

Original Research Article

## A Study on Head Injury

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**Abstract:** As head is the most prominent exposed part of human body by virtue of its Situation, it bears the brunt of violence namely accidents, suicidal and homicides. The diagnosis of the exact lesion due to trauma is a difficulty even to the neuro-physician. The treatment is an enigma to Neuro-surgeon. And the evaluation of mechanism is a puzzle to the medico-legal expert. The present study conducted in 200 cases in the department of forensic medicine, Osmania Medical College, Hyderabad. During the period of June 2009 to the end of May 2012. 60 cases of head injury presented with oedema, 6 cases shown severe tubular degeneration, in 5 cases liver changes were present and 13 cases died due to pulmonary embolism.

**Keywords:** accidents, head injury, histo pathological changes

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### INTRODUCTION

India is now passing through a process of tremendous industrial revolution drawing the rural population into urban areas. Not only the urban roads have become congested with pedestrians, but the movement of goods in the plain period has made the roads unsafe. Modern craze mad race for great speed have made the situation worse. Accidental injuries have progressively and alarmingly increased with modernization in transport and industry. With the advancement of mechanization in agriculture and other walks of life the accidental injuries in general and "head injury" in particular are bound to become a major problem in India as in the west.

Though the history of mechanism and effects of physical violence on the Head dates back to 25 centuries B.C, Craniocerebral injuries were probably first scientifically studied by Hippocrates (in the 5<sup>th</sup> century B.C.) who chronicled his experience by greater understanding of physiology and anatomy of skull and the Brain and the correlation of Post-mortem findings and clinical observation. The progress was extremely slow and many false conclusions were drawn, which later had to be corrected by slow process of trial and error. Polson (1955) said "*It is prudent to assume that every unconscious patient has sustained a Head injury*

*and that his condition is due to this cause, unless or until it is shown to be due to some other cause"* [1].

Moreover opportunity for Post-mortem examination of the head injuries was delayed until early 1308 A.D. The introduction of microscope has aided greatly in the interpretation of the histological alterations in the brain incidental to trauma. The experimental methods introduced little over a century ago, have contributed to solve only some of the perplexing questions regarding the mechanism, pathological physiology and morbid anatomy. It is the object of this piece of work to deal with the more salient views on the pathology of cranio cerebral trauma and their forensic concern, besides clinical approach.

### AIMS AND OBJECTIVES

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### MATERIAL AND METHODS

The present work done in the upgraded department of forensic medicine, Osmania Medical College, Hyderabad. During the period of June 2009 to the end of May 2012 over 200cases.

### RESULTS AND DISCUSSION:

Total number of post mortem examination done in Osmania mortuary during the period 2009 is 4491; in 2010 it is 4445; and in 2011 till date 4217.

This figures are include mortality rates in Osmania only.

Type of cases	2009	2010	2011
Accident	2439	2801	2698
Hanging	315	267	274
Burns	674	623	590
Railway	188	204	168
Poisoning	360	312	295
Fall from height	45	38	51
Others	470	200	141
Total	4491	4445	4217

These are overall cases for which post mortem examination done in OGH during concerned years. The

burden will be higher when we compare the cases in different hospitals all over Hyderabad.

**Total number of cases in both sex:**

Type of cases	2009		2010		2011	
	Male	Female	Male	Female	Male	Female
Accident	1926	513	2156	645	2077	621
Hanging	170	145	144	123	147	127
Burns	277	397	255	368	241	349
Railway	157	31	171	33	141	27
Poisoning	147	213	127	185	121	174
Fall 4m ht	22	23	21	17	28	23
Natural	9	2	11	6	10	4
Others	397	73	168	32	126	15

**Victims involved in different accidents**

	2009		2010		2011	
	Male	Female	Male	Female	Male	Female
Pedestrians	58	12	64	17	62	19
Cyclist	38	5	43	12	41	12
2 wheelers	1136	153	1272	246	1225	254
3 wheelers	192	12	215	23	207	31
4 wheelers	346	61	388	57	373	111
Buses	96	32	107	28	103	31
Lorry & heavy vehi.	154	8	172	24	166	25

During 2009-11 the incidence of accidents have been increased drastically, it may be due to negligence driving, speed, fast track culture, ill maintenance of roads and drainages, in proper road sense, not following traffic rules, not maintaining condition of vehicles and

safety measures. There is predominant increase in female death rates as they are also coming out to work places; they maintain safety measures, slow driving, others negligence can also property and lives.

**Total deaths ingeneral:**

	Spot	During travel	hospital
Pedestrians	64	74	94
Cyclist	42	48	61
2 wheelers	2057	1203	1028
3 wheelers	190	285	205
4 wheelers	374	507	435
Bus/lorry	408	139	36
Heavy vehi.	308	21	34
Train	418	21	0

Three patterns of injury often seen in **pedestrians:**

**1) Primary impact-** in this parts involved depend on position of person in relation to vehicle. Normally victims are struck by front of the vehicle and sustain so called bumper injuries on legs. These are severe with fracture and extensive soft tissue injuries.

**2)Second collision:** impact of the person against an object after the vehicle itself has hit. Most extensive fractures are produced by head striking a flat surface.

**3)Rolling injuries:** produced when vehicle with low chassis rolled the victim along the road way as it passed over him

**Cyclist and motor cyclist:** caused by turning in front of the vehicle from one side to other. The cycle is hit and the person is thrown violently to the ground. Many injuries to legs, groin, abdominal through protruding objects. Injuries to legs occur in low speed. Falling from vehicle at speed cause visceral damage.

**HELMETS**

It reduces friction of head against ground and makes deceleration less drastic by allowing the protected head to skid across the ground. Helmets

reduce the risk of head injuries by about 30% and fatalities by about 40% .when a crash helmet is worn, the crown may be protected, but the whole head may be egg shelled on to the base or cervical spine with fracture of spine. Although helmets do decrease fatalities, there greatest value is protection at lower speed or tangential impact. About 50% of helmet less motor cyclist sustain head injury. The brain is crushed or torn and bleeds from multiple sites. Impact on the crown of the head may produce **RING FRACTURE** around the foramen magnum. In about 25% of cases cervical spine fractures occur. Severe brain damage may occur even when a helmet is worn. Cortical contusions and laceration are common. Brain tissue may extrude through compound fractures of the skull.The classical fatal injuries in both motor cyclist and pillion passengers is fracture of the skull, usually from secondary impact with ground. **Temporoparietal fractures** are common with countercoup brain injury. A fracture is produced through the pituitary fossa, often associated with fissured fracture passing upwards to the temporal bones. At autopsy, the base of the skull is seen divided into two halves, each moving independently of each other like a **Hinge**, the so called **MOTOR CYCLIST FRACTURE**.

**Body parts involve in different accidents:**

	Head			neck	chest	UL&LL	spine	pelvis
	Scalp	Skull	Brain					
Pedestrians	15%	12%	11%	.5%	14%	38%	2.5%	7%
Cyclist	12%	18%	14%	.5%	11%	40%	1%	3.5%
2 wheelers								
Helmet	5%	4%	2%	32%	8%	38%	3%	7%
Without helmet	14%	19%	17%	.5%	8%	33%	2%	6.5%
3 wheelers	15%	10%	14%	.5%	23%	24%	8%	6%
4 wheelers	9%	14%	13%	.6%	28%	30%	2.4%	3%
Bus/lorry	18%	21%	20%	.5%	18%	14%	3.5%	5%
Train	21%	26%	30%	.5%	3%	14%	2.5%	3%
Assault	14%	18%	24%	8%	18%	11%	3%	2%

The application of the blunt force to the head may result in injury to the contents of the skull; outer table is twice the thickness of inner. A blow on head may cause linear acceleration which produce compressional/rarefractional force. Most closed chest injuries are caused by blunt force forming contusion,

abrasion, laceration, penetrating injuries. Abdominal injuries may be penetrating or closed injuries by blunt force. Abrasion, contusion, laceration involving muscles and fracture of long bones of limbs occur more in 2 wheelers, cyclist, and pedestrians.

**SKULL FRACTURES:**

	Fiss Ure #	Depre Ssed #	Commi Nuted #	Po nd	Gutter	Ring	Sutural	Pene Trating#
Pede Strain	159	4	27	1	1	6	18	13
Cyclist	105	1	9	0	1	4	13	8
2 wheel	3000	27	217	1	1	293	167	43
3 &4 Wheel	476	3	24	0	0	31	12	6
Heavy Veh. & train	384	1	41	0	0	25	8	3
Assult	21	64	38	12	9	3	13	86

### MICROSCOPIC APPEARANCE OF BRAIN

From histological study of the brain in 200 cases of head injury, the lesions observed were:

1. edema	60
2. Ring hemorrhage	20
3. contusion	25
4. Petichial haemorrhages	30
5. sub-arachnoid hemorrhage	30
6. sub-arachnoid hemorrhage and intracerebral hemorrhage(combined)	25
7. Intracerebral hemorrhage	10

The importance of cerebral edema as a cause of death is emphasized by some authors and discounted by others. It is believed that low oxygen levels in the blood may be an important cause of cerebral edema following head injury. On the other hand obvious tentorial herniation is rarely seen in fatal head injuries unless there is also an intra or extra cerebral hemorrhage. It has not always been recognized that intracerebral hemorrhages are constantly surrounded by a zone of edema which may be quite extensive around the larger hemorrhages and brain swelling due to this combination of hemorrhage and edema had been attributed to primary edema. Multiple small hemorrhages in the white matter sometimes of ring or ball type are not uncommonly found both in the cerebral and cerebellar hemispheres in cases of fatal head injury. Although they are most probably caused by moulding of the brain their mechanism is not clear. Some authors have found degenerative changes in the media and thrombosis of vessels in hemorrhagic areas. Diffuse neuronal injury of the brain has been observed in six cases and degeneration of the brain tissue in only one case. Petichial hemorrhages were also observed in cerebellum. The comparative incidence of intra cranial lesion, as come across by **Rowbotham, Reddy** and the present work [2, 3]. There is not much difference in the incidence of extra Dural hemorrhage among these workers. The incidence of sub dural haemorrhage is almost similar.

### INCIDENCE OF KIDNEY CHANGES ASSOCIATED WITH HEAD INJURY.

From the histological studies of the kidney in the cases of head injury, six cases present a picture of severe tubular degeneration which has been called shock kidney. Haemorrhage in the intertubular tissue is present. Tubules are filled with rbc's and rbc cast. The earliest time such a change is observed in this series is 15 min. oliguria and other evidence of renal failure is one of the complications of head injury which may result in death. The mechanism by which these lesions are produced has been a matter of debate for a long time. There seems to be grounds for believing that the most constant etiological factor is diminution of the renal circulation. For this reason the name renal anoxia

has been suggested. There can be no question that renal arteries respond to nervous and possibly hormonal stimuli by vasoconstriction. Reflex anuria from both kidneys which follows obstruction of one ureter by a calculus, must be attributed to reflex vascular changes in the other kidney. The same mechanism operates in the shock kidney and crush syndrome.

### INCIDENCE OF LIVER CHANGES ASSOCIATED WITH HEAD INJURY

From gross and histological studies of the liver, pathological changes were observed in 5 cases. Patchy lemon yellow areas which were firm were present on the surface and in the substance of the liver. Microscopically focal necrosis of the central type was observed. These types of lesion are due to severe type of bacterial infection. The earliest time such a lesion observed in these series is 5 days. Clinically there was evidence of respiratory infection also.

### INCIDENCE OF PULMONARY CHANGES ASSOCIATED WITH HEAD INJURY.

From gross and histological studies of the lungs in head injury, the lesions observed were:

1. pulmonary edema.....	10
2. broncho-pneumonia with edema.....	06
3. pulmonary hemorrhages into alveolar spaces..	04

Pulmonary edema and pulmonary hemorrhages into alveolar spaces were observed in cases that survived for a short period and in some cases of instantaneous deaths. In none of them injury to the chest wall or lungs could be detected. Those who survived over a day or more invariably developed broncho-pneumonia although the same was observed, in one case 20 hours after injury to the head. **D.J Reddy** has observed, in 11 hours after head injury. Pulmonary hemorrhages and edema of the lungs form a fruitful soil for bacteria to thrive and cause broncho pneumonia. Evidence of aspiration pneumonia was wanting in all these cases. The probable pathogenesis of the pulmonary lesions in cases of head injury is damage to the brain stem; direct by laceration or hemorrhage or indirect by cerebral edema or compression by sub-tentorial hemorrhage are likely to induce abnormal or ineffective types of respiration owing to adverse effects on the respiratory centre. These results in sustained increase in pulmonary capillary pressure and with heightened permeability due to anoxia, abnormal transudation of fluid occurs into the lungs. **Rowbotham** has recorded pneumonia in 6.7% of 163 deaths as a result of head injury.

### Incidence of fat embolism in cases of head injury

13 persons are died due to pulmonary embolism. Of the 13 cases, two persons died

instantaneously or within a few minutes. The remaining 11 survived for 15 minutes to 7 days. In one case a person who died in 15 minutes showed lung embolism of +++. The maximum number of cases i.e., six cases showed lung embolism in 1 to 24 hours after trauma. The degree of trauma definitely bore a relation to the degree of embolism in the present series.

### CONCLUSIONS

The subject of "cranio-cerebral trauma" has assumed paramount importance in recent times owing to the enormous mechanization of various aspects of life, increasing instances of brutal assault and innumerable and variegated accidents in the air, in water and so on. The consequence of injury to the brain is of very great diversity and complexity and they offer many veering diagnostic problems to the clinicians and contribute often thought provoking necropsy material to the forensic pathologist. They are also important because of many other important medico-legal implications that arise in connections with these injuries like time of survival, acts of volition, compensation settlements etc. In spite of the tremendous advance made during the past 50 years, we are still unable to evaluate all the lesions which are exposed on the autopsy table. As cranio-cerebral traumas from a considerable percentage of deaths and as even trivial injury looks apparently normal in built force injuries, only a careful autopsy will enable us to assess every important fact on the autopsy table, which helps the public, police and court of law in administering justice. In my experience as autopsy surgeons it is "CRANIO-CEREBRAL TRAUMAS" "by all means really baffled me and at the same time inculcated in me the aptitude to investigate and elucidate as much as possible on this intricate problem.

The present work is based on the observation and study made on 200 bodies that died of CRANIO-CEREBRAL TRAUMAS, 60 of which from the clinical series and 140 of those who died before being admitted to any hospital. Various data were arrived at after detailed post-mortem examination and have been discussed and conclusions have been drawn regarding the age, sex and their relation to traumatic injuries. Incidence of changes in the lung, liver and kidney in relation to head injury has been discussed. Cause of death in all these cases has been inferred. Generally the clinicians are misled or may fail to diagnose where head injury is associated with alcoholic intoxication and especially while the patient is in deep coma. In these cases it is always advisable to take the x-ray and CT of the of the skull and brain and L.P done, without which investigation will not be complete and justice cannot be meated out to the patient .There is also question of pre-existing organic disease such as arterio-sclerosis, intra-cranial tumors and intra-cranial aneurysms and other pathological processes should always be excluded

before final opinion as to the cause of death is given. Lastly in cases of alleged infanticide, to exclude birth injuries and accidental trauma to the head, the Forensic Pathologist role is indispensable.

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