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CRANIO CEREBRAL DAMAGE WITH SPECIAL REFERENCE TO CIRCLE OF WILLIS IN FATAL ROAD TRAFFIC ACCIDENTS

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CRANIO CEREBRAL DAMAGE WITH SPECIAL REFERENCE TO CIRCLE OF WILLIS IN FATAL ROAD TRAFFIC ACCIDENTS

THESIS

FOR THE DEGREE OF

DOCTOR OF MEDICINE

(FORENSIC MEDICINE)

CHAPTER 1: INTRODUCTION

Accidents are world's most serious health problem. The development of science in various aspects of human life like household items, atomic energy etc have evolved far more superior, sophisticated, and lethal weapon of assault. The motorized transportation media like vehicles, trains, aeroplanes etc, with fast moving vehicular traffic, vast urbanization and changing social patterns, have contributed to increase in the incidence of trauma to human body. Traffic accident is an endemic disease which affects mainly young people. A middle aged male is more likely to die from injuries received in traffic accident than from any other cause and motor vehicle accidents are single leading cause of death.

In Delhi, vehicular accidents are the most common contributory factors to head trauma because of multifericity of vehicles viz; power driven as well as body and hand driven running on the some road, increased density of traffic specially in peak hours, faulty roads, poor maintenance of vehicles etc. The vehicular accidents usually involves young people between the age group 20-40 years.

Delhi is one of fastest growing cities in India. The population of city exceeds 122 lacs. The rapid increase in population can be seen from the fact that in 1954 population was only 20 lacs. The motor vehicular population has registered a phenomenal growth from 5.21 lakhs in 1980 to 31.67 lacs in 1998 and it is growing exponentially. Thus there has been 600% rise in number of motor vehicles on Delhi roads. Delhi has a total road length of 25948 km[51].

Last year, one out of every three road accident deaths in Delhi was caused by a hit and run motorist. In other words, thirty three percent of those who killed people in road accidents here just drove away. In most cases, the victim lay on the road, bleeding to death.

Pedestrians, cyclists and two wheeler drives are most vulnerable lot to the capital killer vehicles. This category of road users accounted for as high as 83 percent or 1623 out of 1956 of all fatal road accident victims in Delhi last year. Though there were 111 road deaths in last year, the roads here continue to dominate the nation wide list of fatal accidents. Delhi has highest number of vehicles registered accounting to more than 35 lakhs (Source HT dt 6 Jan 2001). There are following facts noted by traffic police department.

1. Number of registered vehicles in 1999 was around 33 lakhs.

The absolute number of vehicular accidents registered a decline in last four years. Total number of persons killed in road accidents fell from: -

1. 2361 in 1996 to 2040 in 1999, because of decrease in average speed of vehicle.
2. Hit and run accidents have began to decline and so have pedestrian death. The number of pedestrian deaths was pegged at 1025 in 1999 almost half the total accident victims – followed by scooterists anders 21 percent anent.
2. More accidents occur in the day time. In 1999 4908 aring day as agait of these 967 were fatal. The number of fatal accidents shows a gradnts shows a gradnts shows a gradnts shows a gradnts shows a gradnts shows a gradual upward swing as the day progresses, with the number peaking between 8-10 pm. It has been attributed to people who are in a hurry to reach home, besides being tired.
4. Although among the five metros Mumbai topped the list of total accidents (25,945), Delhi topped the list of fatal accidents with 1969 as against 411 in Mumbai.

Since the cranial cavity contains the brain with its all vital centres for the life, trauma to this region challenges the integrity and even the viability of the individual. Because of its size and anatomical position, it is a major site of trauma in road accidents. Even with the improvement in safety measures in vehicles and greater availability of state of art resuscitative measures, the mortality rte in crush injuries to head has not declined.

We, in the Department of Forensic Medicine, Maulana Azad Medical College and associated hospitals, New Delhi, have undertaken to study the pattern of craino cerebral damage with special reference to the involvement of Circle of Willis in trauma cases occurring in Delhi and we would try to establish relationship between the extent, nature and type of craino-cerebral injuries and

other injuries to fatality, survival time, age, sex, socio-economic status, type of trauma involved and the pattern of injuries form autopsy findings so as to widen the knowledge of the medical faculty in the field of early diagnosis and management of such injuries.

GENERAL CONSIDERATION:

Brain damage following head injury constitutes a major problem throughout the world. Head injury is now the most important cause of death and severe disability in children and adults under 45. Multicentre outcome studies have shown that about 35% of severely head-injured patients will die and between 1 and 5% will remain vegetative. Between 5 and 18% are left severely disabled 6 months after injury.

Dynamic pathophysiology studies have also led to advances in head injury prevention, via legislation (e.g. seat belts, halmets) and the design of vehicles. However, our understanding of mechanisms which allow recovery and regeneration within injured nervous system are still very poor despite extensive research.

BRAIN ANATOMY:

The human brain weighs about 1.2 kg and is poorly supported by the falx and tentorium within the skull. Its soft consistency and absence of intrinsic fibrous supporting structures make it especially vulnerable to shearing forces. The brain restrained only by the crainal nerves and brain stem at the base, and by the parasagital bridging veins along the interhemispheric convexity. It floats within the CSF and is thus free to move with its large cranial cavity in relative to lower animals.

The skeleton of head is called the skull. It consist of several bones that are joined together to form the cranium. The skull can be divided into two main parts:-

(a) The calvaria (brain box) is the upper part of the cranium which encloses the brain.

(b) The facial skeleton constitutes the rest of the skull and includes the mandible.

THE CALVARIA CONSIST OF 8 BONES

	Paired		Unpaired
1.	Parietal	1.	Frontal
2.	Temporal	2.	Occipetal
		3.	Sphenoid
		4.	Ethmoid

SKULL:

The adult cranium consist of two parallel tables of compact bone called the dipole, the outer being about twice the thickness of the inner. The cranium varies in thickness in adults and varies from place to place. The more venerable areas lie in the parietotemporal, lateral frontal and lateral occipetal zones. The thinnest area in of temporal bone where bone thickness may of only 4 mm.

BRAIN MEMBRANES:

The pachymeninges consist of the duramater and the leptomeninges, the archanoid and pia mater. The dura is formed of two layers of tough collagenous tissue, outer attached to the skull and inner

merges with the archanoid. The dura forms the falx and tentorium, and the cranial venous sinuses run within it, The arcanoid is a thin, vascular meshwork that is intimately applied to inner surface of dura. Sheaths of archanoid follow vessels into brain as they penetrate into neural surface and their surface is covered with the cerebrospinal fluid.

The pia is not a true membrane, but a surface felt work of glial fibers that are inseparable from the underlying brain.

THE BRAIN CONSIST OF

(i) The cerebrum, comprising two large cerebral hemispheres

(ii) The cerebellum

(iii) The midbrain

(iv) The pons

(v) The medulla oblogata

The mid brain, pons, and medulla together from the brainstem. The medulla is continuous, below with the spinal cord.

THE CEREBRUM:

The cerebrum is made of two cerebral hemispheres which are incompletely separated from each other by the median longitudinal fissure. The two hemispheres are connected to each other across the median plane by the corpus callosum. Each hemisphere contains a cavity, called the lateral ventricle.

Each cerebral hemisphere is divided into four lobes –frontal, parietal, occipetal and temporal. Their positions corresponds, very roughly, to that of the corresponding bones.

ARTERIES SUPPLYING THE BRAIN:

The brain is supplied by branches of the internal carotid and the vertebral arteries. Each internal carotid artery gives off two major branches to the brain. These are the anterior cerebral and middle cerebral arteries. The two vertebral arteries ascend on the anterolateral aspect of the medulla. At the lower border of pons they unite to form the basilar artery. The basilar artery lies in the midline, ventral to pons. At the upper broder of the pons it bifurcates into two posterior cerebral arteries. The internal carotid had vertebro-basilar systems are connected by the posterior communicating arteries. The two anterior cerebral arteries are connected by the anterior communicating artery. As a result of these anastomoses an arterial ring; the circuls arteriosus (or Circle of Willis) is formed in relation to the base of brain.

The anterior, middle and posterior cerebral arteries give rise to two sets of branches, cortical and central. The cortical branches ramify on the surface of cerebral hemispheres and supply the cortex. The central branches passes deep into the substance of cerebral hemisphere to supply structures with in it.

MOTOR ACCIDENTS: Mechanism can be divided into three phases

Preliminary phase – before the accident-which may be of long duration and contains the genesis of accident.

a) Accident phase of 0.2 to 1.5 sec from impact to cessation of vehicle movement.

b) The phase which will no exist for the subject if he dies, but which in other cases extends from 1st aid to rehabilitation and in either case is valuable for retrospective data.

MECHANISMS OF BRAIN DAMAGE:

The forces acting on brain during head injury produce complex movements and deformation when the freely mobile head is struck by a blunt object, an acceleration injury occurs. If the moving head suddenly strikes a blunt object a deceleration injury occurs.

PHASE A: DEPENDS UPON THE FOLLOWING FACTORS

1) Riders and passenger factor: i.e. age, sex, alcohol drugs etc. Under 25 years males are more effective especially if they are under influence of alcohol or any other drug.

2) Machine factors: more powerful the machine, the higher the accident rate.

3) Road and environmental factor shows that motor accidents occur in summer peaks.

PHASE B: CRASH PHASE:

Injuries to vehicle are effective in three ways:

1) At impact or in skids or in overturn the vehicle does not remain upright.

2) At impact

There are two main hypothesis that have been advanced to account for brain damage incurred at the moment of injury :-

1. The skull distortion/head rotation hypothesis

2. The head translation/cavitation head rotation hypothesis

Holburn (1945) explained coup lesions (contusions that occur directly below the site of injury) and the counter coups lesions (contusions located on the side of the brain diametrically opposite the point of injury). He postulated that brain damage was due to rotational acceleration forces. The main requisite for countercoup damage was rotational movement of the head in the coronal, sagittal or horizontal plane, or a combination of these. At these sites shear strains develop to cause contusion of the brain and tearing of blood vessels.

Recent work has shown that skull distortion and rotation of head are more important in the production of coup and counter coup injuries than either rotation alone or translation/cavitation. Contusions is produced much more readily by angular acceleration than by translation or linear acceleration.

It has been showed that at low and moderate acceleration levels, cerebral concussion with or without contusions in the frontal and temporal lobes was readily produced, but at high acceleration, the animal died rapidly from massive subdural haematomas.

These studies have shown that all the principal neuropathological features of head injury can be induced by the two phenomena of contact and acceleration. Contact as a result from an object striking the head, and produce local effects such as scalp laceration, skull fracture entradural haemorrhage. Acceleration, in contrast result in movement of the brain which leads to intra cranial and intracerebral pressure graident. These mechanism causes the subdural haematoma resulting from tearing of subdural bridging veins and diffuse damage to axons.

CLASSIFICATION OF BLUNT HEAD INJURY:

The blunt head injury has been classified as :

1. Primary 2. Secondary

Primary damage occurs at moment of injury, usually takes the form of cortical surface contusions, haematomas lacerations, diffuse axonal injury and damage to cranial nerves. Secondary damage is usually produced by complicating processes that are initiated at the moment of injury. The major causes are intra cranial haemorrhage brain swelling, ischemic brain damage, infection and raised intracranial pressure.

PRIMARY DAMAGE:

The principle lesions are scalp lacerations, skull fracture and surface contusions.

SCALP LACERATIONS:

These are of medico legal importance as the indicator of site of injury. They are important sources of blood loss, particularly in children.

SKULL FRACTURES:

The more severe the injury the greater the likelihood of skull fracture. The vault is involved three times more often than the base but there may be fractures in both sites. The importance of skull fracture can not be over emphasized because a skull fracture have a high incidence of intra cranial haematoma. In about 10% the fracture of vault is depressed and is classified as compound if an associated laceration of skull, and penetrating if there is also a tear of the dura. In very severe head injury there may be a hinge fracture that extends across the base of the skull, usually in the region of posterior part of the pituitary fossa and adjacent flat part of the temporal bones.

SURFACE-CEREBRAL CONTUSIONS AND LACERATIONS:

Surface contusions have a very characteristic distribution affecting particularly the frontal poles, the orbital surfaces of the frontal lobes, the temporal poles, the lateral and inferior surfaces of the temporal lobes. Contusions of the parietal and occipetal lobes and of the cerebellum and uncommon unless they are directly related to a fracture of the skull.

The pia-archanoid is intact over the contusions but form in cortical lacerations. The decease may die from the diffuse axonal injury, yet display only minimal or even no surface contusion. Surface contusions are most severe at the crests of gyri but may also extend through the cortex into the subcortical white matter. Surface contusions and lacerations are due mainly to contact between the surface of the brain and the rough and irregular bony surface of the base of the skull as a result of sudden deceleration of skull in which the brain has bone rotating. Their size depends on the area of contact between the striking object and the skull. By contrast, counter coup contusions are due to sudden deceleration of the moving head.

PRIMARY DAMAGE TO BRAIN STEM:

The lower brain stem may be focally injured in traumatic hyperextension of the head or fracture – dislocation of alanto-occipetal joint, or in complex basal skull fractures. Most patients who sustain sufficiently severe head injury to damage the brain stem do not survive.

INJURY TO CEREBRAL VASCULATURE:

The acceleration/deceleration impulses selectively damage blood vessels in brain. This type of damage usually occurs in road traffic accident. The injury to dura leads to thrombosis of meningeal arteries and veins and venous infarction. Massive subarchanoid haemorrhage due to traumatic rupture of a vertebral artery may rarely occur from shearing forces. A bruise or laceration below and behind one ear is usual in these cases.

SECONDARY DAMAGE:

The most important categories are ischemia, haematoma brain swelling and infection.

INTRACRANIAL HAEMATOMA:

Internal haematoma is a common complication of head injury. Haematomas are present in about 55% of trauma cases and is most common cause of death in patients with a lucid interval after injury.

Following trauma there may be bleeding into extradural, subdural or subarchanoid space; into the brain parenchyma or into the ventricles. Some degree of subarchnoid haemorrhage invariably occur as a result of surface cerebral contusions.

EXTRADURAL HAEMATOMA:

Extradural haematoma is present in about 5-15% of fatal head injuries, an overlying fracture of the skull was present in 90% of adult patients.

Extradural haematoma is a fracture haematoma which forms because of bleeding from vascular supply to the dura and which accumulates in the space created when the dura is stripped from inner surface of the skull as a consequence of impact deformation. It most commonly develops in relation to a bleed from a meningeal artery – usually the middle meningeal. Extradural haematoma occur most commonly under the thin bone of the squamous part of the temporal bone, and can occur in other regions also like frontal and parietal regions or posterior fossa.

SUBDURAL HAEMATOMA:

Subdural haematomas are caused by rupture of bridging veins or by a small tear in a cortial artery. When caused by the former mechanism, there may be very little evidence of other brain damage. When acute subdural haematoma is associated with massive contusions, usually of the temporal or frontal poles, the pial surface becomes pulped and small arteries may tear causing bleeding into both contused brain and subdural space.

SUBARACHNOID HAEMORRHAGE:

This is more common type of brain membrane bleeding even more than subdural. Whenever there is damage to the cortex there will be some degree of subarchanoid haemorrhage also the shear stresses and rotational movements of brain causing the rupture of bridging veins that leaves the cortex and penetrate the archanoid. Where laceration, contusion or infarction of the cortex is present, bleeding will come from cortical veins and small arteries directly into subarchanoid space.



Beginning of thesis