INCIDENCE AND PREDISPOSING FACTORS OF BIRTH TRAUMA IN A TERTIARY CARE HOSPITAL: A PROSPECTIVE STUDY

Dissertation submitted to

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

In partial fulfillment of the regulations

For the award of the degree of

M.D.BRANCH- VII

PAEDIATRICS

GOVT. STANLEY MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI, INDIA.

MARCH 2009

CERTIFICATE

This is to certify that the dissertation entitled "**INCIDENCE AND PREDISPOSING FACTORS OF BIRTH TRAUMA IN A TERTIARY CARE HOSPITAL:A PROSPECTIVE STUDY"** is a bonafide original work of **Dr. R. SENTHIL PRABHU**, in partial fulfillment of the requirements for **M.D.BRANCH VII (PAEDIATRICS)** examination of the Tamilnadu Dr. M.G.R. Medical University to be held in march 2009.

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DECLARATION

I, **DR.R.SENTHIL PRABHU**, solemnly declare that dissertation titled, "**INCIDENCE AND PREDISPOSING FACTORS OF BIRTH TRAUMA IN A TERTIARY CARE HOSPITAL: A PROSPECTIVE STUDY"** is a bonafide work done by me at Institute of Social Pediatrics, Govt. Stanley Medical College, Chennai-1 during the period of October 2007 to September 2008 under the supervision of my **Prof. DR. M.L.VASANTHAKUMARI, M.D, D.C.H,** Director, Institute of Social Pediatrics, Govt. Stanley Medical College, Chennai. The dissertation is submitted to Tamilnadu Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **M.D. Degree(Branch-VII) in pediatrics**.

Place: Chennai

Date: **(Dr. R.SENTHILPRABHU)**

ACKNOWLEDGEMENT

I owe my thanks to the **Dean Dr. MOHANASUNDARAM, M.D, Phd**, Govt. Stanley Medical College, Chennai, for granting permission to conduct this study at Govt. RSRM Hospital, Royapuram, Chennai.

I thank my respected **Prof. Dr. M.L.VASANTHA KUMARI, M.D, D.C.H,** Professor, Institute of Social Pediatrics, Govt. Stanley Medical College for having been very much supportive and encouraging for conduct of this study.

I thank Prof. **Dr. SUJATHA SRIDHARAN, M.D, D.C.H.,** Additional Professor, Institute of Social Paediatrics for guiding me to conduct this study.

I also thank **Prof. Dr. KARUNAKARAN, M.D, D.C.H,** Additional Professor, Institute of Social Pediatrics, **Prof. Dr. SUNDARI, M.D, D.C.H,** Professor, Dept. of Neonatology, Govt. RSRM Hospital for their invaluable suggestions & support.

I would like to offer my gratitude to the Registrar, **Dr.C.N.KAMALARATHINAM, M.D, D.C.H,** for his kindness and gratitude.

I offer my special thanks to my Asst. Prof. **Dr. M.A.ARAVIND, M.D (Paed),** for his invaluable help and suggestions throughout my study.

I also thank my Assistant Professors **Dr. J.GANESH, M.D., D.C.H., Dr. ANBU, M.D, D.C.H, Dr. ELANGO, M.D., D.C.H., Dr. SAMBATHKUMAR, M.D (Paed), Dr.**

EKAMBARANATH, M.D(Paed), Dr. RADHIKA, M.D (Paed)., Dr. KUMAR, D.C.H., for their critical reviews and suggestions.

I also thank **Prof. Dr. STEPHEN ABRAHAM SURESHKUMAR, M.D., D.C.H., D.M.,** Professor & HOD, Dept. of Pediatric Neurology and **Prof. Dr. JOHN SOLOMON, M.D., D.C.H.**, Professor, Dept. of Pediatric Haematology & Oncology, Institute of Social Pediatrics for their valuable suggestions.

I also thank **Mr. A.VENKATESAN,** Lecturer in statistics, Madras Medical College, Chennai for his invaluable help in analyzing the values.

I am greatly indebted to all my co- postgraduates who have been the greatest source of encouragement, support, enthusiasms, criticism and friendly concern and timely help.

Last but not the least I owe my sincere thanks and gratitude to all the children and their parents without whom this study would not have been possible.

CONTENTS

INTRODUCTION

The concepts of health are important to Obstetrics. Labor is an intensive care situation. The woman and her unborn infant are at potential risk from unpredictable acute emergencies. In the context of raising level of expectation, knowledge and medico legal problems, it is the right of every prospective parent to be blessed with normal newborn. However there are clinical situations inherent to that particular pregnancy when birth injuries are expected. These inherent factors could be maternal, fetal, type of assisted deliveries and lastly the experience of the health worker conducting the delivery.

The 19th century was witness to many detailed autopsy clinical studies relating birth trauma to fetal presentation and mode of delivery. Despite a declining incidence due to improvements in obstetrical care and prenatal diagnosis, birth injuries remain a significant cause of morbidity and mortality.

The significance of birth injuries may be assessed by review of mortality data. In 1981, birth injuries ranked 6th among major causes of neonatal death, resulting in 23.8 deaths per 100,000 live births[71]. During ensuing decade because of refinements in obstetric techniques and the increased use of caesarean deliveries over difficult deliveries dramatic decline occurred in birth injuries as a cause of neonatal death. Statistics for 1993 revealed a reduction to 3.7 deaths per 100,000 live births[72]. The most recent figures available for 2005, the mortality rate in USA were 0.6 per 100,000 live births[9].

The overall incidence of birth trauma reported from USA ranges from 6-8 injuries per 1000 live births (Perlow et al 1996)[57]. The diagnosis and its notification in Indian setup is

nearly impossible as the majority of deliveries are still conducted by unskilled, self acclaimed birth attendants and even in tertiary medical institutions, autopsies are seldom performed. The Indian literature is deplete with information on birth trauma. Guha et al 1970, observed the incidence of BT as 6.8 per 1000 live births[28].

Injuries to the infant that result from mechanical forces (i.e., compression, traction) during the birth process are categorized as mechanical birth trauma[47]. Factors responsible for mechanical injury may coexist with hypoxic-ischemic insult; one may predispose the infant to the other. Nearly one half are potentially avoidable with recognition and anticipation of obstetric risk factors. Infant outcome is the product of multiple factors.

Many injuries such as soft tissue trauma are minor and self limiting but others such as liver lacerations, SGH or large subdural haemorrhage can be life threatening and require prompt recognition and intervention. Mechanical BT can result in both physical and neuro developmental handicap.

REVIEW OF LITERATURE

BIRTH TRAUMA DEFINITION:

An impairment of the infant's body function or structure due to adverse influences that occurred at birth. This definition is coined by National Vital Statistics, U.S.A[37].

Birth injuries may be avoidable, or they may be unavoidable and occur despite skilled and competent obstetric care, as in an especially hard or prolonged labor or with an abnormal presentation. Fetal injuries related to amniocentesis and intrauterine transfusions and neonatal injuries after resuscitation procedures are not considered birth injuries[23]. However, injuries related to the use of intrapartum monitoring of the fetal heart rate and collection of fetal scalp blood for acid-base assessment are included.[23]

INCIDENCE: Morbidity: 6 - 8 per 1000 live births (USA)[57].

Mortality: 0.6 per 100,000 live births[37].

Despite a reduction in related mortality rates, birth injuries still represent an important source of neonatal morbidity[68] and neonatal intensive care unit admissions. Of particular concern are severe intracranial injuries after combined methods of vaginal delivery (vacuum-assisted and forceps delivery) and failed attempts at operative vaginal delivery[61].

The clinician should consider the broad spectrum of birth injuries in the differential diagnosis of neonatal clinical disorders. Although many injuries are mild and self-limited, others are serious and potentially lethal. The birth process is a blend of compression, contractions, torques, and traction. When fetal size, presentation, or neurologic immaturity

complicates this event, such intrapartum forces may lead to tissue damage, edema, hemorrhages, or fractures in the neonate. The use of obstetric instrumentation may further amplify the effects of such forces or may induce injury alone. Under certain conditions, cesarean delivery can be an acceptable alternative but does not guarantee an injury-free birth.

PREDISPOSING FACTORS[54]:

11. Prima gravida
12. Cephalo pelvic disproportion, small maternal stature, maternal pelvic anomalies
13. Prolonged or rapid labor
14. Deep transverse arrest of descent of presenting part of the fetus
15. Oligohydramnios
16. Abnormal presentation (breech)
17. Use of midcavity forceps or vacuum extraction
18. Versions and extractions
19. Very low birth weight infant or extreme prematurity
20. Fetal macrosomia
21. Large fetal head

22. Fetal anomalies

Birth injuries can be divided into two categories based on their etiology[47].

1) Anoxic birth trauma, insults from hypoxia and ischemia,

2) Mechanical birth trauma, insults from mechanical forces during the process of labor and delivery.

TYPES OF BIRTH TRAUMA:

1) Head & Neck injuries

2) Cranial Nerve, Spinal cord and Peripheral nerve injuries

3) Bone injuries

4) Intra abdominal injuries

5) Skin and soft tissue injuries

1) HEAD & NECK INJURIES:

INJURIES RELATED TO INTRAPARTUM FETAL MONITORING:

Continuous monitoring of the fetal heart rate and the intermittent sampling of fetal scalp blood for determination of acid-base status often are used to monitor the fetus during labor.

Injuries Related to Direct Fetal Heart Rate Monitoring[23]:

Direct monitoring of the fetal heart rate during labor depends on application of an electrode to the fetal scalp or other presenting part. Superficial abrasions, lacerations, and hematomas can occur rarely at the site of application of the electrode. These complications require no specific therapy beyond local treatment. Rarely, abscesses of the scalp may follow application of scalp electrodes. These abscesses usually have been sterile and have required only local treatment. Systemic signs or symptoms require evaluation for possible septicemia.

Injuries Related to Fetal Scalp Blood Sampling[23]:

Fetal biochemical monitoring requires puncture of the presenting part, usually the scalp, with a 2-mm blade. Major complications that may occur rarely are excessive bleeding and accidental breakage of the blades. The bleeding can be stopped by pressure, but on occasion this may require sutures. Rarely blood replacement may be required. The second major complication has been breakage of the blade within the fetal scalp. Removal soon after delivery has been recommended to prevent secondary infection.

INJURIES TO THE HEAD:

EXTRA CRANIAL INJURIES:

CAPUT SUCCEDANEUM:

Caput succedaneum, a frequently observed lesion, is characterized by a vaguely demarcated area of edema over that portion of the scalp that was the presenting part during a vertex delivery. Serum or blood or both accumulate above the periosteum in the presenting part during labor. This extravasation results from the higher pressure of the uterus or vaginal wall on those areas of the fetal head that border the caput. The soft swelling is usually a few millimeters thick and may be associated with overlying petechiae, purpura, or ecchymoses. After an especially difficult labor, an extensive caput may obscure various sutures and fontanelles. Usually no specific treatment is indicated. A caput succedaneum usually resolves within several days.

CEPHALHEMATOMA:

Cephalhaematoma is an infrequently seen subperiosteal collection of blood overlying a cranial bone. The incidence is 0.4% to 2.5% of live births[23]; the frequency is higher in male infants and in infants born to primiparous mothers. Hughes[8] et al found 0.5% as incidence of cephalhaematoma. Thacker [67]KE et al found 2.5% as incidence of cephalhaematoma in study conducted over period of 10 years. A cephalhaematoma is caused during labor or delivery by a rupture of blood vessels that traverse from skull to periosteum. Repeated buffeting of the fetal skull against the maternal pelvis during a prolonged or difficult labor and mechanical trauma caused by use of forceps in delivery has been implicated. The bleeding is sharply limited by periosteal attachments to the surface of one cranial bone; there is no extension across suture

lines. The bleeding usually occurs over one or both parietal bones. Less often it involves the occipital bones and, very rarely, the frontal bones. The overlying scalp is not discolored. Because subperiosteal bleeding is slow, the swelling may not be apparent for several hours or days after birth. The swelling is often larger on the second or third day, when sharply demarcated boundaries are palpable. The cephalhaematoma may feel fluctuant and often is bordered by a slightly elevated ridge of organizing tissue that gives the false sensation of a central bony depression. In 1974, Zelson et al[73] noted an underlying skull fracture in 5.4% of Cephalhaematomas. These fractures are almost always linear and nondepressed. X ray skull indicated if skull fracture suspected.

Therapy is not indicated for the uncomplicated cephalhaematoma. Significant hyperbilirubinemia also may result, necessitating phototherapy or other treatment of jaundice. The most common associated complications are skull fracture and intracranial hemorrhage. Routine incision or aspiration of a cephalhaematoma is contraindicated because of the risk of introducing infection. If a local infection is present, surgical drainage and specific antibiotic therapy should be instituted. Most cephalhaematomas are resorbed within 2 weeks to 3 months, depending on their size; most of these are resorbed by 6 weeks. In a few patients, calcium is deposited, causing a bony swelling that may persist for several months and, rarely, up to 1½ years.

SUBGALEAL HAEMORRHAGE:

Subgaleal hemorrhage (SGH) is a collection of blood in the soft tissue space between the galea aponeurotica and the periosteum of the skull. The incidence is approximately 4 per

10,000 deliveries[23], with an even higher incidence after instrumental deliveries. Ng and et al[53] have reported an incidence of 64 per 10,000 deliveries when vacuum extraction is performed compared with an overall incidence of 0.8 per 1000 deliveries.

The most common predisposing factor is difficult instrumental delivery, particularly midforceps delivery and vacuum extraction.[27,59] The risk of SGH may be reduced by use of softer silicone vacuum cups instead of the original rigid metallic ones.[13] Other factors include coagulopathies,[16,61] prematurity, macrosomia, fetal dystocia, and precipitous labor.[37] The loose connective tissue of the subgaleal space can accommodate as much as 260 ml of blood.[59] SGH may result from an associated skull fracture or rupture of an interosseous synchondrosis (primarily between the parietal bones), in turn causing injury to major intracranial veins or sinuses. Another possible mechanism results from distortion of or traction on emissary veins bridging the subdural and subgaleal spaces.[27]

Early manifestations may be limited to pallor, hypotonia, and diffuse swelling of the scalp. The development of a fluctuating mass straddling cranial sutures, fontanelles, or both is highly suggestive of the diagnosis.[27] Because blood accumulates beneath the aponeurotic layer, ecchymotic discoloration of the scalp is a later finding.[45] This often is associated with pitting edema and progressive posterior spread toward the neck and lateral spread around the ears, frequently displacing the ears anteriorly. Periorbital swelling and ecchymoses also are commonly observed.[59] Eventually, hypovolemic shock and signs of cerebral irritation develop. Massive lesions can cause extracranial cerebral compression, which may lead to rapid neurologic decompensation.[7, 53]

SGH should be considered in infants who show signs of hypoperfusion and falling hematocrit after attempted or successful vacuum delivery, even in the absence of a detectable fluctuant mass. Standard radiographs of the skull may identify possible associated fractures. CT scanning may demonstrate abundant epicranial blood, parieto-occipital bone dehiscence, bone fragmentation, and posterior cerebral interhemispheric densities compatible with subarachnoid hemorrhage.[27] Prompt restoration of blood volume with fresh frozen plasma or blood is essential. If the bleeding continues, gentle compression wraps may be applied to the head; however, the value of this therapy is only anecdotal.[8] In the presence of continued deterioration, surgery may be considered as a last resort. A bicoronal incision allows for exposure of the subgaleal space. Bipolar cauterization of any bleeding points can then be accomplished, and a drain can be left in the subgaleal space. Nearly 25% of infants with SGH die.[35,59] More experience with aggressive and timely surgical intervention may help to improve outcomes.

CRANIAL INJURIES:

SKULL FRACTURES:

Fracture of the neonatal skull is uncommon because the bones of the skull are less mineralized at birth and thus more compressible. In addition, the separation of the bones by membranous sutures usually permits enough alteration in the contour of the head to allow its passage through the birth canal without injury. Padmini et al[55] reported an incidence of 0.18 per 1000 live births. Skull fractures usually follow a forceps delivery or a prolonged, difficult labor with repeated forceful contact of the fetal skull against the maternal symphysis pubis, sacral

promontory, or ischial spine. They have also been described after a vacuum extraction delivery.[32] Most of the fractures are linear. Depressed fractures almost always result from forceps application. However, they may occur spontaneously after cesarean section[21,25] or vaginal delivery without forceps. Factors that have been implicated include pressure on the fetal skull by a maternal bony prominence (e.g., sacral promontory) or uterine fibroid, a fetal hand or foot, or the body part of a twin. Occipital bone fractures usually occur in breech deliveries as a consequence of traction on the hyperextended spine of the infant when the head is fixed in the maternal pelvis. Linear fractures over the convexity of the skull frequently are accompanied by soft tissue changes and cephalhaematoma.

Uncomplicated linear fractures over the convexity of the skull usually do not require treatment. Fractures at the base of the skull often necessitate blood replacement for severe hemorrhage and shock in addition to other supportive measures. If cerebrospinal fluid rhinorrhea or otorrhea is present, antimicrobial coverage is indicated to prevent secondary infection of the meninges. Small (less than 2 cm) “Ping-Pong” fractures may be observed without surgical treatment. Loeser et al[43] reported on three infants with depressed skull fractures in whom spontaneous elevation of the fractures occurred within 1 day to 3½ months of age. Several nonsurgical methods have been described for elevation of depressed skull fractures in certain infants.

Simple linear fractures usually heal within several months without sequlae. Basal fractures carry a poor prognosis. When separation of the basal and squamous portions of the occipital bone occurs, the outcome is almost always fatal; surviving infants have an extremely high incidence of neurologic sequelae. The prognosis for a depressed fracture is usually good

when treatment is early and adequate. When therapy is delayed, especially with a large depression, death may occur from pressure on vital areas of the brain.

INTRA CRANIAL INJURIES:

INTRACRANIAL HEMORRHAGE:

The incidence of symptomatic ICH in term infants is approximately 5.1 to 5.9 per 10,000 live births.[62,68] Traumatic ICH include Epidural(EDH), Subdural(SDH), Subarachnoid(SAH), and less commonly intraventricular, intracerebral, or intracerebellar. Risk factors include forceps delivery, vacuum extraction, precipitous delivery, prolonged second stage of labor, and macrosomia.[60,62,68] The incidence of birth associated ICH has decreased in recent years, secondary to discontinuation of midforceps deliveries and softer more pliable vacuum extractor devices.[62] The most common presenting symptoms are apnoea and seizures.[60] Although symptoms may not be evident immediately after birth, 87% of infants with ICH become symptomatic within 48 hours of birth.

EPIDURAL HAEMORRHAGE:

The incidence is rare, occurring in only 2.2% of autopsies with an ICH.[66] EDH primarily arises from injury to middle meningeal artery, and is frequently associated with a skull fracture or a cephalhaematoma.[52] The rarity of EDH in newborns is due to the absence of middle meningeal artery groove in the neonatal cranial bones, making the artery less susceptible for injury. Although some investigators have postulated that the low incidence of EDH may also be related to the tight attachment of the dura to periosteum, this is disputed by Takagi et al's

finding in autopsy specimens that the dura was easily separated from the periosteum.[66] Clinical manifestations include diffuse neurological symptoms with increased ICP and bulging fontanalle, or more localized symptoms such as lateralizing seizures and eye deviation. Diagnosis by cranial CT, showing high density lentiform lesion in the temporo parietal region. Most infants require surgical drainage; however Negishi et al described 4 infants in whom nonsurgical treatment was successful.[52]

SUBDURAL HAEMORRHAGE:

SDH is the most frequent ICH related to BT. Polina et al[60] reported that SDH accounted for 73% of intracranial birth injuries in term newborns. The incidence of SDH ranges from 2.9 per 10,000 live births in spontaneous deliveries to 8 to 10 per 10,000 live births in vacuum and forceps deliveries.[68] SDH arises from traumatic tearing of veins and venous sinuses at 4 possible locations. The most common locations for SDH are tentorial and interhemispheric.[60] Occipital osteodiastesis is associated with breech delivery. Respiratory symptoms such as apnoea and dusky episodes are the initial clinical findings in 40% to 60% 0f infant.[60] Seizures, focal neurological deficits, lethargy and other neurological symptoms are the initial clinical manifestations. SDH over cerebral convexity tend to produce focal neurologic dysfunction. Posterior fossa SDH are more likely to produce signs of increased ICP, including apnoea, unequal pupils, eye deviation and coma. The onset of symptoms is usually within 24 hours of birth, but some infants may not become symptomatic until 4 to 5 days after delivery.[33, 58]

Cranial CT is the procedure of choice. Many neonates can be treated conservatively.[58] The need for surgery depends on the size of the lesion and presence of brainstem

compression.[58] In one series, 53% of patients with posterior fossa SDH required surgical evaluation. Occasionally VP shunt placement is required, due to progressive hydrocephalus. The long term prognosis depends on the size of the lesion and the presence of intraparenchymal lesions. In a series of 45 infants without an associated intracerebral haemorrhage, Hayashi et al [31]found that 70% were neurologically normal at follow up. In a series of 15 patients with posterior fossa SDH with follow up at mean of 4.5 years, Perrin et al[58] found that 20% were mildly developmentally delayed, 13% were moderately delayed, and 20% were severely delayed.

SUBARACHNOID HAEMORRHAGE:

The incidence of symptomatic SAH ranges from 1.3 per 10000 live births in spontaneous vaginal deliveries to 2 or 3 per 10,000 live births in vacuum and forceps deliveries.[68] Increased incidence occurs with prematurity and asphyxia[1]. In neonates, SAH is caused by rupture of the bridging veins of the subarachnoid space or small leptomeningeal vessels. Haemorrhage from ruptured intra cranial aneurysm or bleeding AV malformation is very rare in newborns. Although SAH may be asymptomatic, the most common presentation is seizures, often occurring on the 2nd day life[24]. Neurologic examination is usually normal during inter ictal periods; however irritability or depressed level of consciousness may be present. Underlying contusion may cause focal neurologic signs. Diagnosis is confirmed by cranial CT. Unless bleeding is massive, SAH in term neonates resolves without intervention[30]. Usually there are no long term sequelae if underlying cortical injury and hypoxic injury are not present[1,24]. If the SAH is large, posthaemorrhagic hydrocephalus may develop; therefore, monitoring head growth and ventricular size with cranial USG is indicated[30].

Face

FRACTURES AND DISLOCATIONS OF FACIAL BONES :

Facial bone fractures may occur during passage through the birth canal, during forceps application and delivery, and during obstetric manipulation (most often the Mauriceau maneuver for delivery of the fetal head in a breech presentation). Manipulation may result in mandibular fractures and mandibular joint damage but is rarely severe enough to cause separation of the symphysis of the mandible. Fracture of the nose may result in early respiratory distress and feeding difficulties.

The most frequent nasal injury is dislocation of the cartilaginous part of the septum from the vomerine groove and columella. The reported incidence is 0.6% to 0.9% of deliveries[8]. This may result from intrauterine factors such as a uterine tumor or persistent pressure on the nose by fetal small parts or during delivery from pressure on the nose by the symphysis pubis, sacral promontory, or perineum. The presence of nasal septal dislocation may be differentiated from the more common normal variant of a misshapen nose by a simple compression test, in which the tip of the nose is compressed[18]. Fractures of the maxilla, lacrimal bones, and nose warrant immediate attention because they unite quickly, with fixation in 7 to 10 days. Nasal trauma frequently requires extensive surgery. While waiting, the pediatrician should provide an oral airway to relieve respiratory distress.[65]

Eyes

Mechanical trauma to various regions of the neonatal eye usually occurs during

abnormal presentation, in dystocia from cephalopelvic disproportion, or as a result of inappropriate forceps placement in normal deliveries. Most of the injuries are self-limited and mild and require no specific treatment. But significant ocular injury occurs in approximately 0.19% of deliveries[47]. Subconjuctival haemorrhage may be found after a difficult delivery but often is noted after easy, completely uncomplicated deliveries. This finding is considered to result from increased venous pressure in the infant's head and neck, produced by obstruction to venous return consequent to compression of the fetal thorax or abdomen by uterine contractions during labor.[40] If the infant is otherwise well, management consists of reassuring the parents. The blood is usually absorbed within 1 to 2 weeks. Trauma at birth may result in retinal hemorrhage, hyphema, or vitreous hemorrhage, with retinal hemorrhage the most common. The cause is most likely compression of the fetal head, resulting in venous congestion. The fetal head is compressed two to four times more forcefully than other fetal parts during the second stage of labor. Retinal hemorrhage is more common in primiparous deliveries and after forceps or vacuum extraction; it is rare after cesarean section. It may occur in normal deliveries. It usually disappears within 1 to 3 days (occasionally 5 days) with no residual effects. Rarely, hemorrhages may take as long as 21 days to resolve.

Ears

The proximity of ears to the site of application of forceps makes them susceptible to injury at birth. Most of the injuries are mild and self-limited, but serious injuries may occur because of slipping or misplacement of forceps. Abrasions must be cleansed gently to minimize the risk of secondary infection. Hematomas of the external ear, if not treated promptly, liquefy slowly, followed by early organization and development of cauliflower ear. Lacerations of the

auricle may be repaired by the pediatrician if they are superficial and involve only skin. If the laceration involves cartilage, surgical consultation should be obtained because of the tendency toward postoperative perichondritis, which is refractory to treatment and leads to subsequent deformities.

Injury to the Sternocleidomastoid Muscle:

Injury to the sternocleidomastoid muscle is designated muscular torticollis, congenital torticollis, or sternocleidomastoid fibroma. Incidence[36] 0.4%. The birth trauma theory suggests that the muscle or fascial sheath is ruptured during a breech or difficult delivery involving hyperextension of the muscle. A haematoma develops and is subsequently invaded by fibrin and fibroblasts with progressive formation of scar tissue and shortening of the muscle. A mass in the midportion of the sternocleidomastoid muscle may be evident at birth, although usually it is first noted 10 to 14 days after birth. It is 1 to 2 cm in diameter, hard, immobile, fusiform, and well circumscribed; there is no inflammation or overlying discoloration. The mass enlarges during the following 2 to 4 weeks and then gradually regresses and disappears by age 5 to 8 months. A transient torticollis produced by contracture of the involved muscle appears soon after birth. The head tilts toward the involved side, and the chin is somewhat elevated and rotated toward the opposite shoulder. The head cannot be moved passively into normal position.

Treatment should be instituted as early as possible. The involved muscle should be stretched to an overcorrected position by gentle, even, and persistent motion with the infant supine. Conservative therapy should be continued for 6 months. If the deformity has not been

fully corrected, surgery should be considered to prevent permanent skull and cervical spine deformities. Akazawa et al[4] reported favorable results after partial resection. Most infants treated conservatively show complete recovery within 2 to 3 months. If surgery is necessary and if it is performed early, the facial asymmetry will disappear almost entirely. Infants treated before their first birthday has a better outcome than those treated later, regardless of the type of treatment. Nonsurgical treatment after 1 year is rarely successful.

2) NERVE CRANIAL NERVE, PERIPHERAL AND SPINAL CORD INJURIES:

BRACHIAL PALSY:

Incidence of brachial palsy decreased from 1.56 per 1000 live births in 1938 to 0.38 per 1000 in 1962, reported by Adler and Patterson in New York. Subsequent reports have suggested an increase in incidence, possibly because of increase in mean birth weight. Bennett and Harrold reported an incidence of 0.61 per 1000 in the United Kingdom in 1976, Greenwald et al reported an incidence of 2.0 per 1000 in USA in 1984, Sjoberg et al reported an incidence of 1.9 per 1000 in Sweden in 1988, and in 1990 al-Rajeh et al reported an incidence of 1.19 per 1000 in Saudi Arabia, Evans jones et al, 1998-99 reported an incidence of 0.4 per 1000 live births in UK.

3 main forms of brachial palsy [23] occur, depending on the site of injury: (1) Duchenne-Erb, or upper arm, paralysis, which results from injury of the C5 & C6 and is by far the most common(90%); (2) Klumpke, or lower arm, paralysis, which results from injury of the eighth cervical and first thoracic roots and is extremely rare(<1%); and (3) paralysis of the entire arm, Total Brachial Palsy occurs approximately 10% of cases.

Most cases of brachial palsy follow a prolonged and difficult labor culminating in a traumatic delivery. The affected infant is frequently large, relaxed, and asphyxiated and thereby vulnerable to excessive separation of bony segments, overstretching, and injury to soft tissues. Injury of the fifth and sixth cervical roots may follow a breech presentation with the arms extended over the head; excessive traction on the shoulder in the delivery of the head may result in stretching of the plexus. The same injury may follow lateral traction of the head and neck away from one of the shoulders during an attempt to deliver the shoulders in a vertex presentation. More vigorous traction of the same nature results in paralysis of the entire arm. The mechanism for isolated lower arm paralysis is uncertain; it is thought to result from stretching of lower plexus nerves under and against the coracoid process of the scapula during forceful elevation and abduction of the arm. Excessive traction on the trunk during a breech delivery may result in avulsion of the lower roots from the cervical cord. In most patients the nerve sheath is torn and the nerve fibers are compressed by the resultant hemorrhage and edema. Less often the nerves are completely ruptured and the ends severed, or the roots are avulsed from the spinal cord with injury to the spinal gray matter. One study, while confirming the well-known association of shoulder dystocia and brachial plexus injury in macrosomic infants, also identified an increased incidence of other malpresentations in low- and normal-weight infants with brachial plexus injury.[26]

The infant with upper arm paralysis holds the affected arm in a characteristic position, reflecting involvement of the shoulder abductors and external rotators, forearm flexors and supinators, and wrist extensors. The arm is adducted and internally rotated, with extension at the elbow, pronation of the forearm, and flexion of the wrist. When the arm is passively

abducted, it falls limply to the side of the body. Moro, biceps, and radial reflexes are absent on the affected side. There may be some sensory deficit on the radial aspect of the arm, but this is difficult to evaluate in the neonate. The grasp reflex is intact. Any signs of respiratory distress may indicate an accompanying ipsilateral phrenic nerve root injury.

Lower arm paralysis involves the intrinsic muscles of the hand and the long flexors of the wrist and fingers. The hand is paralyzed, and voluntary movements of the wrist cannot be made. The grasp reflex is absent; the deep tendon reflexes are intact. Sensory impairment may be demonstrated along the ulnar side of the forearm and hand. Frequently, dependent edema and cyanosis of the hand and trophic changes in the fingernails develop. After some time there may be flattening and atrophy of the intrinsic hand muscles. Usually an ipsilateral Horner syndrome (ptosis, miosis and enophthalmos) also is present because of injury involving the cervical sympathetic fibers of the first thoracic root. Often this is associated with delayed pigmentation of the iris, sometimes of more than 1 year's duration.

When the entire arm is paralyzed, it is usually completely motionless, flaccid, and powerless, hanging limply to the side. All reflexes are absent. The sensory deficit may extend almost to the shoulder.

A careful radiographic study of the shoulder, including an examination of the lower cervical spine, clavicle, and upper humerus, should be made to exclude tearing of the joint capsule, fracture of the clavicle, and fracture, dislocation, or upper epiphyseal detachment of the humerus.

The basic principle of treatment historically has been conservative, with initial emphasis

on prevention of contractures while awaiting recovery of the brachial plexus. During the last decade, this approach has been replaced by a more comprehensive program that combines initial conservative management with closer follow-up and earlier decision regarding surgical intervention. This is best represented by the care plan developed by Shenaq et al[64]. If improvement in deltoid, biceps, and triceps function has not occurred by the third month of life, functional outcome without surgery is unlikely. Consequently, a decision for surgery should be made by the end of the third month, followed by primary brachial plexus exploration during the fourth month.

Initial surgical intervention beyond 12 months of age at the level of the cervical root alone has resulted in disappointing outcomes. However, when infants referred at this age have been offered a combined cervical root and infraclavicular exploration with neurolysis, graph reconstruction, and nerve transfer of appropriate elements in both anatomic compartments, improved outcomes have been noted. Blaauw and Slooff have reported their experience with transfer of pectoral nerves to the musculocutaneous nerve in 25 patients, 22 of whom had upper root avulsions. Seventeen patients, including one who went to surgery at 3 months of age, had excellent outcomes, five had fair outcomes, and two were considered treatment failures. This aggressive approach has resulted in up to 90% of patients demonstrating useful function of muscle groups above the elbow. Function below the elbow has been characterized by 50% to 70% recovery because of the increased distance required for nerve regeneration.

Although most (93% to 95%) infants achieve return of function with conservative management, the remainder with persistent deficits may go on to development of long-term severe handicaps of the affected extremity. Early treatment offers significant improvement for

approximately 90% of these children. Later treatment reduces this number to 50% to 70%. To avoid missing the window of opportunity for timelier and more successful treatment, infants with brachial plexus palsy should be referred to centers that have an established comprehensive management protocol.

FACIAL NERVE PALSY:

The incidence of facial nerve palsy ranges from 1.8 to 7.5 per 1000 live births. Traumatic facial nerve palsy most often follows compression of the peripheral portion of the nerve, either near the stylomastoid foramen, through which it emerges, or where the nerve traverses the ramus of the mandible. The nerve may be compressed by forceps, especially when the fetal head has been grasped obliquely. The condition also occurs after spontaneous deliveries in which prolonged pressure was applied by the maternal sacral promontory. This condition may occur rarely with simultaneous ipsilateral brachial plexus palsy, most likely secondary to compressive forces during delivery.[19] Contributing factors include prolonged second stage of labor and midforceps delivery. Traumatic facial nerve palsy may follow a contra lateral injury to the CNS, such as a temporal bone fracture, or hemorrhage, tissue destruction, or both to structures within the posterior fossa. This CNS injury is less frequent than peripheral nerve injury.

Central paralysis is a spastic paralysis limited to the lower half or two thirds of the contra lateral side of the face. Usually other manifestations of intracranial injury appear most often sixth cranial nerve palsy.

Peripheral paralysis is flaccid and, when complete, involves the entire side of the face.

When the infant is at rest, the only sign may be a persistently open eye on the affected side, caused by paralysis of the orbicular muscle of the eye. With crying, the findings are the same as in a central facial nerve injury, with the addition of a smooth forehead on the involved side. Because the tongue is not involved, feeding is not affected.

A small branch of the nerve may be injured, with involvement of only one group of facial muscles. Paralysis is then limited to the forehead, eyelid, or mouth. Peripheral paralysis caused by nerve injury distal to the geniculate ganglion may be accompanied by a hematotympanum on the same side.

Central and peripheral facial nerve palsies must be distinguished from nuclear agenesis (Möbius syndrome). The latter frequently results in bilateral facial nerve palsy; the face is expressionless and immobile, suggesting muscle fibrosis. Other cranial nerve palsies and deformities of the ear, palate, tongue, mandible, and other bones may be associated with Möbius syndrome. Congenital absence or hypoplasia of the depressor muscle of the angle of the mouth also may simulate congenital facial palsy and has been associated with an increased incidence of other congenital anomalies.

No specific therapy is indicated for most facial palsies. If the paralysis is peripheral and complete, initial treatment should be directed at protecting the cornea with an eye pad and instilling 1% methylcellulose drops every 4 hours. The functional state of the nerve should be followed closely. Falco and colleagues[22] proposed the comprehensive approach; neuro surgical repair should be considered only after lack of resolution during 1 year of observation.

Most facial palsies resolve spontaneously within several days; total recovery may

require several weeks or months. Electro diagnostic testing is beneficial in predicting recovery; repeatedly normal nerve excitability indicates a good prognosis, but decreased or absent excitability early in the course suggests a poor outlook. The subsequent appearance of muscle fibrillation potentials indicates nerve degeneration. The prognosis in surgically treated infants worsens with increasing age at treatment.

RECURRENT LARYNGEAL NERVE PALSY:

Recurrent laryngeal nerve injury causes vocal cord paralysis. 5% to 26% of congenital vocal cord paralysis is due to BT[16, 47]. Unilateral vocal cord paralysis may be a consequence of excessive traction on the head during a breech delivery or lateral traction with forceps in a cephalic presentation. The recurrent laryngeal branch of the vagus nerve in the neck is injured. The left side is involved more often because of this nerve's lower origin and longer course in the neck. Bilateral paralysis may be caused by peripheral trauma involving both recurrent laryngeal nerves, but more frequently it is caused by a CNS insult such as hypoxia or hemorrhage involving the brainstem.

An infant with a unilateral paralysis may be completely free of symptoms when resting quietly, but crying is usually accompanied by hoarseness and mild inspiratory stridor. When associated with difficulty in feeding and clearing secretions, concurrent involvement of the 12th (hypoglossal) cranial nerve should be suspected. Bilateral paralysis results in more severe respiratory symptoms. At birth the infant may have difficulty in establishing and maintaining spontaneous respiration; later, dyspnea, retractions, stridor, cyanosis, or aphonia may develop. Infants with unilateral paralysis should be observed closely until there is evidence of

improvement. Gentle handling and frequent small feedings aid in keeping the infant quiet and minimizing the risk of aspiration. Bilateral paralysis necessitates immediate tracheal intubation to establish an airway. Tracheostomy is required subsequently in most patients. Laryngoscopic examinations then should be performed at intervals to look for evidence of return of vocal cord function; early extubation may be attempted if complete return occurs within a short time. Unilateral paralysis usually resolves rapidly without treatment, and complete resolution occurs within 4 to 6 weeks. The prognosis for bilateral paralysis is more variable.

PHRENIC NERVE PARALYSIS:

Phrenic nerve paralysis results in diaphragmatic paralysis and rarely occurs as an isolated injury in the neonate. Most injuries are unilateral and 75% are associated with an ipsilateral upper brachial plexus palsy[8,20]. The most common cause is a difficult breech delivery. Lateral hyperextension of the neck results in overstretching or avulsion of the third, fourth, and fifth cervical roots, which supply the phrenic nerve.

The first sign may be recurrent episodes of cyanosis, usually accompanied by irregular and labored respirations. In a severe injury, tachypnea, weak cry, and apneic spells may occur. Radiographs show the apparent elevation of the diaphragm. Early diagnosis can be confirmed by real-time ultrasonographic examination of the diaphragm, which reveals abnormal motion of the affected hemidiaphragm. Fluoroscopy should be reserved for the equivocal case.

Infants require only nonspecific medical treatment. The infant should be positioned on the involved side, and oxygen should be administered for cyanosis or hypoxemia. Intravenous fluids may be necessary for the first few days. If the infant begins to show improvement,

progressive oral or gavage feedings may be started. Antibiotics are indicated if pneumonia occurs. Infants with more severe respiratory distress, particularly those with bilateral phrenic nerve palsy, may require assisted ventilation shortly after delivery. de Vries Reilingh et al[20] have reviewed their experience with 23 infants who incurred phrenic nerve injury as neonates. Infants who had not recovered diaphragmatic function after 30 days of conservative treatment did not demonstrate spontaneous recovery thereafter. Infants in this category should be considered candidates for plication of the diaphragm early in the second month of life. Many infants recover spontaneously.

INJURIES TO THE SPINE AND SPINAL CORD:

Birth injuries to the vertebral spine and spinal cord are rarely diagnosed. It is not certain whether the low incidence is real, reflecting improved obstetric techniques, or represents a tendency for postmortem examination to overlook spine and spinal cord lesions. Incidence reported 0.14 per 10,000 live births[47]. These injuries almost always result from breech deliveries,[15] especially difficult ones in which version and extraction were used. Other predisposing factors include brow and face presentations, dystocia (especially shoulder), prematurity, primiparity, and precipitous delivery. Although cesarean delivery has been recommended as optimal for infants in breech presentation with a hyperextended head, Maekawa et al[44] documented spinal cord injury after cesarean section. Difficulty in delivery of the shoulders in cephalic presentations may result in a similar mechanism of injury. The lower cervical and upper thoracic regions are most often involved, but occasionally the entire length of the spinal canal contains a heavy accumulation of blood.

Affected infants may follow one of four clinical patterns. Those in the first group are either stillborn or in poor condition from birth, with respiratory depression, shock, and hypothermia. They deteriorate rapidly; death occurs within several hours, often before neurologic signs are obvious. The second group consists of infants who at birth may appear normal or show signs similar to those of the first group; these infants die after several days. The brachial plexus is involved in approximately 20% of all cases. The third group, with lesions at the seventh cervical to first thoracic vertebra or lower, comprises infants who survive for long periods, some for years. Paraplegia noted at birth may be transient. Infants in the fourth group have subtle neurologic signs of spasticity thought to represent cerebral palsy. MRI is the only

procedure that provides a direct image of the spinal cord and clearly is the most reliable modality available to evaluate presumptive cervical spinal cord injury in the infant.[41,48]

Treatment is supportive and usually unsatisfactory. The infant affected at birth requires basic resuscitative and supportive measures. Infants who survive present a therapeutic challenge that can be met only by the combined and interested efforts of the pediatrician, neurologist, neurosurgeon, urologist, psychiatrist, orthopedist, nurse, physical therapist, and occupational therapist. The prognosis varies with the severity of the injury. Most severe injuries result in death shortly after birth. Infants with cord compression from vertebral fractures or dislocations or both may recover with reasonable return of function if prompt neurosurgical removal of the compression is performed. Infants with mild injuries or partial transections may recover with minimal sequelae. MRI evidence of hemorrhage in the cervical spinal cord portends a poor neurologic outcome.[48] If MRI reveals extensive edema in multiple spinal cord segments without concurrent hemorrhage, complete recovery is possible.[48] Many die in infancy of ascending urinary tract infection and sepsis.

3) INJURIES TO INTRA-ABDOMINAL ORGANS:

Intra-abdominal injuries are rare and involve rupture or sub capsular hemorrhage into the liver, spleen, or adrenal gland. Liver injury is the most common of these injuries. In an analysis of 783 neonatal deaths, French et al found that 15% had large hepatic sub capsular hematomas or a hemoperitoneum from a ruptured liver[47]. When screening ultrasounds are performed, the incidence of adrenal hemorrhage is 1.9 per 1000 live births[47]. The incidence of intra-abdominal injury is increased in complicated deliveries, prematurity, and presence of hepatosplenomegaly, coagulation disorders, and asphyxia. Three potential mechanisms lead to intra-abdominal injury: (1) direct trauma, (2) compression of the chest against the surface of the spleen or liver, and (3) chest compression leading to tearing of the ligamentaous insertions of the liver or spleen.

Clinical presentation depends on the degree of blood loss. With hepatic or splenic rupture, patients develop sudden pallor, hemorrhagic shock, abdominal distention, and abdominal discoloration. Presentation of a liver rupture with scrotal swelling and discoloration has been described. Sub capsular hematomas may present more insidiously, with anemia, poor feeding, tachypnea, and tachycardia. Signs and symptoms may be delayed as a sub capsular hematoma gradually enlarges and then subsequently ruptures, leading to an acute deterioration. Adrenal hemorrhage may present as a flank mass.

Diagnosis is best made by abdominal ultrasound. Computed tomography is also useful, but requires transport of an often critically ill infant. Abdominal radiographs may how nonspecific intra peritoneal fluid or hepatomegaly. Abdominal paracentesis is diagnostic if a

hemoperitoneum is present. Treatment begins with volume replacement and correction of any coagulopathy. If the infant is hemodynamically stable and a sub capsular hematoma is present, conservative management is indicated. With rupture or hemodynamic instability, a laparotomy is required to control the bleeding. Patients with adrenal hemorrhage may require hormone replacement therapy.

4) BONE INJURIES:

Fracture of the Clavicle:

The clavicle is the most frequently fractured bone during labor and delivery. Most clavicular fractures are of the greenstick type, but occasionally the fracture is complete. The incidence is 0.3% to 2.9% of newborns[48].The major causes of clavicular fractures are difficult delivery of the shoulders in vertex presentations and extended arms in breech deliveries. Vigorous, forceful manipulation of the arm and shoulder usually has occurred. However, fracture of the clavicle may also occur in infants after apparently normal labor and delivery.[39]

Most often a greenstick fracture is not associated with any signs or symptoms but is first detected after the appearance of an obvious callus at 7 to 10 days of life. Thus the majority of neonatal clavicular fractures are diagnosed at discharge or at the first follow-up visit.[38] Complete fractures and some greenstick fractures may be apparent shortly after birth; movement of the arm on the affected side is decreased or absent. Deformity and, occasionally, discoloration may be visible over the fracture site with obliteration of the adjacent supraclavicular depression as a result of sternocleidomastoid muscle spasm. Passive movement of the arm elicits cries of pain from the infant. Palpation reveals tenderness, crepitus, and

irregularity along the clavicle. Moro reflex on the involved side is characteristically absent. Radiographs confirm the diagnosis of fracture.

Therapy is directed toward minimizing the infant's pain. The affected arm and shoulder should be immobilized with the arm abducted more than 60 degrees and the elbow flexed more than 90 degrees. A callus forms, and pain usually subsides by 7 to 10 days, when immobilization may be discontinued. Prognosis is excellent, with growth resulting in restoration of normal bone contour after several months.

Fracture of the Humerus:

After the clavicle, the humerus is the bone most often fractured during the birth process. Incidence 0.05 per 1000 live births[48]. The most common mechanisms responsible are difficult delivery of extended arms in breech presentations and of the shoulders in vertex presentations. Besides traction with simultaneous rotation of the arm, direct pressure on the humerus also is a factor. This may account for the occurrence of fracture of the humerus in spontaneous vertex deliveries.

The fractures are usually in the diaphysis. They are often greenstick fractures, although complete fracture with overriding of the fragments occasionally occurs. A greenstick fracture may be overlooked until a callus is noted. A complete fracture with marked displacement of fragments presents an obvious deformity that calls attention to the injury. Often the initial manifestation of the fracture is immobility of the affected arm. Palpation reveals tenderness, crepitation, and hyper mobility of the fragments. The ipsilateral Moro response is absent. Radiographs confirm the diagnosis.

The affected arm should be immobilized in adduction for 2 to 4 weeks. This may be accomplished by maintaining the arm in a hand-on-hip position with a triangular splint and a Velpeau bandage, by strapping the arm to the chest, or by application of a cast. The prognosis is excellent. Healing is associated with marked formation of callus. Moderate overriding and angulation disappear with time because of the excellent remodeling power of infants. Complete union of the fracture fragments usually occurs by 3 weeks.

Fracture of the Femur:

Fracture of the femur is by far the most common fracture of the lower extremity in the newborn. Incidence 0.13 per 1000 live births[50]. Fracture of the femur usually follows a breech delivery when the leg is pulled down after the breech is already partially fixed in the pelvic inlet or when the infant is improperly held by one thigh during delivery of the shoulders and arms. Femoral fracture even may occur during cesarean delivery.[5] Usually an obvious deformity of the thigh is seen; as a rule the bone breaks transversely in the upper half or third, where it is relatively thin. Less often the injury may not be appreciated until several days after delivery, when swelling of the thigh is noted; this swelling may be caused by hemorrhage into adjacent muscle. The infant refuses to move the affected leg or cries in pain during passive movement or with palpation over the fracture site. Radiographs almost always show overriding of the fracture fragments.

Optimal treatment is traction-suspension of both lower extremities, even if the fracture is unilateral. The legs are immobilized in a spica cast; with Bryant traction the infant is suspended by the legs from an overhead frame, with the buttocks and lower back just raised off

the mattress. The legs are extended and the thighs flexed on the abdomen. The weight of the infant's body is enough to overcome the pull of the thigh muscles and thereby reduce the deformity. The infant is maintained in this position for 3 to 4 weeks until adequate callus has formed and new bone growth has started. During the treatment period, special attention should be given to careful feeding of the infant and to protection of bandages and casts from soiling with urine and feces. The prognosis is excellent; complete union and restoration without shortening are expected.

Dislocations:

Dislocations caused by birth trauma are rare. Often an apparent dislocation is actually a fracture displaced through an epiphyseal plate. Because the epiphyseal plate is radiolucent, a fracture occurring adjacent to an unmineralized epiphysis gives a radiographic picture simulating a dislocation of the neighboring joint. This type of injury has been termed pseudodislocation.[29] Because the humeral and proximal femoral epiphyses are usually not visible on radiographs at birth, a pseudodislocation can occur at the shoulder, elbow, or hip. A true dislocation resulting from birth trauma is that involving the radial head. This has been associated with traumatic breech delivery.

Epiphyseal Separations:

As with dislocations, epiphyseal separations are rare. They occur mostly in primiparity, dystocic deliveries, and breech presentations, especially those requiring manual extraction or version and extraction. Any delivery associated with vigorous pulling may predispose the infant to this injury. The upper femoral and humeral epiphyses are most often involved.

Usually on the second day the soft tissue over the affected epiphysis develops a firm swelling with reddening, crepitus, and tenderness. Active motion is limited, and passive motion is painful. Early radiographs will show only soft tissue swelling, with occasional superolateral displacement of the proximal femoral metaphysis. After 1 to 2 weeks, extensive callus appears, confirming the nature of the injury; during the third week, subperiosteal calcification appears. If possible, treatment should be conservative. Closed reduction and immobilization are indicated within the first few days before rapidly forming fibrous callus prevents mobilization of the epiphysis. The hip is immobilized in the frog-leg position as in congenital dislocation. Poorly immobilized fragments of the proximal or distal femur may require temporary fixation with a Kirschner wire[45]. Union usually occurs within 10 to 15 days. Untreated or poorly treated epiphyseal injuries may result in subsequent growth distortion and permanent deformities such as coxa vara. Mild injuries carry a good prognosis.

5) INJURIES TO SOFT TISSUES:

Erythema and Abrasions:

Erythema and abrasions frequently occur when dystocia has occurred during labor as a result of cephalopelvic disproportion or when forceps have been used during delivery. The affected areas should be kept clean to minimize the risk of secondary infection. These lesions usually resolve spontaneously within several days with no specific therapy.

Petechiae:

Petechiae are observed more frequently after breech deliveries. Petechiae are probably caused by a sudden increase in intrathoracic and venous pressures during passage of the chest through the birth canal. No specific treatment is necessary. Traumatic petechiae usually fade within 2 or 3 days.

Ecchymoses:

Ecchymoses may occur after traumatic or breech deliveries. The incidence is increased in premature infants, especially after a rapid labor and poorly controlled delivery. No local therapy is necessary. The rise in serum bilirubin that follows severe bruising may be decreased by the use of phototherapy. The ecchymoses usually resolve spontaneously within 1 week.

Subcutaneous Fat Necrosis:

Subcutaneous fat necrosis is characterized by well-circumscribed, indurated lesions of the skin and underlying tissue. The cause of subcutaneous fat necrosis is uncertain, although obstetric trauma is considered a possibility. Many affected infants are large and have been delivered by forceps or after a prolonged, difficult labor involving vigorous fetal manipulation. They occur on the cheeks, neck, back, shoulders, arms, buttocks, thighs, and feet, with relative sparing of the chest and abdomen. The lesions vary in size from 1 to 10 cm; rarely, they may be more extensive. They are irregularly shaped, hard, plaquelike, and nonpitting. The overlying skin may be colorless, red, or purple. The affected areas may be slightly elevated above the adjacent skin; small lesions may be easily moveable in all directions. There is no local tenderness or increase in skin temperature.

These lesions require only observation. Surgical excision is not indicated. The lesions slowly soften after 6 to 8 weeks and completely regress within several months. Affected infants should be followed closely during the first 6 weeks for potential development of hypercalcemia. It is important to treat this complication without delay to prevent central nervous system (CNS) and renal sequelae.[17]

Lacerations:

Accidental lacerations may be inflicted with a scalpel during cesarean section. They usually occur on the scalp, buttocks, and thighs, but they may occur on any part of the body. If the wound is superficial, the edges may be held in apposition with butterfly adhesive strips. Deeper, more freely bleeding wounds should be sutured with the finest material available, preferably 7–0 nylon. Rarely, the amount of blood loss and depth of wound require suturing in the delivery room. After repair, the wound should be left uncovered unless it is in an area of potential soiling, such as the perineal area; in such locations the wound should be sprayed with protective plastic. Healing is usually rapid, and the sutures may be removed after 5 days.

AIM OF THE STUDY

1. To study the incidence, morbidity & mortality of birth trauma.

2. To analyze the factors predisposing to birth trauma.

MATERIALS & METHODS

Study design: Prospective, case control study.

Study place: Govt. RSRM Hospital, Stanley Medical College, Chennai.

Study period: October 2007-September 2008.

Inclusion criteria:

All babies with Mechanical Birth Trauma delivered in RSRM hospital during the study period.

Exclusion criteria:

1. Still births
2. Anoxic Birth Trauma
3. Caput Succedaneum

Methodology:

- All babies born during the study period were examined for mechanical BT & included as cases in this study.
- Detailed antenatal history & intrapartum history will be obtained for the cases.
- This is followed by complete physical examination & relevant investigations done for obtaining diagnosis.
- Cases were followed till discharge to record the morbidity & mortality.
- During follow up visits if BT babies found, the same included as cases in our study.

- The first normal baby born after 12 A.M daily during the study period was taken as controls.
- Detailed antenatal & intrapartum history were obtained for controls.
- The babies diagnosed as BT were treated based on standard protocols.
- If needed specialist opinion were obtained for diagnosis & treatment.

Statistical methods used:

- Incidence, morbidity pattern & mode of treatment were given in frequencies and their percentage.
- Maternal weight, parity, weight, height, oxytocin use, duration of labor, shoulder dystocia, mode of delivery, neonatal variables like sex, maturity, size of the, birth weight and resuscitation requirements were analyzed using Pearson chi-square test and Yates corrected chi-square test(X^2).
- Obstetrical complications, late referral, presentation and asphyxia were analyzed using Normal test (Z test).
- Risk factors for accident were identified using multivariate logistic regression analysis.
- P value less than 0.05 was taken as significant.

OBSERVATION & RESULTS

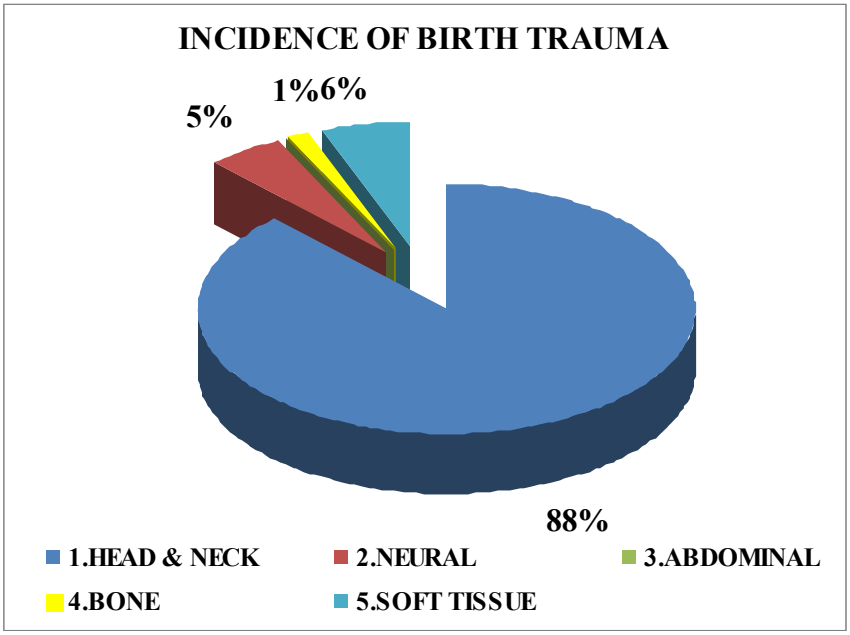
Total deliveries during the study period were 12826, from which 12968 babies were born. 233 cases of still birth were recorded. So the study group constitutes 12735 babies. Out of 12735 babies examined, 283 babies having 288 birth injuries were found as per inclusion criteria. 5 neonates had more than one injury. The incidence of birth trauma was 22.22 per 1000 live births.

Head and neck injuries were commonest with 253(88%) injuries, followed by skin and soft tissue injuries 17(6%), nerve injuries 14(5%), bone injuries 4(1%). No intra abdominal injury has been recorded.

Table I. Incidence of Birth Trauma based on types

Type of birth trauma	Cases* n=283	Incidence % n=12735	Incidence per 1000 live births
1.Head & Neck injuries	248(88)	1.95	19.5
2.Skin & Soft tissue injuries	17(6)	0.13	1.3
3.Nerve injuries	14(5)	0.11	1.1
4.Bone injuries	4(1)	0.03	0.3
5.Intra abdominal injuries	0	0	0
		2.22	22.2

*Figures in parenthesis indicate percentage.



Considering the individual injuries Subconjuctival haemorrhage recorded in 107(37%) babies with an incidence of 0.84% of live births. Cephalhaematoma found in 72 (25%) babies, an incidence of 0.57%. In Head & Neck injuries, abrasions, ecchymoses, laceration found in 31(10.8%), 23(7.9%), 8(2.8%) babies an incidence of 0.24%, 0.18% & 0.06% respectively. Soft tissue contusion and laceration recorded in 13(4.51%), and 4(1.39%) respectively, an incidence of 0.13% when combined together. SGH found in 6(2.08%) neonates an incidence of 0.47 per 1000 live births. Auricle injury noted in 5(1.74%), an incidence of 0.4 per 1000 live births. Sternomastoid tumor found in 1(0.35%) baby, an incidence of 0.08 per 1000 live births. Brachial palsy found in 10(3.47%), an incidence of 0.8 per 1000 live births. Out of this total brachial palsy found in 3(30%) cases. Facial nerve palsy found in 4(1.39%) neonates an incidence of 0.31 per 1000 live births. In bony injuries Fracture clavicle, femur, humerus recorded in 1(0.35%), 2(0.7%), 1(0.35%) babies respectively an incidence of 0.08, 0.16, 0.08 respectively.

Table II. Morbidity pattern in birth trauma

S. No	Injury	n *	Incidence %	Incidence per 1000 live births
1.	Sub conjuctival haemorrhage	107(37)	0.84%	8.4
2.	Cephalhaematoma	72(25)	0.57%	5.7
3..	H & N abrasion	31(10.8)	0.24%	2.4
4.	H & N ecchymoses	23(7.9)	0.18	1.8
5.	Soft tissue contusion	13(4.51)	0.102%	1.0
6.	Brachial palsy	10(3.47)	0.079%	0.8
7.	H & N laceration	8(2.8)	0.06%	0.6
8.	Sub galeal haemorrhage	6(2.08)	0.047%	0.47
9.	Auricle injury	5(1.74)	0.039%	0.4
10.	Facial N palsy	4(1.39)	0.031%	0.31
11.	Soft tissue laceration	4(1.39)	0.031%	0.31
12.	Fracture femur	2(0.7)	0.016%	0.16
13.	Fracture clavicle	1(0.35)	0.008%	0.08
14.	Fracture humerus	1(0.35)	0.008%	0.08
15.	Sternomastoid tumor	1(0.35)	0.008%	0.08

* Figures in parenthesis indicate percentage.

Table III. Distribution of maternal age in birth trauma

S. No	Age group	Cases* n=283	Controls* n=366	p value

1.	15-19 yr	20(7.1)	31(8.5)	X^2=5.08 p=0.16 (NS)
2.	20-24 yr	180(63.6)	215(58.7)	
3.	25-29 yr	74(26.1)	95(26.0)	
4.	30-34 yr	8(2.8)	25(6.8)	
5.	35-39 yr	0	0	
6.	>40 yr	1(0.4)	0	

*Figures in parenthesis indicate percentage.

The common age groups were BT found was 20-24 yrs with 180(63.6%) mothers followed by 25-29 yrs with 74(26.1%) mothers coinciding with common age groups where deliveries are maximum. Maternal age between groups was not significant. (p=0.16).

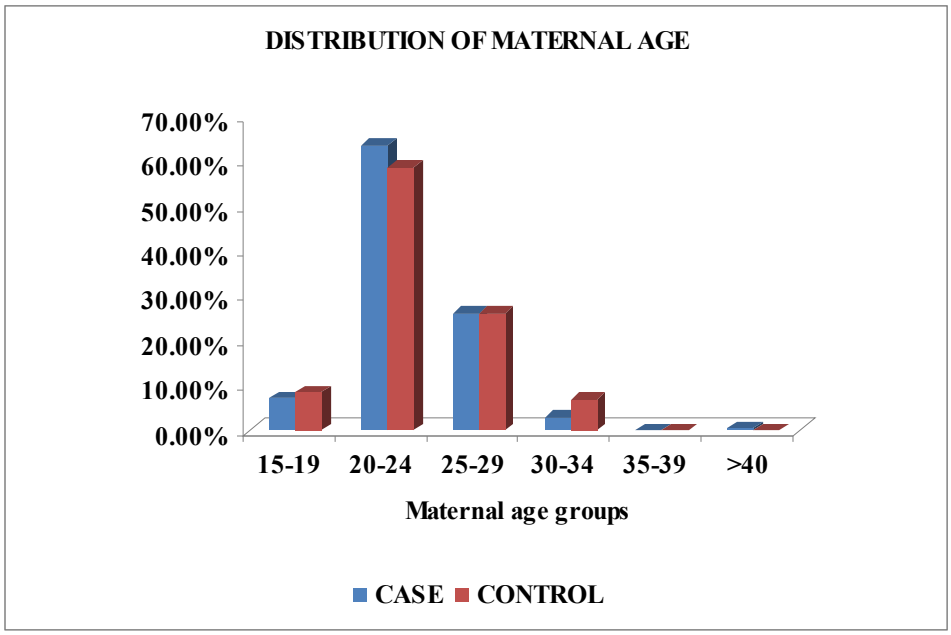
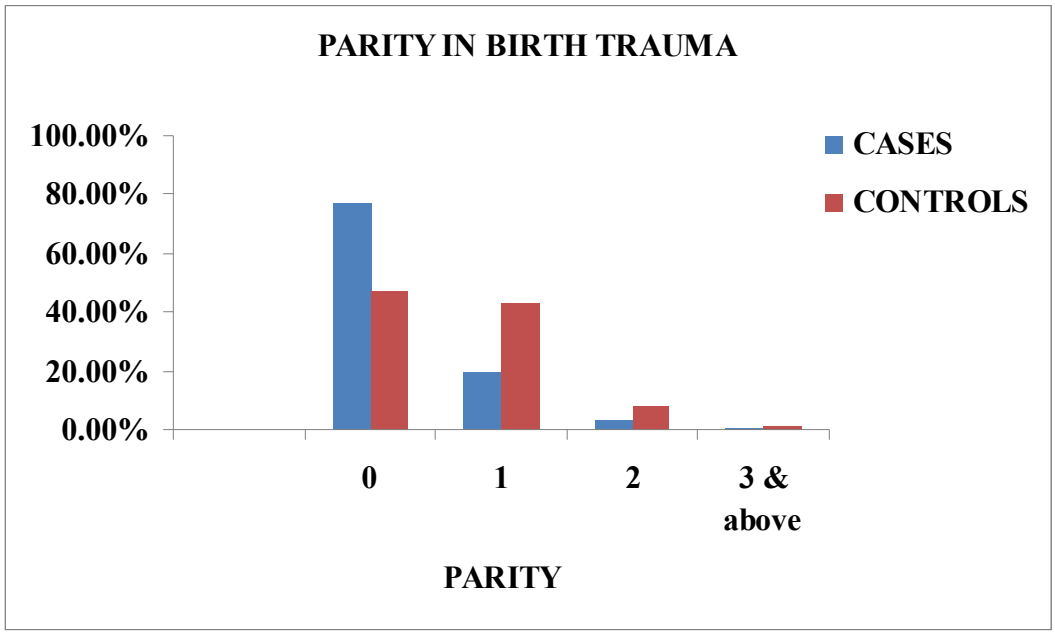


Table IV. Distribution of parity in birth trauma

Parity	Cases* n=283	Controls* n=366	P value

0	218(77.03)	173(47.27)	X^2=57.48 p=0.001 (S)
1	55(19.43)	158(43.17)	
2	9(3.18)	31(8.47)	
3 & above	1(0.35)	4(1.1)	

*Figures in parenthesis indicate percentage.



Maternal parity was 0 in 218(77.03%), 1 in 55(19.43%), 2 in 9(3.18%), 3 & above in 1(0.35%) in cases group. In control group parity was 0 in 173(47.27%), 1 in 158(43.17%), 2 in 31(8.47%), 3 & above in 4(1.1%) was seen. An increased rate of primiparity was present which is statistically significant (p=0.001).

Maternal weight does not have statistical significance. In our study 50-70 kg was the weight commonly observed both in cases [228(80.6%)] and controls [305(83.3%)].

Maternal short stature found in study group was 41(14%) compared to 4(1%) in control group which is statistically significant [p=0.001,Odds Ratio:15(5-51)].

13(5%) of mothers were unbooked in the study group compared to 2(0.5%) in control group [p=0.001, Odds Ratio:9(2-56)].

Most common obstetrical complications found in study group are CPD/Contracted pelvis 48(16.96%), Malpresentation 22(7.77%), PIH/PET/Eclampsia 12(4.24%), GDM in 8(2.83%), Multiple gestation & Oligohydramnios 5(1.77%) each. In control group PIH/PET/Eclampsia 20(5.46%), CPD 18(4.92%), Malpresentation 9(2.46%), multiple gestation & GDM 3(0.82%), nil Oligohydramnios were found in decreasing order of frequency. Statistically significant increased rate of malpresentation (p=0.001), CPD/contracted pelvis (p=0.001), oligohydramnios (p=0.03) were found.

66(23%) of mothers in study group referred late from the peripheral health institutions compared to just 6(1.6%) in control group which is statistically significant (p=0.001).

Table V. Maternal factors in birth trauma

Maternal factor	Group	Cases	Controls	p value

Weight	<50 kg	10(3.6%)	19(5.2%)	X^2=3.46 p=0.18(NS)
	50-70 kg	228(80.6%)	305(83.3%)	
	>70 kg	45(15.9%)	42(11.5%)	
Height	<145 cm	41(14%)	4(1%)	X^2=44.37 p=0.001(S) OR:15(5-51)
	>145 cm	242(86%)	362(99%)	
Antenatal care	Booked	270(95%)	364(99.5%)	X^2=11.58 p=0.001(S) OR:9(2-56)
	Unbooked	13(5%)	2(0.5%)	
Obstetrical complications	Multiple gestation	5(1.77%)	3(0.82%)	Z=0.73 p=0.46(NS)
	Malpresentation	22(7.77%)	9(2.46%)	Z=3.25 p=0.001(S)
	CPD/contracted pelvis	48(16.96%)	18(4.92%)	Z=5.03 p=0.001(S)
	Oligohydramnios	5(1.77%)	0	Z=2.10 p=0.003(S)
	GDM	8(2.83%)	3(0.82%)	Z=1.96 p=0.005(S)
	PIH/PET/Eclampsia	12(4.24%)	20(5.46%)	Z=0.71 p=0.47(NS)
Obstetrical complications	Present	100(35.3%)	53(14.5%)	Z=6.2 p=0.001(S)
Late referral	Yes	66(23%)	6(1.6%)	Z=8.72 p=0.001(S)

Table VI. Perinatal factors in birth trauma

Perinatal factors		**Cases**	**Controls**	**P value**

Presentation	1.Vertex	257(90.81%)	357(97.54%)	Z=3.76 p=0.001(S)
	2.Breech	23(8.13%)	9(2.46%)	Z=3.30 p=0.001(S)
	3.Face	3(1.06%)	0	Z=1.96 p=0.005(S)
	4.Brow	0	0	
Oxytocin use	Yes	181(63.96%)	35(9.56%)	X^2=212.6 p=0.001 (S) OR:16(11-26)
	No	102(36.04%)	331(90.44%)	
Duration of labor	1.Normal	209(73.85%)	365(99.73%)	X^2=104.5 p=0.001(S) OR:124 (19-250)
	2.Obstruted labor	74(26.15%)	1(0.27%)	
	3.Precipitate labor	0	0	
Shoulder dystocia	Yes	19(6.71%)	0	X^2=25.31 p=0.001(S)
	No	264(93.64%)	366(100%)	

Commonest presentation in both study group [257(90.81%] and control group [357(97.54%) were vertex presentation. But increased incidence of malpresentation found in study group [breech 23(8.13%) & face 3(1.06%)] compared to the control group [breech 9(2.46%) & nil face presentation] which is statistically significant (p=0.001).

The use of oxytocin was 181(63.96%) in study group, compared to the control group 35(9.56%) which is statistically significant [p=0.001,Odds Ratio16(11-26)].

The duration of labor was normal in 209(73.85%), 365(99.73%) in study and control group respectively. Obstructed labor was seen in 74(26.15%) in the study group compared to

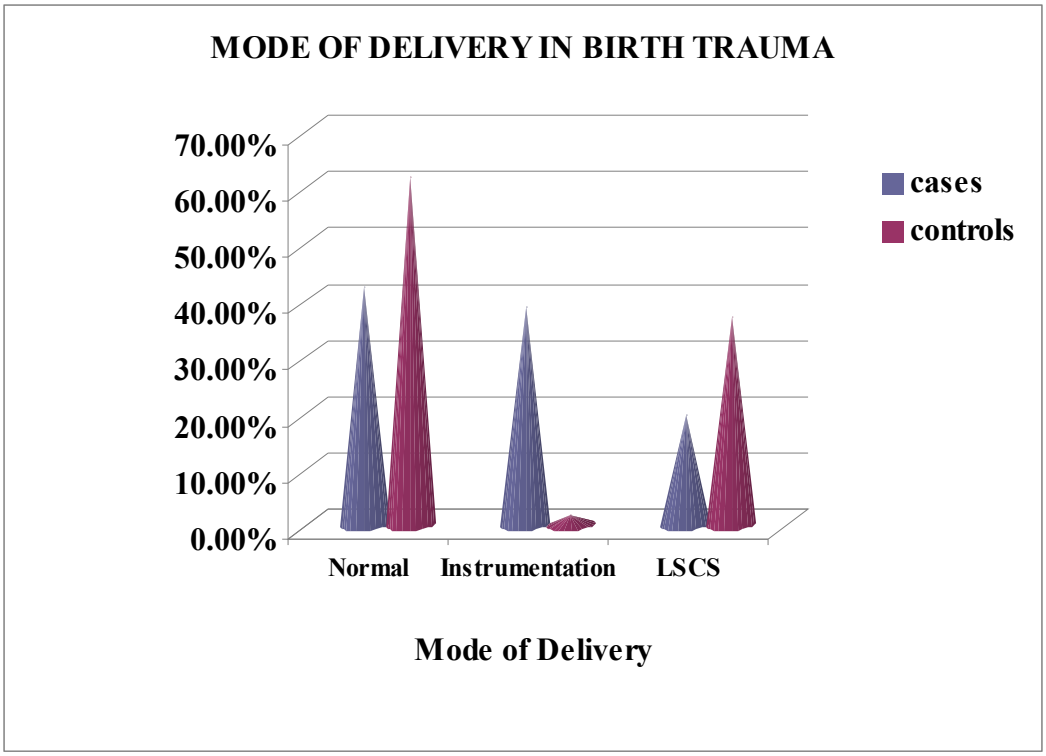
just 1(0.27%) in control group, statistically significant [p=0.001, Odds Ratio: 124(19-250)]. Precipitate labor was not found in both study and control group.

Shoulder dystocia was present in 19(6.71%) deliveries in the study group compared to nil in control group (p=0.001).

Table VII. Mode of delivery in birth trauma

Mode of delivery	Cases* n=283	Controls* n=366	P value
Normal	119(42.05)	226(61.75)	X^2=65.58 p=0.001(S)
LSCS	109(38.51)	134(36.61)	
Instrumentation	55(19.44)	6(1.64)	

*Figures in parenthesis indicate percentage.

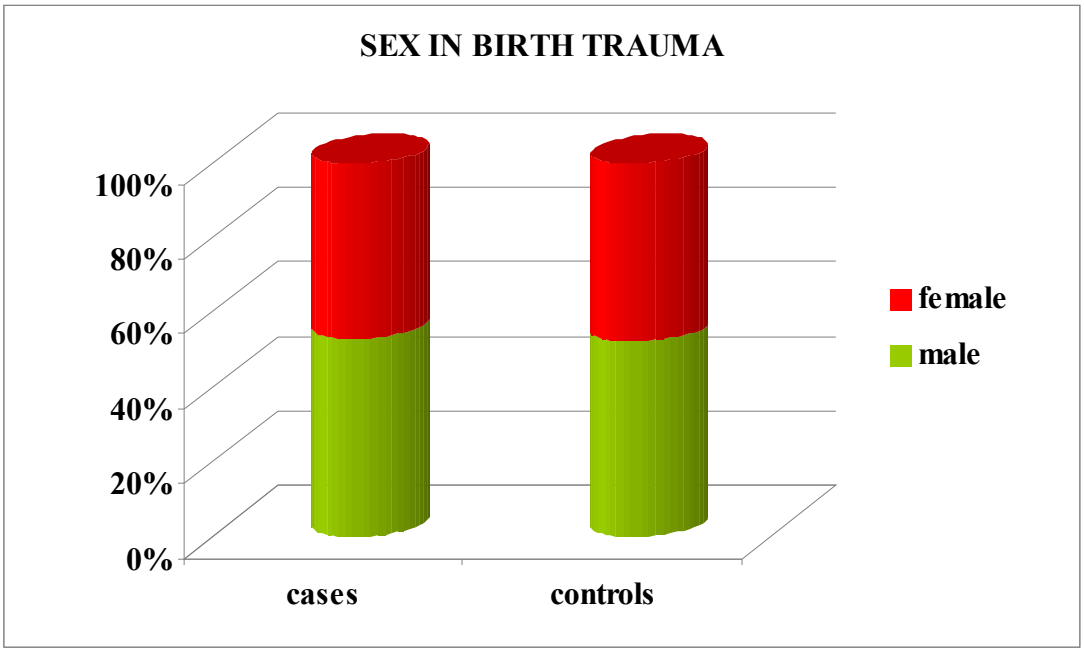


The mode of delivery in order of frequency was normal 119(42.05%0, LSCS 109(38.51%) and instrumentation 55(19.44%) in the study group. Similarly in the control group the mode of delivery was normal 226(61.75%), LSCS(36.61%) and instrumentation 6(1.64%). But statistically increased rate of instrumentation was seen in the study group (p=0.001).

Table VIII. Neonatal factors - sex in birth trauma

Sex	Cases* n=283	Controls* n=366	P value
Male	150(53%)	190(52%)	X^2=0.08 p=0.78(NS)
Female	133(47%)	176(48%)	

*Figures in parenthesis indicate percentage.



Male babies had a higher incidence of BT both in study group [150(53%)] and control group [190(52%)] compared to the female babies,133 (47%) in the study group,176(48%) in the control group, which is not statistically significant (p=0.78).

Term babies are common in both study group [270(95.41%)] and control group [337(92.08%)], compared to the preterm babies 13(4.59%) in the study group, 29(7.92%) in the control group. No post term babies were found in both study and control group. Maturity does not have statistical significance (p=0.09).

Table IX. Maturity in birth trauma

Maturity	Cases* n=283	Controls* n=366	P value
Term	270(95.41)	337(92.08)	X^2=2.92 p=0.09 (NS)
Preterm	13(4.59)	29(7.92)	
Post term	0	0	

*Figures in parenthesis indicate percentage.

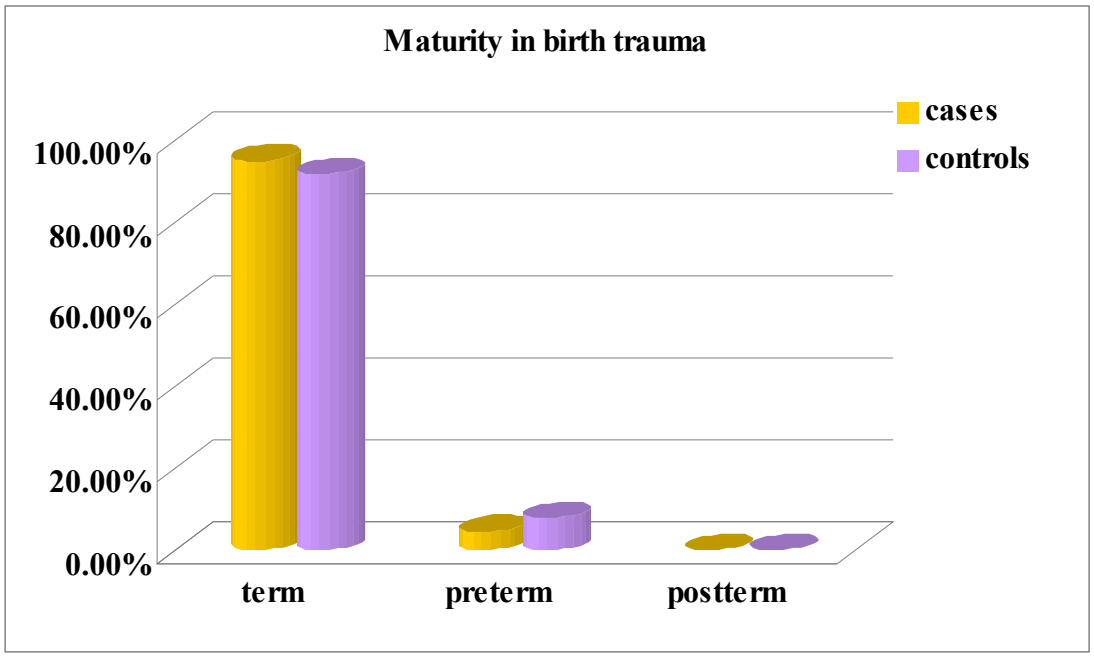
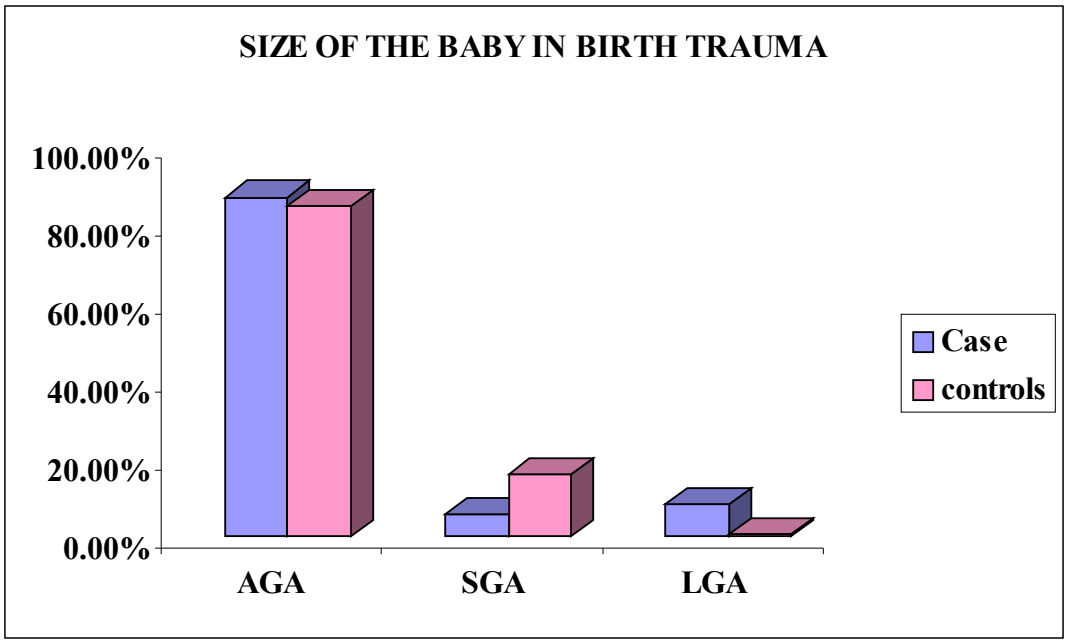


Table X. Size of the neonate in birth trauma

Size of the baby	Cases* n=283	Controls* n=366	P value
AGA	245(86.57)	308(84.51)	X^2=41.91 p=0.001(S)
SGA	15(5.3)	57(15.57)	
LGA	23(8.13)	1(0.28)	

*Figures in parenthesis indicate percentage.



In babies with BT 245(86.57%) were characterized as AGA; 23 (8.13%) as LGA; 15(5.3%) as SGA. In the control group the size of the baby was AGA 308(84.15%), SGA 57(15.57%) and LGA 1(0.28%). LGA babies were significantly increased in the study population [p = 0.001].

Table XI. Birth weight in birth trauma

Birth weight	Cases*(n=283)	Controls*(n=366)	p value
<2.5kg	28(9.9)	87(23.77)	X^2=151.5 p=0.001(S)
2.5-3.0 kg	55(19.43)	197(53.83)	
3.0 – 3.5 kg	139(49.12)	76(20.77)	
3.5 – 4.0 kg	49(17.31)	5(1.37)	
>4 kg	12(4.24)	1(0.27)	

*Figures in the parenthesis indicate percentage

The birth injuries were common in babies with a birth weight more than 3.5 kg. In the study group 49(17.31%) were between 3.5-4.0 kg, 12(4.24%) were more than 4 kg compared to control group 5(1.37%) were between 3.5-4 kg, 1(0.27%) is more than 4 kg. Birth weight distribution between groups was significant (p=0.001).

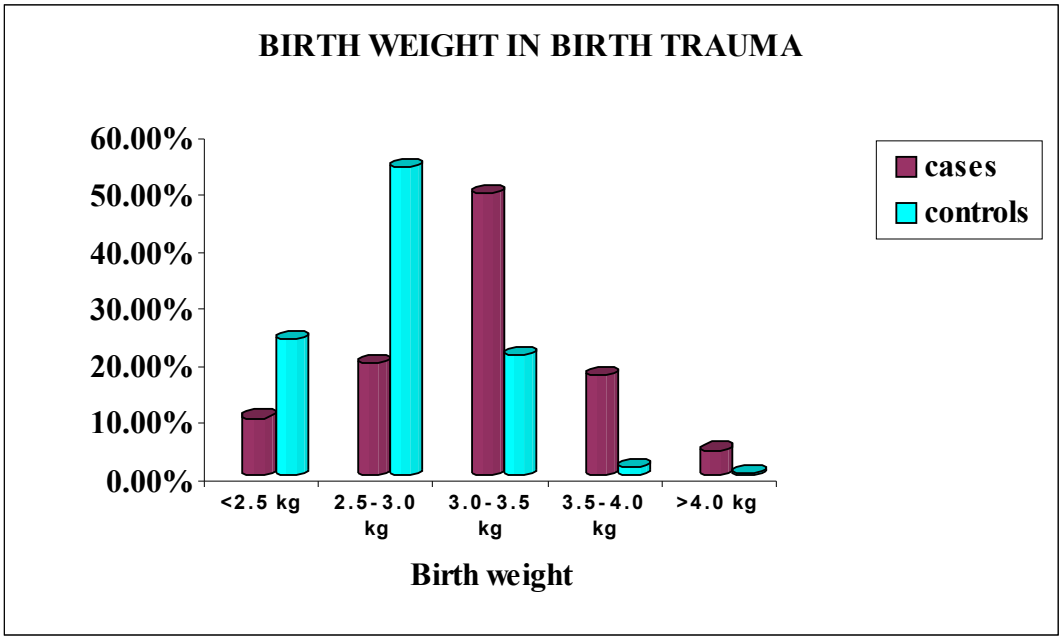


Table XII. Associated Asphyxia in birth trauma

	Group	Cases	Controls	P value
Apgar (at 5 minute)	<4	2(0.71%)	1(0.27%)	X^2=31.40 p=0.001(S)
	4-6	38(13.43%)	8(2.19%)	
Asphyxia		74(26.15%)	14(3.83%)	Z=8.23 p=0.001(S)
Resuscitation requirements	1.Routine care	141(49.82%)	348(95.08%)	X^2=178.68 p=0.001(S)
	2.+Initial resuscitation	68(24.03%)	4(1.1%)	
	3.+PPV	73(25.8%)	13(3.55%)	
	4.+Chest compressions	1(0.35%)	1(0.27%)	
	5.+Medications	0	0	

Apgar score at 5 minute 7 & above in 243 cases (85.86%), between 4 to 6 in 38 cases (13.43%), less than 4 in 2 cases (0.71%) in the study group. In the control group Apgar score 7 & above in 357 (97.54%), between 4 to 6 in 8(2.19%), less than 4 in 1(0.27%). Distribution between groups was significantly significant (p=0.001).

Birth asphyxia [Apgar <7 at 1 minute] were found in 74(26.15%) babies in the study group compared to 14(3.83%) in the control group which is statistically significant (p=0.001).

Neonatal resuscitation requirement, in the study group 73(25.8%) required positive pressure ventilation, 1(0.35%) required chest compressions compared to 13(3.55%) required positive pressure ventilation, 1(0.27%) required chest compressions control group which is statistically significant (P=0.001).

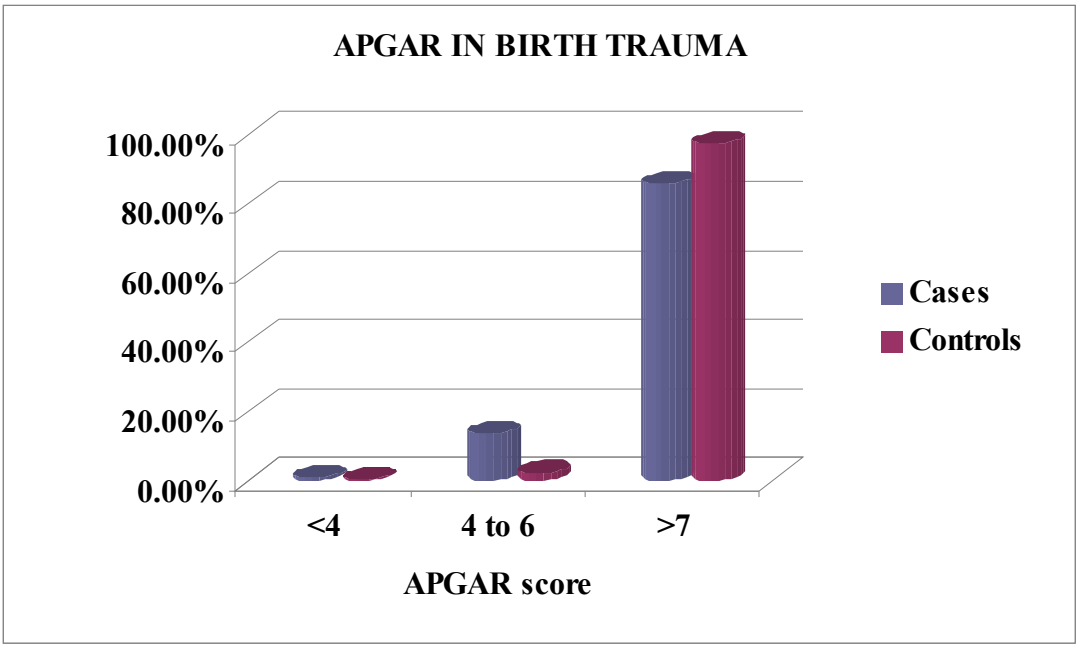


Table XIII. Mode of Treatment in birth trauma

Mode of treatment	N	%
1.Conservative	272	96%
2.Minor surgical/ortho intervention	11	4%
3.Major surgical/ortho intervention	0	0
Total	**283**	**100%**

Out of 283 babies with BT, 272(96%) were treated conservatively i.e reassurance or physiotherapy. 11(4%) cases required minor surgical or ortho intervention like suturing and closed fracture reduction and splinting. No major surgical or orthopedic intervention done to any of the babies.

Table XIV. Morbidity & mortality in birth trauma

	n	%

Morbidity at discharge	101	35.69%
Mortality	Nil	0
Normal at discharge	182	64.31%
Total	283	100%

Morbidity in this study was 101 (35.69%) cases where complete recovery not found. BT was not the direct cause of 241 neonatal deaths during the study period though 1 baby with fracture femur expired on 3rd day of life due to congenital heart disease. The remaining 182(64.31%) cases either completely recovered at discharge or complications are not expected after discharge.

In our study the predisposing factors for mechanical birth trauma were primiparity (p=0.001), short maternal stature (p=0.001), unbooked mother (p=0.001), antenatal obstetrical complications like malpresentation (p=0.001), CPD (p=0.001), oligohydramnios (p=0.03), late referrals from PHI (p=0.001), breech presentation (p=0.001), oxytocin use (p=0.001), obstructed labor (p=0.001), shoulder dystocia (p=0.001), instrumental delivery (p=0.001), size of the baby (p=0.001), birth weight more than 3.5 kg (p=0.001).

DISCUSSION

Incidence:

The incidence of BT in this study was 22.22 per 1000 live births.

Studies	Country	Study duration	Total births	Incidence per 1000 live births
Ali et al[6] 1981-84	Trinidad & Tobago	3.5 years	21000	4.0
Awari et al [11] 1986-96	Saudi Arabia	11 years	31028	6.70
Barrientos et al[12] 1993-98	Spain	5 years	21375	14.50
Mosavat et al [51] 2004-05	Iran	1 year	3340	8.0
Guha et al[28] 1968-70	India	2 years	12000	6.83
Padmini et al[55] 1980-85	India	5 years	16917	7.30
Present study 2007-08	India	1 year	12735	22.22

The different incidences noted in various studies may be due to the different inclusion criteria followed. The higher incidence found in our study is due to the fact that study place is a referral institution, where more number of high risk cases are delivered.

Excluding subconjuctival haemorrhage as done by many studies the incidence of BT in our study was 14.13 per 1000 live births which is comparable with study done by Barrientos et al[12].

Head & neck injuries were commonest with an incidence of 19.47 per 1000 live births in contrast Antony Hughes et al[8] 1991-97 reported an incidence of 9.5 per 1000 live births in his study of BT in the head and neck. If subconjuctival haemorrhage excluded as done by Antony Hughes et al[8] incidence of BT to head and neck was 11.78 per 1000 live births which is marginally higher.

The Nerve injuries had an incidence of 1.1 per 1000 live births in our study.

Studies	Incidence per 1000 live births
Awari et al[11] 1986-96	1.61
Barrientos et al[12] 1993-98	2.34
Guha et al[28] 1968-70	2.25
Padmini et al[55] 1980-85	1.12
Vishnu bhat et al[69] 1983-92	1.81
Present study 2007-08	1.10

The incidence of nerve injuries in our study is comparable with study done by Padmini et al[55], in contrast other studies were marginally higher than the present study (1.61 to 2.34 per 1000 live births).

The incidence of Bone injuries in this study was 0.3 per 1000 live births.

Studies	Incidence per 1000 live births
Ibrahim et al[34] 1986-96	0.67
Awari et al[11] 1986-96	0.64
Adetokunbo et al[3] 2005-06	0.41
Guha et al[28] 1968-70	0.67
Padmini et al[55] 1980-85	1.12
Vishnu bhat et al[70] 1983-93	1.00
Present study 2007-08	0.30

The bony injuries when compared with other studies, higher than the present study. The present study was showing 2 to 3 times reduction in the incidence of bony injuries than the older studies, may be due to the improved obstetrical care and selecting LSCS as mode of

delivery for high risk cases.

The incidence of Skin and soft tissue injuries in this study is 0.13%.

Studies	Incidence
Barrientos et al[12] 1993-98	0.04%
Adetokunbo et al[3] 2005-06	0.32%
Guha et al[28]1968-70	0.06%
Present study 2007-08	0.13%

Both lower and higher incidence of soft tissue injuries were found when comparing to the present study (0.04% to 0.32%)

No case of intra abdominal injuries were recorded in this study in comparable with studies done by Padmini et al[55], in contrast 0.09 to 0.33 per 1000 live births, intra abdominal injuries were found in other studies.

Studies	Incidence per 1000 live births
Barrientos et al[12] 1993-98	0.09
Adetokunbo et al[3] 2005-06	0.13
Guha et al[28] 1968-70	0.33
Padmini et al[55] 1980-85	0
Present study 2007-08	0

The incidence of subconjuctival haemorrhage 0.84% in our study in contrast Adetokunbo et al[3] reported an incidence of 0.27% of subconjuctival haemorrhage.

The incidence of Cephalhaematoma in our study was 0.57%. Other studies reported an

incidence ranging from 0.12% to 2.5%.

Studies	Incidence
Thacker KE et al[67] 1977-87	2.5%
Antony Hughes et al[8] 1991-97	0.5%
Barrientos et al[12] 1993-98	0.49%
Mosavat et al[51] 2004-05	0.3%
Adetokunbo et al[3] 2005-06	0.14%
Guha et al[28] 1968-70	0.12%
Present study 2007-08	0.57%

The incidence of skin and soft tissue injuries in the head and neck in this study was 0.49%. Antony Hughes et al[8] 1991-97 reported an incidence of 0.1% in his study.

The incidence of subgaleal haemorrhage was 0.47 per 1000 live births in this study. Ng et al[53] 1990-93 reported an incidence of 0.8 per 1000 live births. The reason for low incidence may be the drastic reduction of vacuum deliveries, just 5(1.6%) out of 321 instrumental deliveries.

The incidence of brachial palsy in this study was 0.8 per 1000 live births. This was comparable to the study by Perlow JH et al[57]. Other studies incidence ranges from 0.27 to 2.6 per 1000 live births.

Studies	Incidence per 1000 live births
Levine et al[42] 1974-77 & 1979-81	2.6

Perlow JH et al[57] 1985-90	0.9
Antony Hughes et al[8] 1991-97	0.5
Barrientos et al[12] 1993-98	1.2
Adetokunbo et al[3] 2005-06	0.27
Guha et al[28] 1968-70	0.6
Vishnu bhat et al[69] 1983-92	1.0
Present study 2007-08	0.8

The incidence of facial nerve palsy found in our study was 0.31 per 1000 live births. In contrast to this other studies report higher incidence of facial nerve palsy 0.6 to 8 per 1000 live births.

Studies	**Incidence per 1000 live births**
Levine et al[42] 1974-77 & 1979-81	7.5
Perlow JH et al[57] 1985-90	0.6
Antony Hughes et al[8] 1991-97	8.0
Barrientos et al[12] 1993-98	1.2
Guha et al[28] 1968-70	1.5
Vishnu bhat et al[69] 1983-92	0.74
Present study 2007-08	0.31

Fracture femur incidence in our study was 0.16 per 1000 live births, which is comparable to the other studies.

Studies	**Incidence per 1000 live births**
Ibrahim et al[34] 1986-96	0.19
Morris et al[50] 2002	0.13
Vishnu Bhat et al[70] 1983-93	0.14
Present study 2007-08	0.16

The incidence of fracture humerus in our study was 0.08 per 1000 live births, which is comparable to the study done by Ibrahim et al[34], 0.1 per 1000 live births.

Studies	Incidence per 1000 live births
Ibrahim et al[34] 1986-96	0.10
Guha et al[26] 1968-70	0.17
Vishnu bhat et al[70] 1983-93	0.20
Present study 2007-08	0.08

The incidence of clavicle fracture in our study was 0.08 per 1000 live births, in contrast to other studies which report higher incidence, 0.46 to 2 per 1000 live births.

Studies	Incidence per 1000 live births
Levine et al[42] 1974-77 & 1979-81	2.0
Barrientos et al[12] 1993-98	0.5
Guha et al[28] 1968-70	0.5
Vishnu bhat et al[70] 1983-93	0.46
Present study 2007-08	0.08

Distribution of maternal age:

The maximum birth injuries occurred in the maternal age group of 20-24 years, 63.6% in our study, in contrast to Adetokunbo et al[3] who reported 54.5% of injuries in 30 to 34 years age group.

Maternal age group	Adetokunbo et al[3] 2005-06	Present study 2007-08
15-19 yr	Nil	7.1%

20-24 yr	9.09%	63.6%
25-29 yr	18.18%	26.1%
30-34 yr	54.5%	2.8%
35-39 yr	18.8%	0
>40 yr	2.27%	0.4%

Parity:

Nulliparity is the commonest parity in the study group in our study, which is comparable to other studies even though each group differs.

Parity	**Antony Hughes et al[8] 1991-97**	**Adetokunbo et al[3] 2005-06**	**Padmini et al[55] 1980-85**	**Present study 2007-08**
0	51.8%	50%	48.8%	77.03%
1	32.9%	22.7%	22.0%	19.43%
2	10.4%	13.6%	13.8%	3.18%
3 & above	4.3%	13.6%	15.4%	0.35%

Obstetric risk factors were found in 32.5% in our study, which is similar to the study done by Padmini et al[55], 24.4%.

95% of the mothers in study group were booked in our study, in contrast to 34.3% in Vishnu bhat et al[70].

Presentation:

Breech presentation was found in 8.13% of cases in our study, in contrast to Vishnu bhat et al[70], recorded 20% of breech presentation.

Presentation	Vishnu Bhat et al[70] 1983-93	Present study 2007-08
Vertex	62.9%	90.81%
Breech	20%	8.13%
Others	17.1%	1.06%

Duration of labor:

Obstructed labor seen in 26.15% in our study, in contrast to 17% seen in study done by Antony Hughes et al[8].

Duration of labor	Antony Hughes et al[8] 1991-97	Present study 2007-08
Normal	83%	73.85%
Obstructed labor	17%	26.15%
Precipitate labor	0	0

Mode of delivery:

Normal delivery was the maximum mode of delivery in our study, which is comparable to Awari et al[11] & Padmini et al[55]; in contrast Antony Hughes et al[8] reported instrumentation as the predominant mode of delivery in the study group.

Mode of delivery	Awari et al[11] 1986-96	Antony Hughes et al[8] 1991-97	Adetokunbo et al[3] 2005-06	Padmini et al[55] 1980-85	Present study 2007-08
Normal	55%	36.6%	22.61%	46%	42.05%
LSCS	5.2%	17%	50%	28.5%	38.51%

Instrumentation	39.8%	46.4%	27.2%	34.1%	19.44%

Sex:

Male babies are commonly affected in our study which is comparable to studies by Padmini et al[55] and Vishnu Bhat et al[70].

Sex	**Padmini et al[55] 1980-85**	**Vishnu Bhat et al[70] 1983-93**	**Present study 2007-08**
Male	60.2%	60%	53%
Female	39.8%	40%	47%

Maturity:

Term babies are commonly affected in our study, comparable with studies by Vishnu bhat et al[69] and Padmini et al[55].

Maturity	Padmini et al[55] 1980-85	Vishnu bhat et al[69] 1983-92	Present study 2007-08
Term	87.8%	93.2%	95.41%
Preterm	8.9%	6.8%	4.59%
Post term	3.3%	0	0

Size of the baby:

LGA in the study group found in our study was 8.13% in contrast to Antony Hughes et al[8] reported 18.9% of LGA babies.

Size of the baby	Antony Hughes et al[8] 1991-97	Present study 2007-08
AGA	78.1%	86.57%
SGA	3.0%	5.3%
LGA	18.9%	8.13%

Birth weight:

The birth injuries were common among babies with birth weight more than 3.5 kg in our study which is comparable to study by Adetokunbo et al[3] in contrast to western reports where birth injuries common in more than 4 kg babies.

Birth weight	Adetokunbo et al[3] 2005-06	Present study 2007-08
<2.5 kg	0	9.9%
2.5 – 3 kg	4.54%	19.43%
3-3.5 kg	45.54%	49.12%
3.5-4 kg	31.8%	17.31%
>4 kg	18.18%	4.24%

Apgar & Asphyxia:

Apgar score at 5 minutes less than 7 in 14.14% in our study comparable to study by Antony Hughes et al[8] who reported 12.8%.

Birth asphyxia (Apgar <7 at 1 minute) in our study was 26.15% in contrast Vishnu Bhat et al[70] reported 54.3%.

Type of medical care:

Conservative treatment is the predominant modality of care in our study in contrast minor ortho or surgical intervention is the predominant modality care in the study by Adetokunbo et al[3].

Care received	Adetokunbo et al[3] 2005-06	Present study 2007-08
Conservative	41%	96%
Minor ortho/surgical intervention	59%	4%
Major ortho/surgical intervention	Nil	Nil

Even though incidence of BT high in our study, when compared with other studies, there is actually reduction in mortality and major injuries like nerve, bone, intra abdominal and intra cranial injuries, just 30(10.4%) of 288 injuries, all these may be due to the improvement in obstetrical care.

CONCLUSION

1. The incidence of mechanical birth trauma in our study was 22.22 per 1000 live births.

2. Head & Neck injuries are the commonest type of birth trauma.

3. Excluding Subconjuctival haemorrhage cephalhaematoma is the commonest injury to the head and neck.

4. Injury to brachial plexus by far the commonest nerve affected by BT.

5. Femur is the commonest bone injured during birth.

6. Primiparity most important risk factor.

7. Presence of maternal obstetric risk factors increases the likelihood of BT.

8. Early referral from peripheral health instituitions (PHI) may decrease BT.

9. Malpresentation increases the incidence of BT even in skilled and competent hands.

10. Uterine stimulants predisposes to BT.

11. Prolonged and obstructed labor is another risk factor.

12. Possibility of shoulder dystocia should be detected earlier and appropriate intervention must be done to avoid BT.

13. Instrumental delivery especially midforceps and vacuum extraction predispose to

mechanical injuries.

14. LGA and birth weight more than 3.5 kg important risk factors.

15. The association of birth asphyxia is high in babies with mechanical injuries.

16. Most of the injuries are self limiting.

17. Appropriate treatment should be started earlier if indicated to avoid sequlae.

18. Early identification of the risk factors and appropriate timely intervention will be rewarding.

LIMITATIONS

- Our study conducted at tertiary care hospital, which may not reflect the incidence at primary and secondary care level, where majority deliveries are conducted. The incidence may be either high in this institution, since high risk cases are delivered or may be low since competent and skilled care was available.

- Autopsies were not performed for the neonatal deaths.

- Imaging modalities like CT scan and MRI scan, which is more sensitive for CNS imaging, were not freely available.

- Certain birth injuries may be missed, because few injuries may not be clinically evident at birth, later medical attention can be seeked in other instituition.

RECOMMENDATIONS

- Adequate antenatal care must be provided to identify high risk babies and refer them to the appropriate institutions for expert care.
- Good intra partum care is essential to reduce the incidence of birth injuries.
- Avoidance of midforceps and vacuum extraction is necessary to decrease the incidence of BT.
- If instrumental delivery is indicated, the same should be performed under the guidance of the most experienced personnel.
- Since the incidence of BT not reduced significantly in spite of refinements in obstetric care, a special approach must be formulated, for which more studies are required in future.

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