THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

A STUDY ON DECOMPRESSIVE CRANIECTOMY IN MODERATE TO SEVERE HEAD INJURY PATIENTS

Dissertation submitted in partial fulfillment of the requirements for the degree
Of

M.Ch. Branch –II

NEUROSURGERY

Examination in AUGUST 2012

INSTITUTE OF NEUROLOGY

MADRAS MEDICAL COLLEGE

CHENNAI – 3.

CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON DECOMPRESSIVE CRANIECTOMY IN MODERATE TO SEVERE HEAD INJURY PATIENTS"** is the bonafide original work of **Dr.S.JOTHIKUMAR** in partial fulfillment of the requirements for Branch – II, M.Ch Neurosurgery, examination of THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY to be held in August 2012. The period of post graduate study and training was from July 2009 – August 2012.

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DECLARTION

I solemnly declare that this dissertation **"A STUDY ON DECOMPRESSIVE CRANIECTOMY IN MODERATE TO SEVERE HEAD INJURY PATIENTS"** was prepared by me in the Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of Professor of Neurosurgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai between 2009 and 2012.

This dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai in partial and fulfillment of the university requirements for the award of degree of M.Ch. Neurosurgery.

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Date:

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To

Dr. S. Jothi Kumar
PG in MCH Neurosurgery
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Dear Dr. S. Jothi Kumar

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "A study on Decompressive craniectomy in moderate to sever head injury patients" No. 06032011.

The following members of Ethics Committee were present in the meeting held on 17.03.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. V. Kanagasabai .MD Dean, Madras Medical college, Chennai -3.	-- Deputy Chairman
3.	Prof. A. Sundaram, MD Vice Principal, Madras Medical College, Chennai -3	-- Member Secretary
4.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
5.	Prof. C. Rajendiran .MD Director , Institute of Internal Medicine, MMC, Ch-3	-- Member
6.	Prof. Geetha Subramanian, MD,DM Prof. & Head , Dept. of Cardiology, MMC, Ch-3	-- Member
7.	Prof. Mohammed Ali MD DM Prof & HOD of MGE, MMC , Ch-3.	-- Member
8.	Thiru. A. Ulaganathan Administrative Officer, MMC, Chennai -3	-- Layperson
9.	Thiru. S. Govindasamy . BA.BL	-- Lawyer
10.	Tmt. Arnold Soulina	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

CONTENTS

INTRODUCTION

An injury to the brain may cause edema and produce swelling of brain. Pressure within the skull then increases as the brain has no room to expand; this excess pressure, known as high intracranial pressure, can cause further secondary brain injury. High intracranial pressure is the most frequent cause of death and disability in brain-injured patients.

The management of increased intracranial pressure is common clinical scenario in neurosurgery. If high intracranial pressure (ICP) cannot be controlled using general or first-line therapeutic measures, second-line treatments are initiated, one of these procedure is decompressive craniectomy (DC) and also performed while intracranial hematoma evacuation. DC involves the removal of a section of skull so that the brain has room to expand and the ICP decreased. There is however still clinical uncertainty regarding the use of DC and a lack of consensus on the optimal management of traumatic brain injury[1].

The present study was undertaken to analyze the factors that affects the patient's outcome in our setup, and to analyze the role of decompressive craniectomy and also the factors predicting the outcome.

AIM OF THE STUDY

To assess the effects of primary decompressive craniectomy on outcomes and quality of life for patients with moderate to severe traumatic brain injury. To analyze the factors that affects the outcome in our setup, and to analyze the role of decompressive craniectomy and factors predicting the outcome.

REVIEW OF LITERATURE

TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) affects up to2% of the population per year and constitutes the major cause of death and severe disability among young people[2]. Road traffic injuries account for 2.1% of global mortality. The developing countries bear a large share of burden and account for about 85% of the deaths as a result of road traffic crashes. India accounts for about 10% of road accident fatalities worldwide[3].

PATHOPHYSIOLOGY OF TBI

The tissue damage at the moment of brain trauma is the primary injury, whereas secondary mechanisms lead to brain edema. Disruption of the BBB is the most important prerequisite for edema formation[4]. Both vasogenic and cytotoxic edema results in increased intracranial pressure and eventually decreases cerebral perfusion pressure. This is in line with the Monroe-Kellie hypothesis which states that ‘the sum of the intracranial volumes of blood, brain, CSF and other components is constant and that an increase in any one of these must be offset by an equal decrease in another. Elevated ICP diminished cerebral perfusion and can lead to tissue ischemia. Ischemia in turn may lead to vasodilatation via auto regulatory mechanisms designed to restore cerebral perfusion. However vasodilatation increases cerebral blood volume, which in turn then increases ICP, lower CPP and provokes further ischemia[5]. After Traumatic brain injury, CBF autoregulation is impaired or absent in most patients. When pressure autoregulation is impaired or absent, ICP decreases and increases with change in cerebral perfusion pressure (CPP) [6]. Also, autoregulatory vasoconstriction seems to be more resistant compared with autoregulatory vasodilatation which indicates that patients are more sensitive to damage from low rather than high CPP.

CASCADE OF EVENTS IN THE PATHOPHYSIOLOGY OF TBI

1. Initially there is direct tissue damage and impaired regulation of cerebral blood flow and metabolism.

2. Decreased CBF leads to accumulation of lactic acid due to anaerobic glycolysis, increased membrane permeability and consecutive edema formation.

3. Anaerobic glycolysis leads to depleted ATP stores and failure of energy dependent brain ion pumps.

4. Hypoxia leads to release of excitatory neurotransmitters like glutamate and aspartate.

5. These and other neurotransmitters activate the ionotropic (NMDA) and metabotropic receptors

6. Consequently Ca++ and Na+ influx with K+ efflux

7. Ca++ also activates lipid peroxidase, resulting in accumulation of free fatty acids and oxygen free radicals.

8. Prostaglandins and kinins initiate an inflammatory response.

9. Further activations of caspases, translocases and endonuclease initiate progressive structural changes of biological membranes and nucleosomal DNA.

10. There is a depression of metabolic activity of neural tissue resulting in suppressed neuronal activity.

11. Role of aquaporin-4 channels, decreased Mg++ levels and vassopressor-2 receptor channels and erythropoietin in the pathophysiology of post traumatic brain edema is being studied.

Collectively these events lead to BBB disruption and degradation of cellular structures and ultimately necrotic or programmed cell death[4].

HISTORICAL BACKROUND OF DC

Kocher was the first to propose decompressive craniectomy for patients with clinical symptoms of persistent elevated ICP in1901[1,7,8]. Later, in 1905, Harvey Cushing made a detailed report on subtemporal and suboccipital decompression procedure to relieve high ICP in patients with inoperable brain tumors [1,7,8]. A comprehensive historical review of the first few patients who underwent DC was published by Spiller and Frazier in 1906. Decompressive craniectomy in TBI was initially described by Miyazaki in1966 and later popularized by Kjellberg and Prieto in1971[9].

DC involves the removal of a section of skull so that the brain has room to expand and the pressure decreases. Removal of a section of skull bone after a severe traumatic brain injury in patients with persistent raised intracranial pressure that has not responded to conventional medical treatments. Strategy for management of ICP by decompressive craniectomy is to remove the mechanical constrains imposed by the cranial vault.

Types of surgical decompression

1. Primary decompressive craniectomy (P-DC) or Prophylactic decompression is defined in this review as any surgical

decompression performed, with or without brain tissue removal, in patients undergoing surgery primarily for the evacuation of any type of intradural lesion. The aim of prophylactic craniectomy is not only to control refractory ICP but also to avoid expected postsurgical increases in ICP. In these procedures the decision taken by the surgeon is generally independent of ICP and is usually based on a CT scan or intraoperative surgical findings (brain swelling, a ’tight’ brain, or difficulties in repositioning the bone flap), or both[10, 11].

2. Secondary decompressive craniectomy (S-DC) or Therapeutic decompression is defined as the procedure performed in patients in whom continuous ICP monitoring is conducted and in whom high ICP is refractory to medical treatment. This therapeutic option is used in some centers after first- or second- line therapeutic measures have failed to control ICP. In the category of S-DC we also included patients who had undergone a first surgical procedure to evacuate a space-occupying lesion and who had later developed delayed massive unilateral or bilateral brain swelling. Although previous surgery might have been performed in these patients, the purpose of surgical decompression is to control high ICP[12].

The mechanism by which decompressive craniectomy provides reliefs in raised ICP are[7]:

1. It lowers the ICP immediately.
2. It adds vector of expansion to cerebral hemispheres which relieves brain herniation.
3. Allows exploration of subdural space.
4. In addition it provides quick tapering of medical treatment, in order to avoid potential complications.

EFFECTS OF DECOMPRESSIVE CRANIECTOMY

1. Improving cerebral perfusion
2. Preventing ischemic damage
3. Avoiding mechanical compression of the brain (brain herniation)

The overall effects of decompressive craniectomies are to increase volume-buffering capacity of the cranial vault by allowing for centripetal herniation. The centripetal herniation in turn minimizes centrifugal compression of the brain stem structures[13]. Decompressive craniectomy reduces intracranial pressure by 50%, duratomy further enhances intracranial pressure reduction by an additional 35%[14].

The rationale for decompressive surgery is based on the Monro-Kellie law. According to this theory intracranial volume should remain constant and volumetric compensations should be achieved by shifts in CSF, cerebral blood volume, or brain herniation. Removing a variable amount of bone, with or without leaving the duramater open or augmented by a duraplasty, is a fast and effective means of increasing intracranial volume; reducing elevated intracranial pressure and increasing the compliance of the intracranial space. In the Aarabi et al study, mean ICP decreased from 24 to 14.6 mm Hg after decompressive craniectomy[15].

AANS RECOMMENDATIONS

The American Association of Neurological Surgeons has recommended decompressive craniectomy for patients with traumatic brain injury and refractory intracranial hypertension if some or all of the following criteria were met[7]:

1. Diffuse cerebral swelling on CT imaging.
2. Within 48 hrs of injury.
3. No episodes of sustained intracranial hypertension (ICP) > 40 mm Hg before surgery.
4. GCS > 3 at some point subsequent to injury.
5. Secondary clinical deterioration, and
6. Evolving cerebral herniation syndrome.

INDICATIONS FOR DECOMPRESSIVE CRANIECTOMY

1. DC has most commonly been performed in patients with traumatic brain injury and cerebral infarction associated with intractable intracranial hypertension.

2. Other indications, which have mostly been described in single case reports or small case series includes aneurysmal SAH, ICH, palliation for brain tumors, meningitis, subduralempyema, encephalitis, acute disseminated encephalomyelitis, encephalopathy due to Reye syndrome, toxoplasmosis, and cerebral venous and dural sinus thrombosis[16,13].

SURGICAL TECHINIQUE

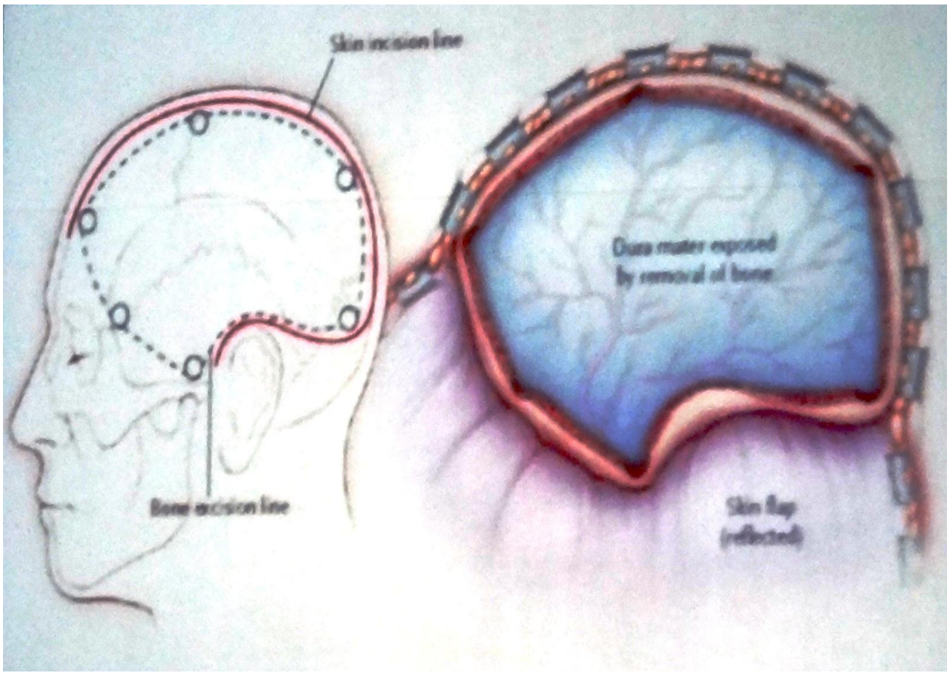
Wide variability has been reported in the surgical procedures for performing decompressive surgery. Nine different types of craniectomies were reported. These variations include small to massive amounts of bone removal, unilateral or bilateral bone decompression, opening the duramater or leaving it closed, scarifying the duramater to decrease its rigidity, and sectioning of the falx among others. Localization of bone removal can be unilateral, bilateral, bifrontal, or subtemporal; or it can be expanded to what has been called ’circumferential decompression’.

In general, these decompression techniques can be divided into three approaches[13]:

Frontaltemporo-parietal approach, frontal approach and temporal approach. All the three approaches can be performed unilaterally or bilaterally.

Frontaltemporo-parietal approach

The patient is placed in supine with head elevated and rotated 30 to45 degrees. Vertex of the head is directed downwards to bring the zygomatic arch to the uppermost plane. The skin incision can be in the form of trauma flap, with the goal of exposing the following margins of craniectomy: anteriorly to the superior border of orbital roof (avoiding entry into frontal sinus); posteriorly to at least 2cm lateral to the external auditory meatus; medially to 2cm lateral to midline (avoiding sagittal sinus); and inferiorly to the floor of middle cranial fossa. Temporalis muscle is reflected anteriorly. Burr holes are placed at the keyhole, the root of the zygoma and along the planned craniectomy margin, and these are connected. The sphenoid wing is fractured and removed to the superior orbital fissure. The dural edges are tacked up to bony margin and dura is opened in a stellate manner.



Duraplasty is crucial that dural closure be nonconstraining and loose to allow for further expansion of intra cranial contents. The recommendation of dimension of cranial vault removal is 10 x 15 cm craniectomy, with the lower margin extending to less than 1cm from middle cranial fossa. The lower margin of the craniectomy, relative to middle cranial fossa floor, directly correlates to the state of mesencephalic cisternal decompression[17].

Bifrontal craniectomy is most widely used approach in decompression of diffuse traumatic brain injury as described by Polin and colleagues[18]

COMPLICATIONS OF DC

I. Perioperative Complications

1. Blossoming of Contusions

Pre-Op Post-Op

Hemorrhagic expansion of contusions is inherent in the injury process and has been demonstrated on serial CT scanning in patients with TBI. In contrast, relief of the tamponade effect with bone removal in patients with severe TBI may facilitate growth and expansion of contusions following decompressive craniectomy

2. Evolution of Contralateral Mass Lesion.

Surgical decompressive craniectomy for TBI may incite a new mass lesion, contralateral or remote to the decompressed hemisphere. Reduction in ICP after decompression likely plays an important role.

Piepmeier and Wagner, however, have pointed out that if tamponade relief underlies contralateral bleeding, then one would expect delayed EDH lesions more frequently. Theoretically patients may be at higher risk for developing a contralateral EDH following decompressive craniectomy than following craniotomy. Decompressive surgery may relieve the tamponade effect on a contralateral bleeding site and predispose the patient to an EDH.

3. External Cerebral Herniation

Expansion of the brain with external cerebral herniation through the craniectomy defect is often observed in the early period after decompression. There is no consensus on how to measure external cerebral herniation. In the study by Yang et al., herniation through the craniectomy defect was measured at the middle of the cranial defect[19]. Herniation was defined as presence of brain tissue in the center of the bone defect > 1.5 cm above the plane where the outer table of the cranium would normally lie. The brain swelling may correspond to hyper perfusion, as detected by CT perfusion imaging[20]. In addition, loss of resistance in brain tissue lacking a protective skull invokes a higher hydrostatic pressure gradient that may permit transcapillary leakage of edema fluid[21]. include compression of cortical veins within the herniated segment of brain and subsequent venous infarction of the herniated tissue.

II. Postoperative Complications within 30 Days

1. Subdural hygromas

Decompressive craniectomy alters the dynamics of CSF circulation. This may exacerbate the occurrence of subdural hygromas and hydrocephalus. Subdural hygromas develop early after decompressive surgery. In the study by Aarabi et al, subdural hygromas developed in 25 (50%) of 50 patients after a mean of 8 days following decompressive craniectomy[15]. Hygromas are generally ipsilateral to the skull defect with volumes ranging from 10 to 120 ml (mean 51 ml). While most authors favor a mechanism of altered CSF dynamics to account for the occurrence of hygromas, others have suggested that increased cerebral perfusion pressure that accompanies decompressive craniectomy may play a role. Duraplasty at the time of decompression has been observed to lower the incidence of subdural effusions.

2. Paradoxical Herniation

3. Paradoxical herniation with compression of the brainstem and neurological deterioration may present in a delayed fashion after a lumbar puncture in patients with decompressive craniectomy. The concept that a negative pressure gradient between the cranial and spinal compartments, provoked by a spinal CSF leak, can precipitate downward herniation, even in the absence of raised ICP, has been carefully documented by many groups.

III. Delayed Complications after 1 Month

1. Wound Healing and Infection

   There are several factors associated with decompressive craniectomy that should lead one to expect a higher rate of infection than with standard craniotomy for general neurosurgical procedures. The incision varies but the typical, large, reverse question mark incision with a long scalp pedicle on a comparatively small base predisposes to wound breakdown along the parietal and posterior temporal limbs farthest along the flap. Bone removal is needed low in the temporal fossa to decompress the basal cisterns. To expose the scalp and temporalis muscle down to the level of the zygoma, the incision is carried to 1 cm below the zygoma anterior to the tragus. The urgency to decompress may not facilitate careful dissection and preservation of the superficial temporal artery. Sacrifice of the artery may impair perfusion of the scalp pedicle and negatively impact wound healing. The dura is not closed primarily; duraplasty using a dura substitute is associated with an increased risk of infection. If the dura is left open without duraplasty, a foreign synthetic material should be laid over the brain surface to prevent adherence of the scalp to the underlying brain.

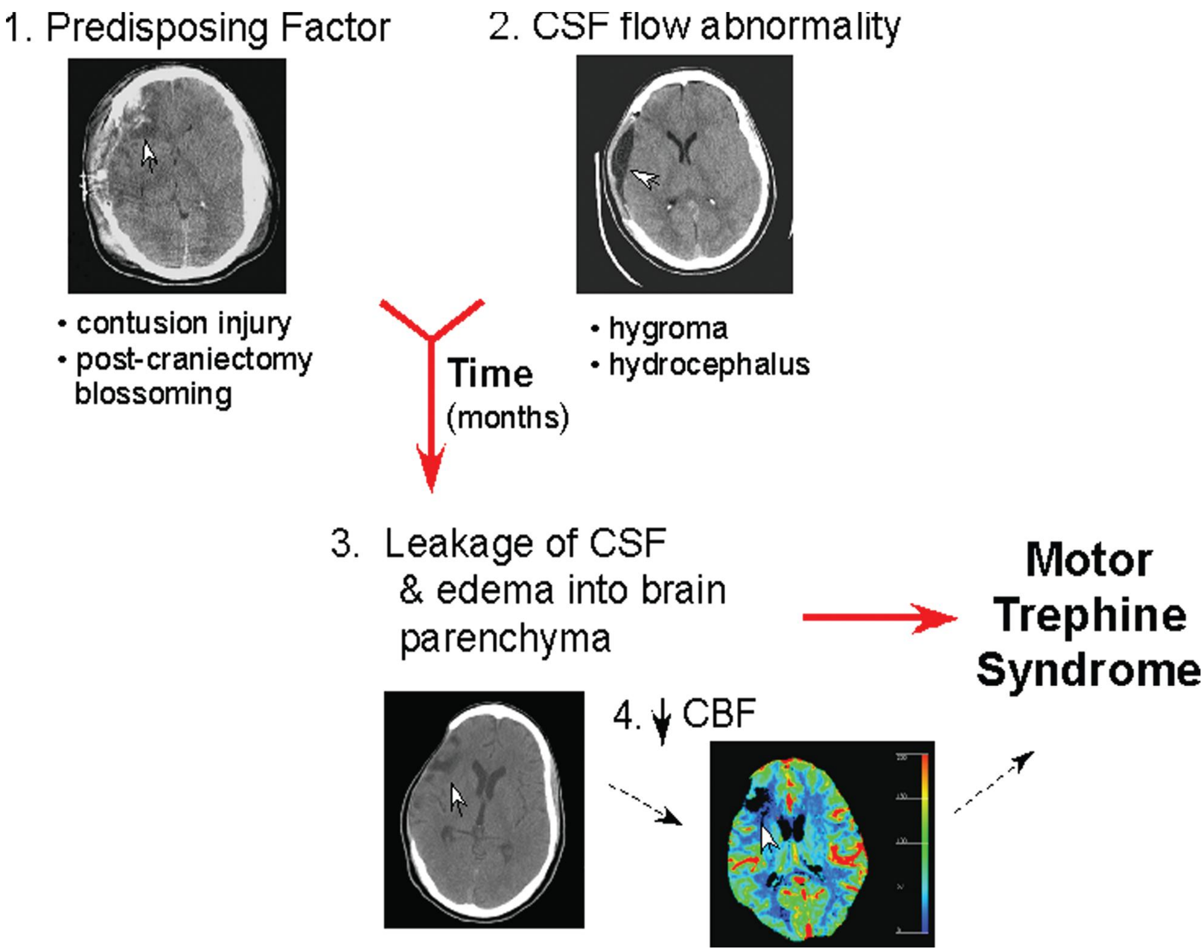
2. Hydrocephalus

Hydrocephalus and syndrome of the trephined are the most frequent complications of decompressive craniectomy beyond 1 month. Decompressive craniectomy has been identified as a risk factor for CSF alterations and development of posttraumatic hydrocephalus. Hydrocephalus has been associated with poorer outcome following TBI. Relatively few patients require ventriculoperitoneal shunt treatment before the bone flap has been replaced. Ventriculoperitoneal shunt treatment of hydrocephalus in the setting of a large cranial defect may also risk neurological deterioration consistent with a paradoxical herniation phenomenon.

3. Syndrome of the Trephined

Syndrome of the trephined is a frequent, delayed complication of decompressive craniectomy. Common symptoms include headaches, dizziness, irritability, concentration difficulty, memory problems, and mood disturbances, which typically arise weeks to months following decompressive craniectomy. The diagnosis is often overlooked, as many of these symptoms are also common sequelae to postconcussion and posttraumatic stress syndromes that accompany TBI. The pathophysiological mechanisms underlying syndrome of the trephined have been a subject of debated theories. Changes in atmospheric pressure, altered CSF circulation, and changes in CBF have all been proposed to explain the pathophysiology underlying the syndrome. In early studies, a sunken scalp was noted in many patients with syndrome of the trephined.

Schematic diagram of motor trephine syndrome[22]



4. Bone Resorption

In decompressive craniectomy, bone resorption of free bone flaps is common and may approach an incidence as high as 50% in long-term follow-up. Skull fractures identified at the time of the original decompression should raise concern for possible bone resorption following cranioplasty.

5. Persistent Vegetative State.

   Decompressive craniectomy is very effective in ameliorating raised ICP as a life-saving measure. While decompressive craniectomy reduces mortality, it may fail to rescue neurological function from devastating injury incurred by either the primary impact or secondary damage that evolves during the early resuscitation period. Risks of survival with an outcome of a persistent vegetative state after decompressive craniectomy have been reported to range upwards of15 to 20% in many series. Preoperative GCS scores < 6, brainstem dysfunction, older age, and longer time to decompression have been reported to be associated with a higher risk of persistent vegetative outcome[23].

OUTCOME FOLLOWING DECOMPRESSIVE CRANIECTOMY FOR TBI

Early reported results of DC performed on TBI were not very encouraging. However, recently, the use of DC has regained popularity as a treatment modality of TBI with associated increased ICP, refractory to medical treatment. Furthermore, some authors advocate that DC could be performed prophylactically, especially in developing countries, where neurointensive care resources and ICP monitoring may not be readily available[24].

There is no Class I evidence to support the use of DC, and prospective studies are being organized by both the European and American Brain Injury Consortiums. There are many studies in the literature with Class II and III evidence that have shown that DC might play a role in severe brain injury refractory to medical therapy. Our understanding of the different factors that determine prognosis after severe brain injury has allowed for improvement in the management of brain injury.

In 2001, a small randomized study originating from the Royal Children's Hospital in Melbourne was published[25]. Patients were randomized to standard treatment alone or with decompression. Those in the standard treatment group had a mean ICP reduction of 3.7 mm Hg and a favorable outcome (normal or mild disability) in 14%; patients in the standard treatment plus decompression (performed at 19 hours post injury) group had a mean ICP reduction of 8.9 mm Hg and a favourable outcome rate of 54%.[16] Two multicenter prospective randomized studies are ongoing: the RESCUEicp study and the DECRA study.

Literature Summary of outcome following DC in TBI

Authors & Year	Indication	Patients%			
		Favourable	Severe disability	Vegetative	Mortality
Aarabi et al., 2006	Primary (20%) & Secondary (80%)	40%	18%	14%	28%
Guerra et al, 1999	Primary (68%) & Secondary(32%)	58%	12%	9%	19%
Howard et al, 2008	Primary (60%) &secondary(40%)	30%	15%	-	55%
Huang et al, 2008	Primary DC	76%	5%	5%	13%
Jiang et al, 2005	Primary DC	40%	30%	4%	26%
Meier et al,2006	Primary (63%) &secondary(37%)	26%	20%	14%	40%
Munch et al, 2000	Primary (63%) &secondary(37%)	20%	33%	14%	33%

MATERIALS AND METHODS

It is a prospective analytical study; study period is from August 2009 to February 2012 in the Institute of Neurology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai. All Patients admitted in our hospital trauma ward with moderate to severe head injury who are undergoing primary decompressive craniectomy according to brain trauma foundation guidelines are included in this study. Categorization of head injury severity is based on Glasgow coma scale (GCS) score, GCS 9-13=moderate, GCS 3-8=severe.

Inclusion criteria

- Age 12-70 years and within first 48 hrs from time of injury.
- Only traumatic causes.
- Post resuscitation GCS 4-13.
- CT scan with evidence of Acute SDH, unilobar or multilobar contusions with diffuse cerebral edema, midline shift >5mm, and effacement of basal cisterns.

Exclusion criteria

- Age less than 12 years and more than 70 years.

- Nontraumatic causes like infarct, spontaneous ICH or aneurysmal bleed.
- Post resuscitation GCS 3.
- Bilateral fixed and dilated pupils.
- Absent brain stem reflexes.
- Devastating injury not expected to survive for 24 hrs.
- Patients who are not willing for surgery or study.

All patients were initially seen in our emergency services. Hemodynamic stabilization and intubation was done where necessary and the post resuscitation GCS was noted. A CT scan was done as soon as possible. Patients with moderate to severe head injury requiring decompressive craniectomy considered for this trail, entry will be determined using the above inclusion and exclusion criteria after resuscitation, and data were entered in proforma. Consent for surgery and study was obtained from next of kin after detail explanation about the study. Approval for the study was obtained from the ethics committee. After the surgery patient treated in head injury ICU, then CT scan brain was done with in 24hr to 48 hrs and compared to pre op CT scan. The postoperative GCS and GOCS (Glasgow outcome score) at discharge from the hospital were noted, primary and secondary outcomes were analyzed.

Primary outcome measures:

- Proportion(%) of favourable outcomes (GOCS4&5), unfavourable outcome (GOCS1,2&3)

Secondary outcome measures:

- Assessing post op GCS, adequacy of bone removal, reduction of mid line shift, basal cisterns compression, residual hematomas in post op CT scan and complications.

The clinical parameters analyzed in relation to the outcome were age, sex, mode of injury, GCS after resuscitation, pupillary status, associated major injuries and the time interval between trauma and surgery. Abnormalities in size and light reflex were considered as abnormal pupil. The variables analyzed on CT scan were the midline shift, status of basal cisterns, presence of residual hematomas and adequacy of bone removal. The midline shift was measured as the largest perpendicular distance between an imaginary reference line joining the frontal crest and internal occipital protuberance and the most shifted point of the septum pellucidum. Suprasellar and perimesencephalic cisterns were taken for basal cistern assessment. The extent of craniotomy and the details of duraplasty were noted. Adequacy of bone removal was measured by the margins of craniectomy in CT scan: anteriorly to the superior border of orbital roof; posteriorly to at least 2cm lateral to the external auditory meatus; medially to 2cm lateral to midline; and inferiorly to the floor of middle cranial fossa.

Statistical analysis

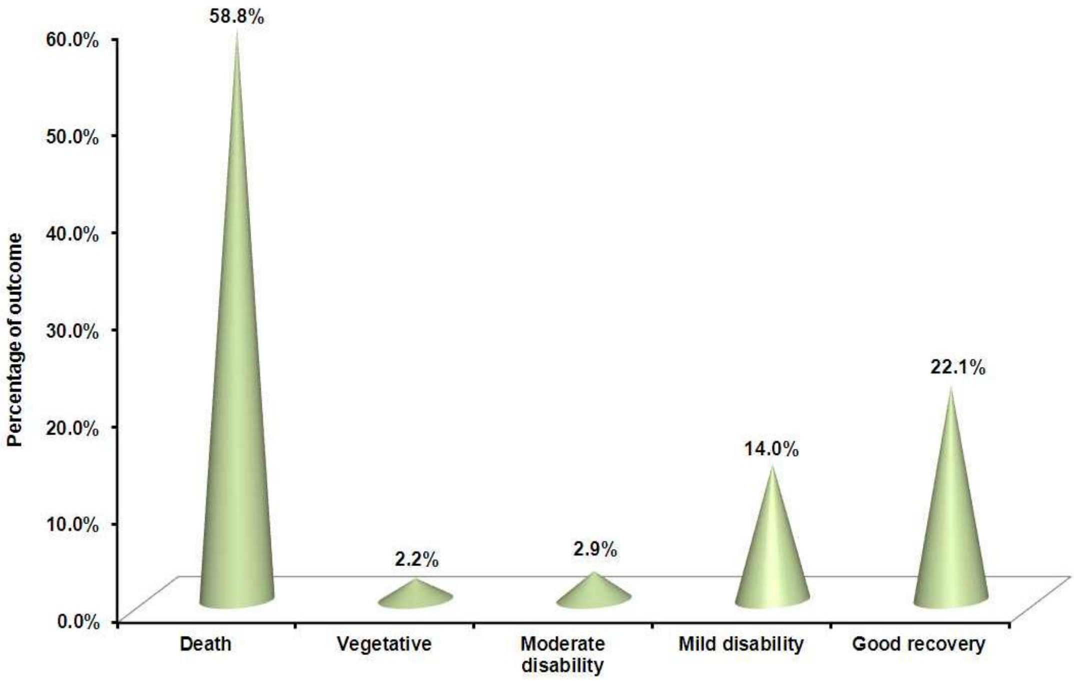
Statistical analysis was performed by using MANOVA test. Multivariate analysis of variance (MANOVA) is a statistical test procedure for comparing multivariate means of several groups. A statistically significant difference was indicated by a p-value of less than 0.05.

RESULTS

Table 1: Outcome distribution

	Outcome GOCS	No of patients	Percentage
1	Death	80	58.8%
2	Vegetative	3	2.2%
3	Moderate disability	4	2.9%
4	Mild disability	19	14.0%
5	Good recovery	30	22.1%
	Total	136	100.0%

Spectrum of Outcome



Favourable outcome rate was 36.1% (GOS 4 & 5), unfavourable outcome rate was 63.9 % (GOS 1, 2 & 3).

Table 2: Survival / death distribution

Survival/Death	No of patients
Survival	56
Death	80
Total	136

Percentage of survival & death

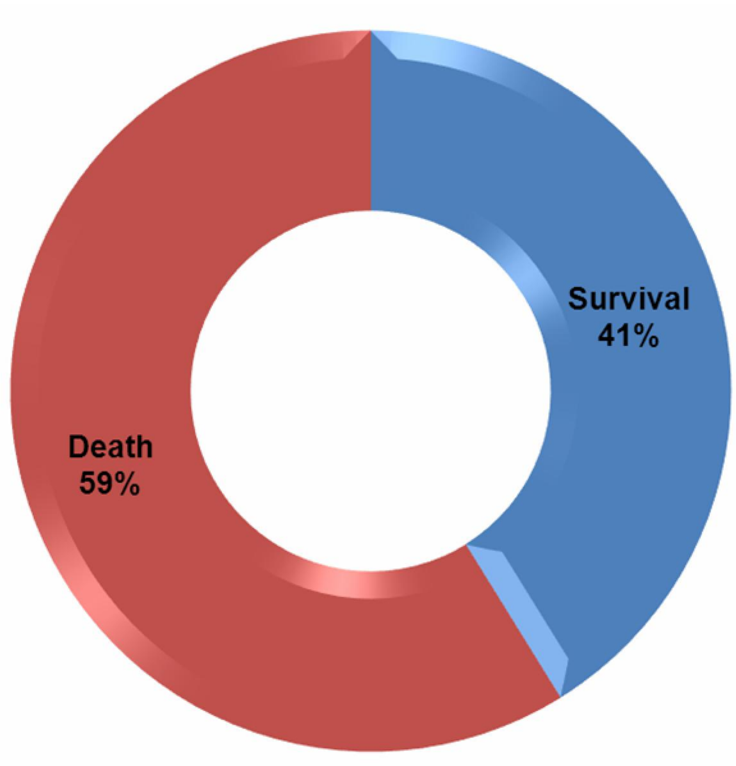


Table 3a: Age Distribution

AGE	No of patients
< = 20	3
21 - 30	34
31 - 40	35
41 - 50	32
51 - 60	20
61 - 70	12
Total	136

Number of patients in each age group

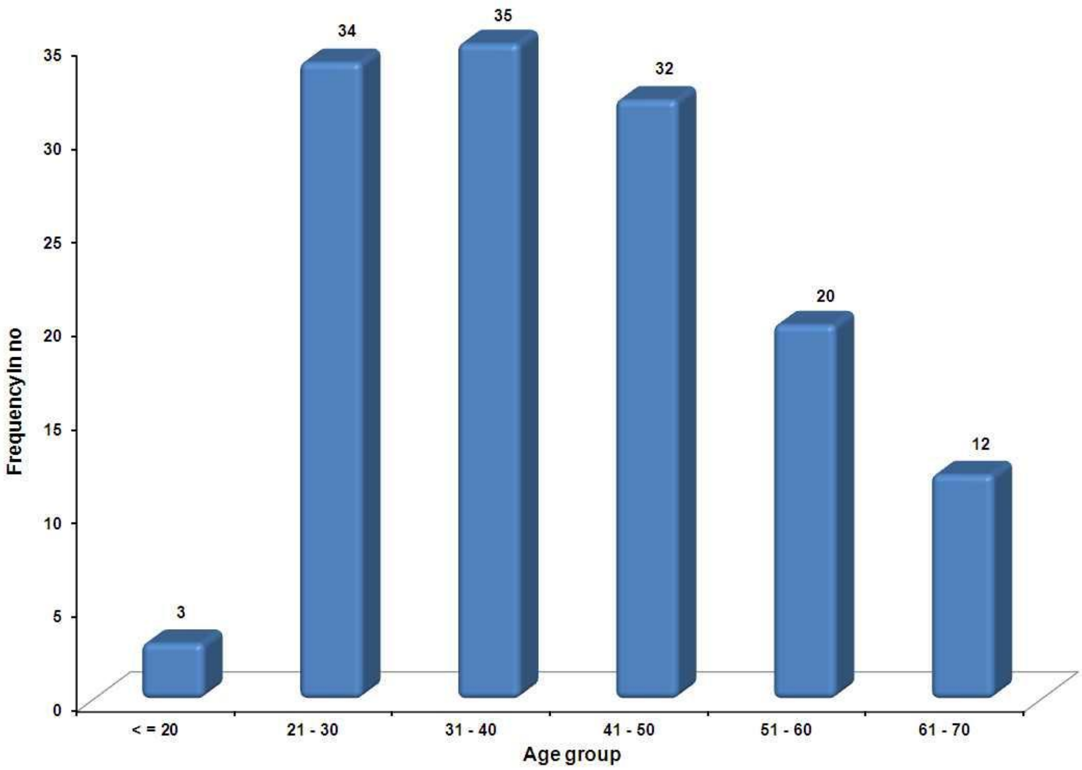
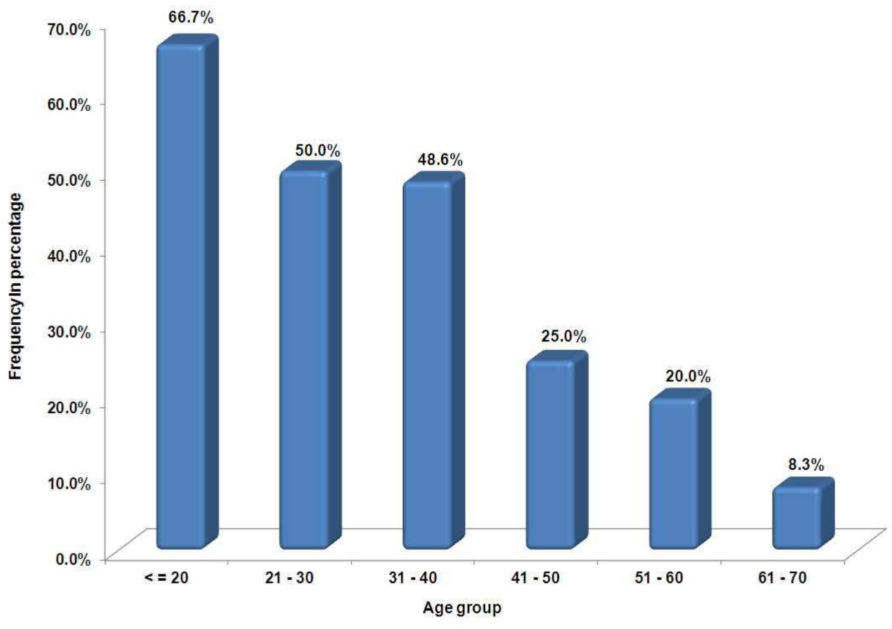


Table 3b: Age vs. outcome

AGE	No of patients		Total
	Favourable outcome	Unfavourable outcome	
< = 20	2	1	3
21 - 30	17	17	34
31 - 40	17	18	35
41 - 50	8	24	32
51 - 60	4	16	20
61 - 70	1	11	12
Total	49	87	136

Percentage of favourable outcome

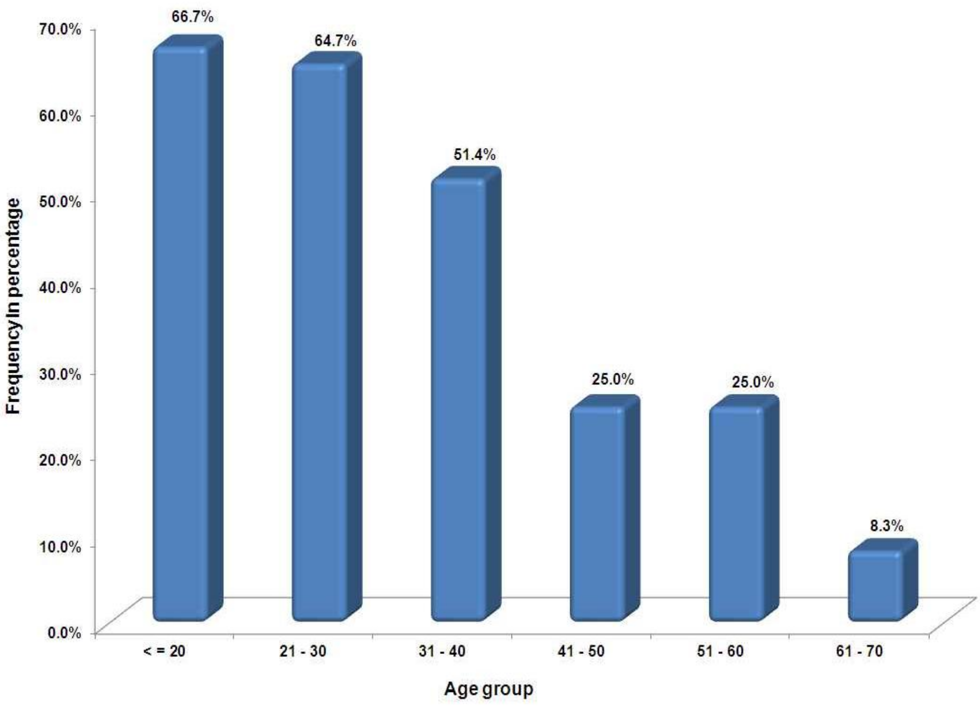


There is statistical significance between Age and outcome (P-value – 0.001 < 0.05).Above data showed lesser age group had better outcome.

Table 3c: Age vs. Survival / Death

AGE	No of patients		Total
	Survival	Death	
< = 20	1	2	3
21 - 30	12	22	34
31 - 40	17	18	35
41 - 50	24	8	32
51 - 60	15	5	20
61 - 70	11	1	12
Total	56	80	136

Percentage of Survival



There is statistical significance between Age and survival (P-value – 0.000 < 0.05)

Table 4a : Distribution of gender

Sex	No of patients
Male	123
Female	13
Total	136

Percentage of Gender Distribution

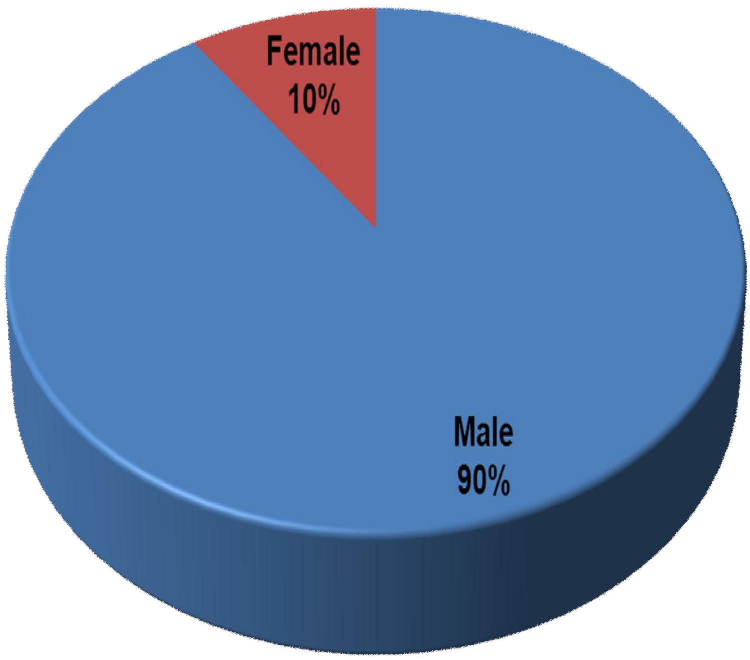
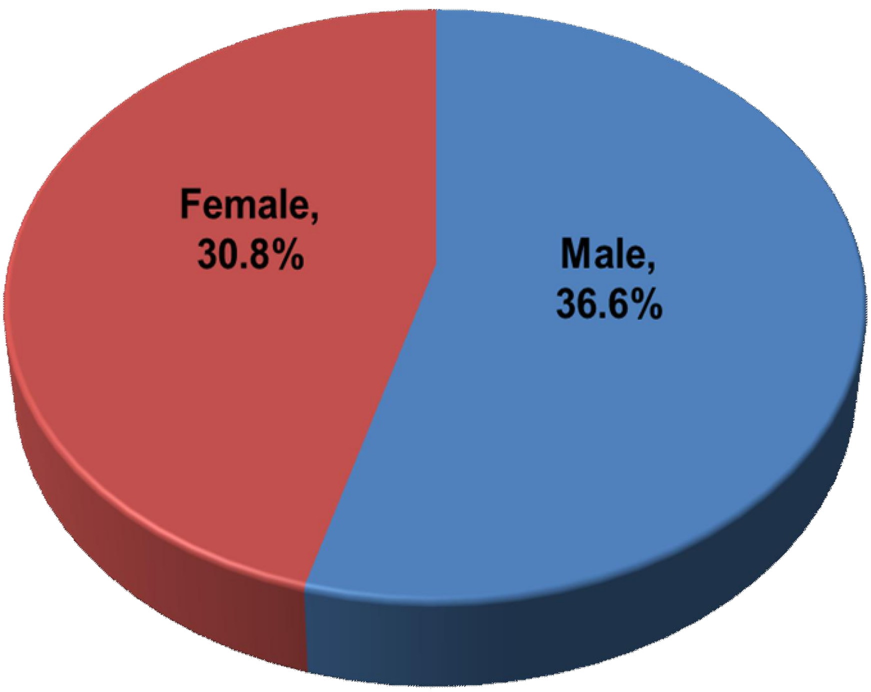


Table 4b : Sex vs. Outcome

Sex	No of patients		Total
	Favourable Outcome	Unfavourable Outcome	
Male	45	78	123
Female	4	9	13
Total	49	87	136

Percentage of favourable outcome



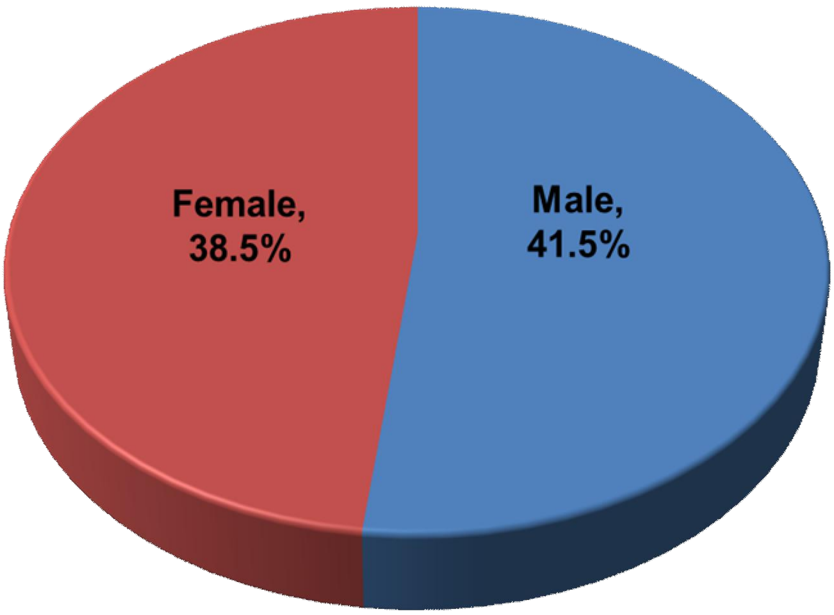
There is no statistical significant between sex and outcome

(P-value – 0.508> 0.05)

Table 4c :Sex vs. Survival / Death

Sex	No of patients		Total
	Survival	Death	
Male	51	72	123
Female	5	8	13
Total	56	80	136

Percentage of Survival



There is no statistical significance between sex and survival (P-value – 0.836 > 0.05)

Table 5a :Distribution of mode of injury (MOI)

MOI	No of patients
RTA	103
Fall	26
Assault	7
Total	**136**

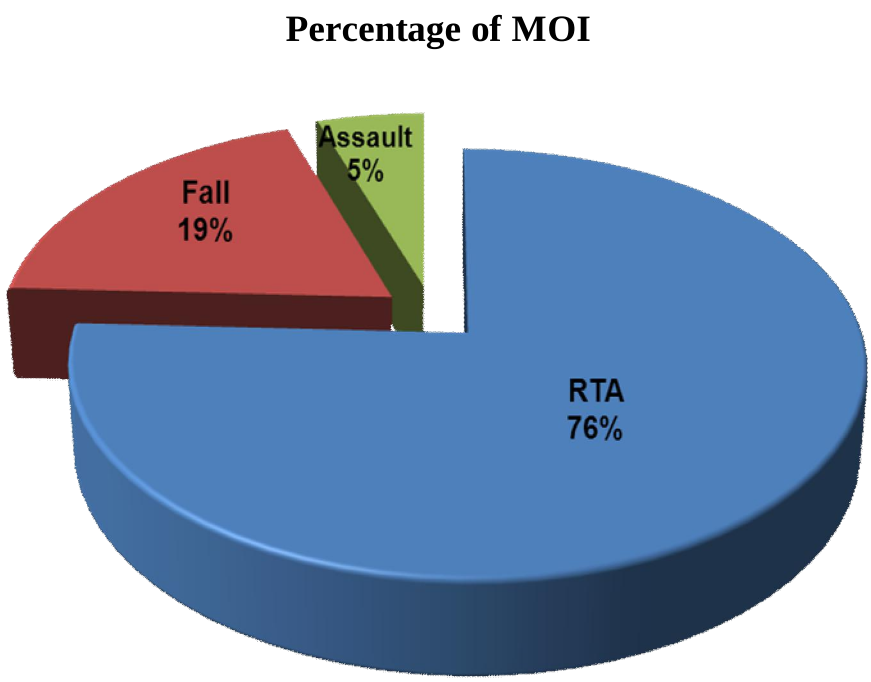
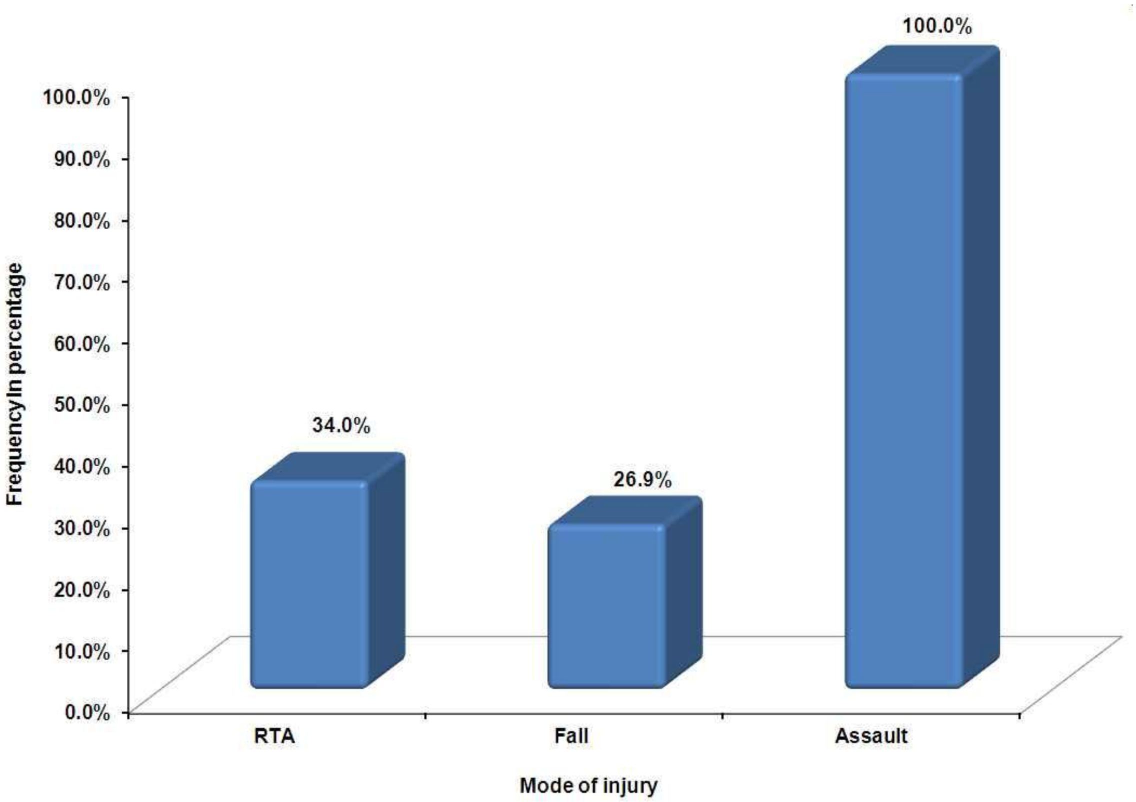


Table 5b :MOI vs. Outcome

MOI	No of patients		Total
	Favourable outcome	Unfavourable outcome	
RTA	35	68	103
Fall	7	19	26
Assault	7	0	7
Total	49	87	136

Percentage of favourable outcome

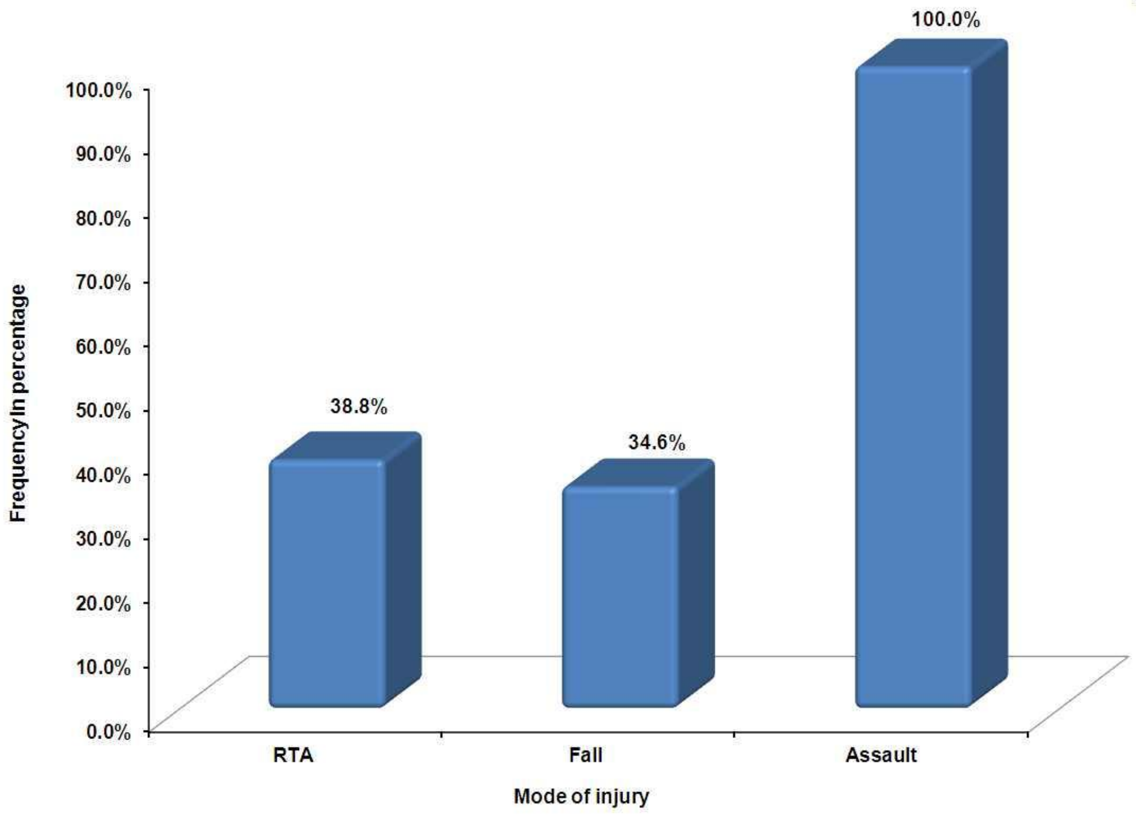


There is statistical significance between MOI and outcome (P-value – 0.000 < 0.05)

Table 5c :MOI vs. Survival / Death

MOI	No of patients		Total
	Survival	**Death**	
RTA	40	63	103
Fall	9	17	26
Assault	7	0	7
Total	56	80	136

Percentage of Survival



There is statistical significance between MOI and survival (P-value – 0.004 < 0.05)

Table 6a :Time of surgery distribution

Time of surgery	No of patients
< 24hrs	119
> 24hrs	17
Total	136

Percentage of time of surgery

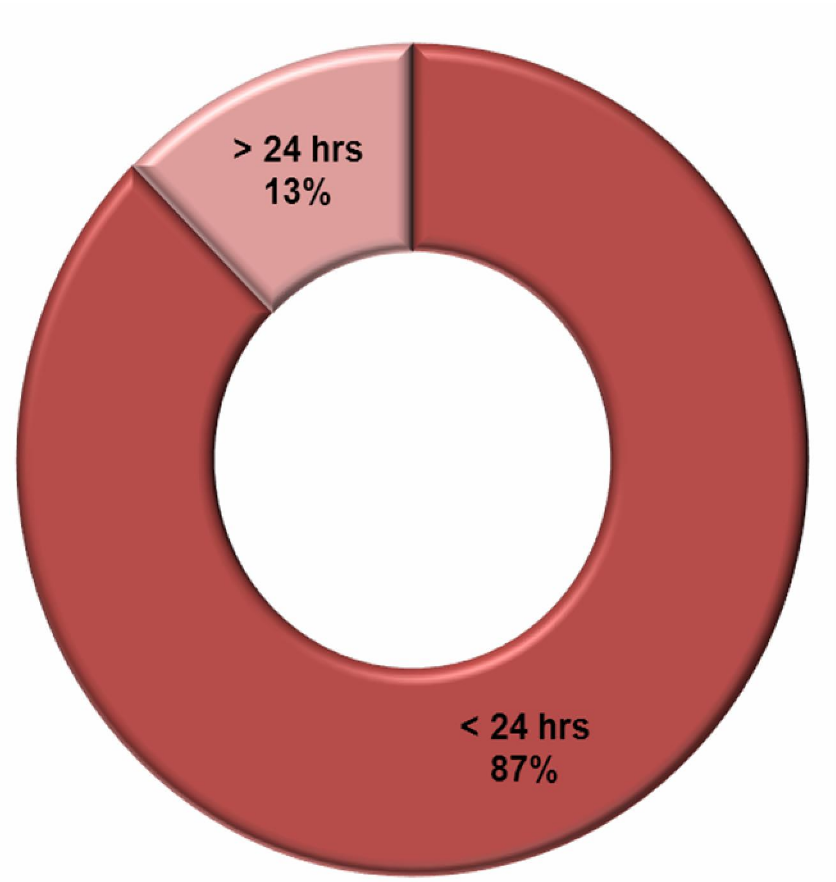
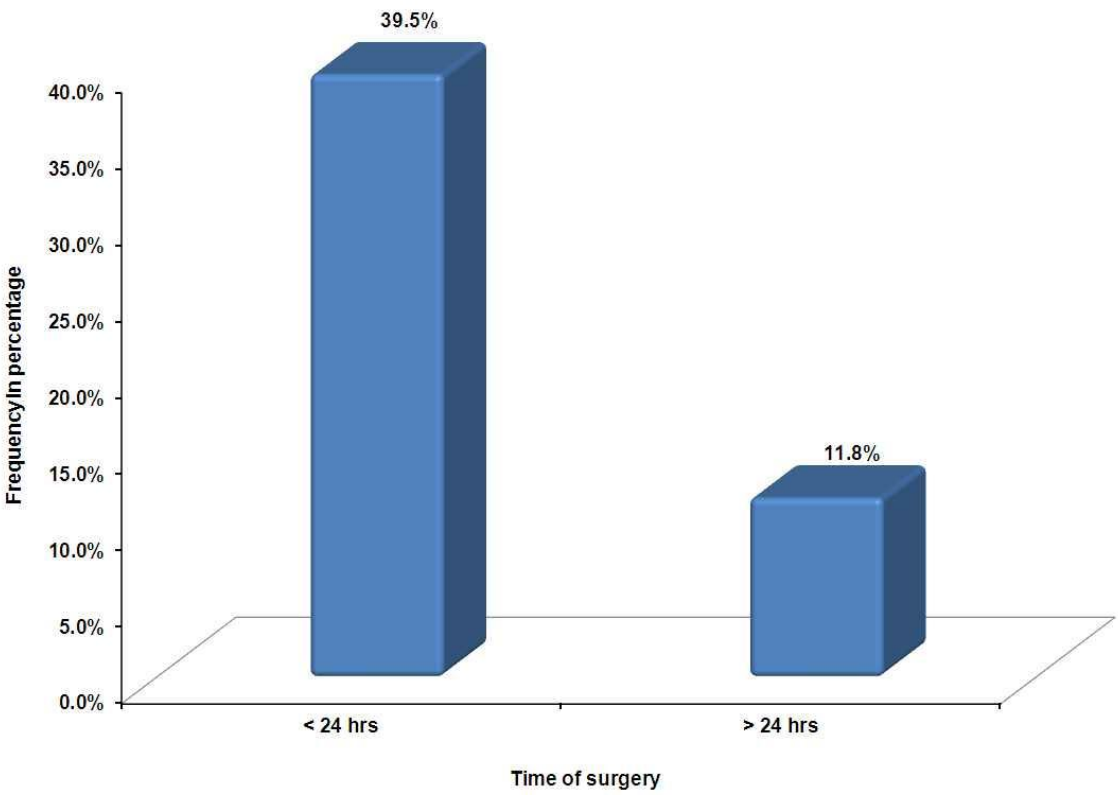


Table 6b :Time of surgery Vs. Outcome

Time of surgery	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
< 24hrs	47	72	119
> 24hrs	2	15	17
Total	49	87	136

Percentage of favourable outcome

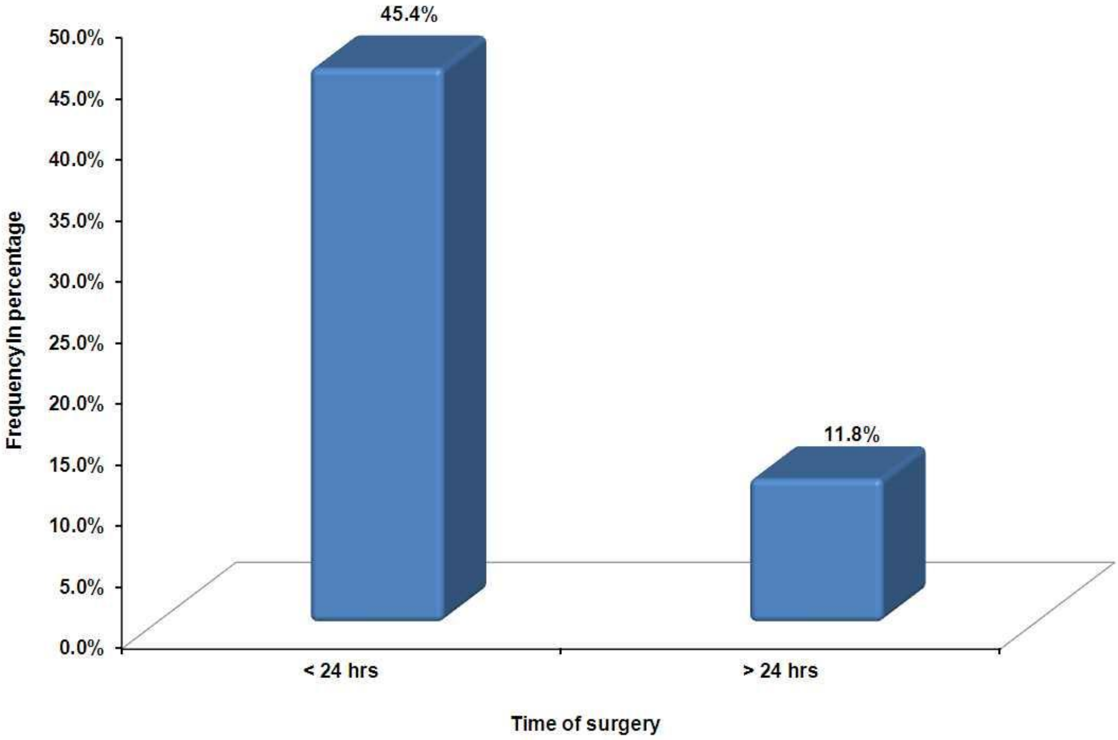


There is statistical significant difference between time of surgery and Outcome (P-value – 0.009 < 0.05)

Table 6c :Time of surgery Vs. Survival / Death

Time of surgery	No of patients		Total
	Survival	**Death**	
< 24hrs	54	65	119
> 24hrs	2	15	17
Total	56	80	136

Percentage of survival



There is statistical significance between time of surgery and survival (P-value – 0.008 < 0.05)

Table 7a :Associated Injury

Associated Injury	No of patients
present	30
Not present	106
Total	136

Persentage of associated injury

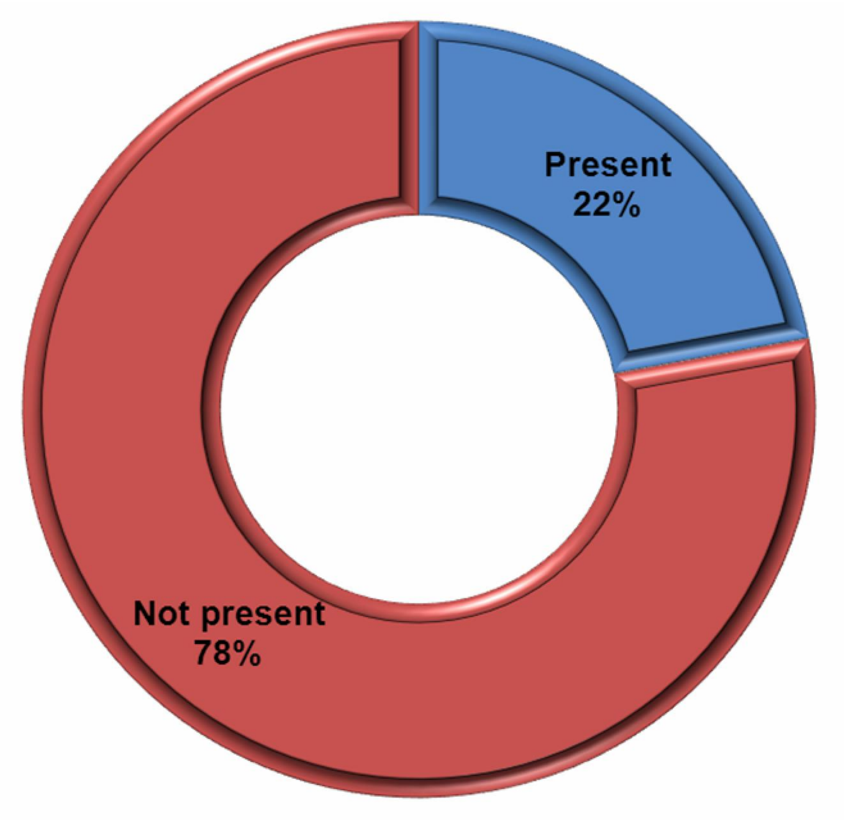
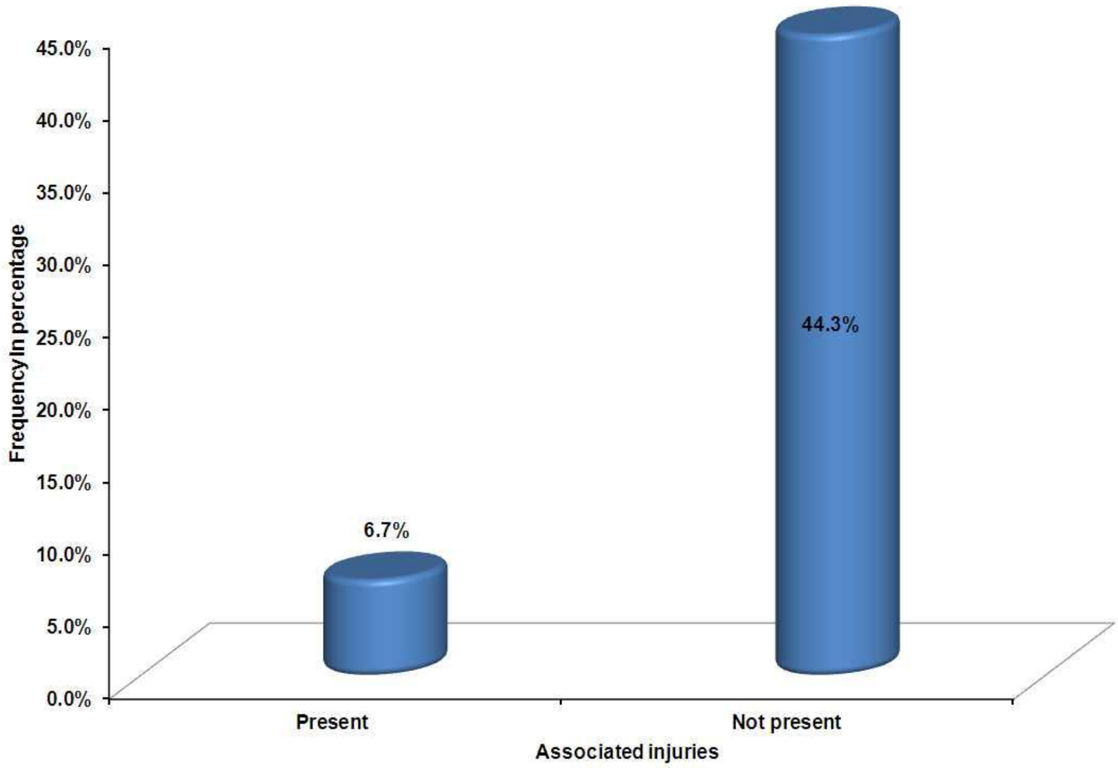


Table 7b :Associated injury vs outcome

Associated Injury	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
present	2	28	30
Not present	47	59	106
Total	49	87	136

Percentage of favourable outcome

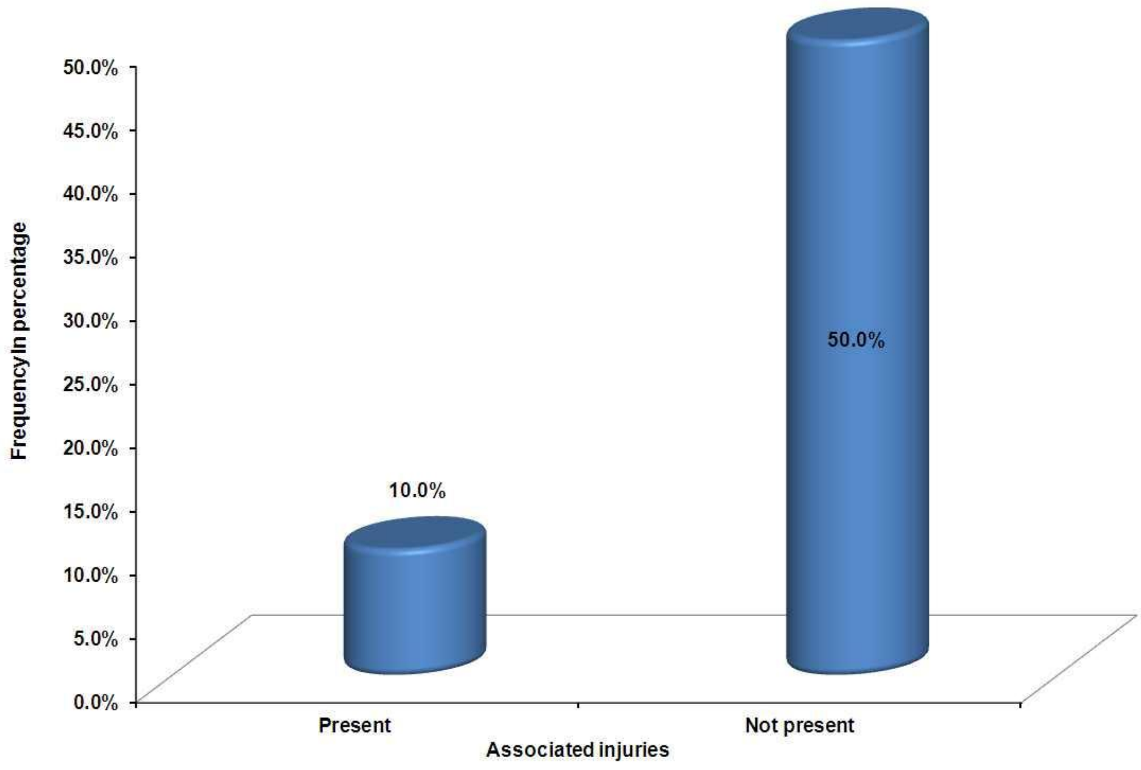


There is statistical significance between associated injury and outcome (P-value – 0.000 < 0.05)

Table 7c :Associated injury Vs. Survival / Death

Associate Injury	No of patients		Total
	Survival	Death	
present	3	27	30
Not present	53	53	106
Total	56	80	136

Percentage of survival



There is statistical significance between Associate injury and survival (P-value – 0.000 < 0.05)

Table 8a :Pre-operative GCS distribution

Pre-op GCS	No of patients
4 - 8	82
9 - 13	54
Total	136

Percentage of Pre Op GCS

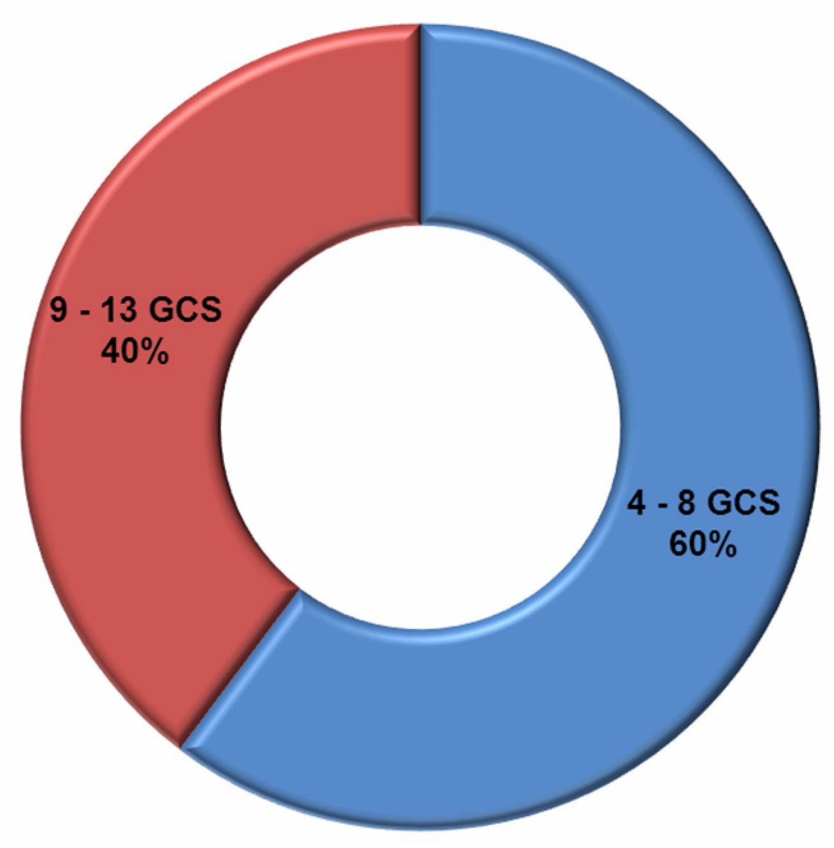
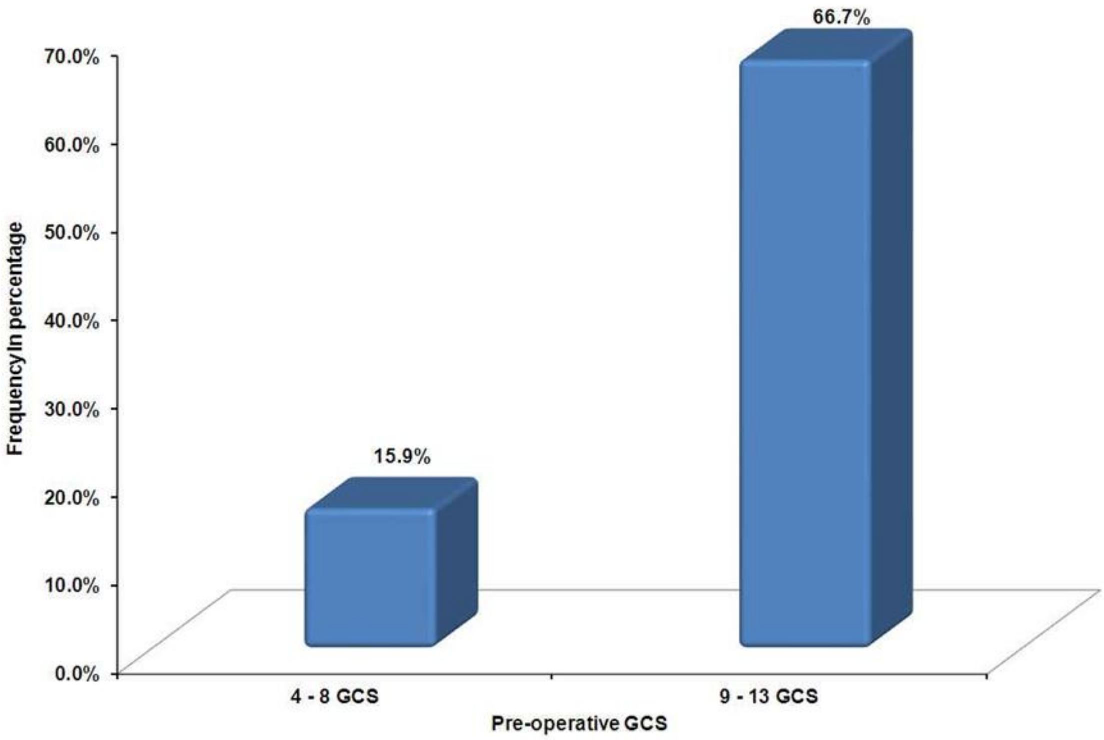


Table 8b :Pre-operative GCS vs. outcome

Pre-operative GCS	No of patients		Total
	Favourable Outcome	Unfavourable Outcome	
4 – 8	13	69	82
9 – 13	36	18	54
Total	49	87	136

Percentage of favourable outcome

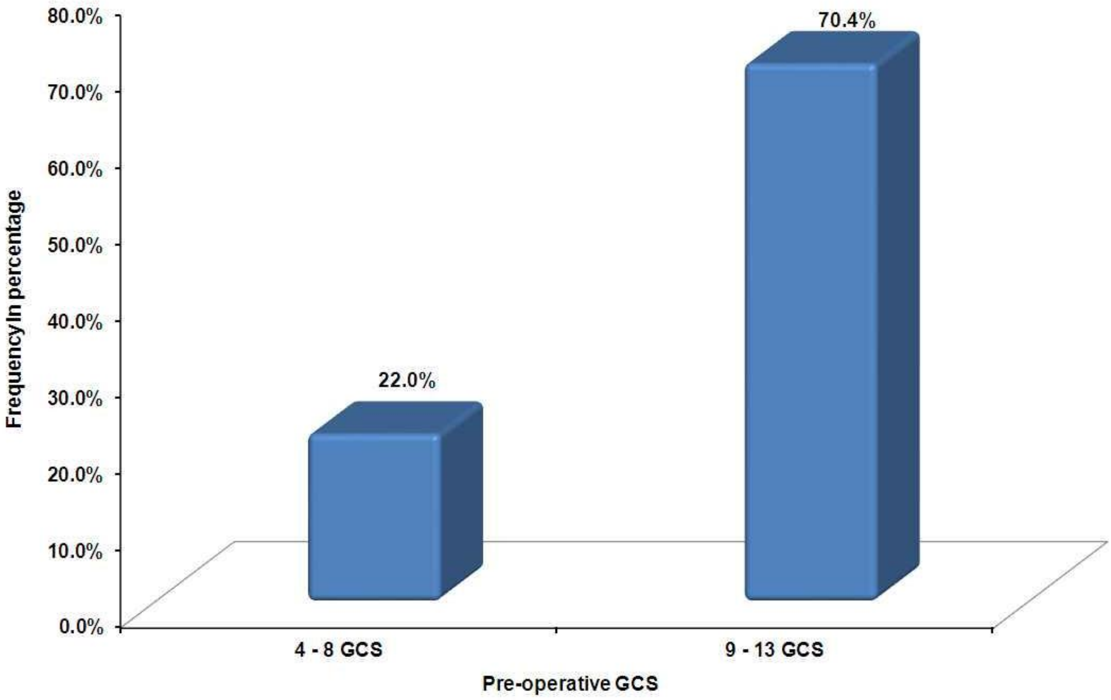


There is statistical significance between pre-operative GCS and outcome (P-value – 0.000 < 0.05)

Table 8c : Pre-operative GCS Vs. Survival / Death

Pre-op GCS	No of patients		Total
	Survival	Death	
4 - 8	18	64	82
9 - 13	38	16	54
Total	56	80	136

Percentage of survival



There is statistical difference between pre-operative and survival (P-value – 0.000 < 0.05)

Table 9 a : Pupillary Reaction distribution

Pupillary Reaction	No of patients
Normal	23
Abnormal	113
Total	136

Perentage of Pupillary Reaction distribution

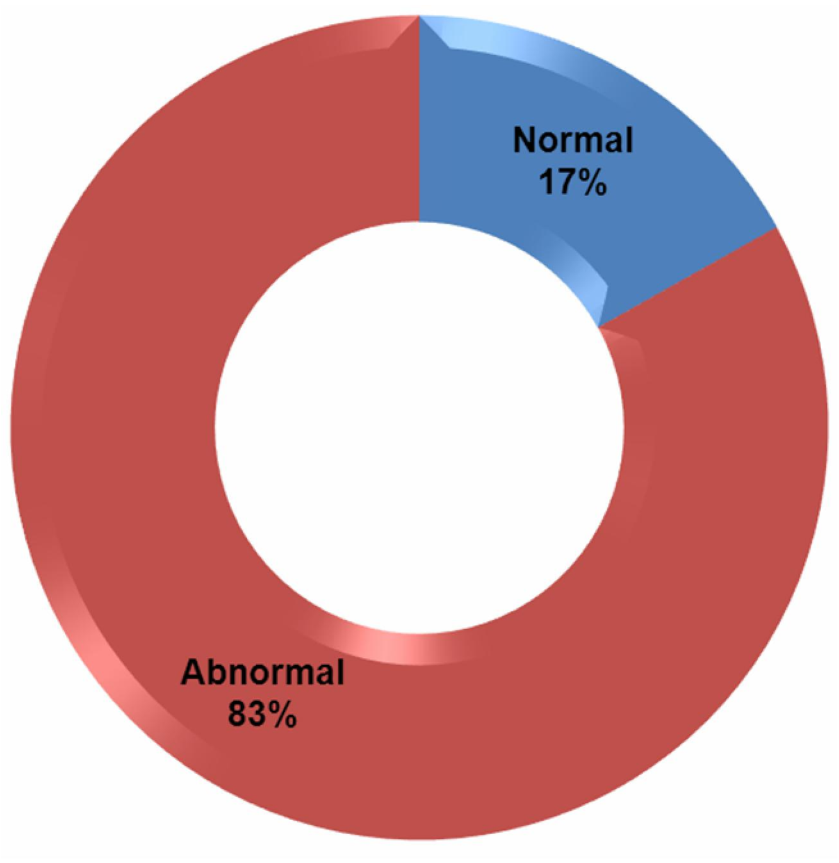
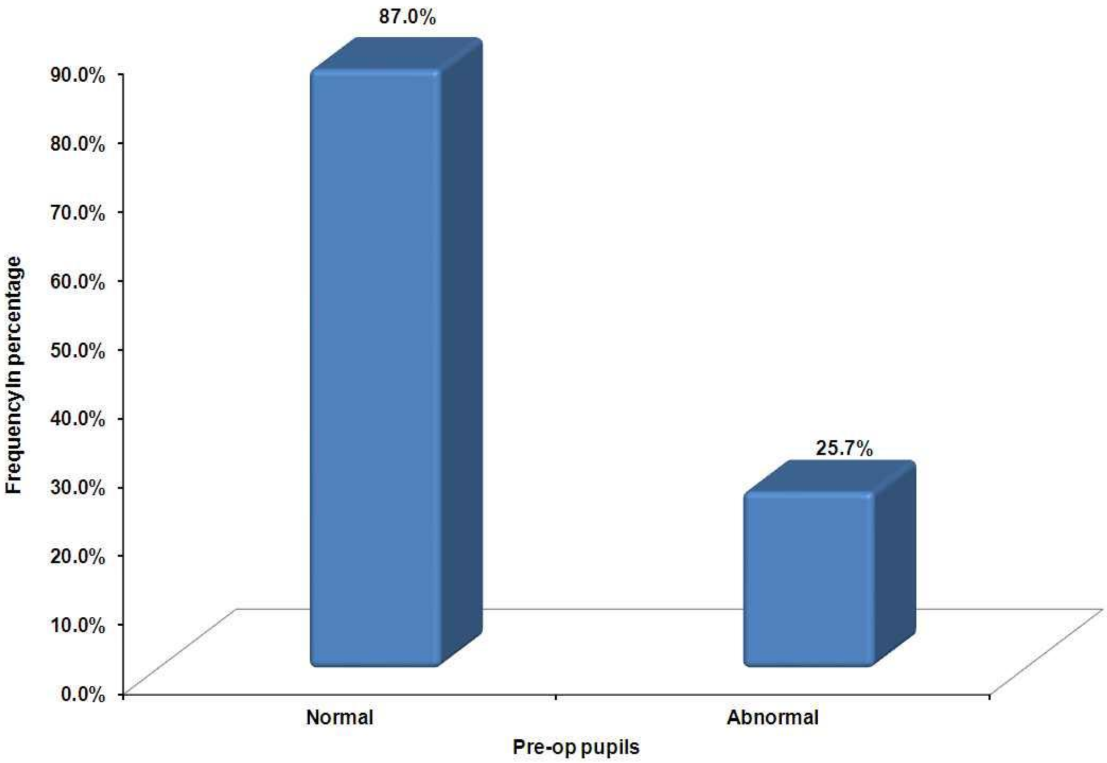


Table 9 b : Pupillary Reaction vs. outcome

Pre-op pupils	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
Normal	20	3	23
Abnormal	29	84	113
Total	49	87	136

Percentage of favourable outcome

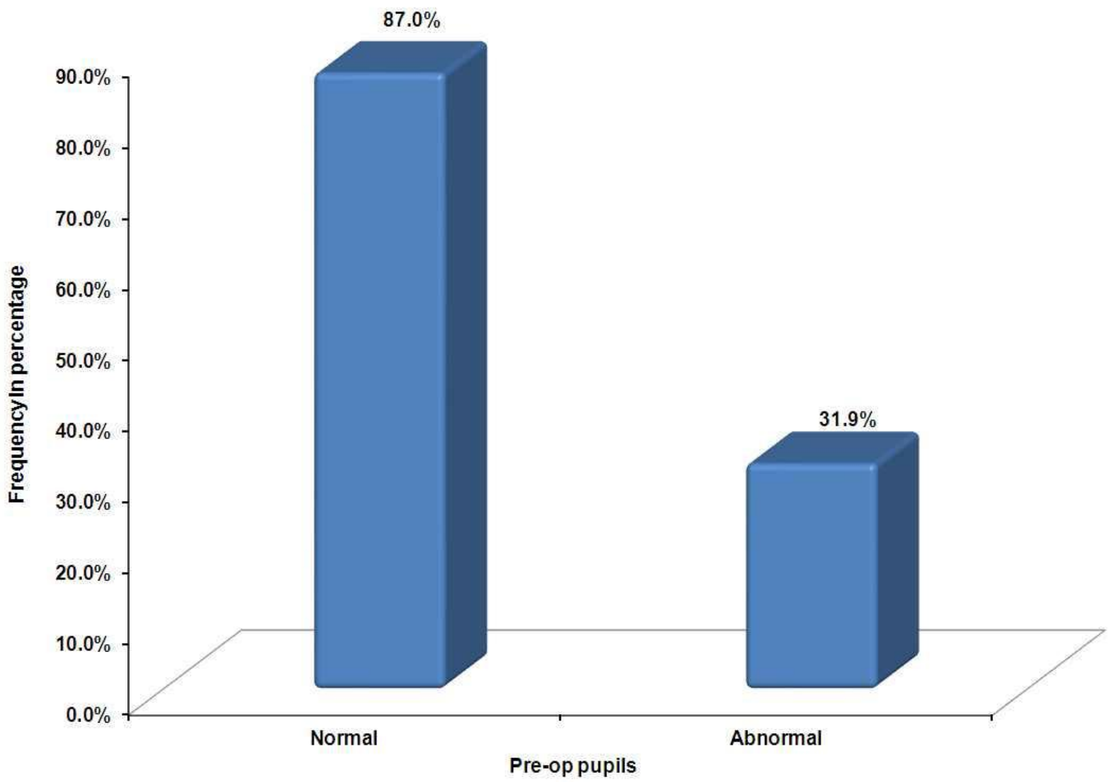


There is statistical significance between Pupillary reaction and outcome (P-value – 0.000 < 0.05)

Table 9 c : Pupillary Reaction Vs. Survival / Death

Pre-op pupils	No of patients		Total
	Survival	Death	
Normal	20	3	23
Abnormal	36	77	113
Total	56	80	136

Percentage of survival



There is statistical significance between pupillary reaction and survival (P-value – 0.000 < 0.05)

Table 10 a : Post op Shift

Post op Shift	No of patients
Not reduced	41
Reduced	95
Total	136

Percentage of Post op shift

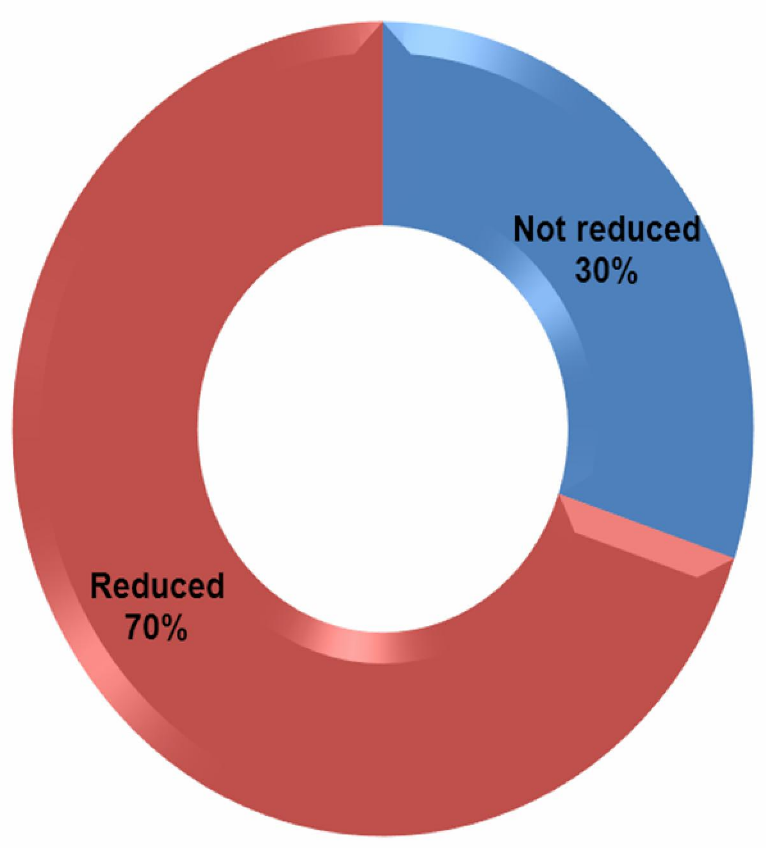
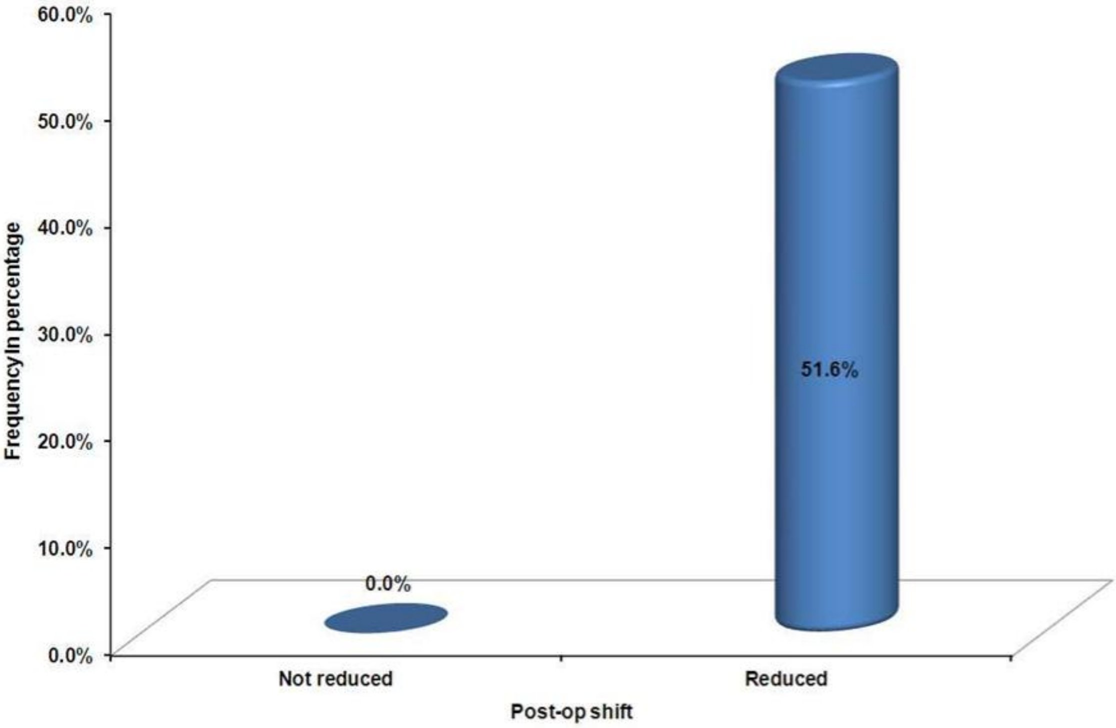


Table 10 b : Post op shift vs outcome

Post-op shift	No of patients		Total
	Favourable Outcome	Unfavourable Outcome	
Not reduced	0	41	41
Reduced	49	46	95
Total	49	87	136

Percentage of favourable outcome

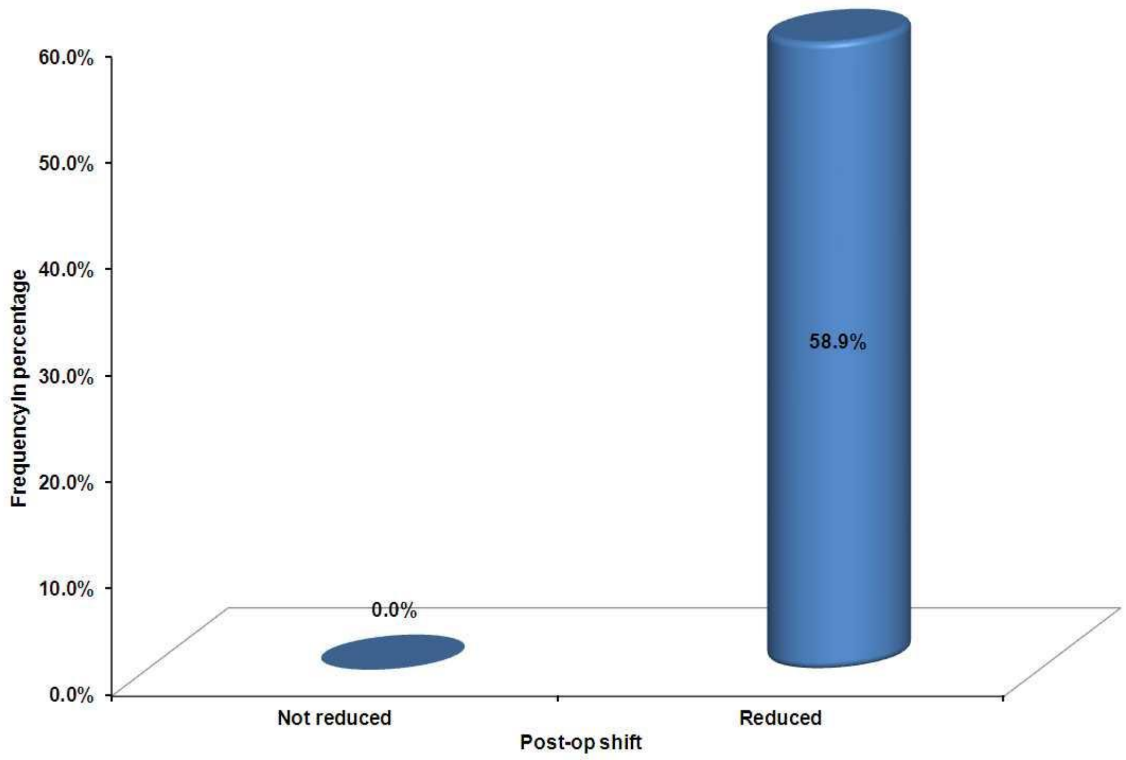


There is no statistical significance between post operative Shift and outcome (P-value – 0.062 > 0.05)

Table 10 c : Post op shift vs Survival / Death

Post-op shift	No of patients		Total
	Survival	Death	
Not reduced	0	41	41
Reduced	56	39	95
Total	56	80	136

Percentage of survival



There is no statistical significance between post op Shift and survival (P-value – 0.166 > 0.05)

Table 11 a :Post op B.Cisterns

B.Cisterns	No of patients
Not opened	91
Opened	45
Total	136

Percentage of Post op B.Cisterns

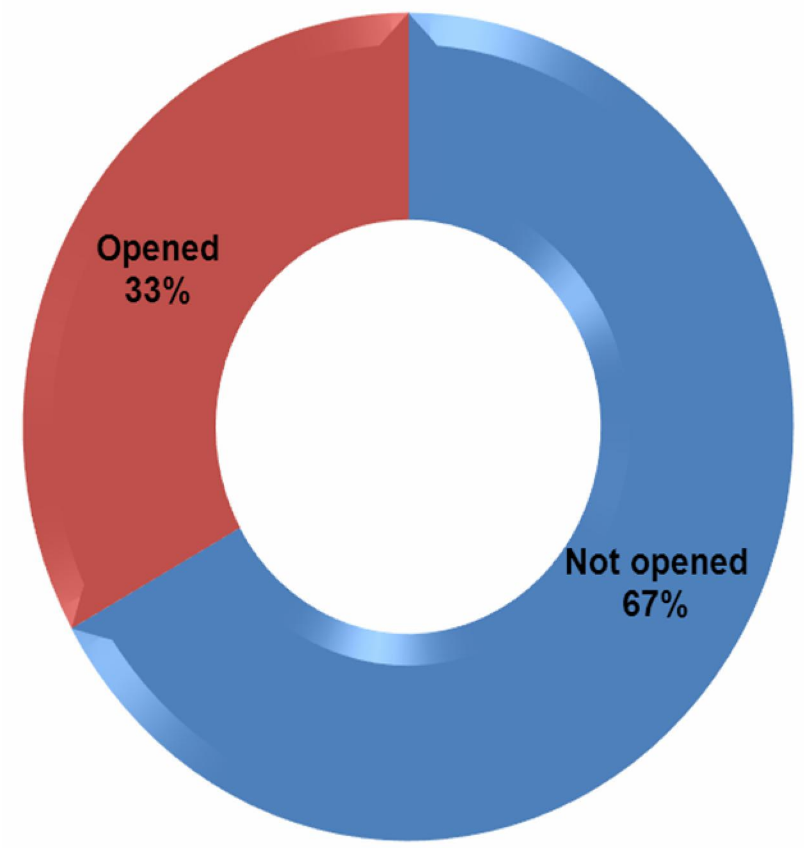
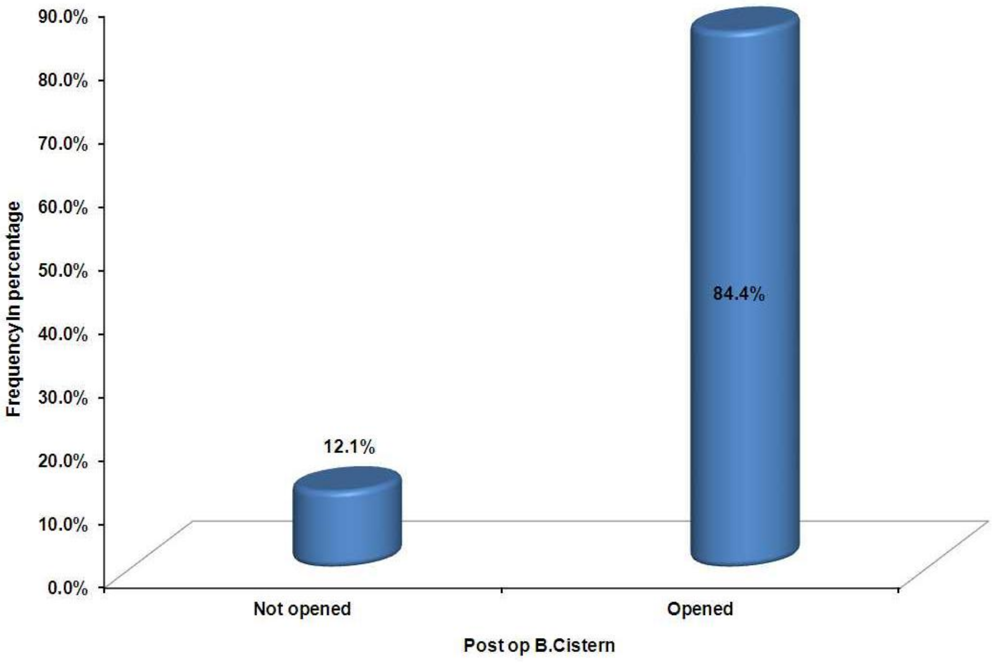


Table 11 b : Post op B.Cistern Vs. Outcome

B.Cistern	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
Not opened	11	80	91
Opened	38	7	45
Total	49	87	136

Percentage of favourable outcome

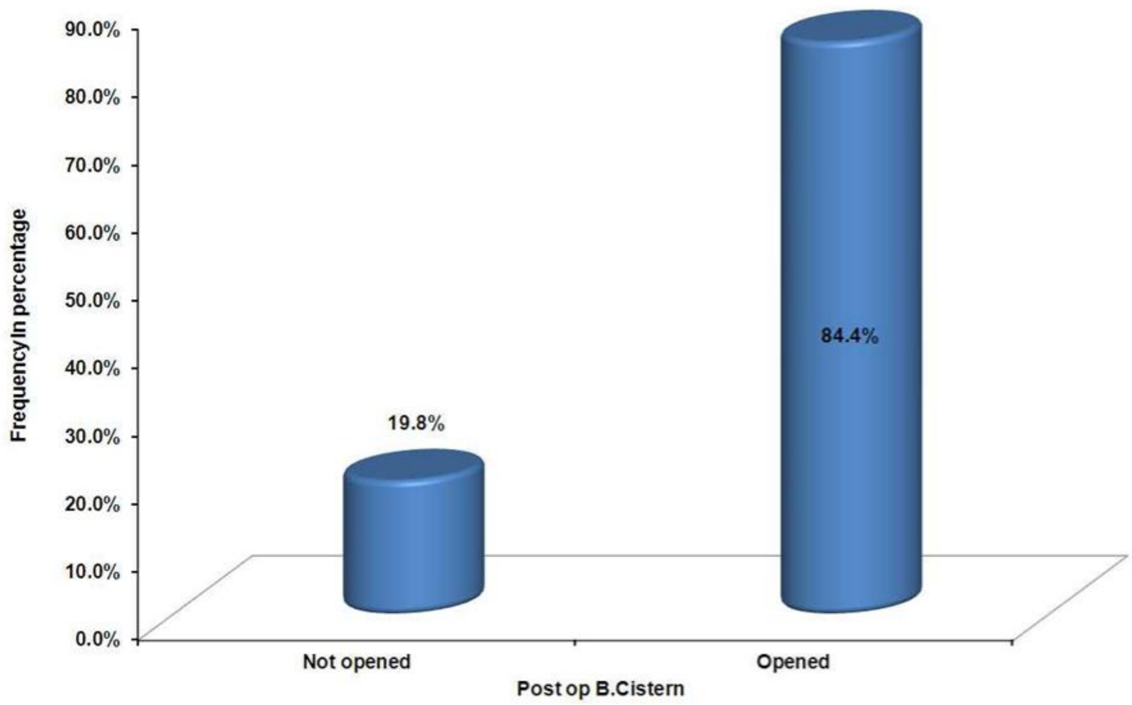


There is statistical significance between post op B.Cistern opening and outcome (P-value – 0.000 < 0.05) – MANOVA

Table 11 c : Basalcistern vs Survival / Death

B.Cistern	No of patients		Total
	Survival	Death	
Not opened	18	73	91
opened	38	7	45
Total	56	80	136

Percentage of survival



There is statistical difference between post op Basal Cistern and survival (P-value – 0.000< 0.05)

Table 12 a :Bone removal distribution

Bone Removal	No of patients
Adequate	126
Inadequate	10
Total	136

Percentage of Bone removal distribution

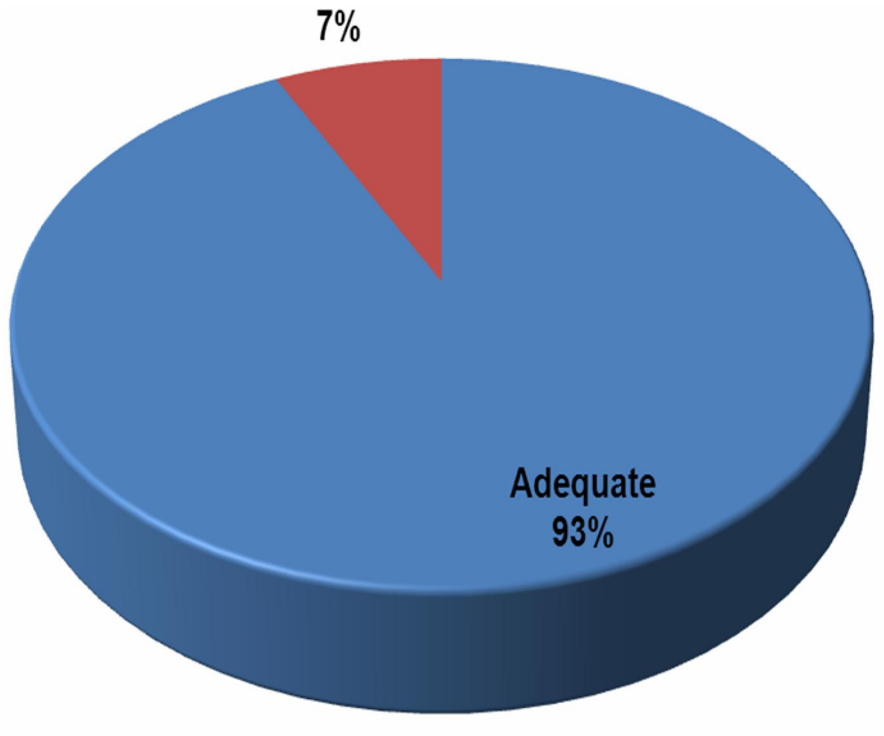
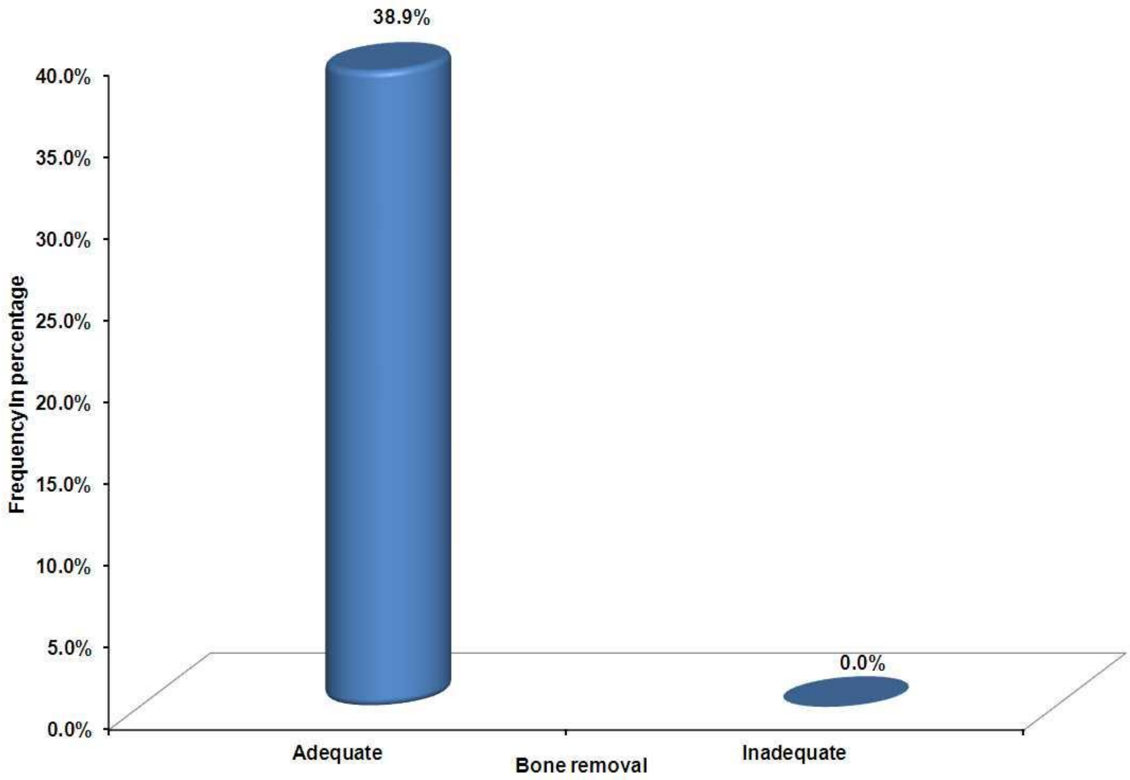


Table 12 b : Bone removal vs. outcome

Bone Removal	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
Adequate	49	77	126
Inadequate	0	10	10
Total	49	87	136

Percentages of favourable out come

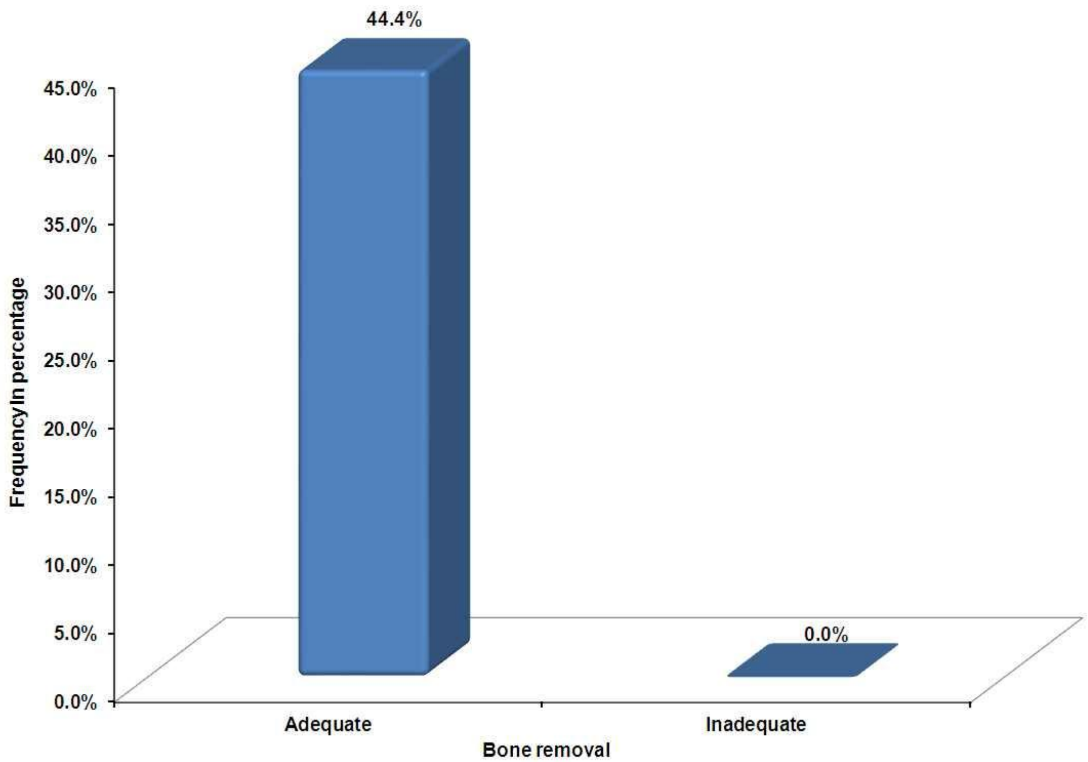


There is statistical significance between Adequate Bone removal and outcome (P-value – 0.009 < 0.05)

Table 12 c : Bone removal Vs. Survival / Death

Bone Removal	No of patients		Total
	Survival	Death	
Adequate	56	70	23
Inadequate	0	10	113
Total	56	80	136

Percentage of Survival



There is significant statistical difference between Bone removal and survival (P-value – 0.006 < 0.05)

Table 13 a : Duraplasty distribution

Duraplasty	No of patients
Not done	116
Done	20
Total	136

Percentage of Duraplasty distribution

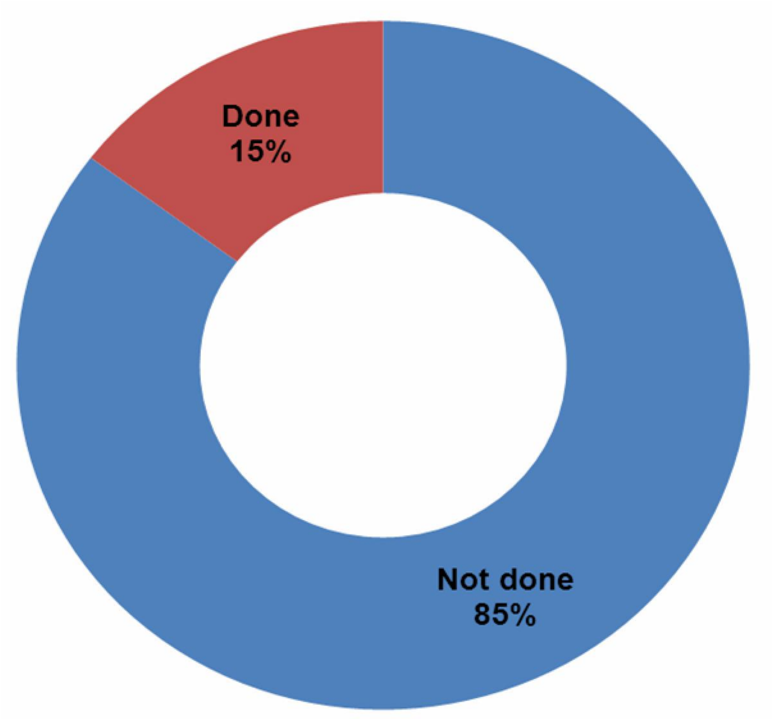
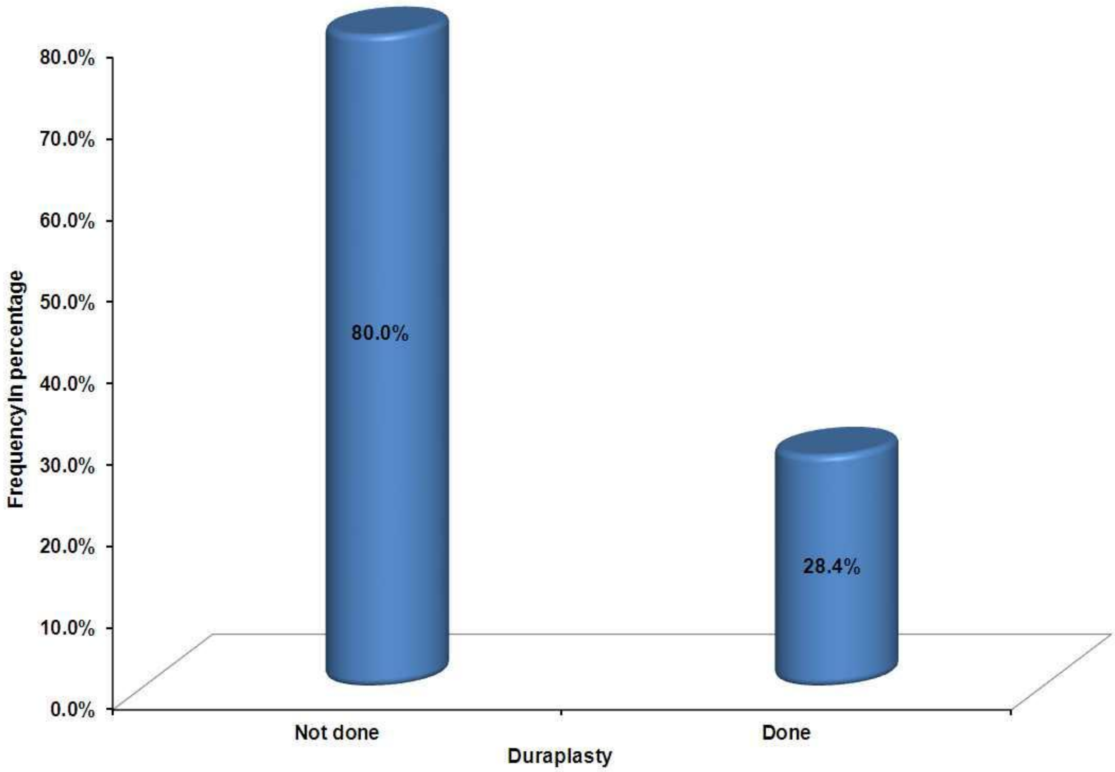


Table 13 b :Duraplasty vs. outcome

Duraplasty	No of patients		Total
	Favourable Outcome	**Unfavourable Outcome**	
Done	16	4	20
Not done	33	83	116
Total	49	87	136

Percentages of favourable out come

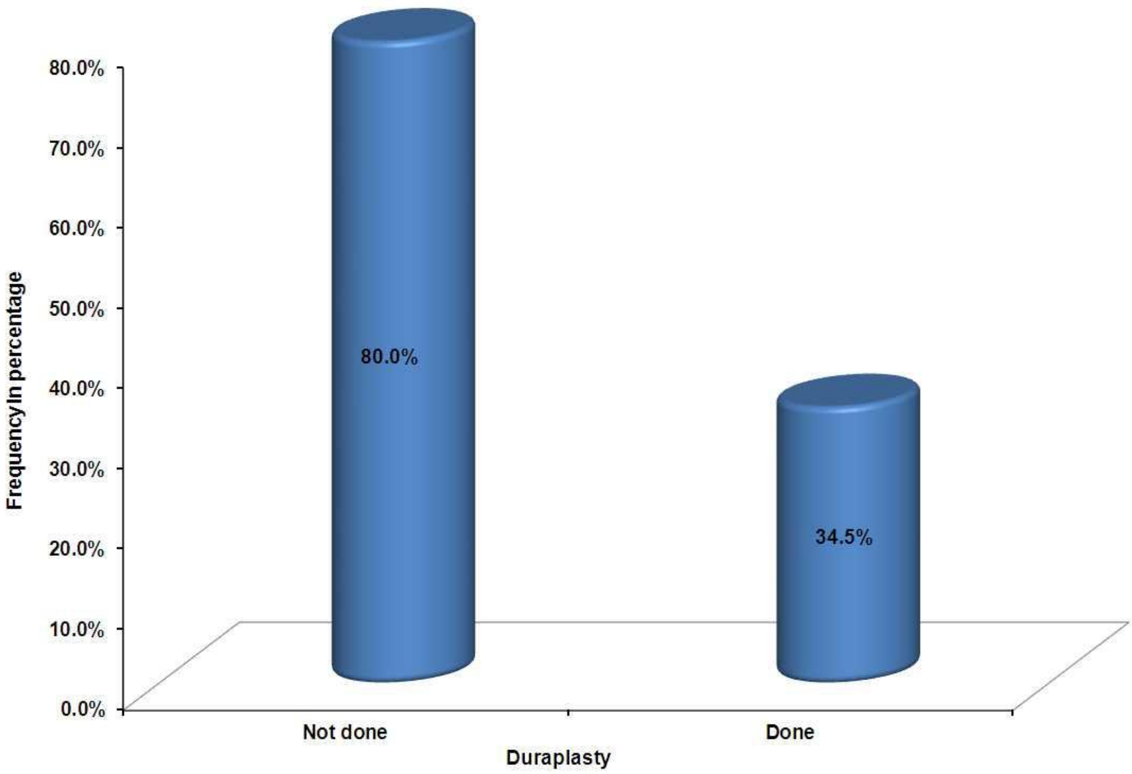


There is statistical significant difference between Duraplasty and outcome (P-value – 0.000 < 0.05)

Table 13 c :Duraplasty vs. Survival / Death

Duraplasty	No of Patients		Total
	Survival	Death	
Done	16	4	20
Not done	40	76	116
Total	56	80	136

Percentage of Survival

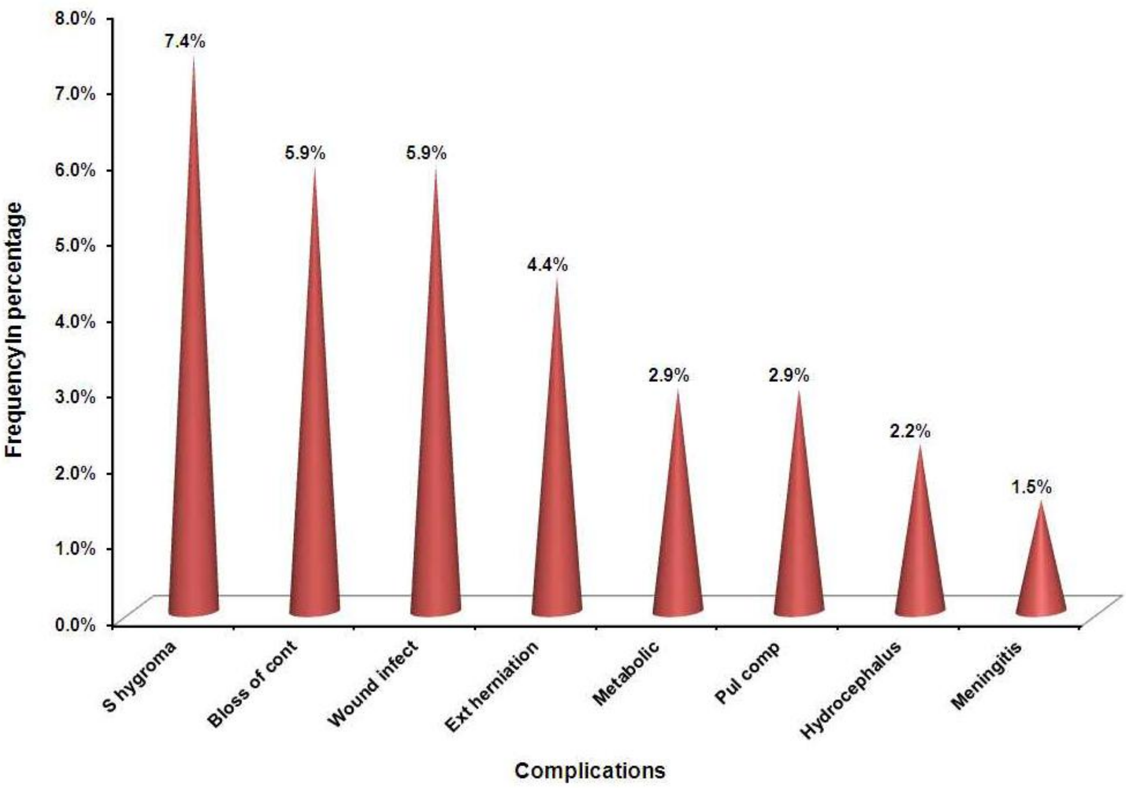


There is statistical difference between Duraplasty and survival (P-value – 0.000 < 0.05)

Table : 14 Complications distribution

Complications	Frequency	Percentage
S hygroma	10	7.4%
Bloss of contusion	8	5.9%
Wound infection	8	5.9%
Ext herniation	6	4.4%
Metabolic	4	2.9%
Pul .complication	4	2.9%
Hydrocephalus	3	2.2%
Meningitis	2	1.5%
Trephine syndrome	1	0.7%

Percentage of Complications distribution



DISCUSSION

In our present study there were 136 cases underwent decompressive craniectomy, of these 90% were males and the remaining 10% were females. The most common mode of injury was road traffic accident (76%). Out of 136 patients 56 patients were survived (41%), of whom 30 patients had good recovery (22.1%), 19 patients had mild disability (14%), 4 patients had moderate disability (2.9%) and 3 patients were in vegetative state (2.2%) at the time of discharge. 80 patients died accounting for a mortality rate of 58.8%. Favourable outcome rate was 36.1% (GOS 4&5), unfavourable outcome rate was 63.9 %(GOS1,2&3).

Age

The age of the patient is one of the main prognostic factor. Schneider et al estimated age as the single most important factor determining post operative outcome. Polin et al found that the pediatric TBI population responded better than the adult population to decompressive craniectomy[18]. In our present study, age was found to be predictor of favourable outcome, as the mortality rates were decreased in younger patients and increased in older patients. This was statistically significant.

Sex

In our present study there is no statistical significant between gender and outcome, male 36.6% and female 30.8%.

Mode of injury

The mortality is higher in road traffic accidents than in injuries due to other mechanism. Because most of the high speed motor vehicle accidents are the result of head injury sustained due to angular acceleration. Similar mechanism when prolonged for a longer duration results in diffuse axonal injury. In our study, road traffic accidents were the most common mode of injury (76%) followed by fall (19%). A small proportion of patients were injured due to assault (5%). There is statistical significance between mode of injury and favourable outcome, for RTA (34%), fall (26.9%) and for assault (100%).

Time of surgery

It is been noted that chances of improved outcome are enhanced if decompressive craniectomy is performed earlier. Polin et al, patient who underwent surgery within 48 hours post trauma had a significant better out come[18,24]. Munch et al reported similar results. Burkert and Plauman showed that there was more pronounced improvement of cerebral oxygenation if earlier decompressive craniectomy was performed[26,24]. Nevertheless, Kunze et al performed late DC, and obtained favorable outcome. In our patients who underwent surgery within 24 hours after trauma had a favourable outcome (39%) when compared to more than 24 hours (11.8%).

Preoperative GCS

Glasgow coma score was the important prognosticating factor and guide to surgical intervention. Guerra found that the most sensitive parameter was the GCS score obtained on the first post traumatic day[12]. In our present study favourable outcome among GCS 4-8 group was 15.9% and GCS 9-13 group was 66.7%. This indicates that the preoperative GCS was one of the most important parameters determining the patient's outcome.

Associated injury

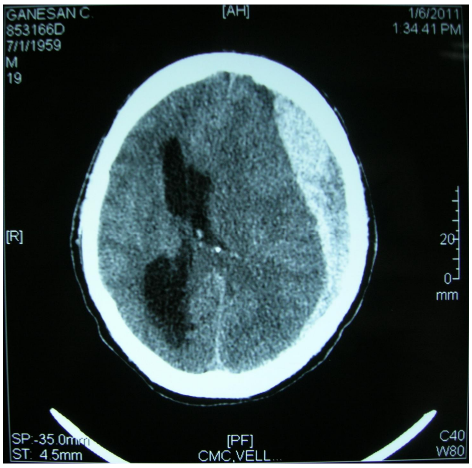
In our study patient who had associated long bone injuries, chest injuries, abdominal injuries and facial injuries, favourable outcome was 10% and patient not having associated injury, favourable outcome was 50%. It indicates associated injury also as one of the predictors of outcome.

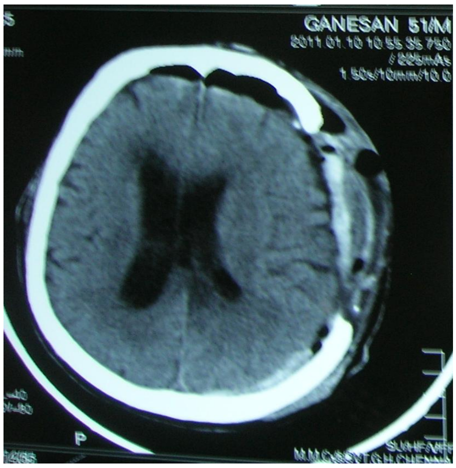
Papillary reaction

Papillary abnormalities due to TBI are associated with a significantly worse outcome. In our study papillary abnormality had high mortality. Favourable outcome was better among patients with normal papillary reaction (87%), and worst among abnormal papillary reaction (31.9%).

CT scan parameters

Eisenberg et al reported that mid line shift is very strong predictor of persistent raised ICP. Munch et al reviewed the effect of DC on computed tomography parameters and noted reduction of shift from 9.7 to 6.2 mm





Pre op mid line shift **Post op reduction in shift(24hrs)**

and a reduction in basal cistern compression, both known to predict poor outcome. In our present study post operative mid line shift reduction was seen in 78% of patients and post operative basal cistern opening was seen in 33% of patients, it indicates decompressive craniectomy decreases the ICP.

Adequacy of bone removal

DC creates a window through which brain tissue under direct mechanical compressive forces can protrude. ICP reduction varies from 15 to 70 % after DC. Munch et al calculated the gained volume after DC to be between 15.9 and 347.4 cm^3 , with a median volume of 73.6 cm^3. Obviously, the larger the DC, the more effective ICP reduction will be[17]. In our study, inadequate bone removal patient had 100% mortality. Favourable outcome was better among adequate bone removal patient (38.9%).

Duraplasty

Duraplasty at the time of de compression has been observed to lower the incidence of subdural effusion. Augmentation of craniectomy with duraplasty has been suggested as a mechanism to prevent or limit external cerebral herniation. Techniques of lattice duraplasty have also been suggested to limit external cerebral herniation[27]. In our present study patient with duraplasty had better favourable outcome (80%), only 15% percentage of patients underwent duraplasty (15%).

Complications

Most common complications observed in postoperative period were subdural hygromas, blossoming of contusion and wound infection. In our study external herniation and subdural hygromas were not occur in patients who had underwent duraplasty. Three patients developed postoperative hydrocephalus and underwent ventriculo peritoneal shunt.

CONCLUSION

The management of post traumatic uncontrollable brain swelling remains a challenge for neurosurgeons. Primary decompressive craniectomy is a therapeutic option for patients who had moderate to severe head injury with the clinical and radiological features of persistent raised ICP. The age, mode of injury, timing of surgery, clinical parameters like Glasgow coma score, associated injury, pupillary status, adequacy of bone removal and duraplasty, are important in predicting the outcome of decompressive craniectomy.

A generous craniotomy and augmented duraplasty facilitating ICP reduction and better outcome of decompressive craniectomy. Randomized studies will provide Class I evidence that will aid the decision making process in treating patients with refractory intracranial hypertension and brain edema.

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DECOMPRESSIVE CRANIECTOMY PROFORMA

NAME AGE SEX

ADDRESS

DATE& TIME OF INJURY:

DATE& TIME OF SURGERY:

MODE OF INJURY:

HISTORY OF	LOC	VOMITING
	FITS	ENT BLEED

CONDITION ON ADMISSION

GCS	MHIPS	PUPILS
PR	BP	RS

ASSOCIATED INJURIES

CT BRAIN

Volume/Thickness	
Midline shift	
Basal cisterns	

SURGICAL MANAGEMENT

TIMING OF SURGERY

PROCEDURE DONE

INTRA OP FINDING

DURAPLASTY

POST OP FOLLOW UP

	GCS	PUPILS	RS	PR/BP
IMMEDIATE				
1^{st} DAY				
3^{rd} DAY				
7^{th} DAY				
10^{th} DAY				

COMPLICATIONS

FOLLOW UP CT SCAN

FINDINGS	POST OP DAYS		
Bone removal			
Midline shift			
Basal cisterns			
Residual hemato			

CONDITION ON DISCHARGE

DATE GCS GOCS

Score1	Death	
2	Vegetative	
3	Moderate disability	
4	Mild disability	
5	Good recovery	

CAUSE OF DEATH

FOLLOW UP

MASTER CHART

								Pre op CT brain		Post op CT brain									
Sl.No	**Age**	**Sex**	**Mode of Injury**	**Time of Surgery**	**Pre Op GCS**	**Associated injury**	**Pupiliary Reaction**	**Volume/Thickness**	**B.Cistern**	**Bone Removal**	**Shift**	**B.Cistern**	**Residual Hameatoma**	**Duraplasty**	**Post Op GCS**	**Complications**	**Days in Hospital**	**Survived/Death**	**Outcome GOCS**
1	43	m	RTA	<24hr	6	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	4	-	2	D	1
2	50	m	RTA	<24hr	11	-	Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	13	-	10	S	5
3	25	m	RTA	<24hr	10	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	15	-	9	S	5
4	70	m	RTA	<24hr	6	-	Abnormal	>1cm	Effaced	adequate	not	not	-	-	4	-	3	D	1
5	40	m	RTA	<24hr	7	Facial injury	Abnormal	>1cm	Effaced	adequate	not	not	-	-	5	-	5	D	1
6	21	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	10	-	35	S	3
7	45	m	Fall	<24hr	10	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	Duraplasty	12	-	10	S	5
8	55	f	RTA	>24hr	7	-	Abnormal	>1cm	Effaced	adequate	not	not	-	-	7	-	5	D	1
9	50	m	RTA	<24hr	9	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	7	-	6	D	1
10	30	m	Fall	>24hr	5	H thorax	Abnormal	>1cm	Effaced	adequate	not	not	-	-	4	-	1	D	1
11	60	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	Inadequate	not	not	-	-	5	-	4	D	1
12	32	m	RTA	<24hr	11	Fore arm#	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	13	-	9	S	5
13	40	m	RTA	<24hr	9	-	Normal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	-	13	-	13	S	4
14	50	m	Fall	<24hr	7	-	Abnormal	>30ml	Effaced	adequate	Redused	not	-	-	7	Wound infection	12	D	1
15	50	m	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	adequate	not	not	-	-	4	-	5	D	1
16	35	m	RTA	>24hr	12	lung cont	Abnormal	>30cm	Effaced	adequate	Reduced	not	-	Duraplasty	10	-	7	D	1
17	40	m	RTA	<24hr	7	Tibia#	Abnormal	>30cm	Effaced	Inadequate	not	not	-	-	7	-	5	D	1
18	40	m	RTA	<24hr	13	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	14	-	10	S	5
19	34	m	RTA	<24hr	12	-	Normal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	-	13	-	8	S	5
20	37	m	Fall	<24hr	8	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	8	PVS	35	S	2
21	70	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	Pul complication	7	D	1
22	48	m	RTA	<24hr	4	H throax	Abnormal	>1cm	Effaced	adequate	not	not	-	-	3	-	3	D	1
23	22	m	RTA	<24hr	6	Femer#	Abnormal	>1cm	Effaced	adequate	not	not	-	-	5	Ext herniation	6	D	1
24	54	m	RTA	<24hr	12	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	15	-	8	S	5
25	60	m	RTA	>24hr	9	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	7	DKA,ARF	6	D	1
26	30	m	RTA	<24hr	8	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	-	6	Wound infection	25	S	5
27	25	m	RTA	<24hr	12	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	15	-	10	S	5
28	55	m	RTA	>24hr	5	-	Abnormal	>1cm	Effaced	adequate	Redused	not	sig ICH	-	4	-	4	D	1
29	45	m	RTA	<24hr	9	B.Bleg#	Abnormal	>1cm	Effaced	adequate	Redused	not	-	Duraplasty	8	Bloss of contusion	3	D	1
30	70	m	Fall	<24hr	5	-	Normal	>1cm,30ml	Effaced	adequate	Redused	not	-	-	4	Pul complication	1	D	1

MASTER CHART

								Pre op CT brain		Post op CT brain									
Sl.No	Age	Sex	Mode of Injury	Time of Surgery	Pre Op GCS	Associated injury	Pupiliary Reaction	Volume/Thickness	B.Cistern	Bone Removal	Shift	B.Cistern	Residual Hameatoma	Duroplasty	Post Op GCS	Complications	Days in Hospital	Survived/Death	Outcome GOCS
31	56	f	fall	<24hr	11	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	10	-	21	S	4
32	50	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	5	-	4	S	5
33	70	m	RTA	>24hr	6	-	Abnormal	>1cm	Effaced	adequate	Not	Not	-	-	4	-	2	D	1
34	10	m	fall	<24hr	4	-	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	3	-	1	D	1
35	28	m	RTA	<24hr	9	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	-	12	S hygroma	15	S	5
36	36	m	ASS	<24hr	11	-	Normal	>1cm,30ml	Effaced	adequate	Reduced	Opened	sig ICH	-	12	Bloss of contusion	18	S	4
37	40	m	RTA	<24hr	7	H thorax	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	6	-	4	D	1
38	50	m	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	Inadequate	Not	Not	-	-	4	Ext herniation	2	D	1
39	30	m	RTA	<24hr	13	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	14	-	9	S	4
40	34	m	RTA	<24hr	9	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	-	12	-	11	D	1
41	50	m	RTA	<24hr	7	BB leg#	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	5	-	8	D	1
42	51	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	8	HydrocepH,Trephine s	26	S	3
43	53	m	fall	>24hr	8	-	Abnormal	>30ml	Effaced	adequate	Not	Not	-	-	5	Bloss of contusion	4	D	1
44	70	m	fall	<24hr	6	-	Abnormal	.1cm	Effaced	adequate	Not	Not	-	-	4	Pul complication	6	D	1
45	30	m	RTA	<24hr	4	-	Abnormal	>1cm	Effaced	adequate	Not	Not	-	-	4	-	1	D	1
46	28	m	RTA	<24hr	7	-	Abnormal	>30cm	Effaced	adequate	Reduced	Not	-	-	5	Wound infection	4	D	1
47	30	m	RTA	<24hr	8	-	Abnormal	>30cm	Effaced	adequate	Reduced	Not	-	-	10	Ext herniation	18	S	3
48	55	m	RTA	<24hr	4	Facial inj	Abnormal	>1cm	Effaced	Inadequate	Not	Not	-	-	3	-	4	D	1
49	40	m	RTA	<24hr	6	-	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	5	-	6	D	1
50	30	m	RTA	<24hr	6	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	10	-	16	S	4
51	47	m	RTA	<24hr	9	-	Abnormal	>1cm	Effaced	adequate	Not	Not	-		5	-	6	D	1
52	19	m	ASS	<24hr	12	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-		13	-	12	S	5
53	27	m	RTA	<24hr	12	-	Abnormal	>30ml	Effaced	adequate	Not	Not	sig ICH	-	7	Bloss of contusion	3	D	1
54	60	f	fall	>24hr	7	-	Abnormal	>30ml	Effaced	adequate	Reduced	Not	-	-	5	-	4	D	1
55	52	f	RTA	<24hr	10	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	12	S hygroma	16	S	4
56	54	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	7	S hygroma'Pul	15	D	1
57	44	m	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	adequate	Reduced	Not	-	-	13	Wound infection	16	S	4
58	45	m	RTA	<24hr	5	Lung con	Abnormal	>1cm	Effaced	adequate	Not	Not	-	-	4	-	2	D	1
59	40	m	fall	<24hr	12	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	15	-	8	S	5
60	35	m	ASS	<24hr	11	-	Abnormal	>30ml	Effaced	adequate	Reduced	Corr	-		13	-	10	S	5

MASTER CHART

								Pre op CT brain		Post op CT brain									
Sl.No	Age	Sex	Mode of Injury	Time of Surgery	Pre Op GCS	Associated injury	Pupiliary Reaction	Volume/Thickness	B.Cistern	Bone Removal	Shift	B.Cistern	Residual Hameatoma	Duroplasty	Post Op GCS	Complications	Days in Hospital	Survived/Death	Outcome GOCS
61	63	m	TTA	<24hr	4	Rib#	Abnormal	>1cm	Effaced	adequate	not	not	-	-	3	-	1	D	1
62	55	m	RTA	>24hr	6	-	Abnormal	>30ml	Effaced	adequate	Reduced	not	-	-	4	-	3	D	1
63	25	m	RTA	<24hr	13	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	13	Wound infection	15	S	5
64	36	m	RTA	<24hr	12	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	13	S hygroma	23	S	5
65	40	f	Fall	<24hr	7	-	Abnormal	30ml	Effaced	adequate	not	not	sig ICH	-	5	Bloss of contusion	3	D	1
66	55	m	RTA	<24hr	9	Femur#	Abnormal	>1cm,30ml	Effaced	adequate	not	not	-	-	7	DM 'ARF	7	D	1
67	30	f	RTA	<24hr	10	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	8	PVS, Hydrocephalus	36	S	2
68	30	m	RTA	<24hr	7	-	Abnormal	>1cm,30ml	Effaced	Inadequate	not	not	-	-	5	Ext herniation	6	D	1
69	52	m	RTA	<24hr	8	Facial inj	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	5	Pul complication	7	D	1
70	31	m	RTA	<24hr	10	H thorax	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	S hygroma,pul com	22	D	1
71	34	m	Ass	<24hr	12		Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	13	-	9	S	5
72	35	m	RTA	<24hr	8	-	Abnormal	>1cm	Effaced	adequate	Reduced	not'	-	-	5	-	3	D	1
73	45	m	RTA	<24hr	7	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	6	-	2	D	1
74	28	m	RTA	<24hr	11	-	Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	13	-	10	S	5
75	70	m	RTA	<24hr	10	Tibia#	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	Duraplasty	7	-	5	D	1
76	45	m	Fall	>24hr	5	-	Abnormal	>30cm	Effaced	adequate	not	not	-	-	4	-	3	D	1
77	55	m	RTA	>24hr	7	-	Abnormal	>30cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	10		12	S	4
78	45	m	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	Inadequate	not	not	-	-	3	-	2	D	1
79	50	m	Fall	<24hr	7	Fore arm#	Abnormal	>1cm	Effaced	adequate	not	not	-	-	5	-	4	D	1
80	27	m	RTA	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	10	S hygroma	30	S	4
81	60	f	Fall	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	5	-	4	D	1
82	70	m	RTA	<24hr	7	Femur#	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	4	-	4	D	1
83	35	m	RTA	<24hr	11	-	Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	13	-	12	S	5
84	34	m	RTA	<24hr	6	-	Abnormal	>1cm	Effaced	Inadequate	not	not	-	-	4	-	2	D	1
85	28	m	RTA	<24hr	9	H thorax	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	Wound infection	7	D	1
86	50	f	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	adequate	not	not	-	-	3	-	3	D	1
87	50	f	Fall	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	5	-	2	D	1
88	45	f	RTA	>24hr	9	-	Abnormal	>30ml	Effaced	adequate	Reduced	not	-	-	7	-	3	D	1
89	22	m	RTA	<24hr	12	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	14	-	10	S	5
90	31	m	Fall	<24hr	8	-	Abnormal	>30ml	Effaced	adequate	Reduced	not	-	-	10	S hygroma	15	S	4

MASTER CHART

								Pre op CT brain		Post op CT brain									
Sl.No	Age	Sex	Mode of Injury	Time of Surgery	Pre Op GCS	Associated injury	Pupiliary Reaction	Volume/Thickness	B.Cistern	Bone Removal	Shift	B.Cistern	Residual Hameatoma	Duroplasty	Post Op GCS	Complications	Days in Hospital	Survived/Death	Outcome GOCS
91	22	m	RTA	<24hr	13		Abnormal	>1cm	Eff	adequate	Reduced	Opened		Duraplasty	14	-	9	S	5
92	47	m	RTA	<24hr	9	Humerus#	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	7	Wound infection	14	D	1
93	27	m	RTA	<24hr	8		Normal	>30ml	Effaced	adequate	Reduced	Opened	-	-	12	Bloss of contusion	12	S	4
94	40	m	RTA	>24hr	7		Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	-	4	D	1
95	24	m	Fall	<24hr	8	Rib#	Abnormal	>1cm,30ml	Effaced	adequate	not	not	sig ICH	-	6	Bloss of contusion	5	D	1
96	25	m	RTA	<24hr	5		Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	4	-	3	D	1
97	29	m	RTA	>24hr	7		Abnormal	>1cm,30ml	Effaced	adequate	not	not	-	-	5	-	2	D	1
98	45	f	Fall	<24hr	11		Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	10	-	13	S	4
99	65	m	RTA	<24hr	8		Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-		10	-	26	S	4
100	34	m	ASS	<24hr	7		Abnormal	>1cm,30ml	Effaced	adequate	Reduced	Opened	-	Duraplasty	12	-	13	S	5
101	40	m	RTA	<24hr	8		Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	13	S hygroma	12	S	5
102	45	m	RTA	<24hr	5	Fascial inj	Abnormal	>1cm	Effaced	Inadequate	not	not'	-	-	3	-	1	D	1
103	42	m	Fall	>24hr	7		Abnormal	>30ml	Effaced	adequate	Reduced	not	-	-	9	-	23	S	4
104	45	m	RTA	<24hr	4	BB leg#	Abnormal	>1cm,30ml	Effaced	adequate	not	not	-	-	3	Ext herniation	5	D	1
105	35	m	ASS	<24hr	12		Normal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	13	-	8	S	5
106	40	m	Fall	<24hr	9	B.I.Abd	Abnormal	>1cm,30cm	Effaced	adequate	Reduced	not	-	-	7	ARF	3	D	1
107	70	m	RTA	<24hr	7	-	Abnormal	>1cm,30cm	Effaced	Inadequate	not	not	-	-	5	-	7	D	1
108	58	m	Fall	<24hr	7	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	6	-	5	D	1
109	32	m	RTA	<24hr	12	-	Abnormal	>1cm	Effaced	adequate	not	not	-	-	7	-	1	D	1
110	70	m	Fall	<24hr	6		Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	4	DKA	4	D	1
111	47	m	RTA	<24hr	9	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	8	Trephine s	25	S	4
112	42	m	RTA	<24hr	7	-	Normal	>1cm	Effaced	adequate	not	not	-	-	5	-	1	D	1
113	40	m	RTA	<24hr	5	-	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	5	-	3	D	1
114	37	m	RTA	<24hr	11	-	Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	Duraplasty	13	-	8	S	5
115	55	m	RTA	>24hr	4	Femur#	Abnormal	>1cm	Effaced	adequate	not	not	-	-	3	-	1	D	1
116	41	m	RTA	<24hr	7	-	Abnormal	>30ml	Effaced	adequate	Reduced	not	-	-	7	Meningitis	9	D	1
117	40	m	RTA	<24hr	10	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	10	S hygroma	15	S	4
118	45	m	RTA	<24hr	9	-	Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	7	-	6	D	1
119	22	m	RTA	<24hr	6	-	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	10	-	17	S	4
120	38	m	RTA	>24hr	7	Forearm#	Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	5	-	2	D	1

MASTER CHART

								Pre op CT brain		Post op CT brain									
Sl.No	Age	Sex	Mode of Injury	Time of Surgery	Pre Op GCS	Associated injury	Pupiliary Reaction	Volume/Thickness	B.Cistern	Bone Removal	Shift	B.Cistern	Residual Hameatoma	Duroplasty	Post Op GCS	Complications	Days in Hospital	Survived/Death	Outcome GOCS
121	22	m	RTA	<24hr	11		Normal	>30ml	Effaced	adequate	Reduced	Opened	-	Duraplasty	13	-	9	S	5
122	42	m	RTA	<24hr	8		Abnormal	>30ml	Effaced	adequate	not	not	-	-	7	Bloss of contusion	5	D	1
123	40	m	RTA	<24hr	7		Abnormal	>1cm	Effaced	adequate	not	not	-	-	7	Ext herniation	5	D	1
124	29	m	Fall	<24hr	10		Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	10	-	15	S	4
125	29	m	RTA	<24hr	9	Fore arm#	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	9	PVS	28	S	2
126	25	m	RTA	<24hr	12		Normal	>1cm	Effaced	adequate	Reduced	not	-	Duraplasty	13	Wound infection	15	S	5
127	68	f	Fall	<24hr	7		Abnormal	>1cm,30ml	Effaced	Inadequate	not	not	-	-	5	-	3	D	1
128	50	m	RTA	<24hr	9	Facial inj	Abnormal	>1cm,30ml	Effaced	adequate	Reduced	not	-	-	10	S hygroma	16	S	4
129	19	m	RTA	<24hr	10		Abnormal	>1cm	Effaced	adequate	Reduced	Opened	-	-	12	Meningitis	17	S	5
130	23	m	RTA	<24hr	6		Abnormal	>1cm	Effaced	adequate	not	not	-	-	5	-	5	D	1
131	30	m	Fall	<24hr	7		Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	Hydrocephalus	38	S	3
132	25	m	RTA	<24hr	12		Normal	>1cm	Effaced	adequate	Reduced	Opened	-	-	13	-	11	S	5
133	32	m	RTA	<24hr	5		Abnormal	>1cm,30ml	Effaced	adequate	not	not	-	-	4	-	3	D	1
134	26	m	RTA	<24hr	7	Rib#	Abnormal	>1cm	Effaced	adequate	Reduced	not	-	-	7	-	4	D	1
135	35	f	ASS	<24hr	13		Abnormal	>30ml	Effaced	adequate	Reduced	Opened	-	-	13	-	10	S	5
136	55	m	RTA	<24hr	4		Abnormal	>1cm	Effaced	adequate	not	not	-	Duraplasty	3	-	4	D	1