# Hip Instability In Cerebral Palsy

An essay submitted in fulfillment of the requirements for the degree of M.Sc of orthopedics

## By

Shaker Mohamed Jabr M.B.B.Ch

Under supervision of

#### Prof. Dr. Naguib Yousif Basha

Professor Of Orthopedics
Faculty Of Medicine
Cairo University

## Prof. Dr. Hasan Magdy El-barbary

Professor Of Orthopedics
Faculty Of Medicine
Cairo University

#### Prof. Dr. Mohamed Mahmoud Hegazy

Assistant Professor Of Orthopedics
Faculty Of Medicine
Cairo University

2009

## Abstrac<sub>#</sub>

CP is a fixed non progressive damage of the brain, which occur before, during, or after delivery. There are two major problems in CP patients, hip instability and gait abnormalities. Hips usually dislocate posteriorly, due to the overactivity of the adductors and flexors of hip. The causative factors of hip problems in CP are the combinations of the following: muscle imbalance, acetabular dysplasia, pelvic obliquity and excessive femoral anteversion. The hip is normal at birth. Most often, the hip dislocates when the child is between 5 - 7 years of age. There are many surgical options: Hip contracture releases, varus or rotational osteotomy, and if necessary, a pelvic osteotomy.

#### **Key words:**

Cerebral palsy, CP, Hip dysplasia, Pelvic osteotomy, Hip dislocation, Surveillance.

## Contents

Contents
Lists of Figures
Lists of tablesVI
Lists of abbreviations
Acknowledgment
English summary
Chapter 1
INTRODUCTION
Etiology of CP
Classification of CP
Hip disorders in CP
Chapter 2
ANATOMY OF THE HIP JOINT
Capsule of the hip joint
Ligaments of the hip joint9
Vascular supply and lymphatic drainage
Innvervation of the hip joint
Movements and muscles of the hip joint11
Chapter 3
PATHOPHYSIOLOGY OF SPASTICITY1
Spasticity
Hip pathology in CP
Windblown hip
Windblown hip deformity and pelvic obliquity26
Anterior dislocation2
Inferior dislocation29
Hyperabducted and extended deformity29
Anteversion and coxa valga relationship30
Hypotonic hip
Special hip problems34
Gait cycle
Gait pathology in CP38
Chapter 4
NATURAL HISTORY, DIAGNOSIS AND EXAMINATION44
Natural history44
Examination4
Measuring anteversion4
Radiological assessment5

## Chapter 5

1 3	
Treatment	59
Introduction	59
Recent advances in management of CP	60
Prevention	63
Hip surveillance	64
Specific treatment	69
Neuromuscular blocking agents	71
Selective dorsal rhizotomy	77
Abduction orthoses	78
Hyperbaric oxygen therapy	79
Reconstruction	81
Pemberton osteotomy	83
Dega osteotomy	85
Shelf osteotomy	86
Chiari osteotomy	86
Steel osteotomy	86
Proximal femoral osteotomy	87
Mid-shaft and distal femoral osteotomies	92
Complications of reconstruction	92
Salvage	94
Total hip replacement	94
Interposition arthroplasty	94
Resection arthroplasty	95
Femoral head resection	96
Hip fusion	96
Protocol of management	97
Type I anterior hip dislocation	99
Type II anterior hip dislocation	99
Type III anterior hip dislocation	99
Inferior hip dislocation	100
Hyperabducted and extended hip deformities	100
Windblown hips	101
Windblown hip and pelvic obliquity	103
Hypotonic hips	104
REFERENCES	105
rahis summaru	V

## Figures

Number	Figure		
1	Diplegic spastic CP	4	
2	Quadriplegic spastic CP	5	
3	Hemiplegic spastic CP	5	
4	Dyskinetic CP	5	
5	Ataxic CP	6	
6	Hip joint (lateral view)	9	
7	Ligaments of the hip joint (anterior view)	10	
8	Vascular supply of the hip joint	11	
9	Nerve supply of the hip joint	11	
10	Hyperexcitability of the lower motor neuron	16	
11	Normal and abnormal hip in CP	19	
12	Hip joint reaction force	21	
13	Primary pathological changes of hip joint in CP	23	
14	Hip joint reaction force according to age group	23	
15	Windblown hip	26	
16	Windblown hip and pelvic obliquity	27	
17	Type I anterior hip dislocation	28	
18	Type II anterior hip dislocation	28	
19	Type III anterior hip dislocation	28	
20	Inferior hip dislocation	29	
21	Hyperabducted and extended hip deformity	29	
22	Normal and weak hip flexors during gait	38	
23	Inspection of the hip joint	47	
24	Evaluation of the iliac crest	47	
25	Side examination of the hip	47	
26	Pelvic tilt	47	
27	Trendelenburg sign	48	
28	Measurement of leg lengths	48	
29	Hip flexion contracture	49	
30	Thomas test	49	
31	Measurement of the femoral anteversion	50	

32	Ely's test	50
33	Palpation of the hip joint	51
34	Flexion/extension examination	51
35	Abduction/adduction examination	51
36	Internal/external rotation examination	51
37	AP radiography of the pelvis in patient with CP	52
38	Fixed flexion deformity of the hip	52
39	Measurements of MP and AI	53
40	Metaphyseal/teardrop distance	53
41	Shenton's line	54
42	The center-edge angle of Wiberg	54
43	Horizonral toit externe HTE angle	54
44	CT showing anterior dislocation of the hip	55
45	Measurement of the femoral anteversion by CT	55
46	Fluoroscopy of the hip	56
47	U/S of the hip	57
48	GMFCS	65
49	GMFCS I	66
50	GMFCS II	66
51	GMFCS III	67
52	GMFCS IV	67
53	GMFCS V	68
54	WGH IV	68
55	Obturator neurectomy	70
56	Botulinum toxin injections	74
57	Phenol, local anesthesia and botulinum toxin injections	75
58	Continuous baclofen injection	70
59	Abduction orthoses	79
60	Pemberton osteotomy	83
61	Pemberton osteotomy line	84
62	Bikini incision	84
63	Subcutaneous tissue	84
64	Bone graft from iliac crest	85
65	Dega osteotomy	85
66	Shelf osteotomy	86
67	Chiari osteotomy	86
68	Steel osteotomy	86

69	Skin incision for proximal femoral osteotomy	
70	Fascia of vastus lateralis	87
71	Flat surface of the femur	88
72	Insertion technique of blade plate	88
73	Osteotomy of the femur using a chisel	89
74	Markers for osteotomy	90
75	1 <sup>st</sup> cut of osteotomy	90
76	Bone wedge removal	90
77	Abduction of the proximal segment in femoral osteotomy	91
78	Blade plate in place	91

## Tables

Number	Table	Page
1	Time of brain injury	1
2	Manifestations of cerebral palsy	1
3	Risk factors of CP	2
4	Grading system of brain hemorrhage	3
5	Movements and muscles of the hip	11 – 14
6	Upper motor neuron syndrome	15
7	Measurements in spasticity	16
8	Modified Ashworth Scale	17
9	Tardieu scale	17
10	Difference between CP and DDH	44
11	The gross motor function classification system	65
12	What to tell the family before soft tissue surgery	71
13	Local anesthetic block	71
14	Hints on using phenol	73
15	Symptoms of acute baclofen withdrawal	76
16	The ideal SDR candidate	77
17	Goals of orthoses	78

## Lists of abbreviations

Ach	Acetyl-choline
Al	Acetabular index
AP	Antero-posterior
ASIS	Anterior superior iliac spine
CMV	Cytomegalovirus
CNS	Central nervous system
СР	Cerebral Palsy
CT	Computed tomography
DDH	Developmental dysplastic hip
DJD	Degenerative joint disease
GMFCS	Gross Motor Function Classification System
GMH	Germinal matrix hemorrhage
НВОТ	Hyperbaric oxygen therapy
HIE	Hypoxic-ischemic encephalopathy
HTE angle	Horizonral toit externe
ITB	Intrathecal baclofen
IVH	Intra-ventricular hemorrhage
MI	Migration index
MP	Migration percentage
MRI	Magnetic resonance imaging
NSA	Neck shaft angle
OSSCS	Orthopedic Selective Spasticity Control Surgery
PIVH	Periventricular-intraventricular hemorrhage
PVL	periventricular leukomalacia
ROM	Range of motion
SCFE	Slipped capital femoral epiphysis
SDR	Selective dorsal rhizotomy
US	Ultra-sound
VDRO	Varus derotational osteotomy
WGH IV	Winters, Gage and Hicks hemiplegia group IV

## Acknowledgement

The valuable contribution of gratitude from a variety of disciplines must be acknowledged, in particular Prof. Dr. Naguib Basha. Prof. Dr. Hasan El-Barbary remains a constant source of inspiration in striving for excellence in the provision of all aspects about the hip in cerebral palsy. I would like to express my gratitude to Prof. Dr. Mohamed Hegazy for his sensitive patronage contribution, guidance and constructive comments to every word in this essay.

## English Summary

CP is a fixed non progressive damage of the brain, which occur before, during, or after delivery. There are two major problems in CP patients, hip instability and gait abnormalities. Hips usually dislocate posteriorly, due to the overactivity of the adductors and flexors of hip. Bony deformities occur later in a response to the spasticity. The causative factors of hip problems in CP are the combinations of the following:

- Muscle imbalance
- Acetabular dysplasia
- Pelvic obliquity
- Excessive femoral anteversion
- Increased femoral neck valgus
- Lack of weight bearing

These conditions cause hip pain, restriction of motion, and difficulties with sitting and perineal hygiene.

The hip is normal at birth. Most often, the hip dislocates when the child is between 5 - 7 years of age, taking approximately two years from first evidence of subluxation. Often the first clinical indication of subluxation is heralded by inability to abduct more than 45°.

Clinical evaluation of the hip joint spasticity does not provide an objective reproducible data. In the radiological measurements of the hip joint instability the migration percentage and migration index is used.

Hip contracture releases should be carefully tailored, taking into consideration the severity, location, child's age, and walking potential. Release of all contractures at one operative setting is usually best. Avoid overcorrection. The soft tissue releases may be combined with varus, rotational osteotomy, and if necessary, a pelvic osteotomy.

In acetabular insufficiency, pelvic osteotomy is necessary to obtain the stability. In older patients with painful subluxation or dislocation, hip arthrodesis or total hip replacement should be recommended.

## Chapter 1



# ntroduction



#### Introduction

**Cerebral Palsy** (CP) is a disorder of movement and posture that appears during infancy or early childhood. It is caused by non-progressive damage to the brain before, during, or shortly after birth. CP is not a single disease but a name given to a wide variety of static neuromotor impairment syndromes occurring secondary to a lesion in the developing brain. The damage to the brain is permanent and cannot be cured but the consequences can be minimized. Progressive musculoskeletal pathology occurs in most affected children. The lesion in the brain may occur during the prenatal, perinatal, or postnatal periods [Table1]. Any non-progressive central nervous system (CNS) injury occurring during the first 2 years of life is considered to be CP. In addition to movement and balance disorders, patients might experience other manifestations of cerebral dysfunction. [1] [Table2]

[Table 1] Time of brain injury		
Prenatal period	Conception to the onset of labor	
Perinatal period	28 weeks intrauterine to 7 days postnatal	
Postnatal period	First 2 years of life	

[Table 2] Manifestations of cerebral palsy		
Neurological	Musculo-skeletal	Associated problems
Muscle weakness	Contractures	Intellectual impairment
Abnormal muscle tone	Deformities	Epilepsy
Balance problems		Visual problems
Loss of selective control		Hearing loss
Pathological reflexes		Speech and communication
		problems
Loss of sensation		Swallowing difficulty
		Feeding difficulty, failure to thrive
		Respiratory problems

CP is the most common cause of childhood disability in Western societies. The incidence is **2-2.5/1000** live births. Some affected children do not survive and the prevalence varies between **1-5/1000** babies in different countries. It was previously thought that improvements in perinatal and obstetric care would decrease the incidence of CP. However, the incidence has not declined and the overall prevalence

increased during the 1980s and 1990s. This is explained by increased survival of premature and very low birth weight infants and by a rise in the number of multiple births. [2]

The **etiology** of CP can be identified only in **50**% of the cases. Certain factors in the history of the child increase the risk of CP. The incidence of CP among babies who have one or more of these risk factors is higher than among the normal population. The clinician should therefore be alerted to the possibility of the presence of CP in a patient with these factors. [1] [Table3]

[Table3] Risk factors in CP		
Prenatal	Perinatal	Postnatal
Prematurity (gestational	Prolonged and difficult	CNS infection
age less than 36 weeks)	labor	(encephalitis, meningitis)
Low birth weight (less than	Premature rupture of	Hypoxia
2500 g)	membranes	
Maternal epilepsy	Presentation anomalies	Seizures
Hyperthyroidism	Vaginal bleeding at the time of admission for labor	Coagulopathies
Infections (TORCH)	Bradycardia	Neonatal hyperbilirubinemia
Bleeding in the third trimester	Нурохіа	Head trauma
Incompetent cervix		
Severe toxemia, eclampsia		
Drug abuse		
Trauma		
Multiple pregnancies		

A whole group of **congenital developmental deformities** lead to CP. These deformities result from defects that occur in normal development and follow patterns based on failures of normal formation. A defect of the neural tube closure is the earliest recognized deformity leading to survival with motor defects. In the brain, the neural tube defect is called an encephalocele, and may be anterior, with a major midface or nasal defect. Most children with significant encephaloceles have very

significant motor impairments, usually quadriplegic pattern involvement with more hypotonia than hypertonia. [3]

**Segmental defects** in the brain are called schizencephaly, meaning there is a cleft in the brain. [4] These schizencephalies vary greatly, from causing minimal disability to causing very severe quadriplegic pattern involvement, usually with spasticity and mental retardation.

**Lissencephaly**, meaning a smooth brain, or a child with decreased cerebral gyri usually leads to severe spastic quadriplegic pattern involvement, but there is a significant range of involvement. [3]

Another part of normal development of the brain in the neonatal and prenatal period requires formation of the synapses and then subsequent remodeling of this neuronal synapse formation. As the cells migrate into the correct position and initially form their synapses, many of these premature synapses need to be remodeled through the influence of external stimuli for normal function to develop.

**Prematurity** and **brain hemorrhages** are much better understood since the widespread use of cranial ultrasound, in which the infant brain can be imaged through the open anterior fontanel. A common grading system for the severity of these hemorrhages shown in [Table 4]. [5]

[Table 4] Grading system of brain hemorrhage	
Grade I	Germinal matrix hemorrhage only
Grade II	Hemorrhage in the lateral ventricle and dilation of the lateral ventricle
Grade III	Ventricular system enlargement
<b>Grade IV</b>	Periventricular hemorrhage and infarctions

**Hypoxic** events occurring around delivery, usually in full-term infants, also lead to disability, with most of these children developing severe quadriplegic pattern involvement with severe mental retardation.

**Shaken baby syndrome** occurs usually in a child less than 1 year of age when a caretaker shakes the baby back and forth to quiet the crying. This vigorous shaking causes stretching, shearing, and tearing of the long axons and capillaries in the cortex of the brain. If these babies survive,