SPASTICITY MANAGEMENT ANDNEUROPHYSIOLOGIC CHANGES IN CEREBRAL PALSY CHILDREN

Thesis

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{ وَأَنْزَلَ اللهُ عَلَيْكَ الْكِتَابَ وَالْحِكْمَةَ وَعَلَّمَكَ مَا لَمْ تَكُنْ تَعْلَمُ وَكَانَ فَضْلُ اللهِ عَلَيْكَ عَظِيمًا }

صدق الله العظيم

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@ Lamis Mohamed El-Shakankery

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List of Abbreviations

AAN	American Academy of Neurology
ADF-MMST	Ankle DorsiFlexor muscle manual strength test
ADLs.....	Activities of Daily Living
AJDF-ROM.....	Ankle Joint DorsiFlexion range of motionby hand held goniometer
AS	Ashworth Scale
CP.....	Cerebral palsy
EMG	Electromyography
GABA	Gamma Aminobutyric Acid
GABAB	Gamma Amino Buteric Acid B receptor
GMFCS.....	Gross motor functional classification system for CP
H-reflex.....	Hoffman reflex
MAS.....	Modified Ashworth Scale
Mmax.....	Supramaximal
NDT	Bobath neurodevelopmental treatment
OFC	Occipito frontal circumference
ROM.....	Range of Motion
SD.....	Standard deviatio

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INTRODUCTION

Cerebral palsy (CP) is a static encephalopathy that may be defined as anonprogressive disorder of posture and movement resulting from a defect or lesion of the developing brain. It is a common disorder, with an estimated prevalence of two in 1000 population ***(Dzienkowski et al.,1996)***.

CP is caused by a group of developmental, genetic, metabolic, ischemic, infectious and other acquired etiologies that produce a common group of neurologic phenotypes ***(Johnston Michael, 2007).***

The incidence of CP has varied in different series according to criteria of selection, time and community studied. A figure of between 1 and 3 cases per 1,000 live births has been *quoted* ***(Nettina, 2001; Reddihough and Collins, 2003).***

Upper motor lesion produces musclespasticity which increases the resistance againstpassive movements. Spasticity disturbs walking and functional abilities of patients***(Feng and Mak,1997).***

Spasticity is characterized by the increase in tendon reflex and tonic stretch reflex because of the hyper-excitability of the stretch reflex after upper motor neuron lesion ***(Brunstrom,2001).***

Different methods are used to evaluate spasticity. These include subjective methods such as passive goniometric measurement and clinical ratio scales(modified Ashworth scale), and objective methods such as electrophysiologic tests(Hoffman(H)reflex and H/M ratios) ***(Moore,1998).***

There is a general agreement that spasticity treatment is important***(Dones et al.,2006).***

Various treatments have been recommended to reduce spasticity, including surgical, medical and physiotherapy techniques***(Albright,2003).***

Methods such as drug therapy, chemical nerve block or neurosurgical treatments may reduce spasticity but may cause muscle weakness or paralysis***(Carmick,1993).***

The aims of physiotherapy techniques used for the treatment of spasticity are to favor sensorimotor recovery, which leads to optimal independence in daily life activities***(Bakke,1995).***

AIM OF THE WORK

To investigate the effect of antispasticity management on clinical and neurophysiologic measurements in cerebral palsy children with lower limb spasticity.

Chapter(1)

CEREBRAL PALSY

Cerebral palsy (CP) is the most common chronic disability of childhood today. It is ubiquitous and it occurs all around the world.In developed nations, the incidence is about 1 to 2 per 1000 births ***(Nadire et al., 2011).***

Definition

Cerebral palsy (CP) describes a group of disorders of the development of movement and posture, causing activity limitations, which are attributed to non-progressive disturbances that occurred in the developing foetal or infant brain. The motor disorders of CP are often accompanied by disturbances of sensation, cognition, perception, behaviour and/or seizure disorders and by secondary musculoskeletal problems***(Rosenbaum et al.,2011).***

Epidemiology

The incidence of CP has varied in different series according to criteria of selection, time and community studied. A figure of between 1 and 3 cases per 1,000 live births has been quoted ***(Reddihough and Collins, 2003)***.

Risk Factors

A multitude of risk factors both environmental and genetic has been associated with the development of CP(Box 1).

Table (1): Cerebral palsy risk factors:

Prenatal risk factors:

Prenatal risk factors include hyperemesis gravidarum, pregnancy-induced hypertension, threatened fetal loss, placenta previa, abruptio placentae, teratogenic drugs, intrauterine bacterial and viral infections and maternal malnutrition.

Natal risk factors:

Natal risk factors include breech delivery, multiple gestation, asphyxia, low Apgar score and especially prematurity and low birth weight.

Postnatal risk factors:

Postnatal risk factors include head trauma, seizures, hyper-bilirubinemia, intracranial infections, toxic encephalopathies and cerebral and intraventricular hemorrhages.

(Jacobsson and Hagberg, 2004).

Despite this extensive list, 17-60% of infants with CP have experienced no recognizable adverse event. Although risk factors analysis is not a very specific or sensitive predictor of CP, risk factors should not be ignored. It is important to take a careful history of prenatal, natal and postnatal events ***(Jacobsson and Hagberg, 2004)***.

Other risk factors associated with an increased risk of CP include patent ductusarteriosus, hypotension, blood transfusion, prolonged ventilation, pneumothorax, sepsis, hyponatremia and total parenteral nutrition. Seizures were associated with an increased risk of CP as were parenchymal damage and an appreciable ventricular dilatation ***(Stelmach et al., 2005)***.

Pathology

The site of lesion and the type of disability varies with the gestational age. In preterm infants, the injury usually involves the white matter and the motor fibers of the lower extremities; thus injured preterm infants generally have a spastic diplegia, primarily of the legs, with normal or near-normal cognitive development because the gray matter of the cortex is not injured. This injury in term infants, usually from a hypoxic ischemic insult such as perinatal asphyxia, usually results in a parasagittal cortical lesions and leads to involvement of the upper extremities, face and tongue with impairment of speech ***(Stelmach et al., 2005)***.

Classification

Table (2): Cerebal palsy subtypes.

Type	Subtype
Spastic	***Diplegia:*** 30% - 40% of spastic CP; 50% were born preterm
	Hemiplegia: 20% - 30% of spastic CP; associated withstrokes, vascular malformations.
	Quadriplegia: 10% - 15% of spastic CP; associated with severe asphyxia in all infants
	Monoplegia/Triplegia
Nonspastic	***Dyskinetic:*** Damage to basal ganglia or thalamus (deep motor neurons)
	Ataxic: Damage to neurons in cerebellum**.**

(Pueyo et al., 2003).

Another classification:

Table (3): Another classification for cerebral palsy

Pyramidal	*Extrapyramidal*	*Mixed forms*	*Other*
Spastic diplegia	Dyskinetic		Hypotonic
Spastic tetraplegia	Athetosis		
Spastic hemiplegia	Ataxia		

(Panteliadis and Thessaloniki, 2009)

The CP is generally classified using a combination of physiologic and anatomic types. Under the physiologic categorization are the pyramidal, extrapyramidal or mixed types.

The pyramidal type generally indicates injury of the cortical system and commonly results in a spastic presentation. Such patients demonstrate hyperreflexia, with the typical "clasp knife" type of hypertonia. Because of the resultant spastic musculature, the growing child with pyramidal CP is prone to contractures ***(Sankar and Mundkur, 2005)***.

Extrapyramidal type injuries, such as those to the basal ganglia and cerebellum, may result in disorders of motion, such as athetosis and ataxia. The tone abnormality is the "lead pipe" rigidity rather than spasticity. In presence of excessive motion, such as athetosis, contractures are uncommon. However, some investigators believe that the great variability of motion patterns leads to less predictable surgical results ***(Sankar and Mundkur, 2005)***.

CP may be classified by clinical type of the motor handicap in term of physiologic, topographic, etiologic categories and functional capacity (table 4).