvement of the neurological deficit may not occur despite successful elevation of the fracture. This is as a result of permanent damage to underlying neuronal structures rather than the mass effect of the depressed skull fracture.

Through this study we aim to objectively advocate for certain management principles based upon the results.

1.8.7) Rehabilitation

A THI may affect all aspects of a patient's life. A THI may result in physical, psychological and social deficits with far reaching consequences for both the patient and their family. The process of rehabilitation is based on the principle of education and requires the involvement of both the patient and family if any real long term significance is to occur

Modern rehabilitation is based around the concepts of impairment, disability and handicap as outlined by the World Health Organization in 1980 (86). There are three basic approaches that rehabilitation adopts in order to limit the disabilities incurred from a THI. The first is an approach to reduce disability. Secondly an approach is designed to acquire new skills and strategies that will reduce the impact of the disability. Lastly the aim is to adopt an approach that alters the environment, both physical and psychological, so that a given disability carries as little handicap as possible. To achieve such goals a multidisciplinary team including psychologists, physical therapists, occupational therapists, speech therapists etc., need to be involved. It is often a long term process with improvements and good outcomes occurring up

to 2 years after the initial injury. In view of this, adequate counselling and reasonable expectations should be highlighted at the very early stages of the rehabilitation process (86).

1.9) Hypothesis

Patients who have incurred post traumatic (non-missile) depressed skull fractures experience an improvement in their neuro-physical deficits after surgical elevation of the fracture.

1.10) Aims

1) To determine if patients with post traumatic depressed skull fractures with resultant neuro-physical deficits show improvement in their respective deficits after surgical elevation of the fracture

2) To determine the extent to which individual patient/radiological variables impact on clinical outcomes.

1.11) Objectives

1) Critically analyse data to objectively determine the effect of surgery on neuro-physical deficits.

2) Critically analyse data to determine the statistical significance of variables and their impact on neuro-physical outcomes post-surgery.

2) Methods

2.1) Design

Prospective observational study

2.2) Site of Study

Chris Hani Baragwanath Academic Hospital

2.3) Study Population

The study included patients who presented directly to or were referred to the neurosurgical unit at Chris Hani Baragwanath Hospital for the treatment of their THI.

2.4) Sampling

2.4.1) Sampling size including statistical rationale

- During a period of six months, the data from all patients meeting the inclusion criteria were included in the study.
- Thirty-eight patients were included in the study. Four patients were however lost to follow up at the six month follow up assessment. Three patients were excluded due to late presentation i.e. > 48 hours from injury to presentation. One patient was excluded due to the presence of neurological sepsis at presentation.
- In a period of six months we were able to include thirty patients with the above mentioned condition undergoing surgical elevation.

- The results obtained over this time period were statistically significant given that the number of patients accounted for 50% of all patients treated in a year with this condition by the neurosurgery unit at Chris Hani Baragwanath Academic Hospital.
- The time period for data collection was not extended as the estimated number of patients expected in the protocol planning stage was met during this time period.

2.4.2) Inclusion Criteria

- Patients who presented to or were referred to the neurosurgical unit at the Chris Hani Baragwanath Academic Hospital for neurosurgical assessment.
- Adult patients: 18 years and above
- Patient/next of kin who consented to both the proposed surgical procedure and use of medical data for research purposes
- Mechanism of injury: traumatic/non missile head injury
- Patients having sustained a depressed skull fracture
- Patient with neuro-physical deficits as a result of the injury sustained

2.4.3) Exclusion criteria

- Patients presenting for assessment more than 48 hours from time of injury
- The presence of neurological sepsis or local sepsis post injury (influences disease process and is not being investigated as an independent variable)
- Patients with previous neurological deficits from co-morbid disease

2.5) Measuring Tool or Instrument

- Data sheet designed to record clinical and radiological criteria (Appendix A)
- Medical Research Council scale (Appendix B)

2.6) Data Collection

- Written consent from the medical superintendent of the hospital was obtained prior to conduction of the study at the Chris Hani Baragwanath Academic Hospital (Appendix D).
- Data collection occurred over a six month period.
- Patients were screened on presentation to the Chris Hani Baragwanath Academic Hospital to determine if he/she would meet the inclusion criteria
- The patient was approached for consent to their surgical procedure and medical records as well as to allow this information to be used for research purposes
- In cases where the patient was not compos mentis, consent was obtained for both the surgical procedure and the use of medical records by the next of kin or first degree relative
- In the case where either the patient or the next of kin were unable to understand English, a translator was used. The translator was asked to sign the consent form as a witness as well.
- Data from patients' medical records were used to establish clinical and radiological details on admission and a comparison was made to post-operative assessments six months later.
- Clinical examinations were conducted by the researcher as well as fellow registrars of equal qualification to create uniformity amongst assessments.
- Appendix A and B were used as tools to record relevant discrete and continuous data.

2.7) Sources of Bias

- To avoid bias, routine management practices previously adopted by the unit remained valid for the course of the study. It is an observational study critically analysing data relating to patient outcomes that would have otherwise been available.

2.8) Confounder

- Timing of injury to time of surgery. Depending on the time of injury and presentation to our hospital, the depressed skull fracture may have resulted in both a primary and secondary neuronal injury. We have tried to limit the effect of this confounder with a time limit for surgery. The time limit used was 48 hours from time of injury to time of surgery. This forms part of the inclusion criteria.
- The elevation of depressed skull fractures at our institution is usually done on an emergency basis by registrars. There were five surgeons including the researcher who were involved in performing the surgical procedures. In an attempt to create uniformity in surgical experience and outcomes, the registrars involved shared similar experience and training in neurosurgery.

2.9) Variables

These are the pre-operative variables analysed in relation to motor outcomes at 6 months:

- Age
- Gender
- Time between injury and surgery
- Mechanism of injury
- Open vs closed injury
- Presence of a dural tear
- Underlying pathology e.g. extra dural hematoma, brain contusion

2.10) Data analysis

- Data acquired was divided into either discrete or continuous data

- Continuous variables e.g. (age) were summarised using mean/median methods depending on the distribution of data.

- Discrete variables e.g. gender, were summarised using either percentages or frequencies.

- Based on the data obtained in the data sheet, intra-class correlation was used to determine the following:

1) If a neuro-physical benefit was present after surgery

2) To objectively quantify the neuro-physical benefit after surgery using the MRC scale

- Generalised linear/mixed models were used to identify variables that are statistically significant in influencing neuro-physical outcomes before and after surgery

- Strata 2014 software was used to determine the statistical significance of the data

- The statistical significance level of all analysed data was a p value of < 0.05

2.11) Ethics

An ethics protocol submission was made to the Human Research Ethics Committee (Medical) at the University of Witwatersrand. The study was approved and researchers subsequently issued with a clearance certificate M161022 (Appendix C). Consent to use hospital patient records for research purposes were obtained from the medical superintendant at the Chris Hani Baragwanath Academic Hospital (Appendix D). Informed consent for the right to use medical records for research purposes were obtained from the patient or next of kin (Appendix E). All identifying information such as names and hospital numbers were removed from the data sheet and only a patient allocation tracking number was used.

3) Results

3.1) Records Reviewed

Screening of patients on presentation to the Chris Hani Baragwanath Academic Hospital commenced in January 2017 and ended in July 2017. The screening process isolated 38 possible patients. However, after the exclusion criteria was applied and the patients lost to follow up were accounted for, the final study sample was reduced to 30 patients. Table 4 is used to illustrate the total data collected in relation to the motor improvement of patients at 6 months. This group of patients was subsequently divided into two groups based on their presenting diagnosis. Group A accounted for patients with post traumatic isolated depressed skull fractures. Group B accounted for patients with post traumatic depressed skull fractures and associated underlying intracranial haematomas. Table 5 and Table 6 highlight the discrete and continuous data included for each group. The medical records pertaining to these patients were analysed and the results are as follows:

Table 4: Evaluation of demographic, radiological and clinical data in relation to the improvement in motor neurological function of patients after 6 months

	Total	**Improvement in motor function**	**No motor improvement**
Patients	30	13	17
Age		Range: 18-34 years Mean 26,2 years	Range: 18-67 years Mean 36,0 years
Mechanism of injury			
Assault	22	11	11
Motor vehicle accident	6	2	4
Pedestrian vehicle accident	2	0	2
Classification			
Open	11	6	5
Closed	19	7	12
Surgical findings			
Dural tear present	7	3	4
Time period between injury and surgery		Range: 4-18 hours Mean: 10,5 hours	Range: 8-43 hours Mean: 16,3 hours
Location of injury			
Frontal	10	3	7
Parietal	29	12	17
Occipital	0	0	0
Temporal	5	2	3
Radiological findings			
Effacement of sulci/gyri	30	13	17
Midline shift	16	5	11
Effacement of ventricle	13	5	8
Associated intracranial haematomas			
Extradural haematoma	7	3	4
Subdural haematoma	6	1	5
Cerebral contusion	5	1	4
Intracerebral haematoma	4	2	2
Intraventricular haemorrhage	0	0	0
No associated haematoma	11	6	5

T able 5: Group A including patients with post traumatic isolated depressed skull fractures

(Group A) Patients with isolated skull fractures	**No**
Total number of patients in group	11
Mechanism of injury	
Motor vehicle accident	2
Pedestrian vehicle accident	0
Assault	9
Classification	
Open injury	5
Closed injury	6
Surgical Findings	
Dural tear	1
No dural tear	10
Radiological findings	
Effacement of Sulci/ Gyri	11
Midline shift	2
Effacement of ventricle	1
Location of injury	
Parietal	6
Frontal/Parietal	3
Temporal/Parietal	2
Clinical findings/Medical Research Council grading (0-5 power)	
Upper limb motor improvement	
6 of 11 patients improved post op (54%)	
Range: 1-3 points improvement	
Average: 1.8 points improvement amongst the 6 patients	
Lower limb motor improvement	
6 of 11 patients improved (54%)	
Range: 1-3 points improvement	
Average: 1.7 points improvement amongst the 6 patients	

Table 6: Group B including patients with post traumatic depressed skull fractures with an associated underlying haematoma

(Group B) Patients with skull fractures and associated intracranial haematomas	**No**
Total number of patients in group	19
Mechanism of injury	
Motor vehicle accident	4
Pedestrian vehicle accident	2
Assault	13
Classification	
Open injury	6
Closed injury	13
Surgical Findings	
Dural tear	6
No dural tear	13
Radiological findings	
Effacement of Sulci/ Gyri	17
Midline shift	13
Effacement of ventricle	12
Location of injury	
Parietal	8
Frontal/Parietal	7
Temporal/Parietal	4
Associated intracranial haematomas	
Extra dural haematoma	7
Subdural haematoma	4
Cerebral Contusion	5
Intracerebral haematoma	4
Intraventricular haemorrhage	0
Clinical findings/Medical Research Council grading (0-5 power)	
Upper limb motor improvement	
7 of 19 patients improved post op (36%)	
Range: 1-5 points improvement	
Average: 1.8 points improvement amongst the7 patients	
Lower limb motor improvement	
6 of 19 patients improved (31%)	
Range: 1-3 points improvement	
Average: 1.3 points improvement amongst the 6 patients	

3.2) Age

The age range among the entire group of patients ranged from 18 years to 67 years of age. Figure 5 and Table 7 show the age distribution of patients among the isolated skull fracture group and the skull fracture with associated haemorrhage group. The mean age in the isolated skull fracture group was 29.09 years. The mean age in the skull fracture with underlying haemorrhage group was 30.26 years. The student's t-test was applied to the two samples with equal variance and the p value was 0.76. The age difference between these particular samples was not significant. Table 8 illustrates the age distribution between those patients with motor improvements versus those with no motor improvements. The mean age of the patients who improved was 26,2 years and those patients that did not improve was 36 years. The student's t-test was applied, considering age in relation to motor improvement. The p value was calculated to be 0.12. This value shows a trend towards statistical significance.

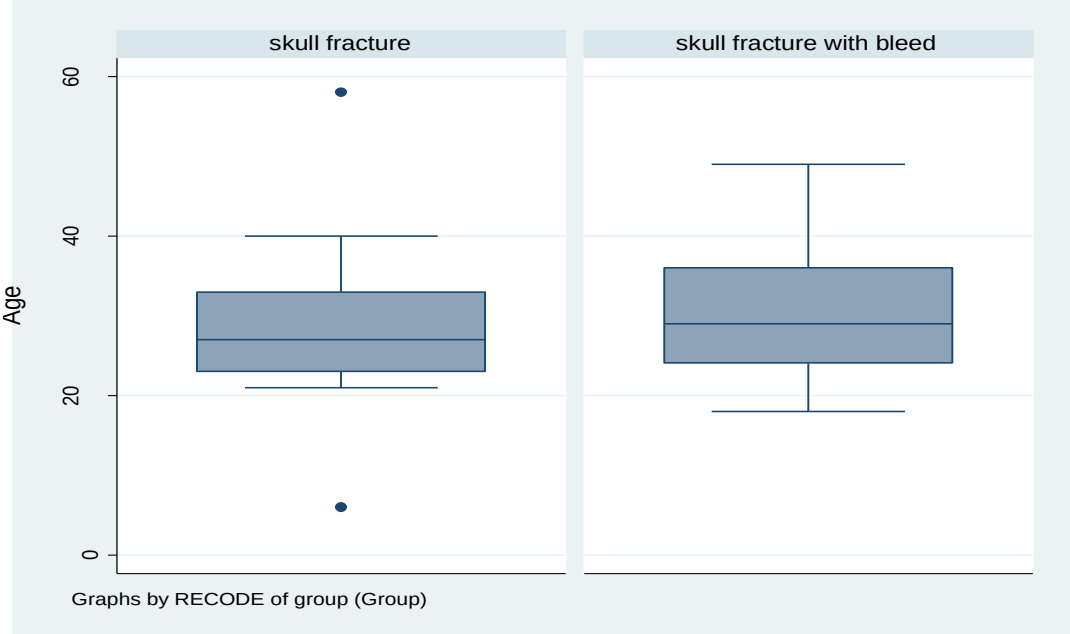


Figure 5: Box Plot graph illustrating age distribution in the groups of patients with isolated skull fractures and those with a skull fracture with an associated intracranial haemorrhage.

Table 7: The mean age distribution and standard deviation between patients with skull fractures and those with associated haemorrhage

Group	Patient Number	Mean age	Std. Err.	Std. Dev.	[95% Conf. Interval]	
Skull fracture	11	29.09091	3.843423	12.74719	20.52723	37.65459
Skull fracture with associated haemorrhage	19	30.26316	1.913085	8.338945	26.24391	34.2824
Combined	30	29.83333	1.821382	9.976121	26.10819	33.55848
Difference		-1.172249	3.840146		-9.038431	6.693933

Table 8: The age distribution between those patients with improvement in motor function vs patients with no improvement in motor function

Group	Patient number	Age range (years)	Mean (years)
Motor improvement	13	18-34	26,2
No motor improvement	17	18-67	36

3.3) Gender

There were 29 males and 1 female included in the study. Table 9 highlights the frequency of gender within the sample. Table 10 highlights the frequency of gender between the skull fracture and skull fracture with associated haemorrhage groups. The Fisher's exact test was applied to the data included in table 10. The p value was calculated to be 1. Gender as a variable between these two groups was not statistically significant.

Table 9: The frequency of gender within the total sample group

Gender	Frequency	Percent	Cumulative
Female	1	3.33	3.33
Male	29	96.67	100.00
Total	30	100.00	

Table 10: The frequency of gender distribution between the skull fracture and skull fracture with associated haematoma groups

Gender	Skull fracture group	Skull fracture with bleed group	Total
Female	0	1	1
	0.00%	5.26%	3.33%
Male	11	18	29
	100%	94.74%	96.67%
Total	11	19	30
	100.00%	100.00%	100.00%

3.4) Open versus closed injury

Traumatic skull fractures were classified as either open or closed fractures. There were 11 patients with open injuries and 19 patients with closed injuries. Table 11 illustrates the frequency of injuries among the patients included in the study. Table 12 illustrates the distribution of patients when considering injury classification in comparison with motor improvement in the isolated skull fracture group. The chi-squared test was used to calculate the p value. The p value was calculated as 0.12 showing a trend towards statistical significance. Table 13 illustrates the distribution of patients when considering injury classification in comparison with motor improvement in the skull fracture with associated bleed group. The chi-squared test was used to calculate the p value. The p value was calculated as 0.41 showing a trend towards statistical significance.

Table 11: The frequency of open versus closed injuries in the study sample

Injury classification	Frequency	Percentage	Cumulative
Closed	19	63.33	63.33
Open	11	36.67	100.00
Total	30	100.00	

Table 12: The distribution of patients with isolated skull fractures stratified by classification of skull fracture versus presence/absence of an improvement in motor function

Isolated skull fracture group	Closed	Open	Total
No Motor improvement	4	1	5
	66.67%	20.00%	45.45%
Motor improvement	2	4	6
	33.33%	80.00%	54.55%
Total	6	5	11
	100%	100%	100%

Table 13: The distribution of patients with skull fractures and associated haematomas stratified by classification of skull fracture versus presence/absence of an improvement in motor function

Skull fracture with associated haematoma group	Closed	Open	Total
No Motor improvement	9	3	12
	69.23%	50.00%	63.16%
Motor improvement	4	3	7
	30.77%	50.00%	36.84%
Total	13	6	19
	100%	100%	100%

3.5) Time of injury to time of surgery

The period between time of injury and time of surgery ranged from 4 hours to 45 hours. The majority of patients (60%; n=18) were operated on between 8 and 12 hours after their injuries. Table 14 illustrates the distribution of time periods between time of injury and time of surgery in this study. Table 15 illustrates the relationship between patients with skull fractures and associated haematomas and that of time to surgery. The student's t-test was used to calculate the p value. The p value was found to be 0.56. Table 16 illustrates the relationship between the improvement in patient's motor function and time to surgery. The unpaired student's t-test was used to calculate the p value. The p value was found to be 0.66. This result was not significant.

Table 14: The distribution of time periods between time of injury and time of surgery

Hours	Number of patients	Percent	Cumulative %
4	1	3.33	3.33
7	1	3.33	6.67
8	5	16.67	23.33
10	13	43.33	66.67
11	2	6.67	73.33
12	3	10	83.33
18	1	3.33	90.00
28	1	3.33	90.00
42	1	3.33	93.33
3	1	3.33	96.67
45	1	3.33	100.00
Total	30	100.00	

Table 15: The comparison between patients with skull fractures only and those with skull fractures with associated haematomas versus time to surgery

Group	Patient Number	Mean time to surgery	Standard (Std) Error (Err)	Std Deviation (Dev)	[95% Confidence. Interval (CI) respectively]	
Skull fracture	11	15.36364	4.31756	14.31973	5.743514	24.98376
Skull fracture with associated haematoma	19	12.94737	1.929266	8.409477	8.89413	17.00061
Combined	30	13.83333	1.966433	10.7706	9.811527	17.85514
Difference		2.416268	4.127678		-6.038898	10.87143

Table 16: The relationship between the improvement in patient's motor function and time to surgery

Group	Patient Number	Mean time to surgery	Std Err.	Std Dev.	[95% CI respectively]	
No motor improvement	17	14.58824	2.778672	11.45676	8.697713	20.47876
Motor improvement	13	12.84615	2.82127	10.17223	6.699134	18.99317
Combined	30	13.83333	1.966433	10.7706	9.811527	17.85514
Difference		1.742081	4.025097		-6.502956	9.987119

3.6) Dural tear

There were 23 patients without dural tears and 7 patients who presented with dural tears. Table 17 illustrates the frequency of patients who presented with dural tears. There were 6 patients with dural tears in the group of patients with skull fractures and associated haematomas. Five patients had improvements in their motor score and 1 patient had no improvement in their motor score following surgical intervention. Table 18 shows the relationship between patients with dural tears and motor improvement in the group of patients with skull fractures with associated haematomas. The chi squared test was used to determine the p value. The p value was calculated to be 0.90 showing no statistical significance. There was 1 patient with a dural tear in the isolated skull fracture group. This patient had an improvement in their motor score. Table 19 shows the relationship between the patient with a dural tear and motor improvement in the isolated skull fracture group. The chi squared was used to determine the p value. The p value was calculated to be 1.32 and thus was not statistically significant.

Table 17: The frequency of patients who presented with traumatic dural defects

Dural defect	Frequency	Percent	Cumulative
Absent	23	76.67%	76.67%
Present	7	23.33 %	100.00%
Total	30	100.00%	

Table 18: The relationship between the frequency of traumatic dural defects and an improvement in motor neurological function in the group of patients with skull fractures with an associated intracranial haematoma

Skull fracture with associated haematoma group	Dural defect absent	Dural defect present	Total
Motor improved	8	5	13
	61.54%	83.33%	68.42%
No motor improvement	5	1	6
	38.46%	16.67%	31.58%
Total	13	6	19
	100%	100%	100%

Table 19: The relationship between the frequency of traumatic dural defects and an improvement in motor neurological function in the group of patients with isolated skull fractures

Isolated skull fracture group	Dural defect absent	Dural defect present	Total
Motor improved	4	1	5
	40.00%	100.00%	45.45%
No motor improvement	6	0	6
	60.00%	0%	54.55%
Total	10	1	11
	100%	100%	100%

3.7) Mechanism of injury

The mechanism of injury varied among patients. Twenty-two patients were assaulted, 6 patients were involved in MVA and 2 patients were involved in PVA. Table 20 illustrates the frequency of distribution of mechanism of injury among patients within the total sample. Table 21 illustrates the relationship between mechanism of injury and motor improvement in the total sample of patients. The chi squared test was used to calculate the p value. The p value was calculated to be 0.43 showing no statistical significance.

Table 20: Mechanism of injury in the total patient sample

Mechanism of injury	Frequency	Percent	Cumulative
Assault	22	73.33%	73.33%
MVA	6	20.00%	93.33%
PVA	2	6.67%	100.00%
Total	30	100%	

Table 21: The relationship between mechanism of injury and improvements in motor function in the total sample of patients

Group	Assault	MVA	PVA	Total
Motor improvement	12	3	2	17
	54.55%	50.00%	100.00%	56.67%
No motor improvement	10	3	0	13
	45.45%	50.00%	0%	43.33%
Total	22	6	2	30
	100.00%	100.00%	100.00%	100.00%

3.8) Isolated skull fracture versus skull fracture with associated haemorrhage

There were 11 patients with isolated skull fractures of which only 54.55% (n=6) had an improvement in their motor deficit. There were 19 patients with skull fractures and associated haematomas of which only 31.58% (n=6) had an improvement in their motor score. Table 22 illustrates the distribution of patients in the skull fracture and skull fracture with associated haemorrhage groups relative to improvements in their respective motor scores. The chi squared test was used to calculate the p value. The p value was calculated to be 0.21 showing a trend towards statistical significance. Patients in the isolated skull fracture group had an average improvement of 0.90 points in their motor score. Patients in the skull fracture with associated haemorrhage group had an average improvement of 0.56 points in their motor score. Table 23 below illustrates the comparison of mean number of points improved on the MRC scale between the skull fracture group and the skull fracture with associated haemorrhage group. The student's t-test was applied to determine the p value. The p value was calculated to be 0.30 showing a trend towards statistical significance.

Table 22: The distribution of patients stratified by the presence of an isolated skull fracture vs a skull fracture with an associated intracranial haematoma, relative to improvements in their respective motor scores.

	Isolated skull fracture	**Skull fracture with associated haematoma**	**Total**
No motor improvement (n)	5	13	18
%	45.45%	68.42%	60.00%
Motor improvement (n)	6	6	12
%	54.55%	31.58%	40.00%
Total	11	19	30
	100.00%	100.00%	100.00%

Table 23: The comparison of mean number of points improved on the MRC scale between the isolated skull fracture group and the skull fracture with associated haematoma group.

Group	Number of patients	Mean number of points improved on the MRC scale	Std error	Std deviation	95% CI	
Isolated skull fracture	**11**	**.9090909**	**.3149183**	**1.044466**	**.2074091**	**1.610773**
Skull fracture with associated haematoma	**19**	**.5263158**	**.2075817**	**.9048279**	**.0902027**	**.9624289**
Combined	**30**	**.6666667**	**.1750752**	**.9589266**	**.3085976**	**1.024736**
Difference		**.3827751**	**.3625916**		**-.3599601**	**1.12551**

3.9) Upper limb versus lower limb

Table 24 illustrates the distribution of patients relative to the points improved on the MRC scale in the upper limb. Table 25 illustrates the distribution of patients relative to their respective points improved on the MRC scale in the lower limb. The average motor score in the upper limb and lower limb at 6 months follow up was found to be 3.06 and 2.20 respectively. Table 26 illustrates the average motor scores on the MRC scale postoperatively in patients at 6 months relative to the upper limb and lower limb. The student's t-test was used to calculate the p value. The p value was calculated to be 0.07 showing a trend towards statistical significance

Table 24: The distribution of patients relative to the respective improvement in points on the MRC scale in the lower limb

Points improved on the MRC scale in the lower limb	**Number of patients**	**Percentage**	**Cumulative**
0	18	60.00%	60.00%
1	6	20.00%	80.00%
2	4	13.33%	93.33%
3	2	6.67%	100.00%
Total	30	100.00%	

Table 25: The distribution of patients relative to the respective points improved on the MRC scale in the upper limb

Points improved on the MRC scale in the upper limb	Number of patients	Percentage	Cumulative
0	17	56.67%	56.67%
1	5	16.67%	73.33%
2	5	16.67%	90.00%
3	2	6.67%	96.67%
5	1	3.33%	100.00%
Total	30	100.00%	

Table 26: Comparison between the average post-operative (6 months) MRC scale motor scores of the upper and lower limbs

Group	Number of patients	Average motor score on the MRC scale postoperatively at 6 months	Std error	Std deviation	95% CI	
Motor score lower limb	30	2.2	.2218004	1.214851	1.746367	2.653633
Motor score upper limb	30	3.066667	.2488287	1.362891	2.557755	3.575578
Difference	30	-.8666667	.2286056	1.252125	-1.334218	-.3991156

4) Discussion

4.1) Study Sample

The sample size originally used for this particular study included 38 patients. Eight patients were excluded from the study due to the exclusion criteria being enforced or patients being lost to follow up. This sample of 30 patients was obtained over a six month screening period and patients were subsequently followed up for a period of six months. The six month follow up period allowed for patients to undergo the appropriate rehabilitation including physical therapy. Patients were then subjected to a clinical examination after this six month period to adequately assess for motor improvements. The examinations were conducted by the same clinician both pre and post operatively to avoid inter-examiner discrepancy in findings.

The decision was made early in the protocol development phase of this study to include a time period for data collection rather than a target of a fixed number of patients. This rational was based on the idea that the sample obtained over a six month period would account for 50% of all patients presenting with this condition to the Chris Hani Baragwanath Academic Hospital in a given year. In so doing, the results obtained would be statistically significant to the treating centre.

The results obtained thus far in evaluating trends and the effect of variables on motor deficits has shown promise. In retrospect however, statistically significant results were not obtained due to the small sample size.

It is in the opinion of the author that this study should serve as a pilot study. The data obtained and trends illustrated in this study show promise and a larger study sample obtained from multiple centres would likely yield statistically significant results of a similar nature.

4.2) Age

The mean age of patients who experienced an improvement in motor function was 26.2 years. The mean age of patients who did not experience an improvement in motor function was 36 years as illustrated in Table 8. This shows a mean age difference of 9.8 years between the above mentioned groups. The p value was calculated to be 0.12 showing a strong trend towards statistical relevance. The inference from the above is that advances in age can be used pre-operatively as a poor prognostic factor for potential motor outcomes.

Age was then assessed in relation to patients with skull fractures and patients with skull fractures and associated haematomas. The mean age of the isolated skull fracture group was 29.09 years. The mean age of the skull fracture with associated haematoma group was 30.26 years. The mean age difference between these groups was found to be 1.17 years as shown in Table 7. The p value was calculated to be 0.76 using the student's t-test. The age difference between each group was not statistically significant. The inference from the above is that age was not an influencing factor in prognosticating motor improvements when patients are subdivided into these groups.

4.3) Gender

The bias in the sample towards males could not be avoided as the sample was obtained over a 6 month period using census sampling. The male predominance in this study however correlates positively with the findings quoted by Naidoo in 2013, showing that male gender was indeed a risk factor for a THI in South Africa (1).

The results reflect a bias towards male gender as well as a poorly reflective sample of both gender groups within the sample group. The statistical relevance of gender in prognosticating motor improvements pre operatively thus could not be determined. This represents an avenue for further potential research in this field using gender as an isolated variable.

4.4) Open versus closed injury

Of the 11 patients with open injuries, 7 (63.63%) had improvements in motor function post-operatively. Of the 19 patients with closed injuries, 6 (31.57%) had improvements in motor function post operatively. The inference from the above is that patients with open injuries are more likely to have improvements in motor function post operatively (p value of 0.45). Interestingly this fact goes against traditional thinking when considering the implications associated with open injuries.

In the isolated skull fracture group, 2 of the 6 patients (33.3%) with closed injuries experienced improvements in motor function. Similarly, in the isolated skull fracture group 4 of the 5 patients (80%) with open injuries experienced improvements in motor function (p value 0.12).

In the skull fracture with associated haemorrhage group, 4 out of 9 (30.7%) patients with closed injuries experienced improvements in motor function. Similarly, in the skull fracture with associated haemorrhage group, 3 out of 6 patients (50%) with open injuries experienced improvements in motor function (p value 0.41)

The inference from the above shows correlating results in that patients with open injuries are more likely to have improvements in motor function post operatively. This fact has a higher statistical significance in the isolated skull fracture group and is thus clinically relevant.

4.5) Time of injury to time of surgery

Patients were first screened by the neurosurgical registrar on arrival to the CHBAH. A diagnosis was made and the patient underwent surgery after adequate resuscitation measures were completed. Patients were operated on as a matter of urgency. Reasons for delays included delayed presentations, transportation delays from referring institutions and the lack of availability of emergency operating theatres.

Table 16 illustrates that the average time to surgery in those patients who experienced improvements in motor function (n=17) was 12.84 hours. Similarly, the average time to surgery in those that did not experience any improvements in motor function (n=13) was 14.58 hours. This showed a difference on average by 1.74 hours between both groups. The inference from the above was that a shorter time to surgery correlated positively with improvements in motor function experienced post operatively (p value 0.66)

Table 15 illustrates the average time to surgery between the isolated skull fracture group and the skull fracture with associated haemorrhage. It was observed by the author that the general tendency among anaesthetists and neurosurgeons was to prioritise patients with skull fractures with associated haematomas rather than the isolated skull fracture group for surgery. The average time to surgery in the isolated skull fracture group (n=11) was 15.36 hours. The average time to surgery in the skull fracture with associated haemorrhage group (n=19) was 12.94 hours. The average difference between groups was calculated to be 2.41 hours (p value 0.56). We know now from the above inference that time to surgery correlates positively with motor improvements post operatively. These results may thus be seen as a limiting factor to the actual motor improvements seen in the isolated skull fracture group.

4.6) Dural tear

The inference from the above results indicates that patients with dural tears are more likely to experience improvements in their motor deficits post operatively. This goes against traditional thinking. It is in the opinion of the author that a plausible reason behind this finding could be that mass effect on eloquent brain is reduced when there is a dural breach allowing for expansion of inflamed tissue. This trend is also continued when patients are further subdivided into the isolated skull fracture group and the skull fracture with associated haemorrhage group.

4.7) Mechanism of injury

Twelve of the 22 patients (54%) who were assaulted experienced motor improvements post operatively. Three of the 6 patients (50%) who were involved in a MVA experienced motor improvements post operatively. 100% of the patients involved in a PVA experienced motor improvements post operatively.

When considering patients who were assaulted and those involved in MVA, the likelihood of having motor improvements was 54.55% and 50.00% respectively. The sample size in patients who were involved in a PVA was just 2 and thus was not considered for analysis as a factor influencing motor improvements. The inference from the above findings was that mechanism of injury could not be used to predict motor improvements post operatively (p value 0.43).

4.8) Isolated skull fracture versus skull fracture with associated haemorrhage

There were 6 of 11 patients (54.5%) with isolated skull fractures who experienced improvements in their motor deficits. There were 6 of 19 (31.58%) patients with skull fractures and associated haematomas who experienced improvements in their motor deficits.

The average points improved on the MRC motor scale in the isolated skull fracture group and the skull fracture with associated haematomas group was 0.90 and 0.52 respectively.

Therefore, patients with isolated skull fractures are more likely to experience motor improvements in comparison to the group of patients with skull fractures and associated haemorrhage 54.55% versus 31.58% (p value 0.21).

It may also be inferred that the degree of improvement was higher in the isolated skull fracture group as opposed to the skull fracture with associated haematoma group when measured on the MRC motor scale, 0.90 points vs 0.52 points improved respectively (p value 0.30).

4.9) Upper limb vs lower limb

There were 43.33% (n=13) of patients of the total group who had motor improvements in the upper limb as illustrated in Table 25. There were 40% (n=12) of patients of the total group who had motor improvements in the lower limb as illustrated in Table 24. Table 26 also reflects the average upper limb and lower limb motor scores on the MRC scale at six months evaluation as 3.06 and 2.2 respectively.

Therefore, the above findings show a slight predilection for upper limb motor improvement as compared to lower limb motor improvement post operatively. The degree of improvement as measured by the MRC motor scale was also slightly higher in the upper limb by 0.86 points as compared to the lower limb group (p value 0.07).

4.10) Limitations

- A number of patients were lost to follow up during the period of research. Clinical examination and comparison of results at six months was therefore not possible.

- On occasion, patients presented with altered levels of consciousness making clinical examination of the patient's motor system difficult. The patient's best motor score was then used for data collection.

4.11) Conclusion

A motor deficit sustained after a traumatic depressed skull fracture is often one of the first major objective findings that is identified on clinical examination by health care providers. Patients and their families are often distressed and overwhelmed. They realise that these motor deficits have occurred as a result of their injury and that the possibility of these deficits being permanent is real. Health care workers would then be subjected to a battery of leading questions by patients and their families with the intent to determine if these deficits would improve with treatment. In the past, however, there was no objective way to quantify the potential benefit from various treatment options or to use individual variables to prognosticate on outcomes. The answers to such questions would therefore be uncertain.

The aim of this study was to determine if patients with post traumatic depressed skull fractures with resultant neuro-physical deficits showed improvement in their respective motor deficits after surgical elevation of the fracture. Secondly the aim was to determine the extent to which individual clinical/radiological variables would impact on clinical outcomes. To the authors knowledge this is the first research paper to analyse neuro-physical benefits in patients with post traumatic skull fractures and resultant motor deficits.

Results showed that 66% (n=12) of patients with post traumatic depressed skull fractures did in fact experience a motor improvement post operatively. Patients with isolated skull fractures were more likely to experience motor improvements than patients with skull fractures and underlying haematomas (p value 0.21). The degree of motor improvement based on the MRC scale was higher is the isolated skull fracture group as compared to the skull fracture with associated haemorrhage group (p value 0.30).

Prognostic variables analysed pre-operatively showed that a younger age (p value 0.12), shorter time to surgery (p value 0.66), a dural tear (p value 0.90) and an open injury (p value 0.45) compared favourably with improved motor outcomes post operatively.

Upper limb vs lower limb motor improvements were analysed. The results indicate that there is also a slight predilection for upper limb motor improvement as opposed to lower limb motor improvement post operatively. The average degree of improvement as measured by the MRC motor scale was slightly higher in the upper limb by 0.86 points as compared to the lower limb group (p value 0.07)

Statistically significant results could not be achieved as the sample size in this study was too small. It was however motivating to notice trends towards statistical significance in the analyses of the results of this study. This study should serve as a pilot study for future research based on the positive trends that were found. It is the hope of the author that these trends guide future treatment strategies and help serve as a knowledge base to counsel both patients and their families preoperatively.

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6) Appendix

Appendix A: Data sheet page 1

Research project:

Analysis of the neuro-physical benefits of surgically elevating depressed skull fractures in patients who have sustained non-missile traumatic head injuries at the Chris Hani Baragwanath Academic Hospital.

Researcher:

Dr N Munthree

Supervisor:

Dr S Gowan, Dr J Ouma

Patient Details:

Name: _____

Age: _____

Gender: _____

Hospital File Number: _____

Date of admission: _____

Patient allocation tracking number: _____

Data sheet page 2

Patient allocation tracking number: _____

Clinical Data:

Mechanism of injury: _____

Open vs Closed injury: _____

Dural tear present: _____

Site of injury: Occipital/ Frontal/ Parietal/ Temporal

Time period between injury and surgery: _____

Motor Deficit using the Medical research Council scale on admission:

Upper limb left: _____/5 Upper limb right: _____/5

Lower limb Left: _____/5 Lower limb right: _____/5

Motor Deficit using the Medical research Council scale on six month review:

Upper limb left: _____/5 Upper limb right: _____/5

Lower limb Left: _____/5 Lower limb right: _____/5

Radiological Data:

Depth of depression of skull fracture greater than width of skull table: Yes / No

Clinical motor deficit accounted for and related to the site of skull fracture: Yes / No

Dural tear present: Yes/ No

Underlying Pathology: Yes/ No

Type of underlying pathology if applicable:

Intra-Ventricular Bleed, Brain Contusion, Intra-Cerebral Haemorrhage,

Extra Dural Haemorrhage, Subdural Haemorrhage, Pneumocephalus

Mass effect from depressed skull fracture present:

Nil, Effacement of underlying Sulci and Gyri, Midline Shift, Effacement of Ventricle

Appendix B: Medical research council scale

Motor strength evaluation

Grade

0) No contraction
1) Flicker of movement
2) Active movement with gravity eliminated
3) Active movement against gravity
4) Active movement against gravity and resistance
5) Normal power

Grade 4 may be divided into 4- , 4 and 4+ to indicate slight, moderate or strong resistance respectively

Appendix C: Ethics Approval

R14/49 Dr Nash Munthree

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M161022

NAME: (Principal Investigator)	Dr Nash Munthree
DEPARTMENT:	Neurosurgery Chris Hani Baragwanath Academic Hospital
PROJECT TITLE:	Analysis of the Neuro-Physical Benefits of Surgically Elevating Depressed Skull Fractures in Patients who have Sustained Non-Missile Traumatic Head Injuries at the Chris Hani Baragwanath Academic Hospital
DATE CONSIDERED:	28/10/2016
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Dr Shamil Gowan
APPROVED BY:	Professor P. Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	03/04/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 10004,10th floor, Senate House/2nd floor, Phillip Tobias Building, Parktown, University of the Witwatersrand. I/We fully understand the the conditions under which I am/we are authorised to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated,from the research protocol as approved, I/we undertake to resubmit to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. in this case, the study was initially review October and will therefore be due in the month of October each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature Date 4/4/17

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix D: Consent from Medical Superintendant for the use of medical records

Chris Hani Baragwanath Academic Hospital

Department of Neurosurgery

3 October 2016

To whom it may concern:

Re: Consent authorising the use of patient medical records at the Chris Hani Baragwanath Academic Hospital for research purposes

Research project:

Analysis of the neuro-physical benefits of surgically elevating depressed skull fractures in patients who have sustained non-missile traumatic head injuries at the Chris Hani Baragwanath Academic Hospital.

Researcher: Dr N Munthree

Supervisors: Dr S Gowan, Dr J Ouma

Please be advised that this letter serves as proof authorising the above mentioned researchers access to and the anonymous use of patient medical records at the Chris Hani Baragwanath Academic Hospital for research purposes.

Medical Superintendant - Chris Hani Baragwanath Academic Hospital:

Name: DR. K.A. MUSTAFA

Signature: K A. [signature]

Email address: kmdrmustafa747@gmail.com

Contact Number: (011) 933 0000

Appendix E: Informed consent page 1

Patient Consent Form: use of clinical information for research

Good Day Madam/Sir

My name is Dr Nash Munthree. I am a neurosurgical registrar at the Chris Hani Baragwanath Hospital. I am conducting a research project titled below.

You are currently admitted at the Chris Hani Baragwanath Academic Hospital for treatment of problems you are currently experiencing. The Chris Hani Baragwanath Academic Hospital not only renders treatment but is also actively involved in conducting research aimed at improving the quality of care that we deliver. From time to time such research involves the use of patient records from which information is extracted. The use of such information is subject to the following:

1. Approval from the Human Research Ethics Committee (Medical) of the University of the Witwatersrand.
2. Identity of a patient from whose file information is extracted is never revealed to anyone but the researcher unless specific consent is obtained to do so. The information gathered does not contain the name of the patient.

We would like to obtain your consent to use information from your file for the purpose of research, subject to the aforementioned conditions. If you choose not to give consent, this will not compromise your treatment in any way. If at any time you choose to withdraw consent you are free to do so and will not be prejudiced in any way.

Informed consent page 2

Research project title:

Analysis of the neuro-physical benefits of surgically elevating depressed skull fractures in patients who have sustained non-missile traumatic head injuries at the Chris Hani Baragwanath Academic Hospital.

Aim

1) To determine if patients with post traumatic depressed skull fractures with resultant neuro-physical deficits show improvement in their respective deficits after surgical elevation of the fracture.

(neuro-physical deficits - refers to weakness of the patient's limbs or face)

2) To determine the extent to which individual patient/ radiological variables impacts on clinical outcomes.

Objectives

- Critically analyze data to objectively determine effect of surgery on neuro-physical deficits.
- Critically analyze data to determine statistical significance of variables and their impact on neuro-physical outcomes post-surgery.

Informed consent page 3

Patient requirements:

- Participation in the above study is completely voluntary. Consent may be withdrawn at any stage without prejudice. The standard of care afforded to the patient will remain the same irrespective of participation in the study.
- In cases where patients are unable to consent on admission, deferred consent will be obtained from the patient if possible.
- In cases were family members have consented to the use of the patients medical records due to incapacity of the patient, an attempt will be made at a later stage to obtain the patients consent if possible.
- Clinical follow up after six months as part of routine management

Human research and ethics committee protocol reference number: M161022

Should you wish to contact us at any stage regarding consent, contact:

Researcher: Dr N Munthree Tel: (011) 933 0000

Chris Hani Baragwanath Academic Hospital

Informed consent page 4

Patient Consent form:

Use of clinical information for research

A. Consent Given

I hereby give consent for patient …….

record's to be used as per the above mentioned conditions for the purposes of research:

PATIENT: _____ DATE: _____

SIGNATURE: _____

B. Consent Not Given

I hereby do not give consent for patient …….

record's to be used for the purposes of research:

PATIENT: _____ DATE: _____

SIGNATURE: _____

Appendix F: Turnitin Report

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ORIGINALITY REPORT

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