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Study of the Alteration in Skeletal Maturation in Children with Cerebral Palsy

Thesis

Submitted for Partial Fulfillment of Master Degree in Childhood Studies Medical Department (Child Health and Nutrition)

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بسم الله الرحمن الرحيم

(وَأَنْزَلَ اللهُ عَلَيْكَ الكِتَابَ وَالحِكْمَةَ وَعَلَّمَكَ مَا لَمْ تَكُنْ تَعْلَمُ وَكَانَ فَضْلُ اللهِ عَلَيْكَ عَظِيمًا)

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ABSTRACT

Cerebral palsy is a group of nonprogressive motor impairment syndromes that can be manifested in the early years of life and caused by damage to the motor control centres of the young developing brain. Bone age is a way of describing the degree of maturation of the child's bone, it is the average age at which children reach this stage of bone maturation. Skeletal maturation has been shown to be altered in children with CP. The aim of the present study was designed to assess bone age in children with different types of CP, and it was conducted on forty children with CP. X-ray of the left wrist and hand was taken from all children included in the study and bone age was assessed according to Greulich and Pyle Atlas method. Anthropometric measures were taken to all children. Statistical analysis for the data was done including descriptive statistics and significance was determined as $P<0.05$. The most common type of CP according to the topography in the present study was the diplegic type which constitutes 47.5% of the studied cases and the least type was the triplegia, and the most common type according to the muscle tone was the spastic type which formed 70% of cases and the least type was the dyskinetic type which formed 5% of cases. There was 50% ambulant and 50% non ambulant CP children included in the study .

The result of the present study revealed that there was delayed bone age in comparison with the chronological age of the children with CP included in the study . Also, delayed bone age could be related to the level of motor dysfunction according to Gross Motor Function Classification System, where greater bone age delay was more frequently associated with nonambulant CP children (Level IV and V of dysfunction according to GMFCS) more than ambulant CP children (Level II and III of dysfunction according to GMFCS) and the difference was statistically significant .

List of Abbreviation

BA .....Bone Age

CA .....Chronological Age

CP .....Cerebral Palsy

CT .....Computerized Tomography

EEG .....Electroencephalogram

EMG.....Electromyelogram

EMPP.....Early Motor Pattern Profile

GMFCS.....Gross Motor Function Classification System

GP method.....Greulich and Pyle method

MRI.....Magnetic Resonance Image

PVL.....Peri-ventricular leukomalacia

ROIs.....Twenty Regions Of Interest

SCPE .....Serveillance of Cerebral Palsy in Europe

TW2 ..... Tanner and Whitehouse method

BMI.....Body Mass Index.

SDS.....Standard Deviation Score

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Introduction

INTRODUCTION

Cerebral palsy (CP) is a common developmental disability. It is a diagnostic term used to describe a group of motor syndromes resulting from disorders of early brain development ***(Tein ,2007).***

CP was first identified by English surgeon William Little in 1860. Little raised the possibility of asphyxia during birth as a chief cause of the disorder ***(Mutch, 1992).***

CP is the most common and costly form of chronic motor disability that begins in childhood ***(Johnston, 2008).***

It is caused by damage to the motor control centers of the young developing brain. Intrauterine exposure to maternal infection (chorioamnionitis, inflammation of placental membranes, umbilical cord inflammation, maternal sepsis, urinary tract infections) is associated with significant increase in the risk of CP in normal birth weight infants. Fewer than 10 % of children with CP had evidence of intrapartum asphyxia ***(Paul, 2008).***

In the industrialized world, the incidence of CP is about 2-2.5 per 1000 live birth ***(Barbara,2008)***. The prevalence of CP among preterm and very preterm infant is substantially higher ***(Susan, 2008)***. In the developing world, the prevalence of CP is not well established but

estimates are 1.5-5.6 cases per 1000 live births. These figures may represent an under estimation because of a paucity of data, the lack of health care access and in consistent diagnostic criteria ***(Stanley, 2000)***.

The prevalence of CP is increased among low birth weight infants, particularly those weighing less than 1000 gm at birth, primarily because of intracerebral hemorrhage and peri-ventricular leukomalacia (PVL). Although the incidence of intracerebral hemorrhage has decline significantly, PVL remains a major problem ***(Tein , 2007)***.

There is no known cure for CP, medical intervention is limited to the treatment of complications possible from C P and a team of physicians from various specialties as well as occupational and physical therapists, speech pathologists, social workers educators, and developmental psychologists provide important contributions to treatment of these children ***(Miller et al., 2005)***.

Bone age is a way of describing the degree of maturation of a child's bones, it is the average age at which children reach this stage of bone maturation ***(Anderson et al., 2000)***.

Different studies were done to assess skeletal maturation in children with CP and there is controversy

between the results of these studies, A study was done by ***Gollapundi et al. (2002)*** who concluded that bone age was advanced compared with chronological age in most of ambulatory CP patients while, another study done by ***Roberts et al. (1994)***, found that bone age delay has been observed in children with CP.

It is common in children with CP to undergo surgical intervention as a part of their rehabilitation program to correct deformities in a way of improving the function or preventing complications ***(Roberts et al., 1994).*** One of these intervention, is to do limb equalization in case of hemiplegic CP where there is difference in limb length due to weakness and spasticity in the hemiplegic side. To accurately predict when limb length equalization should be undertaken, serial bone length and bone age are necessary, these data then allow the surgeon to predict the leg lengths discrepancy at skeletal maturity as well as when to intervene with limb length equalization procedures ***(Miller, et al., 2005).***

AIM OF THE STUDY

The aim of the present study was to assess skeletal maturation using bone age in a sample of CP children.

CHAPTER (I)

CEREBRAL PALSY

Modern accounts of cerebral palsy (CP) begin with the attribution of CP to difficult deliveries ***(William Little, 1843),*** i.e. to perinatal causes. Fifty years later **(Freud , 1893)** speculated that CP could represent the effects of deeper – lying influences on the development of the fetus, i.e. antenatal causes. Because of the medicolegal implications, controversy still rages as to how much prenatal and how much intrapartum factors contribute ***(Kuban and Leviton 1994).***

Definition:

CP is a diagnostic term used to describe a group of motor syndromes resulting from disorders of early brain development. CP is caused by a broad group of developmental, genetic, metabolic, ischemic, infectious, and other acquired etiologies that produce a common group of neurologic phenotypes ***(Johnson , 2008).***

Firstly, all children with CP have suffered some form of brain damage and this has involved the motor pathway. The term (cerebral) palsy differentiate such children from those with muscle palsies, orthopedic, and spinal palsy as

seen in the spina bifida complex, traumatic paraplegia or familial paraplegia, etc ***(Roeleveld ,1998).***

Secondly, it indicates that the pathology is nonprogressive, thus, excluding conditions such as cerebral tumors, the degenerative brain diseases or progressive multisystem diseases (e.g. Friedreich,s ataxia). This doesn't mean that CP is a static condition, the clinical pattern changes as brain maturation continues throughout childhood resulting in dynamic clinical pattern despite a static pathology, so, the neurological features of CP often change or progress over time ***(Johnson , 2008).***

Prevalence and Incidence:

CP is a common neurodevelopmental disorder of childhood with a prevalence of 2 – 2.5 per 1000 live births worldwide ***(Johnson , 2008).***

Recent advances in neonatal management and obstetric care have not shown decline in the incidence of CP where with decline in infant mortality rate, there is an increase in incidence of CP ***(Barbara,2008).***.

Cerebral palsy and related developmental disorders are more common in males (M) than in females (F). The Surveillance of Cerebral Palsy in Europe (SCPE) reports a M: F ratio of 1.33: 1 but the reasons for this disparity are uncertain ***(Barbara,2008).***

Males born very preterm also appear to be more vulnerable to white matter injury and intraventricular hemorrhage than females. Experimental studies in adult animals and data from adult patients with stroke indicate that sex hormones such as estrogens provide protection against hypoxic-ischemic injury, and the neonatal brain is also influenced by these hormones. However, hormonal influences on the fetus and neonates are substantially different from those on adults ***(Johnston ,2008).***

Other reports demonstrated major differences between male and female neurons grown separately in cell culture, suggesting that sex differences in the fetal or neonatal period result from intrinsic differences in cell death pathways. This new information indicates that there are important neurobiological differences between males and females with respect to their response to brain injuries. This information is relevant to understanding the pathogenesis of CP as well as to the design of future clinical trials of potential neuroprotective strategies ***(Andrew,1996)).***

Pathology:

Cerebral palsy results from a permanent static lesion of the cerebral motor cortex that occur prenatal, natal, postnatal or within the first two years of life ***(Flett, 2003)*** Lesion itself doesn't change but the clinical picture change

while the child grows and develops. The rate of growth of the child with CP is slower than in unaffected child ***(Thorngren , 2006).***

A specific hypoxic event associated with irreversible cell death explains the etiology of CP in less than 50 % of cases ***(Bruce , 2000).***

Some areas of the brain are more susceptible to damage than others. For example, variations in blood supply and unique metabolic requirement in some brain areas increase the sensitivity to hypoxia in the presence of bacterial and viral infection of the fetus, maternal infection or chorioamniotitis ***(Gaudet , 2001).*** Selective vulnerability of the periventricular white matter occurs between 26- 34 weeks of gestation, so fetal insults occurring during this period can produce periventricular leukomalacia with spastic diplegia. Similarly, the unique metabolic demands of the basal ganglia in the fetus at 38 – 40 weeks create selective vulnerability that can result in dystonia and movement disorder ***(Foster , 2001).***

Attempts to reduce the incidence of CP in term infants should be directed towards increasing understanding of fetal developmental biology and awareness that strategies need to be developed for premature infants to protect the vulnerable developing white matter ***(Barbara , 2008).***

Injury to upper motor neurons decrease input to the reticulospinal and corticospinal tracts which in turn affects motor control, and produce abnormal muscle control and weakness. Also, the loss of descending inhibitory input through reticulospial tract produce spasticity ***(Goldstein, 2001)*** which has been defined as a velocity – dependent resistance of muscle to stretch ***(Sanger , 2003)*** or an excessive, inappropriate involuntary muscle activity associated with upper motor neuron paralysis.

Spasticity in patient with CP may lead to musculoskeletal complications such as contractures, pain, subluxation **(Rosenbaum,2002).**

Injury to extrapyramidal tract results in movement disorder such as athetosis, chorea, dystonia or rigidity. Clinical manifestations depends on the extent and the type of CNS damage, the location of the irreversible insult and the ability of the CNS to reorganize after the insult e.g. movement disorder occurs after hyperbilirubinemia and basal ganglia injury, diplegia occurs in association with periventricular leukomalacia, and quadriplegia occurs with diffuse brain injury ***(Fedrizzi , 2003)***.

Etiology and risk factors of CP:

It is usual to divide the cerebral palsy into congenital and acquired types. In congenital CP, damage may arise

from several causes (e.g. genetic defects, migration defects, cerebral malformation, hypoxic ischemic encephalopathy, nutritional deficiency, trauma, infection, infarction and hemorrhage). Migration defects may result from mutation of genes responsible for the mechanisms that control the neuronal migration, like the radial glial fiber system that guides neurons to their proper site ***(Barbara , 2008).***

The injury to the developing brain may be prenatal, natal, or postnatal. As much as 75% - 80 % of the cases are due to prenatal injury with less than 10 % being due to significant birth trauma or asphyxia ***(Maclennan , 1999).***

The most important risk factor seems to be prematurity and low birth weight with risk of CP increasing with decreasing gestational age and birth weight. CP is seen in 10 – 18 % of babies in 500 – 999 grams birth weight ***(Johnson, 2004).*** CP occurs more commonly in children who are born very prematurely. Although term infants are at relatively low absolute risk, term births constitute the large majority of all births, as well as approximately half of all births of children with CP. Prenatal maternal chorioamnionitis is also a significant risk factor accounting for as much as 12 % of CP in term infants and 28 % in premature infants ***(Colford , 2000).***

Table (1) : Shows Risk Factors of C.P.

Prenatal	Perinatal	Postnatal
Intrauterine infections Teratogenic exposures Placental complication Multiple births. Maternal conditions like seizures,hyperthyroidism **(Donovan , 1998)**	Infections Intracranial Hemorrhage Seizures, Hypoglycemia, Hyperbilirubinemia, Birth asphyxia. Perinatal arterial ischemic stroke. **(Garite, , 2004)**	Meningitis Encephalitis Trauma Drowning. Coagulopathies causing cerebral infarction . Postnatal events account for 12 % - 21% of CP. But in a large number of cases, the cause of CP remains unknown **(Sankar., 2005).**

Classification of CP:

There are many ways of classification of CP

1. According to pathology: genetic syndrome, malformation, infective, intrauterine encephalitis or vasculitis, hemorrhage, infarction, hypoxic ischemic damage, periventricular leukomalacia
2. According to site of brain injury: cortical, subcortical-periventricular, white matter, basal ganglia, cerebellar, brain stem and global.
3. According to topography of signs: monoplegia, diplegia, triplegia, quadriplegia and hemiplegia.
4. According to severity: mild, moderate and severe ***(Brunstram et al., 1998).***
5. According to neurology: spastic, dystonic, dyskinetic (choreoathetoid) hypotonic and / or ataxic.

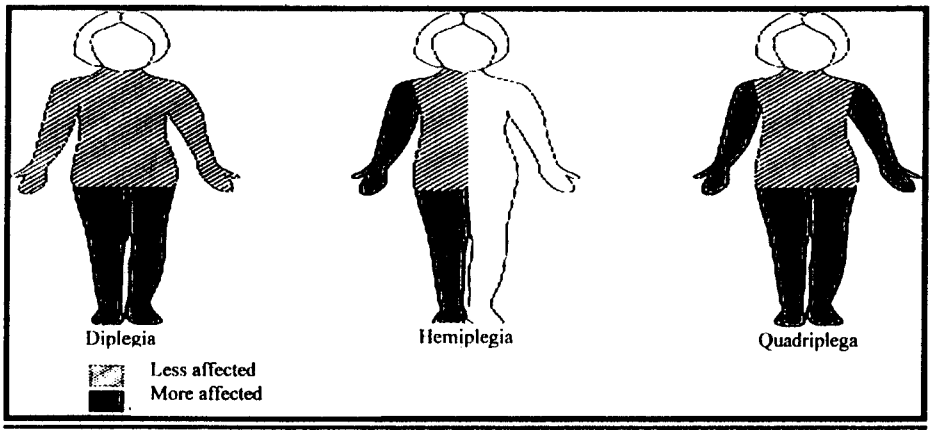

Figure (1): The classification of cerebral palsy according to topography of signs ***(Freeman ,2005).***

I: Spastic Cerebral Palsy:

It is the most common type and accounts for 70 % - 75 % of cases It is characterized by upper motor neuron signs, namely clasp knife hypertonia, exaggerated deep tendon reflexes and extensor planter responses ***(Wollach ,1997).***

Types of Spastic Cerebral Palsy:

Spastic quadriplegia:

These patients are severely disabled. All four limbs are affected with upper limbs being equally or more affected than lower limbs. The majority of the children have severe mental handicap, pseudo bulbar palsies, microcephaly, growth failure and may be associated with visual and hearing difficulties and epilepsy ***(Menkes , 2000).*** They have hypertonicity leading to arching of the

back and scissoring of legs (either spontaneous or when vertically suspended). Hip subluxation or dislocation may occur because of severe spasticity. Arms are internally rotated, elbows extended or lightly flexed and hands fisted. Later flexion contractures develop at ankles, knees and elbows ***(Eicher , 1993).***

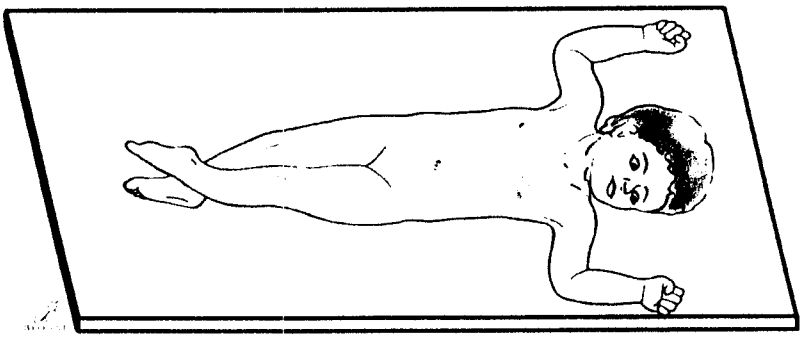

Figure (2): Signs of spastic quadriplegia ***(Freeman ,2005).***

Spastic Diplegia:

The lower limbs are more affected than upper limbs. Mild cases may present with toe walking due to dorsiflexion of the feet with increased tone of the ankles. The first indication of spastic diplegia is often noted when an affected infant begins to crawl, The child uses the arms in a normal reciprocal fashion but tends to drag the legs behind more than using the normal four – limbs crawling movement. If the spasticity is severe, application of diaper is difficult because of the excessive adduction of the hips.

In severe cases there is flexion of the hips, knees, and for a lesser extent elbows, when the child is held vertically, rigidity of the lower limbs is more evident and adductor spasm of the lower extremities causes scissoring of the legs. The prognosis for normal intellectual development is excellent for these patients, and the likelihood of seizures is minimal ***(Johnson , 2008).***

Spastic Hemiplegia:

It is a unilateral paresis with upper limbs more severely affected than lower limbs, it is seen in 56 % of term infants and 17% of preterm infants, it involves one side of the body, the arm is usually more affected than leg except in preterm with periventricular hemorrhagic infarction where the leg may be more affected than the arm.

It is noticed that the right side involvement is twice as the left side in congenital but not acquired cases and in most series males outnumber females by 3: 2.The most common cerebral injury is in the middle cerebral artery distribution ***(Hensen , 1998).***

Spastic Triplegia:

Is a type of spastic cerebral palsy that affects three of the limbs, usually one leg and both arms. Spastic Triplegia is one of the rarest forms of spastic cerebral palsy ***(Hensen , 1998)..***

Abnormal signs are noticed first at the hand as normal development is cephalocaudal.

There are early signs which may help in diagnosis:

1. Eighty percent of infants who will develop a hemiplegia later will show some abnormalities of neonatal behavior with fits, apnea, cyanotic attacks and hypothermia after birth.
2. Fisting of the hands beyond 3 months of life and minimal movement in the affected side.
3. Hand preference in child less than 12 months of age.
4. Sitting and crawling are not much delayed, walking is delayed generally by 2-3 months (from 18 – 24 months) with circumductive gait.
5. When the child is supine, the affected lower limb may be extremely rotated ***(Hensen, 1998).***

Examination of the extremities may show growth arrest, especially if the contralateral parital lobe is abnormal because extremity growth is influenced by this area of the brain.

In severe established cases, the arm is held adducted, flexed and internally rotated at the shoulder, with the elbow

flexed, arm pronated, wrist flexed and thumb adducted. The legs are adducted, semiflexed at knee and the ankle is planter flexed, eqinovarus deformity of the foot. In long standing cases asymmetry of limb growth may occur ***(Pratibha , 1998).*** Seizures occur in more than 50%, visual field defect, homonymus hemianopia, cranial nerves abnormalities most commonly facial nerve palsies are seen in CP children with spastic hemiplegia. 25 % of those children have cognitive abnormalities including mental retardation ***(Johnson , 2008).***

II: Athetoid Cerebral Palsy:

It is called choreoathetoid or extrapyramidal CP. Affected infants are characteristically hypotonic with poor head control and marked head lag and develop increased variable tone with rigidity and dystonia over several years ***(Champman , 2008).***

Feeding may be affected and drooling may be prominent. Speech is typically affected because the oropharyngeal muscles are involved. Speech may be absent or slurred and voice modulation is impaired, seizures are uncommon and intelligence is preserved. It is also called dyskinetic CP and is the type most likely to be associated with birth asphyxia. Athetoid CP can also be caused by

kernicterus secondary to high level of bilirubin and in this case MRI show lesions in the globus pallidus bilaterally. Extrapyramidal CP can also be associated with lesions in basal ganglia and thalamus caused by metabolic genetic disorder such as mitochondrial disorders, so, metabolic tests are important in the evaluation of child with athetoid CP ***(Michael V , 2008).***

Although intelligence is often preserved, the presence of severe physical and communicative disabilities give a mistaken diagnosis of mental retardation ***(Singhi , 1997).***

III: Ataxic Cerebral Palsy:

This occurs due to predominant involvement of the cerebellum. These infants are hypotonic and inactive. Walking is delayed. The gait is ataxic, wide – based and accompanied by exaggerated balancing movements of arms. Cerebellar signs are present. Ataxia may occur in pure form or coexists with spasticity (ataxic diplegia) ***(Champman , 2008).***

IV: Hypotonic Cerebral Palsy:

This is extremely rare type of CP. In many cases it may in fact represent an evolving form of dyskinetic or spastic CP. Others causes of hypotonia should be excluded ***(Johnson ,2008).***

II: Mixed Cerebral Palsy:

Children with **mixed cerebral palsy** usually have both the tight muscle tone of spastic cp and the involuntary movements of athetoid cerebral palsy. This is caused by injury to both the pyramidal and extra pyramidal areas of the brain. Spasticity is usually the more obvious type, with the involuntary athetoid movements increasing when the child is between nine months and three years old. It usually takes months or years to notice the presence of mixed cerebral palsy more obvious ***(Johnson ,2008).***

Figure (3): Different types of CP according to the neurological effect ***(Freeman, 2005)..***

Associated Disorders:

CP is also commonly associated with a spectrum of developmental disabilities, including mental retardation, epilepsy, as well as visual, hearing, speech, cognitive and behavioral abnormalities. The motor handicap may be the least of the child's problem ***(Freeman, 2005).***

Behavior Disturbance:

In the early months of life child with brain damage often shows a reverse sleep pattern. This may persist into later childhood with child at first wants to sleep during the day and then appearing to require less total sleep, awaking in the night, being difficult to get off to sleep and showing resistance to hypnotic drugs. The infant is also likely to be irritable and slow at feeding. The older child will show poor concentration span and a decreased threshold for fight and flight so that he may have violent attacks over mild anxieties ***(Gould , 2001).***

Visual Problems:

Squint is very common in children with CP and may be paralytic. In twenty five percent of low birth weight infants squint associated with myopia occur. Optic atrophy is a common finding but is not usually associated with

complete loss of vision or visual acuity. A significant percentage of multiple handicapped children have a major degree of cortical blindness, however, specific difficulties with concepts of space, shape, and direction are characteristic of the brain damaged child and give rise to the classic verbal performance discrepancy on IQ testing.

Visuospatial problems by themselves are not a common cause of reading and writing difficulties but produce untidiness in writing. Visuomotor disorders are also common. These may include the inability, in the absence of spasticity, to imitate gestures difficulties with fine finger coordination such as tying shoe laces ***(Freeman , 2005).***

Communication Problems:

The child may suffer from pseudobulber, hyperkinetic, hypokinetic, or ataxic bulbar palsy or a combination of these. Dysarthria occurs in 100% of children with dyskinetic CP ***(Freeman , 2005).*** In children with ataxia or diplegia following prematurity a speech disorder is more likely to be developmental than a true dysarthria. Both receptive and expressive language deficits are common. The speech therapist should be involved in positioning and feeding program from an early age, trying to encourage proper lip closure and bite. Formal speech therapy will be required later and the child may need an

alternative communication system such as one of the sign language e.g. Makaton, Paget Gorman or Bliss symbol system .The speech therapist can also help the child with severe drooling in a combination of exercises, the use of drugs such as propanthine, biofeedback systems, orthodontic appliances such as palatal lifts or plastic surgical operations such as pharyngoplasty may be needed in individual cases ***(Pennington , 2004).***

Hearing impairment occurs in approximately 12% of children with CP. This occurs more commonly if the etiology of CP is related to very low birth weight, kernicterus, neonatal meningitis or severe hypoxic ischemic results ***(Golomb , 2000).***

Feeding Problems:

Oromotor problems with feeding difficulties, swallowing dysfunction and drooling are also present ***(Reilly , 1996).***

Many children with CP are likely to have difficulties with eating certain foods because of chewing and/or swallowing problems. So, it's difficult for them to gain weight. Many children with eating and drinking difficulties don't receive an adequate or balanced diet. It's common for children with CP to suffer from constipation, and to be both short and underweight for their age. To some extent, poor

weight gain may be minimized by providing many calories as possible in the texture that the child can eat .

Poor fluid intake often contributes to the tendency of many children with CP to be constipated. Increasing fluid intake often requires development of drinking skills as outlined earlier and the introduction of drinking thickened liquits ***(Christos , 2004)***.

Epilepsy:

The incidence of epilepsy in children with CP ranges between 20% and 50% and it occurs in the majority of patients who suffer from bilateral hemiplegia and in about 50% of patients who suffer from a hemiplegia. It is rarest in children with pure diplegia, ataxia, or dyskinesia ***(Aicardi, 1990)***.

Epilepsy is more common in certain forms of CP, i.e., epilepsy is more common in children with tetraplegia and less common in children with simple ataxia. Lesions of left hemisphere (right hempiplegia) are more often associated with epilepsy ***(Pearson RD ,2003)***.

In spastic or ataxic diplegia, the risk of epilepsy is lower (16%-27%) ***(Aicardi, 2003)***.

In the acquired spastic hemiplegia the incidence of epilepsy is twice that of the congenital one. Convulsions

may occur as early as the neonatal age, the presence of neonatal seizures has been a useful marker for subsequent epilepsy in CP children, the convulsions may be second to prenatal asphyxia, neonatal meningitis, stroke or due to brain malformation ***(Kwong , 2000).***

Status epilepticus is not uncommon in childhood epilepsy and children at highest risk are those having a background of neurological impairment.

Epilepsy in children with tetraplegia appears earlier compared to those with diplegia or hemiplegia ***(Aicardi , 2003).***

Types of seizures and epilepsy:

1. Pure generalized tonic-clonic movement in spastic froms of CP, least in the other types.
2. Focal epilepsia in hemiplegia.
3. Infantile convulsions.
4. Early childhood myoclonic epilepsy.
5. Epilepsy of the temporal robe.

Sensory Disorders:

A normal child cannot tell differences in weights placed in his hands and doesn't recognize shapes drawn on

his hands or recognize which fingers are touched (finger agnosis). Sensory inattention is common until about seven years of age. Brain damaged children are much older before they acquire these abilities, so that sensory defects may be diagnosed in a child and yet subsequently disappear. There is no doubt that children with chronic motor disorders may experience a disordered body image and unlike normal children will not get the normal feedback from utilizing the limb during the critical periods of parietal maturation in infancy by exploring objects, textures.. etc. It is for that reason, many programs include this type of sensory stimulation, e.g. sensory stimulation to hemiplegic limb. The limbs may also show growth disturbance and be blue and cold so that in cold whether, a pair of shoes a size larger than the child needs (to accommodate an extra pair of socks) may be required to prevent cold pains or in extreme cases, ischemia ***(Todman, 2008).***

Educational Problems:

The majority of children with moderate to severe CP are of subnormal intelligence, only about a third of the children are found to be of average or superior intelligence and between a quarter and a third have IQ of less than 55. Children with bilateral hemiplegia or diplegia with involvement of upper limbs are less intelligent than those with hemiplegia or diplegia, confined to the lower limbs or dyskinesia.

In addition to overall decrease in intellectual function, many of children show specific learning difficulties, especially in arithmetic, and don't attain the level of competence predicted from their IQ. At the same time, many children and adults with CP function at high educational and vocational level,without any signs of cognitive dysfunction ***(Neil , 2008).***

Deformities:

Deformities of the spine e.g. scoliosis, kyphosis, and lordosis are associated with CP, Although scoliosis can lead to serious outcomes, kyphosis and lordosis are not associated with significant comorbidity ***(Renshaw , 1995).*** The frequency of scoliosis in CP patient is 25% with the rate about 60-75% in patient with severe involvement and quadriplegia ***(Thomson and Banta, 2001).*** The natural course of scoliosis in CP is different, and characterized by curve progression after skeletal maturity ***(Thometz and Simon, 1988).*** Progression of scoliosis can lead to pain, interference with sitting, and less commonly cardiopulmonary compromise. Progression of curves beyond 50 degrees is frequent, with curves beyond 100 degrees a risk factor for complications is high ***(Sarwahi , 2001).***

Personal and Social Independence:

Personal independence means the ability to dress, undress, look after the personal hygiene, toileting and in girls to cope with periods at adolescence. By social we mean the ability to use the telephone, go to shops, cook, and use kitchen aids. All these aspects will be affected in cases of CP and require training to be achieved ***(Hansen , 2000).***

Gross Motor Function Classification System (GMFCS):

GMFCS has been applied to children with CP to measure the severity of motor impairment objectively. Measurement of severity of impairment is important when evaluating intervention strategies ***(Palisano , 1997).***

GMFCS is a classification according to severity of the motor impairment or resulting disability. This is a recently developed system which has been found to be a reliable and valid system that classifies children with CP by their age – specific gross motor activity. This GMFCS describe the functional characteristics in five levels, from I to V, level I being the mildest in the following age groups: up to 2yrs, 2 – 4 yrs, 4 – 6 yrs and between 6 – 12 yrs. For each level, separate descriptions are provided ***(Palisano , 1997)***.

Children in level III usually require orthoses and assisting mobility devices, while children in level II do not require assisting mobility device after the age of 4 yrs. Children in level III sit independently, have independent floor mobility, and walk with assisting mobility devices. In level IV, affected children function in supported sitting but independent mobility is very limited. Children in level V lack independence even in basic antigravity postural control and need power mobility.

Gross motor function Classification System for CP is based on self–initiated movement with particular emphasis on sitting (trunkal control) and walking. When defining a 5 level classification System, the primary criterion was that the distinction in motor function between levels must be clinically meaningful. Distinctions between levels of motor function are based on functional limitation, the need for assistive technology, including mobility devices (such as walker, crutches and canes) and wheeled mobility, and to much lesser extent quality of movement.

Criteria for Classification:

Before second birthday:

Level 1

Infants move in and out of sitting and floor sit with both hands free to manipulate objects. Infants crawl on hands and knees.

Pull to stand and take steps holding on to furniture. Infants walk between 18 months and 2 years of age without the need for any assistive mobility device.

Level 2

Infants maintain floor sitting but may need to use their hands for support to maintain balance. Infants creep on their stomach or crawl on hands and knees. Infants may pull to stand and take steps holding on to furniture.

Level 3

Infants maintain floor sitting when the low back is supported. Infants roll and creep forward on their stomachs.

Level 4

Infants have head control but trunk support is required for floor sitting. Infants can roll to supine and may roll to prone.

Level 5

Physical impairments limit voluntary control of movement. Infants are unable to maintain antigravity head and trunk postures in prone and sitting. Infants require adult assistance to roll.

Between second and forth birthday:

Level 1

Children floor sit with both hands free to manipulate objects. Movements in and out of floor sitting and standing are performed without adult assistance. Children walk as the preferred method of mobility without the need for any assistive mobility device.

Level 2

Children floor sit but may have difficulty with balance when both hands are free to manipulate objects. Movements in and out of sitting are performed without adult assistance. Children pull to stand on a stable surface. Children crawl on hands and knees with a reciprocal pattern, cruise holding onto furniture and walk using as assistive mobility device as preferred methods of mobility.

Level 3

Children maintain floor sitting often by "W-sitting" (sitting between flexed and internally rotated hips and knees) and may require adult assistance to assume sitting. Children creep on their stomach or crawl on hands and knees (often without reciprocal leg movements) as their primary methods of self-mobility. Children may pull to stand on a stable surface and cruise short distance. Children may walk short distances indoors using an assistive mobility device and adult assistance for steering and turning.

Level 4

Children sit on a chair but need adaptive seating for trunk control and to maximize hand function. Children move in and out of chair sitting with assistance from an adult or a stable surface to push or pull up on with their arms. Children may at best, walk short distance with a walker and adult supervision but have difficulty turning and maintaining balance on uneven surfaces. Children are transported in the community. Children may achieve self-mobility using a power wheel chair.

Level 5

Physical impairment restricts voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At level V, Children have no means of independent mobility and are transported, some children achieve self-mobility using a power wheelchair with extensive adaptation.

Between forth and sixth birthday:

Level 1

Children get into and out of, and sit in a chair without the need for hand support. Children move from the floor and from chair sitting to standing without the need for objects for support. Children walk indoors and outdoors, and climb stairs. Emerging ability to run and jump.

Level 2

Children sit in a chair with both hands free to manipulate objects. Children move from the floor to standing and from chair sitting to standing but often require a stable surface to push or pull up on with their arms.

Children walk without the need for any assistive mobility device indoors and for short distance on level surfaces outdoors. Children climb stairs holding onto a railing but are unable to run or jump.

Level 3

Children sit on a regular chair but may require pelvic or trunk support to maximize hand function. Children move in and out of chair sitting using a stable surface to push on or pull up with their arms. Children walk with an assistive mobility device on level surfaces and climb stairs with assistance from an adult. Children frequently are transported when traveling for long distances or outdoors on uneven terrain.

Level 4

Children sit on a chair but need adaptive seating for trunk control and to maximize hand function. Children move in and out of chair sitting with assistance from an adult or a stable surface to push or pull up on with their arms. Children may at best, walk short distances with a walker and adult supervision but have difficulty turning and maintaining balance on uneven surfaces. Children are transported in the community. Children may achieve self-mobility using a power wheelchair.

Level 5

Physical impairments restrict voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At Level V, children have no means of independent mobility and are transported. Some children achieve self-mobility using a power wheel chair with extensive adaptations.

Between sixth and twelfth birthday:

Level 1

Children walk indoors and outdoors, and climb stairs without limitations. Children perform gross motor skills including running and jumping but speed, balance and coordination are reduced.

Level 2

Children walk indoors and outdoors, and climb stairs holding onto a railing but experience limitations walking on uneven surfaces and inclines, and walking in crowds or confined spaces. Children have at best only minimal ability to perform gross motor skills such as running and jumping.

Level 3

Children walk indoors or outdoors on a level surface with an assistive mobility device. Children may climb stairs holding onto a railing. Depending on upper limb function. Children propel a wheelchair manually or are transported when traveling for long distances or outdoors on uneven terrain.

Level 4

Children may maintain levels of function achieved before age six or rely more on wheeled mobility at home, school, and in the community. Children may achieve self-mobility using a power wheel chair.

Level 5

Physical impairments restrict voluntary control of movement and the ability to maintain antigravity head and trunk postures. All areas of motor function are limited. Functional limitations in sitting and standing are not fully compensated for through the use of adaptive equipment and assistive technology. At level V, children have no means of independent mobility and are transported. Some children achieve self-mobility using a power wheelchair with extensive adaptations.

Distinction Between Levels:

Distinctions between Levels I and II:

Compared with children in Level I, children in Level II have limitation in the ease of performing movement transitions; walking outdoors and in the community; the need for assistive mobility devices when beginning to walk; quality of movement; and the ability to perform gross motor skills such as running and jumping.

Distinction between Levels II and III:

Differences are seen in the degree of achievement of functional mobility. Children in Level III need assistive mobility devices and frequently orthoses to walk, while children in Level II do not require assistive device after age 4.

Distinctions between Levels III and IV:

Differences in sitting ability and mobility exist, even allowing for extensive use of assistive technology. Children in Level III sit independently, have independent floor mobility, and walk with assistive mobility devices. Children in Level IV function in sitting (usually supported) but independent mobility is very limited. Children in Level IV are more likely to be transported or use power mobility.

Distinction between Level IV and V:

Children in Level V lack independence even in basic antigravity postural control. Self – mobility is achieved only if the child can learn how to operate an electrically powered wheelchair

Evaluation of Children with Cerebral Palsy:

Evaluation of a child with cerebral palsy (CP) requires a multidisciplinary approach with a team of

professionals comprising of a pediatrician or pediatric neurologist, occupational therapist, a physiotherapist, child psychologist, and a social worker. The assessment is necessary to confirm the diagnosis, determine the cause, assess the motor function and associated problems. The diagnosis of CP is clinical but selected investigations may be required for determine the cause. Evaluation includes assessment for common medical problems of childhood particularly nutritional disorders and assessment of family functioning.

Additional disabilities are common. Routine assessment of vision and hearing is required in children with CP. Since CP is a changing disorder, some limitations may not be evident early in life but manifest in the school age or later. The evaluation of a child with CP is an ongoing process and should be a part of continuing care as

the child grows from infancy to adolescence ***(Freeman , 2005).***

Abstract Strategies for the early detection and diagnosis of cerebral palsy include multiple measures of the underlying brain abnormalities and their neuro-developmental consequences. These measures can be grouped into the categories of pathogenesis, impairment, and functional limitation. Neuroimaging techniques are the most predictive measures of pathogenesis of cerebral palsy in both the preterm and term infant. Measures of neurological impairment focusing on muscle tone, reflexes, and other features of the neurological examination are poorly predictive in the first months of life. Detection of functional limitations manifested by motor developmental delay is sensitive and specific for later cerebral palsy, but not until well into the second six months of life ***(Freeman , 2005).***

Abnormal spontaneous general movements in the infant 16 to 20 weeks postterm and earlier reflect functional limitations in the first months of life and have been shown to predict later cerebral palsy. Recognition of abnormal spontaneous general movements may improve early detection and diagnosis of cerebral palsy if these techniques can be successfully incorporated into organized follow-up programs and developmental surveillance ***(Ashwal , 2004).***

The diagnosis of CP is essentially clinical and involves detailed prenatal, natal, and postnatal history and careful physical and neurolodevelopmental examination *(Wollach , 1997).*

Signs Useful in Early Diagnosis of CP ***(Singhi et al., 2002)***:

1. Warning Signs:

- Lack of alertness.
- Decreased spontaneous mobility.
- Stereotyped abnormal movements.
- Constant fisting after 2 months of age.
- Poor quality of sleep.

2. Abnormal Signs:

- Reduced head circumference.
- Delayed social smile.
- Excessive extensor tone,dystonia.
- Primitive reflexes persisting beyond six months.

Primitive reflexes are:

- Asymmetric tonic neck reflexes (ASTNR).
- Symmetric tonic neck reflexes
- Moro reflex.
- Tonic labyrinthine reflex.

Postural reactions:

- Foot placement.
- Parachute reaction.

The following figures will show some of the primitive reflexes.

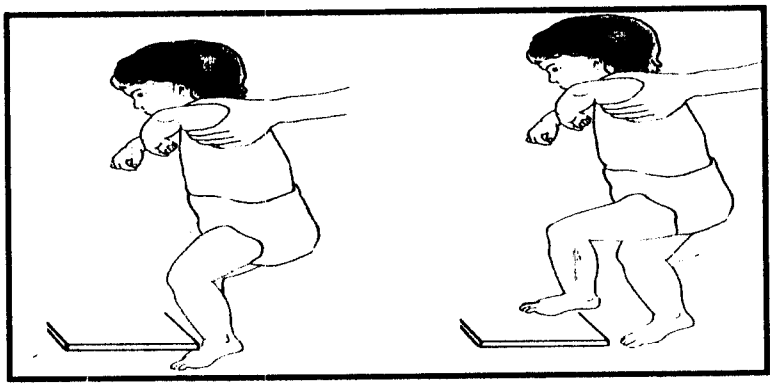

Figure (4): The foot placement reaction or step reflex.

(Freeman,2005)

It is initiated with the child held under the arms or by the chest. When the dorsum of the foot is stimulated at the edge of a table, the child will flex the hip and knee, simulating a stepping action.

Figure (5): The tonic labyrinth reflex.

(Freeman,2005)

The baby with abducted shoulders, flexed elbows, adducted extended hips, and extended knees and ankles. This posture primarily occurs with the baby in the supine position.

- Persistent asymmetric tonic neck reflex (ATNR)
- Delayed appearance of postural reflexes and development milestones
- Persistent asymmetry in posture, movement and reflexes.

3. Associated Signs:

- Occulovisual problems: visual problems, persistent squint.
- Lack of auditory response.

Early Motor Pattern Profile (EMPP)

It is a group of common neurological findings that have been organized into a systemic format to provide an objective picture of child's neurological status ***(Andrew , 1996).***

The 15 items of EMPP are:

1. Head Lag- pull to sitting position from supine, and assess alignment of head with trunk.
2. Slip through–support in vertical suspension with hands in axillae, and assess the need for lateral pressure to prevent the child from slipping through.
3. A stasis – place in supported standing and assess weight bearing.
4. Hip abduction – with leg extended, flex foot at ankle and assess resistance.
5. Ankle dorsiflexion – with leg extended, flex foot at ankle and assess resistance.

6. Deep tendon reflexes – assess the response at ankle.

7. Asymmetric tonic neck reflexes –flex head to one side in supine and observe position of extremities.

8. Tonic labyrinthine Reflex – place hands under shoulders in supine, lift slightly and observe efforts of flex forward.

9. Equilibrium in sitting –support in sitting on lap, shift weight to one side, observe efforts to maintain a neural position.

10. Protective extension – pushes to one side in sitting and observe efforts to stop falling with lateral propping.

11. Fisting – hands remain tightly clenched at rest.

12. Shoulder retraction – arms flexed and shoulders retracted posteriorly in sitting.

13. Tonic extension – backward thrusting in sitting or lifted from supine.

14. Scissoring – legs adduct in a scissoring motion in vertical suspension or standing

15. Equines – up on toes in supported standing.

A three point scale was used for each EMPP item, a score of 2 was given if the abnormality was severe or present all the time, a score of 0 was given if the abnormality was never present, a score of 1 was given if the abnormality was inconsistent or partial. The optimal cutoff score at six months between 9 and 10 at which the positive predictive value was 89.4, sensitivity was 87.1 and specificity was 97.8. The optimal cutoff score at 12 months was between 3-4 at which the positive predictive value was 91.0, sensitivity was 91.5 and specificity was 97.9 ***(Andrew , 1996).***

Investigations Needed for Children with CP:

1. Brain MRI and CT scan.
2. Audiological investigations: audiometry ,otoacoustic emissions, brainstem evoked audiometry (ABR).
3. Ophthalmic examination: may reveal chorioretenitis suggestive of cytomegalovirus, rubella, toxoplasmosis infection.
4. EMG to differentiate between CP and myopathy.
5. EEG if there are symptoms suggesting seizure activity.
6. Color Doppler imaging of intracranial vessels in neonates.

7. Laboratory Investigations:

- In attempt to find the cause e.g. thyroid function, serum uric acid is indicated in children who have hypotonia. To rule out other causes of hypotonia like Lesch-Nyhan syndrome (X-linked uric aciduria enzyme deficiency) ***(Hladniku, 2008)***.
- Metabolic investigations and quantitative amino acids in children with hypotonia.
- Serological tests to identify congenital infections.
- Serum copper ceruloplasmin determinations in children with unexplained choreoathetosis.

Treatment of Cerebral Palsy:

Multi disciplinary team is needed in rehabilitation of children with cerebral palsy. Care providers are roughly organized around the education system, primary medical care provider, the cerebral palsy specialized medical team, and community support services. Significant overlap and good communication provide the best resources to the child and the family.

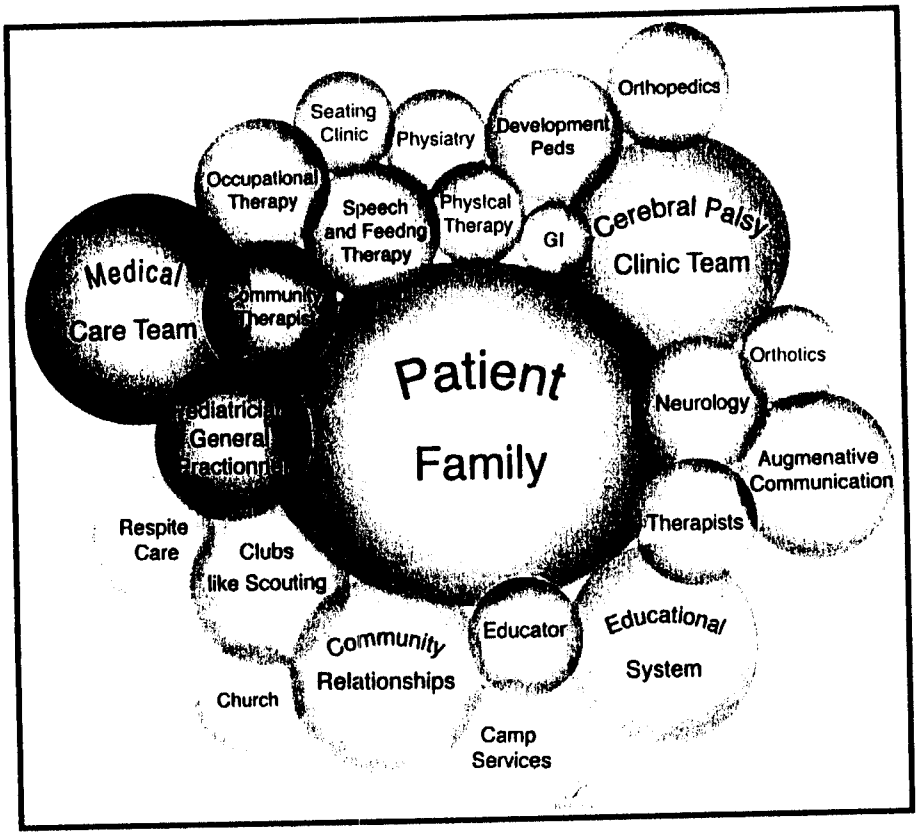

Figure (6): Shows a large and extensive care team surrounds the family with a child who has cerebral palsy ***(freeman, 2005).***

The overall objective of treatment in CP is to improve function and prevent deformity. The improvement in function, if they can walk must mean that as a result of treatment they can walk further, faster, or more efficiently, i.e. using less energy.

Prevention of deformity is important as otherwise there is deterioration in function. Not all deformity needs treatment and the straightening of the child with CP may seriously decrease the function ***(Freeman , 2005).***

Physiotherapy, Exercise and Mobility:

Many techniques are used to decrease the muscle tone e.g. positioning to reduce primitive reflexes, the local application of ice or vibration to spastic muscles, the avoidance of pressure over the extensor surfaces.

The value of physiotherapy is to prevent the gross contractures which can result from absence of therapy. Also physiotherapy can be used in conjunction with a knowledge of appliances, the management of postoperative cases, serial plastering, splinting, and the prevention of positional deformity.

Many children will develop independent walking. This may be with an abnormal gait or with aids such as tripods, or a rollator, other children may only be able to achieve very limited independent mobility, for example go on and off the toilet, in and out of bath or around the house, whilst they remain too slow and ponderous for independent ambulation. A child with CP may become mobile in the

swimming bath and be able to ride a tricycle before he can walk.

Mobility depends on the type of CP and the age at which certain milestones are achieved ***(Miller , 2005)***.

Orthotics:

Night splints can be expected to work in the same way as physiotherapy i.e. stretching a muscle over a prolonged period. the muscle needs to be relaxed during stretching so, sleep, medication or botulinus toxin may allow a period of stretch without the muscle fighting back: it is thought that at least 8 hours of stretching over 24 h period are necessary to encourage increase in muscle length.These measures can also be used to improve the position of the wrist by gradually increasing the range of dorsiflextion ***(Rang , 2000).***

Medications:

The marked extensor spasms which occur with the onset of the dystonic type of rigidity in the small baby may make handling very difficult. As soon as the infant is put into the bath, opens his bowels or experiences anxiety, rigid arching of the back occurs. This can often be lessened by small regular doses of benzodiazepine. Over the years many drugs have been claimed to reduce the amount of spasticity in muscles ***(Hawamdeh , 2007).***

*** Baclofen:**

It reduces the severity of spasticity and the increased muscle tone of dynamic deformities. It will not affect fixed deformities for which some form of plastering or orthopedic surgery is usually required. The main difficulty with drugs such as baclofen is knowing the age at which treatment should start, whether they are safe drugs during the period of rapid brain growth in the first four years of life, whether they influence synaptic development and whether they effect sufficient improvement in function to warrant medication in young children over many years. The decrease in muscle tone may result in child developing different deformities such as increasing internal rotation or being unable to stand when previously he could ***(Hawamdeh , 2007).***

*** L-Dopa:**

May have beneficial effect in specific circumstances. It has been claimed that L dopa will occasionally produce dramatic effect in children with athetoid CP. The majority of cases may show little effect and there may be an increase in the involuntary movement as a side effect ***(Hawamdeh , 2007).***

*** Intrathecal baclofen:**

Baclofen, a GABA receptors agonist first used in treatment of spasticity in1997s, it reduce the dynamic sensitivity, reduce muscle spasm ***(Miller et al., 2005).***

*** Botulinum toxin:**

The use of botulinum toxin, which began with treatment of blepharospasm, laryngeospasm, and torticollis in patient suffering from dystonia, appears ideal for the management of dynamic deformities and postures. It inhibit the release of acetylcholine from the presynaptic nerve ending of the neuromuscular junction, This effect reaches a maximum over a few weeks but lasts for several months, It has the potential benefit of being repeatable if and when signs recur. An additional advantage is that since the muscle is final common pathway of all motor activity, botulinum toxin abolishes voluntary and involuntary movements, dystonia, spasticity and reflex excitability. It is therefore suitable for the treatment of dynamic hypertonus of any origin. However, by definition, treated muscles will become weak, if not paralyzed ***(Hawamdeh et al., 2007).***

Surgery:

Orthopedic surgery in CP is used to improve ambulation, improve seating, help nursing and to treat pain or discomfort. Deformity which might lead to irreversible loss of function may be prevented e.g. scoliosis, hip dislocation with secondary acetabular dysplasia ***(Farmer et al., 2007).***

Summary of treatment in Cerebral Palsy:

1. Physical therapy
2. Orthosis – sol and hinged
3. Plaster immobilization – serial casting
4. Drugs – Diazipam, Beclofen oral and intrathecal, dantrolene, L- Dopa
5. Botulinum toxins – intramuscular injection
6. Functional electrical stimulation
7. Orthopedic surgery – single and multiple soft tissue release with or without bone surgery
8. Selective dorsal rhizotomy.
9. Aids for posture and mobility
10. Positioning program
11. Neurosurgery

12. Psychotherapy, feeding program.

Principles of Surgery in Cerebral Palsy:

- Surgery will not make a child weak.
- Surgery may prevent a child from ever walking.
- Deformity need not stop a child walking.
- Tenotomy may relieve spasticity but grossly weakens the muscle
- Surgery is better for fixed deformity.
- Surgery has little part to play in dystonia, athetosis, or ataxia
- The primary aim is to improve function, not increase range at a joint
- Combined iliacus and psoas surgery may stop child walking.
- There is a high chance of recurrence if operation undertaken under the age of 4 years.
- Always use intense postoperative physiotherapy.
- If the operation is for pain e.g. in hip, the outcome must be absence of pain, not radiological correction of deformity.
- Start at the top of the patient's body and work down.

- Surgery in one joint will affect distant joint which may not be predictable.
- Soft tissue surgery alone will not correct a bony deformity or dislocation.

Prognosis for Walking in Child with Cerebral Palsy:

The ambulatory potential of children with CP is a concern of both parents and health professional involved in CP children's treatment.

There are many factors which may help the physician in prediction of walking in children with CP, one of these factors is the type of CP:

Table (1): Shows prediction of walking in relation to types of Cerebral Palsy.

Type	Prediction
Hemiplegia	100%
Diplegia	90%
Ataxic diplegia	88%
Dyskinesia	80%
Quadriplegia	18%

((Badell, 1985).

On the basis of the type of CP, children with spastic hemiplegia have the prognosis for becoming ambulatory

(100 %). They are followed closely by children with spastic diplegia, whose outcome as favorable is greater than 85 % of the cases. The prognosis is less positive for children with spastic quadriplegia. SO, the type of CP serves as a descriptor of the degree of the child's motor involvement (***(Badell, 1985)***.

Other factor affecting the prediction of walking in CP children is the gross motor skills of the child ***(Campos, 1994)***:

Table (2): Prognosis of walking in Cerebral Palsy children according to gross motor skills.

Sit supported before 2 Yrs	97%
Sit unsupported 2-4 Yrs	50% walk(less efficiently)
Sit unsupported after 4Yrs	3% walk unaided

There is a relationship between the age of acquisition of various gross motor skills and ultimate ambulatory status. The sitting (defined as the ability to maintain sitting without support when placed) by 2 years of age is associated with a good prognosis for ambulation. However, not sitting by 2 years does not preclude achievement of ambulation, but makes it substantially less likely. The maintenance of sitting requires adequate trunk control and balance to sustain the upright posture. As these are motor

skills essential for ambulation, children's sitting ability would be a good indicator of their ambulatory potential.

Prognosis for Walking According to the Presence of Primitive Reflexes and Postural Reflexes *(Badell, 1985):*

The persistence of primitive reflexes and the absence of postural reactions are associated with a poor prognosis for ambulation. The critical age for examining for these signs to determine ambulatory potential is two years old. These been integrated into volitional movements at an earlier age, may interfere with the development of more advanced gross motor skill. ASTNR, Moro reflex are the most consistently with the ability to ambulate.

CHAPTER (II)

BONE GROWTH

Growth and development describe the changes an individual progresses through from conception until death. Growth is defined as a change in physical size of the organism as a whole or any of its parts, Example of such growth may be an increase in height, an increase in weight, or an increase in head circumference ***(Johnston, 2008)***.

Skeletal growth and development is a complex process that includes not only the obvious increase in size but also changes in skeletal maturation. Changes in size occur with linear growth at epiphyses and increase in diameter under the periosteum ***(Caetano, 2007).***

Skeletal maturation refers to the development of the epiphysis and the primary and secondary ossification centers. Skeletal maturation is most commonly assessed with radiographic appearance of growth centers of the hands and wrist, but the radiographic appearance of growth centers in other regions, including the knee ***(Anat, 1954),*** pelvis ***(Charles C Thomas, 1979),*** and ankle ***(Acheson, 1957),*** may also be used to assess skeletal maturity.

Growth of the individual may be divided into three major intervals: infancy, childhood and adolescence. Each intervals is distinguished by its own characteristic rate of growth ***(Karberg , 1985).***

Physiologic Age Assessment:

Bones in the skeleton may be analyzed throughout an individual's life from birth, through skeletal maturation, finalized with the end of life ***(Greulich, 1959).*** Chronological age, or person's age in calendar years, serves as the standard by which most of persons reach maturity. However, this measurement often dose not adequately reflect a person's biological maturity or developmental status, particularly when considering those periods of infancy and childhood. The majority of children may be seen as "average maturers", with a strong association between their chronological and biological ages. Some children, however, may appear developmentally delayed. Conversely, "early maturers" is the term that describes those children whose developmental growth precedes their chronological age ***(Tanner, 1962).***

The skeleton has been chosen in the past merely because radiographic technique provides a ready, easily applicable and noninjurious method of determination. Skeletal age thus becomes a measure of bodily maturation and not a goal in itself ***(Todd, 1937).*** Skeletal age or

biological age, also termed "developmental age and physiological age, reflects the level of maturity achieved by the individual.

Average bone or skeletal ages illustrate the maturation status in which normal children, male and female, match up with their corresponding calendar or chronological age ***(Jimenez, 1996).*** Similar to the growth of organs in human body, bones of the skeleton progress through their morphological development at different points in time. Knowing initiation times and morphological changes of bones in the hand and wrist provide a means of relating skeletal age to chronological age.

Bone Age (BA):

Is a way of describing the degree of maturation of a child's bones. As a person grows from fetal life through childhood, puberty, and finishes growth as a young adult, the bones of the skeleton change in size and shape. These changes can be seen by x–ray.

The bone age of a child is the average age at which children reach this stage of bone maturation ***(Donoghue, 2002)***.

Physiology of Bone growth:

At birth, only the metaphyses of long bones are present, the long bones are those that grow primarily by elongation at one end of the growing bone. The long bones include the femurs, tibias, and fibulas of the lower limbs, the humeri, radii, and ulnas of the upper limbs and the phalanges of the fingers and toes. The other primary skeletal component of height is the spine and skull ***(Green ,2004).***

It is important to know the sites and time of formation of primary and secondary ossific centers to determine the bone age. Figure 7 will show formation of ossific centers.

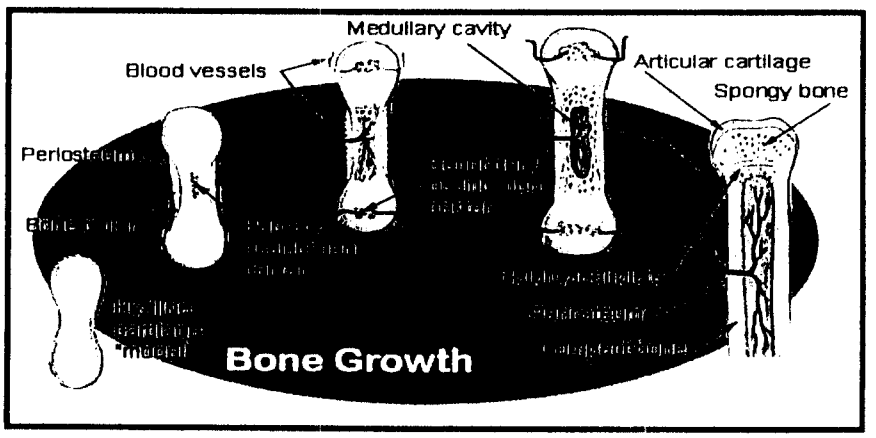

Figure (7): Formation of the primary ossification centers, secondary ossification centers and the growing of bone mass.

(http://www. web.books.com/Bone development.htm)

As a child grows , the epiphysis become calcified and appear on the X – ray separated by a layer of invisible cartilage from the diaphysis, where most of the growth is occurring.

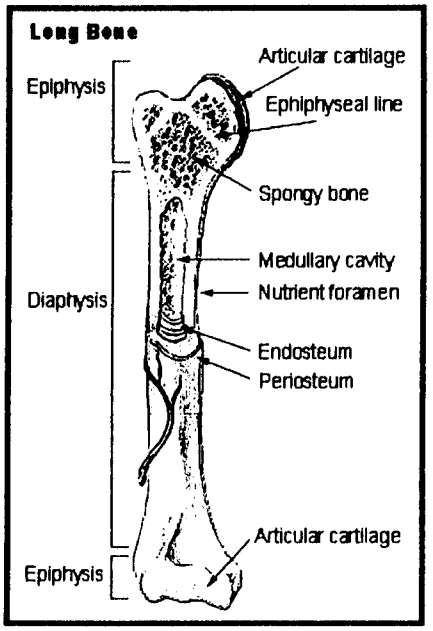

Figure (8): Increase in length of long bones at the epiphysial end and increase in mass below the periosteum.

(http://www.heightforum.com/upload-images/bone_stru.)

As sex steroids levels rise during puberty, bone maturation accelerates. As growth nears conclusion and attainment of adult height, bones begin to approach the size and shape of adult bones. The remaining cartilaginous portions of the epiphyses become thinner. As these cartilaginous zones become obliterated, the epiphyses are said to be closed and no further lengthening of the bones

will occur. A small amount of spinal growth concludes growth *(Bertelloni , 1998).*

There is a sequence of appearance of centers of ossification, and fusion of epiphysis according to which the bone age is calculated.

Appearance of Ossific centers:

a. **At Birth:** there are 6 centers present at birth:

1. Distal end of the femur
2. Proximal end of tibia
3. Talus
4. Calcaneous
5. Cuboid
6. Head of humerus

b. **Carpal Bones:** They are 8 in number in each hand:

- 1st carpal center appears within 1st 6 months
- 2nd carpal center appears within 2nd 6 months
- Then one carpal center roughly appears each year, except for the eighths center which is delayed to the age of 12Y

Table (3): Number of carpal bones in relation to age in years .

Age in years	Number of bones
2 years	3 carpal bones
3years	4 carpal bones
4years	5 carpal bones
5years	6 carpal bones
6years	7 carpal bones
7years	8 carpal bones

(Corpus, 2000).

Skeletal Bone Age Assessment:

Bone age assessment is a procedure frequently performed in pediatric radiology. Based on a radiological examination of skeletal development of the left hand and wrist, bone age is assessed and then compared with the chronological age. A discrepancy between these two values indicates abnormalities in the skeletal development. The procedure is often used in the management and diagnosis of endocrine disorders and it can also serve as an indication of the therapeutic effect of treatment. Generally, it can indicate whether the growth of a patient is accelerating or decreasing. In many cases the decision whether to treat a

patient with growth hormones depends on the outcome of this test ***(Charles , 1979)***.

This radiological examination of left wrist and hand is universally used due to its simplicity, minimal radiation exposure, and the availability of multiple ossification centers for evaluation of maturity.

Automatic skeletal age assessment using Computer – Assisted Diagnosis module(CAD) has the potential to reduce the time required to examine the image and to increase the reliability of the analysis ***(Zhang , 2007)***.

Clinical Methods:

The main clinical methods for skeletal bone age evaluation are the Greulich and Pyle **(GP)** method. ***(Greulich, 1971)*** and The Tanner and Whitehouse **(TW2)** method ***(Tanner, 1975)***.

There are several differences between the two methods. The Gerulich and Pyle method is most widely used in The Netherlands. This is mainly because the Gerulich and Pyle method is faster than the TW2 method. However, research has shown that the two methods produce different values for skeletal age and these differences are significant in clinical practice. The TW2 method is the more reproducible of the two, and also potentially more accurate ***(Johnson, 1989)***.

Both methods rely on radiographs taken from the left hand.

The Greulich and Pyle Method:

In 1929 preliminary studies were started at the Western Reserve University School of Medicine in Ohio. These studies were the base for long term investigation of human growth and development. A large number of children of different ages were enrolled in the study. These children had radiographs taken on their left shoulder, elbow, hand, hip, and knee. In the first postnatal year, an examination was conducted every three months, from twelve months to five years they were examined each 6 months and annually thereafter. In total the study ran from 1931 until 1942.

In 1937 an atlas, "Atlas of Skeletal Maturation of the Hand" was published by **Todd *(1937)*.** This atlas was based on a part of the data collected in the above mentioned study. Greulich and Pyle based their atlas partly on the atlas by ***Todd (1937)***. Most institutions use a more rapid modified version of the original which is also potentially less accurate ***(Bocchi, 2003)***.

In order to determine the skeletal age using the modified ***Greulich and Pyle method (1971)*** one uses the atlas that they have developed. The sex of the patient is one

of the most important pieces of information, because females develop quicker than males. The atlas is divided into two parts, one for the male patients and one for the female patients. Each part contains standard radiographic images of the left hand of children by chronological age. The first step in an analysis is to compare the given radiograph with the image in atlas that corresponds closest with the chronological age of the patient. Next one should compare it with adjacent images representing both younger and older children. When comparing the radiograph against an image in the atlas there are certain features a physician should use as maturity indicators.

These features vary with the age of the child. In younger children the presence of certain carpal or epiphyseal ossification centers are often pointers for the physician about the skeletal age of a child. In older children the shape of the epiphyses and the amount of fusion with the metaphyses is a good indicator of skeletal age.

Once the atlas image that most resembles the radiograph is found, the physician should conduct a more detailed examination of the individual bones and epiphyses. When the physician is sure that the matching radiograph has been found, he can find the skeletal age printed at the top of the page.

Tanner and Whitehouse Method:

The TW2 method does not use a scale based on the age, rather, it is based on a set of bone's standard maturity for each age population. In details, in the TW2 method twenty regions of interest (ROIs) located in the main bones are considered for the bone age evaluation. Each ROI is divided into three parts: epiphysis, metaphysis, and diaphysis, it is possible to identify these different ossification centers in the phalanx proximity.

The development of each ROI is divided into discrete stages and each stage is given a letter (A,B,C,D ...).

A numerical score is associated with each stage of each bone. By adding the scores of all ROIs, an overall maturity score is obtained. For TW2 method three scores systems have been developed:

- TW2 20 Bones: characterized by twenty bones including the bones of the first, third and fifth fingers and the carpal bones.
- RUS: considers the same bones of the TW2 method except the carpal bones.
- CARPAL: considers only the carpal bones.

Factors Affecting Bone Development:

Normal bone growth and development is affected by factors including:

1. Genetic factors
2. Nutrition, appropriate levels of some nutrients and hormones (e.g., vitamin D, calcium, estrogen, parathyroid hormone),
3. Mechanical loading through weight bearing and muscle tension.

In weight-bearing bones, where locomotion efficiency depends, in part, on bone mass, dynamic strains are essential to maintain bone mass.

Dynamic strains are repetitive forces that cause minute deformation of the bone. Activity level has been found to be a major determinant in the development of BM (bone mass).

Disuse, decreased activity, and non-weight bearing have been shown to precipitate a loss of 0.4% to 0.6% per month in adults without developmental disabilities. The effects of mechanical forces on the development and remodeling of the skeleton have been studied extensively.

Clinical application of bone age readings:

An advanced or delayed bone age doesn't always indicate disease or (pathologic) growth. Conversely, the bone age may be normal in some conditions of abnormal growth. Children don't mature at exactly the same tempo. Just as there is wide variation among the normal population in age of losing teeth or experiencing the first menstrual period, the bone age of healthy child may be a year or two advanced or delayed.

An advanced bone age is common when a child has had prolonged elevation of sex steroid levels, as in precocious puberty or congenital adrenal hyperplasia. The bone age is often marginally advanced with premature adrenarche, when a child is overweight from a young age or when a child has lipodystrophy.

Bone maturation is delayed with the variation of normal development termed constitutional delay of growth and puberty, but delay also accompanies growth failure due to growth hormone deficiency and hypothyroidism.

Skeletal Maturation in Children with Cerebral Palsy:

Cerebral palsy is a diagnostic term used to describe a group of motor syndromes caused by developmental, genetic, metabolic, ischemic, infectious, and other acquired etiologies ***(Krigger , 2006).***

In addition to abnormalities of speech, vision, intelligence, and neurological problems, children with CP have significant alterations in skeletal growth and development. In addition, low bone mineral density and decrease linear growth rate in CP patients have been described in multiple previous studies ***(Henderson , 2005).*** These changes have been attributed to both nutritional and non nutritional factors such as poor feeding, altered pubertal progression, growth hormone deficiency, as well as functional and disease severity ***(Coniglio , 1996).***

Skeletal maturation has also been shown to be altered in children with CP, but data are conflicting ***(Gilbert , 2004).*** Several studies have reported delay in bone age relative to chronological age, whereas others have found there to be no difference between bone age and chronological age, but others have observed a large prevalence of significantly advanced skeletal maturation ***(Henderson , 2005).***

Most of these studies have focused on non ambulatory children with severe CP. However, ambulation has shown to be a critical factor that improves both bone growth and quality in CP children ***(Baer , 1997).*** Many studies were done to determine the bone density and bone mass in CP children and to find causes of alteration of bone density in those children. Osteopenia in children with CP was attributed to a combination of factors including

immobility ***(Wilmshurst , 1996),*** low intake of minerals ***(Slemenda , 1991)***, and abnormal vitamin – D metabolism in those treated with anticonvulsants ***(Chung ,1996).***

Skeletal health in children is made up of a number of factors including the overall pattern of the skeleton, the appropriate size and shape of individual bones, bone mass, bone metabolism and turnover ***(Bertelloni , 1998).***

A markedly advanced or delayed skeletal maturity may raise questions as to hormonal or other systemic disturbances that require further investigation in the individual patient. Such disturbances are often associated with other effects on bone that require attention.

Skeletal growth and development may be significantly altered in children with CP. Diminished linear growth ***(Samson, 1989)*** and low bone density ***(Henderson , 2002)*** have been well documented in children with moderate to severe CP. Both nutritional and non nutritional factors contribute to growth disturbances in these children ***(Shapiro , 1989),*** including poor feeding that results in malnutrition ***(Sullivan , 2002),*** altered pubertal progression ***(Worley , 2002)*** and growth hormone deficiency ***(Coniglio, 1996).***

There have been concerns that delayed bone age may result in reduced final height and bone mass.

The correlation of reduced skeletal maturity with reduced height and bone density is not surprising and it reflects also the small bone size. A well reported feature of reduced muscle activity is reduced bone size, reduced stature and overall bone dimensions in CP would therefore be expected ***(Henderson , 2005).***

The use of anticonvulsant medication, is associated with a reduction in bone mass in children who are not ambulant. Anticonvulsant medication may accelerate the metabolism of vitamin D and vitamin D intake from diet and sunlight exposure may be reduced in this population, increasing the risk of fractures ***(Bischof ,2002).***

Assessing vitamin D status in children who are immobilized with CP should be a routine event with appropriate supplements being given to all affected children. The American Academy of Pediatrics recently suggested that all members of the population should receive 200 IU /day of vitamin D irrespective of race or age ***(Greer , 2004).***

Such a dose would be unlikely to accelerate any immobilization – induced calciuria.

There is no doubt that exercises increase bone size in proportion to an increase in muscle cross – sectional area. Physiotherapy is routinely used to maintain range of motion

in children with CP. and studies using vibrating plates suggest that the action of such micro-strain directly on bone can increase bone mass in the appendicular skeleton ***(Caulton , 2004).***

There are suggestions however, that over – vigorous physiotherapy could put patient at risk for fractures at the ends of long bone and certainly, physiotherapy regimens should be tailored to the state of the individual patient.

In short term, skeletal health in children with CP might be judged in terms of propensity to fractures, and on the metabolic side the development of osteomalacia or rachitic changes. Also, skeletal maturity need to be assessed regularly in these patients during the planning of limb lengthening procedures and when information on growth potential in individual limb is required ***(Erickson , 2003)***.

PATIENTS AND METHODS

The present study was conducted on children attending the day care in Prince Sultan Rehabilitation Center in Saudi Arabia during the period between January 2008 to May 2008.

Forty children were recruited according to the following inclusion criteria:

Inclusion criteria:

1. Children of preschool and school age, with age ranged between 3 – 12 years old.
2. Both genders.
3. Cases diagnosed as CP
4. A written consent from the parents was obtained.

Exclusion Criteria:

1. Chronic systemic disease which may affect general health and growth of the child.
2. Motor disabilities other than CP.

Study Design:

Descriptive study.

Ethical Issues:

A written consent was taken from parents before each child was recruited in the study.

Method:

All patients were subjected to the following:

1. Thorough history taking laying stress on perinatal, natal and postnatal incident in an attempt to find the cause of CP ***(Miller et al., 2005)***.

2. Clinical examination, both general and neurological examination, laying stress on anthropometric measures include BMI which can be calculated as weight over height , the presence or absence of congenital abnormalities, abnormal postures, abnormal movements, deformities, muscle power, tone and deep tendon reflexes to determine the type of CP and its effects on the child.

3. The use of Gross Motor Function Classification System (GMFCS) which is used to classify children according to their age specific gross motor activity

and functional characteristics into five levels, from I to V, level I being the mildest in the following age group : up to 2 yrs , 2-4 yrs , 4-6 yrs and between 6-12 yrs . For each level , separate descriptions are provided ***(Palisano et al., 1997)***.

4. Bone age determination: by plain X- ray on left wrist and hand done to all children included in the study. Interpretation of X – ray was done to determine the skeletal maturation and bone age according to ***Greulich and Pyle (1971)*** using atlas of skeletal development. The focus of the bone age analysis centered on determining whether the tempo of growth in the patient with CP is altered in the different types of CP.

Statistical Analysis:

The data were coded, entered and processed on an IBM-PC compatible computer using *SPSS* (version 15). The level $P < 0.05$ was considered the cut-off value for significance.

The following tests were done:

Chi-Square test X^2 was used to compare between groups with different types of CP according to muscle tone, convulsions, between groups with different types of CP according to GMFCS, between gender, different types of CP, and GMFCS according to delayed bone age and normal bone age.

Fisher exact test was performed in table containing value less than 5.

Student's *t-test* was used to assess the statistical significance of the difference between male and female cases as regards the difference between bone age and chronological age and in comparison between chronological age, height, bone age and difference (BA-CA) according to delayed bone age and normal bone age.

Correlation analysis: assessing the strength of association between two variables. Correlation between chronological age and bone age.

Limitation of the Study:

1. Number of the patients available.
2. Failure to obtain written concept taken from parents.
3. Cost.

RESULTS

Results of the present study are shown in tables (1) to (16) and figures (1) to (10):

Table (1): Frequency of descriptive and clinical data of CP children included in the study

		N	%
Sex	**Male**	18	45.0
	Female	22	55.0
Age group	**Preschool**	20	50
	School	20	50
Type of CP according topography	**Diplegia**	19	47.5
	Hemiplegia	2	5.0
	Quadriplegia	17	42.5
	Triplegia	2	5.0
Type of CP according to muscle tone	**Spastic**	28	70
	Dyskinetic	2	5
	Mixed	10	25
Level of dysfunction according to GMFCS	**II**	11	27.5
	III	9	22.5
	IV	6	15.0
	V	14	35.0
Ambulation	**Ambulant(level II,III)**	20	50
	Non ambulant (IV,V)	20	50
Convulsions	**Present**	12	30
	Absent	28	70

From table (1) it can be seen that the most common type of CP according to the topography was the diplegic type which constitutes 47.5% of the studied cases and the least type was the triplegia, and the most common type according to the muscle tone was the spastic type which

formed 70% of cases in the present study and the least type was the dyskinetic type which formed 5% of cases. There was 50% ambulant and 50% non ambulant CP children included in the study .

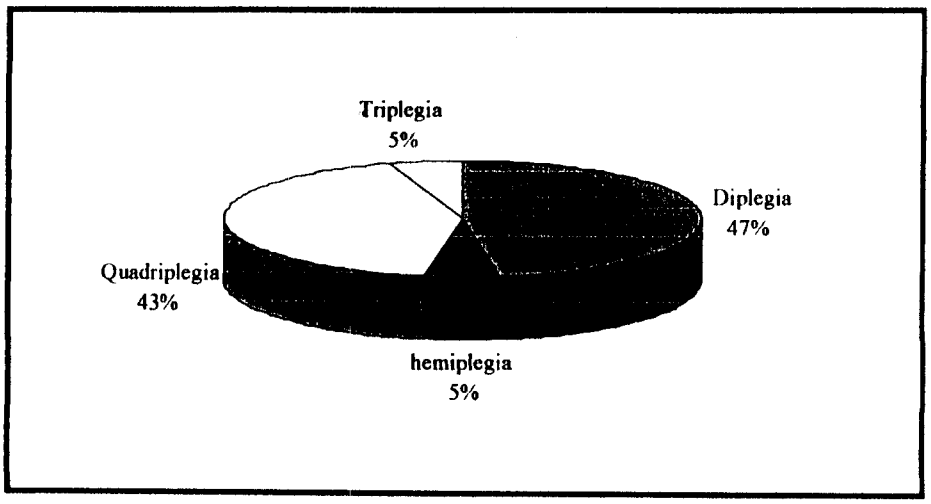

Figure (1): Types of CP in children included in the study.

Table (2): Frequency of delayed skeletal maturation in CP children included in the study

		N	%
Skeletal maturation	**Delayed**	21	52.5
	Normal	18	45.0
	Advanced	1	2.5

Results of the present study showed that 52.5 % of children with CP had delayed bone age according to plain X-ray , while 45 % of CP children had normal skeletal maturation .

Table (3): Comparison between preschool and school age children included in the present study.

		Pre-school			School	X2	P	Sig.
		N	%					
Sex	Male	9	45.0%	9	45.0%	0	1.00	NS
	Female	11	55.0%	11	55.0%			
Types of CP	Diplegia	9	45.0%	10	50.0%	2.11	0.55	NS
	Hemiplegia	2	10.0%	0	0.0%			
	Quadriplegia	8	40.0%	9	45.0%			
	Tripegia	1	5.0%	1	5.0%			
Level of dysfunction according to GMFCS	II	3	15.0%	8	40.0%	6.00	0.11	NS
	III	7	35.0%	2	10.0%			
	IV	2	10.0%	4	20.0%			
	V	8	40.0%	6	30.0%			
Skeletal maturation	Delayed	8	40.0%	13	56.0%	3.08	0.21	NS
	Normal	11	55.0%	7	35.0%			
	Advanced	1	5.0%	0	0.0%			

Chi-square test

There was no statistical significant difference between the two groups as regards the distribution of different types of CP, sex, GMFCS and skeletal maturation (P>0.05).

Table (4): Descriptive statistics (Z- score) of height SDS, head circumference SDS and BMI SDS.

	N	Minimum	Maximum	Mean	Std. Deviation
z-score height	40	-2.07	2.74	0.00	0.99
z-score HC	40	-1.52	2.37	0.00	1.002
z-score weight	40	-1.28	2.008	0.03	0.99
z-score BMI	40	-1.63	2.27	0.04	1.012
Valid N (listwise)	40				

Table (5): Difference between mean chronological age and mean bone age, of the children included in the study.

	Mean	±SD	Minimum	Maximum
Chronological age in years	6.98	2.69	.60	13.00
Bone age in years	5.23	2.71	1.00	11.00
Difference (BA-CA)	-1.75	2.25	-8.40	2.40

From table (5) it can be seen that the mean chronological age was 6.98 while the mean bone age was 5.23.

Table (6): Comparison between chronological Age and bone Age in children with cerebral palsy.

	Cases		Paired Differences		*t*	P	Sig.
	Mean	±SD	Mean	±SD			
Chronological age	6.98	±2.69	1.75	±2.25	4.93	<0.0001	HS
Bone age	5.23	±2.71					

From table (6) it can be seen that there was a highly statistical significant difference between mean chronological age and mean bone age (P<0.0001).

Table (7): Anthropometric measures of CP children included in the study.

	Mean	±SD	Minimum	Maximum
Height	109.97	14.87	80.00	149.00
Weight	17.86	4.55	12.00	27.00
BMI	14.93	2.62	10.18	21.36
Head circumference	49.79	2.22	46.50	55.00

From table (7) it can be seen that maximum height was 149 cm and minimum was 80 cm while maximum weight was 27 Kg and minimum was 12 Kg while maximum BMI is 21.36 and the minimum was 10.18.

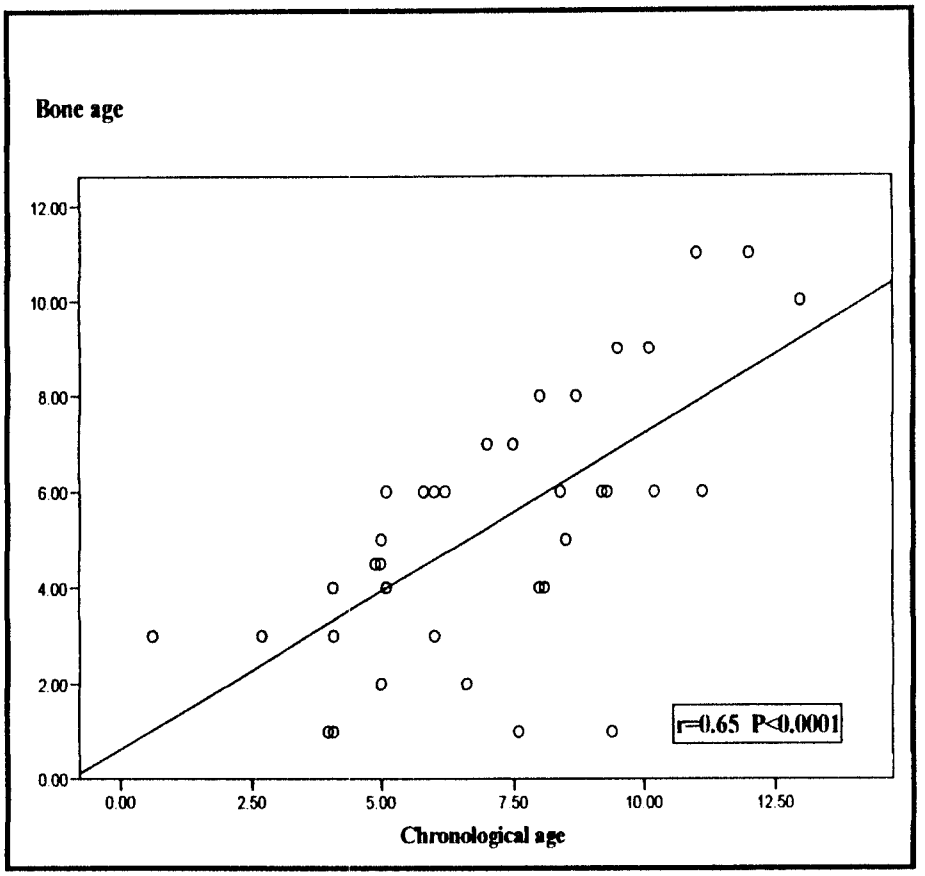

Figure (2): Scatter Diagram Showing Chronological Age and Bone Age of CP children included in the study.

From figure (2) it can be seen that there was a positive significant correlation between chronological age and bone age in CP children included in the study. ($P<0.0001$)

Table (8): Comparison between male and female CP cases as regards the difference between bone age and chronological age ,and other anthropometric measures .

	Male		Female		*t*	P	Sig.
	Mean	±SD	Mean	±SD			
Chronological age	6.37	±2.53	7.47	±2.77	1.29	0.20	NS
Bone age	3.75	±2.12	6.43	±2.57	3.54	0.001	S
Difference (BA-CA)	-2.62	±2.66	-1.04	±1.57	-2.35	0.02	S
Height	104.28	11.11	114.36	15.23	2.34	0.02	S
Weight	17.06	3.93	18.48	4.98	0.96	0.34	NS
BMI	16.01	2.79	14.04	2.15	2.51	0.02	S
Head circumference	49.28	1.56	50.18	2.58	1.27	0.21	NS

From table (8) it can be seen that there was no statistical significant difference between males and females cases as regards the mean chronological age ,weight and head circumference . (P>0.05).

Also male cases showed a statistically significant greater delay in bone age when compared to female cases ($P<0.05$).Also there was a statistical significant greater difference between bone age and chronological age in male cases than in female cases ($P<0.05$).

Female cases showed a statistically significant lower mean BMI and height when compared to female cases ($P<0.05$)

There was no statistical difference between male cases and female cases as regards the mean weight and head circumference ($P>0.05$)

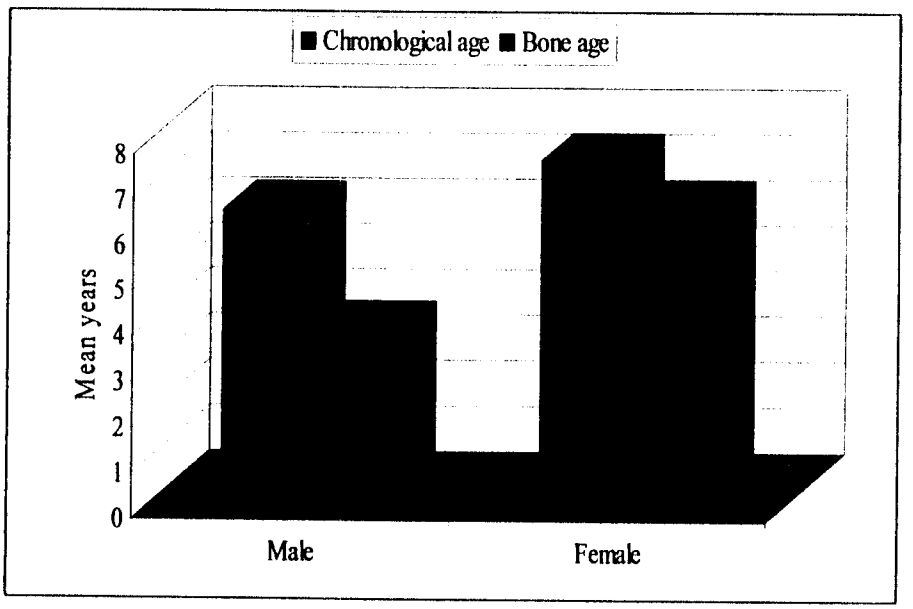

Figure (3): Comparison between chronological age and bone age in female and male CP children included in the study.

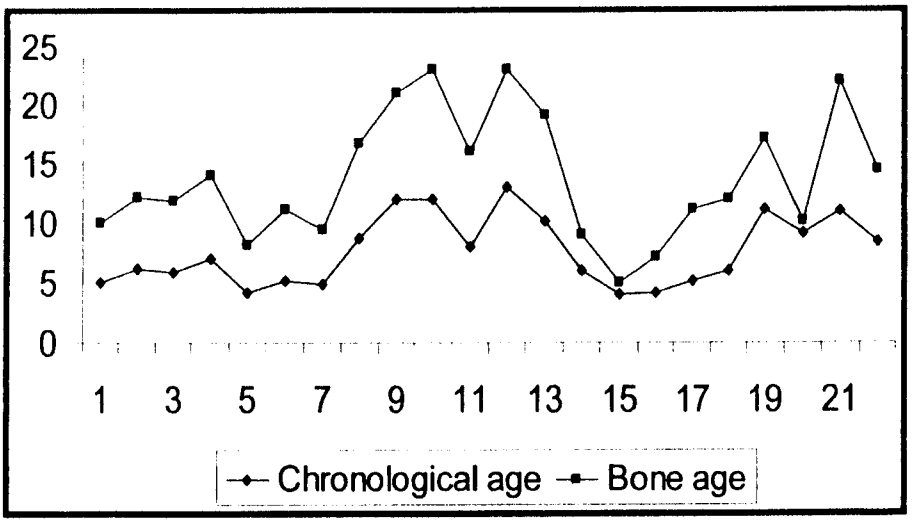

Figure (4): Relation between chronological age and bone age in female CP children included in the study.

From figure (4) it can be seen that there was delayed bone age in comparison to chronological age in female CP children included in the study.

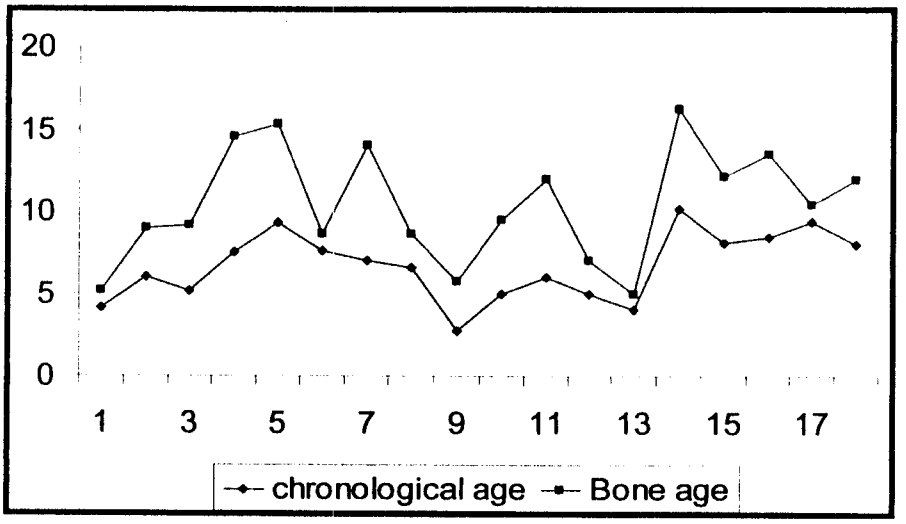

Figure (5): Relation between chronological age and bone age in male cases included in the study.

From figure (5) it can be seen that there was delayed bone age in comparison with chronological age of the male CP children included in the study.

Table (9): Classification of CP patients according to topography of CP in relation to muscle tone.

Tone	Diplegia		Hemiplegia		Quadriplegia		Triplegia	
	N	%	N	%	N	%	N	%
dyskinetic	0	0%	0	0%	2	11.8%	0	0%
Mixed	5	26.3%	0	0%	4	23.5%	1	50%
Spastic	14	73.7%	2	100%	11	64.7%	1	50%
χ^2=4.21 P=0.65								

From table (9) it can be seen that the most common type of CP was the spastic diplegic type which formed 73.7% of cases.

Table (10): Comparison between types of CP and the presence of convulsions.

Convulsions	Diplegia		Hemiparesis		Quadriplegia		Triparesis	
	N	%	N	%	N	%	N	%
-ve	14	73.7%	1	50%	12	70.6%	1	50%
+ve	5	26.3%	1	50%	5	29.4%	1	50%
χ^2=0.89 P=0.83								

There was no statistical significant difference between patients with different types of CP as regards the presence or absence of convulsions (P>0.05).

Table (11): Comparison between Difference of chronological age and bone age as regards the presence or absence of convulsions

	Convulsions			
	Negative	Positive	P	Sig.
	Median	Median		
Difference (BA-CA)	1.10	0.67	0.51	NS

Mann-Whitney test , Data were presented as median due to high variation among +ve cases of convulsions .

From table (11) it can be seen that there was no statistical significant difference between cases with and without convulsions as regards the difference between chronological age and bone age (P>0.05)

Table (12): Association between different types of CP and level of dysfunction according to GMFCS.

GMFCS	Diplegia		hemiplegia		Quadriplegia		*Triplegia*	
	N	%	N	%	N	%	N	%
II	8	42.1%	2	100%	0	.0%	1	*50%*
III	9	47.4%	0	0%	0	.0%	0	*0%*
IV	2	10.5%	0	0%	4	23.5%	0	*0%*
V	0	0%	0	0%	13	76.5%	1	*50%*
χ^2=37.80 P<0.0001								

GMFCS = Gross Motor Function Classification Scale.

From table (12) it can be seen that level IV and V of dysfunction according to GMFCS were more commonly associated with quadriplegia while level II and III of dysfunction according to GMFCS (mild affection) were commonly associated with CP hemiplegia and diplegia in the children included in the present study.

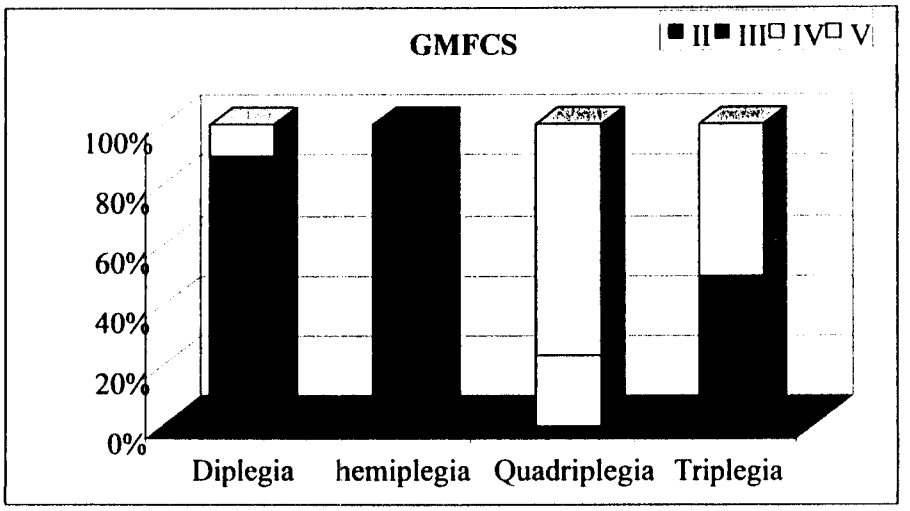

Figure (6): Association between different types of CP and the level of dysfunction according to GMFCS.

From figure (6) it can be seen that hemiplegia was less associated with severe level of motor dysfunction according to GMFCS among CP children included in the study, while quadriplegia was more associated with severe levels of motor disability.

Table (13): Comparison between gender, types of CP, and level of dysfunction according to GMFCS in relation to delayed and normal skeletal maturation.

		Delayed bone age		normal		χ^2	P	Sig.
		N	%	N	%			
Sex	Male	12	70.6%	5	29.4%	3.40	0.07	NS
	Female	9	40.9%	13	59.1%			
Types of CP	Diplegia	7	38.9%	11	61.1%	7.47	0.06	NS
	hemiplegia	0	.0%	2	100.0%			
	Quadriplegia	13	76.5%	4	23.5%			
	Triplegia	1	50.0%	1	50.0%			
Level of motor dysfunction according to GMFCS	II	6	54.5%	5	45.5%	8.98	0.03	S
	III	1	12.5%	7	87.5%			
	IV	3	50.0%	3	50.0%			
	V	11	78.6%	3	21.4%			

Chi-square test
NS: non-significant

From table (13) it can be seen that male CP cases showed a higher frequency of delayed bone age (more than one year) when compared to female CP cases, but the difference was not statistically significant (P=0.07).

Also it can be seen that there was no statistical significant difference between patients with different types of CP as regards the presence or absence of delayed skeletal maturation (P>0.05).

Also it can be seen that there was a statistical significant difference between level of dysfunction according to GMFCS as regards the presence or absence of delayed skeletal maturation (P=0.02).

Also, delayed bone age was significantly associated with grade V level of dysfunction according to GMFCS (P = 0.03).

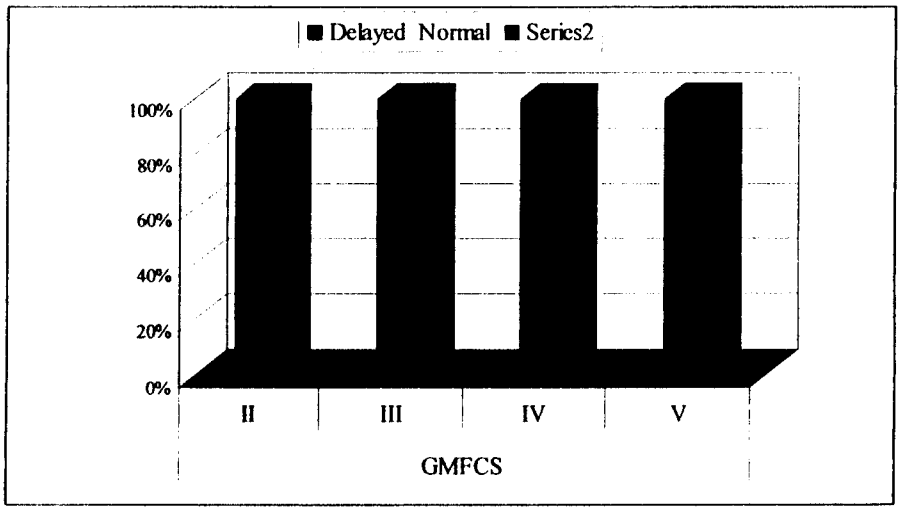

Figure (7): Comparison between levels of motor dysfunction according to GMFCS in relation to delayed skeletal maturation.

From figure (7) it can be seen that level V was the level most commonly associated with delayed skeletal maturation, showing that 78.6% of cases have delayed bone age.

Table (14): Relation between levels of motor dysfunction according to GMFCS and the delay of bone age.

	Difference (BA-CA)		F	P	Sig.
	Mean	±SD			
II	-1.49	2.10	1.29	0.02	S
III	-0.19	1.39	3.54		
IV	-1.64	2.32	-2.35		
V	-3.01	2.24			

GMFCS = Gross Motor Function Classification Scale
ANOVA Test

There was a statistical significant variation between difference of bone age and chronological age (BA-CA) and the levels of motor dysfunction according to GMFCS (p=0.02)

On performing multiple comparison between different levels a significant difference was detected between level III and level V of dysfunction according to GMFCS (P=0.003).

There was a statistical significant difference as regards delayed bone age and ambulation where greater bone age delay was more frequently associated with non ambulant CP children (Level IV and V of dysfunction according to GMFCS) than ambulant CP children (Level II and III of dysfunction according to GMFCS) and the difference was statistically significant (p= 0.02)

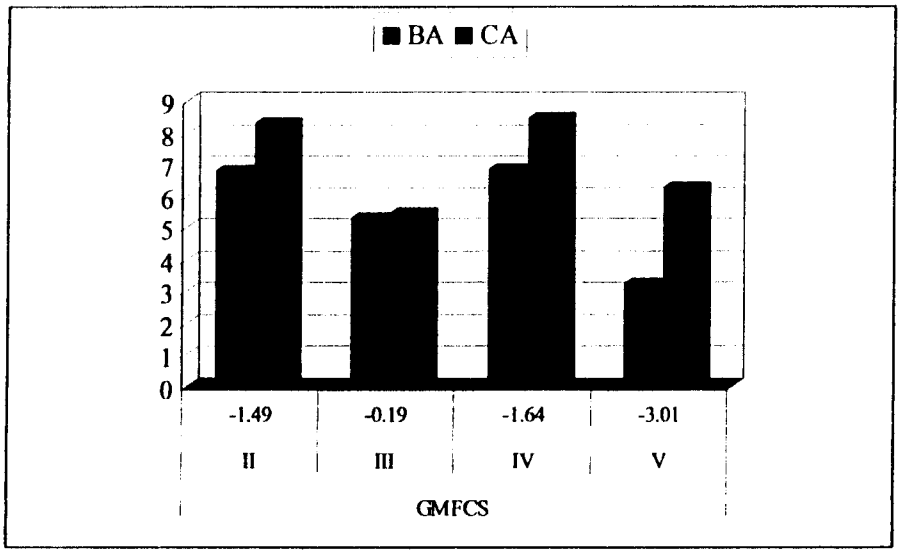

Figure (8): Relation between levels of motor dysfunction according to GMFCS and the delay in bone age.

From figure (8) it can be seen that greater bone age delay was more frequently associated with non ambulant CP children (Level IV and V of dysfunction according to GMFCS) more than ambulant CP children (Level II and III of dysfunction according to GMFCS) and the difference was statistically significant (p= 0.02)

Table (15): Relation between difference of bone age and chronological age (BA-CA) of CP children included in the study and different types of CP according to topography.

	Difference (BA-CA)		F	P	Sig.
	Mean	±SD			
Diplegia	-.92	1.66	3.52	0.02	S
Hemiplegia	.45	.64			
Quadriplegia	-2.72	2.27			
Triplegia	-3.55	4.31			

ANOVA Test

There was a statistical significant variation between difference of bone age and chronological age (BA-CA) of CP children included in the study and different types of CP according to topography (P =0.02).

On performing multiple comparison , quadriplegia showed a higher significant difference than diplegia triplegia and hemiplegia (P=0.01, 0.05).

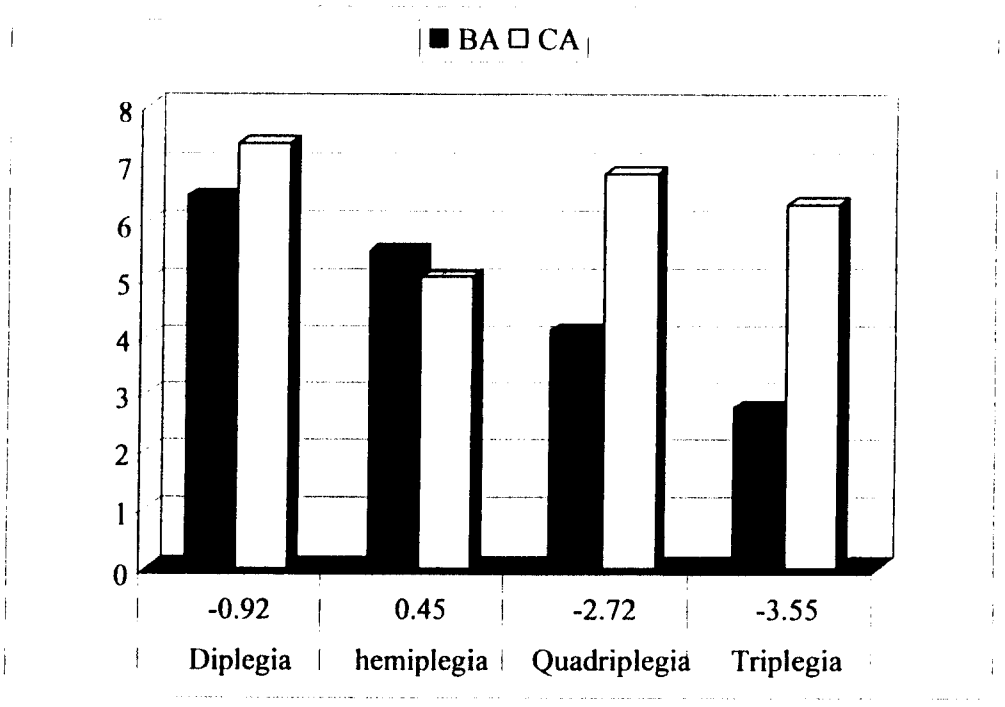

Figure (9): Relation between difference of bone age and chronological age (BA-CA) of CP children included in the study and different types of CP according to topography.

From figure (9), it can be seen that quadriplegia and triplegia were associated with more delay in bone age.

Table (16): Correlation between difference between bone age and chronological age (BA-CA) and anthropometric measures .

	Difference (BA-CA)		
	r	P	Sig.
Height	0.05	0.78	NS
Head circumference	0.19	0.24	NS
weight	0.36	0.02	S
BMI	0.47	0.002	S

Difference (BA-CA): difference (bone age-chronological age)
Pearson Correlation coefficient: r

There was no correlation between difference (BA-CA) and height and head circumference ($P>0.05$)

There was a positive correlation between difference of (BA-CA) and weight, and BMI ($P<0.05$)

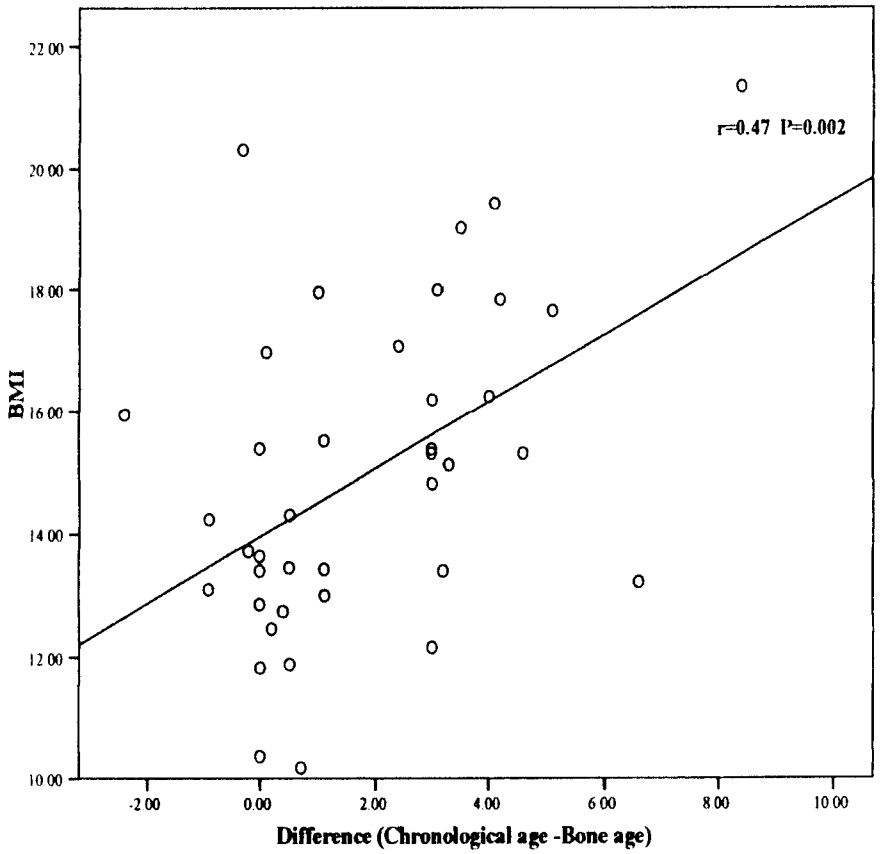

Figure (10): Scatter diagram showing difference (chronological age -bone age) and BMI

There was a positive correlation between (chronological age - bone age) difference and BMI , so, higher BMI was associated with bigger difference between chronological age and bone age (P=0.002)

Table (17): Comparison between groups of ambulant and non-ambulant CP children included in the study and the difference between bone age and chronological age

	Difference (BA-CA)		t	P	Sig.
	Mean	±SD			
Ambulant (26)	1.07	±1.97	2.82	0.008	S
Non-ambulant (14)	3.01	±2.24			

Student's t test

There was a statistical significant variation between difference (BA-CA) in ambulant and non-ambulant children ($P<0.05$)

Non-ambulant children showed a statistically significant higher difference between bone age and chronological age than ambulant cases ($P=0.008$) . So, non ambulant CP children had more delay in bone age than ambulant CP children in the present study .

DISCUSSION

Cerebral palsy is a childhood condition in which there is a motor disability caused by a static, nonprogressive lesion in the brain. The causative event has to occur in early childhood usually defined as less than 2 years of age ***(Miller , 2005)***. It is a common neurodevelopmental disorder of childhood with prevalence of 2 – 2.5 per 1000 live births ***(Johnson , 2008)***.

Understanding the medical and anatomical problem in the individual with CP is important, however, always keeping in mind the greater long term goal, which is similar to that for all normal children is to be independent in life ***(Miller , 2005)***.

The bone age of a child is the average age at which children reach this stage of bone maturation. ***(Donoghue , 2002)***.

Patients with CP have a high likelihood of under-going an orthopedic procedures, so, accurate prediction of skeletal maturity is especially important in this patient population. Determining skeletal maturation is important for planning the timing for surgical procedures such as limb lengthening.

The high prevalence of altered skeletal maturation in children with CP can be of direct clinical significance in situations where treatment decisions involve issues of "growth remaining". Altered skeletal maturation may be of indirect

clinical significance if it reflects problems such as malnutrition, poor general health, or endocrine dysfunction. Of particular relevance to the orthopedic care of these children is the potential relationship between altered skeletal maturation and other skeletal growth problems such as osteoporosis ***(Henderson , 2002).***

The purpose of this study was to assess skeletal maturation in children with CP using Greulich and Pyle Atlas of skeletal maturation .

Also, Gross Motor Function Classification System was used to assess the severity of motor dysfunction in every child included in the study . GMFCS is a classification according to severity of the motor impairment or resulting disability. This is a recently developed system which has been found to be a reliable and valid system that classifies children with CP by their age – specific gross motor activity. This GMFCS describe the functional characteristics in five levels, from I to V, level I being the mildest in the following age groups: up to 2yrs, 2 – 4 yrs, 4 – 6 yrs and between 6 – 12 yrs. For each level, separate descriptions are provided ***(Palisano , 1997).***

Children in level III usually require orthoses and assisting mobility devices, while children in level II do not require assisting mobility device after the age of 4 yrs. Children in level III sit independently, have independent floor mobility, and walk with assisting mobility devices. In level

IV, affected children function in supported sitting but independent mobility is very limited. Children in level V lack independence even in basic antigravity postural control and need power mobility.

In the sample of the study the most common type of CP according to the topography was the diplegic type which constitutes 47.5% of the studied cases and the least type was the triplegia, and the most common type according to the muscle tone was the spastic type which formed 70% of cases in the present study and the least type was the dyskinetic type which formed 5% of cases. There was 50% ambulant and 50% non ambulant CP children included in the study . Anthropometric measures and X-ray of left wrist and hand were taken to all children included in the study , it can be seen that the mean chronological age was 6.98 while the mean bone age was 5.23 (Table 1)

The mean height was 109.97 cm , mean weight was 17.86 Kg and mean BMI was 14.79 .

Results of the present study showed that 52.5 % of children with CP had delayed bone age , while 45 % of CP children had normal skeletal maturation (Table 2) . This result agreed with **(Ihkkan and Yalcin (2001) and Gilbert (2004)** who reported that skeletal age generally lags behind the chronological age of children with moderate to severe CP (level IV , V of motor dysfunction according to GMFCS)

Also , in the present study there was a highly statistical significant difference between mean chronological and mean bone age ($P < 0.0001$) (Table 6). This agreed with **(C Kong , 2007)** who stated that there was positive significant correlation between chronological age and bone age which was expected , as the child becomes older there will be increase in bone maturation . But in another study **(Clement, 2002)** it was found that no significant difference between a child's skeletal age and chronological age , however , there was a high prevalence of individual participants in whom skeletal age was advanced (7%) or delayed (10 %) relative to chronological .

Previous studies have demonstrated altered skeletal maturation primarily in non ambulatory children with moderate to severe CP, however, skeletal maturation in ambulatory CP patients has not been clearly defined ***(Henderson , 2005)***. This study found that 7% of non ambulatory CP patient had advanced bone age greater than 2 years when compared with chronological age ***(Henderson ,2005).***

In the present study ,there was a statistical significant difference as regards delayed bone age and ambulation where greater bone age delay was more frequently associated with non ambulant CP children (Level IV and V of dysfunction according to GMFCS) than ambulant CP children (Level II

and III of dysfunction according to GMFCS) and the difference was statistically significant (p= 0.02) (Table 14)

Also , there was a statistical significant difference between level of dysfunction according to GMFCS as regards the presence or absence of delayed skeletal maturation (P=0.02) (Figure 7) , more severe motor dysfunction was frequently associated with more delayed skeletal maturation .

There was a statistical significant variation between difference of bone age and chronological age (BA-CA) and the levels of motor dysfunction according to GMFCS (p=0.02) (Table 14)

On performing comparison a significant difference was detected between level III and level V of dysfunction according to GMFCS (P=0.003).

Also , it was found that level V of dysfunction according to GMFCS was commonly associated with delayed skeletal maturation, showing that 78.6% of cases have delayed bone age while level III was the least level associated with bone age delay showing that 12.5 % of cases have delayed bone age (Table 13). This was concordant with study done by **Gollapudi (2007)** who demonstrated that most of the ambulatory CP patients had advanced bone age compared to chronological age , level III of motor dysfunction according to GMFCS and low BMI significantly associated with advanced bone age in boys and girls (P<0.05).

In the present study ,there was a statistical significant difference as regards delayed bone age and ambulation where greater bone age delay was more frequently associated with non ambulant CP children (Level IV and V of dysfunction according to GMFCS) (Table 14)

Baer (1997) showed that bone age values in non ambulatory children were significantly lower than those in ambulatory children regardless of presence or absence of convulsions and regardless of anticonvulsive use , suggesting that ambulation is a critical factor in determining skeletal maturity .

In the present study , level IV and V of dysfunction according to GMFCS were more commonly associated with quadriplegia , while level II and III of dysfunction according to GMFCS were commonly associated with CP hemiplegia and diplegia in the children included in the study (Table 12).

Also , spastic hemiplegia was found to be less associated with severe level of motor dysfunction according to GMFCS among CP children included in the study, while spastic quadriplegia was more associated with severe levels of motor disability (Table12).

There was a statistical significant variation between difference of bone age and chronological age (BA-CA) of CP children included in the study and different types of CP according to topography (P =0.02) (Table 15) .

On performing comparison , quadriplegia showed a higher significant difference than diplegia and hemiplegia (P=0.01) .

This results showed difference from other study done by **C Kong (2007)** , where dyskinetic type of CP was found to be associated with more marked delay in bone age , although the results had to be interpreted with caution due to the relatively small number of children with dyskinetic CP .

In contrast, other studies have found that there is no difference regarding bone age and chronological age in CP children ***(Gilbert , 2004).***

In the present study , there was no statistical significant difference between males and females cases as regards the mean chronological age (P>0.05).

Also , it was found that male CP cases showed a higher frequency of delayed bone age (more than one year) when compared to female CP cases, but the difference was not statistically significant (P> 0.05)(Table 13) . This results was in consistent with the results of studies done by **Gollapudi and Norman (2007)** where they found that there was no significant difference in the alteration of skeletal maturation in both sexes .

Also male cases showed a statistically significant greater delay in bone age when compared to female cases (P<0.05).

Also there was a statistical significant greater difference between bone age and chronological age (BA-CA) in male cases than in female cases ($P<0.05$).

Male cases showed a statistically significant lower mean BMI and height when compared to female cases ($P<0.05$)

There was no statistical difference between male cases and female cases as regards the mean weight and head circumference ($P>0.05$) (Table 8) .

In the current study, there was no statistical significant difference between patients with different types of CP and the presence of convulsions ($P> 0.05$) (Table 10). Also , it can be seen that there was no statistical significant difference between cases with and without convulsions as regards the difference between chronological age and bone age ($P>0.05$) (Table 11)

The results were consistent with results of studies done by **Gollapudi (2007)and Baer (1997)** who showed that bone age values in non ambulatory children were significantly lower than those in ambulatory children regardless of presence or absence of convulsions and regardless of anticonvulsive use.

There was no correlation between (BA-CA) difference and height and Head circumference ($P>0.05$)

There was a positive correlation between (BA-CA) difference and weight ($P<0.05$) .

There was a positive correlation between (chronological age -bone age) difference and BMI , so, higher BMI was associated with bigger difference between chronological age and bone age ($P=0.002$) (Table 16).The results were in accordance with results done by **C kong (2007) and Worley (2006)** who demonstrated that low BMI was associated with better skeletal maturation .

In non ambulatory CP patients, two previous studies have found delayed bone age compared with chronological age ***(Ihkkan, 2001 and Kong, 1999).***

The delay in bone age may be multifactorial, it may be due to decreased physical activity, hormonal factors, nutritional factors which may affect the growing process of the child where many studies have focused on the effect of diet, vitamin D intake, calcium and phosphorus during early growth ***(Susan , 2003).***

In the current study, there was no statistical significant difference between patients with different types of CP and the presence of convulsions ($P> 0.05$).

Many studies were done to find the effect of anticonvulsants on the skeletal maturation and bone mineral density and it revealed that bone mass was adequate in children who have uncomplicated idiopathic epilepsy and who receive monotherapy with carbamazepine or valporic acid even for long time, but delay skeletal maturation and decrease bone mineral density were present in none ambulant children with epilepsy ***(Marion , 1992 and Gabriele, 2001)***.

In the current study, there was a statistical significant difference between GMFCS and the skeletal maturation ($p=0.03$) where, higher degree of motor dysfunction according to GMFCS, was found to be associated with more delay in skeletal maturation.

Many studies were done to find the relation between level of motor dysfunction according to GMFCS and the skeletal maturation resulted in statistically significant correlation between them, where GMFCS I, II, III which means ambulatory patient have better degree of skeletal maturation than level IV and V (non-ambulatory patient) ***(Gollapndi , 2007 and Goodgold, 2007)***.

SUMMARY

Cerebral palsy is a group of non progressive but often changing motor impairment syndromes secondary to lesions or anomalies of brain arising in early stages of its development.

It is the most common and costly form of chronic motor disability that begins in childhood. It is caused by damage to the motor control centers of the young developing brain.

Bone age is a way of describing the degree of maturation of a child's bones, it is the average age at which children reach this stage of bone maturation.

Skeletal maturation has been shown to be altered in children with CP, but data are conflicting. Several studies have reported delays in bone age relative to chronological age, whereas others, have found there to be no difference between bone age and chronological age, but others have observed a large prevalence of significantly advanced skeletal maturation.

In view of such data, the aim of the present study was designed to assess skeletal maturation using bone age in a sample of CP children. The present study was conducted on forty children recruited from day care in Prince Sultan Rehabilitation Center in Dammam in Saudi Arabia .

Plain X – ray of left wrist and hand was done to all children included in the study and bone age was assessed according to Greulich and Pyle atlas method.

The result of the present study revealed that, there was delayed bone age in comparison with the chronological age in % 52.5 of children with CP included in the study.

In the current study, there was a statistical significant difference between GMFCS and the skeletal maturation ($p=0.03$) where, higher degree of motor dysfunction according to GMFCS, was found to be associated with more delay in skeletal maturation where 78.6% of children with grade V in GMFCS with higher degree of motor dysfunction have delayed bone age while 54.5% of children with grade II in GMFCS with mild degree of motor dysfunction have delayed bone age .

There is a statistical significant higher difference between bone and chronological age in male cases than in female cases i.e., delayed bone age was more frequently associated in male gender (70.6%) than in female gender (40.9 %) CP patients.

In the present study, there was also a relation between ambulation and the degree of skeletal maturation where it could be found that non ambulatory CP children included in the study have more delayed skeletal maturation than the ambulatory CP children ($P<0.05$) .

In the present study, there was a statistical significant difference between patients with different types of CP and the levels of motor dysfunction according to GMFCS ($P < 0.0001$), where, quadriplegic type of CP was found to be more affected regarding the motor dysfunction level, where 76.5% of quadriplegic type are classified as grade V according to GMFCS which was the most severe type regarding motor disability .

CONCLUSION

In conclusion, delayed bone age was observed in children with CP , and this delay could be related to the level of motor dysfunction according to GMFCS . Children with severe level of motor dysfunction were found to have greater delay in skeletal maturation. Also, non ambulatory CP children (level IV and V) were found to have more delay in skeletal maturation when compared to ambulatory CP children (level II , III) included in the present study. Also, spastic quadriplegic type of CP was found to be associated with greater delay in skeletal maturation than other types of CP in children included in the present study.

RECOMMENDATIONS

In the view of the findings obtained in the present study.

It is recommended to put in mind that delayed bone maturation may be present in cases of CP, while making decisions regarding physiotherapy to the child or before doing any orthopedic surgery helping in the rehabilitation plan of the child.

Follow up is needed for children with CP by frequent X- rays of left wrist and hand to determine progress in bone age maturation.

Further studies are needed to determine the significance of nutritional and non nutritional factors on skeletal maturation in children with CP.

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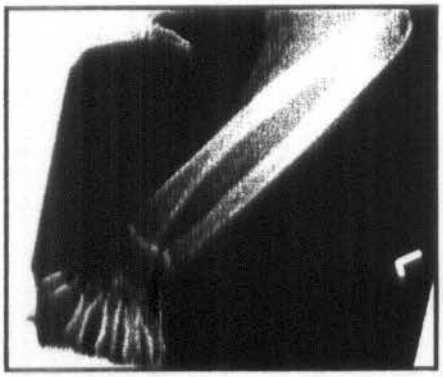
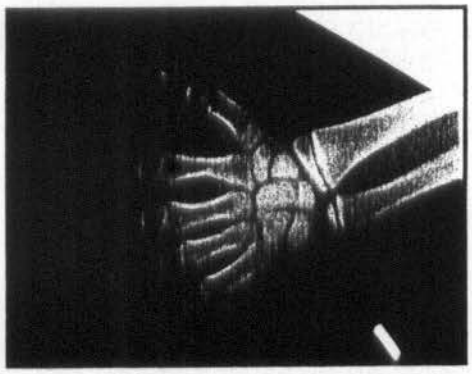


Case 1
Delayed bone age



Case 2
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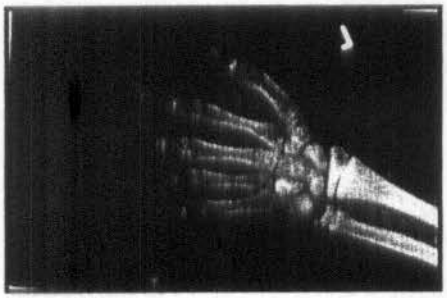

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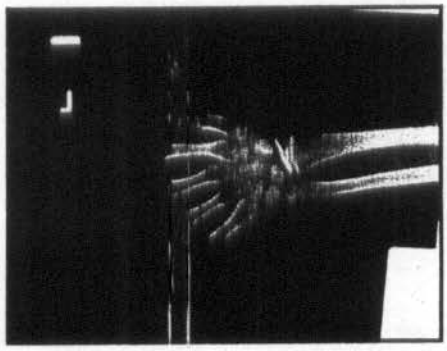

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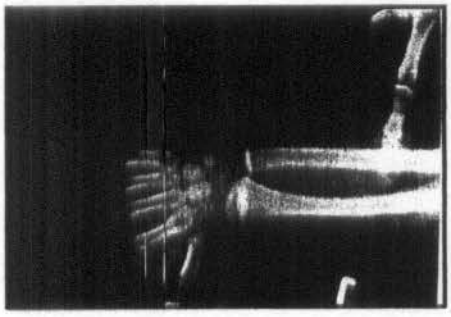

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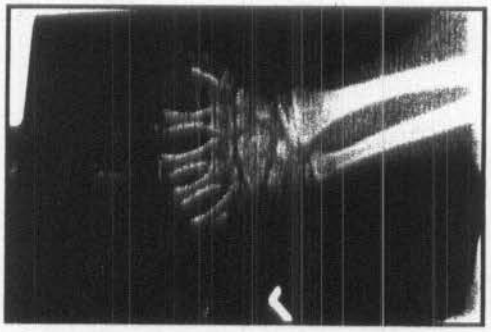
Delayed bone age



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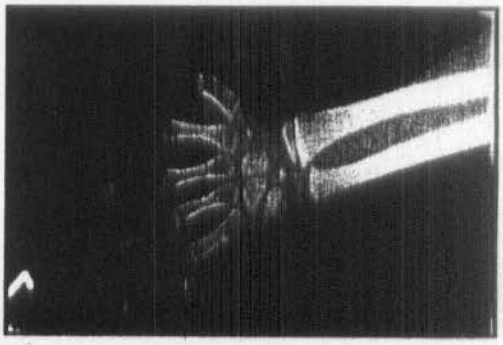

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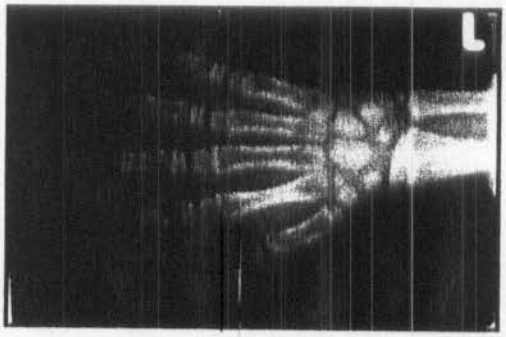
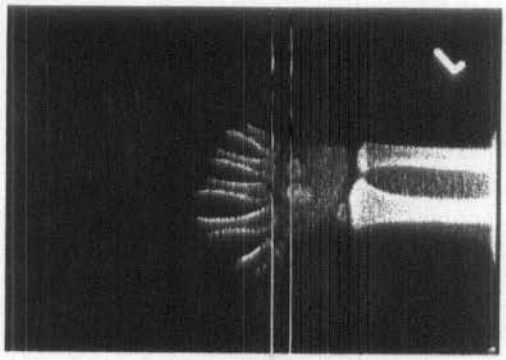
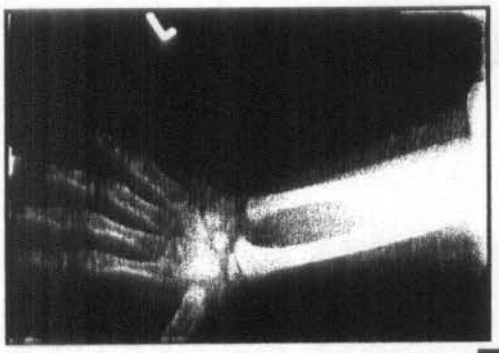
Delayed bone age



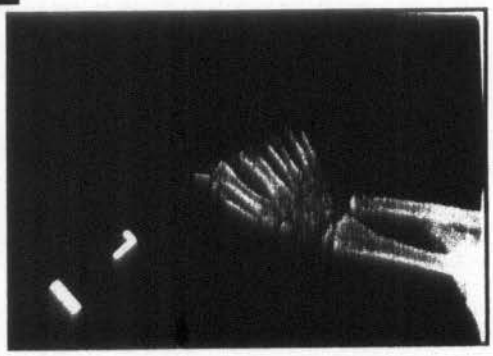
Case 8
Delayed bone age



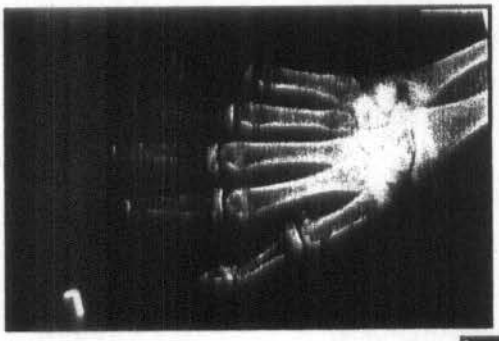
Case 9
Delayed bone age



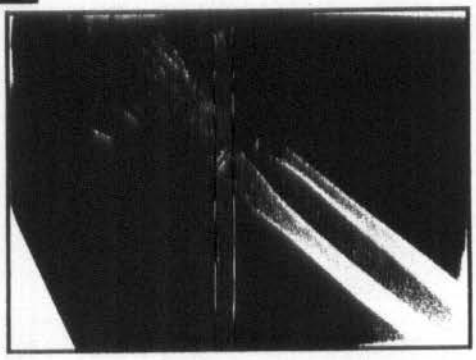
Case 10
Normal bone age



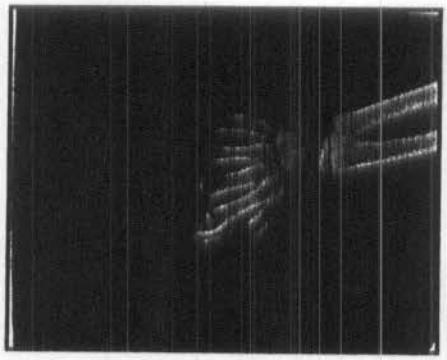
Case 11
Delayed bone age- wrist and bone deformity



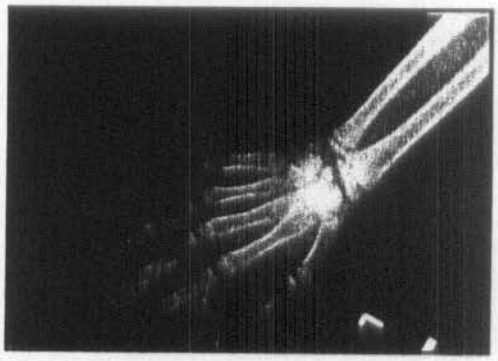
Case 12
Normal bone age



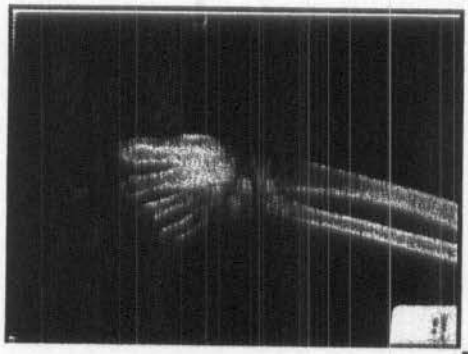
Case 13
Delayed bone age



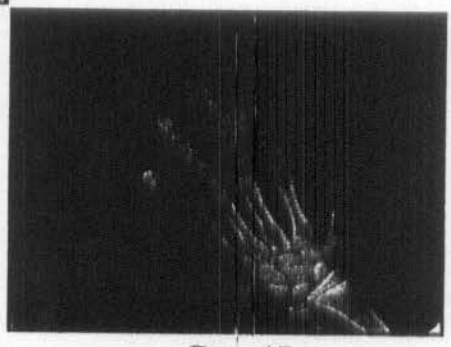
Case 14
Delayed bone age – deformity of Finger's bones



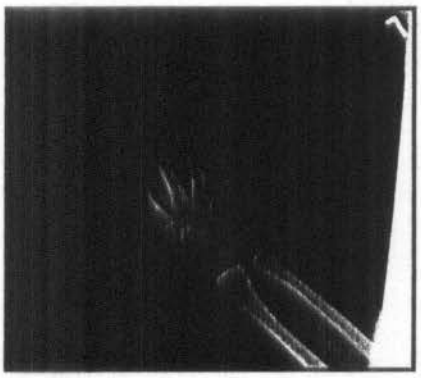
Case 15
Delayed bone age- bone deformity



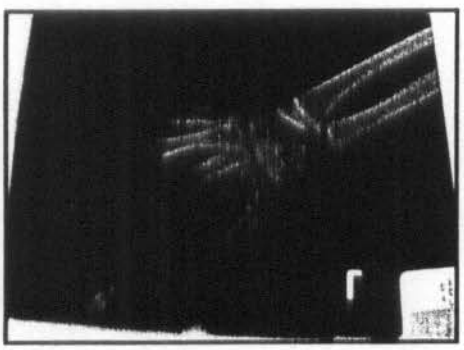
Case 16
Delayed bone age –bone deformity



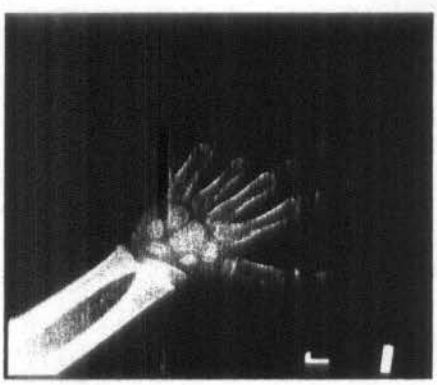
Case 17
Normal bone age – deformity of little finger's bone



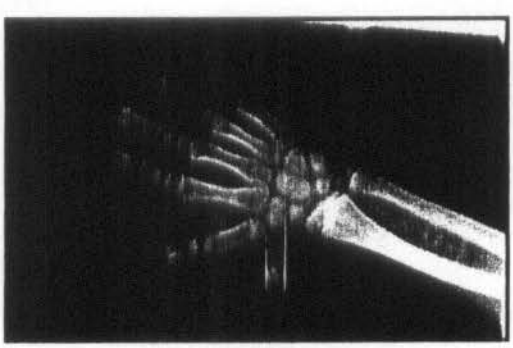
Case 18
Normal bone age



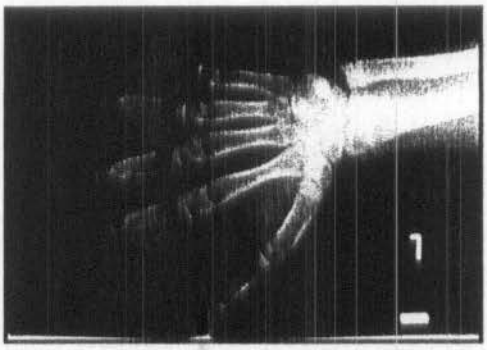
Case 19
Delayed bone age –bone deformity



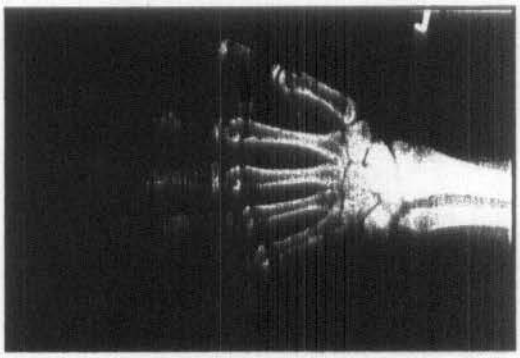
Case 20
Normal bone age



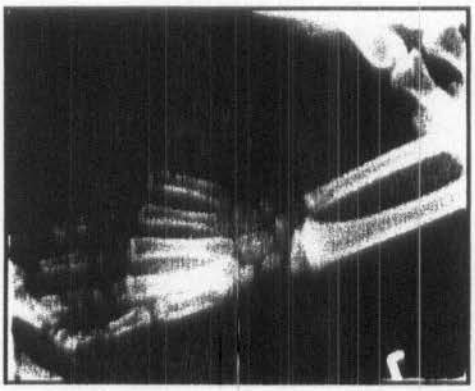
Case 21
Normal bone age



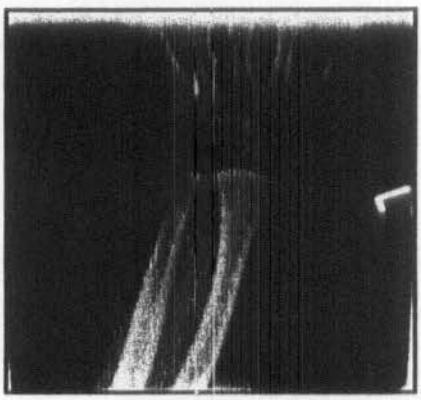
Case 22
Normal bone age



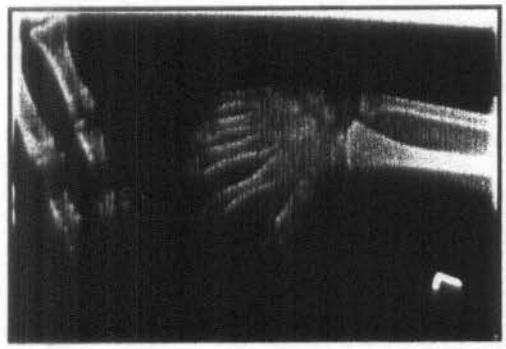
Case 23
Normal bone age



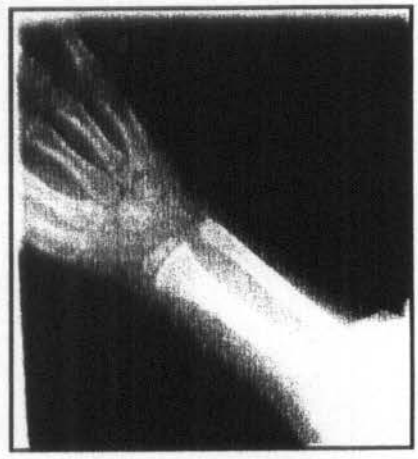
Case 24
Delayed bone age – bone deformity



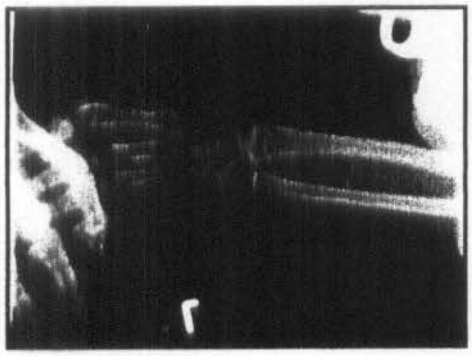
Case 25
Delayed bone age



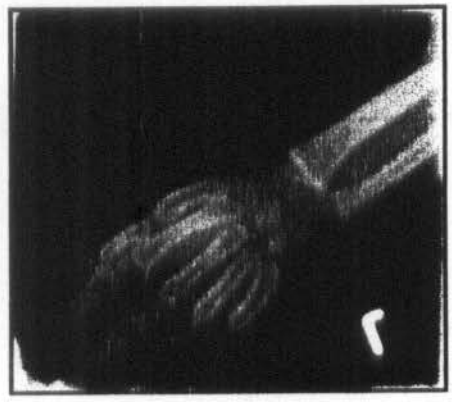
Case 26
Delayed bone age-bone deformity



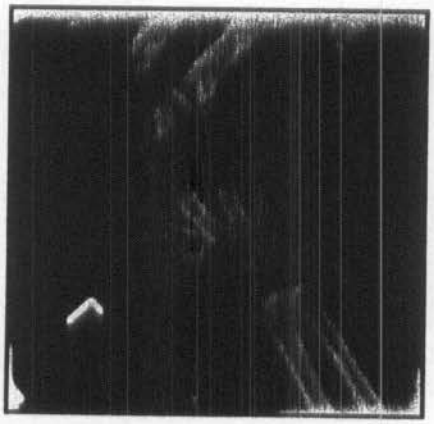
Case 27
Delayed bone age



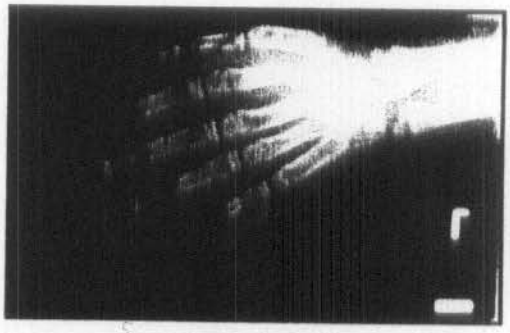
Case 28
Delayed bone age-bone deformity



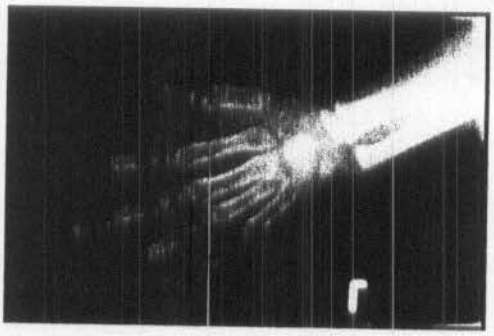
Case 29
Delayed bone age



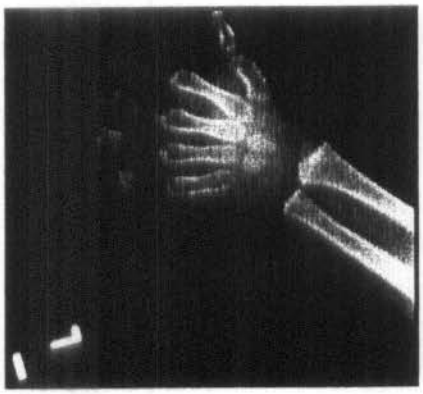
Case 30
Delayed bone age – bone deformity



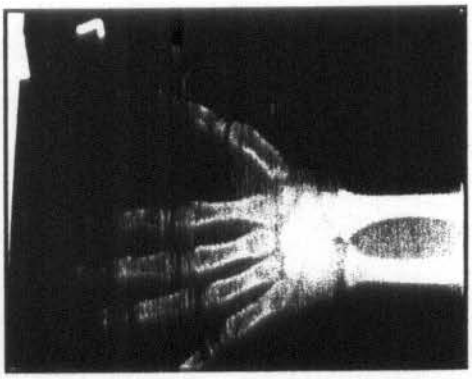
Case 31
Normal bone age



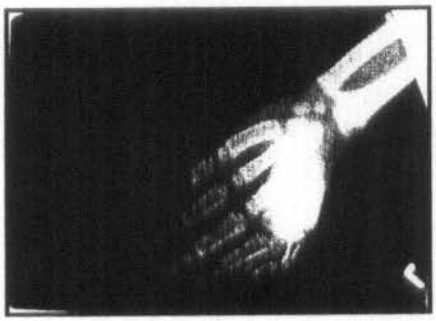
Case 32
Delayed bone age



X-ray show delayed bone age



X-ray of normal bone age



X-ray delayed bone age and deformity
of thumb bones.

Arabic Summary

الملخـــص العـــربي

المقدمة :

الشلل الدماغي هو اضطراب حركي ناتج عن ضمور في خلايا المخ حدث في وقت ما أثناء المراحل الأولي من النمو، فنتجت عنه اضـــطرابات حركية ثابتة و لكن قد تتغير صورتها مع مرور السنوات و نمو الطفل.

يحدث الشلل الدماغي بنسبة 2- 2.5 في كل 1000 مولود ، وهذه النسبة مقربة في كل دول العالم مع بعض الاختلافات البسيطة.

لا يوجد علاج شاف حتى الآن للشلل الدماغي و لكن الهـدف مـن العلاج والتأهيل في هذه الحالات هو تحسين القدرات الوظيفية الحركيـة، و يمكن تحقيق هدا الهدف بالتدخل المبكر في العملية التأهيلية، كمـا يجـب العمل مع الطفل والأسرة من خلال فريق متعدد التخصصات مثل: طبيـب الأطفال، والأعصاب، والعظام، والعيون، وأخصـائي العـلاج الطبيعـي والوظيفي والنطق، والأخصائي النفسي، أخصـائي السـمعيات و التغذيـة و الأخصائي الاجتماعي.

إن العمر العظمي هو متوسط العمر الذي يصل إليه الطفـل عنـد مرحلة معينة من نضوج الهيكل العظمي.

ولقد لوحظ أن هناك تغيرات تحدث في عظام الأطفـال المصـابين بالشلل الدماغي وقد أجريت عدة دراسات لمعرفة هـذه التغيـرات، و قـد اختلفت نتائج هذه الدراسات فمنها ما سجلت تأخر في نمو العظام و منها ما

سجلت تقدم في نمو العظام ومنها ماذكر أنه لا يوجد اختلاف مابين العمر الزمني وتطور العظام عند الأطفال المصابين بالشلل الدماغي.

الهدف من الدراسة :

أجريت هذه الدراسة لمحاولة معرفة التغيرات التي تحدث في العظام عند الأطفال المصابين بالشلل الدماغي 0

الطرق و الوسائل :

أجريت هذه الدراسة على عدد أربعون طفلا من الأطفال المصابين بالشلل الدماغي و الملتحقين بوحدة الرعاية النهارية بمجمع الأمير سلطان للتأهيل بالمملكة العربية السعودية و قد اجريت لهم أشعة سينية علي اليد اليسري بعد الحصول على موافقة كتابية من الأهل و تم تحديد عمر العظام لديهم مستخدمين طريقة أطلس جرلش و بايل. كما تم مقارنة عمر العظام مع العمر الزمني لكل طفل من الأطفال المشاركين في الدراسة.

النتائج :

كانت من نتائج هذه الدراسة وجود تأخر في نمو العظام عند معظم الأطفال المصابين بالشلل الدماغي بالمقارنة بالعمر الزمني لهم.

و لقد وجد أن هناك ارتباط بين التأخر في نمو العظام و مستوى الاعاقة الحركية كما تصنف بمقياس GMFCS والذي يتكون من خمس مستويات تحدد القدرات الحركية الكبرى للطفل.

وجد أن هناك ارتباط بين الفرق بين العمر الزمني و عمر العظـــام عند الطفل و درجة الإعاقة الحركية كما تصـــنف بمقيـــاس GMFCS فكلما زاد الفرق بين العمرين كلما زادت درجة الإعاقة.

وجد ارتباط أيضا بين الفرق بين العمر الزمني و عمر العظام عند الذكور و الإناث فقد وجد أن التأخر عند الذكور أكثر مـــن الإنـــاث فـــي الأطفال المشاركين في هذه الدراسة.

الخلاصة :

يعاني نسبة كبيرة من الأطفال المصابين بالشلل الدماغي من تأخر في نمـــو العظام و قد كانت هده النسبة 52.5 % من الأطفال الدين شملتهم الدراسة0

التوصيات :

توصي هده الدراسة بالأخذ في الاعتبار احتمالية وجود تأخر في نمو العظام عند الأطفال المصابين بالشلل الدماغي قبل اتخاذ قرار بأي تدخل جراحـــي للطفل كما توصي في هده الحالة بمتابعة نمو العظام لدى الطفل بعمل أشعة سينية على اليد على فترات متباعدة لتحديد نمو العظام 0

مستخلص

الشلل الدماغى هو اضطراب حركى ناتج عن ضمور فى خلايا المخ حدث فى وقت ما أثناء المراحل الأولى من النمو، فنتجت عنه اضطرابات حركية ثابتة ولكن قد تتغير صورتها مع مرور السنوات ونمو الطفل.

لوحظ أن هناك تغيرات تحدث فى عظام الأطفال المصابين بالشلل الدماغى وقد أجريت عدة دراسات لمعرفة هذه التغيرات، وقد اختلفت نتائج هذه الدراسات فمنها ما وجدت تأخر فى نمو العظام ومنها ما وجدت تقدم فى نمو العظام ومنها ما ذكر انه لا يوجد اختلاف ما بين العمر الزمنى وتطور العظام عند الأطفال المصابين بالشلل الدماغى.

وفى ضوء هذه الاختلافات، فقد أجريت هذه الدراسة لمحاولة معرفة التغيرات التى تحدث فى العظام عند الأطفال المصابين بالشلل الدماغى، وقد أجريت هذه الدراسة على عدد أربعون طفلا من الأطفال المصابين بالشلل الدماغى وقد أجريت لهم أشعة سينية على اليد اليسرى وتم تحديد عمر العظام لديهم مستخدمين طريقة أطلس جراش وبابل. كما تم مقارنة عمر العظام مع العمر الزمنى لكل طفل من الأطفال المشاركين فى الدراسة. .

كانت من نتائج هذه الدراسة وجود تأخر نمو العظام عند معظم الأطفال المصابين بالشلل الدماغى بالمقارنة بالعمر الزمنى لهم.

وجد أن هناك ارتباط بين الفرق بين العمر الزمنى وعمر العظام عند الطفل ودرجة الإعاقة الحركية كما تصنف بمقياس GMFCS فكلما زاد الفرق بين العمرين كلما زادت درجة الإعاقة.

وجد ارتباط أيضا بين الفرق بين العمر الزمنى وعمر العظام عند الذكور والإناث فقد وجد أن التأخر عند الذكور أكثر من الإناث فى الأطفال المشاركين فى هذه الدراسة

الكلمات الكاشفة

(نمو العظام – الشلل الدماغى – الإعاقة الحركية – طريقة أطلس جرلش وبايل)

شكر

اشكر السادة الأساتذة الذين قاموا بالإشراف وهم:

أ.د / هيام كمال مصطفى نظيف — أستاذ طب الأطفال بقسم الدراسات الطبية للأطفال – بالمعهد.

د / ريم بسيونى محمد — مدرس الأشعة – كلية طب – جامعة عين شمس.

ثم الأشخاص الذين تعاونوا معى البحث

وهم:

١-

٢-

٣-

وكذلك الهيئات الآتية:

١-

٢-

٣-

صفحة العنوان

اسم الطبيبة: إيمان خليل إبراهيم عبد الله

الدرجة العلمية: ماجستير

القسم التابع له: الدراسات الطبية

أسم المعهد: معهد الدراسات العليا للطفولة

الجامعة: عين شمس

سنة التخرج: ٢٠٠٩

سنة المنح: ٢٠٠٩

رسالة: الماجستير

اسم الطبيبة: إيمان خليل إبراهيم عبد الله

عنوان الرسالة: (دراسة التغيرات التى تحدث فى نمو العظام فى الأطفال المصابين بالشلل الدماغى)

أسم الدرجة:الماجستير

لجنة الحكم والمناقشة:

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أ.د / هيام كمال مصطفى نظيف	أستاذ طب الأطفال بقسم الدراسات الطبية للأطفال – بالمعهد.
أ.د / نيرة إسماعيل إسماعيل عطية	أستاذ طب الأطفال بقسم الدراسات الطبية للأطفال – بالمعهد.

تاريخ البحث : ٢٥/ ١ / ٢٠٠٨

الدراسات العليا

ختم الإجازة:

أجيزت الرسالة بتاريخ:

٢٠١٩ /٦/٢٩

موافقة مجلس المعهد

موافقة مجلس الجامعة

٢٠١٩ /٧/٢٧

/ / ٢٠٠٩

معهد الدراسات العليا للطفولة
قسم الدراسات الطبية للأطفال

دراسة التغيرات التي تحدث في نمو العظام في الأطفال المصابين بالشلل الدماغي

رســـــالـــة

توطئة للحصول على درجة الماجستير في دراسات الطفولة

قسم الدراسات الطبية – الأطفال ذوي الاحتياجات الخاصة

مقدمة من

الطالبة/ ايمان خليل ابراهيم عبد الله

دبلوم طب الأطفال- جامعة عين شمس

تحت إشراف

أ.د./ هيـــام كمال نظيـــف

أستاذ طب الأطفال

معهد الدراسات العليا للطفولة

جامعة عين شمس

د./ ريم بسيونى محمد

مدرس الأشعة

كلية الطب

جامعة عين شمس

٢٠٠٩