

Chapter 2

Epidemiology of cerebral palsy



ลิขสิทธิ์มหาวิทยาลัยเชียงใหม่
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“Cerebral” comes from the Latin word meaning brain. “Palsy” refers to weakness or paralysis or lack of muscle control. Cerebral palsy (CP) is sometimes called static encephalopathy. “Static” means the brain damage is permanent and does not worsen over time. Encephalopathy comes from the term “encephalo-” meaning brain and “-pathy” which is a problem or an abnormal condition. When the brain damage, the brain sends incorrect signals to the child's muscles, resulting in abnormal movement patterns.^{1,2} Therefore, CP is a disorder of muscle control as a result of damage to several parts of the brain. The term CP is used when a problem occurs in early life to the development of the brain.²

1. Classification of cerebral palsy

Classification means “the basic cognitive process of distributing children with CP into classes or categories of the same type”.³ Traditional classification schemes, with the same focus of the little club model in 1959 is based on the distributional pattern of affected limbs (e.g. hemiplegia, diplegia) with an added modifier describing the predominant type of tone or movement abnormality (e.g., spastic, dyskinetic). Although model of classification is quite similar but the most popular by Ingram (1955, 1964), Crothers and Paine (1959), Michaelis and Edebol-Tysk (1989), the Australian school (1989, 2000). And is widely known in Europe, is distinguished by the Swedish school (1975) and the classification by SCPE - Surveillance of Cerebral Palsy in Europe (2000).^{4,5} There is no classification system is the most obvious way, depending on the purpose that it may use certain characteristics or a combination. The purposes of classification include: (1) Description: provide the level of detail about individual with CP that will clearly identify the characteristics of the problem and the severity of it; (2) Prediction: providing information that can inform health care professionals in providing current and future needs of individuals with CP; (3) Comparison: to provide sufficient information to permit a proper comparison of a series of cases of CP assembled in different places; and (4) evaluation of change: providing information that will allow a comparison of the same individuals with CP at different points in time. However, it has become clear that additional characteristics will be carried for the report of the classification scheme to support the understanding and management of this disease. Data for classification of the features of CP are different in each age span and across geographic regions and settings. Factors other than age will affect the classification of information, including past history, especially pregnant. In year 2004, the International

Workshop by an Executive Committee and participated with the revised classification system for CP and reported by Rosenbaum and colleagues in 2007.⁴ They focus on quantitative assessment of the clinical imaging of the brain and nervous system disabilities. The classification of the CP for the four dimensions that are categorized as follows.

1.1 Motor abnormalities

1.1.1 Nature and topology of the motor disorder

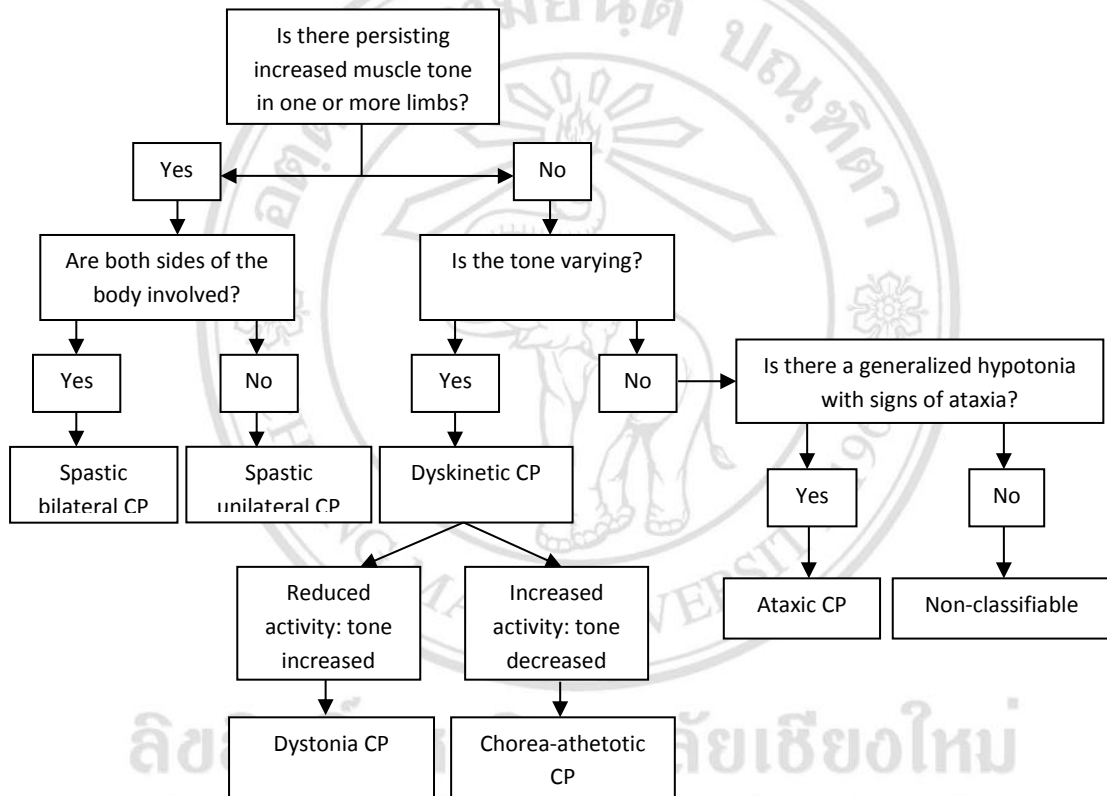


Figure 2.1 Hierarchical classification tree of cerebral palsy sub-types (Reproduced with permission from: SCPE working group. Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. Dev Med Child Neurol. 2000;42:816-24.)⁶

Types of abnormal muscle tone or involuntary movement disorder observed or displayed prominently, often associated with the underlying pathophysiology of the disorder. SCPE⁶ has developed a classification system of CP into three main groups, which are based on clear neurological symptoms indicative of pathology in the brain motor system, including spastic, dyskinetic and ataxic CP.^{3, 4} Resulting in neuronal loss can be: (1) cortical (pyramidal),

resulting in spasticity; (2) basal ganglial (extrapyramidal), resulting in dyskinesia; and (3) cerebellar, resulting in hypotonia and ataxia.^{7, 8} And to promote this standardized way of classifying CP types, SCPE developed a classification tree (Figure 2.1).⁶

(1) Spastic CP is the most common type generally correlates with a fixed lesion in the motor portion of the cerebral cortex.^{9, 10} Approximately 70% to 80% of children with CP are spastic.^{8, 11} Spasticity is a velocity-dependent increase in muscle tone and pathological reflexes, either increased reflexes, e.g. hyper-reflexia or pyramidal signs, such as Babinski response. Sanger et al.¹² have defined spasticity as hypertonia in which one or both of the following signs are present: (1) resistance to externally imposed movement increases with increasing speed of stretch and varies with the direction of joint movement; and/or (2) resistance to externally imposed movement rises rapidly above a threshold speed or joint angle. Spastic will be some time after the onset of the movement. Clonus is often associated with hyper-reflexia. Spasticity is different depending on a child's state of alertness, activity, or posture. Spasticity can be increased by anxiety, emotional state, pain, surface contact, or other nonnoxious sensory input. Spasticity may aggravate with movement of the involved muscles or maintenance of the limb against gravity, but it is not specific to particular attempted tasks. Be considered pathological when it happened a long time or does not stop spontaneously. Pathological posturing of lower limbs is characterized by: (1) internal rotation of the hip; (2) hip adduction; and (3) equinus foot, resulting in a "scissored" position.^{12, 17}

(2) Dyskinetic CP accounts for approximately 10% to 15% of all cases of CP. Athetosis, chorea, and dystonia are the main movement disorders seen in dyskinetic children.^{8, 11} Dyskinesia refers to disorder of movement, which athetosis is the most common dyskinetic syndrome. Children with dyskinesia lack the postural stability required to allow purposeful movements to be controlled for the completion of functional tasks. Muscle tone is often fluctuates from low to high to normal to high, such that children who have difficulty in maintaining a balance in all but the most firmly supported positions and exhibits slow, repetitive involuntary movements.¹⁰ Athetoid CP is characterized by the stability of the postural decline in both static and dynamic.¹² Chorea refers to rapid involuntary, jerky, often fragmented movements³. For some case, it may be difficult to delineate between athetosis and chorea of the existing features. It is defined as the dyskinetic CP is more appropriate. Dystonia is a disorder of involuntary movements sustained or periodic muscle contractions cause twisting and repetitive movements, abnormal postures, or both. In general, dystonia is activated or made

worse by attempted voluntary movement and may fluctuate in presence and severity over time. Severity of dystonic postures, depending on body position, specific tasks, emotional state, or level of consciousness.¹²

(3) Ataxic CP means a lack of coordination of muscle activity in the execution of voluntary movements, So that movements are carried out by unnatural force, rhythm, and accuracy. Clinically important discoveries include wide-based gait by a bug in the range and force of the limbs (dysmetria), error rates and the regulation of repetitive and switch movement (dysdiadochokinesia) and tremor that is usually most marked at the end of the movement. Anatomically associated with the most markedly pathology of the cerebellum and / or the afferent or efferent it. They are hypotonic in the first 2 years of life. Muscle tone becomes normal and ataxia appears at the age of 2 to 3 years.^{3, 8, 13}

However, there are arguments that many children have mixed presentations. And the status of each tone and or movements abnormalities may have clinical utility and cause even more, as recommended by the NINDS 2001 workshop on childhood hypertonia.¹² It is recommended that the case was still under classified by the dominant type of tone or movement abnormality.^{3, 12} Mixed term should not be used without elaboration of the component motor disorders.⁴ Children with mixed types of CP are generally mild spasticity dystonia and / or the athetosis. Ataxia may be a component of motor abnormalities in patients in this group. Ataxia and spasticity often occur together. Spastic ataxic diplegia is a type of compound, which is often associated with hydrocephalus.⁸

1.1.2 Functional motor abilities

The WHO's International Classification of Functioning, Disability and Health, known as ICF.¹⁴ The overall aim of the ICF classification is to provide a unified and standard language and framework for the description of health and health-related states. For classification, ICF domains for different groups of people in a given health condition (e.g. what the person has a disease or disorder does do or can do). Functioning as an umbrella term covering all body functions, activities and participation; similarly, disabilities to act as an umbrella term for impairments and activity limitations or participation restrictions. While the classification of the impact of the involvement of the upper and lower limbs should be separated

using the objective scales. Therefore, it is classified as follows and will be discussed again in Chapter 4.

(1) Ambulatory function: For the key function of ambulation, including the GMFCS¹⁵, which is widely used internationally for the group with CP were divided into five levels based functional mobility or activity limitation.

(2) Upper extremity function: The classification in parallel, the Bimanual Fine Motor Function Scale (BFMF)¹⁶ has been developed for the evaluation of the upper limb in CP. It has not been extensively studied as the GMFCS.

(3) Hand and arm function: New instrument for assessing hand and arm function - the Manual Ability Classification System (MACS)¹⁷ - has been developed in recent years, in 2006, how to identify children with CP use their hands when handling objects in daily activities.

1.2 Accompanying impairments

While the motor deficit in CP is predominant, a number of associated conditions are frequently present. Different impairments meddle with the capacity to work in everyday life, and may on occasion create much more prominent movement restriction than the engine hindrances that are the sign of CP. Therefore, must be considered in the overall developmental needs of the affected child. These impairments may have arisen from the same or comparative pathophysiologic forms that prompted to the engine issue, yet they all things considered require isolate list.^{4, 9} In addition, a finding by Samson-Fang and Stevenson¹⁸ suggests the presence of unique growth patterns, with evidence of poor linear growth in this population that requires further investigation. In every sense, therefore, the term “cerebral palsy” conveys the concept of a broadly based, multiply handicapping condition. Therefore, the parents should ensure the presence of this issues involved and get referrals for appropriate treatment. These problems as much as possible and as early as possible to prevent the development of disability. These impairments are as follows.^{4, 7, 8, 10, 19-21}

(1) Seizure disorders: Area of cerebral harm in CP may turn into a concentration of anomalous electrical movement, which can bring about seizures. Central nervous system infections, central nervous system malformations, or gray matter damage will probably show seizures in children with CP than white matter damage.²² Approximately 30 to 50% of children

with CP experience seizures. They are most regular in quadriplegic and hemiplegic CP, in patients with intellectual disability and in postnatally gained CP.²³ This thesis classifies seizures as experience seizures or absent seizures.

(2) Visual impairments: The prevalence of visual impairments ranged from 39 to 100%, which common in children with CP.²⁴ In the event that there is harm to the visual cortex, the tyke will be practically visually impaired in light of the fact that he will be not able translate driving forces from the retinas. In serious cases, the optic nerves may likewise be harmed. Loss of coordination of the muscles controlling eye developments is extremely normal. The children can't settle his look on a matter. About half of cases, binocular vision does not create and nearsightedness is a companion issue. Vision is critical for the advancement of adjust amid the initial 3 years of life, therefore children with visual impairments may have more trouble in creating head and trunk control and in investigating their prompt environment. This thesis identifies visual impairment as impaired or unimpaired.

(3) Hearing impairments: Hearing impairment was less common in children with CP. This group found sensorineural hearing misfortune about 10%, which high risk in children born prematurely. It is difficult to diagnose early hearing because of other disabilities. This thesis identifies hearing impairment as impaired or unimpaired.

(4) Communication problems and dysarthria: Dysarthria, referring to a speech problem, which the child has difficulty in producing sound and clear words. Dysarthria happens in 40% of children with CP. Some speech problems can be optional to poor engine control of oral muscles or respiratory disability. Spasticity or athetosis of the muscles of the tongue, mouth and larynx cause dysarthria. It is important that all children are provided with a choice of communication as soon as possible to avoid further paralysis. This thesis identifies communication problems as being able to speak single words or sentences and cannot communicate speech.

(5) Oromotor dysfunction: Oromotor impairments included sucking, swallowing, and chewing. Drooling, dysarthria, and inability to eat, result in children with retarded growth and nutrition in poor oral hygiene. Abnormal oromotor development is present by persistence of infantile oral reflexes, for example, rooting or suck-swallow or exaggerations of normally occurring reflexes, for example, a tonic bite or tongue thrust. Oromotor impairments are related with dental malocclusion and trouble with oral cleanliness, prompting to an expanded danger of periodontal illness. This thesis considers oromotor dysfunction from its ability to eat independently and eat by helping.

(6) Gastrointestinal problems and nutrition: In common, children with CP often have problems with growth and development, especially in children with dyskinesia and spastic quadriplegia as failure to thrive. This is related to dietary deficiency with recurrent vomiting as well. In addition, children also have difficulties in swallowing (dysphagia), hyperactive gag reflex, spasticity or loss of fine motor control impair feeding. Pneumonia can be caused by gastroesophageal reflux and swallowing impairments in this children. Many children with CP have a high metabolic rate, coupled with difficulty in feeding, thus causing malnutrition. It was determined by the body mass index.

(7) Respiratory problems: Children with CP will be pneumonia from aspiration because of difficulty swallowing. Premature babies with pulmonary dysplasia, most of which can lead to infection of the upper respiratory tract. In addition, symptoms of respiratory muscle spasms cause pulmonary problems.

(8) Cognitive and attention deficit: Cognition alludes to specific features of higher cortical function, including namely, attention, memory, problem solving, and language. Intellectual disability and learning disability caused by a deficiency of cognition. Although there is no direct relationship between the intensity of the level of involvement of motor and mental disabilities, the percentage of children with CP who have intellectual disability has been reported to be in the range of 38 to 92% depending on the category. This most often occurs in spastic quadriplegia. This thesis classifies this problem as having intellectual disability and absent intellectual disability with its level.

(9) Emotional and behavioral problems: Children with CP have prevalence of emotional and behavioral differences in the range of 30 to 80%.²⁵ Attention deficit disorder, passivity, immaturity, anger, sadness, impulsivity, emotional lability, low self-esteem, and anxiety are a variety of behavior and emotional disorders. A population-based study in Europe found a similar prevalence of significant emotional and behavioral symptoms in 26% of children with CP.²⁶ The most well-known issues recognized were in peer relationships (32%), hyperactivity (31%), and emotion (29%). It is seen that children with CP who are at high risk for psychological disorders, they should be introduced to a mental health professional for evaluation assessment and treatment.

(10) Sensory Impairments: Children with hemiplegic are the most sensory deficits.²⁷⁻²⁹ A study of children with spastic hemiplegia showed that the spastic limbs had a stereognosis deficit (97%), a two-point discrimination deficit (90%), and a proprioception deficit (46%), and

these sensory deficits were more commonly present in limbs with a greater size discrepancy.²⁹ Sensory deficits are important to recognize because they may affect the functioning of the extremity.

(11) Later-developing musculoskeletal problems: In this age, children with CP have a bone growth. The spastic CP children have tightened muscles are shortened, resulting in an imbalance between bone and muscle. So often have deformities of the hip and foot. In addition, this children may be complications such as scoliosis and other bone fractures due to osteoporosis or osteomalacia.

(12) Genitourinary Disorders: The development of urinary incontinence is usually delayed in children with CP. More than one-third of children with CP are experienced to present with dysfunctional voiding symptoms.³⁰ The most vital elements related with urinary incontinence were quadriplegia (tetraplegia) and hindered cognizance. Incontinence was the most well-known grumbling, however recurrence, criticalness, aversion, and urinary maintenance may likewise be available. Recurrence and criticalness are regularly connected with spasticity of the detrusor muscle, creating little, and visit voids.³¹

1.3 Anatomical and neuro-imaging findings

1.3.1 Anatomical distribution^{4, 5, 7, 11, 32, 33}

Really, the most well-known foundation to group CP has dependably been founded on the topographic (geographic) circulation of the motor impairment: monoplegia, hemiplegia, diplegia, triplegia, and quadriplegia. The expression word "plegia" is utilized alongside a prefix to assign whether four appendages, two appendages, one appendage, or a large portion of the body is influenced by loss of motion or shortcoming.¹⁰ Monoplegia and triplegia rare and is associated with the body parts that overlap (Figure 2.2).

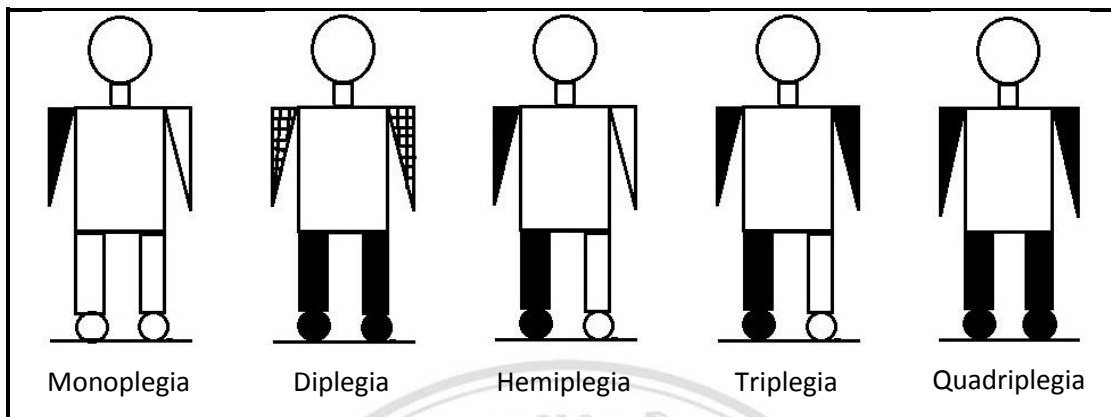


Figure 2.2 Classification of cerebral palsy

(1) Monoplegia: Monoplegia refers to single extremity involvement. For all intents and purposes, this circumstance is likely the aftereffect of a hemiplegia in which the disability of the leg is indistinct.

(2) Diplegia: Diplegia is available when the lower furthest points are more seriously influenced than the furthest points, despite the fact that the furthest points are not totally saved. Spastic diplegia is the most widely recognized kind of spastic CP around half and is related with prematurity and low birth weight.^{7, 32} In general, these children no problems about mental function, communication, oromotor function, and gastrointestinal function. In any case, they are regularly seen shortfall and strabismus. In addition, they also tend to fall backwards because the reaction equilibrium development. However, the main problem in spastic diplegia is walking difficulties, due to disturbances, muscle weakness, spasticity and balance disorders resulting in an abnormal gait pattern. In general, diplegic children walking are increasing the use of energy, causing fatigue. Most diplegic children start crawling at the age of two years and started walking at the age of four. Being able to walk the kids up until the age of seven years. Children who cannot walk, even with appropriate treatment will be necessary to use a walking aids.

(3) Hemiplegia: Anomalies that occurred in this group, as seen in adults after a stroke is an unusual one side of the body affected by the upper extremity rather than the lower. One side brain damage that causes of hemiplegic children, which are most common in term infants (more than 38%).³⁴ There are problems associated with these children very little. However, they may have seizures, learning, and behavior problems. These children are good prognosis compared with other types because one side of the body is normal. The triple flexion

posture is normal pattern of this group with related to arm flexion and adduction of the wrist, elbow, and shoulder, however hemiplegic children learned to walk at the age of three years. They become able to support themselves in the activities of daily living independently.

(4) Triplegia: Triplegia is uncommon and ordinarily comes about because of milder and extremely lopsided double hemiplegia (saving one leg) or milder uneven diplegia (saving one arm).

(5) Quadriplegia (tetraplegia): Quadriplegia is the contribution of neck, trunk and every one of the four furthest points. By and large, the legs are more required than the arms. Children with quadriplegic CP much of the time give truncal hypotonia and limit hypertonicity. The prevalence rate of quadriplegic children range from 10 to 15% of children with CP, these children have severe motor impairment and other signs and symptoms of central nervous system (CNS) disorders such as cognitive impairments, seizures, speech, and swallowing difficulties. Primitive reflexes persist lifelong, and extrapyramidal signs are common. It also found an intellectual disability, seizures, visual deficits, strabismus, bulbar disorders manifested by drooling, difficulty swallowing, dysarthria, and medical complications were frequent. The severity of disease is the key factor for the child not being able to sit or no control head and trunk, including the ability to walk independently. Only about 15% of these children have the potential to walks and the rest have to use wheelchair. Most of these children have to be cared for very much.³⁵

Children with CP are different individual person. These classifications only help organize the treatment approach for a child with CP. Each child's strengths and challenges are unique and individual. In spite of the fact that these assignments appear to concentrate on the quantity of furthest points or the side of the body included, the limits are associated with the storage compartment. The storage compartment is constantly influenced to some degree when a child has CP. The storage compartment is essentially influenced by strange tone in hemiplegia and quadriplegia, or it is optionally influenced, as in diplegia, when the storage compartment adjusts for absence of controlled development in the included lower furthest points.¹⁰

1.3.2 Neuro-imaging findings

Today, the relationship between neuro-imaging disclosures and clinical offer in CP were mild. In spite of advances both in imaging innovation and in the quantitative assessment of the engine change this photo. American Academy of Neurology³⁶ indicates that neuro-imaging results in children with CP may help determine the cause. However, the current evidence is insufficient to demonstrate the need for an investigation. Electroencephalography (EEG) is not recommended in children with symptoms of seizure, or epilepsy. Because children with CP may have deficits related intellectual disabilities, visual and hearing impairment, speech and language disorders, and disorders in the oral motor screening, therefore these conditions should be a part of the initial evaluation.

1.4 Causation and timing

Obviously CP may come about because of the cooperation of various hazard components and much of the time, no cause can be distinguished. Hence, the grouping of the cause is unmistakably not practical right now. For the present, timing of affront ought to be watched when an adequately solid confirmation to demonstrate that the causative operator. While recording unfriendly occasions in the prenatal, perinatal, and postnatal existence of a child with CP is important doctors ought to abstain from making the presumption that the nearness of such an occasion is adequate to permit etiologic characterization. So this will be discussed again in the etiology or risk factors.

In conclusion, the assortment of CP according to motor abnormalities, impairments, and anatomical distribution is commonly used. But it is not yet clear classification using neuro-imaging findings and causation. Bax and colleagues³⁷ contend that this order plans will encourage better comprehension of CP, and subsequently an enhanced administration of the condition. A few measurements, for example, radiological discoveries, are as yet not well catered for and further work is required to develop appropriate classification schemes for each dimension. Similarly as with any arrangement framework, the present terminology does not totally portray what is found practically speaking. Numerous people with CP have a mix of the already depicted development variations from the norm. This is alluded to as blended example CP and is available in numerous people with CP. Since epidemiologic reviews regularly utilize an

essential analysis for grouping, blended example CP is probably going to be underreported. Furthermore, as a child grow up, the pattern of CP and particular finding may change.

2. Prevalence and Trend over time

The role of perinatal factors that might be the cause of CP, so the frequency of CP was measured as a rate per thousand live births and not per thousand population alive at the time of diagnosis. This rate is called the prevalence, not incidence, because it is difficult to calculate the incidence of CP because of the huge gap of time between the onset of CP and their diagnosis and many of the dead or lost to follow-up prior to diagnosis were not counted.⁹ Most CP prevalence studies using population-based study, especially in developed countries. However, there are some countries that do not register, but it can also be used to survey study, especially in developing or undeveloped countries. The making of registers for screen CP rate is vital for example keeping in mind the end goal to guarantee that the expanding survival rate in exceptionally untimely newborn child is not at cost of an expanding "morbidity" rate.³⁸ In addition, trends in CP is a major source of etiological hypotheses for congenital CP, give supportive confirmation to existing speculations and may guide methodologies to anticipate post neonatally obtained CP. In developed countries in the course of the most recent three decades prior, the likelihood of survival has expanded even in children with severe disabilities. Due to the generally utilization of electronic fetal heart rate checking there are 5-crease increment in the cesarean conveyance rate over a similar timeframe. Conversely, the commonness of CP has not diminished but rather stayed consistent for around 2 to 3 for each 1,000 live births for a very long while (Table 2.1).^{19, 39, 40} However, recent data shows that the overall prevalence of CP declined both in the European⁴¹ and Australian⁴² Registers. In addition, the survival rate of at-risk preterm infants has expanded. CP prevalence rate was about 2.0 per 1,000 live births, as obtained from two systematic reviews.^{43, 44}

Table 2.1 Data for overall cerebral palsy prevalence from worldwide studies

Country	Design	Time period	Prevalence (N per 1,000)	Trend
Europe				
Eastern Denmark ⁴⁵	population-based	1983 – 1998	3.00 to 2.10	decrease
United Kingdom ⁴⁶	population-based	1984 – 1995	2.50 to 1.70	decrease
Southern Sweden ⁴⁷	census	1990 – 1993	2.40	NA
Iceland ⁴⁸	population-based	1990 – 1996	2.20	NA
		1997 – 2003	2.30	
Northern Ireland ⁴⁹	population-based	1981 – 1997	2.30 to 2.10	not change
Sweden	population-based ⁵⁰	1991 – 1994	2.12	NA
	population-based ³⁴	1995 – 1998	1.92	NA
	population-based ⁵¹	1999 – 2002	2.18	NA
Norway ⁵²	population-based	1996 – 1998	2.10	NA
SCPE	population-based ⁶	1980 – 1990	2.08	increase
	population-based ⁵³	1980 – 1998	1.16 to 0.99	not change
Europe ⁴¹	population-based	1980 – 2003	1.90 to 1.77	decrease
Western Sweden ³⁴	population-based	1980 – 1998	1.92	decrease
West of Ireland ⁵⁴	population-based	1990 – 1999	1.88	NA
North-east England ⁵⁵	survey	1964 – 1993	1.68 to 2.45	increase
Netherlands ⁵⁶	cross-sectional population- based	1977 – 1988	1.51	increase
Asia				
Turkey ⁵⁷	survey	1996	4.40	NA
South Korea ⁵⁸	survey	2004 – 2008	2.60	NA
Japan ⁵⁹	population-based	1995 – 2001	2.30	NA
Hong Kong ⁶⁰	survey	2003 – 2004	1.30	NA
China ⁶¹	survey	1998	1.28	NA
North America				
British Columbia ⁶²	population-based	1991 – 1995	2.68	NA
United States ⁶³	population-based	1975 – 1991	1.7 to 2.0	increase
Victorian Cerebral Palsy Register ⁴²	population-based	1983 – 2009	1.75 to 1.62	decrease
Africa				
Egypt ⁶⁴	survey	2010	2.04	NA

A study on the prevalence of children with CP in the past, there have been controversy about the relationship between the rate of decline in neonatal mortality and the incidence of CP. Therefore, it is unclear whether the reduction in neonatal and infant mortality has led to changes in the occurrence of CP. Reporting prevalence or incidence of CP is dependent on a variety of factors, including region, population, age, severity, time period, migration, stillbirths and neonatal mortality rates, which may restrict the generalizability of population-based outcomes.^{19, 65} Therefore, the reported prevalence rates of CP are different depending on these factors. Prevalence of children with CP overviews might be underreported in light of the fact that seriously weakened newborn children may pass on before creating discoveries that are analytic of CP or before their irregularities meet criteria for a determination of CP. Likewise, if children who have mild CP are not gotten by their folks for assessment, might be lower than the real predominance.^{60, 65} For example, a cross-section survey study of Hong Kong in the local prevalence of 1.3 per 1,000 children with CP, which is lower than other countries, possibly because of differences in study design or a low true prevalence. Setting up a CP registry to help determine the prevalence of disability in childhood is more accurate.⁶⁰ As with China, the prevalence is lower than other countries is 1.28 per 1,000, which may be related to the lower neonatal survival and higher infant mortality.⁶¹ Prematurity and low birth weight is an important risk factor for CP, so much so that the epidemiology of CP differs greatly among children with normal birth weight and low birth weight.

2.1 Gestational age – specific prevalence and trends over time

Approximately half (55%) the children with CP are born mature, 20% have a moderately preterm and 25% are very preterm. These results came from the number of 9,701 CP with SCPE comprehensive database of children born in 1977 to 1996.¹⁹ The prevalence of CP for the geographical differences, but the overall population report showed a fairly stable rate among children born at term, 1 to 1.5 per 1,000 live births.^{48, 49, 66-68} Considering the trend of CP in children born preterm has reported inconsistent results. The report both concluded that the prevalence of CP is increased^{48, 69, 70} and decreased^{42, 66, 69, 71} in infants born prematurely (Table 2). These fluctuations are likely to comply with the advances in obstetric and neonatal care improved survival of very premature infants.

2.2 Birth weight – specific prevalence and trends over time

Table 2.2 Data for specific cerebral palsy prevalence from worldwide studies

Country	Design	Time period	Prevalence (N per 1,000)	Trend
Europe				
United Kingdom ⁴⁶	population-based	1984 – 1995	90.0 to 57.0	decrease (ELBW)
			77.0 to 40.0	decrease (VLBW)
			24.6 to 12.5	decrease (MLBW)
Iceland ⁴⁸	population-based	1990 – 2003	33.7 to 114.6	increase (VPT)
			north-east	survey
England ⁵⁵	survey	1964 – 1993	3.9 to 11.5	increase (MLBW)
			Eastern Denmark ⁶⁶	population-based
SCPE	population-based ⁷¹	1980 – 1998	12.2 to 4.5	decrease (MPT)
			population-based ⁷²	1980 – 1996
North America				
Northern Alberta ⁶⁹	population-based	1974 – 1994	0 to 110.0	increase
		1994 – 2003	110.0 to 22.0	decrease (VPT)
Nova Scotia ⁷⁰	population-based	1993 – 2002	44.0 to 100.0	increase (VPT)
Victorian Cerebral Palsy Register ⁴²	population-based	1983 – 2009	41.5 to 32.4	decrease (MPT)
			92.1 to 70.6	decrease (VPT)

* ELBW = Extremely Low Birth Weight, VLBW = Very Low Birth Weight, MLBW = Moderately Low Birth Weight, VPT = Very Preterm, MPT = Moderately Preterm

Gestational age and birth weight is associated with two factors affect the risk of CP.⁴⁸ CP prevalence rates range from 3.9 to 90.0 per 1,000 live births for LBW infants, the prevalence of which is inversely related to birth weight.^{46, 55, 72} For example, the study of infants born in the Metropolitan Atlanta area year 1986 to 1991, the prevalence rate of normal infants birth weight ($\geq 2,500$ gram) was 1.1 per 1,000 live births. In infants who low birth weight (1,500 to 2,499 gram), the prevalence rate was 6.1 per 1,000 live births. For infants who very low birth weight ($< 1,500$ gram), the prevalence rate was 39.7 per 1,000 live births.⁶³ Trends in the prevalence of CP in low birth weight infants have been reported to vary as well as the prevalence of CP in preterm infants (Table 2.2). However, the format was changed to CP diplegia and spastic quadriplegia more and more away from hemiplegia and athetosis.⁵⁵ This change may reflect expanded medicinal care with better care, labor, and the expanded occurrence of survivors of

neonatal emergency unit mind unit. Additionally, various births have expanded with expanding maternal age, and these numerous births have a generously higher danger of developing CP.⁷³

3. Impact

3.1 Children impacts

Children who have been diagnosed with CP, is one that has been most affected. They will have to work hard everything it for the rest of their lives. These children will be treated several times a week and will most likely need help when it comes time to attend school. Most kids with CP will probably need to manage with the teasing from classmates and even adults look foolish. They will need a lot of medical appointments and must have assistance with daily life when they are of legal age.⁷⁴

3.2 Family impacts

CP is a condition that changes enormously from extremely gentle engine impacts to exceptionally serious engine inabilities with comorbidities.⁷⁵ Families of children who have been diagnosed with CP will be affected directly.⁷⁴ Because they will have to provide more care for children with CP than normal children need. Although the parents of the children must have a role in children care. But this role in children with CP will take a completely different with normal children. One of the main challenges for parents of children with CP is to effectively manage and help them with daily living. Thus, the task of caring for a child with CP at home is hard, especially for the children with CP are severely impaired.⁷⁶ Such care may demonstrate impeding to both physical wellbeing and mental prosperity of guardians of children with incapacities, unending consideration, some of which may be as good as the others cannot. Different reactions of parents may cause stress that leads to high levels of divorce, which is found most often in children aged 1 to 4 years. But no concrete evidence is clear that the divorce rate for these families is higher than in the normal population. In order to take proper care for children with CP, the doctor should have an understanding of the structure of family, because most children with CP develop problems in infancy and childhood, so the family is important.⁷⁵ The family is a key element in the early management of the infant with CP. There are many factors, the significance of family contribution in the early treatment. These include the following.¹⁰

- (1) Parents are the most critical individuals for the child's life.
- (2) Parents have the privilege to settle on choices about their child.
- (3) Parents have a legitimate appropriate to the data and basic leadership in the instructive procedure.
- (4) Parental contribution is fundamental for improving early intervention.
- (5) Positive parenting experience to support their children at home.
- (6) Parents can be enabled to utilize group assets successfully.
- (7) Participation of parents to ensure coordination of services more fully.
- (8) Participation of parents will save a lot of resources.

One study⁷⁷ concluded that African caregivers who care for children with CP has been a negative impact on health than those who did not care for children with CP. The caregivers of this study were mothers (93.62%) fathers (4.95%) and grandmothers (1.42%). The proportion of caregivers shows that it is the duty of these children to become the administrator by default. As well as education in private orthopedic clinic in the Piedmont region of North Carolina⁷⁸ found that mothers of children with CP have a higher risk of depression. The recommendation for treatment should take into account the impact of social support may increase an adaptation of the mother. In addition, a study has indicated that the most important predictors for the well-being of patients, including children's behavior caregiving demands, and family function.⁷⁶ In this way, medicinal care suppliers ought to keep on being delicate to their anxiety administration. This anxiety can bring about sickness of other relatives, money related weights, work changes, conjugal anxiety, and, most ordinarily, the impacts of maturing on the guardians, kin, and people with CP.

4. Etiology or risk factors

The goal of epidemiological studies is to determine the cause of the disease with a view to preventing successful on the basis of population, while the usual clinical goals are predicted treatment and/or rehabilitation of the individual.⁷⁹ It is well known that there are many causes of CP, and know for sure. The cause is not very important for physicians to manage motor impairments. But the cause is important in determining the prognosis of the child. Therefore, parents need to know why it is important because it comes as part of a deal with the question of why the CP is going on with their child.^{75, 80} Despite the fact that CP is not an ailment with a

solitary cause, similar to chicken pox or measles. But it is a neurological condition caused by a brain injury that occurs before the brain development is complete caused similar problems in control of movement. Therefore, CP can be caused by many factors and complex because of the development of the brain during the first two years of life, CP is a result of brain injury that occurs during the prenatal, perinatal or postnatal time period.^{10, 21, 32, 75, 79-84} Prior to 1980, the medical information that is the most common cause of CP is hypoxic-ischemic encephalopathy at birth⁸⁵, but at present, the most common cause of CP is premature labor and problems in the prenatal period, such as maternal illness, or late risk factors, such as chronic lung disease.^{86, 87} Current information affirmed that as much as 75 to 80% of the cases are because of pre-birth damage, however has under 10% being because of birth injury or asphyxia.^{81, 88} Although the causes of CP cannot know about 30 to 40% of the all CP, but a physician can diagnose CP by modern imaging techniques (computerized tomography (CT) and magnetic resonance imaging (MRI)) that some cases of CP are caused by stroke or brain hemorrhage in late stages of development of the fetus.⁸⁹ Risk factors different from causes, while the risk is to increase the probability of a child born with CP, the cause is the act that resulted in development of CP. Along these lines, it is critical to recognize risk factors and causes.⁹⁰

4.1 Prenatal period

The important known causes of CP in prenatal period are maldevelopment of the brain. Others known causes are vascular insults and maternal infections such as rubella, herpes simplex, and cytomegalovirus. In addition, less common causes of CP include maternal problems (e.g. diabetes, metabolic disorders, toxemia, and hyperthyroidism) and a rare genetic disorder. Finally, CP created by Rh contradiction happens when an Rh negative mother conveys a child who is Rh positive.^{10, 90}

The risk factors for the occurrence of CP occur before pregnancy is associated with maternal factors, for example, deferred onset of monthly cycle, unpredictable feminine cycle or long intermenstrual interims,⁹⁰ and maternal age older than 40 years.⁹¹ One study found that low social class is associated with the occurrence of CP in children born at term.⁹² In addition to factors related to the mother, there are other risk factors for CP include multiple pregnancies,^{93, 94} twin pregnancies,^{91, 95} antenatal infections,⁹¹ abruptio placentae,^{91, 96} chorioamnionitis,⁹⁷ primiparity,⁹¹ and maternal risk behaviors such as smoking.⁹¹

4.2 Perinatal period

An infant may experience asphyxia resulting from hypoxia during labor and delivery. Although there is evidence of a modest association between perinatal asphyxia and CP, but it is difficult to demonstrate a causal relationship in special child. Because most cases of CP occur in infants without a history of adverse perinatal events, and most babies experience birth asphyxia recover without CP.^{9, 10, 90} It is well known that asphyxia is a major cause of CP. In any case, there are just a little rate of instances of CP are because of asphyxia around the season of birth.⁹⁸

There are several risk factors for CP during labour which these occasions are probably going to bring about perinatal asphyxia, including obstructed labour, antepartum hemorrhage, preeclampsia, cord prolapse, premature rupture of membranes, prolonged labor, preeclampsia, instrumental delivery, emergency cesarean delivery, and breech delivery.^{91, 95, 99, 100} It also has two main risk factors for CP is premature and low birth weight.¹⁰ In addition, studies have found that low Apgar scores are strongly associated with CP.^{85, 91, 96}

4.3 Postnatal period

Sometimes the postnatal causes of CP could not be separated as they may overlap somewhat with the prenatal and perinatal, but asphyxia, infections, cerebral hemorrhage, and trauma are considered as etiologies in this group. The main causes are found in developed countries tend to be involved in car accidents, acts of violence against children in the form of shaking the baby, near drowning, or exposure to lead.^{9, 10, 75, 101, 102} For developing countries, meningitis, septicemia, and malaria remains a major causes of CP.⁹⁰ A large study in Europe found that the cause of CP is the most common postnatal infection accounted for 50%, followed by vascular episodes for 20% and head injury for 18%, respectively.¹⁰³

There is also a risk factor, including seizures,¹⁰² sepsis, respiratory distress, patent ductus arteriosus, blood transfusion, hypotension, prolonged ventilation, hyponatremia, pneumothorax, total parenteral nutrition, and parenchymal damage with appreciable ventricular dilatation were detected by cerebral ultrasound are associated with CP.¹⁰⁴

Table 2.3 Causes or risk factors associated with cerebral palsy

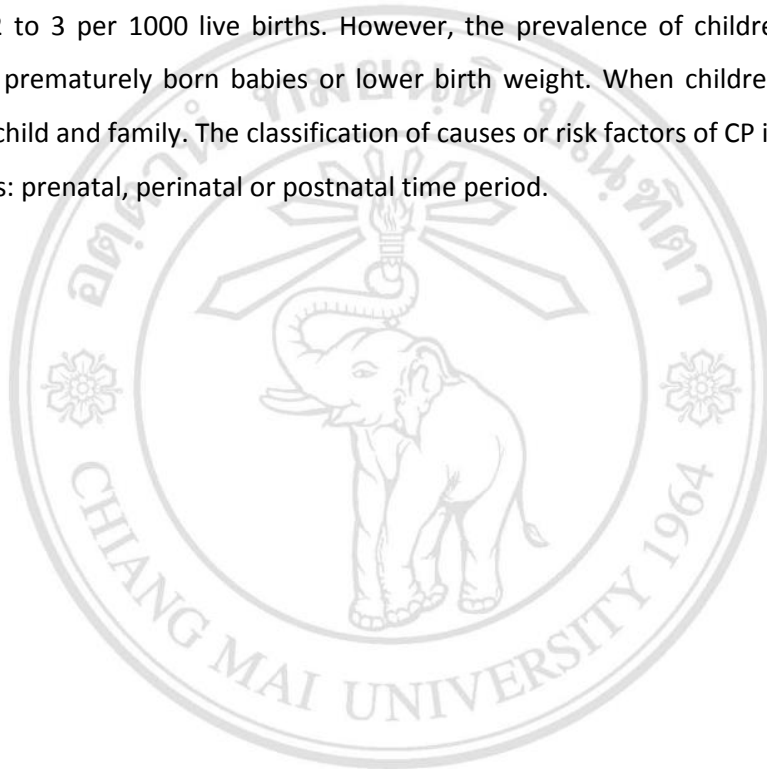
Prenatal	Perinatal	Postnatal
Congenital brain Malformations*	Birth asphyxia*	Asphyxia* (near drowning)
Vascular insults*	Prematurity	cerebral hemorrhage*
Maternal infections* (rubella, herpes simplex, toxoplasmosis, cytomegalovirus)	Low birth weight	Infections* (meningitis, Encephalitis, septicemia, sepsis)
Maternal problems* (diabetes, toxemia, metabolic disorders, hyperthyroidism, mental retardation, seizures)	Low Apgar score	Head injury* (motor vehicle accidents, Shaken baby syndrome, non-accidental injury)
Genetic disorders* Rh incompatibility*	Obstetric emergencies - obstructed labour	Malaria* Respiratory disease
Multiple / twin pregnancy/ primiparity	- antepartum hemorrhage	Seizures within 48 hours of birth
Abruptio placentae	- cord prolapse	Hypotension
Thrombophilic disorders chorioamnionitis	- prolonged labor	Blood transfusion
Intrauterine infections	- home birth	Hyperbilirubinemia/ kernicterus
Intrauterine growth restriction	- premature rupture of membranes	Methylmercury / lead exposure / postnatal steroids
Maternal fever	- preeclampsia	Patent ductus arteriosus
Abdominal trauma	- instrumental delivery	Parenchymal damage with appreciable ventricular dilatation
Maternal risk behaviors (smoking, alcohol, radiation exposure, use of recreational drugs)	- breech delivery	
Delayed onset of menstruation / irregular menstruation / long intermenstrual intervals	- emergency cesarean delivery	Hyponatremia Total parenteral nutrition Prolonged ventilation

* Cause, data from: Mary M et al. (2009)²⁰; Nancy ND (2008)²¹; Nelson KB (2008)¹⁰⁵; Jones MW et al. (2007)⁸²; Miller F (2007)⁸⁰; Martin SC and Kessler M (2006)¹⁰; Parker J & Parker P (2002)⁸⁴; Scherzer AL (2001)⁹

In conclusion, factors causing CP vary in degree according to gestational age group and the CP subtype.¹⁰⁵ This review of the causes or risk factors of the CP was not finalized by such groups, but concluded by the time period (Table 2.3). However, knowing these warning signs will help physicians monitor the children face a higher risk. Parents should not be too alarmed if their child has a problem with one or more of these factors. Most children who have these risk factors do not develop CP, only to become a child with CP.⁸⁴ Now it is generally accepted that CP is usually caused by a series of causal pathways is a combination of events that led to the

injury in the brain development of infants. However, no causal pathway of CP is the best. Although the causal pathway to CP to help prevent and help treat effectively.^{79, 106}

In summary, the content of this chapter mentions definition of CP refers to the term for a range of non-progressive syndromes of brain damage. The classification of cerebral palsy by motor abnormalities, accompanying impairments, anatomical and neuro-imaging findings, and causation and timing. The frequency of CP was measured as a rate per thousand live births. The prevalence is 2 to 3 per 1000 live births. However, the prevalence of children with CP will increase when prematurely born babies or lower birth weight. When children have CP, the impact is both child and family. The classification of causes or risk factors of CP is divided into 3 main categories: prenatal, perinatal or postnatal time period.



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REFERENCES

1. Leonard JF, Cadenhead SL, Myers ME. Keys to parenting a child with cerebral palsy: Barron's Educational Series; 1997.
2. Bagnara C, Bajraszewski E, Carne R, Fosang A, Kennedy R, Ong K, Randall M, Reddihough D, Touzel B. Cerebral palsy an information guide for parents. second ed. Melbourne: Royal Children's Hospital; 2000.
3. Cans C, Dolk H, Platt MJ, Colver A, Prasausk1Ene A, RÄGeloh-Mann IK. Recommendations from the SCPE collaborative group for defining and classifying cerebral palsy. *Dev Med Child Neurol*. 2007;49:35-8.
4. Rosenbaum P, Paneth N, Leviton A, Goldstein M, Bax M. A report: the definition and classification of cerebral palsy April 2006. *Dev Med Child Neurol*. 2007;49:8-14.
5. Ferrari A, Cioni G. The spastic forms of cerebral palsy: a guide to the assessment of adaptive functions: Springer; 2009.
6. SCPE working group. Surveillance of cerebral palsy in Europe: a collaboration of cerebral palsy surveys and registers. *Dev Med Child Neurol*. 2000;42:816-24.
7. Jan MM. Cerebral palsy: comprehensive review and update. *Ann Saudi Med*. 2006;26:123-32.
8. Nadire B, Selim Y. The HELP guide to cerebral palsy: Avrupa Medical Bookshop Co. Ltd. & Global-HELP Organization; 2005.
9. Scherzer AL. Early diagnosis and interventional therapy in cerebral palsy: an interdisciplinary age-focused approach: M. Dekker; 2001.
10. Martin SC, Kessler M. Cerebral palsy. *Neurologic interventions for physical therapy*. St. Louis: Elsevier Saunders; 2006. p. 124-52.
11. Nadire B, Selim Y. Cerebral palsy: orthopedic aspects and rehabilitation. *Pediatric clinics of North America*. 55: Elsevier; 2008. p. 1208-25.
12. Sanger TD, Delgado MR, Gaebler-Spira D, Hallett M, Mink JW. Classification and definition of disorders causing hypertonia in childhood. *Pediatrics*. 2003;111:e89-e97.
13. Donald LG. Ataxia. *Treatment of pediatric neurologic disorders*: Taylor & Francis; 2005. p. 415-22.
14. World Health Organization. International classification of functioning, disability, and health. Geneva: World Health Organization; 2001 [cited 2011 6 Febuary]. Available from: <http://www.who.int/classifications/icf/en/>.

15. Palisano R, Rosenbaum P, Walter S, Russell D, Wood E, Galuppi B. Development and reliability of a system to classify gross motor function in children with cerebral palsy. *Dev Med Child Neurol.* 1997;39:214-23.
16. Beckung E, Hagberg G. Neuroimpairments, activity limitations, and participation restrictions in children with cerebral palsy. *Dev Med Child Neurol.* 2002;44:309-16.
17. Ann-Christin Eliasson, Lena Krumlinde-Sundholm, Birgit Rösblad, Eva Beckung, Marianne Arner, Ann-Marie Öhrvall, Rosenbaum P. The Manual Ability Classification System (MACS) for children with cerebral palsy: scale development and evidence of validity and reliability. *Dev Med Child Neurol.* 2006;48:549-54
18. Samson-Fang L, Stevenson RD. Linear growth velocity in children with cerebral palsy. *Dev Med Child Neurol.* 1998;40:689-92.
19. Cans C, De-la-Cruz J, Mermet M-A. Epidemiology of cerebral palsy. *Paediatr Child Health.* 2008;18:393-8.
20. Mary M, David P, Jilda V-A. Cerebral palsy. *Pediatric rehabilitation: principles and practice: Demos Medical Publishing, LLC; 2009.* p. 165-97.
21. Nancy ND. Cerebral palsy: medical aspects. *Pediatric clinics of North America.* 55: Elsevier 2008. p. 1189-207.
22. Carlsson M, Hagberg G, Olsson I. Clinical and aetiological aspects of epilepsy in children with cerebral palsy. *Dev Med Child Neurol.* 2003;45:371-6.
23. Humphreys P, Deonandan R, Whiting S, Barrowman N, Matzinger MA, Briggs V, Hurteau J, Wallace E. Factors associated with epilepsy in children with periventricular leukomalacia. *J Child Neurol.* 2007;22:598-605.
24. Stiers P, Vanderkelen R, Vanneste G, Coene S, De Rammelaere M, Vandenbussche E. Visual-perceptual impairment in a random sample of children with cerebral palsy. *Dev Med Child Neurol.* 2002;44:370-82.
25. McDermott S, Coker AL, Mani S, Krishnaswami S, Nagle RJ, Barnett-Queen LL, Wuori DF. A population-based analysis of behavior problems in children with cerebral palsy. *J Pediatr Psychol.* 1996;21:447-63.
26. Parkes J, White-Koning M, Dickinson HO, Thyen U, Arnaud C, Beckung E, Fauconnier J, Marcelli M, McManus V, Michelsen SI, Parkinson K, Colver A. Psychological problems in children with cerebral palsy: a cross-sectional European study. *J Child Psychol Psychiatry.* 2008;49:405-13.
27. Krumlinde-Sundholm L, Eliasson AC. Comparing tests of tactile sensibility: aspects relevant to testing children with spastic hemiplegia. *Dev Med Child Neurol.* 2002;44:604-12.

28. Sanger TD, Kukke SN. Abnormalities of tactile sensory function in children with dystonic and diplegic cerebral palsy. *J Child Neurol.* 2007;22:289-93.
29. Van Heest AE, House J, Putnam M. Sensibility deficiencies in the hands of children with spastic hemiplegia. *J Hand Surg Am.* 1993;18:278-81.
30. Karaman MI, Kaya C, Caskurlu T, Guney S, Ergenekon E. Urodynamic findings in children with cerebral palsy. *Int J Urol.* 2005;12:717-20.
31. Roijen LE, Postema K, Limbeek VJ, Kuppevelt VH. Development of bladder control in children and adolescents with cerebral palsy. *Dev Med Child Neurol.* 2001;43:103-7.
32. Sankar C, Mundkur N. Cerebral palsy-definition, classification, etiology and early diagnosis. *Indian J Pediatr.* 2005;72:865-8.
33. Sarah W. Cerebral palsy. *Handbook of intellectual and developmental disabilities: Springer* 2007. p. 61-80.
34. Himmelmann K, Hagberg G, Beckung E, Hagberg B, Uvebrant P. The changing panorama of cerebral palsy in Sweden. IX. Prevalence and origin in the birth-year period 1995–1998. *Acta Paediatr.* 2005;94:287-94.
35. Liptak GS, Accardo PJ. Health and social outcomes of children with cerebral palsy. *The Journal of Pediatrics.* 2004;145:S36-S41.
36. Ashwal S, Russman BS, Blasco PA, Miller G, Sandler A, Shevell M, Stevenson R. Practice parameter: diagnostic assessment of the child with cerebral palsy: report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology.* 2004;62:851-63.
37. Bax M, Goldstein M, Rosenbaum P, Leviton A, Paneth N, Dan B, Jacobsson B, Damiano D. Proposed definition and classification of cerebral palsy, April 2005. *Dev Med Child Neurol.* 2005;47:571-6.
38. Krägeloh-Mann I, Cans C. Cerebral palsy update. *Brain Dev.* 2009;31:537-44.
39. Hutton JL, Cooke T, Pharoah PO. Life expectancy in children with cerebral palsy. *BMJ.* 1994;309:431-5.
40. Clark SL, Hankins GDV. Temporal and demographic trends in cerebral palsy—Fact and fiction. *Am J Obstet Gynecol.* 2003;188:628-33.
41. Sellier E, Platt MJ, Andersen GL, Krageloh-Mann I, De La Cruz J, Cans C. Decreasing prevalence in cerebral palsy: a multi-site European population-based study, 1980 to 2003. *Dev Med Child Neurol.* 2016;58:85-92.
42. Reid SM, Meehan E, McIntyre S, Goldsmith S, Badawi N, Reddihough DS. Temporal trends in cerebral palsy by impairment severity and birth gestation. *Dev Med Child Neurol.* 2016;58 Suppl 2:25-35.

43. Oskoui M, Coutinho F, Dykeman J, Jette N, Pringsheim T. An update on the prevalence of cerebral palsy: a systematic review and meta-analysis. *Dev Med Child Neurol.* 2013;55:509-19.
44. Odding E, Roebroek ME, Stam HJ. The epidemiology of cerebral palsy: incidence, impairments and risk factors. *Disabil Rehabil.* 2006;28:183-91.
45. Ravn SH, Flachs EM, Uldall P. Cerebral palsy in eastern Denmark: declining birth prevalence but increasing numbers of unilateral cerebral palsy in birth year period 1986-1998. *Eur J Paediatr Neurol.* 2010;14:214-8.
46. Surman G, Bed HN, Johnson A. Cerebral palsy rates among low-birthweight infants fell in the 1990s. *Dev Med Child Neurol.* 2003;45:456-62.
47. Nordmark E, Hagglund G, Lagergren J. Cerebral palsy in southern Sweden I. Prevalence and clinical features. *Acta Paediatr.* 2001;90:1271-6.
48. Sigurdardottir S, Thorkelsson T, Halldorsdottir M, Thorarensen O, Vik T. Trends in prevalence and characteristics of cerebral palsy among Icelandic children born 1990 to 2003. *Dev Med Child Neurol.* 2009;51:356-63.
49. Dolk H, Parkes J, Hill N. Trends in the prevalence of cerebral palsy in Northern Ireland, 1981–1997. *Dev Med Child Neurol.* 2006;48:406-12.
50. Hagberg B, Hagberg G, Beckung E, Uvebrant P. Changing panorama of cerebral palsy in Sweden. VIII. Prevalence and origin in the birth year period 1991-94. *Acta Paediatr.* 2001;90:271-7.
51. Himmelmann K, Hagberg G, Uvebrant P. The changing panorama of cerebral palsy in Sweden. X. Prevalence and origin in the birth-year period 1999-2002. *Acta Paediatr.* 2010;99:1337-43.
52. Andersen GL, Irgens LM, Haagaas I, Skranes JS, Meberg AE, Vik T. Cerebral palsy in Norway: prevalence, subtypes and severity. *Eur J Paediatr Neurol.* 2008;12:4-13.
53. Sellier E, Surman G, Himmelmann K, Andersen G, Colver A, Krageloh-Mann I, De-la-Cruz J, Cans C. Trends in prevalence of cerebral palsy in children born with a birthweight of 2,500 g or over in Europe from 1980 to 1998. *Eur J Epidemiol.* 2010;25:635-42.
54. Mongan D, Dunne K, O'Nuallain S, Gaffney G. Prevalence of cerebral palsy in the West of Ireland 1990-1999. *Dev Med Child Neurol.* 2006;48:892-5.
55. Colver AF, Gibson M, Hey EN, Jarvis SN, Mackie PC, Richmond S. Increasing rates of cerebral palsy across the severity spectrum in north-east England 1964–1993. *Arch Dis Child Fetal Neonatal Ed.* 2000;83:F7-F12.
56. Wichers MJ, van der Schouw YT, Moons KG, Stam HJ, van Nieuwenhuizen O. Prevalence of cerebral palsy in The Netherlands (1977-1988). *Eur J Epidemiol.* 2001;17:527-32.
57. Serdaroglu A, Cansu A, Ozkan S, Tezcan S. Prevalence of cerebral palsy in Turkish children between the ages of 2 and 16 years. *Dev Med Child Neurol.* 2006;48:413-6.

58. Park MS, Kim SJ, Chung CY, Kwon DG, Choi IH, Lee KM. Prevalence and lifetime healthcare cost of cerebral palsy in South Korea. *Health Pol.* 2011;100:234-8.
59. Touyama M, Touyama J. Prevalence of cerebral palsy in Okinawa between 1995 and 2001. *No To Hattatsu.* 2008;40:387-92.
60. Yam WK, Chan HS, Tsui KW, Yiu BP, Fong SS, Cheng CY, Chan CW. Prevalence study of cerebral palsy in Hong Kong children. *Hong Kong Med J.* 2006;12:180-4.
61. Liang Y, Guo X, Yang G, Yan X, Li X, Li G, Lan D, Li S, Wang Y, Ding H, Liu Y, Liu J, Lin Q. Prevalence of cerebral palsy in children aged 1 - 6 in Guangxi, China. *Zhonghua Yu Fang Yi Xue Za Zhi.* 2002;36:164-6.
62. Smith L, Kelly KD, Prkachin G, Voaklander DC. The prevalence of cerebral palsy in British Columbia, 1991-1995. *Can J Neurol Sci.* 2008;35:342-7.
63. Winter S, Autry A, Boyle C, Yeargin-Allsopp M. Trends in the prevalence of cerebral palsy in a population-based study. *Pediatrics.* 2002;110:1220-5.
64. Hamdy NE-T, Wafaa MAF, Ghaydaa AS, Nabil AM, Tarek AR, Noha A-E. Epidemiology of cerebral palsy in El-Kharga District-New Valley (Egypt). *Brain Dev.* 2011;33:406-11.
65. McAdams RM, Juul SE. Cerebral palsy: prevalence, predictability, and parental counseling. *NeoReviews.* 2011;12:e564-e74.
66. Topp M, Uldall P, Greisen G. Cerebral palsy births in eastern Denmark, 1987-90: implications for neonatal care. *Paediatr Perinat Epidemiol.* 2001;15:271-7.
67. Wu YW, Croen LA, Shah SJ, Newman TB, Najjar DV. Cerebral palsy in a term population: risk factors and neuroimaging findings. *Pediatrics.* 2006;118:690-7.
68. Drummond PM, Colver AF. Analysis by gestational age of cerebral palsy in singleton births in north-east England 1970-94. *Paediatr Perinat Epidemiol.* 2002;16:172-80.
69. Robertson CM, Watt MJ, Yasui Y. Changes in the prevalence of cerebral palsy for children born very prematurely within a population-based program over 30 years. *JAMA.* 2007;297:2733-40.
70. Vincer MJ, Allen AC, Joseph KS, Stinson DA, Scott H, Wood E. Increasing prevalence of cerebral palsy among very preterm infants: a population-based study. *Pediatrics.* 2006;118:e1621-6.
71. Andersen G, Romundstad P, De La Cruz J, Himmelmann K, Sellier E, Cans C, Kurinczuk J, Vik T. Cerebral palsy among children born moderately preterm or at moderately low birthweight between 1980 and 1998: a European register-based study. *Dev Med Child Neurol.* 2011;53:913.
72. Platt MJ, Cans C, Johnson A, Surman G, Topp M, Torrioli MG, Krageloh-Mann I. Trends in cerebral palsy among infants of very low birthweight (<1500 g) or born prematurely (<32 weeks) in 16 European centres: a database study. *Lancet.* 2007;369:43-50.

73. Keith LG, Oleszczuk JJ, Keith DM. Multiple gestation: reflections on epidemiology, causes, and consequences. *Int J Fertil Womens Med.* 2000;45:206-14.
74. Bodine A. Who does cerebral palsy affect? : Demand Media; 2011 [cited 2012 23 October]. Available from: <http://www.essortment.com/cerebral-palsy-affect-26930.html>.
75. Miller F, Browne E. *Cerebral palsy*: Springer; 2005.
76. Raina P, O'Donnell M, Rosenbaum P, Brehaut J, Walter SD, Russell D, Swinton M, Zhu B, Wood E. The health and well-being of caregivers of children with cerebral palsy. *Pediatrics.* 2005;115:e626-e36.
77. Hamzat T-hK, Mordi EL. Impact of caring for children with cerebral palsy on the general health of their caregivers in an African community. *Int J Rehabil Res.* 2007;30:191-4
78. Manuel J, Naughton MJ, Balkrishnan R, Paterson Smith B, Koman LA. Stress and adaptation in mothers of children with cerebral palsy. *J Pediatr Psychol.* 2003;28:197-201.
79. Blair E, Watson L. Epidemiology of cerebral palsy. *Semin Fetal Neonatal Med.* 2006;11:117-25.
80. Miller F. *Physical therapy of cerebral palsy*: Springer; 2007.
81. Krigger KW. Cerebral palsy: an overview. *Am Fam Physician.* 2006;73:91-100.
82. Jones MW, Morgan E, Shelton JE, Thorogood C. Cerebral palsy: introduction and diagnosis (Part I). *J Pediatr Health Care.* 2007;21:146-52.
83. Early Support. Information for parents: cerebral palsy. 3rd ed: DCSF Publications; 2010.
84. Parker J, Parker P. *The official parent's sourcebook on cerebral palsy*: Icon Health; 2002.
85. Moster D, Lie RT, Irgens LM, Bjerkedal T, Markestad T. The association of Apgar score with subsequent death and cerebral palsy: A population-based study in term infants. *J Pediatr.* 2001;138:798-803.
86. Croen LA, Grether JK, Curry CJ, Nelson KB. Congenital abnormalities among children with cerebral palsy: More evidence for prenatal antecedents. *J Pediatr.* 2001;138:804-10.
87. Paneth N. Cerebral palsy in term infants—birth or before birth? *J Pediatr.* 2001;138:791-2.
88. MacLennan A. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. *BMJ.* 1999;319:1054-9.
89. Miller F, Bachrach SJ. *Cerebral palsy: a complete guide for caregiving*: Johns Hopkins University Press; 2006.
90. Reddihough DS, Collins KJ. The epidemiology and causes of cerebral palsy. *Aust J Physiother.* 2003;49:7-12.
91. Thorngren-Jerneck K, Herbst A. Perinatal factors associated with cerebral palsy in children born in Sweden. *Obstet Gynecol.* 2006;108:1499-505

92. Dolk H, Pattenden S, Johnson A. Cerebral palsy, low birthweight and socio-economic deprivation: inequalities in a major cause of childhood disability. *Paediatr Perinat Epidemiol.* 2001;15:359-63.
93. Pharoah PO. Risk of cerebral palsy in multiple pregnancies. *Obstet Gynecol Clin N Am.* 2005;32:55-67.
94. Topp M, Huusom LD, Langhoff-Roos J, Delhumeau C, Hutton JL, Dolk H. Multiple birth and cerebral palsy in Europe: a multicenter study. *Acta Obstet Gynecol Scand.* 2004;83:548-53.
95. Ozturk A, Demirci F, Yavuz T, Yildiz S, Degirmenci Y, Dosoglu M, Avsar Y. Antenatal and delivery risk factors and prevalence of cerebral palsy in Duzce (Turkey). *Brain Dev.* 2007;29:39-42.
96. Jacobsson B, Hagberg G, Hagberg B, Ladfors L, Niklasson A, Hagberg H. Cerebral palsy in preterm infants: a population-based case-control study of antenatal and intrapartum risk factors. *Acta Paediatr.* 2002;91:946-51.
97. O'Shea TM, Klinepeter KL, Dillard RG. Prenatal events and the risk of cerebral palsy in very low birth weight infants. *Am J Epidemiol.* 1998;147:362-9.
98. Pschirrer ER, Yeomans ER. Does asphyxia cause cerebral palsy? *Semin Perinatol.* 2000;24:215-20.
99. Sukhov A, Wu Y, Xing G, Smith LH, Gilbert WM. Risk factors associated with cerebral palsy in preterm infants. *J Matern Fetal Neonatal Med.* 2012;25:53-7.
100. Andersen GL, Irgens LM, Skranes J, Salvesen KA, Meberg A, Vik T. Is breech presentation a risk factor for cerebral palsy? A Norwegian birth cohort study. *Dev Med Child Neurol.* 2009;51:860-5.
101. Radell U, Tillberg E, Mattsson E, Amark P. Postnatal cerebral infection leading to hemiplegic cerebral palsy: functional limitations and disability of 13 children in Sweden. *Disabil Rehabil.* 2008;30:1910-9.
102. Walstab JE, Bell RJ, Reddihough DS, Brennecke SP, Bessell CK, Beischer NA. Factors identified during the neonatal period associated with risk of cerebral palsy. *Aust New Zeal J Obstet Gynaecol.* 2004;44:342-6.
103. Cans C, McManus V, Crowley M, Guillem P, Platt M-J, Johnson A, Arnaud C, group Sc. Cerebral palsy of post-neonatal origin: characteristics and risk factors. *Paediatr Perinat Epidemiol.* 2004;18:214-20.
104. Murphy DJ, Hope PL, Johnson A. Neonatal risk factors for cerebral palsy in very preterm babies: case-control study. *BMJ.* 1997;314:404.
105. Nelson KB. Causative factors in cerebral palsy. *Clin Obstet Gynecol.* 2008;51:749-62.
106. Blair E, Stanley F. Causal pathways to cerebral palsy. *Current Paediatrics.* 2002;12:179-85.



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