

Faculty of Medicine

University of Dhaka

ANTENATAL AND INTRAPARTUM RISK FACTORS OF CHILDREN WITH CEREBRAL PALSY

By

Md. Obaidul Haque

Master of Science in Physiotherapy

Session: 2012-2013

Registration No: 88

Roll No: 206



Department of Physiotherapy

Bangladesh Health Professions Institute (BHPI)

May 2016



Faculty of Medicine

University of Dhaka

ANTENATAL AND INTRAPARTUM RISK FACTORS OF CHILDREN WITH CEREBRAL PALSY

By

Md. Obaidul Haque

Master of Science in Physiotherapy

Session: 2012-2013

Registration No: 88

Roll No: 206

Submitted in Partial Fulfillment of the requirements for the Degree of Master

of Science in Physiotherapy



Department of Physiotherapy

Bangladesh Health Professions Institute (BHPI)

May 2016

We the undersigned certify that we have carefully read and recommended to the Faculty of Medicine, University of Dhaka, for acceptance of this thesis entitled, **"Antenatal and Intrapartum Risk Factors of Children with Cerebral Palsy",** submitted by Md. Obaidul Haque, for the partial fulfillment of the requirements for the degree of Master of Science in Physiotherapy.

Dr. Kamal Ahmed

Associate Professor

BHPI, CRP, Savar, Dhaka-1343.

Firoz Ahmed Mamin

Assistant Professor of Physiotherapy

BHPI, CRP, Savar, Dhaka-1343

Dr. Parviz Shahidi

Professor and Head, Department of Orthopedics

Shaheed Monsur Ali Medical College

Uttara Model Town Sector-11, Uttara, Dhaka- 1230

Nasirul Islam

Associate Professor and Principal (Acting) BHPI,CRP, Savar, Dhaka-1343

Date of approval: July 02, 2016

Declaration Form

- This work has not previously been accepted in substance for any degree and is not concurrently submitted in candidate for any degree
- This dissertation is being submitted in partial fulfillment of the requirements for the degree of Master of Science in Physiotherapy.
- This dissertation is the result of my own independent work/investigation, except where otherwise stated. Other sources are acknowledged by giving explicit references. A Bibliography is appended.
- I confirm that if anything identified in my work that I have done plagiarism or any form of cheating that will directly awarded me fail and I am subject to disciplinary actions of authority.
- I confirm that the electronic copy is identical to the bound copy of the Thesis.
- In case of dissemination the finding of this project for the future publication, research supervisor will highly concern and it will be duly acknowledged as graduate thesis.

Signature:
Name: Md.Obaidul Haque
Date:

Acknowledgement

At first, I commit to memory the almighty Allah for giving me ability to conclude the study. I would like to gratitude to my honorable supervisor **Dr. Kamal Ahmed** for his unremarkable guidelines and for not only assisting me but also giving me courage in completion of the study. I am grateful to the Chairman and members of the thesis defense committee for kindly accepting the topic and giving me the opportunity to conduct the study. I am especially indebted with the study participants for giving me valuable time. Very struggle and challenging phenomena of the participants have been stretched me and shown me to be empathetic. I must salute them. I would like to thank to Firoz Ahmed Mamin, Assistant Professor & Course coordinator, M.Sc in Physiotherapy program, Department of Physiotherapy, BHPI. I would like to pay my special thanks & gratitude to Ehsanur Rahman, Assistant Professor, Department of Physiotherapy, for his remarkable help and support to accomplish the study. I would like to gratitude to Md. Fazlul Karim Patwary, Associate Professor, Jahangir Nagar University, for his special contribution in data analysis. My special gratitude to the staffs of the BHPI library for their cordial help to find out the books and for collecting literature of the study. I would like to confer my thanks to Mohammad Habibur Rahman, Assistant Professor, department of Physiotherapy, BHPI for his incredible suggestions and support through the study. I specially thank to all Clinical Physiotherapist Paediatric Unit and all the intern physiotherapists of Paediatric Unit for their inconceivable support.

Finally I would like to thanks all of my friends and colleagues who always inspired and encouraged me and individual who are directly or indirectly involve with this study.

Contents

Торіс	Page No.
List of Tables	i
List of figures	ii
List of Abbreviations	iii
Abstract	iv-v
CHAPTER-I: INTRODUCTION	1-10
1.1 Background	1-4
1.2 Justification of the study	5-6
1.3 Operational definition	7
1.4 Research question	8
1.5 Objectives	9
1.6 List of Variables	10
CHAPTER-II: LITERATURE REVIEW	11-33
CHAPTER -III: METHODOLOGY	34-39
3.1 Study design	34
3.2 Study site	35
3.3 Study duration	35
3.4 Study population & sample population	35-36
3.5 Inclusion criteria	36
3.6 Exclusion criteria	37
3.7 Sampling technique	37
3.8 Data collection Procedure	37
3.9 Data analysis	38
3.10 Ethical consideration	39
CHAPTER-IV:RESULTS	40-48
CHAPTER-V:DISCUSSION	49-53
5.1 Limitations	54
CHAPTER-VI: CONCLUSION AND	55
RECOMMENDATION	
6.1 Conclusion	55

6.2 Recommendation	55
REFERENCES	56-68
APPENDIX	i-xviii
Inform consent (English)	vii
Questionnaire (English)	viii-xii
Inform consent (Bangla)	xiii
Questionnaire (Bangla)	xiv-xvii
Permission Letter	xviii
Ethical Approval letter of IRB	xix

List of Tables

Table	Торіс	Page no.
Table 1	Characteristics of children age and gender ratio	40
Table 2	Shows age of mother and their educational status	42
Table 3	Risk factor of antenatal and Intrapartum factor	44-45

List of Figures

Figure	Торіс	Page no.
Figure - 1	Mother age during child birth	41
Figure - 2	Place of Child birth	41
Figure - 3	Type of Physical problem	43

List of Abbreviations

AAP	: American Academy of Paediatrics
ACOG	: American College of Obstetricians and Gynecologists
BHPI	: Bangladesh Health Professions Institute
BMRC	: Bangladesh Medical and Research Council
СР	: Cerebral Palsy
CRP	: Centre for the Rehabilitation of the Paralysed
IFB	: Impact Foundation Bangladesh
IRB	: Institutional Review Board
OR	: Odds Ratio
SPSS	: Statistical Package for the Social Science
WHO	: World Health Organization

Abstract

Purpose: The aim of the study was to identify common antenatal and intrapartum risk factors among the Children with Cerebral Palsy (CP). Objectives: To identify and analyse possible antenatal and intrapartum risk factors affecting the development of cerebral palsy. Methodology: Antenatal and intrapartum events were compared between 25 children with CP case and 25 control in a retrospective case-control method. Antenatal and intrapartum factors were expressed as odds ratios, 95% confidence intervals and chi-square test. Factors associated with an increased risk of CP identified as antenatal and intrapartum risk factors were: maternal eclampsia, maternal hypertension, maternal diabetes, physical problem (e.g. fall down), maternal viral diseases, low birth weight(< 2500 gm), delayed crying, birth asphyxia, neonatal seizures were associated with an increased risk of CP in the neonatal period. Results: Data was analyzed by using SPSS version 20 and odds ratio was used. Microsoft Excel Work sheet 2013 and had a two group case and control. A total of 50 participants with cerebral palsy minimum age group of mother were 18 years and maximum age was 35 years. Among case the mean age of the participants was 26.5 years and boy ratio 54% and girl ratio 46%. Highest mother education were primary 46% (n=23). The frequency of child born in area there are 42% (n=22) home, 22% (n=11) born hospital, 36% (n=18) born clinic and their physical problem 22.0% (n=11) fall down, 16% (n=8) weight lifting, 10% (n=5) traumatic, no related cause 52% (n=26). The factors significantly associated with the cerebral palsy were antenatal care (OR- 1.833; 95% CI, 0.387-8.674, chi-0.22,P>0.05), Maternal Eclampsia (OR 1.313; 95% CI, 0.308-5.598, chi-0.13, P >0.05), Medicine taken during pregnancy (OR 3.188; 95% CI, 0.99-10.17, p<0.05) cerebral palsy which is statistically significant, Physical problem (OR 5.76; 95% CI, 1.36- 24.36, chi-6.34,

p<0.05), cerebral palsy which is statistically significant, maternal hypertension (OR-11.15; 95% CI 2.86 to 43.46, p<0.05) cerebral palsy which is statistically significant. Maternal diabetes (OR 13.5; 95% CI- 3.55- 51.22, p<0.05) statistically significant. Birth weight (OR 46.00; 95% CI-8.02-263.62, p<0.05) cerebral palsy which is statistically significant. Delayed crying (OR .80; 95% CI-0.016-0.416; Chi-11.52 p<0.05), Birth asphyxia (Chi-10.976, p<0.05), Neonatal seizure (Chi-21.42; p<0.05) cause of cerebral palsy which is statistically significant. Illness or infection of child after birth (OR 3.778; 95% CI- 1.170 -12.194; Chi-5.128; p<0.05) which means 3.778 higher risk factor for cerebral palsy. *Conclusion:* Study demonstrated that perinatal asphyxia, child birth weight, delayed crying and any pathology during pregnancy were independent factors associated with CP in term newborns.

Keywords: Cerebral Palsy, Intrapartum, Antenatal care.

1.1 Background

Cerebral Palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour, by epilepsy, and by secondary musculoskeletal problems (Rosenbaum, et al., 2007). The term Cerebral palsy refers to any one of a number of neurological disorders that appear in infancy or early childhood and permanently affect body movement and muscle coordination but don't worsen over time (Keynes, 2006). The most recent consensus definition states that cerebral palsy is an "umbrella term covering a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development" (Wolraich, et al., 2008).

Majnemeer and Mazer (2004), reported it is the most common and chronic form of early disability that begins in childhood with a prevalence of 2 per1000. Another study showed that the prevalence of cerebral palsy is 1.2 - 2.5 per 1000 live births although, the rates vary from country to country and also within the countries (Wolraich, et al., 2008). In United States, there are living almost 800,000 children in with one or more of the symptoms of cerebral palsy estimated the Foundation of the United Cerebral Palsy (UCP). Every year about 10,000 children born in the United States had developed cerebral palsy according to the federal government's (Elkamil, et al., 2011). In New Zealand approximately 7000 children are affected by some degree of Cerebral palsy. Cerebral palsy has a prevalence of 2 to 2.5 per 1000 live births and affects males and females in equal numbers (Damiano, 2004). In the Norwegian counties there were 494 children with CP born between 1st January 1996 and 31st December 2003, corresponding to a prevalence of 2.65 per 1000 live births (Elkamil, et al., 2011). In developed countries, International assessments propose that CP affects between 1.2 to 3.0 per 1000 children (Hustad, et al., 2011). In developing countries the incidence of CP children is 2 per 1000 children (Serdaroglu, et al., 2006). The incidence of CP is considered to be 2 to 2.5 in 1000 live births and the prevalence of CP in the developing countries tends to be in a similar range (Bialik and Givon, 2009). In one study found that prevalence of cerebral palsy in Bangladesh was 6.1/1000 children (Tabib, 2009). Bangladesh has recently seen an increase in the number of children diagnosed with cerebral palsy. Most of the population are illiterate and not be aware about health (Ackerman, et al., 2005). Cerebral palsy (CP) is now familiar to most health and social service professionals, as well as to many members of the general public, as a physically disabling condition. In fact, although CP only affects between 2 and 3 per 1000 live births, it is thought to be the most common cause of serious physical disability in childhood (Morris, 2007).

According to disability profile, the client assess in the Shishu Bikash Clinic (Rural Centre) during January to December 1998 showed a report of child disability were 42% of total disability was cerebral palsy, among these Spastic cerebral palsy is 9%, Athetoid cerebral palsy is 2%, Ataxic cerebral palsy is 3% and rest of the patient is other type of cerebral palsy (Khan & Rahman, 2000). Service for disabled children are meagre in relation to their needs. A large number of children with cerebral palsy need better physiotherapy treatment for better survival in the community. Cerebral

palsy cannot be cured but treatment can improved child capability. The earlier treatment can be made more improvement of the child with cerebral palsy. In realizing this truth some NGO's such as Centre for Rehabilitation of the Paralysed (CRP), Bangladesh Protibondhi Foundation (BPF), BRAC Inclusive Education Programme, Assistant for Blind Children (ABC), Impact Foundation Bangladesh (IFB), Shishu Bikash, Shishu Pally, Shishu Hospital, ICMH (Institute of Child and Mother Health) and also some other organization have taken step to provide physiotherapy service (Tanner & Harpham, 2013). Among these NGO's only CRP have an individual paediatric unit for the children with cerebral palsy which provide Physiotherapy, Occupation therapy and Speech and language therapy service.

Perinatal asphyxia has long been believed to be a major cause of CP. Advances in perinatal care have led to decreased mortality rates among newborns. However, recent epidemiologic assessments indicate that the incidence of CP is stable or increasing in some industrialized countries. The pathology of CP in term newborns is very different from preterm infants. Brain mal developments are seen in 16% of term and 2.5% of preterm infants with CP and gray matter lesions are more often seen in term (33%) than preterm (3.5%) CP infants. However periventricular white matter lesions occur significantly more often in preterm (90%) than in term (20%) infants (Krageloh Mann, 2008). Early brain injury in CP frequently results life-long disability, with serious adverse effects and implications for the child, family, and society (Bax, et al., 2007). In the absence of a known pathophysiological mechanism, only supportive care is provided; there is no evidence for the effectiveness of preventive strategies. Even if the pathology of neonatal encephalopathy is well-recognized, numerous questions remain regarding the causes and risk factors for CP in term infants differ from

premature infants (Andersen, et al., 2008) and in order to conduct preventive measures, it is necessary that the risk factors, etiology and the pathophysiology of the insult in this group be determined.

Gage's study stated that cerebral palsy is primarily characterized by central nervous system abnormalities, such as loss of selective motor control and abnormal muscle tone. As a result of growth these primary characteristics often lead to secondary deficits, including bony deformities, muscle contractures and gait abnormalities, and among all type of cerebral palsy spastic cerebral palsy is the most common type of cerebral palsy (Behrman, 2004). Andersen, et al. (2008) reported that risk factors for CP in term infants differ from premature infants and in order to conduct preventive measures, it is necessary that the risk factors, etiology and the pathophysiology of the insult in this group be determined. However, controversy exists regarding many of these risk factors. This study aims to determine the maternal and neonatal factors associated with term infants diagnosed with CP born in Savar, Bangladesh.

1.2 Justification of the study

The incidence of cerebral palsy worldwide is between 2 to 2.5 cases per 1,000 births (Marron, et al., 2013) and gives burden on parents both physically and psychologically. Cerebral palsy is a chronic condition that have serious consequences for physical, cognitive and behaviour functioning. Identifying risk factors for a disease is one of the methods used to gain understanding of its etiology. Identification of the risk factors of children with cerebral palsy will give us evidence by which we can take necessary measure to manage this condition as well as it can help to take preventive measures to minimize the sufferings of this condition. Since antenatal period maternal chronic diseases especially diabetes and cardiovascular diseases and intrapartum period perinatal asphyxia, prematurity and neonatal low birth weight are emerging which eventually make them at higher risk of developing cerebral palsy. By conducting this research it is expected that some of these risk factors can be identified to minimize the cost of treatment, mortality, morbidity, however physical and psychosocial distress. Much other Health professional will get update knowledge about factors which causing Cerebral Palsy and this knowledge also will benefit a large number of people.

Many studies have done about perception, quality of life, burden, physical and psychological stress, depression, efficacy of Proprioceptive Neuromuscular Facilitation (PNF) stretching program with cerebral palsy. But there is lack of researches about the risk factors of cerebral palsy patient in our country. This study also can be helpful in making Physiotherapist to aware about risk factors for developing cerebral palsy. So that Physiotherapist can provide better treatment as well as essential advice to the parents of cerebral palsy. As a health professional it improves our knowledge. Research makes the profession strongest. So there is no alternative option to do research as a professional to develop the profession.

After conclusion of this study, parents would become aware about the pregnant mother complications, infections, drugs, and importance of place of delivery. Eventually, other peoples as well as the members of the family would become aware about the necessity of obstetrics care in Bangladesh. So it was considered to find out the antenatal and intrapartum risk factors of children with cerebral palsy in Bangladesh.

1.3 Operational Definitions:

Cerebral Palsy: Cerebral Palsy is defined as a group of non-progressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in the early stages of its development. It occurred before birth, during birth or after birth at the age of 2 years of child age.

Perinatal: It is occurring during or pertaining to the phase surrounding at the time of birth, from the 20^{th} week of gestation to the 28th day of new born life.

Antenatal care is the cares receive from healthcare professionals during pregnancy period. It is offered a series of appointments with a midwife, or sometimes with a doctor who specialises in pregnancy and birth (an obstetrician).

Intrapartum factors: Preterm labour, caesarean section and low birth weight (<2500 gm) were associated with an increased risk of CP.

1.4 Research question

What are the common antenatal and intrapartum risk factors for the development of cerebral palsy?

1.5 Objectives

1.5.1 General objective

To identify the antenatal and intrapartum risk factors for the development of cerebral palsy.

1.5.2 Specific Objectives

1. To explore socio demographic characteristics of the participants.

2. To identify association between developing cerebral palsy and maternal age.

3. To find out the association between developing cerebral palsy and having maternal hypertension.

4. To find out the association between developing cerebral palsy and maternal Diabetes.

5. To determine the association between developing cerebral palsy and maternal eclampsia.

6. To determine the association between high risk pregnancy and development of cerebral palsy.

7. To discover the association between developing cerebral palsy and having medication during antenatal period.

8. To identify association between neonatal seizure and cerebral palsy.

9. To identify the association between neonatal head injury and cerebral palsy.

10. To find out the association between perinatal asphyxia and cerebral palsy.

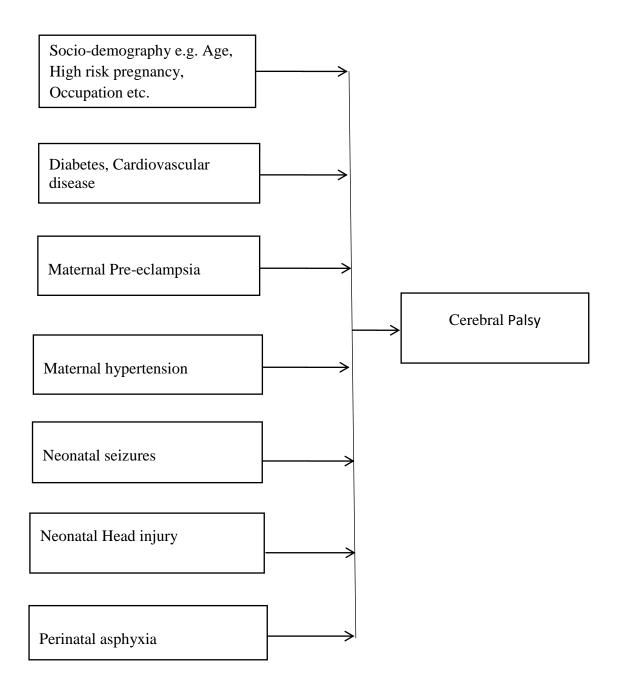
1.6 List of Variables

Conceptual framework

The anticipation for choosing this design is Cerebral Palsy because this disorder is common in our country. On the other hand the risk factors take long latent period for develop the disease. The conceptual framework of this study is presented below.

Independent Variables

Dependent Variable



CHAPTER-II

LITERATURE REVIEW

Cerebral palsy is the most common neuro developmental motor disability in children. The condition requires medical, educational, social, and rehabilitative resources throughout the lifespan (Hurley, et al., 2011). Amin et al. (2015) stated cerebral palsy (CP) describes a group of permanent disorders of the development of movement and posture, causing activity limitations that are attributed to nonprogressive disturbances that occurred in the developing fetal or infant brain. The motor disorders of cerebral palsy are often accompanied by disturbances of sensation, perception, cognition, communication, and behaviour, by epilepsy, and by secondary musculoskeletal problems (Rosenbaum, et al., 2007). According to the Surveillance of CP in Europe (SCPE) definition, cerebral palsy is a group of permanent and nonprogressive disorders of movement and posture caused by a central nervous lesion, damage or dysfunction originating early in life (Elkamil, et al., 2011). Cerebral palsy is the most common chronic motor disorder of childhood, affecting approximately 2 to 2.5 infants per 1,000 live births. The increase in survival rates for preterm infants has amplified the risk of brain injuries that potentially cause CP. In addition to immeasurable health, social, and psychological problems that the affected children and their families suffer CP has a huge economic impact (Faria, et al., 2011).

There is no definite cause of cerebral palsy rather some risk factors contribute to the development CP prenatal, natal or postnatal period (Tatla, et al., 2013).70 to 80% of cerebral palsy cases are acquired prenatally with unknown causes and birth complications, including asphyxia, are currently estimated to account for about 6 % of patients with congenital cerebral palsy, on the other hand neonatal risk factors for cerebral palsy include first cousin marriage birth after fewer than 32 weeks gestation,

birth weight of less than 5 lb with intrauterine growth retardation, intracranial haemorrhage and trauma and about 10 to 20% patients (Chen, et al., 2013), Preeclampsia affects 3-5% of pregnant women and is characterized by maternal hypertension and proteinuria occurring after 20 weeks of gestation (Melheim, et al., 2013). The pathological changes start when the specific causes resulting in neural damage and ending up with impaired neural connectivity as well as transmission. 10-15% of cerebral palsy cases are found during birth including prolongs labour, sudden birth, birth asphyxia, baby did not cry immediate after birth or by forceps delivery (Bangash, et al., 2014). Postnatal causes include toxic, infectious meningitis, encephalitis, traumatic such as drowning. There is also a relation between coagulopathies causing cerebral infarction and particularly hemiplegic type of CP. Postnatal events account for 12% - 21% of CP. But in a large number of cases, the cause of CP remain unknown (Kulak, et al., 2014). Before birth, occurs the disruption of normal development of the brain result of CP in about 70% of cases. According to a 2003 report by the American College of Obstetricians and Gynecologists (ACOG) and the American Academy of Paediatrics (AAP) conflicting to common belief that lack of oxygen reaching the foetus during labor and delivery contributes to only a small minority of cases of cerebral palsy. A slight number of babies also develop brain injuries in the first months or years of life result in cerebral palsy. In child the cause of cerebral palsy is unknown in many cases (American pregnancy association, 2013). We know the cause of CP is unknown. Brain injury or brain malformation is the cause of cerebral palsy that occurs while the brain is developing before, during or after birth. Muscle control, muscle coordination, muscle tone, reflex, posture and balance also disturbed due to cerebral palsy. It can also impact fine motor skills, gross

motor skills and oral motor functioning (My child, 2013). In many cases, the cause of congenital cerebral palsy is not identified. According to the timing of the brain insult, CP is valuable to classify the known causes where the prenatal, perinatal or postnatal. Congenitally brain malformations which including malformations of cortical development are caused by antenatal of CP. In general congenital malformations are strongly connected with cerebral palsy and children with congenital brain malformations also have more anomalies outside of the central nervous system. Metabolic disorders, maternal ingestion of toxins and rare genetic syndromes are less common cause of CP (Tan, et al., 2010). During a baby's development in the womb, congenital cerebral palsy results from brain injury. It is present at birth although it may not be detected for months. It is responsible for about 70% of children of cerebral palsy. Children are more likely to develop cerebral palsy when any of the following circumstances is present: Bleeding in the brain, Illnesses that cause an infant to go into shock, Infections of the central nervous system (such as meningitis or encephalitis), Interruptions in oxygen supply or blood flow to the brain, Maternal infections (chorioamnionitis), Physical trauma or injury, Poisoning from drugs or other toxic substances, Premature birth, Seizures. Although cerebral palsy isn't inherited, some genetic disorders can cause brain damage early in life. Such damage, in turn, can lead to cerebral palsy. In addition, research is uncovering genetic components to diseases that mimic the effects of cerebral palsy (Stephens &Vohr, 2009). CP is classified into four categories. They are Spastic, Athetoid, Ataxia and Mixed type of CP. Spastic cerebral palsy is the most common type of CP. Spastic cerebral palsy refers to the increased tone, or tension, in a muscle when normal muscles work in pairs. Allowing free movement in the desired direction when one group contracts and the other group relax. The flow of muscle tensions disrupted, due

to complications in brain-to-nerve-to-muscle communication. Muscles affected by spastic cerebral palsy become active together and restricted in actual movement. This causes the muscles in spastic cerebral palsy patients to be constantly tense or spastic. Mild cases of spastic cerebral palsy patients may have affect only a few movements or severe cases that can affect the whole body (Darsaklis, et al., 2011). The second most common type of cerebral palsy is athetoid or dyskinetic. Injuries to the basal ganglia can result in athetoid cerebral palsy, which causes involuntary muscle movements. The movements often interfere with speaking, feeding, grasping, walking and other skills requiring coordination. Now-a-days about 4% of people have cerebral palsy. Inability to activate the correct pattern of muscles during movement ataxia is defined. Injuries to the cerebellum can result in ataxic cerebral palsy, which causes poor coordination. That, in turn, affects balance, posture and controlled movements. Ataxic cerebral palsy can cause unsteadiness when walking and difficulties with motor tasks. Other type of CP is mixed CP. Injuries to multiple brain areas usually the cerebral cortex and basal ganglia can result in more than one kind of abnormal muscle tone. For example, someone could have spasticity and dystonia, or dystonia and rigidity. Cerebral palsy is a neurological disorder the signs or symptoms of cerebral palsy

may appear soon after birth or may take several months (Mandal, 2013). The most common early sign of cerebral palsy is developmental delay. Delay in reaching key growth milestones such as rolling over, sitting, crawling and walking are cause for concern. Physicians will also look for signs such as abnormal muscle tone, unusual posture, persistent infant reflexes and early development of hand preference (My child, 2013). Common signs of severe CP that may be noticed shortly after birth include: problems sucking and swallowing, weak or shrill cry, seizures and unusual positions. Often the body is either very relaxed or floppy or very stiff. In some severe cases many signs and symptoms are not readily visible at birth except and may appear within the first three to five years of life as the brain and child developed (My child, 2013). Severe motor and coordination impairment also occur (Mandal, 2013). Drooling is another but common symptom among children with CP. Children has movement and postural disorder associated with many disabilities such as- including intellectual disability, hearing and visual deficits, nutrition, feeding and swallowing problems, respiratory infections and epilepsy. Cerebral palsy suffers for long term and it affect activities of daily living and quality of life (Bell, et al., 2010). The symptoms of cerebral palsy include: excessive drooling, difficulty swallowing, sucking or speaking, tremors, and trouble with fine motor skills such as fastening buttons or holding a pencil, stiff or tight muscles, low muscle tone, exaggerated reflexes, uncontrolled body movement, toe walking, limping or dragging a foot while walking, walking with a scissor gait, turning in their legs as they walk. Children with cerebral palsy can also have feeding problems, mental retardation, seizures, learning disabilities and problems with their vision and hearing. The symptoms don't worsen with age but symptoms can range from mild to severe (Iannelli, 2008). Signs can appear during several stages of early life. They include: neonatal early Infancy (0-3 Months): high pitched cry, poor neck control, excessive lethargy or irritability, weak suck or tongue thrust or tonic bite, oral hypersensitivity, decreased interest in surroundings, stiff or floppy posture, abnormal or prolonged reflexes. Later infancy-inability to perform motor skills control of hand grasp by 3 months, rolling over by 5 months and independent sitting by 7 months. Abnormal developmental patterns: hand preference by 12 months, excessive arching of back, prolonged or abnormal parachute response, and logrolling. Abnormal developmental patterns after 1 year of age: W sitting means

both knee flexion, legs extremely rotation, bottom shuffling means scoots along the floor, tiptoe walking or hopping (Gershon, et al., 2013).

Muscle tone is defined as the tension of a muscle due to involuntary contractions of its motor units; it is determined both by the passive elasticity of muscular tissues, the visco elastic properties of the fibrillary proteins contained within each muscle fibre and by the active (though not continuous) contraction of muscle in response to the reaction of the nervous system (Kassolik, et al., 2009). Muscle tone is a result of both muscular components and neural components: it is the tension in a muscle due to the activity of some muscle fibres, and is controlled by the nervous system (Canning, 2006). Contraction is activated by a stimulatory nerve impulse from the central nervous system (CNS) (Allen, 2008). It triggers an action potential which stimulates the muscle fibre, causing it to contract. A muscle fibre is a single, elongated cell which extends the length of the muscle. A muscle is composed of 10,000 to 450,000 muscle fibres (Gracies, 2005). Myofibrils, contained in copious amounts in muscle fibres, are the contractile element of the muscle (Prado, et al., 2005). They are contained within the muscle fibre cytoplasm and extend the length of the cell. Not only can myofibrils contract, but they can elongate to endure stretching of the muscle. Each myofibril consists of a linked chain of sarcomeres. Sarcomeres contain myofilaments which are chains of contractile proteins. The myofilaments are either thin or thick, and lie in parallel layers, partially overlapping. The thinner myofilament mainly consists of actin; the thicker myofilament mainly consists of myosin. As proposed by the Sliding Filament Theory, muscle contraction occurs because the thick and thin filaments slide past one another increasing the amount of overlap between them. Myosin cross-bridges attach onto the actins filament, rotate towards the centre of the sarcomere, and slide

the actins filament towards the centre of the sarcomere. The actin layers are anchored to both ends of the sarcomere: pulling in of the actin filament subsequently draws in the ends of the sarcomere, reducing its length (Valle, et al., 2007). Billions sarcomeres shortening simultaneously results in contraction of the myofibril and because all myofibrils respond together, this causes contraction of the muscle fibre. Because sarcomeres, myofibrils, and muscle fibres all extend longitudinally within the muscle, the contraction and shortening of sufficient sarcomeres causes the entire muscle to contract and shorten in the same direction. It is this contraction which generates tension: without tension, no voluntary movement could take place (Allen, 2008). When tension develops, the ends of the muscle are drawn in towards the centre which causes it to shorten and produce movement. The increase in tension increases tone, which may then instigate movement (Windhorst, 2007). When tone is high, bony points move closer together which is also true when observing tension. It can therefore be concluded that increased tension, generated by increased contraction, increases tone (Bloemsaat, et al., 2005). Muscle tone increases as a result of the increased number of activated myosin cross bridges. This increases the proportion of actins filament which overlaps the central myosin myofilament. This results in more contracted muscle fibres which increases tension (Lee, et al., 2005). Muscle tone thus results from neural pathways and the Central Nervous System (CNS), the number of contracted muscle fibres and the amount of overlap between actins and myosin myofilaments. For this reason, it becomes apparent why children with physical disabilities resulting from neurological impairments often have abnormal muscle tone: it is an indirect resultant of abnormal development or damage to motor areas in the brain which disrupt the brains ability to adequately control tone

(Stevens, et al., 2009). Therefore one can postulate that high muscle tone is a result of excessive tension caused by excessive contraction, and low muscle tone is a result of insufficient tension caused by insufficient contractions. Muscle tone keeps muscle firm but it does not result in a force strong enough to produce movement. At complete rest, a muscle has not lost its tone although there is no neuromuscular activity in it (Chang, et al., 2010). When muscles in the back of the neck are in normal tonic contraction, the head is kept upright. To execute fine motor skills, a low degree of contraction is required; to execute gross motor movements, a large degree of contraction is required. Muscles thus need to vary their tone (by varying contraction) ascertain times throughout gross movements to ensure smooth movement. The ability to alter muscle tone is therefore very important. Russell, et al. (2011) stated that there are 4 predominant motor types of CP such as spastic, ataxic, dyskinetic and mixed types of CP. Spastic CP is the commonest and accounts for 70%-75% of all cases, dyskinetic -10% to 15% and ataxic is less than 5% of cases. Spasticity occurs when muscles have increased tone and appear stiff. This is the most common type of CP. In contrast to spastic ataxia affects balance and coordination. Children with ataxic CP may appear shaky and unsteady. In addition, dyskinesia causes a person to have involuntary movements, which generally increase when they try to move and the person can present with any combination of motor types. CP can also be classified according to the part of the body affected: quadriplegia (affects all four limbs), diplegia (affects both legs) and hemiplegia (one side of the body is affected). Mcintyre, et al., (2012) stated that quadriplegic CP is the most severe form involving all four limbs, and the trunk upper limbs are more severely involved than the lower limbs. Voluntary movements are few; vasomotor changes of the extremities are common. Most children have psuedobulbar signs with difficulties in swallowing and recurrent aspiration of food material. In hemiplegic CP, spastic hemiparesis is a unilateral paresis with upper limbs more severely affected than the lower limbs. It is seen in 56% of term infants and 17% of preterm infants. Voluntary movements are impaired with hand functions being most affected. Pincer grasp of the thumb, extension of the wrist and supination of the forearm are affected. In the lower limb, dorsiflexion and aversion of the foot are most impaired. There is increased flexor tone with hemiparetic posture, flexion at the elbow and wrist, knees and equines position of the foot. Palmer grasp may persist for many years. Sensory abnormalities in the affected limbs are common. Seizures occur in more than 50%. Visual field defects, homonymous hemianopia, cranial nerve abnormalities most commonly facial nerve palsies are seen. Spastic diplegia is associated with prematurity and low birth weight. Spastic types exhibit pyramidal involvement with upper motor neuron signs, weakness, hypertonia, hyperreflexia, clonus and positive Babinski. Dyskinesia is characterized by extra pyramidal involvement in which rigidity, chorea, choreo athetosis, athetoid and dystonic movements are seen. This type of CP is also associated with birth asphyxia. The severity of dystonic postures may vary with body position, emotional state and sleep. Clonus and Babinski are absent. Primitive reflexes are more prominent and persist for a longer time in dyskinetic CP. These movement patterns are eliminated in sleep, with a decrease in tone of the affected limbs. There are also abnormalities of posture control and coordination. Those children who are hypotonic to start with may develop into this type by 1 to 3 yrs of age. In majority of this group, there is no cognitive impairment. Dysarthria or motor problems with drooling and swallowing difficulties are seen. 30% of children with CP have a mixed pattern of involvement. While contractures are common in spastic group, they are uncommon in the extra pyramidal group. Hypotonic CP is characterized by generalized muscular hypotonia that persists beyond 2 to 3 yrs of age that does not result from a primary disorder of muscle or peripheral nerve. The deep tendon reflexes are normal or hyperactive, and the electrical reactions of muscle and nerve are normal. More than half the children develop frank cerebellar deficits with in coordination, ataxia and impaired rapid succession of gross motor functional movements (Sankar & Mundkur, 2005).

The component of diagnosis is physical examination and medical history taking. Development and any other problem of child are assessed. Tests such as a CT scan, MRI, and ultrasound are used to find out the cause of cerebral palsy (Health Link, 2013). There is no cure for CP but treatment can improve the lives of those who have the condition. It is important to begin a treatment program as early as possible (Centre for Disease Control and Prevention, 2012).

A multidisciplinary team approach is effective for the treatment of CP. The multidisciplinary team includes health care professionals such as paediatricians, rehabilitation specialists, neurologists and physiotherapists, occupational therapists and speech therapists. The multidisciplinary team develops an individualized treatment plan depending on the severity of cerebral palsy (Physician & Nurses, 2013). To achieve their goal strive to: 1) Help children with cerebral palsy achieve maximum physical, intellectual and emotional development 2) Educate patients, parents and the community about children with cerebral palsy 3) Develop and promote clinical research programs that will advance the pharmacological, surgical and therapeutic treatment of cerebral palsy 4) Continue making advances in the diagnosis, management and treatment of cerebral palsy (Children's Hospital, 2013).

Health care professionals usually collectively set up common goal while patient is placed in the centre. Then, their goal of treatment would be to make at least near to the development to that particular age or modified independent.

Medical specialists may prescribe medications that assist movement issues. Some medications are taken orally (e.g. diazepam) and others are injected or delivered through surgically implanted pumps (e.g. Baclofen). Many children with cerebral palsy benefit from Botulinum toxin type injections into muscles affected by spasticity. This intervention is used from about two years of age and is most effective when used in conjunction with therapy. Selective Dorsal Rhizotomy (SDR) is a neurosurgical procedure that is used in a small percentage of children with cerebral palsy to permanently reduce spasticity in their legs (Cerebral Palsy Alliance, 2015).

Physiotherapists focus on encouraging a person's day-to-day movement skills such as sitting, walking, playing, dressing and toileting. Practicing the gross and upper extremity exercise for example using constraints induced movement therapy (CIMT), weight bearing exercise, proprioceptive exercise, balance exercise etc. They will use a range of specialist interventions such as movement training and equipment, e.g. walking frames, wheelchairs, supportive seating, footwear and orthotics. Physiotherapist uses daily range-of-motion (ROM) exercises to prevent or delay contractures that are secondary to spasticity and to maintain the mobility of joints and soft tissues. Stretching exercises are performed to increase range of motion (Thorogood & Alexander, 2013).

Orthopaedic surgeons correct joint deformities and lengthen muscles. Surgery usually takes place in a child's late primary years or early adolescence to improve walking quality and reduce pain. Paediatric rehabilitation specialists support the management of some of the conditions associated with cerebral palsy, such as spasticity, musculoskeletal issues and growth. Physiotherapists may also focus on preventing impairments that might affect movement. They use casts, orthotics and muscle strengthening exercises (Cerebral Palsy Alliance, 2015).

Progressive resistance exercises should be taught in order to increase strength. The use of age-appropriate play and of adaptive toys and games based on the desired exercises are important to elicit the child's full cooperation. Strengthening knee extensor muscles helps to improve crouching and stride length. Postural and motor control training is important and should follow the developmental sequence of normal children (that is, head and neck control should be achieved, if possible, before advancing to trunk control) (The Ontario Federation for Cerebral Palsy, 2011).

Psychologists can provide assessment of a child's learning and development. Special educators work with families to develop a program of interventions to address each child's learning needs. Occupational therapists can facilitate a child's active participation in these learning activities. Psychologists and occupational therapists can provide assessment and recommend learning strategies to compensate for perceptual difficulties. Psychologists can offer assessment and support for emotional and behavioural challenges. Psychologists may also assist with behaviour therapy or help families to establish routines to help resolve children's sleep issues. For children with postural issues that make sleep difficult, occupational therapists and physiotherapists may prescribe special sleep systems to help children feel more comfortable in bed.

Social workers support people with cerebral palsy and their families in many ways, including strengths-based counselling and mutual aid group work. Treatment (intervention) for severe difficulties with digestion, such as gastro-oesophageal reflux (GOR) includes medications or sometimes surgery. If the individual has severely limited eating skills or experiences an unsafe swallow, non-oral feeding may be

recommended. This involves a surgically inserted feeding tube to the stomach or the small intestine. Speech pathologists provide assessment and treatment (intervention) for eating, drinking and swallowing difficulties. Examples of these are learning to use the muscles of the mouth better, using specially designed utensils and sitting in an optimal position. Dieticians provide advice on improved and balanced nutrition. Speech pathologists may suggest various strategies to help people manage saliva loss. These include remembering to wipe their mouth and wearing age-appropriate clothing protection. They may also recommend special exercises for the face and mouth, which may lead to better ability to control saliva. Medical specialists may prescribe medication for saliva control. This is not usually considered a long term treatment option, but can be useful to dry up saliva temporarily. Botulinum Toxin Type A, injected into the salivary glands has also shown to reduce the secretion of saliva. 2.8.9. Interventions for hearing and vision impairment. Hearing and Vision impairment are managed as they are for the general population by Ophthalmologists, Optometrists, Audiologists and Doctors (GPs). Interventions include corrective eye wear (glasses), contact lenses, hearing aids and in some cases medication or surgery. Hwang et al. (2011) stated that caregivers of children with CP may be under more physical, psychological, and financial burdens compared with those who provide care for children who develop in a typical manner because their responsibilities are greater.

In addition to providing direct daily care and support, caregivers of children with CP invest time and effort in assisting with interventions such as physical, occupational, and speech therapy. Thus caregivers perceptions of their child's needs and of efforts related to the daily care of their child are likely to have substantial impact on the selection and success of the child's rehabilitative management

In Western countries, cerebral palsy is diagnosed in two to three infants in every 1000 live born Children. Cerebral palsy is diagnosed on the basis of clinical symptoms only and is defined as "a group of permanent disorders of the development of movement and posture, causing activity limitation, that are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain." Rosenbaum et al. (2007) mentioned that perinatal hypoxia was previously considered the major cause of cerebral palsy, whereas current knowledge suggests that prenatal causes are most important. Children with low birth weight and asymmetrical body proportions are at increased risk of intrapartum complications and neonatal morbidity, including intraventricular haemorrhages of grades III to IV, compared with children whose birth weight is appropriate for gestational age. Dashe, et al. (2000) mentioned that a study by Blair and Stanley concluded that, overall, spastic CP in 44% of all sufferers could be attributed to growth deviation at birth. Despite extensive research, the causes of CP are known in only a minority of cases. In children born very preterm, the typical brain lesion is focal periventricular leukomalacia in deep white matter, leading to the characteristic spastic bilateral CP subtype with lower limb involvement (diplopia). In term-born infants, the most common CP subtype is spastic unilateral CP, considered to be mainly the sequel of antenatal or perinatal stroke. In contrast, global hypoxicischaemic injury in children born at term, for instance due to fetopelvic disproportion, is considered to be the main cause of dyskinetic and spastic bilateral subtypes with four-limb involvement (quadriplegia). However, in the majority of cases, CP probably has an antenatal cause; while only about 10% of cases of CP have intrapartum causes (Jarvis, et al., 2005). Previous studies on the relationship between growth deviations and the risk of CP have primarily studied decreased or increased birth weight. We wanted to expand previous knowledge by exploring the association

between deviations of other measures of foetal growth and the risk of CP in singletons born at term, and, therefore, we assessed not only birth weight, but also birth length, head circumference, and body proportions. Since in most cases, CP in children born at term is thought to be of antenatal origin, we hypothesized that children with CP would be shorter, would have lower birth weight, and would have a smaller head circumference at birth. The increase in survival rates for preterm infants has amplified the risk of brain injuries that potentially cause CP. In addition to immeasurable health, social, and psychological problems that the affected children and their families suffer CP has a huge economic impact (Faria, et al., 2011). In many cases, the cause of congenital cerebral palsy is not identified. According to the timing of the brain insult, CP is valuable to classify the known causes where the prenatal, perinatal or postnatal. Congenitally brain malformations which including malformations of cortical development are caused by antenatal of CP. In general congenital malformations are strongly connected with cerebral palsy and children with congenital brain malformations also have more anomalies outside of the central nervous system. Metabolic disorders, maternal ingestion of toxins and rare genetic syndromes are less common cause of CP (Reddihough& Collins, 2003).

Kadhim, et al., (2005) reported that cerebral palsy (CP) which is a non-progressive condition affecting approximately 3 in 1000 new-borns, is characterized by acquired brain damage and it affects motor and cognitive functions. However, Krageloh-Mann (2008) stated that Perinatal asphyxia has long been believed to be a major cause of CP. Advances in perinatal care have led to decreased mortality rates among newborns. However, recent epidemiologic assessments indicate that the incidence of CP is stable or increasing in some industrialized countries. The pathology of CP in term newborns is very different from preterm infants. Brain mal-developments are seen in

16% of term and 2.5% of preterm infants with CP and gray matter lesions are more often seen in term (33%) than preterm (3.5%) CP infants. However periventricular white matter lesions occur significantly more often in preterm (90%) than in term (20%) infants (Krageloh-Mann, 2008). Moreover, Bax et al. (2007) mentioned that early brain injury in CP frequently results life-long disability, with serious adverse effects and implications for the child, family, and society. Neonatal encephalopathy (NE) in the term newborn is a clinical syndrome of disturbed neurologic function that presents in early life and occurs in 1-6 per 1000 live term births. NE secondary to hypoxia-ischemia is the most common etiology of this condition in term newborns and is a major cause of morbidity and mortality. As many as 20% of affected infants with NE die during the neonatal period, whereas permanent neuro- developmental disability will be seen in 25% of the surviving children. Even if the pathology of NE is well-recognized, numerous questions remain regarding the causes and risk factors for pre-, peri-, and postnatal predictors of outcome. Because risk factors for CP in term infants differ from premature infants (Andersen, et al., 2008). In order to conduct preventive measures, it is necessary that the risk factors, etiology and the pathophysiology of the insult in this group be determined. However, controversy exists regarding many of these risk factors (Soleimani, et al., 2013). The exact etiology of CP is not yet well understood, and brain lesions are thought to be associated with perinatal events of varying causes. Several predictors for the development of CP have been identified in various settings, such as maternal age, preeclampsia, chorioamnionitis, small for gestational age, multiple births, assisted reproduction, a relative with CP, breech position, bleeding at any time in pregnancy, male sex, and multiple miscarriages (O'Callaghan, et al., 2011).

The increase in survival rates for preterm infants has amplified the risk of brain injuries that potentially cause CP. In addition to immeasurable health, social, and psychological problems that the affected children and their families suffer CP has a huge economic impact (Faria, et al., 2011). In many cases, the cause of congenital cerebral palsy is not identified. According to the timing of the brain insult, CP is valuable to classify the known causes where the prenatal, perinatal or postnatal. Congenitally brain malformations which including malformations of cortical development are caused by antenatal of CP. In general congenital malformations are strongly connected with cerebral palsy and children with congenital brain malformations also have more anomalies outside of the central nervous system. Metabolic disorders, maternal ingestion of toxins and rare genetic syndromes are less common cause of CP (Reddihough & Collins, 2003).

During a baby's development in the womb, congenital cerebral palsy results from brain injury. It is present at birth although it may not be detected for months. It is responsible for about 70% of children of cerebral palsy. Children are more likely to develop cerebral palsy when any of the following circumstances is present: Bleeding in the brain, Illnesses that cause an infant to go into shock, Infections of the central nervous system (such as meningitis or encephalitis), Interruptions in oxygen supply or blood flow to the brain, Maternal infections (chorioamnionitis), physical trauma or injury, poisoning from drugs or other toxic substances, premature birth, seizures. Although cerebral palsy isn't inherited, some genetic disorders can cause brain damage early in life. Such damage, in turn, can lead to cerebral palsy. In addition, research is uncovering genetic components to diseases that mimic the effects of cerebral palsy (Stephens and Vohr, 2009). A clinical presentation of Cerebral palsy (CP) may result from an underlying structural abnormality of the brain, early prenatal, perinatal, or postnatal injury due to vascular insufficiency, toxins of infections, pathophysiologic risks of prematurity. Evidence suggests that prenatal factors result in 70-80% of cases of cerebral palsy (Mc Conachie, et al., 2006). Cerebral palsy, expect in its mildest forms, can be seen in the first 12-18 months of life. It present when children fail to reach movement milestone. Babies most at risk of cerebral palsy are those born prematurely or with low birth weight (Chowdhury, 2005). Cerebral palsy which occurs due to antenatal, neonatal or postnatal causes. Therefore, the emphasis would mainly be on the availability of a functional and efficient antenatal care and on the availability of a well-equipped neonatal care units and services to avoid the problem of having babies with cerebral palsy (Saadi, et al., 2012).

Ferriero, (2004) suggested that the neonatal encephalopathy (NE) in the term newborn is a clinical syndrome of disturbed neurologic function that presents in early life and occurs in 1to 6 per 1000 live term births. NE secondary to hypoxia-ischemia is the most common etiology of this condition in term new-borns and is a major cause of morbidity and mortality. As many as 20% of affected infants with NE die during the neonatal period, whereas permanent neuro-developmental disability will be seen in 25% of the surviving children. Early brain injury in CP frequently results life-long disability, with serious adverse effects and implications for the child, family, and society. In the absence of a known pathophysiological mechanism. Soleimani et al. (2009) state that only supportive care is provided; there is no evidence for the effectiveness of preventive strategies. Even if the pathology of NE is wellrecognized, numerous questions remain regarding the causes and risk factors for pre-, peri-, and postnatal predictor of outcome. Because risk factors for CP in term infants differ from premature infants11 and in order to conduct preventive measures, it is necessary that the risk factors, etiology and the pathophysiology of the insult in this group be determined. However, controversy exists regarding many of these risk factors. This study aims to determine the maternal and neonatal factors associated with term infants diagnosed with CP born in Tehran, Iran. Cerebral palsy (CP) is an umbrella term that describes impaired muscle control of movement and posture caused by early insults to the developing brain (Surveillance of cerebral palsy in Europe, 2000). The incidence is approximately 2.1 per 1000 live births. Normal intrauterine growth is essential for later normal growth and health, and intrauterine growth restriction has repeatedly been identified as an independent risk factor for CP (Stoknes, et al., 2012) .However, at the other extreme, excess growth resulting in high birth weight has also been shown to be a risk factor for CP. Jarvis, et al. (2005) state that Children with above or below average birth weight who later develop CP will often exhibit the most severe motor and cognitive impairments. Deviation in foetal growth is difficult to diagnose during pregnancy; thus, weight at birth is used as a proxy for foetal growth, and statistical cut-offs based upon birth weight charts are used to diagnose deviations. Moreover, children with deviating birth weight may be further divided into those with symmetrical and those with asymmetrical body proportions. In suspected foetal growth restriction, different body proportions are considered to reflect the cause: smoking in pregnancy is associated with symmetrical growth restriction 10 and asymmetrical growth restriction could result from placental conditions, defined in this study by biparietal diameter and mean abdominal diameter. Soleimani, et al.(2009) mention that the Children without CP, and that these deviations would be the typical findings in children with unilateral and diplegic CP subtypes. In contrast, we hypothesized that children with dyskinetic and spastic

quadriplegic CP subtypes would be larger and more often have asymmetrical body proportions, manifesting as low weight for length. Wolraich, et al. (2008) mentioned that the cerebral palsy refers not to a single condition but to a number of different and varied chronic conditions. The traditional definition of cerebral palsy is a nonprogressive impairment in movement or posture caused by in- jury or anomaly of the developing brain. It is the most common neuron motor developmental disability of childhood, affecting as many as 8000 to 12,000 children born in the USA each year, correspond- in to a prevalence rate of between 2 and 3 per 1000 children . Recent improvements in neonatal care have not re- salted in a decline in the overall prevalence of cerebral palsy and, in fact, greater numbers of very preterm, very low birth weight infants are surviving with cerebral palsy and other developmental problems (Glew, et al., 2011).

Gilbert et al. (2010) suggested that the cerebral palsy is a condition which occurs due to antenatal, neonatal or postnatal causes. Therefore, the emphasis would mainly be on the availability of a functional and efficient antenatal care and on the availability of a well-equipped neonatal care units and services to avoid the problem of having babies with cerebral palsy. We were aiming that; this study would highlight the need for more emphasis on antenatal and neonatal care services in Iraq. We also hoped that, this study could detect the real association between the hypothesized risk factors and the occurrence of cerebral palsy among mothers of CP and CP children born in Baghdad. Last but not least, we thought that, this study would give an explanation for the increasing incidence and prevalence of cerebral palsy among Iraqi children.

Those disorders which were classified as chronic disorders did not get the full sponsorship of the government at that time as the priority in health care services was shifted towards more acute health problems. At the same time, the national income was also shifted towards military spending in favorite of spending on other sectors of community like health and education. Maternal and obstetric factors have been discussed in many studies before as potential risk factors for CP. In Iraq however, we don't have any study before about any possible correlation between maternal, obstetric or foetal risk factors with occurrence of CP. The antenatal period extends from conception to the time of birth. Disabling problems can occur at any point in the developmental process between those two events (Glew, et al., 2011).

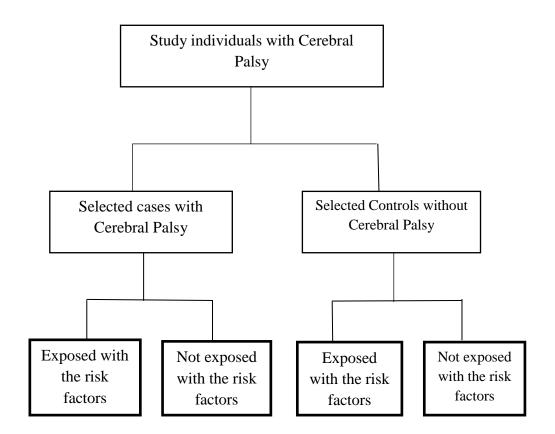
Saadi, et al. (2012) stated that the maternal risk factors and their correlation with occurrence of CP have been highlighted in few studies around the world. In one study, a researcher has found that, of the 6,145,357 deliveries examined during the study 8946 cases of CP were identified (1.45 per 1000 live births). In both CP groups, there were significantly increased risks of CP related to advanced maternal age (>40 y) and increasing parity; Pregnancy risk factors (e.g. chronic hypertension and preeclampsia). Gestational (but not pre gestational) diabetes increased the risk in term. The perinatal period is the time immediately before and after birth. Disabilities originating from this time period are primarily biomedical ones. They may result from many causes like, drugs taken during labour and delivery, prematurity, injury, oxygen deprivation, or infections acquired during the way through the birth canal (Saadi, et al., 2012). There was one very interesting study, which was trying to determine the neonatal predictors of cerebral palsy in extremely low birth weight infants (<1000 g). The out- come variables for this study included maternal demo- graphics, obstetric complications, and neonatal outcomes like (gestational age at delivery, birth weight, APGAR scores, intrauterine growth restriction, respiratory distress syndrome, intraventricular haemorrhage and neonatal sepsis). Interestingly, the results of this study showed that, primigravid (OR = 5.52, 95% CI 1.67 - 18.3), early neonatal sepsis

(OR = 12.9, 95% CI 2.94 - 57.2) and chorioamniomtis (OR = 3.71, 95% CI 1.16 -11.9) all were significantly associated with the development of cerebral palsy. As in any other disease or syndrome, CP risk factors do interact with each other's and do correlate to each other's (Costantine, et al., 2007). It has been well established that both the preterm infant and low birth weight infant are at particular risk for CP. The etiology of a large portion of preterm births, particularly in the very preterm infant, is believed to be intrauterine infection. It is known that microorganisms from the lower genital tract can gain access to the intrauterine cavity and subsequently infect the placenta, mem- branes, and fetus. Recent studies suggest that feto-pla- cental and uterine infection/inflammation play a role in the initiation of preterm labour and contribute to the de- velopment of central nervous system injury and CP (Saadi, et al., 2012). Neonatal asphyxia has also been a known risk factor for developing CP. It may occur during a prolonged or difficult birth, and, because the brain suffers damage very quickly without a fresh and adequate supply of oxygen, brain damage can result. One major danger associated with birth is haemorrhage, which is caused when very strong pressure on the head of the foetus breaks blood vessels in the brain. Another danger is failure of the infant to begin breathing soon after being separated from the maternal source of oxygen. Asphyxia in a new-born is more likely to damage the cells of the brain stem than those of the cortex, and to result in motor defects. The child may experience paralysis of the legs or arms, a tremor of the face or fingers, or inability to use the vocal muscles. In this last case, the child may have difficulty learning to speak. The term cerebral palsy describes a variety of motor defects associated with damage to the brain cells, possibly as a result of lack of oxygen during birth process. It is estimated that about 30% of cerebral palsy cases involve problems that occurred during birth or immediately afterward (Thorngren and Herbst, 2006).

For decades, birth asphyxia was believed to be the predominant etiology of CP. More recently, multiple antenatal factors have been shown to be likely causes of CP in both the preterm and term infant, with birth asphyxia playing a minor role. It is now believed that 70% to 80% of cases of CP are due to antenatal factors with 10% to 28% of CP cases due to birth asphyxia in term and near-term infants as one study suggested that birth asphyxia may not be such an important cause of CP but might constitute one element of a multi-factorial cause, that neonatal signs associated with birth asphyxia might be early manifestations of CP from a variety of causes, of which birth asphyxia is only one (Hagberg, et al., 2001). A very significant finding from another study was, an independent 8 times increased risk for developing cerebral palsy among children whose mothers were employed compared to those whose mothers were unemployed (Saadi, et al., 2012).

3.1 Study design

Hospital based unmatched case control study was conducted at the paediatric unit of Centre for the Rehabilitation of the Paralysed (CRP), Savar. The children who had already been diagnosed with CP (based on the medical history, a clinical examination, and imaging of the brain) and who attended follow-up are interviewed using a pretested questionnaire. Cases with CP were classified into three major categories; spastic, dyskinetic, and ataxic subtypes. Those children who were diagnosed as cerebral palsy were considered as cases and those who did not were kept in the control group. As it was a case control study, so the study began from the outcome or cerebral palsy and the investigator had to find out the exposure. If the exposure among the cases is higher than control then it was said that it was a risk factor. For selection of control of each case were selected from the hospital those who did not have cerebral palsy and matched for time and place of birth as much as possible after exclusion of major congenital anomalies. The investigator used selected same mothers siblings as a control group of the study. After obtaining informed consent, pretested questionnaires are applied for each mother/guardian to gather maternal socio-demographic characteristics, and information on labor and perinatal and neonatal events that are expected to be predictors for CP (age, parity, antepartum haemorrhage, and intra-partum fever, mode of delivery, gestational age, birth weight, and admission to the nursing). There is demonstrating the design of the case control study.



3.2 Study site

The study was conducted at Paediatric unit of outdoor physiotherapy department of Centre for the Rehabilitation of the Paralysed (CRP) at Savar.

3.3 Study Duration:

The study duration was from September 2015 to May 2016

3.4 Study population and sample population

The study population was the children with Cerebral Palsy those who attended in CRP for treatment.

Sampling

In the study area there was no previous study from Bangladesh. Also there was no base line data to calculate the sample size. The sample size was calculated using following formula,

$$n = \frac{\left[Z_{\alpha}\sqrt{(1+m)p(1-p)} + Z_{\beta}p_{1}(1-p_{1}) + mp_{0}(1-p_{0})\right]^{2}}{(p_{1}-p_{0})^{2}}$$

$p^{\sim} = \frac{1}{1 + 1/m}$ $p_{1} = \frac{p_{0}\Psi}{1 + p_{0}(\Psi - 1)}$ $Z_{\alpha} = alpha = 95\% \text{ confidence level} = 1.96$ $Z_{\beta} = 1 - power = 80\% \text{ power} = 0.84$ $\Psi = odds \text{ ratio} = 20 \text{ (Soleimani, et al., 2013).}$ $p_{o} = \text{Prevalence of cerebral palsy in Bangladesh} = 6.1\% \text{ (Tabib, 2009).}$

Using the above formula and the parameter of the sample size calculation is given

below

$$p_1 = \frac{0.061 \times 20}{1 + 0.061 (20 - 1)} = 0.38$$
$$p^{*} = \frac{0.38 + 0.061/1}{1 + 1/1} = 0.22$$

$$n = \frac{\left[1.96(1+1)0.22(1-0.22) + 0.84 \ 0.38(1-0.38) + 0.061(1-0.061)\right]^2}{(0.317_{-}0.085)^2} = 46$$

As duration of data collection was 3 months so total sample size was 50 samples where 25 were cases and 25 were controls.

3.5 Inclusion criteria

- 1. Confirmed cases of cerebral palsy by Paediatrician were considered as case.
- Maternal age was categorized as <18 years, 18-35 years and >35 years. Term and pre-term delivery was defined as delivery ≥36 completed weeks of gestation (Soleimani, et al., 2013).
- 3. Subjects who were siblings or special relatives of cases or other Paediatric problem excluding Cerebral palsy considered as control.
- 4. Both boy and girl were included.
- 5. Subjects who were willingly participated.

3.6 Exclusion Criteria

- Maternal Age was more than 35 years because it was a risk factor for developing birth defect child.
- 2. Maternal Epilepsy
- 3. Other Unstable medical condition.

3.7 Sampling technique

The investigator was using purposive sampling technique to collect data. Selection of case: Those children who have suffered cerebral palsy. Selection of control: The investigator was using selected children's siblings or special relatives. Sample size: For this study 25 cases and 25 controls was selected randomly.

3.8 Data Collection Procedure

A questionnaire was developed after thorough literature review regarding risk factors of developing cerebral palsy. This questionnaire was filled out by the clinician after having interview with the children's mother after obtaining consent.

All new consecutive children who were attending at CRP and were diagnosed as Cerebral Palsy were asked to participate in the study. A structured questionnaire was used for identifying the risk factors. These questionnaires were developed after reviewing literature about the risk factors of Cerebral Palsy. In the questionnaire participants demographic information including age, sex, marital status, level of education, income, occupational history including types of job, bad obstetric history, maternal disease, pre-eclampsia and placenta previa had been asked about developing of CP. Intrapartum related factors such as Oxytocin therapy, preterm labour, caesarean section and low birth weight were asked with an increased risk of CP. Neonatal factors such as prolonged ventilation, septicaemia, hyper-bilirubinia, neonatal seizures and severe cranial ultrasound abnormality were associated with CP.

3.9 Data Analysis

Quantitative data was analysed using SPSS. Descriptive and inferential statistics was used for data analysis. Continuous variables were expressed as mean \pm SD, and categorical variables as percentages. Prevalence rates was presented as percentage and compared among different children groups (case & control; Male & Female etc).

As this was a case-control study for finding the risk factors Odds ratio. An odds ratio was a measure of association typically used to quantify the strength of association between a potential risk or protective factor (exposure) and an outcome. In this study the outcome variable was having Cerebral Palsy or not and analysis was done to identifies different risk factors according to their strength of association.

The odds ratio (OR) was measured by the relative magnitude of the odds of exposure among individuals who have the disease (cases) and the odds of exposure among individuals who do not have the disease (controls) from a typical 2 x 2 table as below:

	Case	Control
Exposure	a	b
N Exposure	С	d

Odds of exposure among cases: a/c

Odds of exposure among controls: b/d

Odds ratio = (a/c)/(b/d)

If odd ratio for certain exposure becomes more than 1 then we could say it is the risk factor. 95% confidence interval was used to identify significance of the OR the risk factors by using following formula:

*i*lnOR±z.SE_{lnOR}

Where e is the base on the natural logarithms (e $\approx 2.71828...$), z is a Standard normal deviate corresponding to the desired level confidence (z = 1.96 for 95%), and

$$SE_{lnOR} = \sqrt{\frac{1}{a} + \frac{1}{b} + \frac{1}{c} + \frac{1}{d}}$$

Confidence interval having 1 between its ranges was considered to be a nonsignificant risk factor.

3.10 Ethical Consideration

The study protocol was submitted to the Institutional Review Board (IRB) of BHPI and board approved the proposal. Besides, permission was also taken from the department of Physiotherapy to collect the data. Written consent was taken from the participant. The participants were informed about the purpose of the study, anonymity, their rights to refuse answering any question, withdrawn from the study at any point of time and other issues mentioned in the form before starting the interviews. The data was kept in a secure place where only the researcher had the access. World Health Organization (WHO) and Bangladesh Medical Research Council (BMRC) guidelines were also followed.

4.1 Socio-demographic Information

An exploratory data analysis was conducted among 50 mothers to have a preliminary idea about the trends of data.

The mean age of the respondents was 1.56 years with a standard deviation of 0.50 (table 1). Majority of the respondents (56%) were 4 to 6 years old followed by 1 to 3 years old (44%). A total 46% respondent was boy. 68% of the cases were girl whereas 40% of the controls were girls.

Table 1: Characteristics of children age and gender ratio								
	Total	Total Case						
	N=50	n=25	n=25					
Age (mean ± SD)	1.56 ± 0.50	1.56 ± 0.50	1.55 ± 0.51					
1-3 Years	22 (44%)	14 (56%)	08(32%)					
4-6 Years	28 (56%)	18 (72%)	10(40%)					
Sex								
Boy	23 (46%)	15 (60%)	8 (32%)					
Girl	27 (54%)	17 (68%)	10 (40%)					

4.2 Mother age during child birth

Among the 50 participants 50% (n=25) participants were between 18-22 years, 32% (n=16) were between 23-26 years, 14% (n=7) were between years 27-30 years and 4% (n=2) were 31-35 years. Average mean age was 26.5 years and minimum age was 18 years and maximum age was 35 years (Figure-1).

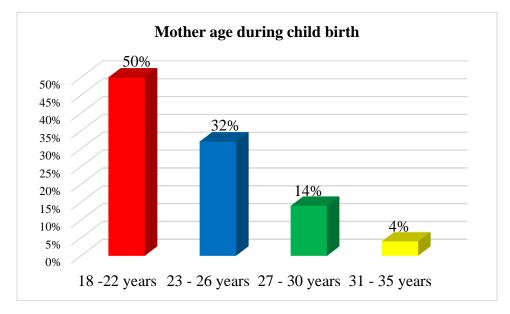
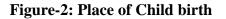
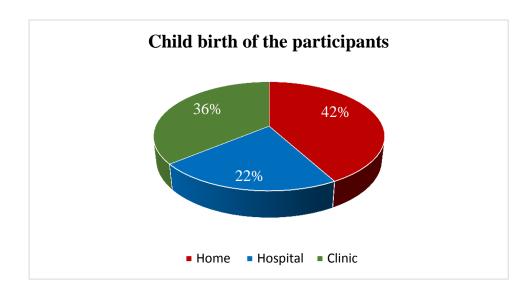


Figure-1: Mother age during child birth.

4.3 Place of Child birth

The study identify that 50 participants there 42% (n=21) participants were born at home, 22% (n=11) participants were born hospital, 36% (n=18) participants were born clinic. (Figure-2)





4.4 Age & Educational Status of the participants

Out of the 50 participants, most of the respondents were at the age of 26 years or below and that was about 80% (n=41). Educational status of mothers showed that 8% (n=4) mothers could do signature, 46% (n=23) completed primary education, 38% (n=19) completed secondary education, 4% (n=2) completed degree education, 4% (n=2) completed master degree (Table-2).

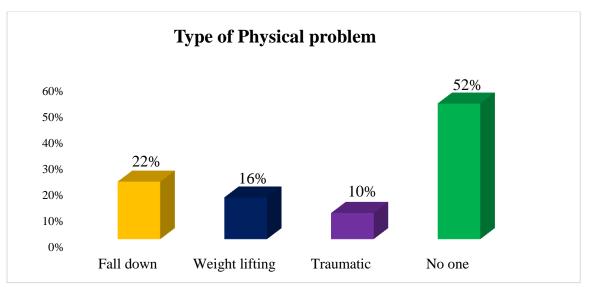
Age(Mother)	Ν	Percentage (%)		
18-26 years	41	80		
27-35 years	09	20		
Educational status (Mother)				
Signature	4	8		
Primary	23	45		
SSC	19	39		
Degree	2	4		
Masters	2	4		

Table-2 shows age of mother and their educational status:

4.5 Type of Physical problem

All the cases and controls performed mother's physical problem 22.0% (n=11), fall down, 16% (n=8) weight lifting, 10% (n=5) traumatic and 52.0% (n=26) no related cause (Figure-3).





Risk factors associated with cerebral palsy:

This study was a case control study and the mode of association between disease and risk factors was Odds ratio. 95% confidence interval was calculated for finding out the significant of the association. If 1 came between the lower bound and the upper bound of confidence interval it was considered as non-significance.

Antenatal care and cerebral palsy:

The Odds ratio for the antenatal care of the study was 1.833 suggesting cerebral palsy is 1.833 times as frequent in the mother who are taking antenatal care compared in the non antenatal care mother (Table 3). The confidence interval of odds ratio was ranging from 0.387 to 8.674 indicating that this association was not significant as 1 came between the intervals.

Exposure	Case	Control	OR	95% of CI		Chi	P-	Significant
				L/L	U/L	Square	Value	
Antenatal Care								Not
Yes	23	22	1.833	.387	8.674	0.22	0.67	Significant
No	2	3						C
Maternal Eclampsia								Not
Yes	5	4	1.313	.308	5.598	0.136	.713	Significant
No	20	21						0
Cousin marriage								Not
Yes	5	3	1.833	.387	.8674	.595	.440	Significant
No	20	22						C
Maternal disease or								Not
complication								Significant
Yes	8	4	2.47	.63	9.62	1.75	.854	C
No	17	21						
Medicine taken during								
pregnancy								
Yes	17	10	3.188	.99	10.17	3.94	0.03	Significant
No	8	15						0
Physical problem								
Yes	11	3	5.76	1.36	24.36	6.34	0.01	Significant
No	14	22						
Maternal Hypertension								
Yes								
No	17	4	11.15	2.86	43.46	13.87	.000	Significant
	8	21						
Maternal Diabetes								
Yes	23	0	13.5	3.55	51.227	42.593	.000	Significant
No	2	25						
Birth weight								
1500 gm. or less or	20	2						
More than 1500 gm	5	23	46.00	8.027	263.62	26.299	.00	Significant
Child crying								
Before 30 min	12	23						
After 30 min	13	2	0.80	.016	.416	11.52	.01	Significant
	1	I	I	I	I	I	I	

 Table 3: Risk factors of antenatal and Intrapartum factors

Birth asphyxia								
Yes	9	0	-	-	-			
No	16	25				10.976	0.01	Significant
Neonatal seizure								
Yes								
No	15	0	-	-	-			
	10	25				21.42	0.00	Significant
Illness or infection child								
Yes								
No	16	8	3.778	1.170	12.194	5.128	0.02	Significant
	9	17						
High risk								
Pregnancy(Miscarriage)								
Yes	21	15	3.5	0.921	13.307	3.571	0.59	Not
No	4	10						Significant
Head injury								
Yes	17	9	3.78	1.170	12.194	5.128	0.01	Significant
No	8	16						

Maternal factors:

Antenatal care and cerebral palsy: Odds ratio of antenatal care was found to be 1.83 suggesting that cerebral palsy is 1.83 higher among mothers who had taking antenatal care compared to control group. The confidence interval of odds ratio was ranging from 0.387 to 8.674 which span 1 indicating that this odd was not significant. It was also supported by the results found in association analysis. In the association test using chi-square, the value was 0.22 which indicates among variables was not significant because p>0.05.

High risk Pregnancy(**Miscarriage**): Odds ratio of high risk pregnancy (miscarriage) was found to be 3.5 suggesting that cerebral palsy is 3.5 higher among mothers who had high risk pregnancy (Miscarriage and still birth) compared to control group. The

confidence interval of odds ratio was ranging from 0.921 to 13.307 which do span 1 indicating that this odds was not significant. It was also supported by the results found in association analysis. In the association test using chi-square, the value was 3.571 which indicates among variables was not significant because p>0.05.

Medicine taken during pregnancy: Odds ratio of medicine taken during pregnancy was found to be 3.188 which indicating that cerebral palsy is 3.188 times more frequent among those who taken antenatal medicine during pregnancy. The confidence interval of odds ratio was ranging from 0.99 to 10.17 indicating that this association was not significant. There is a relationship between having taken medicine during pregnancy and causing cerebral palsy which is statistically significant (p<0.05).

Neonatal seizure: There is a strong relationship between having convulsion disorder of child after birth and causing cerebral palsy which is statistically significant (p<0.05).

Maternal eclampsia and cerebral palsy:

Odds ratio of Maternal Eclampsia was found to be 1.313 which indicating that cerebral palsy is 1.313 highly frequent among those mother who had eclampsia compared with non eclampsia group (table -1). The confidence interval of odds ratio was ranging from 0.308 to 5.598 which spans 1 indicating that odds of CP was not reach statistical significance.

Cousin marriage: Odds ratio of cousin marriage was found to be 1.833 which indicating that cerebral palsy is 1.833% more frequent among those who had cousin marriage. The confidence interval of odds ratio was ranging from 0.387 to 0.8674 indicating that this association was significant.

Maternal disease or complication: Odds ratio of maternal disease or complication was found to be 2.47 which indicating that cerebral palsy is 2.47 times highly frequent among those who had maternal diseases. The confidence interval of odds ratio was ranging from 0.63 to 9.62 indicating that this association was not significant.

Physical problem: Odds ratio of physical problem was found to be 5.76 which indicating that cerebral palsy is 5.76 times highly frequent among those who had physical problem during pregnancy. The confidence interval of odds ratio was ranging from 1.36 to 24.36 indicating that this association was not significant. But here chi square is 6.34 which indicates that there is a relationship between having physical problem during pregnancy and causing cerebral palsy which is statistically significant (p<0.05).

Maternal hypertension: Odds ratio of maternal hypertension was found to be 11.15 suggesting that cerebral palsy is 11.15 higher among mothers who had hypertension compared to control group. The confidence interval of odds ratio was ranging from 2.86 to 43.46 which do not span 1 indicating that this odds was significant. It was also supported by the results found in association analysis. In the association test using chi-square, the value was 13.87 which indicates among variables was significant because p<0.05.

Maternal diabetes: Odds ratio of maternal diabetes was 13.5 indicating that cerebral palsy was 13.5 times more chance among those mothers who had diabetes. The confidence interval of odds ratio was ranging from 3.55 to 51.22 indicating that it was significant and also chi square test was 42.593 and it was also significant (p<0.05).

Birth asphyxia: There is a strong relationship between having child birth asphyxia after birth and causing cerebral palsy which is statistically significant (p<0.05).

Intrapartum factors:

Birth weight: Birth weight has been identified as a risk factor of cerebral palsy (OR=46.00) which means there is 46 times greater chance of having cerebral palsy among those birth weight(1500 gm or less) child compared to those who have weight 1500 gm or more than. There is a relationship between having child weight after birth and causing cerebral palsy which is statistically significant (p<0.05).

Child crying: Child crying has been identified as a risk factor of cerebral palsy (OR=.80) which means there is 0.80 times greater chance of having cerebral palsy among those child (crying before 30 minutes or crying after 30 minutes). There is a relationship between having child crying after birth and causing cerebral palsy which is statistically significant (p<0.05).

Illness or infection of child after birth: It has been identified as a risk factor of cerebral palsy (OR=3.778) which means there is 3.778 times greater chance of having cerebral palsy among those child. The confidence interval of odds ratio was ranging from 1.170 to 12.194 indicating that this association was not significant.

Head injury: Odds ratio of head injury was found to be 3.78 suggesting that cerebral palsy is 3.78 higher among children who had head injury compared to control group. The confidence interval of odds ratio was ranging from 1.170 to 12.194 which do span 1 indicating that this odds was not significant. It was also supported by the results found in association analysis. In the association test using chi-square, the value was 5.128 which indicates among variables was significant because p<0.05.

CHAPTER-V

Several antenatal, intrapartum, and neonatal factors investigated in this study of term and preterm and very preterm babies were associated with an increased risk of CP. According to the results of the present study, maternal diabetes, maternal hypertension, perinatal asphyxia, and high risk pregnancy were independent factors that correlated with CP in term and near-term new-borns. In developing countries, 4 to 9 million infants experience birth asphyxia annually. There are 1 million neonatal deaths attributed to birth asphyxia each year, which comprises 20%–40% of all neonatal deaths.

The increased risk of CP among offspring of women over the age of 35 years in our study was significant compared with offspring of women aged 18 to 35. The increased risk of CP in this group might be related to changes in uterine function seen with advancing age and the state of high risk pregnancy and its multiple covariates.

It was also observed that majority of the mother of cerebral palsy respondents completed at least primary education 46% (23) as well as their low educational status most commonly occur cerebral palsy. Only 4% (2) participants had completed masters education.

In this study maternal hypertension was closely related to cause cerebral palsy, here odds ratio was 11.15 which were significant (95% CI-2.86 to 43.46). Chi square was 13.87 which was also significant (p<0.05).

Study shows that odds ratio of maternal eclampsia was 1.313 which was not significant (95% CI-0.308 to 5.598) to cause cerebral palsy.

Wilson-costello, et al. (1998) stated that maternal age, maternal disease and preeclampsia have not been associated with CP. This result confirms these findings. In contrast, hypertensive disease and pre-eclampsia have been found to be associated with a reduced risk of CP (Jacobson, et al., 2002).

In case of maternal diabetes and causing cerebral palsy it indicates odds ratio was 13.5(95% CI, 3.55-51.22) which was significant and also chi square test was 42.593 and it was also significant (p<0.05).So this study proved that there was strong relationship between maternal diabetes and causing cerebral palsy.

In antenatal period mother was taking medicine 51.9 % (n=27) here mean as 1.46 (\pm .503). Sometime medicine can affect the child to develop cerebral palsy.

Miss use of drug can follow the role of cerebral palsy child and sometime born congenital type of cerebral palsy child.

Association between developing cerebral palsy and having amniotic fluid discharge:

Study demonstrated that among 50 mothers having cerebral palsy it was identified 19% (n=10) mothers were high risk pregnancy as they had miscarriages occur early in pregnancy. Study showed that odds ratio was 1 (95% CI 0.25-3.998), it indicates having miscarriages had no causal effect of developing cerebral palsy.

Erkin, et al.(2008) stated that antenatal and intrapartum risk factors for CP in very preterm babies found a strong association between maternal infection and, in particular, chorioamnionitis and an increased risk of CP. Maternal infection followed by neonatal sepsis was strongly associated with CP in preterm babies. However, in our study we did not find this correlation in the evaluated population.

Neonatal seizure and cerebral palsy:

Relationship between neonatal seizure and having cerebral palsy showed that Chi square was 21.429 which was significant (p<0.05). In the present study spastic

hemiplegic, spastic diplegia, and tetraplegia occurred significantly more frequently in the term group then in the preterm and very preterm groups.

Neonatal head injury and cerebral palsy:

Neonatal head injury of the participants were (OR= 3.778) and mean 1.48 (n= 26) whereas std. deviation (\pm .1.00), chi-square (<0.05). Neonatal head injury was the 3 times risk factor occurring cerebral palsy. Most commonly neonatal jaundice occurring region of poor delivery. In developing country most of the delivery had done at home by some uneducated person and causing cerebral palsy which was statistically significant (p<0.05).

Perinatal asphyxia and cerebral palsy:

Perinatal asphyxia, maternal age >35 years and high risk pregnancy were independent factors that correlated with CP in term and near-term new-borns. In developing countries, 4 to 9 million infants experience birth asphyxia annually (Soleimani, et al., 2013). Study stated that chi square of birth asphyxia was 10.97 and p value < 0.05, which indicated it was significant of developing cerebral palsy owing to birth asphyxia after child birth. In developing countries, 4 to 9 million infants experience birth asphyxia, annually (World Health Organization, 1998).

Majority of the respondents of this study was female but earlier studies also found that female are more affected with cerebral palsy (Hung, et al., 2007) which showed similarities from our findings. This might be because this was a hospital based study and females seek less care than male in least developing countries like Bangladesh (Khan and Rahman, 2000).In another study, Romeo et al. (2011) study showed that boys are most commonly affected and the percentages were 54% in cerebral palsy. Soleimani, et al. (2013) studies proved that low birth weight and IUGR were

significantly associated with the development of CP in Univariate analysis; however

this study make a correlation as independent risk factors. This might be due to the fact that low birth weight and IUGR are background risks for induction of perinatal insults such as birth asphyxia, followed by CP. In this study followed odds ratio 8.027 where it means that eight times risk factor cerebral palsy child born below 1500 mg weight. Perinatal asphyxia, mother's age, and any pathology during pregnancy are independent factors associated with CP in term new-borns.

Association between developing cerebral palsy having medication during antenatal period:

Majority of the respondents of this study was developing cerebral palsy and maternal eclampsia's chi-square was 0.136 and p value <0.05 that was indicated it was not significant and which showed similarities from one study findings (Hung, et al., 2007).

In this study, preterm labour, caesarean section and low birth weight were associated with the increased risk of CP. These results were not consistent with previous studies. Jacobson, et al. (2002) found that no association between caesarean section and Cerebral palsy. The association between low birth weight and CP has also been reported by others.

The idea of this study was to identify the risk factors of cerebral palsy which might help to explore the underlying mechanism of cerebral palsy. This study found that the average age of the incidence of the cerebral palsy was $1.56 (\pm 0.50)$.

Previous studies of antenatal and intrapartum risk factors for CP in very preterm babies found a strong association between maternal infection and in particular, chorioamnionitis and an increased risk of CP (Wu and colford, 2000). Maternal infection followed by neonatal sepsis was strongly associated with CP in preterm babies. In one study found that mothers with a history of fever during labor had a nine times greater risk of having newborns with CP. Therefore, the current study supports several previous studies showing that maternal infection at the time of delivery is an important risk factor for CP (Ali and Adam 2010).

However, in this study did not find that about 23% mothers have maternal disease during pregnancy. The idea of this study was to identify the risk factors of cerebral palsy which might help to explore the underlying mechanism of cerebral palsy. Several antenatal, intrapartum and neonatal factors investigated in this study of term and preterm and very preterm babies were associated with in an increased risk of CP. It has been well established that both the preterm infant and low birth weight infant are at particular risk for CP (Saadi, et al., 2012). We found that children of low birth weight and those with low or high length or small or large head circumstances at birth were at increased risk of Cerebral palsy.

The excess risk of CP in the lowest birth weight group in our study is consistent with the results for 105 children born at term in the Australian study. Jarvis, et al. (2003) included a total of 4503 children with CP, and reported an inverse J-shaped association between birth weight and CP for children born at term when they used customized growth charts. However, when they used conventional growth standards they found that excess risk of CP was associated only with low birth weight and not with high birth weight. Our results are consistent with this finding. Poor education and poor nutrition taken that can burden of cerebral palsy child. Mother ages are the most important factor for CP child born.

5.1 Limitation of the Study:

The main limitation of this study was its small sample size and retrospective in nature. The study was conducted with 50 cerebral palsy children for identifying antenatal and intrapartum risk factors for developing cerebral palsy which was a small number of samples and was not sufficient enough for the study to generalize the wider population of this condition. It was limited by the fact that it was not possible to measure head or arm circumference as after child birth not detecting as cerebral palsy. There was no available research done in this area in Bangladesh and worldwide. So, relevant information about with antenatal and intrapartum risk factors for Bangladesh was very limited in this study. There were some limitations in the study which was not possible to overcome, it was necessary to design the study within the situation. Another limitation of this study was its short duration. The investigator had resource and time constraint to conduct a cohort or other experimental study. Data were collected just from one centre, so the result might lack generalized ability.

CHAPTAR - VI CONCLUSION AND RECOMMENDATION

6.1 Conclusion

This study showed that, perinatal asphyxia, child birth weight, delayed crying and any pathology during pregnancy were independent factors associated with CP in term newborns. We found that children of low birth weight and those with low or high length or small or large head circumferences at birth were at increased risk of CP whereas reduced size measurements were mainly associated with the spastic unilateral and diplegic CP subtypes, large size at birth was particularly associated with the spastic quadriplegic and the dyskinetic CP subtypes. The findings were consistent with the notion that the most frequent causes of CP were adverse antenatal events, followed by restricted foetal growth, while large infants may be more vulnerable to adverse intrapartum events. Previous studies had suggested that improving maternal care improves neonatal outcome. However the extent to which preventing or treating these and other risk factors would reduce the incidence of CP in new-borns is unknown and merits for further study.

6.2 Recommendation

Further multicentre and different geographical region with larger sample size is recommended to assess the antenatal as well as intrapartum risk factors for developing cerebral palsy children in Bangladesh.

REFERENCES

Ackerman, P., Thormann, M.S., and Huq, S., (2005). Assessment of educational needs of disabled children in Bangladesh.Washington : USAID. Available at: http://www.beps.Net/publications/Bangladesh_disbled_children_report040605.pdf [accessed on 12 August 2015].

Ali, A.A., & Adam, I. (2010). Maternal and perinatal outcomes of obstructed labour in kassala hospital Sudan. *Journal of Obstetrica & Gynaecology*, 30(4), pp.376–7.

Allen, D.G., Lamb, G.D., and Westerblad, H., (2008). Skeletal muscle fatigue: cellular mechanisms. *Physiological Reviews*, 88(1), pp. 287-332.

American Pregnancy Association, (2013).Cerebral palsy, [online]. USA: American Pregnancy Association. Available at: http:// american pregnancy. Org/ birth defect/ cerebral palsy.htm[Accessed on 7 September 2015].

Amin, M. R., Rahman, S., Saha, N., Hossain, M. S., Islam, M. J., Ahmed, M., Chakraborty, P. K., Islam, F. A., (2015). Role of Baclofen in Combination with Intensive Rehabilitation in Spastic Cerebral Palsy. *Journal of National Institute of Neurosciences Bangladesh*, 1 (1), pp. 2410-8030.

Andersen, G.L., Irgens, L.M., Haagaas, I., Skranes, J.S., Meberg, A.E., Vik, T. (2008). Cerebral palsy in Norway: prevalence, subtypes and severity. *Europian Journal of Peadiatric & Neurology.*, 12, pp. 4-13.

Aneja, S. (2006).Evaluation of a child with cerebral palsy. *The Indian Journal of Pediatric*, 71(2),pp. 627-634.

Bangash, A. S., Hanafi, M. Z., Idrees, R., and Zehra, N. (2014). Risk factors and types of cerebral palsy. *Journal of Pakistan Medical Association*. 64, p.103.

Bax, M.C., Flodmark, O., Tydeman, C. (2007). Definition and classification of cerebral palsy: from syndrome toward disease. *Developmental Medicine and Child Neurology Supplement*.109, pp. 39 – 41.

Behrman, E.R., (2004), Nelson text book of paediatrics, 17thed, London: Churchill Livingstone.

Bell, K.L., Boyd, R.N., Tweedy, S.M., Weir, K.A., Stevenson, R.D., and Davies, P.S., (2010). A Prospective, longitudinal study of growth, nutrition and sedentary behavior in young children with cerebral palsy. *Bio Med Central Public Health*, 10,pp.179.

Bialik, G.M., and Givon, U. (2009). Cerebral palsy: classification and etiology, *Acta Orthopaedica Traumatologica Turcica*,43(2),pp.77-80.

Bloemsaat, J.G., Meulenbroek, R.G., and Van Galen, G.P., (2005). Differential effects of mental load on proximal and distal arm muscle activity. *Experimental Brain Research*, 167(4), pp. 622-634.

Canning, B.J., (2006). Reflex regulation of airway smooth muscle tone. *Journal of Applied Physiology*, 101(3), pp. 971-985.

Cans, C. (2000). Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. *Developmental Medicine & Child Neurology*, 42, pp. 816–24.

Center for Disease Control and Prevention, (2012).Cerebral Palsy, [Online]. USA: Center for Disease Control and Prevention. Available: http://www.cdc.gov/ncbddd/cp/facts.html [Accessed on 19 October 2015].

Cerebral Palsy Alliance, (2015).Treatment for cerebral palsy (Interventions for Cerebral Palsy). Available: https://www.cerebralpalsy.org.au/what-is-cerebral-palsy/interventions/ [Accessed on18 October 2015].

Chang, H.Y., Chou, K.Y., Lin, J.J., Lin, C.F., and Wang, C.H., (2010). Immediate effect of forearm Kinesio taping on maximal grip strength and force sense in healthy collegiate athletes. *Physical Therapy in Sport*, 11(4),pp. 122-127.

Chen, C.M. Hsu, H.C., Chen, C.L., Chung, C.Y., Chen, K.H., and Liaw, M.Y., (2013). Predictors for changes in various developmental outcomes of children with cerebral palsy. *Research in Developmental Disabilities*, 34, pp.3867-3874.

Children's Hospital Colorado, (2013). Cerebral Palsy Overview, [Online]. USA: Children's Hospital Colorado. http://orthopedics.childrenscolorado.org/conditions/cerebral-palsy [Accessed on18 October 2015].

Chowdhury, M.A., (2005). Birth asphyxia and neurological sequelae: Paeditric Neurology: Services and research in Bangladesh, Shishu Bikash Network: Dhaka.

Costantine, M.M., How, H.Y., Coppage, K., Maxwell, R.A. and Sibai, B.M. (2007). Does peripartum infection increase the incidence of cerebral palsy in extremely low birthweight infants?*American Journal of Obstetrics and Gynecology*. 196(5), pp.6-8.

Damiano, D.J. (2004). Physiotherapy management in cerebral palsy: Moving beyond philosophies, Management of the Motor Disorders of Cerebral Palsy. *Clinician Developmental Medicine No. 161*, 2nd edn, MacKeith Press, London, pp.161-169.

Darsaklis, V., Snider, L., Majnemer, A., and Mazer, B., (2011). Predictive validity of Prechtl's Method on the qualitative assessment of general movements: a systematic review of the evidence. *Development Medicine and Child Neurology*, 53,pp.896–906.

Dashe, J.S., Mcintire, D.D., Lucas, M.J., Leveno, K.J. (2000). Effects of symmetric and asymmetric fetal growth on pregnancy outcomes.*Obstetic and Gynecology*; 96, pp.321–7.

Elkamil, A., Andersen, G.L., Hagglunad, G., Lamvik, T., Skranes, J., and Vik, T. (2011). Prevalence of hip dislocation among children with cerebral palsy in regions with and without a surveillance programme: a cross sectional study in Sweden and Norway. *Bio Med Central Musculoskeletal disorder*, 12,pp.284.

Erkin, G., Delialioglu, S.U., Ozel. S.(2008). Risk factors and clinical profiles inTurkish children with cerebral palsy: analysis of 625 cases. *International Journal of Rehabilitation and Research*, 31, pp. 89–91.

Faria, A.V., Hoon, A., Stachinko, E., Jiang, H., Mashayekh, A., Akhter, K., Hsu, J., Oishi, K., Zhang, J., Miller, M.I., and Mori, S. (2011). Quantitative Analysis of Brain Pathology Based on MRI and Brain Atlases-Applications for Cerebral Palsy. *Neuroimage*, 54(3), pp.1854-1861.

Faria, A.V., Hoon, A., Stachinko, E., Jiang, H., Mashayekh, A., Akhter, K., Hsu, J., Oishi, K., Zhang, J., Miller, M.I., and Mori, S., (2011). Quantitative Analysis of Brain Pathology Based on MRI and Brain Atlases - Applications for Cerebral Palsy. *Neurology Image*, 54(3), pp.1854-1861.

Ferriero, D.M. (2004). Neonatal brain injury. *The New England Journal of Medicine*.351, pp. 1985 – 1995.

Gershon, Z.T., Willoughby, W.M., Getz, R.D., and Smith, R.R., (2013). Cerebral Palsy signs and symptoms, [Online]. USA: Gershon, Willoughby, Getzand Smith,LLC.Available:http://www.cerebralpalsylawdoctor.com/symptoms/ [Accessed on 31August 2015].

Gilbert, W.M., Jacoby, B.N., Xing, G., Danielsen, B. and Smith, L.H. (2010). Adverse obstetric events are associ-ated with significant risk of cerebral palsy. *American Journal of Obstetrics & Gynecology*, 203,p. 328.

Glew, G.M. and Bennett, F.C. (2011).Cerebral palsy grown up. *Journal of Developmental & Behavioral Pediatrics*, 32, pp.469-475.

Gracies, J.M., (2005). Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. *Muscle & Nerve*, 31(5),pp. 535-551.

Hagberg, B., Hagberg, G., Beckung, E. (2001). Changing panorama of cerebral palsy in Sweden. Prevalence and origin in the birth year period 1991-94. *Acta Paediatrica*, 90,pp. 271-277.

Health Link, B.C, (2013). Cerebral Palsy, [Online]. Canada: Health wise. Available: http://www.healthlinkbc.ca/kb/content/major/aa56609. html [Accessed on 19 October 2015].

Hung, J. W., Wu, Y. H., Wu, W. C., Leong, C. and Lau, Y., (2007). Regional Survey of Assistive Devices Use by Children with Physical Disabilities in Southern Taiwan. *Chang Gung Medical Journal*, 30,pp.354-62.

Hurley, D.S., Moulton, T.S., Msall, M.E., Krosschell, K.J., and Dewald, J.P., (2011). The Cerebral palsy Research Registry: Development and Progress toward Collaboration in the United States. Journal of Child Neurology, 26(12), pp. 1534-1541.

Hustad, K.C., Gorton, K., and Lee, J., (2011). Classification of speech and language profile in 4years old children with cerebral palsy.*National Institute of Health Public Access Author Manuscript*, 53(6), pp.1496-1513.

Hwang, M., kuroda, M.M., Tann, B., and Gaebler-Spira, D.J., (2011). Measuring Care and Comfort in Children with Cerebral Palsy: The Care and Comfort Caregiver Questionnaire. The American Academy of Physical Medicine and Rehabilitation, 3(10), pp.912-919.

Iannelli, V., (2008). Cerebral Palsy pediatrics Basics, [Online]. USA: About.com.Available:http://pediatrics.about.com/od/cerebralpalsy/a/cerebral_palsy. htm [Accessed on 31 August 2015].

Jacobson, B., Hagberg, G., Hagberg, B.(2002). Cerebral palsy in preterm infants : a population–based case–control study of antenatal and intrapartial risk factors. *ActaPaediatrica*, 91, pp. 946–951.

Jarvis S, Glinianaia SV, Arnaud C. (2005). Case gender and severity in cerebral palsy varies with intrauterine growth. *Archive of Disease in Childhood*; 90, pp.474–9.

Kadhim, H., Sébire, G., Kahn, A., Evrard, P. and Dan, B. (2005). Causal mechanisms underlying periventricular leukomalacia and cerebral palsy. *CurrPediatrRev*.1, pp. 1-6.

Kassolik, K., Jaskolska, A., Kisiel-Sajewicz, K., Marusiak, J., Kawczynski, A., and Jaskolski, A., (2009). Tensegrity principle in massage demonstrated by electro-and mechanomyography. *Journal of body work and movement therapies*, 13(2), pp. 164-170.

Keynes, M. (2006). No Significant Improvement in Motor Function in Children with CP Following Intensive Physiotherapy or Hyperbaric Oxygen, Bobath Centre for Children with Cerebral Palsy.

Khan, M.R., and Rahman, M.E., (2000).Essence of Pediatrics. Anwara Khan, Dhaka. Krageloh-Mann I. (2008), Understanding causation of cerebral palsy by using magnetic resonance imaging. *Paediatric Child Health*.18, pp. 399 – 404.

Krageloh-Mann, I. (2008). Understanding causation of cerebral palsy by using magnetic resonance imaging. *Paediatric Child Health*.18, pp. 399 – 404.

Kułak, P., Maciorkowska, E., and Goscik E. (2014). Selected risk factors for spastic cerebral palsy in a retrospective hospital–based case control study. *Progress in Health Science*. 4 (2),pp. 7-13.

Lee, P.J., Rogers, E.L., and Granata, K.P., (2005). Active Trunk Stiffness Increases with Co-contraction. *Journal of Electromyography and Kinesiology*, 81(5),pp.460-465.

Majnemer, A. and Mazer, B.(2004). New directions in the outcome evaluation of children with cerebral palsy, *Semin Pediatric Neurology*, 11,p. 7

Mandal, A., (2013).Cerebral Palsy symptoms, [Online]. UK: News-Medical.net. Available: http://www.news-medical.net/health/Cerebral-Palsy-Symptoms.aspx [Accessed on 6 September 2015].

Marron, M., Redolar-Ripoll, E., Boixados, D., Nieto, M., Guillamon, R., Hernandez, N., Eulalia; Gómez, E. (2013). Burden on Caregivers of Children with Cerebral Palsy: Predictors and Related Factors. *Universitas Psychologica*, 12(3), pp.767-777.

McConachie, H., Smith, D., Bax, M. (2006). Services for children with disabilities in European countries.Conclusions and recommendations, *Devidson Medicine Child Neurology*, 39(76), pp. 5-7.

Mcintyre, S., Taitz, D., Keogh, J., Goldsmith, S., Badani, N., and Blair, E. (2012). A systematic review of risk factors for cerebral palsy in children born at term in developed countries. *Developmental Medicine & Child Neurology*. 12, pp. 499-503.

Melheim, K., Heimstad, R., Austgulen, R., Lydersen, S., Andersen, G.L., Irgens, L.M., and Vik, T., (2013). Mediators of the association between pre-eclampsia and cerebral palsy: population based cohort study. *Bio Medical Journal*, 10, p.1136.

Morris, C., (2007). The Definition and Classification of Cerebral Palsy. UK: Department of Public Health.

My child, (2013).Signs and symptoms of Cerebral Palsy, [Online]. USA: Stern Law Group, PLLC. Available: http://cerebralpalsy.org/about-cerebral-palsy/symptoms/ [Accessed on 7 September 2015].

O'Callaghan, M.E., MacLennan, A.H., Gibson, C.S., McMichael, G.L., Haan, E.A., Broadbent, J.L., Goldwater, P.N., Dekker, G.A. (2011). Australian Collaborative Cerebral Palsy Research Group: Epidemiologic associations with cerebral palsy. *Archives of disease in childhood-feotal and neonetal edition*, 118(3),pp.576–582.

Physician and Nurses, (2013). Information for parents of Children with Cerebral Palsy, [Online]. USA: Cerebral Palsy Injury.com – Med Law Legal Team of Janet, Jenner & Suggs, Attorneys at Law Medical Malpractice, Pain Pump Lawyers, Birth Injury Attorneys, Physician / Lawyers - Site by Consultwebs.com, Inc. Available: http://www.cerebral-palsy-injury.com/treatment-of-cerebral palsy.html [Accessed on18 October 2015].

Prado, L.G., Makarenko, I., Andresen, C., Kruger, M., Opitz, C.A., and Linke, W.A., (2005).Isoform diversity of giant proteins in relation to passive and active contractile properties of rabbit skeletal muscles. *The Journal of general physiology*, 126(5), pp. 461-480.

Reddihough, D.S., Collins, K.J. (2003). Epidemiology and causes of cerebral palsy. *Austrailan Journal of Physioteraphy*, 49,pp.7–12.

Romeo, D.M., Cioni, M., Battaglia, L.R., Palermo F, Mazzone D.(2011). Spectrum of gross motor and cognitive functions in children with cerebral palsy: Gender differences. *Europeon Journal of Paediatric Neurology*.15, pp.53 – 58.

Rosenbaum, P., Paneth, N., and Leviton, A. (2007). The definition and classification of cerebral palsy. *Developmental Medicine in Child Neurology*, 49,pp. 8–14.

Russell, S.K., Martha, S.W., Kim, V.N.B., Nancy, S.D., Carrie, L.A., Ruth, E.B., Beverly.M., Maureen, S.D., Robert, T.F., Matthew, J., Jean, A.P., and Marshalyn, Y.A. (2011).Prevalence and functioning of children with cerebral palsy in four areas of the United States in 2006. *Research in Developmental Disabilities*. 32(2),pp. 462-469.

Saadi, H.R., Sutan, R., Dhaher, A.M., Alshaham, S.A. (2012). Maternal and foetal risk factors of cerebral palsy among Iraqi children: A case control study. *Open Journal of Preventive Medicine*, 2(3), pp.350-358.

Sankar, C., and Mundkur, N., (2005). Cerebral Palsy–Definition, Classification, Etiology and Early Diagnosis. *Indian Journal of Pediatrics*, 72 (10), pp. 865-868.

Serdaroglu, A., Cansu, A., and Tezcan, S., (2006). Prevalence of cerebral palsy in Turkish children between the ages of 2 and 16 years. *Developmental Medicine & Child Neurology*, 48(6),pp. 413-416.

Soleimani F, Vameghi R, Hemmati S, Salman Roghani R.,(2009). Perinatal and neonatal risk factors for neurodevelopmental outcome in infants in Karaj. *Archive of Iranian Medicine*,12,pp.135 – 139.

Soleimani, F., Vameghi R, Biglarian A. (2013). Antenatal and Intrapartum Risk Factors for Cerebral Palsy in Term and Near-term Newborns. *Archieve of Iranian Medicine*, 16(4), pp. 213 – 216.

Stephens, B. and Vohr, B.R., (2009). Neurodevelopmental outcome of the premature infant. *Pediatric Clinic of North America*, 56, pp. 631–46.

Stephens, B. and Vohr, B.R., (2009). Neurodevelopmental outcome of the premature infant. *Pediatric Clinical North American*, 56,pp.631–646.

Stevens, R.D., Marshall, S.A., Cornblath, D.R., Hoke, A., Needham, D.M., de Jonghe, B., Ali, N.A., and Sharshar, T., (2009). A framework for diagnosing and classifying intensive care unit-acquired weakness. *Critical Care Medicine*, 37(10), pp. 299-308.

Stoknes, M., Andersen, G.L., Dahlseng, M.O.(2012).Cerebral palsy and neonatal death in term singletons born small for gestational age. *Pediatrics*, 130, pp.1629–35.

Tabib, S. M. S. B., (2009). Prevalence of childhood disabilities and cerebral palsy in the community. Available: http://www.jldd.jp/gtid/acmr_19/pdf/57.pdf [accessed on 26 December 2015]

Tan, R.Y.L., Neligan, A., and Shorvon, S.D., (2010). The uncommon causes of status epilepticus: a systematic review. *Epilepsy Research*, 91(2), pp. 111-122.

Tanner, M. and Harpham, T., (2013).Urban health in developing countries: progress and prospects, 2nd ed., New York: Earthscan.

Tatla, S.K., Sauve, K., Virji-Babul, N., Holsti, L., Butler, C., and Loos, H.F. M., (2013). Evidence for outcomes of motivational rehabilitation interventions for children and adolescents with cerebral palsy: an American Academy for Cerebral Palsy and Developmental Medicine systematic review. *Developmental Medicine & Child Neurology*, 55(7), pp.593-601.

The Ontario Federation for Cerebral Palsy, (2011).A Guide to Cerebral Palsy. Available: http://www.waikatodhb.health.nz/assets/for-health-professionals/Primarycare-management-guidelines/Cerebral-Palsy-Clinical-Practice-Guideline.pdf [Accessed on 19 October 2015]

Thorngren, J. K. and Herbst, A. (2006). Perinatal factors associated with cerebral palsy in children born in Sweden. *Obstetrics & Gynecology*, 108(6), pp.1499-1505.

Thorogood, C., and Alexander, M. A., (2013). Rehabilitation and Cerebral Palsy. Available: http://emedicine.medscape.com/article/310740-overview#a3 [Accessed on 19 October 2015]

Valle, F., Sandal, M., and Samori, B., (2007). The interplay between chemistry and mechanics in the transduction of a mechanical signal into a biochemical function. *Physics of Life Reviews*, 4(3), pp. 157-188.

Wilson-Costello, D., Borawski, E., Friedman, H.(1998). Perinatal correlates of cerebral palsy and other neurologic impairment among very low birth weight children. *Pediatrics*, 102, pp. 315–322.

Wind horst, U., (2007). Muscle proprioceptive feedback and spinal networks. *Brain Research Bulletin*, 73(4),pp. 155-202.

Wolraich, M., Droter, D., Dworkin, P. and Perrin, E. (2008).Developmentalbehavioral pediatrics. *Evidence and Practice*, 14, pp.483-517.

World Health Organization: World Health Report 1998: Life in the twenty first century: A vision for all. Geneva. .

Wu, Y.W., Colford, J.M., Jr. (2000). Chorioamnionitis as a risk factor for cerebral palsy: A meta-analysis. *JAMA*, 284, pp.1417-24.

APPENDIX

- 1. Consent form (English)
- 2. Questionnaire (English)
- 3. Consent form (Bangla)
- 4. Questionnaire (Bangla)
- 5. Permission Letter

Consent Form

Assalamualaikum,

I am Md. Obaidul Haque, Final part M.Sc.in Physiotherapy student of Bangladesh Health Professions Institute (BHPI) under the Faculty of Medicine, University of Dhaka. To obtain Masters Degree, I have to conduct a research and it is a part of my study. The participants are requested to participate in the study after briefing the following.

My research title is "Antenatal and Intrapartum Risk Factors of Children with Cerebral Palsy". Through this study I will find the Antenatal and Intrapartum Risk Factors of term and near term born Children with Cerebral Palsy. If I can complete this study successfully, patients may get benefits who are suffering from Cerebral Palsy.

To fulfill my research project, I need to collect data. So, you can be a respected participant of this research. I want to meet you a couple of sessions, during your regular therapy schedule.

I would like to inform you that this is a purely academic study and will not be used for any other purposes. I assure that all data will be kept confidential. Your participation will be voluntary. You may have the rights to withdraw consent and discontinue your participation at any time of the study. You have the rights to reject any particular question that you don't like.

If you have any query about the study as a participant, you may contact with me (Md. Obaidul Haque) Department of Physiotherapy, BHPI, CPR, Savar, Dhaka-1343. Do you have any questions before I start?

So, may I have your consent to proceed with the interview?

Yes No

Signature of the mother and date Signature of the researcher and Date...... Signature of the witness and Date.....

Research Questionnaire

Antenatal and Intrapartum Risk Factors for Children with Cerebral Palsy.

Respondent Name:	Children's Age:
Date of the interview:	Father's Age: Mother's Age:
Address:	Child diagnosed by:
Mobile no:	

Part-1: Socio Demographic Information

Patient ID:

Question	Questions/ Information on	Coding Category
Number		
1	Sex	Male=1,Female=2
2	Mother's Educational level	Illiterate=1,Literate=2,Primary=3,SSC=4,HSC=5, Graduation=6, Masters and Above=7
3	Have you got cousin marriage?	Yes=1, No=2
4	What was your age during birth of this child	18-22 year=1; 22-26 year=2; 26-30 year=3; 30-35 year=4
5	What was your area of living	Urban=1; Rural=2

Question	Questions/ Information on	Coding Category
Number		
1	Did you take antenatal care	Yes=1, No=2
	during pregnancy?	
2	If yes, which type of care	Antenatal=1, Natal=2, Post natal=3, All=4, None
	you had taken.	of these=5
3	Did you suffer any	Yes=1, No=2
	disease/infection/complicati	
	on during pregnancy?	
4	Did you take any medicine	Yes=1, No=2
	during pregnancy?	
5	If yes, which type of	Allopathy=1, Homeopathy=2, Kabiraj=3
	medical treatment?	
6	Did you get any physical	Yes=1, No=2
	assault during pregnancy?	
7	If yes, then which type?	Fall down=1, Lifting heavy object=2, Others=3
8	Deliver of birth is attended	Doctor=1, Nurse=2, Midwife=3
	by-	
9	In which place your child	Home=1, Hospital=2, Clinic =3
	was born?	
10	What was your child's birth	Premature=1, Term=2, Post term=3
	history?	
11	What was about your labour	Prolonged labour (> 10 hr) =1, Short labour
	period?	(2-3 hr)=2, Sudden birth(few mins)=3

Part- 2: Antenatal Information

Question	Questions/ Information on	Coding Category		
Number				
1	Did you have any complications during	Bleeding=1,		
	delivery?	Prolonged de	elivery=2,	
		Breech prese	entation=3,	
		Mothers Ecc	lampsia=4,	
		Epilepsy=5,		
		Meconium st	taining=6,	
		None=7.		
2	Did your child get any head injury after	Yes=1	No=2	
	delivery?			
3	Did your child get any birth asphyxia?	Yes=1	No=2	
4	If yes, what was time until baby cried?	Less than ten minute=1		
		More than te	n minute=2	
5	What was your child's weight after	Didn't know=1, 2.5 kg = 2,		
	birth?(kg/lb)	Below 2 kg= 3, Above 3		
		kg=4		
6	Did your child get any disease after	Genetic disorder=1,		
	birth?	Rh disease=2	2, Jaundice=3,	
		Convulsion=	-4,	
		Pneumonia=5,		
		Diarrhoea=6, Fever=7, Post		
		natal infection or		
		trauma=8, TORCH		
		etiology=9, Others=10		
7	After birth which treatment did you take	Physician = 1		
	for this child?	Physiotherapy = 2		

Part- 3: Intrapartum Information

Part-4: Other relevant information

1.	Have you	had 2 or more miscarriages or a stillborn child?	Yes	No
2.	Do you or	your partner have any birth defects, mental		
	Retardatio	n or learning problems?	Yes	No
3.	Has anyon	e in your family or your partner's family ever had:		
	A. Dowr	a syndrome?	Yes	No
	B. Spina	bifida or Neural tube defects and/or Anencephaly?	Yes	No
	C. Hydro	ocephalus?	Yes	No
	D. Heart	defect present at birth?	Yes	No
	E. Menta	al retardation or learning problems?	Yes	No
	F. Hemo	philia or other blood disorders?	Yes	No
	G. Musc	ular dystrophy?	Yes	No
	H. Cysti	c fibrosis?	Yes	No
	Metal	polic/chemical disorder or need for a special diet?		
	I. (ex. P	KU)	Yes	No
	J. Other	genetic or inherited condition not listed here?	Yes	No
4.	Do you ha	ve thalassemia?	Yes	No
5.	Do you ha	ve Tay- Sach's Disease?	Yes	No
6.	Do you ha	ve any siblings child which is CP?	Yes	No
7.	During thi	s pregnancy have you:		
	A. Dranl	alcohol?	Yes	No
	B. Smok	ed cigarettes?	Yes	No
	C. Taker	any medication?	Yes	No
	D. Used	any "recreational" street drugs?	Yes	No
	E. Had a	ny X-rays? (including dye studies, CAT scan)	Yes	No
	F. Had a	ny illnesses, infections, rash or fever greater	Yes	No
	than 1	01degree for 2 or more days?		
	G. Had c	contact at home or at work with cats, cat litter, mice	Yes	No
	or ha	nsters?		
	H. Been	exposed to chemical or toxic substances?	Yes	No
8.	Have you	had diabetes during pregnancy?	Yes	No
9.	Have you	had hypertension during pregnancy?	Yes	No

10.	Have you had high blood sugar during this pregnancy	Yes	No
	or during a previous pregnancy?		
11.	Do you have any seizure disorder that requires medication?	Yes	No
12.	Do you feel threatened at home either physically or verbally?	Yes	No
13.	Did your child cry after birth?	Yes	No
14.	How long duration?	Before 30 minutes	After 30 minutes
15.	Did your child had birth asphyxia after birth?	Yes	No
16.	Did your child had neonatal seizure after birth?	Yes	No
17.	Did your child had other problem after birth?	Yes	No
18.	What was your child's weight after birth?	Yes	No

Yes=1, No=2

সম্মতিপত্র

আসসালামুয়ালাইকুম, আমি মোহাঃ ওবায়দুল হক, ঢাকা বিশ্ববিদ্যালয়ের চিকিৎসা অনুষদের অধিভুক্ত বাংলাদেশ হেলথ প্রফেশনস্ ইন্সিটিটিউট এর এম.এস.সি ইন ফিজিওথেরাপি কোর্সের চূড়ান্ত বর্ষের একজন শিক্ষার্থী। অধ্যায়নের অংশ হিসেবে আমাকে একটি গবেষণা সম্পাদন করতে হবে এবং এটা আমার প্রাতিষ্ঠানিক কাজের একটা অংশ। নিম্নোক্ত তথ্যাদি পাঠ করার পর অংশগ্রহণকারীদের গবেষণায় অংশগ্রহনের জন্য অনুরোধ করা হলো।

আমার গবেষণার বিষয় হল **"সেরিব্রাল পালসি সম্বলিত বাচ্চাদের জন্মপূর্ব এবং জন্মের সময়কার ঝুঁকি** "এই গবেষণার মাধ্যমে সেরিব্রাল পালসি সম্বলিত বাচ্চাদের জন্মপূর্ব এবং জন্মের সময়কার ঝুঁকিসমুহ নির্ণয় করব। আমি যদি আমার গবেষণাটি সার্থকভাবে সম্পূর্ণ করতে পারি তবে যেসব রোগীরা সেরিব্রাল পালসি রোগে ভুগছেন তারা উপকৃত হবেন এবং এটি হবে একটি পরীক্ষামূলক প্রমাণ। গবেষণাটি সম্পাদনের জন্য, আমার তথ্য সংগ্রহ করা প্রয়োজন হবে। গবেষণার ক্ষেত্র বিবেচনা করে আপনার মাঝে আমার গবেষণায় অংশগ্রহণ করার জন্য প্রয়োজনীয় বৈশিষ্ট্য লক্ষ্য করা গেছে। এজন্য, আপনি আমার গবেষণার একজন সম্মানিত অংশগ্রহণকারী হতে পারেন এবং আমি আপনাকে আমার গবেষণায় অংশগ্রহন করতে অনুরোধ

জানাচ্ছি|

আমি প্রতিজ্ঞা করছি যে,এই গবেষণা আপনার জন্য ঝুঁকিপূর্ণ হবে না অথবা আপনার কোন ক্ষতি করবে না। গবেষণা চলাকলীন সময়ে কোন রকম দ্বিধা বা ঝুঁকি ছাড়াই যেকোন সময়ে আপনি এটাকে বাদ দিতে পারবেন। এই গবেষণার প্রাপ্ত তথ্য সম্পূর্ণভাবে গোপনীয় থাকবে এবং অংশগ্রহণকারীর ব্যক্তিগত তথ্য অন্য কোথাও প্রকাশ করা হবে না। যদি কোনও প্রশ্ন আপনার পছন্দ না হয় তাহলে ওই প্রশ্নের উত্তর না দেয়ার অধিকার আছে।

আপনার গবেষণা সম্পর্কে কোনো জিজ্ঞসা থাকে তবে আপনি অনুগ্রহপুবক যোগাযোগ করতে পারেন গবেষক মোহাঃ ওবায়দুল হক, ফিজিওথেরাপি বিভাগ, বিএইচপিআই, সিআরপি, সাভার, ঢাকা-১৩৪৩

গবেষণা শুরু করার আগে আপনার কি কোন প্রশ্ন আছে ?

আমি কি শুরু করতে পারি ?

হ্যাঁ না

অংশগ্রহণকারীর স্বাক্ষর ও তারিখ	
গবেষকের স্বাক্ষর ও তারিখ	
স্বাক্ষীর স্বাক্ষর ও তারিখ	

গবেষণা-প্রশ্নাবলী

সেরিব্রাল পালসি সম্বলিত বাচ্চাদের জন্মপূর্ব এবং জন্মের সময়কার ঝুঁকি

(গবেষণায় অংশগ্রহণকারী নম্বর:.... কেস নং......কন্ট্রোল নং.....)

বাচ্চার নামঃ	বাচ্চার বয়সঃ
সাক্ষাতের তারিখঃ	বাচ্চার বাবার বয়সঃ
ঠিকানাঃ	বাচ্চার মায়ের বয়সঃ
মোবাইল নং-	রোগ সনাক্তকারিনি,শিশু ডাক্তারঃ
আই ডিঃ	ফিজিওথেরাপিস্টের স্বাক্ষরঃ

বাচ্চার মায়ের স্বাক্ষরঃ

<u> পর্ব-১ : সামাজিক-বৈষয়িক তথ্যাবলী</u>

51	লিঙ্গঃ	ছেলে=১, মেয়ে=২
২	মায়ের শিক্ষাগত যোগ্যতা-	নিরক্ষর =১, স্বাক্ষরজ্ঞান =২, প্রাইমারি= ৩, এস এস সি =৪, এইচ এস সি =৫, ডিগ্রী = ৬, মাস্টার্স= ৭
৩।	আপনার কি আত্মীয়ের মধ্যে বিয়ে হয়েছে?	হাঁ = ১, না=২
81	আপনার বয়সকতছিলএইবাচ্চাজন্মেরসময়?	১৮২২ বছর =১, ২২-২৬ বছর=২,২৬-৩০ বছর=৩, ৩০-৩৫ বছর=৪,৩৫-৪০ বছর=৫
٥I	আপনি কোথায় বাস করেন ?	গ্রাম=১, শহর=২

পর্ব-২: জন্মপূর্ব

প্রশ্ননম্বর	প্রশ্ন	কোড
51	আপনি কি জন্মপূর্ববর্তী যত্ন নিয়ে ছিলেন?	হাঁ = ১, না=২
২৷	যদি হ্যাঁ হয়, তাহলে কি ধরনের যত্ন নিয়েছিলেন?	জন্মপূর্ববর্তী=১ ,জন্মকালীন=২, জন্মপরবর্তী =৩, সবগুলো=৪, একটিও নয় =৫
৩।	গর্ভাবস্থায় আপনি কি কোন রোগ বা সংক্রমণ বা জটিলতায় ভুগেছেন?	হাঁ = ১, না=২
81	গর্ভাবস্থায় আপনি কি কোন ওষুধ গ্রহণ করতেন?	হাঁ = ১, না=২
¢I	যদিহ্যাঁহয়, তাহলেকোনধরনেরচিকিৎসাপদ্ধতি?	এলোপ্যাথি = ১, হোমিওপ্যাথি =২, কবিরাজি= ৩
હ	গর্ভাবস্থায় আপনি কি কোন শারীরিক সমস্যার শিকার হয়েছেন?	হাঁ = ১, না=২
۹	যদি হ্যাঁ হয়, তহলে কি ধরনের সমস্যা?	পড়ে গিয়ে=১, ভারী জিনিস তুলতে গিয়ে = ২, আঘাত =৩, অন্যান্য= ৪
৮ ।	প্রসবের ধরন কেমন ছিল?	স্বাভাবিক = ১, সীজার=২, ফরসেপ=৩
৯	প্রসবের সময় কে উপস্থিত ছিল?	চিকিৎসক= ১, সেবিকা = ২, ধাত্রী = ৩
201	সন্তান কোথায় জন্মগ্রহণ করেছে?	বাড়ী=১, হাসপাতাল=২, ক্লিনিক=৩
221	আপনার সন্তানের জন্ম ধরন কেমন ছিল?	প্রসব বেদনা অনেকক্ষন > ১০ ঘন্টা = ১, যথাসময়ে = ২, হঠাৎ করে (অল্প সময়ের মধ্যে) =৩
১২।	আপনার প্রসবকালীন সময় কেমন ছিল?	দীর্ঘায়িত সময় =১,যথাসময় =২, হঠাৎ জন্ম=৩

পর্ব-৩:জন্মের সময়

21	বাচ্চার জন্মের সময় কি আপনার বয়স ৩৫ বা তদুর্ধ ছিল?	হ্যাঁ	না
২া	আপনার কি ২ বা তার বেশি গর্ভপাত অথবা মৃত সন্তান হয়েছে?	হ্যাঁ	না

৩।	আপনার বা আপনার সঙ্গীর কি কোন জন্মগত ব্রুটি, মানসিক প্রতিবন্ধিতা বা শেখার	হ্যাঁ	না
	সমস্যা আছে?		
8	আপনার পরিবার বা আপনার সঙ্গীর পরিবারে কারো কি এগুলো কখনো ছিলঃ	হ্যাঁ	না
	ক. ডাউন উপসগ?		
	খ স্পাইনা বাইফিডা সিন্ড্রোম অথবা নেউরাল টিউব সমস্যা ?	হ্যাঁ	না
	গ হাইড্রোসেফালাস	হ্যাঁ	না
	ঘ. হৃদপিণ্ডে ফুটো	হ্যাঁ	না
	ঙ মানসিক ভারসাম্যহীনতা?	হ্যাঁ	না
	চ. হেমফিলিয়া অথবা অন্যান্য রক্তের সমস্যা?	হ্যাঁ	না
	ছ. মাংস পেশীর সমস্যা?	হ্যাঁ	না
	ঝ. মেটাবলিক সমস্যা/রাসায়নিক সমস্যা ?	হ্যাঁ	না
	ট. জেনেটিক সমস্যা?	হ্যাঁ	না
3	আপনার কি থ্যালাসেমিয়া রয়েছে?	হ্যাঁ	না
ક	আপনার কি তাই সেকাস রোগ রয়েছে?	হ্যাঁ	না
۹	গর্ভাবস্থায় আপিনি কিঃ		
	আপনি কি কোন মাদক জাত দ্রব্য গ্রহন করেছেন ?	হ্যাঁ	না
	ধূমপানকরেছেন?	হ্যাঁ	না
	গর্ভাবস্থায় আপনি কি গাইনি ডাক্তার নির্দেশ অনুযায়ী ওষুধ খেয়েছেন কি না?	হ্যাঁ	না
	গর্ভাবস্থায় আপনি কি ব্যাথারওষুধ খেয়েছেন কি না ?	হ্যাঁ	না
	কোন এক্স-রে করেছেন কি না?	হ্যাঁ	না
	কোন অসুস্থতা, সংক্রমণ, ফুঁসকুড়ি কি ছিল ?	হ্যাঁ	না
	বাচ্চার জ্বর কি কখনো ১০২ ডিগ্রীর বা তার বেশি ছিল কি না?	হ্যাঁ	না
	ঘরে বা কর্মস্থলে বিড়াল, বিড়ালের বাচ্চা, ইঁদুর বা হ্যামস্টারের সংস্পর্শে এসেছেন?	হ্যাঁ	না
	কোন রাসায়নিক বা বিষাক্ত বস্তুর সংস্পর্শে এসেছেন কি না ?	হ্যাঁ	না

ש	গর্ভাবস্থায় আপনার কি ডায়বেটিস ছিল?	হ্যাঁ	না
۵I	গর্ভাবস্থায় আপনার কি উচ্চ রক্ত চাপ ছিল?	হাাঁ	না
२०।	আপনার গর্ভাবস্থায় বা পূর্বের গর্ভাবস্থায় ব্লাডসুগার লেভেল কি বেশি ছিল?	হ্যাঁ	না
221	আপনার কি খিঁচুনি রোগ আছে যার জন্য চিকিৎসা নিয়েছেন?	হাাঁ	না
১২।	আপনি কি কখনো শারীরিক বা মানসিক আঘাত পেয়েছেন ?	হ্যাঁ	না
১৩।	বাচ্চা জন্মের সাথে সাথে কি কান্না করেছিল?	হ্যাঁ	না
281	কতক্ষন পরে?(৩০ মিনিট পূর্বে অথবা পরে)	৩০ মিনিট পূর্বে	৩০ মিনিট পরে
261	বাচ্চা জন্মের সাথে সাথে কি শ্বাস কষ্ট হয়েছিল?	হ্যাঁ	না
<u></u> ଧତା	বাচ্চা জন্মের সাথে সাথে কি খিঁচুনি হয়েছিল?	হ্যাঁ	না
୪୩	বাচ্চা জন্মের সাথে সাথে কি অন্য কোনও সমস্যা হয়েছিল?	হ্যাঁ	না
2F I	বাচ্চা জন্মের পর ওজন কত ছিল?	হ্যাঁ	না
l			

হ্যাঁ =১, না=২

Permission Letter

February 17, 2016 Head of Physiotherapy Department Center for the Rehabilitation of the Paralyged (CRP) Savar, Dhaka-1343. Subject: Regarding permission to collect data from paediatric unit to conduct a research project.

Through: Course Coordinator, MSc in Physiotherapy Program. Sir,

Greetings from Bangladesh Health Professions Institute (BHPI).

It is your kind attention that Bangladesh Health Professions Institute (BHPI)- an academic institute of CRP, has been conducting M.Sc. in Physiotherapy under Faculty of Medicine of University of Dhaka (DU) since 2014. My thesis entitled "Antenatal and Intrapartum Risk Factors of term and near term born Children with Cerebral Palsy" under honorable supervisor, Dr. Kamal Ahmed, Associate professor, Department of health service management, Bangladesh Health Professions Institute (BHPI). The purpose of study is toidentify possibleantenatal and intrapartum risk factors affecting the development of cerebral palsy. It is a case control research study. Data collection will require the patients and a small space of your reputed paediatric unit and will occur for six weeks from 20th February, 2016. Data collectors will receive informed consents from all participants. Any data collected will be kept confidential. Ethical approval is received from the Institutional Review Board (IRB) of Bangladesh Health Professions Institute. I have chosen Pediatric Unit to collect required data. Now I am looking for your kind approval to start my data collection. I would like to assure that anything of my research project will not harmful for the participant.

Therefore I look forward to your cooperation by giving me permission for data collection at paediatric unit, CRP, Savar.

Yours faithfully

(Obaidul Haque) Part-2, M. Sc. in physiotherapy Program Session: 2012-13 BHPI, CRP, Savar, Dhaka-1343

Le Jordado



Ref. CRP/BHPI/IRB/02/16/021

Date: 27.02.2016

То

Md. Obaidul Haque Part – II, Student of M.Sc. in Physiotherapy Session: 2012-2013, DU Reg. No.: 88 BHPI, CRP, Savar, Dhaka-1343, Bangladesh

Subject: Approval of the thesis proposal – "Antenatal and Intrapartum Risk Factors of Children with Cerebral Palsy" by ethics committee.

Dear Md. Obaidul Haque

The Institutional Review Board (IRB) of BHPI has reviewed and discussed your proposal to conduct the above mentioned thesis, with yourself, as the Principal investigator. The Following documents have been reviewed and approved:

SI. No.	Name of the Documents	
1	Thesis Proposal	
2	Questionnaire (English and Bengali version)	
3	Information sheet & consent form.	

Since the study involves answering a questionnaire that takes 15 minutes, have no likelihood of any harm to the participants and have possibility of identifying the antenatal and intrapartum risk factors of cerebral palsy children. However, the members of the Ethics committee have approved the study to be conducted in the presented form at the meeting held at 8.30 am on February 25, 2016 at BHPI.

The institutional Ethics committee expects to be informed about the progress of the study, any changes occurring in the course of the study, any revision in the protocol and patient information or informed consent and ask to be provided a copy of the final report. This Ethics committee is working accordance to Nuremberg Code 1947, World Medical Association Declaration of Helsinki, 1964 - 2013 and other applicable regulation.

Best regards,

S. M. Ferdous Alam Assistant Professor, Dept. of M. Sc. in Rehabilitation Science Member Secretary, Institutional Review Board (IRB) BHPI, CRP, Savar, Dhaka-1343, Bangladesh

সিআরপি-চাপাইন, সাভার, ঢাকা-১৩৪৩, বাংলাদেশ, ফোন ঃ ৭৭৪৫৪৬৪-৫, ৭৭৪১৪০৪ ফ্যাক্স ঃ ৭৭৪৫০৬৯

CRP-Chapain, Savar, Dhaka-1343, Tel: 7745464-5, 7741404, Fax: 7745069, E-mail: contact@crp-bangladesh.org, www.crp-bangladesh.org