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Cerebral palsy: Physical therapy and medical treatment issues

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1. Introduction

Cerebral palsy (CP) is a broad term used for a number of developmental disorders that affect a child's movement, posture maintenance and balancing abilities. These categories of disorders are the result of a nonprogressive abnormality of the immature brain. CP is sometimes caused by a brain injury that occurs before birth, during birth or within the first few years after birth. This type of injury is classified as neurological damage and is often associated with premature births, difficult deliveries, neonatal medical complications, or brain trauma. CP may also be caused by developmental brain malformations. Sometimes the brain may not develop the usual number of brain cells, migration of the brain cells may be flawed, or genetic disorders and chromosome abnormalities may come into play. Developmental brain malformations in areas of the brain which control voluntary movement are another potential cause.[19]

CP affects the ability of the brain to control the muscles but does not directly damage the muscles or nerves connecting them to the spinal cord. Depending on which areas of the brain have been damaged, the child may have one or more symptoms including muscle tightness or spasms, involuntary movement, difficulty with gross motor skills such as walking or running, difficulty with fine motor skills such as writing or buttoning a shirt, and difficulty in perception and sensation.[26] Impairment can vary considerably in that no two individuals with CP are affected in exactly the same way. At its mildest, CP may result in a slight awkwardness of movement or hand control. At its most severe, it may result in virtually no muscle control, profoundly affecting movement and speech. This disorder may also have comorbidities including mental retardation, language disorders, seizures, learning disabilities, behavioral challenges and vision and hearing problems. Recognizing and managing these is as important as treating the motor disabilities.

More people in the United States have cerebral palsy than any other developmental disorder, including Down Syndrome, epilepsy and autism.[15] It is the most common physical disability in childhood, and the incidence of CP in developed countries is about 2-2.5 per 1000 live births.[23,30] In the United States, this translates to approximately 10,000 babies born with CP each year.[29] The prevalence of this disorder is not decreasing, therefore it is imperative to continue to undertake scientific and clinical trials to measure the efficacy of commonly used medical and physical therapy interventions.

Studies using Magnetic Resonance Imaging (MRI) have shown that as many as 77% of children diagnosed with CP have brain malformations that developed during pregnancy.[29] This contrasts the long-held belief that CP results from improper care or hypoxia during delivery. Risk factors for CP include premature birth, low birth weight, inability of the placenta to provide the developing fetus with oxygen and nutrients, lack of growth factors during intra-uterine life, Rh and A-B-O blood type incompatibility between mother and infant, infection of the mother with viral diseases or bacterial infection during early pregancy, prolonged loss of oxygen during the birthing process and severe jaundice shortly after birth.[37] Pinpointing which, if any, risk factor caused a child's brain injury is difficult. There is no combination of risk factors that always result in CP, and the majority of the cases are of unknown etiology.

A number of prevention measures for CP are in place in today's medical community. Pregnant women are now tested for Rh factor and, if they are Rh negative, they can be immunized within 72 hours of giving birth. This prevents any adverse consequences of blood incompatibility in a subsequent pregnancy.[26] Newborns with jaundice can also be treated with phototherapy. In addition, educational programs stress the importance of optimal well-being prior to conception and adequate prenatal care. Safety campaigns advise parents on ways to protect children from accidents and injury.

There are many issues associated with the diagnosis and assessment of children with CP. Development of the brain starts in early pregnancy and continues until about age 3. Damage to the brain in this time period may result in CP.[26] The onset of the disorder frequently occurs before the diagnosis is made, with early signs usually appearing by 18 months of age. Parents are generally the first to suspect that the infant is not developing motor skills normally. Medical experts use a number of diagnostic techniques to assess whether or not a child is afflicted with CP.

Computerized Tomography (CT) is an imaging technique that uses X-rays and a computer to create a three-dimensional picture of the brain's tissues and structures. MRI, mentioned earlier, uses magnetic fields to provide detailed pictures of the brain and spine.[19] Both of these tests help to identify lesions on the brain. Children with CP have brain scars, cysts and other changes which show up on scans more frequently than in normal children.[35] In addition, head sonograms use sound waves to visualize structures of the brain and electroencephalograms (EEGs) measure the electrical activity of the brain and help identify seizure activity.

Children with CP fail to reach motor milestones and show qualitative differences in motor development, such as asymmetric gross motor function and muscle stiffness or floppiness. A lack of variability of limb movements or sustained cramped postures also indicate possible motor problems.[23] Most clinicians delay the formal diagnosis of CP until the child is 2 years old, because signs may remain hidden until the nervous system is more fully developed. The National Collaborative Perinatal Project in the USA endorses this caution when considering the fact that two-thirds of children diagnosed with spastic diplegia, and half of all children with signs of CP at one year of age, "outgrow" their symptoms by age 7.[23]

The complexity of the disorder warrants a team approach to care. Investigators from several areas of medicine and health care are using their expertise to help improve the treatment

and prevention of CP. Children diagnosed with one of the many forms of CP may see pediatricians, orthopedic surgeons, physical and occupational therapists, speech-language pathologists, social workers and educators. Physical therapists, in particular, are essential members of the team and help children with CP optimize development and live with as few limitations as possible. The goal of physical therapy (PT) includes identifying and treating problems with movement and body position. Physical therapists treat movement problems such as high or low muscle tone or weak muscles and facilitate the development of movement skills such as sitting, rolling, crawling and standing. They generally deal with gross motor skills that require the use of larger muscles. It is crucial that physical therapy begin at an early age for children with CP because the nervous system is most susceptible to change during the first five years of life.[15]

Early intervention services are often recommended for children under age two. They are intended to minimize effects of neurological conditions that make it difficult to learn and acquire the normal developmental skills. Physical therapists often recommend special equipment to make tasks such as feeding, moving and speaking easier. They also develop exercise programs to prevent complications that often come along with CP such as contractures, hip dislocations, and spine curvatures. Physical therapy enables children with CP to experience activities they most likely would not otherwise be able to do on their own. The creativity involved in pediatric PT seems never-ending, involving techniques such as special adaptations to favorite toys, teaching sign language, and the use of bubbles and games to keep children motivated and interested. Physical therapy programs also serve to educate the family of a child with CP as to how to make the most of his or her abilities.

In recent years, there have been several advances and new methods developed regarding treatment of children with CP. Evidence-based practice calls for the evaluation of these

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interventions on the basis of the effect that they have on the health and lives of the children who receive them. Studies explore the effect of the treatment on level of impairment, functional limitation, disability, spasticity and societal limitations. It is important to remember that CP is a disorder, is neither contagious nor hereditary, and people with this disorder may have a long and full life. However, children with severe forms of CP are more vulnerable to major respiratory illness and early mortality.[23] The standardized mortality rate is defined as the ratio of observed deaths to expected deaths. The standardized mortality rate for 15-34 year olds with CP is 5 to 16 times more than expected.[23] Children with CP who survive to 8 years of age have a life expectancy that ranges from 29 to 62 years, depending on their functional ability.[29]

A discussion of the various terms associated with CP as well as the different classifications will precede a review of several randomized controlled trials, case studies, cohort studies and other related articles regarding treatment methods. Specifically, the use of Intrathecal Baclofen Therapy (ITB), Botulinum toxin injections (Botox), Selective Dorsal Rhizotomy, Threshold Electrical Stimulation (TES), and Pediatric Constraint-Induced Movement Therapy will be examined.

2. Definitions and Types of Cerebral Palsy

The definition of cerebral palsy has been a major source of controversy in past years. "Cerebral" emphasizes the importance of the brain in the labeling of the condition, but "Palsy" is usually associated with paralysis, which generally suggests complete loss of movement.[15] This is not typical of CP. Even though the term is somewhat inaccurate, it has been proven useful in the design of medical and therapy programs, scientific studies, and for legislation and advocacy at the societal level. Three elements make up the complete definition of CP:[15]

1. *There is a significant problem with motor function.* CP is a motor impairment syndrome in that all individuals with CP have difficulties controlling movement and posture.

2. *The motor impairment is a result of a disturbance or anomaly to the brain during early development.* The brain develops at its quickest pace during the prenatal period and during the early postnatal years. Brain injury occurs in the motor cortex, cerebellum or basal ganglia.

3. *The disturbance to brain development occurred over a specific period of time and does not represent a continuing, recurrent, or progressive process.* Motor impairment of functional consequences of CP may change over time, but the underlying brain injury does not worsen. CP is thus a static condition, not a progressive condition.

Mutch et al. combine all of these elements into a more complete definition of CP that is widely accepted in the medical community:

Cerebral palsy is an umbrella term covering a group of nonprogressive, but often changing, motor impairment syndromes secondary to lesions or anomalies of the brain arising in early stages of development.[15]

Several different classification systems and labels have been developed to describe varying forms of CP. Muscle tone is a key term in understanding these classifications. This term is found in almost all literature associated with CP and refers to the amount of tension or resistance to movement in a muscle, or its stillness. In general, muscle tone allows an individual to keep a certain posture and to complete a movement smoothly. The tone in all muscle groups

must be balanced for proper movement to occur. All individuals with CP have a certain degree of damage to the area of the brain that controls muscle tone, and they may experience asymmetric muscle tone. The location of the brain damage determines the parts of the body affected by abnormal muscle tone. The following is a list of the types of muscle tone abnormalities that may occur.

- *High tone:* This is also known as *spasticity or hypertonia* and is characterized by increased muscle tone. Spasticity causes stiff and awkward movements due to tone imbalance and tight muscles.

- *Low tone:* This is also known as *floppiness or hypotonia* and is characterized by muscles that are too relaxed and do not contract enough. This causes problems with posture maintenance without support. If low tone affects abdominal and respiratory muscles, it can cause problems with speech development.

- *Fluctuating tone:* This is also sometimes called *variable tone* and results in a combination of high and low tone. Tone is generally low when at rest but often increases to high with active movement. Often, too many muscle groups tighten, which makes certain movements impossible.

It is necessary to look at the different types of classifications based on the type of muscle tone problem, location of brain injury, and according to areas of the body affected by movement problems. This knowledge is essential to understanding the studies that will be discussed shortly.

2.1 Classifications Based on Location of Brain Injury

Spastic (Pyramidal) cerebral palsy, affecting about 80% of all children with CP, is the most common type. This type is characterized by having one or more tight muscle groups which limit movement. It is due to damage in the motor cortex of the brain. There may also be damage to the pyramidal tracts, which are pathways that link the motor cortex to the spinal nerves that relay motor signals to the muscles. In effect, the brain has difficulty communicating with muscles on one or both sides of the body. Several other symptoms may also occur as a result of spastic CP. Exaggerated stretch reflexes may cause much stronger and faster reflexes than

normal. Ankle clonus is when muscles in the calf and foot rapidly and rhythmically contract if the foot is flexed upward or when standing. This type of CP also involves a tendency to develop contractures, which are abnormal shortenings of the muscles and tendons, limiting movement around a joint. Primitive reflexes may persist much longer than usual.[15,19]

Choreo-Athetoid (Extrapyramidal) cerebral palsy occurs in about 10% of children with CP and is caused by damage to the cerebellum or cerebral nuclei. These areas normally function to process signals from the motor cortex, enabling smooth, coordinated movements and maintaining posture. Specifically, the cerebellum helps to coordinate muscle activity, maintains muscle tone, and controls balance. The cerebral nuclei control adjustments in posture needed during movement.[19] Considering this, it seems logical that damage to these areas causes a child to develop involuntary, purposeless movements. This is especially apparent in the face, arms and trunk. There is often interference with speaking, feeding, reaching, grasping and other skills that require coordinated movements. Children with this type of CP have low muscle tone, which causes problems with posture maintenance for sitting and walking. There are several different types of involuntary movements including the following:[19]

- *dyskinesia* – general term for involuntary movements, used when exact type of movement is difficult to classify.

- *athetosis* – slow, writhing movements, especially in the wrists, fingers and face.

- *chorea* – abrupt, quick, jerky movements of the head, neck, arms or legs.

- *ataxia* – unsteadiness and lack of coordination in standing and walking and problems with balance due to damage to cerebellum.

- *dystonia* – slow, rhythmic, twisting movements.

- *rigidity* – extremely high muscle tone in any position combined with very limited movements.

Mixed-type cerebral palsy is seen in about 10% of children with CP and is characterized by having both spastic muscle tone and involuntary movements due to injuries to both the pyramidal and extrapyramidal tracts of the brain.

2.2 Classifications Based on Location of Movement Problems

CP is usually characterized clinically by the parts of the body affected, such as the face, arms, trunk and legs.[30]

- *Monoplegia* – only one limb (arm or leg) on one side of the body is affected.

- *Diplegia* – motor impairment primarily of the legs (usually with some relatively limited involvement of the arms).

- *Triplegia* – three limb involvement.

- *Hemiplegia* – one side of body is affected. The arm is usually more affected than legs, trunk or face. Arm or leg on affected side may be shorter or less developed.

- *Quadriplegia (tetraplagia)* – all four limbs, in fact the whole body, are functionally compromised. Legs and feet are generally more affected. Difficulty with most activities of daily living due to extent of motor disabilities.

- *Double Hemiplegia* – affects whole body, but the arms are the most affected.

Finally, functional status of individuals with CP is often categorized by using the gross motor classification system for cerebral palsy, which involves five levels of functioning with respect to gross motor activity.[30] This illustrates the severity of the CP presented:

- *Level I* – Walks without restrictions; limitations in more advanced gross motor skills.

- *Level II* – Walks without devices; limitations in walking outdoors and in the community.

- *Level III* – Walks with mobility devices; limitations in walking outdoors and in the community.

- *Level IV* – Self mobility with limitations; children are transported or use power mobility outdoors and in the community.

- *Level V* – Self mobility is severely limited even with the use of supporting technology.

2.3 Physical Therapy Evaluation and Treatment

The American Physical Therapy Association certifies specialists in pediatric physical therapy. There are even physical therapists who further specialize in treating developmental disabilities in infants and children. They may specialize in Neurodevelopmental Treatment (NDT), which is designed specifically for infants and children with motor handicaps including CP. Sensory Integration Therapy (S.I.) is another form of special training.[19] A variety of other specialized techniques are also used by physical therapists to treat children with CP, such as myofascial release (MFR) in which connective tissues around the joints and muscles are stretched, aquatic therapy, manual therapy used to influence the length of soft connective tissue, and hippotherapy (horseback riding).

There are several common problems of children with CP that often require physical therapy treatment. Some main categories include problems with posture, transitional movements (e.g. rolling from front to back), persistent primitive reflexes, balance, sensory impairments and joint mobility. Evaluation involves observing the child's movement patterns and identifying postures the child prefers that may delay acquisition of new skills. A physical therapist will also look for asymmetries, which are differing abilities of the child to use either side of the body caused by tone disturbances.[19] For example, a child with an asymmetry may only roll to one side or lean to one side when sitting.

Standardized tests are often used by physical therapists to assess a child's gross motor development. They also provide information about functional abilities, skeletal differences, posture, physical characteristics, strength, need for special equipment, and sensory and perceptual skills among others. Standardized methods of assessing functional status and

monitoring progress are essential in evaluating new treatments as well as measuring change in children with CP. Currently, many of the tests used are not sensitive enough to detect the small changes that occur over a long period of time. This is changing as evidence-based practice is becoming the standard in physical therapy.

In choosing an outcome measurement, criteria such as purpose of the measure, validity, reliability, responsiveness (ability to detect minimal clinically important change in subjects), and description of qualitative components needs to be taken into consideration. Some common tests include the Gross Motor Function Measure (GMFM), Pediatric Evaluation of Disability Inventory (PEDI), Functional Independence Measure for Children (WeeFIM), Peabody Developmental Motor Scales (PDMS), Toddler and Infant Motor Evaluation (TIME), Bayley Scales of Infant Development, and the Movement Assessment Inventory (MAI).[15,19] Some of these testing measures appear in the clinical studies to be discussed shortly.

After the initial evaluation of a child with CP, the physical therapist will talk with the caregiver about the strengths and impairments of the child and work together with the caregiver to set treatment goals. A baby is generally reevaluated every 3 to 6 months, while an older child is reevaluated every 6 to12 months to measure progress. The therapist stresses the importance of family involvement in treatment at home. Engaging the child with a sibling or another child is encouraged because children are often motivated to try new things if watching another child.

Children with CP generally see a physical therapist at least once per week and more often during times of rapid change in motor development. Physical therapists use repetition and practice heavily during sessions because this helps the child to refine and remember important skills. After a child has surgery to improve function in some way, physical therapy may begin within 24 to 48 hours after surgery in order to maximize progress. In general, a child usually

makes the most progress in the first 6-12 months after surgery.[19] Therefore, a physical therapist may see a child more frequently during this time frame.

2.4 Research

Advances in research are increasing our understanding of the etiology of CP, opportunities for primary prevention, and the effectiveness of specific intervention strategies. In recent years, the biomedical, surgical and therapeutic strategies for treatment of CP have greatly expanded. Four organizations that have developed major research programs into CP are the United Cerebral Palsy Research and Educational Foundation, the National Institutes of Health, the Centers of Disease Control and Prevention, and the National Institute of Disability and Rehabilitation Research.[37]

Ethical concerns for nontreatment control groups as an alternative to treatment have hindered the implementation of randomized controlled studies in the past. However, many researchers and members of the medical community also argue that it is unethical to deliver unproven treatments in that they result in significant monetary and psychological commitments for families. In both the medical and physical therapy communities, this has lead to strong adherence to the theory of evidence-based practice.

2.5 Evidence-Based Practice

Clinicians have a responsibility to be familiar with current research and apply scientific evidence in their practice. Physical therapists are under increasing pressure to remain knowledgeable about the various interventions for children with CP. Evidence-based medicine is defined as the conscientious, explicit and judicious use of the current best evidence in making decisions about the care of individual patients. It involves the integration of the best research evidence with clinical expertise and patient values.[2] In contrast to practicing evidence-based medicine, those who favor nonstandard therapies often provide no anatomical or physiological

basis for their intervention. They may have a broad and diverse target population, ignore side effects and limitations of the approach, or provide no research evidence to support their claims.[2] Clearly, this is not a scientific or effective approach to providing care. Today, physical therapists have to be able to justify what they do by demonstrating the effectiveness of their interventions, which enhances their ability to provide patients with optimal care.

The traditional focus is on impairments, but the model of evidence-based practice encourages physical therapists to look at the effects of a particular intervention on functional limitations and disability. An impairment is defined as a deviation from the normal such as not being able to make a muscle move or to control an unwanted movement.[35] Disability is a term used to define a restriction in the ability to perform a normal activity of daily living which someone of the same age is able to perform, for example, walking to school.[35] A primary focus should be to look at the overall quality of life, which involves the extent to which having a disability affects overall happiness and satisfaction of an individual.

In looking at studies on new interventions in physical therapy, *Levels of Evidence* have been developed and are applied to clinical situations to assess research results based on the level of scientific evidence. Below is a list of the characteristics of each level.[2]

- *Level 1*: Randomized controlled trials (RCTs). These studies have large numbers of subjects randomly assigned to either an intervention group or control group. This generates a great deal of confidence in the positive or negative results, and these can be generalized to patients similar to those studied.

- *Level 2*: Cohort (group) studies. These studies do not involve random group assignment and involve smaller RCTs. Less confidence is placed in these studies.

- *Level 3*: Case-control studies. These are not common in physical therapy.

- *Level 4*: Case series. Series of patients with an outcome of interest are reported, but no control group is used. This type of study is prevalent in PT.

These lower-level research results are only suggestive and provide a basis of higher-level studies.

- *Level 5*: Expert opinion

Currently, there are few studies with *Level 1 Evidence* to support the effects of PT on movement dysfunction in children with CP. However, these are becoming more prevalent and will lead to stronger evidence regarding intervention methods. Strength of the evidence depends on the number of people who have been studied, the consistency of the findings across studies, and the internal validity of the body of evidence. Physical therapists try to keep up to date by attending continuing education courses, reading textbooks, and reading systematic reviews. There are a number of approaches to synthesizing and summarizing available evidence so that PTs can more quickly and easily evaluate a treatment option. A few of the more common ways are described below.[2]

- *Systematic review:* summarizes available research studies following established guidelines for the literature search, assessment, and statistical analyses. Systematic reviews of high-quality RCTs with similar findings are placed in *Level 1*. These help to provide insight into current evidence.

- *Meta-analysis:* one of the best methods of quantitatively assessing the results of multiple studies. In this statistically based analysis, the results of numerous studies are compiled and an overall conclusion is derived from all of the studies analyzed.

- *Consensus conference:* establishes expert agreement on an issue by reviewing the evidence and producing a group synopsis and recommendations. Therefore, the biases of one person or a few people are not as apparent.

2.6 Spasticity

In many of the approaches to treatment of CP that will be examined in the next section, reducing the impairment of spasticity is a primary goal and outcome measure. Therefore, it is important to understand the perceived role that this impairment plays in children with CP. It has been attributed as a major contributor to movement dysfunction in these children. It is also a common problem in many other conditions that affect motor function, including stroke, head

injury, spinal cord injury, multiple sclerosis, and various dystrophies. Spasticity involves the inability of the muscles to relax. Increased muscle tone and resistance to high-velocity stretching characterize spastic type CP. Increased tone results from the hyperexcitability of the alpha motor neurons. Spasticity also causes a reduction in longitudinal muscle growth, which is thought to be caused by an increase in collagen in spastic muscles.[29] The lesion in the central nervous system of individuals with CP frequently results in the spasticity of various muscle groups.[10]

The motor function of children with CP depends on the interaction between spastic agonist muscles and antagonists, which atrophy due to inactivity.[11] Children often develop abnormal posture and gait, which are characterized by spasms in extensor, flexor and adductor muscles that create toe-walking and scissoring of the legs.[21] Spasticity also causes a considerable increase in energy expenditure.[36] It can negatively affect the development, school life, and social life of children with CP. The goal of many interventions involves relaxation of the spastic muscles, which facilitates stretching, promotes growth, prevents contractures, and allows for the strengthening of the weak antagonist muscles.[14]

The measurement of spasticity reduction has differed across studies, which makes comparison difficult. Also, some of these studies have reported positive results in spasticity reduction even though the study had methodological problems or findings that were not statistically significant. However, the most commonly used scale for assessing spasticity is the Ashworth Scale.[1] Certain treatments for spasticity have been shown to have risks and side effects that may limit their clinical use or outweigh the benefits. Physicians, physical therapists and parents need to consider the long-term effects of these medications and procedures due to the fact that children with CP will most likely receive treatment over a lifetime. Few research studies have looked at the treatment of spasticity in children, but this is changing. Most treatments have

been tested in adults. Obviously, there are many differences between children and adults in terms of metabolism, developmental issues, and other major factors.

From this discussion, one can conclude that an underlying assumption in the treatment of spasticity is that it may impede or mask existing motor control. However, some researchers argue that reduction of spasticity may impair rather than improve motor function if individuals rely upon their stiffness for support during walking or standing.[7] This debate is ongoing and requires further research. Many studies present compelling evidence that even with reduced spasticity, there is often no evidence of improved function. This brings into light the fact that physical therapists should also emphasize exercise training principles to improve strength, range of motion, posture and coordination instead of only emphasizing reduced spasticity and tone.[21]

3. Analysis of Specific Treatment Methods for CP

3.1 Intrathecal Baclofen Therapy

Intrathecal baclofen therapy (ITB) is a neurosurgical procedure that involves the use of baclofen introduced directly into the spinal canal. An externally programmable pump is implanted subcutaneously into the lateral abdominal wall and is attached to a catheter that tunnels under the skin and is connected to an intrathecal (within the spinal cord) catheter at the distal thoracic or lumbar spine.[15] The U.S. Food and Drug Administration approved ITB for use in the management of spasticity of cerebral origin in 1996 for patients four years of age and older.[1] Studies of continuous intrathecal baclofen infusion use in children with CP have shown significant improvements in the ability to perform activities of daily living as well as improvements in muscle tone, joint range, hamstring motion, and upper extremity function. It has also been found to be effective in management of dystonia, which is characterized by slow, twisting movements.[15]

Baclofen is an centrally acting GABA (gamma-aminobutyric acid) agonist that appears to reduce spasticity primarily by acting on inhibitory pathways within the spinal cord. Its exact mechanism of action is based on the premise that spasticity is associated with the inadequate release of GABA, which is an inhibitory neurotransmitter. The molecular structure of baclofen closely resembles that of GABA and seems to stimulate GABA b receptors.[7] Direct delivery to the cerebrospinal fluid allows baclofen to penetrate only a few millimeters to receptor sites in the spinal cord. This delivery system reduces systemic effects of oral administration, which often include drowsiness and lethargy. Also, this allows for the doses to be titrated in very small amounts to allow precise dosing.[1] Oral baclofen has been used for spasticity in CP since 1977, but this type of administration results in virtually undetectable levels of the drug in the spinal

cord. Intrathecally administered baclofen, on the other hand, at 1/100th the dose, results in cerebral spinal fluid levels comparable to serum levels following oral medication.[7]

Long-term tolerance to ITB has been good in general, with devices that have been implanted for as long as four years.[15] However, there have been no studies to determine the long-term effects on the developing brain. The American Academy for Cerebral Palsy and Developmental Medicine has completed systematic reviews of intrethecal baclofen using their framework as well as the levels of evidence.[7] They looked at fourteen studies with ITB involving about 200 participants. Overall, these studies showed that ITB reduces spasticity to a greater extent in the lower extremities. Affect on spasticity in the upper extremities was unclear through these studies. It was inconclusive because the findings were inconsistent, with five results indicating improvements and five indicating no change. Higher concentrations of ITB or inserting the catheter at a higher level have been suggested to improve effect in the upper extremities.[7]

Also through this systematic review, dystonia was shown to improve after ITB. Dystonia is characterized by frequent, involuntary sustained muscle contractions that cause abnormal postures or twisting and repetitive movements. These individuals were more comfortable, had less facial grimacing, trunk arching and other abnormal posturing. In addition, function and ease of care improved overall. Improved voluntary movement control was also shown with ITB use by both measures. This review commented that only a small number of people have been studied to date and more rigorous investigation of ITB is needed. A greater understanding of other factors such as age, expense, and dosage level is also needed. The American Academy for CP found consistent evidence that ITB suppressed signs of spasticity in the lower extremities. This suppression was clinically significant and appeared to continue to improve over time.

Some of the studies included in this review, however, utilized research methodology that was relatively weak for several reasons. Only half of the fourteen studies provided data that had been subjected to statistical analysis to calculate the probability of chance findings. 75% of the research studies produced only Level IV and V evidence with half of all studies being Level 5. Only two studies furnished Level 1 evidence.[7]

Roscigno[29] did a study that followed 11 children and adults with age ranging from 8 to 55 years old and who suffered from spasticity due to cerebral trauma. The efficacy of ITB in reducing spasticity and improving function was the focus. The participants were evaluated using the Ashworth Scale, following either a bolus (one-time injection) or continuous infusion. The researchers concluded that bolus injections were dose-dependent and decreased spasticity significantly in both upper and lower extremities, but especially in upper extremities when compared to placebo group. The results of the infusion showed a smaller decrease in spasticity when compared with bolus, but after 12 months, mean Ashworth Scale scores were decreased as compared to baseline. Less of an effect was seen in upper extremities during continuous infusion compared to bolus injections. The participants also reported less pain while receiving treatment.

Awwaad et al.[1] described the outcomes for a case series of 29 patients with CP who received ITB and individualized rehabilitation programs. Outcome measures included use of the Ashworth Scale to assess impairment and the Pediatric Evaluation of Disability Inventory to assess dimensions of functional limitations and disability. Results indicated that all areas of functional skills and caregiver assistance improved; especially self-care and social function. In terms of caregiver assistance, the greatest improvement seen was less reliance on caregivers for transfers and ambulation. A relationship was found between the age and dose of intrathecal baclofen, with older patients receiving higher doses. All participants seemed to have potential to make functional gains and reduce spasticity. This study stressed the importance of allowing time

for improvements to occur and the need for a comprehensive rehabilitation program to allow optimal results.

Overall, several studies thus far show that ITB is often effective in reducing spasticity and associated pain in both children and adults with CP. The invasive nature of inserting a baclofen pump, the expense, and the expertise needed to evaluate and maintain the pump are issues that may not make this a suitable choice for everyone. ITB has been shown to have some side effects including catheter migration, infection, somnolence (extreme lethargy), hypotonia and subjective feelings of sensory or motor loss. Headache, nausea, and vomiting also occurred in some cases. In addition, the influence of ITB on seizure activity has been of concern.[7] Catheter-related problems as well as recurrent cerebrospinal fluid leaks require surgical intervention.[15] The cost of ITB was found to be four times higher than another procedure called selective posterior rhizotomy, to be discussed shortly. ITB also has ongoing costs including percutaneous refilling of the pump reservoir every 2 to 3 months and surgical replacement of the pump every few years.[7]

The body of evidence about ITB for spasticity in children with CP is relatively limited. However, it is showing promising results in many studies. Treatment effect has yet to be firmly established. Medical complications are frequent, but most are manageable. Only a small number of people have been studied to date, so it is imperative that further and more rigorous investigations are conducted. It is also important that future studies look beyond spasticity measures in terms of outcome. Other factors to be explored include age or distribution of CP, ITB technology regarding dosage and level of insertion of catheter tip, effects of maturation, time, cost, and the invasive nature of the treatment. Systematic reports of complications need to be provided to determine rates of adverse effects and to run a risk/benefit analysis of ITB, and there needs to be a clear picture of the surgery, drug, and system effects experienced with this

therapy. Future studies also need strict inclusion and exclusion criteria to promote homogeneous groups of patients for analysis, which are often difficult to find in studies such as this. ITB has definitely shown promising results in reducing spasticity and pain in children with CP and is currently becoming the treatment of choice for many of these children.

3.2 Botulinum Toxin Injections

Botulinum toxin type A, commonly called Botox (BT-A), offers a targeted form of therapy to reduce spasticity in specific muscle groups. It was introduced as a treatment for spasticity in children with CP in the early 1990s, with the first report confirming the efficacy and safety of BT-A in spasticity published in 1989.[12] In its unprocessed form, Botulinum toxin is the most potent neurotoxin known and is produced by the gram negative anaerobic bacterium *Clostridium botulinum*.[12] However, botulinum toxin in a purified and injectable form can be used safely to control conditions associated with involuntary muscle contractions. BT-A is currently being tested in children with CP and is the only locally acting antispasmodic.[29] It has been shown to both reduce spasticity and decrease pain associated with spasticity in children.

More specifically, Botulinum toxin is a protein polypeptide chain that irreversibly binds to the cholinergic terminal in the neuromuscular junction and inhibits the release of the acetylcholine necessary for muscle contraction.[15] This blockade is reversed only after several months when nerves have generated new neuromuscular junctions by terminal sprouting.[10] Because this agent has a natural affinity for the neuromuscular junction, it must be given intramuscularly.[15] The injection is given with a very small needle. In simpler terms, when BT-A is injected at the site of spastic muscles, it binds to nerve endings at the point where the nerves join the muscles. This temporarily prevents the nerves from signaling the muscles to contract, which results in muscle relaxation. BT-A often reverses the failure of longitudinal muscle growth by causing tone reduction in spastic muscles. This was first seen in animal model work. For

children with CP, this means the potential to correct motor imbalance, improve functional position and gait, and delay or obviate the need for surgery.[12]

Success with BT-A injections occurs in 70-82% of children with spastic CP.[20] The main advantages of this intervention are that injections provide localized, short-term results with relatively minimal side-effects. However, the cost of BT-A is high, at greater than $300 per 100 units, using 8-10 units per kilogram.[15] It is now marketed under the trade name Botox and can be prescribed at a physician's discretion without specific FDA approval for many therapeutic and cosmetic purposes.[34] Average duration of the effect is two to three months, but the procedure is usually repeated multiple times.[15] With repeated injections, antibodies to botulinum toxin may develop and cause the injection to become ineffective. This phenomenon is called immunoresistance. In general, with ongoing injections most children are able to maintain gains they made after the beginning of treatment. In one study involving 148 children with CP, over 80% of the children tracked beyond two years of multiple injections continued to demonstrate improvement over pre-treatment symptoms.[34]

The majority of individuals injected with BT-A continue to show responsiveness to repeated treatments, however some do not initially respond (primary non-responders) and others respond initially but fail to respond with repeated injections (secondary non-responders). Injections are generally continued until clinical response becomes insignificant, at which time other methods of treatment are explored. Also, a common cause for secondary unresponsiveness is starting treatment in older children, resulting in development of fixed contractures.[17] Factors that may affect the risk of developing resistance include dosage used and the time interval between injections. Higher doses seem more likely to cause antibody production as do shorter intervals between doses. Therefore, it is generally recommended to use the lowest dose that causes the desired effect and to use longer dosage intervals.[12]

There are seven serotypes (A through G) of botulinum toxin, but only one immunotype is available commercially (as of 1997). This is botulinum toxin type A. If more types are introduced, it may be possible that development of antibodies will be much less of an issue because types could be rotated for different injections.[12,15,36] Other serotypes may have a large part to play in the future, especially in those who fail to respond to BT-A. Studies show that type B is also effective, safe, and well tolerated with mild and transient side effects. It seems to be effective in both A responders and A non-responders. Antibodies to type A do not cross-react with type B.[12] There is also evidence that the efficacy of botox injections could be increased by injecting the toxin nearer to the motor end plates.[15]

BT-A is one of the top pharmacological agents at present due to its reversible effect, low adverse effect rate, and painlessness. It is one of the top agents used to improve lower limb spasticity and gait impairment in CP.[14] Most researchers suggest early treatment, preferably before age 6, in effort to avoid development of fixed contractures.[12] This medication is only suitable for children who can tolerate the invasive and somewhat painful nature of delivery. It also requires access to skilled care and may be quite expensive. Side effects can be either local or systemic. The most common local adverse side effect is weakness, which is generally minimal and transient. It occurs because of local diffusion of the toxin. Systemic adverse affects are rare, and include transient flu-like symptoms, excessive fatigue, anaphylaxis (systemic immune response), and generalized muscle weakness.[12]

Koman et al.[22] conducted a randomized, double-blind, placebo-controlled trial (RCT) of Botox in 114 children between 2 and 16 years of age to determine the efficacy of Botox in the treatment of dynamic equinus foot deformity due to spastic CP. This deformity is characterized by walking on the toes due to a shortening of the calf muscles. Results showed a significant difference in the treatment group when compared to the placebo group at four and twelve weeks.

However, there was no statistically significant difference in passive range of motion. Certain adverse effects were also reported by the treatment group, including weakness at injection site, pain at injection site, and falling. No participants left the study due to adverse effects. A similar double blind placebo controlled trial tested 12 children with dynamic equinus deformities. Results showed significant improvements in muscle tone and motor performance in the treatment group after injections into the gastrocnemius muscle. Also, no side effects were reported. This is often the case in studies with Botox.[12]

Another RCT conducted by Ubhi et al.[36] involved 40 patients with spastic diplegia or hemiplegia examined whether BT-A can improve walking in children with CP. 22 received Botox and 18 received placebo. Video gait analysis showed clinically and statistically significant improvement in initial foot contact following BT-A at 6 weeks and 12 weeks compared to placebo. The Gross Motor Function Measure (walking dimension) showed a statistically significant improvement in favor of the BT-A treated group. This was the first, large, well controlled, randomized study that showed an improvement in walking pattern in children with CP using BT-A targeted at the calf muscles.

Other studies describe a reduction in pain in addition to a reduction in tone after BT-A injections. Barwood et al.[5] studied the effect of Botox for the relief of postoperative spasticity-related pain following adductor-release surgery for children and adults who had both acute and chronic spasticity. They injected half of the participants with Botox and half with normal saline into a tendon of the hips 5 to10 days before the surgery. The treatment group had lower pain scores over their entire hospitalization.

Another study by Wong[40] involving 17 children with spastic CP between 2 and 14 years old resulted in improved range of motion in hip and ankle joints after injection of Botox into one of six sites including the left/right adductor, left/right gastrocnemius, and left/right

hamstring. Yang et al.[41] designed a study involving 38 children between age 3 and 10 with spastic CP. They reported significant improvement of spasticity as measured by the Modified Ashworth Scale at 12 weeks in comparison to a placebo group.

In a case study carried out by Gill et al.[20] of a 2-year-old patient treated with Botox injections and a comprehensive PT program, improvement was reported in almost every manifestation of the child's spasticity. His range of motion increased, reflex hyperreactivity decreased, and muscle tone decreased. The child had the opportunity to strengthen his opposing muscle groups following the Botox injections. Ambulation distance greatly increased and there were no side effects.

BT-A injections are a widely used and accepted practice in managing lower-limb spasticity, but are a relatively recent addition to the management of upper limbs.[39] A randomized double blind placebo controlled trial studied the effects of BT-A injections in the upper limbs. Significant increases in maximum elbow and thumb extension and significant decreases in wrist and elbow tone were obtained. There was also a noted functional change. A cosmetic benefit resulted as well, with reduction in involuntary elbow flexion.[12]

A study by Detrembleur et al.[14] was designed to determine whether the efficacy of BT-A may be enhanced by electrical stimulation. Twelve children with dynamic foot equinus deformity (walking on toes due to a shortening of the calf muscles) were randomly assigned to two groups in a blinded, clinically controlled trial. Group A received electrical stimulation following intramuscular BT-A into calf muscles. Group B did not receive electrical stimulation. Results showed that the combined treatment was not superior to BT-A alone. Improvement in the clinical and gait variables occurred at 1 and 3 months after BT-A injection for all participants in this study.

Suputtitada[33] tested the efficacy of a low dose of botulinum toxin A in 10 children between the ages of 2 and 5 with spastic CP. The results showed that even low dose injections into the legs combined with rehabilitation therapy improved Ashworth Scale scores and Physician Rating Scale scores. In comparing the use of BT-A injections with other forms of treatment such as casting, tone reduction in the BT-A group has generally allowed a more prolonged improvement.[12]

Due to limited experience, the impact of Botox injections on long-term functional outcome remains unclear. Overall, the use of BT-A has proven to be effective, but careful goal planning as well as objective assessment measures need to be utilized in further studies. The reversible yet long lasting-effects, ease of administration, and favorable safety and adverse effect profile are all factors that add to its usefulness. The optimal time to begin treatment as well as the potential for combinations of treatments calls for further research. In addition, response to treatment is difficult to predict and includes such factors as Ashworth scale scores, gross motor function measurement scores, and ambulatory status. This treatment method offers an alternative to surgical intervention for children with CP. When combined with a physical therapy regimen, BT-A injections definitely have the potential to lead to permanent improvement.

3.3 Selective Dorsal Rhizotomy

Selective dorsal rhizotomy (SDR) is frequently performed on children with severe spasticity with the goal of improving daily comfort and care. SDR is the most common surgical procedure for management of spasticity.[29] This procedure is widely practiced in the U.S., but long-term results are raising doubts about sustained benefit. SDR is a neurosurgical method used for reducing spasticity by sectioning a portion of the sensory nerve roots, which are typically restricted to the lumbosacral plexus.[15] These are nerves that innervate the lower extremities. This procedure uses electromyographic readings (EMG), essentially electrical stimulation of the nerve

rootlets, to help identify which subdivisions of the nerve roots are carrying abnormal information back to the alpha motor neurons.[21] Only those nerve rootlets that are identified are sectioned. The dorsal rootlets serve the function of carrying the sensory nerve fibers from the body to the central nervous system. After this surgery, the patient's spasticity level usually decreases, but there is also a decrease in sensory perception. The reduction in spasticity that results often allows for the development of more normal motor patterns in children with CP.

The rationale for performing SDR is based once again on the assumption that spasticity is the underlying cause of disordered movement and that reducing this spasticity will improve movement. Physical therapists also need to direct their efforts into developing adequate assessment, treatment and measurement techniques for assessing motor control in children with CP due to factors other than spasticity. Oftentimes, the underlying weakness and resulting hypotonia after surgery are related to poor functional outcome.[21] This serves as evidence that careful screening is needed to identify patients who have underlying strength or weakness. Some researchers have raised the argument that improved motor function after SDR is more related to the intense physical therapy given after the procedure than to SDR itself. RCTs have yielded conflicting conclusions regarding this argument.[15]

Following SDR, six months to one year of intense rehabilitation is required for motor retraining and addressing the loss of muscle strength that many children experience.[15] An ideal candidate for this procedure is an ambulatory or near-ambulatory preschool-age child with spastic diplegia whose resting muscle tone interferes with quality of gait. SDR may also benefit children with spastic quadriplegia whose spasticity interferes with care or positioning, such as seating. Older children with fixed contractures are less likely to benefit.[15]

Improvement of lower limb range of motion has been the most promising outcome of this surgery.[29] However, there are risks that come along with SDR. Back pain, leakage of

cerebrospinal fluid, incontinence, lower extremity sensory loss, and weakness are reported as side effects of this procedure.[15] There is also a risk of spinal deformity. In a study of 43 participants, it was reported that 36% of participants suffered from spinal deformity and 6% of these required stabilization for their deformities.[29] Due to the seriousness of side effects, this procedure must be used only after careful consideration.

Several studies have reported improved function after SDR, however, many of these studies present anecdotal evidence for improvement. The results do suggest that SDR decreases spasticity and improves movement ability when paired with intensive physical therapy after surgery. Indeed, postoperative PT plays an important role in strengthening, improving function, and developing more normal movement patterns.[3] It has generally been found that decreased spasticity as a result of this surgery cannot lead to improvements without physical therapy.[21]

There has been conflicting evidence regarding the effectiveness of SDR. A randomized, controlled trial of 24 ambulatory children with spastic diplegic CP compared SDR and therapy to therapy alone. The SDR group had greater improvements in gross motor function than the therapy group after one year.[3] A comparative analysis and meta-analysis of three published randomized clinical trials again examined the effect of SDR in treatment of children with spastic diplegic CP. Two studies showed improved function, but one showed no improvement in outcome compared with intensive PT alone.[25]

Several researchers have observed that lower limb strength generally decreases initially after SDR and then gradually increases, resulting in improved function. Fasano et al.[16] conducted a 2 to 7 year follow-up of 80 children who had received SDR. They reported that spasticity returned in only 5% of the cases. A comparative analysis and meta-analysis was done by McLaughlin et al.[25] of three published RCTs examining the effectiveness of SDR in the

treatment of children with spastic diplegic CP. These trials showed a direct relationship between the percentage of dorsal roots transected and functional improvement.

Overall, the effects of SDR documented in many studies supply evidence that SDR reduces spasticity and increases joint range of motion immediately after surgery. Improvements in strength and ability to control and coordinate movement patterns are gained slowly over a long period of time with therapy. The functional potential of SDR may be maximized through various types of exercise including strengthening and practice. For example, one study recently reported that upper extremity strengthening exercises such as pushups for children with CP did not increase spasticity and appeared to improve function.[21] In future studies, researchers need to consider the effects of maturation or intensive therapy alone in looking at the efficacy of SDR.

3.4 Threshold Electrical Stimulation

The use of electrical stimulation for the treatment of children with CP remains controversial. A lack of clearly defined terms and treatment protocols contribute to this uncertainty. Threshold electrical stimulation (TES) is one of the more commonly used types of electrical stimulation for children with CP and is based on low-intensity, long duration, electrical, transcutaneous stimulation using surface electrodes.[11] The electrical stimulation system is composed of three elements including the stimulator unit, electrodes, and connecting wires.[31] It is claimed that low-intensity electrical stimulation increases local blood flow. This, along with the secretion of trophic hormones, may enlarge atrophic muscles which will improve motor function.[11]

There are several reasons that therapists apply electrical stimulation to children with CP. Main goals include maintaining or improving range of motion, facilitating voluntary muscle control, and reducing spasticity. It is not an easy task to interpret the available research on electrical stimulation. A majority of the literature focuses on adults with orthopedic conditions.

Some of the studies show improvement in impairment after electrical stimulation treatment but no improvement in function or changes in disability. For example, range of motion and muscle strength may have improved but there were no significant improvements in gait.[3] Considering these types of outcomes, it is important to remember that improvements in impairment do not always lead to improvements in function.

Dali et al.[11] conducted an RCT that included 57 ambulatory children with CP and examined the effects of TES on motor function, range of motion, spasticity and muscle growth. In this study, TES was applied over a 12-month period. Two-thirds of the children received active stimulators, while one-third received sham devices. Children slept with TES for at least six hours per night, six nights a week. Motor progress was measured by quantitative tests. Range of motion, reflexes, clonus (rapid, rhythmic movments) and degree of spasticity were recorded. Some of the children also underwent Computerized Tomography to determine if the treated leg muscles were enlarged compared to the placebo group. Results stated that there was no significant difference between active and placebo treatment as a whole or in any of the tested subgroups. It was concluded that TES in these patients had no significant clinical effect during the test period.

Pape and Kirsch[27] conducted a 12-month trial of 74 patients with CP. Electrical stimulation was applied nightly to the quadriceps femoris and tibialis anterior muscles of alternating legs. The authors reported improved strength and reduced spasticity, however they did not actually measure these variables in either study. Also, the authors presented no reliable or valid data on the outcome measures, one of which was designed specifically for this study. These factors leave room for many questions regarding the reported results of this study. Much researcher bias may have come into play and there were several methodological problems with their research.[3]

A recent RCT by Cho[9] looked at the effects of electrical stimulation applied to the abdomen and posterior back muscles in young children while sitting. Significantly higher positive changes in the Gross Motor Function Measure and sitting angle were recorded in the treatment group as opposed to the control group after 6 weeks. In another study, electrical stimulation of the gluteus maximus muscle of children with CP did not result in any significant improvement in muscle strength, gait characteristics or motor function when compared to the control group.

Some studies involving TES measure outcomes mainly by the subjective observations of caregivers. In a study conducted by van der Linden et al.[38], 7 of the 11 parents thought that the treatment made a difference to their child, but there was no statistically or clinically significant improvement in the TES group when compared to the control.

Regarding combined treatment, an RCT by Steinbok et al.[32] of 44 children with spastic CP concluded that TES may be beneficial for children after undergoing selective posterior rhizotomy more than one year earlier. This study resulted in increased strength, decreased spasticity and improved motor function in the treatment group.

Several reports have been made regarding electrical stimulation, and they have established a good base for future research studies into this treatment option. However, evidence for the efficacy of electrical stimulation is inconclusive. Many recent studies have failed to find a significant effect in motor function with low-intensity electrical stimulation for children with CP. Studies of homogeneous groups of subjects with high statistical power would provide much better evidence for the effectiveness of this therapy.

Further studies should explore the effectiveness of TES in ambulatory versus non-ambulatory children with spastic CP, as well as investigate the mechanism involved as to how TES may affect motor function. If this treatment is shown to be beneficial for children with CP,

it does have many positive aspects. It is simple to perform, non-invasive, generally well-tolerated by children and their caregivers, and there are no known negative side effects or significant complications involved with this therapy. Other factors that may be important in selecting patients for TES include age and initial muscle strength. A few studies have reported that younger children responded more rapidly to electrical stimulation.[38] Future studies may determine if another more functional way of applying electrical stimulation would lead to better results in motor function improvements for children with CP.

3.5 Pediatric Constraint-Induced Movement Therapy

Constraint-induced movement therapy was designed to improve upper-extremity function in patients with hemiplegia secondary to neurological injuries. This therapy was originally developed for adults over the past sixteen years, based on extensive research with nonhuman primates.[13] In humans, learned nonuse is hypothesized to occur as a result of conditioned suppression of movement. This occurs following an injury, when a patient tries to use the affected extremity but cannot. Repeated failure during the acute phase of a neurological injury leads to learned nonuse, which leads to continued nonuse in the chronic phase of a patient's recovery. Such early disuse may be reversed by constrained inhibition of the less affected upper extremity, forcing use on the less functional limb.[23]

Some studies have recently begun to address the use of this type of therapy in children. In theory, a child with a movement disorder such as CP may not develop neural pathways involved in movement because of the lack of ability to experience age-appropriate sensorimotor stimuli that lead to the development of upper extremity (UE) skills. Charles et al.[8] reported success with three children with hemiplegia after combining intensive training of the more effected UE with restraint of the less affected UE. These children wore a cotton sling on the less affected UE while an investigator played with them for six hours each day for fourteen days in a row. Results

included positive changes in functions such as limb coordination, manual dexterity and sensory discrimination.

A case study of a one-year-old girl with CP by DeLuca et al.[13] applied Pediatric CI Therapy by using a full-arm, bivalved cast on the less affected UE throughout a three-week intervention. Before intervention, she performed all movement tasks using her less affected left UE. She did not use her affected, right UE for any activity. The first intervention involved putting a lightweight, fiberglass cast on the less affected UE from the shoulder to the fingertips. The cast was bivalved so it could be removed once a week to wash the arm, permit active range of motion and check skin integrity. Treatment involved a six hour intervention involving play-based functional activities promoting use of the affected UE provided by a therapist each day for three weeks. Behavioral techniques were also used, and included the use of rewards such as verbal praise, smiles, hugs, cheers, clapping.

When the cast was removed, the child continued to use her more affected (right) UE for reaching and object manipulation. A problem arose in which the child was more willing to use the affected UE with the therapist than with her parents. Addressing this issue involved the therapist teaching the parents how to facilitate the child's new learned skills with her affected UE.

The second intervention took place five months after the first and once again lasted for three weeks, with therapy for six hours per day. At the start of this intervention, the child had maintained her skills, but the frequency of use of the affected UE had declined somewhat. The goal this time was to improve self-help skills and independent initiation in use of the affected UE. After the treatment period, the child used her affected arm more easily and more frequently. Her parents were pleased with the intensity of the therapy, the fact that it was done at home, and the focus on functional activities needed in daily life. She used her affected UE for 100% of the

free choice-trials immediately after the intervention. This was an increase from the first intervention, after which she used it 50% of the time. This decreased somewhat over time, but she continued to use the affected UE much more than before the treatments.

This case report gives evidence that multiple episodes of pediatric CI therapy may be an effective intervention for children with hemiplegic CP. There were major gains in motor abilities, including independent sitting, crawling, use of a walker, bilateral arm and hand use, and use of hands and fingers to play. Other factors may have come into play as well. She had a strong positive relationship with the therapist, therapy was done in the home, therapy was high intensity, her parents and other family members actively promoted maintenance of everyday use of UE skills, and normal maturation occurred. In future studies, these factors will need to be more closely examined. Overall, the constraint of the less affected UE facilitated the child's focus on using the more affected UE, giving a very positive outcome.[13]

The two interventions that have been found to be successful thus far in producing permanent functional use of the limb are restraint of the favored limb for a period longer that seven days and shaping of the affected limb for many consecutive days. Impressive short-term results have been seen, but more evidence is needed. This therapy is now being subjected to a more rigorous evaluation using RCTs designed to include one intervention episode for children with hemplegic CP. This will help to better evaluate the therapy method and understand how all of the components of the intervention and other factors come into play. This therapy needs to be examined with a larger number of children, considering that studies thus far have dealt with relatively few children. It also needs to be determined if such high intensity PT or multiple intervention episodes are needed in order to reap the benefits of this intervention. Cost-benefit analysis should also be a part of future research efforts. Short bursts of intensive therapy in clinical settings would be more cost beneficial. The major issue to be explored in pediatric

constraint-induced movement therapy, however, is the level of long-term functional benefits for the children treated.

4. Conclusions

Evidence-based practice is becoming the norm in Physical Therapy. This is a positive step that demonstrates that the core of the practice should be the scientific basis of the interventions. Physical therapists integrate this scientific knowledge with the art of their practice to provide the best care for children with CP. A larger amount of literature has become available along with several new tests and measures to assess outcomes. New evidence supporting numerous intervention strategies in pediatric physical therapy for children with CP is being gathered at a rapid pace.

Neurosurgical interventions including intrathecal baclofen therapy and selective dorsal rhiztomy are proving effective in treating muscle spasticity related to many types of cerebral palsy. In addition, botulinum toxin injections are often a good choice when looking for a more specific, short-term, targeted form of therapy to reduce spasticity in certain muscle groups. Studies involving other interventions, such as threshold electrical stimulation, show inconclusive evidence regarding efficacy in the treatment of childhood CP. More investigation into this type of treatment is needed. Finally, a very new method, called pediatric constraint-induced movement therapy, is showing promising results in the treatment of the affected upper limb in children with hemiplegic CP.

Research on efficacy of treatment is needed for third-party payers (managed care), patients, families and the medical community. The role of spasticity in limiting motor function is a topic of great debate. Effects of most physical therapy interventions on patients with movement disorders besides spasticity have not been studies and need to be examined. More research into the long-term effects of intrathecal baclofen, botulinum toxin, electrical stimulation and selective dorsal rhizotomy in terms of function, disability and cost-effectiveness is needed. Improvement in terms of impairment is important, but of more immediate concern are the actual improvements

in functional limitation and disability. Quality of life should be the main consideration for each child. In addition, attention to the motor problems of this motor disorder should not cover up the need for a holistic approach to the child's emotional, social, cognitive, visual, auditory, and educational needs.

Independent mobility should be the goal of caregivers and physical therapists. This may not necessarily mean walking, but may involve a wheelchair or other assistive devices. For children with severe CP, ease of care, comfort, and prevention of deformity should be important goals. Therapy should prepare a child for independent adult life to the greatest extent possible.

Further studies should include children of differing age ranges to determine if there are developmental factors that influence treatment efficacy. Individual responses to treatment may vary, as shown by many of the studies discussed. It is important to constantly assess response of a child to a treatment and design the treatment accordingly. In these types of studies, it is often difficult to recruit suitable children who fulfill the inclusion criteria or can commit to a long period of time.

The Americans with Disabilities Act of 1990 ensures the civil rights of persons with disability. The Education for All Handicapped Act in 1975 requires that states provide free and appropriate educational services, including therapies, for all students 3-21 years of age. In 1986, the act was extended to include children from birth to 2 years of age.[3] Physical therapists play an essential role in providing interventions with the goal of addressing the rights of those with disabilities. They do this by working to minimize impairments, optimize function and reduce disability.[3] The challenge remains to determine the effectiveness of interventions for children with CP and pinpoint optimal treatment. Lack of universal diagnostic criteria, standard definitions, and well-defined interventions are obstacles that still remain to the treatment of childhood cerebral palsy.

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