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Three Dimensional Gait Analysis Following the Adeli Treatment for Cerebral Palsy: A Case Report

Troy Lase
Grand Valley State University

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THREE DIMENSIONAL GAIT ANALYSIS FOLLOWING THE ADELI TREATMENT FOR CEREBRAL PALSY: A CASE REPORT

Master's Thesis

By

Troy Lase

Submitted to the School of Health Professions at Grand Valley State University, Allendale, Michigan in partial fulfillment of the requirements for the degree of

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THREE DIMENSIONAL GAIT ANALYSIS
FOLLOWING THE ADELI TREATMENT
FOR CEREBRAL PALSY:
A CASE REPORT
ABSTRACT

This case report describes the use of three-dimensional gait analysis to identify kinematic changes following the Adeli suit treatment. The suit's original design was to decrease the effects of weightlessness in space but has been modified to become the primary modality for the Adeli treatment used to treat cerebral palsy. Data were quantitatively produced by three-dimensional gait analysis before and after treatment were used to identify any changes in gait. Following the Adeli treatment, instrumented gait analysis showed that no clinically significant changes in four critical events of gait following the Adeli treatment occurred.
ACKNOWLEDGEMENTS

I would like to personally thank all of my committee members for allowing me the chance to finish this thesis and also the PT program at GVSU. The last couple years of my life have been to say the least very challenging. I am so very grateful for your help in assisting me to complete my dream. I also would like to thank all my friends and family especially Dan and Laura McCloud, Julie Parker, Steve Leppard, and Craig Vissers for all of their support through this process. A special thanks to my best friend Bret whose guidance, strength and support has helped me in ways that he cannot imagine. May God’s light shine favorable on each and everyone.
DEFINITION OF TERMS

**Afferent Nerves**: Nerve fibers that transmit impulses from the periphery toward the central nervous system.

**Agnosia**: An inability to recognize sensory inputs (sight, sound, touch). The most common agnosia is a result of brain injury damaging the posterior aspect of the brain causing visual agnosia (inability to properly recognize objects by sight).

**Anoxia**: An abnormal condition characterized by a lack of oxygen as a result from an inadequate supply of oxygen in the blood to carry oxygen to the tissue or for the tissue to absorb.

**Apgar Scale**: The evaluation of an infant's physical condition at birth performed at one and five minutes after birth and based on a rating of five factors that reflect the infant's ability to adjust to extrauterine life.

**Apraxia**: A movement disorder due to brain damage that results in an inability to carry out preplanned voluntary sequences of movement.

**Ataxia**: A disturbance in the rate, range force and duration of movement.

**Athetosis**: A neuromuscular condition characterized by slow, writhing, and involuntary movement of the extremities as seen in forms of cerebral palsy and in motor disorders resulting from lesions in the basal ganglia.

**Basal Ganglia**: A collection of nuclei at the base of the cerebral cortex which includes the caudate nucleus, the putamen, the globus pallidus and functionally include the substantia nigra and subthalamic nucleaus.

**Central Nervous System (CNS)**: The nervous system consisting of the brain, spinal cord and processes information to and from the peripheral nervous system and in the main neocortex for coordination and control for the entire body.

**Central Pattern Generator (CPG)**: A network of neurons contained within the spinal cord identified in non-human vertebrates as being a center for generated locomotion without initiation of upper cerebrum input.

**Cerebral Cortex**: A layer of neurons on the surface of the cerebral hemispheres which integrates higher mental function general movement, visceral functions, perception and behavioral reactions.

**Cerebellum**: The posterior portion of the brain in the posterior aspect brain between the cerebrum and the brain stem, that is responsible for coordinated voluntary muscular activity.
Cerebral Vascular Accident (CVA): The sudden death of some brain cells due to lack of oxygen when the blood flow to the brain is impaired by blockage or rupture of an artery to the brain. A CVA is also referred to as a stroke.

Cerebral Palsy (CP): A syndrome of weakness, spasticity, poor coordination of the limbs and other muscles, impaired sensory perception, and sometimes impaired intelligence. The cause of cerebral palsy is not always known, although many cases are linked with lack of oxygen during birth.

Chorioamnionitis: An inflammatory reaction in the amniotic membranes caused by bacteria or viruses in the amniotic fluid.

Chorea: Ceaseless rapid complex body movements that look well coordinated and purposeful but are, in fact, involuntary and non-purposeful movements.

Dysdiadokinesia: An inability to perform rapid alternating movements such as rhythmically tapping the fingers on the knee.

Dysmetria: An inability to gauge distances accurately combined with the inability to control a muscular movement at the desired point.

Dystonia: Involuntary movements and prolonged muscle contraction, resulting in twisting body motions, tremor, and abnormal posture. These movements may involve the entire body, or only an isolated area. Some types of dystonia respond to dopamine, or can be controlled with sedative-type medications, or surgery.

Efferent Nerves: Nerve that transmits impulse away from the central nervous system usually causing a muscle contraction or release of glandular secretion.

Electromyography (EMG): A reading or tracing of the electrical signal elicited by a muscle during contraction.

Extrapyramidal System: The part of the nervous system that include the basal nuclei, substantia nigra, subthalamus parts of the midbrain and the motor neurons of the spine.

Extremely low birth weight baby (ELBW): A baby born very prematurely weighing between 401 and 1000 grams at birth. Extremely low birth weight (ELBW) babies are at the lower limits of viability.

Festinating: A manner of walking in which a person's speed increases in an unconscious effort with a displaced center of gravity and base of support.
**Floppy Baby Syndrome:** A general medical reference to an abnormal condition of newborns and infants manifested by inadequate tone of the muscles. It can be due to a multitude of different neuralgic and muscle problems.

**Hypotonia:** Decreased tone of skeletal muscles. In a word, floppiness. Hypotonia is a common finding in cerebral palsy and other neuromuscular disorders.

**Hypertonia:** Increased resting muscle tone. Untreated hypertonia can lead to loss of function and deformity. Treatment is by physical and/or occupational therapy, and in some cases muscle relaxant medication.

**Hyperbaric Medicine:** Hyperbaric oxygenation is an increased amount of oxygen in organs and tissues resulting from the administration of oxygen in a compression chamber at an ambient pressure greater than 1 atmosphere.

**Incidence Rate:** The frequency with which something, such as a disease or trait, appears in a particular population or area.

**Intention Tremors:** Fine rhythmic purposeless movements that tend to increase during voluntary movements.

**Low birth Weight Infant (LBW):** A baby born prematurely weighing between 2500 and 1501 grams at birth. Low birth weight (LBW) babies are at the upper limits of viability.

**Lower Motor Neuron:** A neuron originating in the motor nuclei of the spinal cord or brainstem and providing innervations to motor units of skeletal muscle.

**Motor Control:** The ability of the CNS to regulate and/or direct the musculoskeletal system in purposeful acts.

**Muscle Spindles:** A sensory receptor found in muscle fibers that are sensitive to changes in muscle length and rate of change during movement and stretch.

**Multiple Sclerosis (MS):** A progressive neurological disorder characterized by the demyelination of the white matter sometimes extending into the gray matter. Impaired bodily functions or altered sensations associated with those demyelinated nerve fibers give rise to the symptoms of MS.

**Neocortex:** The most recent evolved part of the brain, which includes all of the cerebral cortex except for the hippocampal and piriform areas.

**Nuchal Cord:** An abnormal but common condition in which the umbilical cord is wrapped around the neck of the fetus in utero.
Periventricular Leukomalacia (PVL): Necrosis of the periventricular white matter believed to inhibit myelination and develop cysts in preterm infants therefore leading to cerebral palsy.

Perinatal: The time frame pertaining to the time or process of giving birth.

Prenatal: The time frame before the birth of the child.

Postnatal: The time frame following the birth of the child.

Proprioception: The sensations of body movements and awareness of posture, enabling the body to orient itself in space without visual clues.

Pyramidal System: The part of the primary motor system consisting of the corticospinal tracts and the corticobulbar tracts of the spinal cord.

Spasticity: A motor disorder characterized by a velocity dependent increase in tonic reflexes with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex. Spasticity is a component of the upper motor neuron syndrome.

Thrombophilia: The tendency to form blood clots (thrombosis) which can break off, travel through the bloodstream and lodge in any artery of the body (thromboembolism) and impair the normal circulation within these tissues.

Toxemia: Condition in pregnancy, also known as pre-eclampsia (or preeclampsia) characterized by abrupt hypertension (a sharp rise in blood pressure), albuminuria (leakage of large amounts of the protein albumin into the urine) and edema (swelling) of the hands, feet, and face.

Upper Motor Neuron: A neuron originating in a motor area of the cerebrum and descending to influence peripheral motor neurons or cranial nerve nuclei contained within the spinal cord.

Very Low Birth Weight Infant (VLBW): A baby born prematurely weighing between 1500 and 1000 grams at birth. Very low birth weight (VLBW) babies are at the middle limits of viability.
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CHAPTER 1

INTRODUCTION

Background To Problem

As the field of health care enters the next millennium, physical therapy must continue to keep pace with current trends in healthcare. Among these trends is the implementation of prospective payment systems and managed healthcare, which have bolstered the drive for both efficiency and efficacy in patient care. As a result, an area in which physical therapy services will be challenged to adapt is the manner in which treatment protocols are implemented into the rehabilitation plans of the patient.

This implementation will especially be important in regards to the long-term treatment requirements of patients diagnosed with neurological disorders such as stroke, traumatic brain injury, multiple sclerosis and cerebral palsy. More specifically, treatment protocols must improve in children with cerebral palsy (CP) due to extensive long term treatment requirements and lack of functional abilities.\(^1,2,3\)

CP is a non-progressive neurological disorder characterized by a static lesion to the motor cortex of an immature undeveloped brain.\(^4,5,7\) Depending on factors such as location and intensity of the lesion, sensation, motor control, speech and cognitive abilities may all be affected.\(^6,7,9\)

Due to the multiple areas of possible lesions within the central nervous system (CNS), the term CP is used as a medical classification with the specific diagnosis being based on the limb involvement and muscle tone.\(^6,10,11\) Likely
areas of lesion within the CNS are the cerebral cortex, cerebellum, and the basal ganglion.\textsuperscript{5,6} Some of the more common associated problems possibly causing lesions include low birth weight, premature birth, and anoxia at birth.\textsuperscript{5,10-12} Other causes are cerebral mal-development secondary to drug or alcohol abuse, infections, viruses, genetic syndromes and other teratogens.\textsuperscript{1,10,11,15} Risk factors include placental complications, toxemia, maternal bleeding and an Apgar score less then three at ten minutes after birth.\textsuperscript{1,11} Mental retardation is also likely to be seen in about sixty to sixty-five percent of the children with approximately thirty percent being seizure active.\textsuperscript{5,11,12}

An increased amount of spasticity, along with co-contractions of agonist and antagonist muscle groups, is one of the trademark characteristics of CP.\textsuperscript{5,15,16} Spasticity, is defined as a velocity dependent increase in tonic stretch reflexes resulting from a insult to the CNS.\textsuperscript{13,14,16,17} Spasticity when present in children with CP contributes to gait deviations such as crouched and scissors gait and also in toe walking.\textsuperscript{18,19} Upper extremity spasticity can contribute to an inability to reach, therefore the child has limited ability to perform activities of daily living.\textsuperscript{20}

Due to the broad classification and lack of specific diagnostic tools, the incidence of CP varies considerably. Researchers have estimated incidences of CP ranging from 1 to 4.7 cases per 1000 births.\textsuperscript{21,22} Slight increases in incidence rates have been identified in mothers not receiving adequate pre-natal care and also in underdeveloped countries where little or no pre-natal care is available.

Currently there is not a cure for individuals medically diagnosed with CP. While medical researchers work towards a cure, healthcare providers direct
treatment towards improvement of functional abilities such as ambulation, bathing, dressing and feeding. Once these areas of function improve, focus on treatment is then shifted to the sustainment of these abilities. Healthcare providers use a variety of techniques to attain and sustain such functional levels. One of the most common management techniques of cerebral palsy is through rehabilitation services consisting of physical and occupational therapy. Both disciplines focus on the inhibition of abnormal tone and improvement of postural control, as seen in children with cerebral palsy. Occupational therapy tends to focus on activities of daily living (ADLs) such as bathing and eating, while physical therapy focuses on ambulating, balance, postural control and functional training. Pharmaceutical interventions are commonly used to decrease spasticity within affected muscle groups, therefore allowing increased functional activity. Another treatment technique to manage spasticity is to administer a toxin which severely damages the specific afferent nerves which are causing the increase in spasticity. Surgery is another common management technique for CP. Selective dorsal root rhizotomy (SDR) is used to decrease the afferent input into the central nervous system in hopes of decreasing spasticity of the affected limbs. Adductor, gastrocnemius and hamstring releases are orthopedic surgical options for improvement of functional gait by increasing the overall length of the muscle from origin to insertion. Lengthening the muscle also decreases tension on the specific joint and decreases the likelihood of bone deformities. There are a variety of different treatments and therapeutic modalities which have demonstrated improvements in a variety of functional
activities such as orthotics, electrical stimulation, splinting, serial casting, hyperbaric medicine and conductive education.$^{30-36}$

The focus of this project is to investigate a new treatment for cerebral palsy called the Adeli treatment based on using what is known as the Adeli suit. Children receive the Adeli treatment combined with other types of physical therapy and modalities such as strength training, hot packs and acupressure at Euro-Med of Poland. The Adeli suit is an anti-gravitational suit used in the treatment of cerebral palsy. It was originally developed by Russian scientists to deter the effects of weightlessness in outer space. The suit is an external garment consisting of adjustable elastic bands that parallel gross flexor and extensor muscle groups of the trunk and lower extremities. It is proposed by the designers of the Adeli suit that the increased loads imposed by the elastic bands promote possible neuroplastic changes within the CNS that may lead to a decrease in spasticity. It is unclear if the decrease of spasticity caused by the Adeli treatment may then lead to improvements in motor control.

Currently, only one scientific research article is published in medical journal, which investigates the validity and efficacy of the Adeli treatment in relation to children with CP. This study reports marked increases in a variety of functional areas. Unfortunately, the study is questionable because of its poor design such as no control groups, randomization or proper data collection and reporting.$^{37}$ Parents who accompanied their children to Poland for the Adeli treatment have reported improved functional abilities around the home, such as increased ambulation and decreased assistance to perform ADL's. School teachers also
have reported improvements in speech cognitive abilities and increased endurance in children after undergoing the Adeli treatment. Dr. Edward Dabrowski, Medical Director of the Physical Medicine and Rehabilitation Center at the Children's Hospital of Michigan and Gretchen Backer, Director of Children's Hospital of Michigan's Motion Analysis Laboratory, have both traveled to Poland and witnessed the Adeli treatment of children directly under their care. Dabrowski and Backer have initiated a pilot study investigating changes in range of motion and spasticity in children who have participated in the Adeli treatment. The findings for their study have not been reported. The follow-up study to the pilot will also include initial investigations by sociology and psychology researchers from Wayne State University in relation to psychosocial dynamics of the Adeli treatment. Unfortunately, the dependent variables being investigated do not prove that the Adeli treatment provides changes in active motor control components such as gait, balance, and posture. The limited amount of objectively published data especially in active motor control warrants the necessity for this study.

Problem Statement

Undergoing the Adeli Treatment may be causing an increase in the functional ambulation of children with a medical diagnosis of spastic cerebral palsy. The improvement is possibly caused by a combination of interventions, which are proposed to inhibit spasticity and increase the amount of dynamic proprioceptive input into the central nervous system. Due to the plasticity of the CNS this increase in dynamic proprioceptive input may produce changes to the lesion area.
of the central nervous system and controlling motor function. The clinicians who
developed the Adeli treatment believed that by altering lesion areas, which
produce the characteristic symptoms seen in CP, an increase in inhibitory control
of motor response through descending spinal tracts may occur. This increase in
inhibitory control may lead to a decrease in spasticity and possible improvements
in functional ambulation. Unfortunately, research is lacking to support this belief.
Evidence is also nonexistent to support if the Adeli suit itself produces any
possible improvement of descending inhibition that leads to subsequent
improvement in gait or is the numerous hours of treatment combined with the
Adeli suit that lead to reported improvements. Will this improvement of
descending inhibition lead to improvements in the child’s functional ambulation?
If functional improvement in gait does occur after the Adeli treatment, one must
question if the entire Adeli treatment is producing the response or just single
components.

**Purpose / Aims:**

The purpose of this study is two-fold. First, is to provide clinical information
about the specific aspects of the Adeli Treatment including the design of the suit,
secondary modalities used during treatment, treatment plans and protocols, time
duration of each part of the treatment, hypotheses about the mechanisms of
treatment and cost. Second, is to examine changes in gait patterns following the
Adeli treatment using three-dimensional gait analysis in a child with CP.
Significance of the Problem

With the changing health care system today, there have been new demands imposed upon physical therapy. These demands have challenged physical therapists to find more effective / efficient ways to improve patient function. One of these areas would be in children with CP. Unfortunately, children with CP receive different ongoing forms of rehabilitation for most of their lives. The cost and time involved for these treatments are immense and sometimes overwhelming to the family. That is why clinicians are actively conducting research in new treatment techniques for children with CP. One of these new treatments is the Adeli treatment. The Adeli treatment is a relatively new treatment program designed specifically to improve ambulation and functional activities of daily living for children with CP. Unfortunately, the Adeli treatment has not been studied or evaluated thoroughly enough to provide clinicians with the necessary scientific data to make clinical decisions about the potential benefits of the treatment. Not only is the Adeli treatment time intensive for the staff members but also is also extremely expensive. Furthermore, clinicians are unable to provide any scientifically sound recommendations to families who seek information pertaining to the Adeli treatment. This study will provide clinicians with preliminary information to help determine the usefulness/effectiveness of the Adeli treatment. Unfortunately, this study is only a single case report. The true function of this case report will be to help establish the foundation for future research into the validity of the Adeli treatment. Also researchers may want to focus on just specific individual parts of the treatment that will provide the
greatest amount of improvement not only in ambulation but also in other functional activities.

**Purpose of Case Report**

The purpose of this case report is to identify any changes in the gait pattern following the Adeli treatment. Dr. Jacquelin Perry from Ranchos Los Amigo Medical Center and Dr. James Gage from Gillette Children's Hospital are highly regarded specialists in motor control and gait analysis. Perry and Gage have both identified four critical events of gait, which are required for a functional gait pattern. These four requirements of gait are: (1) stability of weight-bearing foot throughout stance phase, (2) clearance of the non-weight bearing foot during swing phase, (3) appropriate pre-positioning during terminal swing for the next gait cycle, and (4) adequate step length.\(^{38,39}\) Using data collected from three-dimensional gait analysis, the four previously mentioned critical events in gait identified by Perry and Gage will be used to evaluate changes in the child's gait pattern following the Adeli treatment.
CHAPTER 2

LITERATURE REVIEW

Introduction to Literature Review

The following literature review will focus on topics germane to this study of cerebral palsy (CP). The review will begin with related anatomy of the CNS, followed by the history of CP, the incidence rates, etiology, detection and diagnosis, and its medical classifications. Next, a survey of different medical management interventions currently being used for cerebral palsy will be reviewed, including rehabilitation programs, surgical procedures and pharmacological interventions. Finally, the literature review will conclude with a thorough explanation of the Adeli treatment to include all the secondary modalities and treatment applications involved. Any current research pertaining to the Adeli treatment program also will be examined.

Related Neuroanatomy

In a child suspected of CP the extent of symptoms presented is based on the area of the CNS affected and the level of involvement. To better understand how different lesions within the CNS may present clinically, a review of functional neurological structures responsible for voluntary and involuntary control of movement is essential. This review of functional neuroanatomy will be organized in a hierarchical fashion starting with the higher functional components including the Limbic System and Cerebral Cortex and progressing to the ascending and descending tracts contained within the spinal cord.
Limbic System and Hypothalamus

The primary responsibility of the limbic system and hypothalamus is to regulate and maintain homeostasis of the body. Arguably, this is the responsibility of the entire CNS but neurons and the control mechanisms vital to this function lie specifically within the limbic system and hypothalamus. The means by which these two structures control homeostasis is through maintaining central control of autonomic nervous system, hormonal release and continual development of emotional and motivational states. Even though structurally the hypothalamus is part of the diencephalon, the functional role in homeostasis of the body is the centerpiece of the limbic system and will be discussed in this section.

The limbic system consists of four components, which includes the cingulate, parahippocampal gyri, amygdala and hippocampus. Unfortunately, due to a lack of clear anatomical structure and understanding of their physiological function, researchers are argumentative on the exact components of the limbic systems. Researchers do agree that the primary function of the limbic system is to bridge voluntary and involuntary output responses in relation to changing internal and external input stimuli. Two primary subsystems provide functional output from the limbic system into the reticular formation of the midbrain. The first subsystem of the limbic system consists of the amygdala. The amygdala is a spherical mass of neurons subdivided into functional units called corticomedial amygdala and basolateral amygdala.
The corticomedial amygdala is interconnected with the olfactory bulb, olfactory cortex, the hypothalamus and the brain stem, allowing this subsystem to help regulate the autonomic nervous system and the pituitary function. The basolateral amygdala receives highly processed sensory information from the sensory association area of the frontal, temporal and occipital lobes. The basolateral also provided input to the corticomedial amygdala.

Functions of the amygdala remain in question but clinical and experimental research suggests two different possibilities. First clinical evidence suggests that the amygdala is an important link between emotional and motivation responses to external stimuli. Second, is the possibility the amygdala may influence both visceral and somatic components of behavior due to the connections between itself, the autonomic nervous system and the neuroendocrine system.

The second subsystem is centered on the hippocampus utilizing the cingulate complex and parahippocampal gyri for communication with the neocortex. The hippocampus has been identified as an important component of declarative learning and memory development within the CNS. Also by receiving highly refined afferent impulses from the cerebral cortex, the hippocampus assists in possibly establishing a cognitive map through which the body can recognize its position in space and time in relation to external stimuli.

The hypothalamus is a structural component of the diencephalon but is functionally organized with the limbic system and is critical in homeostasis. The hypothalamus functions to provide homeostasis and stimulate and coordinate drive-related behaviors such as sex, hunger and thirst. The means by which the
The hypothalamus accomplishes this function is by altering autonomic, endocrine, emotional and somatic functions according to afferent stimuli. The efferent connections from the hypothalamus primarily include the inner limbic systems connecting afferent influences via the pituitary gland.

Afferent connection into the hypothalamus include numerous structures of the CNS and can be functionally categorized into midbrain / limbic structures including the septal nuclei, hippocampus, amygdala, orbital cortex and the retina, and the brainstem / spinal cord. Most input from the hypothalamus parallels the afferent pathway with primary projection also to the midbrain, limbic system, brainstem and spinal cord. A functional control mechanism of the hypothalamus is its ability to regulate hormonal release from the pituitary gland. This mechanism of hormonal release is established through either neural projection via the hypothalamic-hypophyseal tract directly to the posterior lobe of the pituitary gland or indirectly by a vascular link to the anterior lobe of the pituitary gland through the hypophyseal portal system. 40-43

Basal Ganglia

The basal ganglia are one of two structures within the CNS that indirectly influence motor function through descending pathways and cortical modulation. Cortical modulation occurs through complex circuit loops involving the brain stem, cerebellum, thalamus and basal ganglia. Clinicians have divided this complex circuitry into two separate motor systems based on function and identified them as pyramidal and extrapyramidal systems. The pyramidal system consists of the corticobulbar and the corticospinal pathways. The extrapyramidal
system consists of all other projection pathways influencing motor control such as the rubrospinal, reticulospinal, vestibulospinal and tectospinal tracts of the spinal cord. Even though the circuitry of both systems parallels each other and interconnects, the term "extrapyramidal tract" is identified by clinicians as the basal ganglia. Numerous structures and complex terminology is used to identify the variety of components of the basal ganglia. For the purpose of this review only the general structures will be discussed.

The basal ganglia can be anatomically grouped into three major and two minor components. The three major components include the caudate nucleus, putamen, and globus pallidus and the two minor components being subthalamic nucleus and the substantia nigra. The caudate nucleus forms the wall and floor of the lateral ventricle and is comprised of a head, body and tail, which together forms the shape of a horn. The caudate nucleus also has direct connections to the thalamus and limbic system, which provide a unique role in motor control. Also when combined, the putamen and globus pallidus are identified as the lentiform nucleus. The globus pallidus is further divided into either internal or external.

Damage to the basal ganglia results in a variety of motor control deficits including control of movement patterns leading to akinesia, bradykinesia and poorly controlled and sustained amplitude of muscle contraction. Other possible neuromuscular deficits caused by damage to the basal ganglia include abnormal muscle tone presented as rigidity or resting tremors and poor postural control mechanisms as evident by inadequate postural awareness and corrections.
Common gait deviations seen in basal ganglia insults include small shuffling steps, festinating, poor dynamic balance and an inability to adapt to environmental changes. 44-47

**Thalamus**

The diencephalon or the thalamus is a structure within the inner most aspect of the brain. The thalamus is functionally divided into four different parts called the epithalamus, dorsal thalamus, subthalamus and the hypothalamus. The thalamus is identified as a major processing center for the brain of both sensory input and motor control. Through the different nuclei the thalamus acts as a relay station by which afferent and efferent impulses are directed through the CNS. The hypothalamus along with the limbic system plays a significant role in hormonal regulation for homeostasis. The other functionally significant component in motor control of the thalamus is the dorsal thalamus. Numerous afferent pathways pass through this part of the thalamus to include all sensory input to the structure, and many of the anatomical circuits used to connect the cerebellum, basal ganglia and limbic system. Further subdivision of the thalamus includes classifying these different circuitry systems into thalamic nuclei. Thalamic nuclei can be distinguished from each other by topographical location within the thalamus and by specific input and output patterns.

Thalamic nuclei can be generalized into two categories: relay nuclei and association nuclei. The functional role of the relay nuclei is to receive defined bundles of specific input fibers and project this information to the appropriate cortical association areas of the cerebral cortex. Association nuclei do not
receive direct input from sensory or motor systems instead they receive projections from other thalamic nuclei and cortical association areas and assist in relaying these signal to other specific areas of the cerebral cortex.

Input signal into the thalamus can be categorized into two separate classifications; specific inputs and regulatory inputs. Specific inputs are afferent signals processed by the thalamus and directed towards a distinct output. Regulatory inputs differ from specific inputs in its contribution to describe which specific inputs will leave the thalamic nuclei. Most regulatory inputs communicate between other thalamic nuclei but primary inputs come from the cortical association area to which a specific nuclei projects.

Outputs from the thalamus are also divided but, instead of by function, they are categorized by physiological condition and are identified as tonic and burst modes. Tonic neurons act similar to regular neurons in that increased depolarization of the neuron will cause the ability for action potential to travel the length of the neuron with hyperpolarization ceasing the conduction of the action potentials. The unique characteristic of the thalamic output neuron lies in the neuron's ability to function in a burst mode. The burst mode is categorized by specific calcium channels, which are more highly sensitive to depolarization and therefore may cause a depolarization wave that may be sufficient to trigger a burst action potential. 48-52

Cerebral Cortex

The cerebral cortex is a unique component of the CNS that shares the responsibility of higher advanced intellectual functions through sensory and
motor processing. The cerebral cortex is involved in many different aspects of motor, sensory and cognitive functions including memory storage and recall, comprehension and production of language, and artistic and scientific abilities. The cerebral cortex also interprets all perception and conscious understanding entering the CNS via sensory input. Motor control within the cerebral cortex includes the planning and execution of many complex motor activities especially in fine motor movements.

The cerebral cortex is divided anatomically into two regions based on function and separated by the central sulcus. A discrete region of the frontal lobe anterior to the central sulcus consists of primary motor function and is identified as the motor cortex. The motor cortex is divided into three functional areas; the primary motor, premotor and supplementary motor cortices. The region posterior to the central sulcus consists of the sensory function and is identified as the somatosensory cortex. The sensory cortex is divided into primary and secondary somatosensory cortices. The unique functional component of the cerebral cortex lies in the association cortices, which allows sensory input and perception to be translated and directed into functional motor output. The combination of motor and sensory regions coupled by the association areas provides the cerebral cortex with its functional abilities.

The motor cortex is divided into three separate regions; primary motor cortex, the supplemental motor region and the premotor area. The primary cortex is an organization of neurons projecting from the surface of the cortex to the inner region. Each column provides control to one grouping of muscles acting
on a specific joint and not to each muscle individually. Clinical research using electrical stimulation and neurological mapping techniques has identified an organizational representation of muscle groups within the body. This representation starts on the medial aspect of the hemisphere, progresses over the convexity of the cortex with the last representation being on the lateral most aspect of the cortex. The amount of cortex involvement is based on either fine or gross motor control movements. Therefore, the largest area on the motor cortex represents the greatest amount of fine motor control such as intrinsic finger movement.

The supplemental motor area is located anterior to the primary motor cortex and receives input primarily from the putamen of the basal ganglia. Electrical stimulation of this area has identified a complete somatotopic representation of the body. The supplementary motor area is believed to provide support to the primary motor cortex to help support functions of advanced planning of movements especially in coordinated bilateral extremities.

The last area of the motor cortex is the premotor area, which lies anterior to the supplementary area. Most of the input into this area is from the cerebellum. Similar to the supplementary area, the premotor area also plays a vital role in movement. What differs between the two areas is the supplementary area is more commonly used in initiated or sequenced movement. The premotor area is more active when sequential tasks require visual guidance. The literature has also identified that the premotor area is highly associated in reaching and gripping action of the upper extremity. Also, significantly large amount of
neurons within the premotor area are selective for different types of gripping activity.

Anterior to the premotor cortex but not associated with motor function is the prefrontal region. This region is identified as being highly responsive to behavioral inputs by inter-connection of thalamic nuclei. The prefrontal region also plays a key role in the development of cognition and memory. Researchers believe that this area integrates complex sensory stimuli with motivational events. The prefrontal region also is believed to be responsible for inhibition responses to delay motor responses.

The sensory region of the cerebral cortex is located posterior to the central sulcus. The primary responsibility of the somatosensory cortex is to receive sensory input into the CNS and initiate cross-modality processing. The sensory cortex is divided into three functional components; the primary, secondary and the sensory association areas.

The primary sensory area receives afferent input from different area of the body. The primary sensory region is represented similarly to the motor cortex with the leg and foot represented medially, the thigh, trunk arm and hand represented dorsolaterally and the mouth and face being ventolaterally. Afferent signals delivered to the sensory cortex originate from different sensory input structures including joint receptors, muscle spindles and various mechano and thermo-receptors throughout the body.

The secondary sensory areas are organized similar to the primary sensory area with only mild differences in order and size of representation areas.
Secondary sensory areas receive input from the primary sensory area along with direct and indirect sensory inputs from the thalamus. The primary function of the secondary sensory area is in perceiving different sensory input modalities to include touch, pressure and pain. Both acoustic and visual systems have secondary sensory areas, which are located in the temporal and occipital lobes respectively.

Located adjacent to the primary and secondary sensory areas is the sensory association area. This area receives input from the primary and secondary sensory areas and processes and analyses the sensory information. The sensory association area consists of specific or unimodal and nonspecific or heteromodal modality inputs. Information processed through specific or unimodal modality is limited to only a single sensory modality providing the input. Non-specific or heteromodal inputs act opposite of the specific in that multiple types of sensory modalities can produce similarly sensory inputs. Altering sensory rates is also a characteristic of heteromodal inputs. This ability for heteromodal nerves or non-specific to change firing rates proves the interaction between sensory input and motor output. 53-56

Cerebellum

The cerebellum is a bilateral symmetrical structure located on the cranium floor, posterior to the cerebral hemispheres. The cerebellum, along with other components of the CNS, is primarily responsible for a wide variety of motor control functions. Responsibilities critical to the cerebellum include processing afferent inputs to generate adequate correction in voluntary movements, gait,
posture, balance, and muscle tone to maintain functional activities. The
cerebellum is also responsible for the generation and sequencing of coordinated
extremity movement patterns, such as ambulation. Even though the cerebellum
is identified as an important component of motor function its only influence in
movement is performed indirectly.

Sensory and motor communication between the cerebellum and the other
components of the CNS occurs through three pairs of neuron bundles called the
cerebellar peduncles. Sensory inputs from virtually every part of the body pass
through the peduncles and enter the cerebellum. Processing occurs through the
deep cerebellar nuclei and outputs pass back through the peduncles and project
towards different components of the CNS.

Although the cerebellum receives large numbers of afferent fibers from the
body, conscious perception does not occur in the cerebellum. Furthermore,
efferent proprioceptive fibers do not contribute to conscious sensations
elsewhere in the brain. Recent scientific evidence has indicated that the
cerebellum is involved in certain aspects of cognition, more specifically in mental
processes needed for motor control and procedural learning for the execution of
complex movements.

Cerebellar lesions characteristically impair the ability of the individual to
maintain normal functional motor control. Clinical presentations vary due to the
location and extent of cerebellar lesion and could include significant problems in
coordinated movements such as assynergia, dysmetria, dysdiadokinesia and
intention tremors. Decreased functional abilities due to decreased muscle tone or
hypotonic and muscle weakness. Postural control impairments may also be
evident in poor anticipatory control, over reaction in basic movement and the
ability to change environments to task demands. Ataxic gait patterns are also
present with cerebellar insult resulting in increased base of support, staggered
step and weaving.\textsuperscript{57-60}

**Brainstem**

The brain stem is the anatomical structure, which connects the higher levels
of the CNS to the spinal cord. The brainstem also performs spinal cord functions
by containing lower motor neurons for the head and neck. The brainstem
contains the cranial nerves, which provide both sensory and motor aspects to
specific locations of the body.

Organization of the brainstem can be categorized into three functional
components; a conduit function, a cranial nerve function and an integrated
function. The most important role of the brainstem is to provide sustainment of
life through the integrated functions of the brainstem coupled with the cranial
nerves. These integrated functions include respiratory and cardiac regulation
and sustainment. These functions are accomplished by numerous nuclei within
the brain stem providing output to the specific target tissue. Other integrated
functions of the brainstem include regulation in the level of consciousness and
assistance in complex motor planning through the connection with the
cerebellum.

The brainstem also houses the cranial nerves and their nuclei. One of the
responsibilities of the cranial nerves lies in motor control of the head and neck.
Other key responsibilities involving cranial nerves include using sensory receptors to identify special scenes such as taste, hearing, sight and smell and to also provide equilibrium information to the CNS. Cranial nerves couple integrative functions of the brain stem and also help regulate respiratory and cardiac functions of the body.

The simplest function of the brainstem is that of a pathway or a conduit for spinal ascending and descending tracts. Sensory tracts must pass through the brainstem to reach sensory input components of the CNS while motor outputs usually descend to target structures, which they innervate. Relay nuclei within the brainstem and other CNS structures are also involved in these specific pathways. 61-64

Spinal Cord and Sensorimotor Pathways

The spinal cord is a critical component in the CNS because of its function in transmitting signals between the brain and body. Motor and sensory signals are passed through parallel spinal nerves at each segmental level of the vertebral column. Enlargements of the spinal cord within the cervical and lumbosacral regions accommodate the increased number of nerve fibers needed to monitor or control extremity function. Bilateral symmetric organization also allows equal distribution of signals throughout the body.

Apart from general motor / sensory monitoring and control, the intact spinal cord is also responsible for electing various skeletal muscle reflexes. An example of the deep tendon reflex (DTR) is when an afferent signal is produced by quick tendon pressure causing a rapid stretching of a gross muscle. The
resulting afferent signal to the spinal cord stimulates muscle contraction of that specific muscle. The sensory signal synapses to a motor neuron either directly or through interneurons causing the specific target muscle to contract, generating a sudden movement across a specific joint. The DTR is used diagnostically to assess possible CNS damage. The DTR also consists of a proprioceptive feedback mechanism with the CNS, which limits continuous muscle contraction when the reflex is elicited. The feedback mechanism within the CNS involved in the processing of the inhibitory information is critical in understanding different aspects of CP.\textsuperscript{65-68}

Another unique ability of the spinal cord is the functional use of interneurons or central pattern generators (CPG's) to generate locomotion. CPG's are an interneuron circuitry system, which is contained within the spinal cord or in higher components of the CNS. The foundation of how a CPG works is based on the motor program theory of motor control. The motor program theory suggests that a specific neural circuitry exists for all movements. This neural circuitry is also identified as the hardwired concept of the CNS. A CPG works by sending stimuli to target organs such as skeletal muscle. The action of the CPG is initiated either internally which is identified as a open loop system or from outside sensory input which is called a closed loop. The theory or the motor program theory of motor control will be more thoroughly discussed in the motor control section of the literature review.

Currently animal models are being used to investigate CPG's within the spinal cord. Using rhythmic walking stimulation in a supported weight-bearing
environment, an animal with a transected spinal column has demonstrated an ability to generate a functional locomotion pattern. Researchers believe that this movement seen in animals with transected spinal cord is caused by direct sensory inputs to the spinal cord therefore stimulating activity of the CPG. What challenges researchers is how to identify CPG’s and their functional role within the higher components of the CNS.\(^{69-75}\)

Descending pathways primarily responsible for motor control are identified by their specific function and include the corticospinal tract and the ventromedial and dorsolateral brainstem systems. Collectively, the organization of the corticospinal, ventromedial and dorsolateral tracts to their presynaptic junction is clinically defined as the upper motor neuron.

In motor control, the primary descending tract is the corticospinal or pyramidal tract consisting of the lateral and ventral tract. This tract provides the capacity for fine motor control of distal extremities and digits such as independent movements of the fingers. The ventromedial brainstem system is particularly concerned with maintenance of erect posture, integrated movements of the body and limbs and progression movements of the limbs. These pathways generally facilitate the activity of the extensor muscle while inhibiting the flexor muscles.

The ventrolateral brainstem system is concerned with fine manipulative independent movements of the limbs, particularly of the hand and feet. The ventrolateral pathway differs from the ventromedial system in that it primarily stimulates flexor muscles and inhibits the extensors. Involuntary control and
regulation of the autonomic nervous system is predominately passed through the reticulospinal system of the spinal cord.

Sensory information is carried from somatic receptors into the CNS via four major ascending pathways; fasciculus gracilis tracts, fasciculus cuneatus tracts, lateral and ventral anterior spinothalamic tracts and the dorsal and ventral spinocerebellar tracts. The fasciculus gracilis and fasciculus cuneatus are two tracts primarily responsible for carrying proprioceptive input from specific joint mechanoreceptors and muscles receptors to the brain which provide for the ability to perceive position of limbs and/or digits in space. Input from these tracts also provides somatosensory tactile information pertaining to touch and feel in relation to the surrounding environment. The spinothalamic tracts consist of lateral and ventral pathways. The lateral spinothalamic tract is responsible for carrying information pertaining to pain and temperature while the ventral spinothalamic tract transmits sensory information pertaining to the modalities of touch and pressure.

The spinocerebellar tract consisting of paired dorsal and ventral tracts and connects neuromuscular receptors such as joint receptors, golgi tendon organs (GTO) and muscle spindles sensitive to proprioceptive awareness to the cerebellum. The cerebellum then processes information from the spinocerebellar tract at the subconscious level.65-69

It is evident through this review of neuroanatomy that the complexity of the CNS is overwhelming. This section provides a foundation to which further
discussions pertaining to causes, treatments and outcomes of CP may be addressed.

**History of Cerebral Palsy**

An English physician named William Little first discussed CP over one hundred years ago. Little provided the medical community with the first clinical identification of abnormal movement patterns and deformities observed in newborn infants children. Observations made by Little included stiff joints and spastic muscles, which limited functional mobility in the child’s first years of life. These abnormal movements of a child led Little to believe that there was a likelihood a fetus could be exposed to a harmful pathology similar to those exposed to a child after birth. Due to limited advancements in medical technology, Little was unable to identify the exact disease process, which he believed was occurring within an unborn child.\textsuperscript{76-78}

After further investigation Little changed his initial thoughts and hypothesized the abnormal movements and deformities being observed were likely due to a traumatic birthing processes such as premature labor, breech delivery or asphyxia during or immediately after birth, instead of an infection. In 1862 Little presented a paper titled *The Influence of Abnormal Parturition, Difficult Labor, Premature Birth and Asphyxia in Relation to Deformities* to the Obstetrical Society of London.\textsuperscript{79} Little, who for the first time, proposed a direct relationship between various neuromuscular disabilities of neonates and children who had undergone a difficult delivery, neonatal asphyxia and prematurity. During this presentation, Little characterized these abnormal movement patterns causing
deformities as spastic rigidity. Little's conclusions were drawn from the observation of two hundred children who had undergone a traumatic birthing process. As Little observed these children, he identified other secondary physical problems including joint contractors, joint deformities, and scoliosis. Cognitive and other neurological changes consisting of mental retardation, irritability, mild epilepsy, and mood changes were also observed in these children. Physicians began identifying children who presented characteristic symptoms of spastic rigidity as having "Little's Disease." 78

Little's colleagues were reluctant in accepting his hypothesis, questioning how asphyxia at birth was causing neurological and orthopedic deformities later in a child's life. Another problem for Little's colleagues was the difficulty in recognizing and classifying the complex condition with so many variables involved.77 Most physicians during this time were aware that the majority of asphyxiated newborns requiring resuscitation eventually recovered unharmed therefore, leaving Little's ideas highly questionable. Neurologists also admitted to a persistent confusion between the diagnosis of cerebral palsy and poliomyelitis.77,80,81

Little's thought processes were influenced by many great scientific minds, who observed similar abnormal movement pattern in infants years before he was born. Archived medical history revealed that Hippocrates in the fourth and fifth centuries BC used splints and exercise for people with congenital defects similar to those classified as spastic rigidity. Galen was another influence on Little due to his discoveries in neuromuscular physiology in the first century. Egyptian,
Chinese, Hindu, Greek and Romans cultures also have recorded medical literature explaining the importance of bracing and splints in the treatment of deformities, again, similar to the observations of Little which, would later be describe as spastic rigidity. 7,8,28,33

In 1888 research into spastic rigidity within the United States was lead by Dr. William Osler, Professor of Medicine at University of Pennsylvania. Osler delivered the first series of lectures within the United States titled The Cerebral Palsies of Children, which addressed clinical description and presentation of spastic rigidity. These lectures and Osler’s own research that the term cerebral palsy began to be used more commonly to describe spastic rigidity.84-90

Sigmund Freud was another individual who made significant contributions to the understanding of CP. In the late 1800’s Freud published four publications pertaining to CP. Through these publications he presented the first clinical classification categorizing the factors into congenital, acquired during birth, and acquired postnatally.91 Freud suggested that the relationship between the location of a specific lesion could determine the amount of involvement and also provided extensive descriptions of the movement disorders commonly seen in CP. Freud continued in stating that the more superficial the lesion, the increased likelihood of lower extremity involvement in the form of spasticity and contractures. Freud partially agreed with Little in that the majority of causes of CP were related to the birth process itself. Freud believed that cerebral palsy resulted from considerable damage to the brain but reasoned that for each case resulting in severe injury the variability of clinical presentation were unique.92
Freud's objection to what Little proposed was based on children with CP having secondary problems such as mental retardation, visual disturbances and seizure activity. Freud suggested that the disorder might also have roots earlier in life, such as during the development of the brain before birth. Freud took Little's hypotheses one step further in suggesting difficult birthing itself might be in some cases a symptom rather then a cause.

At the end of the nineteenth century medical researchers abandoned work on further classification and diagnoses of CP due to the lack of any specific neuropathological correlation with clinical presentations. This lack of interest in CP gave rise to the neurological description of CP as the "wastebasket" category well into the mid-nineteenth century. Little's Disease was the common name for spastic rigidity until the mid-1940's when the American Medical Association officially accepted the medical term Cerebral Palsy.

In the late 1800's, surgical interventions for the treatment of CP were becoming more viable options for the management of spasticity and subsequent deformities. During this time, neurological procedures such as ramisectomies, ganglionectomies, obturator nerve resection and extra-pyramidal tract resection were being used for the management of spasticity. Numerous orthopedic procedures were also being attempted to alter any orthopedic deformities in the lower extremity caused by the spastic muscle, which inhibited functional mobility.

Lastly, are the contributions of current healthcare providers MacKenzie, Lovett and Wright, and Hughling-Jackson whose techniques in the development of
muscle reeducation have been key components in the treatment of CP. Another commonly used treatment is Neurological Developmental Treatment (NDT) developed by Bobath. Components of NDT are widely used in the treatment of CP and will be discussed in depth in a later section.

**Incidence, Prevalence and Etiology**

Numerous risk and causative factors have been identified which may attribute to the cause of CP. The clinical presentation of CP varies considerably due to the cause, location, extent of neurological damage and the developmental level of the CNS. Therefore, between multiple possible causes and varying clinical presentations, researchers are challenged to account for any specific incidence or prevalence rates in relation to each cause of CP within the pediatric population.

The definitions of incidence and prevalence rates in a given population are commonly misused and not understood, therefore it is important to identify the differences between these terms especially in CP. An incidence rate is defined by the rate at which new cases of a disease occur within a given population during a specified time period. Prevalence rates differ from incidence rates in that prevalence is the actual number of cases of a disease existing in a given population at a specific time. In CP both incidence and prevalence rates are usually compared to a uniform number such as the number of live births.

Due to the many factors in CP, incidence rates will more accurately identify specific trends per medical condition then a prevalence rate. Some researchers report prevalence rates for CP have remained constant since the 1960's per
1000 live births at 1.4 to 2.7 per 1000 live births while other studies identify a slight increase since 1970. Although the overall prevalence of CP has remained constant, increases in incidence rates for CP has occurred especially in preterm births and low birth weight infants. The importance of incidence rates in CP is evident in the literature by the number of publications that extensively investigated incidence rates instead of prevalence rates in relation to specific etiological factors. In addition a review of the literature in relation to incidence rates and etiological factors, numerous authors discuss the challenge of separating the incidence rate of CP from etiological factors. This section will review the primary and secondary risk and causative factors of CP and report incidence rates specific for each component discussed.

Only a few studies have been published which attempt to account for all possible risk factors and report a general incidence rate for CP. Due to the lack of specific diagnostic criteria for CP, variations in these specific incidence rates are evident. The largest research project, which has addressed the incidence rates of CP, is the Collaborative Perinatal Project (CPP) performed by the National Institute of Neurological Disorders (NINDS). Incidence rates of this project were first compiled in a general category of CP and then reorganized to account for incidence rates in each of the causative factors. Nelson et al reported that the incidence rate of CP in infants under the age of seven is 4.6 per 1000 births as reported by the CPP. This incidence rate accounts for deceased children diagnosed with CP but never reported any incidence rates excluding these children. A study by Rosen et al investigated the same
population of children diagnosed with CP but excluded deceased children. Rosen reported that the incidence rate of 2.7 per 1000 live births, which is significantly lower due to the exclusion of deceased children diagnosis with CP. Nelson et al in a second study, which also excluded deceased children, reported an incidence rate very similar to that of the previous study, which were 2.1 per 1000 births. Without these studies either including or excluding the deceased children, it is difficult to determine the accuracy of the count.

Despite tremendous advances in perinatal and neonatal care in the last 20 years, there has been essentially no significant decrease in the overall incidence rates of CP. Medical technology has allowed further investigation into incidence rates due to possible causes of CP. Subsequent reevaluation of causative factors has raised serious questions about the relationship between delivery events and subsequent cerebral palsy. Studies now suggest the perinatal origin of CP is much less common then previously thought. Antepartum events account for a significant portion of CP although in the majority of cases the specific cause is unknown.

Further investigation into incidence rates of CP is categorized by causative factors. Lacking one common etiological factor, medical researchers have identified many likely causes and risk factors for CP. Pschirrer et al reports that through record reviews more than seventy-five percent of the children diagnosed with CP a single causative factor could not be identified.
Primary Causes

Through the literature, three significant causes of CP have been identified which include prenatal infections, perinatal asphyxia with labored birth, and postnatal low birth weight with premature births. Recently medical researchers have become interested in a possible association between intrauterine and fetal infections and the occurrence of CP in newborn children. Gilstrap et al discusses two common medical infections, chorioamnionitis and funisitis which are contracted by the mother during gestation. Chorioamnionitis and funisitis are both suspected in causing possible birth complications which result in CP. Chorioamnionitis is an intrauterine infection contracted by the mother and manifested primarily via a fever of less then thirty-eight degrees Celsius. Chorioamnionitis can be detected either clinically through a culture of the mother's blood or histologically on the skin of the newborn at birth. Over fifty percent of the women who deliver prematurely have been identified as having chorioamnionitis via clinical or histological detection. Numerous studies have investigated a possible link between chorioamnionitis and CP and will be discussed in further detail later in this section. The second type of infection is funisitis and presents as an inflammation of the umbilical cord. Primary means of infection is cellular by bacterial invasion such as a form of streptococcus and normally diagnosed using microscopic imaging after birth. Early detection, diagnosis and treatment of these specific medical pathologies have challenged researchers.
Numerous biochemical markers have been developed and clinically proven to diagnose maternal and fetal infections or inflammatory responses to infection. Several cytokines are suspected and currently one of the more commonly studied inflammatory cytokines include interleukin-1 beta (IL-1\(\beta\)), interleukin-6 (IL-A), and tumor necrosis factor-alpha (TNF-\(\alpha\)) as markers for newborns at risk for periventricular leukomalacia (PVL) which is associated with CP.\(^{116,117}\)

Eastmen et al, in 1955, first questioned the possibility of intrauterine infections being a cause for what was then known as Little’s Disease.\(^{118}\) The idea of intrauterine infections and their possible relationship to CP as a primary cause is extensively investigated throughout the literature with conflicting conclusions amongst researchers. The literature also accounts for numerous studies that investigate the relationship of low birth weight infants with infections and the incidence rate of CP. Some of these studies will be discussed in this section with a thorough review of low birth weight and CP in a later section.\(^{119-123}\)

Dammann et al recently summarized twenty-eight studies between 1972 and 1998 in an attempt to identify any evidence to a possible correlation between fevers during pregnancy or prenatal infections in relation to the incidence of CP. Variables pertaining to neonatal infections including blood borne pathogens, vasculitis, sepses and maternal infections including urinary track infections, clinical chorioamnionitis, clinical sepsis, and placental infection were thoroughly investigated. Dammann et al concluded that there is significant evidence within published research to support that a strong correlation between infections may be a primary cause of CP.\(^{124}\)
Grether et al reported that a significant number of newborn diagnoses with CP and weighing less than 2500 grams experienced a maternal infection during gestation. In forty-six children with CP and 378 controls, these investigators found a significant association with maternal fever during labor or a clinical diagnosis of chorioamnionitis and CP. In mothers with a maternal temperature less than thirty-eight degrees Celsius a relative risk of 9.3; 95% (CI 2.7-31.0). Also, newborns of women with histological evidence of placental infection presented a significant increased risk of CP. Grether concluded that maternal infection is associated with a marked increased risk of CP in infants of normal birth weight and maternal infection is also linked with low Apgar scores, seizures, newborn resuscitation and meconium respiration syndrome.

As mentioned previously, chorioamnionitis is a significant problem in newborns if infected. Fortunately, the incidence rate strictly for chorioamnionitis is not exceptionally high. Alexander et al investigated 101,170 full term infants to establish a specific incidence rate for chorioamnionitis. Out of the total study, 5,144 infants were born to woman with chorioamnionitis. Alexander reported that of the newborns exposed to chorioamnionitis had significantly lower Apgar scores, lower umbilical artery pH levels, increased number of seizures and an increased number of newborns with sepsis. Once Alexander adjusted for all secondary factors, intubation in the delivery room had the strongest correlation to chorioamnionitis. Alexander concluded that neonatal complications of infection at birth are likely due to chorioamnionitis but fetal neurological deficits are likely related to labor complications.
Wu et al recently published results of a meta-analysis to determine if chorioamnionitis was a relevant risk factor for CP.\textsuperscript{127} This study consisted of a MEDLINE search of all relevant studies from 1966 to 1999. Of the studies reviewed nineteen addressed clinical chorioamnionitis and seven histological chorioamnionitis in relation to the incidence of CP in full and pre-term births. Wu identified the relative risk of clinical chorioamnionitis was 1.9; 95% (CI 1.4-2.5) and histological chorioamnionitis relative risk was 1.6; 95% (CI 0.9-2.7). Wu concluded that the presence of chorioamnionitis in either a clinical and / or histological presentations increases the risk of a child developing CP.\textsuperscript{127}

Two studies by Alexander et al and Dexter et al investigated the significance of infection, specifically in mothers with a confirmed diagnosis of chorioamnionitis in relation to the incidence rate of CP.\textsuperscript{114,128} Alexander reported that out of 1,367 very low birth weight (VLBW) infants ninety-five were exposed to chorioamnionitis during gestation. Significant increases in the rates of intraventricular hemorrhage, PVL and early seizures occurred with odds ratios in the first twenty-four hours being 2.8; 95% (CI 1.6-4.8), 3.4; 95% (CI 1.6-7.3) and 2.9; 95% (CI 1.2-6.8) respectively.\textsuperscript{114}

In a study performed by Dexter et al the results contrasted those of Alexander. Dexter comparing the outcomes at seven months of age, in 71 VLBW infants exposed to acute chorioamnionitis to 259 infants who were not exposed and reported that no significant differences in outcome measures existed.\textsuperscript{128}
In two other studies, researchers investigated a possible association between intrauterine infections and the relation to a specific type of CP. Studies by Dammann et al reported that a cohort study of 312 children evaluated six years of age, twenty-nine presented with spastic CP. Of the 29 children 24 were born due to idiopathic preterm labor or premature membrane rupture. Dammann speculated that an infection process contracted during pregnancy induced the premature delivery experienced but lacked any scientific data to conclude such a finding.

O'Shea et al examined the association between spastic diplegic CP and chorioamnionitis and reported finding similar to Dammann. In 815 children one year old, 62 cases of cerebral palsy were identified. Medical records were reviewed for markers indicative of intrauterine infections. O'Shea identified that chorioamnionitis is strongly associated with a relative risk of 2.6; 95% (CI 1.0-6.5). O'Shea also identified that spastic diplegia presented itself with the highest association but this study did not allow a specific investigation. In reviewing the literature it is reasonable to assume that maternal infection is associated with brain damage subsequently leading to CP.

Until recently, perinatal asphyxia has been believed to be a key component in causing CP and commonly discussed in the literature as the primary cause. Even though asphyxia is considered to be one of the common causes of CP new research has identified that perinatal complications are a less prevalent cause of CP then first believed.
One of the first studies to investigate incidence rates of perinatal asphyxia was the Collaborative Perinatal Project (CPP). Nelson et al reported that data collected from the CPP provided supporting evidence that asphyxia did not significantly contribute to a higher incidence rate of CP. In 189 children, Nelson reported that 21 of these children had only one marker suggestive of CP. Conversely, the remaining 79 of the children diagnosed with CP presented with no clinical markers for asphyxia therefore leading Nelson to conclude that asphyxia is significantly less likely to cause CP then initially suspected.

In a review article, Pschirrer et al outlined some of the current markers being used in medicine to identify asphyxia. During labor markers for asphyxia may include fetal heart rate abnormalities and meconium staining of the amniotic fluid. Perinatal markers for asphyxia include low Apgar scores and umbilical artery acidemia. Neonatal markers commonly used to identify asphyxia are early onset seizure activity and encephalopathy. Other asphyxia makers include nucleated red blood cells, urinary lactate:creatinine spectrophotometric ratio and biophysical profile.

Blair et al in another study reported that in 183 children with spastic CP and 549 control children in Western Australia, a relative risk of 2.48; 95% (CI 1.85-4.37) of asphyxia during birth. Of the children with spastic CP, Blair estimated that eight percent or (15/183) were caused by intrapartum asphyxia. Research published by Torfs et al reported incidence rates from the California Child Health and Development Study (CHDS) and compared their findings to the results of the CPP. Results reported from the CHDS identified that in 41 children with CP,
32 or 78% of the children did not experience perinatal asphyxia at birth. The remaining twenty-two percent of the children who sustained asphyxia had secondary prenatal risk factors for CP. In the same study of 561 control infants, 16 or 3 percent had perinatal asphyxia during birth but fully recovered without any complications. Torfs concluded that both the CPP and the CHDS presented significant evidence to conclude that perinatal asphyxia is less likely to cause CP than originally suspected and that further research is needed to investigate prenatal, gestational and neonatal risk factors. Neither the CHDS nor the CPP were able to identify any specific etiological factors for CP.

Postnatal premature or low birthweight (<2500 grams) is one of the most significant causative factors for the development of CP. Rosen et al performed a literature review of eleven studies from 1985 to 1990 and reported an incidence rate of CP in low birthweight infants being 15 to 1000 live births. In neonatal VLBW a significantly higher rate occurred from 15 to 90 per 1000 live births. Overall, in the eleven studies reviewed, 36 percent of low birthweight infants were diagnosed with CP.

Nelson et al used data from the CPP reported that the relative risk of CP in newborns weighing less than 2500 grams is 4.9; 95% (CI 1.72-6.98) and for birthweights less than 2000 grams 13.7; 95% (CI 1.45-6.43). Torfs et al also reported the relative risk of children with CP born weighing less than 2000 grams as being 4.2; 95% (CI 2.34-12.67). Cummins et al reported incidence rates of 192 low birth weight children against a control of 155,636 children. Out of the
given population eight percent of the children, weighting less than 1500 grams and 28 weighting less than 1000 grams were diagnosed with CP.  

Secondary Causes

Few studies in the literature investigate possible secondary causes of CP such as multifetal gestation, placental abnormalities, environmental toxins or trauma following birth. Many of the factors previously mentioned have been identified to possibly cause CP either by themselves or when associated with other risk factors. The following section will further investigate these secondary causes of CP and the specific incidence rates as reported.

Research findings suggest that multifetal gestation appears to be a significant risk factor for CP. Grethner et al reported that CP was twelve times more likely in twin gestation versus single gestation. Specifically, Grethner identified the incidence rates of CP to be 12 per 1000 twins and 1.1 per 1000 for single gestation. Approximately one in five cases of CP in infants less than 1500 grams at birth occurred in twins. Although more twins had low birthweights, the rate of CP in twins with normal birthweight was significantly higher than in single births.

Another important finding in Grether’s study was that the rate of CP within the twins was similar when comparing like and unlike sexes. Grether concluded that the data suggest there might be an increase risk of CP in dizygotic twins versus monozygotic twins.

The incidence rate of CP has been even higher as the number of fetuses increases. Petterson et al reviewed medical records for the prevalence of CP in twins and triplets born in Western Australia. Petterson reported an incidence
rate of 28 per 1000 births in triples, 7.3 per 1000 births in twins, and 1.6 per 1000 births in singletons. Peterson concluded that twin pregnancy resulted in CP 8 times more often and triples 47 times more often in comparison to single pregnancies. Yokoyama et al also reviewed medical records of 705 twins, 96 sets of triplets and 7 sets of quadruplets born in Japan. The prevalence of cerebral palsy was 0.9% in 1410 twins, 3.1% in 287 triplets, and 11.1% among 27 quadruplets. The study also reported the risks of producing at least one child with cerebral palsy were 1.5%, 8.0%, 42.9% in twin, triplet, quadruplet pregnancies, respectively. Pharoah et al reported a prevalence of CP of 2.3 per singles, 12.6 in twins and 44.8 in triplets per 1000 live births. Williams et all studied the effects of twinning, birthweight and gestational age on the incidence rate of CP and concluded that the evidence suggests that each of the three risk factors were independent of each other.

Researchers have identified few cases of CP where genetic abnormalities were the primary cause of CP. Evidence suggests that the risk of a familial recurrence of CP is less than ten percent and those cases identified as having genetic causes presented with ataxic characteristics. Hyperammonemic coma and urea cycle dysfunction are both caused by genetic errors in amino acid sequencing presented in infants at birth and have been reported to manifest into different variations of spastic cerebral palsy.

Limited evidence is available which identifies different environmental toxins as a cause of CP. A study of expecting mothers following nuclear detonations in Nagasaki and Hiroshima, Japan, who were exposed to significant high levels of
radiation, revealed an increase in the number of cases in children born with mental retardation, microcephaly and CP.\textsuperscript{147,148} The use of radiation as a medical option for tumor reduction is common but presents as a risk factor for expecting mothers especially when pelvic treatment is required.\textsuperscript{149} Fetal isolation by a lead contamination field is always used even if the mother only suspects being pregnant. Prenatal gestation, by the mother, of foods containing toxins has also been identified primarily in undeveloped countries as a secondary risk factor. Studies have shown a moderate increase in the number of children diagnosed with CP when the mothers have consumed with food or water with known contaminants.\textsuperscript{150,151}

Abnormal presentations at birth such as breech or face and transverse lie have been reported to be associated with an increased rate of CP. Torfs et al reported a relative risk of 3.8; 95\% (CI 1.6-9.1) of children diagnosed with CP were born in an abnormal presentation.\textsuperscript{153} Nelson et al also reported that in low birthweight infants, breech position is identified as a significant risk for CP, but it is important to note that the presentation of birth is the predictor not the method of delivery.\textsuperscript{154} Nelson et al in another study reports that of the children born with CP and breech position, a third had a major noncerebral malformation.\textsuperscript{132} One of the more recent studies by Kreb et al also investigated breech presentation and CP in children from Denmark and reported that a mildly significant risk of 1.56: 95\% (CI 0.9-1.24) occurred between CP and breech birth.\textsuperscript{155} Kreb also identified there is difference in the rate of CP occurrence due to the mode of
delivery. Kreb concluded in stating that the increased rate of CP in breech presentation is likely due to possible prematurely amongst breech births. 155

Researchers believe that the most common placental abnormality possibly linked to an increased risk of CP is premature separation of the placenta or intrauterine bleeding. 156-157 Nelson et al reported the relative risk of premature separation as record by the CPP as being 3.5;95% (1.3-6.74). 158 Results from the CHDS reported the relative risk of PC associated with premature separation as being 7.6;95% (CI 2.7-21.1). 153

Researchers questioned the possibility of thrombophilia and resultant thrombosis in the placenta with fetal cerebral vessel damage which causes fetal and newborn neurological damage linked to CP. Kraus et al studied the autopsy findings of eighty-four newborns and identified sixteen cases of fetal thrombotic vasculopathy (FTV) in the placenta. 160 Even more important is the evidence of severe brain injury in some cases in which FTV clearly preceded the death of the fetus. Kraus suggested, but has little evidence to support, parental coagulopathy might be a significant unrecognized cause of CP. 160

The nuchal cord also has been associated as a possible cause of CP. Nelson et al reported that in a study of forty children diagnosed with CP from California a significant association of 18; 95% (6.2-48) between tight nuchal cord and spastic cerebral palsy. Nelson reported that the association of CP and tight nuchal cords is only evident in spastic quadriplegia and not in clinical presentations of spastic diplegia or hemiplegia. 161
Researchers estimate that about ten percent of all cases of CP occur postnatal and usually within infancy. Postnatal events which cause CP are categorized into non-traumatic and traumatic occurrences. Non-traumatic events causing CP are manifested by either viral or bacteria infections. Common infections causing CP include exposure to the herpes virus and Beta streptococcus. Older children are more likely to develop CP by contacting viral encephalitis or bacterial meningitis. Researchers also have identified other non-traumatic events such as metabolic abnormalities, that may present as a risk factor for CP. A primary metabolic abnormality leading to CP is the inability for the body to regulate bilirubin leading to bilirubin encephalopathy. Clinical presentation of bilirubin encephalopathy is choreoathetoid movements secondary to high levels of bilirubin affecting proper function of the basal ganglia. Medical advancements in Rh immunizations have nearly eliminated any chance of brain damage caused by increased bilirubin levels. Hypoglycemia is another metabolic abnormality, which used to be identified as possible risk factor for CP. Research has shown that unless the level of hypoglycemia is severe and left untreated for an extensive amount of time there is no risk of manifesting CP. Traumatic events manifesting into CP primarily occur in older children involved in motor vehicle accidents, fall from significant heights or victims of domestic abuse.

In reviewing the literature it is evident that medical researchers are unsure and cannot agree to the exact cause of CP. Researchers have investigated numerous causative factors which all provide supporting evidence to link to the
causes of CP. Due to the complexity of the CNS and multiple causative factors the true etiology of CP is unclear. Until a defined set of clinical diagnostic criteria is established, variations in incidence rates among researchers are likely to remain inconsistent.

**Diagnosis and Detection of Cerebral Palsy**

Currently medical researchers have not been able to develop an exclusive test to definitively conclude a diagnosis of cerebral palsy. Because there is no specific diagnostic test for CP, the means of diagnosis becomes one of exclusion and differential diagnosis. Common neurological deficits, which can clinically be present in newborns, must be excluded in diagnosing CP. This includes neurodegenerative disorders, lesions of the spinal cord, neuromuscular disorders, metabolic abnormalities, and mental retardation. Lacking a specific test and with numerous other possible childhood pathologies, a definitive diagnosis of CP may not occur until upwards of six months. Nelson et al reported that about forty-three percent of CP diagnoses were made in the first six months and seventy percent within the first year. Even with marked increases in medical technology, subjective reports by medical doctors express little change in these figures.

This section will investigate the different diagnostic tools and clinical procedures used to diagnoses CP. The discussion will start by investigating physical exams performed by medical doctors and allied health care providers. The importance of subjective reports by a child’s parents, teachers and family members about movements and behaviors outside of the treatment areas will...
also be discussed. The focus will then shift to investigating the validity of
different imagery equipment and laboratory tests currently being used in
diagnosing CP. Keep in mind that when reading this section that the diagnosis of
CP is based on exclusion criteria and a differential diagnosis and imagery
modalities being discussed only provide information to medical doctors to assist
them in making a diagnosis.

Currently the diagnosis of cerebral palsy is based on a physician’s ability to
identify developmental abnormalities in comparison to other pathologies. By
attaining solid medical histories, conducted thorough physical examinations,
utilizing proper neurological imaging techniques along with exclusion criteria of a
differential diagnosis, physicians tend to be able to quickly conclude a diagnosis
of CP. ¹⁷¹

A clinical marker for medical doctors in the diagnosis CP is to identify the non-
progressive nature of any movement disorders. Through numerous physical
examinations, trained medical specialists will be able to identify any progression
in the development of a childhood movement disorder. Many generalizations do
exist in using differential diagnosis, which may confuse untrained physicians,
therefore the importance of a pediatric neurologist is essential. ¹⁷²

In establishing a possible diagnosis of CP the clinician must identify the
nature of the birth, any prenatal complications or risk factors, time, and location
of the original CNS insult. This information will allow medical doctors to better
conclude the specific type of CP being presented in a child. The specific
classification of CP, along with key symptoms will be discussed in a later section.
Being able to diagnose specific types of CP is important for anticipatory outcomes and functional abilities of the child. Additional factors unique to a child suspected of having CP is their response to different types of therapy, the personality of the child and the psychosocial structure of the family. ¹⁷³⁻¹⁷⁵

Since the amount of time a doctor spends with each child is limited, observational reports from healthcare providers, family, teachers and friends help to provide subjective information, which may help lead to a diagnosis of CP. Lack of, or normal response to, developmental reflexes is very useful in the diagnosing and analyzing motor defects in a child suspected of having CP. In normal child, developmental reflexes have been proven in a variety of scientific studies to be present during specific time frames of a child’s development. Therefore, healthcare providers can use these proven normal reflex patterns as a reference tool in comparison to another child. Some of the commonly used primitive reflexes are Moro’s reflex, tonic neck reflex, placing reflex, Landau reflex and parachute reflex. In a child with CP, developmental reflexes will be present as either normal, abnormal due to incomplete movements or time frames in which the reflex should be occurring, or absent. Assessing these reflexes properly through specific developmental times is critical in the differential diagnosis process. ¹⁷⁶⁻¹⁷⁷

As mentioned earlier in this section, the ability to observe and identify characteristics specific to a child with CP is also important in the differential diagnosis of CP. This is especially important in observing developmental milestones in a child such as the ability to sit, crawl and perform other motor
functions. Common characteristics which have been identified in early infants with CP include excessive lethargy or irritability, high pitched crying, poor head control, lacking a sucking response, tongue thrust and tonic bite, oral hypersensitivity, lack of interest in surroundings, poor postural control, and abnormal primitive relaxes as mentioned previously. Symptoms seen in later infancy or after three months include the delay of gross or fine motor movements. It also included an inability to perform motor skills such as hand grasps by three months, rolling over by four to five months and independence in sitting at seven months and would include excessive arching of the back, log rolling and abnormal or prolong parachute response.\textsuperscript{176-177}

Occasionally, the diagnosis of cerebral injury or abnormality, which results in CP, can be accomplished with antenatal ultrasonograph. This diagnosis can clearly time the insult to the antepartum period in which the injury occurred. Other antenatal assessments include external fetal monitoring and the biophysical profile. Abnormalities of these tests may be associated with cerebral palsy and can be used with other data in differential diagnosis and identification.

Neurological insults to the developing CNS of an infant such as intraventricular hemorrhage, hydrocephalus, intracerebellar hemorrhage, and porencephalic cysts have been detected on antenatal ultrasound.\textsuperscript{178-182} Serial ultrasound examinations also have been able to identify abnormal changes in development of cranial structures such as the ventricles.\textsuperscript{177} Other complications identified through ultrasound include intraventricular hemorrhages and Fetal V complications.\textsuperscript{183} In a study by Manning et al. an inverse relationship was
reported between the last biophysical profile and the incidence rate of CP.\textsuperscript{184} Manning reported when the profile was zero out of ten, the incidence rate of CP was only thirty-three percent and when the profile was six out of ten, the incidence rate was forty-nine percent. Manning cautions the use of biophysical profiling to identify CP since only one third of the newborns scoring a zero actually developed CP.\textsuperscript{184}

Medical doctors have identified that most intrapartum injuries to newborn infants are caused by a hypoxic event.\textsuperscript{185} The American College of Obstetricians and Gynecologists have established specific criteria to be used to determine if an infant suffered an intrapartum injury. The guidelines are as follows: profound metabolic or mixed acidemia in an umbilical blood sample from the umbilical cord, a persistent Apgar score of zero to three for longer than five minutes, evidence of neonatal neurological sequel and evidence of multi-system dysfunction including cardiovascular, gastrointestinal, hematological, pulmonary and renal systems.\textsuperscript{186}

This section discusses the different modalities including Meconium staining, fetal heart rate tracing, Apgar score, acid / base levels and nucleated red blood cell levels, which provide medical doctors with the information needed to determine if an intrapartum ischemic event occurred which may lead to CP.

Meconium is material that collects in the intestines of the fetus consisting of intestinal gland secretions, bile, fatty acids and some amniotic fluids, which forms the first stools of a newborn. Medical doctors have identified that the presence of meconium within the amniotic fluid may indicate fetal distress. Studies have
identified that meconium complicates approximately twenty percent of all labors and thirty percent all post term deliveries. Meconium has been identified as a surrogate for asphyxia during delivery but its reliability as an indicator has not been proven. Nelson et al reported that 99.6% of normal birth weight infants, who presented with meconium in the amniotic fluid, never developed CP. Thus, the presence of meconium within the amniotic fluid at birth is not a viable detection marker for CP.

Prenatal and perinatal use of a fetal heart monitor to record fetal heart rate patterns (FHR) is common especially with mothers classified as having high-risk births. Certain FHR patterns such as fixed baselines with absent variability, severe variable decelerations, repetitive late decelerations and bradycardia have all been associated with abnormal neurological outcomes. In a recent study by Nelson et al investigating FHR and CP, seventy-eight of ninety-five children with CP and 300 of 378 controls undergoing FHR monitoring for heart rate decelerations and beat to beat variability rates had an odds ratio of 3.9; 95% (CI 1.7-9.3) and 2.7;95% (CI 1.1-5.8) respectively. After adjusting other risk factors for CP, Nelson reported that an increased risk of CP still persisted with abnormalities in FHR monitoring 2.7;95% (CI 1.4-5.4). Nelson concluded that specific findings of FHR were associated with CP but warned that numerous false-positives could exist. Another concern addressed in this study was the use of FHR finding to perform a cesarean birth which increased further complications to the both the mother and child. Other studies also have investigated the use
of FHR monitoring in relation to CP, but it is not a valid predictor in when identifying the possibility of CP or if neurological injury has occurred.\textsuperscript{191-192}

The Apgar test is another diagnostic tool being used to identify if asphyxia possibly occurred during birth. The original design of the Apgar test was not to identify if a perinatal asphyxia event occurred, but as a quantitative assessment tool to determine the need for, and effects of, resuscitation immediately following birth.\textsuperscript{193} Through clear operational definition the test evaluated the heart rate, respiratory effort, muscle tone, reflex activity and skin color by assigning each as a one, two or three. Even with clear operation definitions numerous factors, gestation age, maternal medication and the individual scoring the child may affect the score. No correlation exists between Apgar scores at one and five minutes and neurological insults.\textsuperscript{194} Nelson et al have reported that in Apgar scores less than three after twenty minutes, the risk of CP make be as high as fifty-seven percent depending on secondary medical complication. Nelson cautions that low Apgar scores themselves cannot be used to determine an origin of CP due to subjective means of assessment combined with possible preexisting congenital abnormalities.\textsuperscript{195}

Evaluating blood gas levels from the child’s umbilical cord is an effective means to identify any changes in the fetal acid base (FAB). Diagnostically FAB can be used as a marker to identify possible asphyxia events during birth. Studies have identified that metabolic acidosis is associated with seizures and neonatal death.\textsuperscript{196-197} The specific types of acidosis are important in relation to neurological insult. Respiratory acidosis at delivery has no correlation to
neurological insult. Metabolic acidosis in newborns though has lead to systemic complications including seizure activity, cardiac and renal dysfunction and periventricular leukomalacia. Also, metabolic acidosis in the term and preterm fetus is related to an increased risk of death in the first years of life and / or cognitive deficits in those who survive. Researchers have also identified that an increase in metabolic acidosis levels correlates to the progression of severity in a newborn.

Key information to remember is that not all infants with metabolic acidosis lead to neurological impairment. In a control study of fifty-nine infants with severe metabolic acidosis thirty-six had had no newborn complications. Currently the measures of metabolic acidosis levels are only indicative of neurological insult within the intrapartum time frame, thus limiting its usefulness. This leaves no functional use to predict preterm insults or CP.

Elevated levels of nucleated red blood cells (RBC) immediately after birth is another diagnostic measure currently being investigated for validity in detecting perinatal neurological damage in a newborn. Mild elevations of nucleated RBC are common in newborns but researchers believe that significantly higher levels of RBC may be a possible indicator of prenatal or perinatal complications such as asphyxia resulting in CP.

In a controlled study by Korst et al, nucleated RBC levels were measured in newborns with confirmed neurological damage. Korst reported that nucleated RBC levels were significantly higher in the number of newborns with neurological injury when compared to the control group. In this study Korst questioned the
usefulness of elevated nucleated RBC as indicators due to the rapid normalization of RBC levels the first several hours after birth.\textsuperscript{205}

Buonocore et al, in another study investigating the relationship of nucleated RBC to neurological injury, reported findings similar to Korst.\textsuperscript{206} The results of Buonocore's study identified that a significant correlation exists between elevated levels of nucleated RBC and birth weight, blood gas levels, pH levels and Apgar scores in predicting neurological damage. Buonocore concluded that the elevated nucleated RBC levels at birth does reflect perinatal hypoxia and believes that this measure can be used as a reliable indicator for perinatal brain damage.\textsuperscript{206}

Evidence exists which does not support the use of nucleated RBC levels as an indicator for brain asphyxia and perinatal brain damage. In a study by Leikin et al researchers investigated if elevated nucleated RBC is a valid indicator to identify intraventricular hemorrhage (IVH) and periventricular leukomalacia (PVL) in the first weeks of life.\textsuperscript{207} Children identified as high risk for neurological complications were given a neonatal cranial fontanel sonography and diagnosed with either IVH or PVL or the newborns were excluded from the study. Newborns with confirmed diagnoses of IVH or PVL did not exhibit increased nucleated RBC when compared to the control group. Leikin concluded the evidence demonstrated increased nucleated RBC are not a good indicator for PVH or PVL but stressed that further researcher is required.\textsuperscript{207}

Current clinical and biochemical parameters established for antepartum and intrapartum periods are nonspecific and leave many questions unanswered.
Also abnormal values are not a reliable means for medical doctors to determine when or if a neurological insult has occurred. Advances in medical technology have made neuroimaging and electroencephalograms strong diagnostic tools in detecting neurological insults that may possibly lead to CP.

Ultrasound technology is frequently used before birth to follow the developmental progression of the fetus, but ultrasound also has been effective in assessing a newborn's neurological intactness. A unique feature of the ultrasound, which compared to other imaging techniques, is its portability and ease of use. This technology has proven itself in detecting status marmoratus and focal and multifocal neurological injuries, but it is most useful in detecting periventricular leukomalacia. Ultrasonographic and postmortem studies have allowed medical researchers to identify the progression of different cerebral abnormalities, which occur during development. The problem with ultrasound relates principally to limited visualization of the sub arachnoid space, cerebral cortex, and posterior fossa structures. Problems also arise in subjective interpretation of the images, along with difficulty in distinguishing between hemorrhagic and non-hemorrhagic ischemic injuries.

Currently computed tomography (CT) is the most reliable and accurate imagery technique used to identify acute neurological hemorrhage. CT provides a visualization of abnormal ventricular size and surface contours that are characteristic of periventricular leukomalacia (PVL). Hypoxic / ischemic brain lesions also may be identifiable through density changes in comparison to other tissue. When evaluating neonates, CT imagery can provide extensive
information pertaining to decreased tissue attenuation in relation to irreversible cerebral edema and necrosis. Limitations in CT imagery include an inability to detect lesions smaller than five millimeters in size, differentiate between gray and white matter interface and detecting demyelinations of nervous tissue.\textsuperscript{216}

Magnetic Resonance Imaging (MRI) is another powerful diagnostic tool used to provide medical doctors with information due to its ability to identify small anatomical changes and tissue characterization. The unique ability of the MRI in relation to a newborn is that very small subtle changes in the development of neurological structures, such as cortical dysplasia.\textsuperscript{217} MRI imaging is also able to clearly define gray and white matter within the brain.\textsuperscript{218} Unlike the CT, the MRI is able to detect abnormalities in myelination.\textsuperscript{219}

Electroencephalogram (EEG) is another diagnostic tool, which provides medical doctors with a visualization of electrical activity within the brain. The key characteristic in identifying neurological damage is represented by a significant depression of electrical activity followed by a slow recovery of activity.\textsuperscript{220} Through serial EEG records, this technology allows a specific time frame of neurological insult to be determined once the initial EEG has been performed following birth. Limitations in using EEG technology include the identification of normal values in preterm births. Also EEG recordings cannot differentiate between numerous neurological insults before or after birth.\textsuperscript{221}

Until a specific test is established for CP observational skills and differential diagnosis of healthcare providers along with the subjective input from family members will be essential to confirm the proper diagnosis of CP.

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**Associated Disorders and Complications**

In most children diagnosed with cerebral palsy numerous secondary complications and disabilities also become important in establishing the medical treatment for the child. Seizure activity is a common secondary complication seen in thirty-five to fifty percent of all children with cerebral palsy. Seizures usually become evident at the age of two as tonic-clonic or partial complex in nature. Intellectual impairment such as mental retardation is also common and occurs in fifty to seventy-five percent of those or diagnosed with cerebral palsy. It is not uncommon for children with CP to have full cognitive abilities but deficits in sensory processing; attention disorders, depression or emotional lability may hinder cognitive and intellectual growth. Visual strabismus, amblyopia, myopia, nystagmus and blindness are common in fifty percent of diagnosed cases of CP. Hearing difficulties are also common in about ten percent of children diagnosis with CP. Motor deficits in speech and central processing problems lead to disorders of language expression and reception. Oral-motor obstacles to feeding include weak sucking strength, poor coordination of swallowing mechanism, tonic bite reflex, tongue through, hyperactive gag reflex and possible aspiration. Poor fine motor control and limited upper extremity mobility also hinder feeding. Respiratory complications may also be common due to problems associated with immobility, restrictive posturing, and an inability to clear mucous secretions thus increasing the potential for aspiration.\(^{173}\)
Classification of Cerebral Palsy

As defined previously, the term CP is not a specific medical diagnosis but instead a general classification to identify a group of numerous motor and voluntary movement problems caused by developmental abnormalities of the CNS as seen in children who suffered a traumatic birthing process. Many classification systems have been proposed but the development of a single functional classification that is acceptable for medical researchers and healthcare providers is very problematic.\textsuperscript{222-224} One of the more widely accepted and clinically used classification systems is the Swedish system. This system categorizes CP into four groups; spastic, dyskinetic, ataxic and mixed.\textsuperscript{222} The following section will thoroughly discuss each specific diagnosis based on the location of CNS insult, extremities affected, tone involvement and involuntary movement properties.\textsuperscript{222}

Spastic Cerebral Palsy

Spastic CP accounts for approximately seventy percent of the diagnosed cases of CP. Spastic CP is associated with an insult to the cerebral cortex and/or the medullary pyramidal tracts of the CNS, which are responsible for primary motor control. Complications due to an active motor control injury include ambulation, postural control and balance. Spasticity, which was defined, previously is also a clinical manifestation that leads to abnormal movement control. These children often present clinically with increased tone, rigidity, abnormal primitive reflexes and exaggerated deep tendon reflexes. Secondary complications due to these clinical manifestations are the development of joint
contractors, bone deformities and abnormal curvature of the spine. Spastic cerebral palsy is further categorized into three subtypes, which describes specific limb involvement: spastic quadriplegia, spastic diplegia and spastic hemiplegia. These subtypes all involve the manifestations of spasticity, but as the child develops and attempts to compensate for specific motor deficits, he or she begins to display behaviors unique to the specific subtypes.

The spastic diplegic child tends to have adequate strength and control in their upper extremities in comparison to their pronounced motor deficits in the trunk and lower extremities. Increased extensor spasticity along with abduction and inward rotation at the hips makes it difficult for this individual to sit comfortably. Non-ambulatory children may learn to maneuver around by pulling themselves along the floor with flexed arms and in dragging their rigid extended legs. In less severe cases, the upper extremities can be used with assistant devices for ambulation and activities of daily living. Typical gait deviations seen during ambulation are classic scissoring caused by spastic hip adductors and crouch posture or rigid lower extremities caused by spastic hamstrings or quadriceps respectively. Toe walking is another common deviation and is caused by spastic plantar flexors. Other factors that account for gait deviations in spastic CP include motor control deficits and selective movement abnormalities.

In spastic hemiplegia CP, the involvement is limited to the limbs opposite the side of the CNS insult. Early detection is important because the child will tend to gain total orientation to their normal side and neglect the hemiplegic side. This
will eventually lead to the neglect of the involved side due to poor proprioception and delay motor development of sitting, posture control, balance, standing and walking. The child at first lacks awareness and will show a tendency to fall to the involved side due to poor balance reaction of the affected arm and leg. Through therapy, the child will learn how to orient to the sound side and be forced to use that side for increased function and to decrease muscle atrophy. 225-226

Dyskinetic Cerebral palsy

Dyskinetic CP is identified by an insult to the basal ganglia and or extrapyramidal tracts of the CNS. Dyskinetic CP is far less common than spastic CP and accounts for ten to fifteen percent of the cases of CP. Impairment of voluntary muscle control is normally seen in this subtype of CP. In all forms of dyskinetic CP, all four extremities are involved to varying degrees. Clinical manifestations include abnormal tone, partial motor movements along with bizarre twisting motions, tremors and exaggerated posturing. Athetoid, choreoathetoid, and dystonic are terms used with his type of cerebral palsy to describe the lack of reciprocal tone and the nature of the dyskinetic muscle activity. Dyskinetic cerebral palsy occurs less frequently today due to better recognition and prevention of kernicterus, a condition in which elevated levels of bilirubin result in dysfunction of the basal ganglia. 227

Ataxic Cerebral Palsy

Ataxic cerebral palsy is rare and seen in only about one to two percent of the CP population. Ataxic CP is associated with insult to the cerebellum and or the extrapyramidal tracts of the CNS. Cerebellar injury characteristically results in
abnormal muscle coordination along with the lack of balance and position in space. Infants with ataxic cerebral palsy present with marked hypotonia at birth and tend to be misdiagnosed with floppy baby syndrome. In children with ataxia CP, hypotonia is then replaced by a gradual increase in tone and stiffness to the trunk muscles by late infancy. Complications of motor control include the inability to accurately time movements, poor coordination and limited overall postural support. A reduction in hypotonia is normally seen after early infancy but sometimes an old child as well. \(^\text{225-226}\)

**Mixed Cerebral Palsy**

Mixed CP is a combination of any of the previously named types. The two most common types of CP seen together are spastic and dyskinetic. The clinical manifestation, as well as physical, mental and psychological development varies greatly with each individual diagnosed with mixed CP. The child with mixed CP tends to display unique compensatory movements based on combined functional deviations of the two different types. \(^\text{226}\)

**Management of Cerebral Palsy**

In the following section a detailed discussion will outline a wide variety of treatment options for children with CP. As mentioned previously, spastic CP account for about 70% of the case of CP within children. The most common clinical manifestation of spastic CP includes increased tone, rigidity, abnormal primitive reflexes and exaggerated deep tendon reflexes. Other secondary complications are the development of joint contractures, bone deformities and abnormal curvature of the spine. Therefore the focus of medical management of
CP will be primarily directed towards the treatment of spasticity and how to improve a child’s functional activities. The discussion will be divided into two sections, medical management and rehabilitation services. The medical management will discuss treatment options such as pharmacological and surgical interventions while the rehabilitation services will discuss treatments such as NDT, exercise training, treatment modalities and the Adeli Treatment.

Oral Medications

Pharmacological interventions are common for the management of symptoms, such as spasticity, seen in children with CP. Common goals for the use of pharmacological interventions are to control extremity spasticity, muscle spasms, seizure activity, and gastrointestinal disturbances. A variety of drugs have been proven beneficial specifically for the treatment of spasticity by causing a decrease in excitability of spinal reflexes. This reduction in spinal reflexes occurs by decreasing the release of excitatory neurotransmitters, facilitating inhibitory inter-neurons, and altering contractile properties of skeletal muscle. Baclofen, benzodiazepine, clonidine, dantrolene, phenothiazine, and tizanidine are the most commonly used oral medications in children with CP for the management of spasticity and will be discussed throughout this section.

Baclofen

Baclofen is one of the newest drugs to be used in the management of spasticity in children with CP. Baclofen has an affinity to GABA receptors similar to phenothiazine which will be discussed later and therefore share very similar structural properties. Baclofen is unique because of its ability to be administered
orally and through a baclofen pump, which will be discussed in a late section. The precise mechanism of baclofen is not fully known but generally theorized to inhibit the GABA receptors sites. It is believed to inhibit reflexes by acting through the pre-synaptic cleft to reduce the release of excitatory neurotransmitters. The excitatory neurotransmitters are primarily found within the descending corticospinal tracts and the primary afferent nerve endings within the spinal cord. Researchers have also noted that increased amounts of oral baclofen will act in the postsynaptic cleft to inhibit the activation of motor neurons. A common side effect includes mild sedation but it is eliminated when dosages are started low and then titrated up slowly. Another common side effect is muscle weakness but in patients on baclofen these effects are far less severe then what is observed in patients on dantrolene. Other side effects of oral baclofen include ataxia, confusion, headaches, and hallucinations along with respiratory and cardiac depression. Unfortunately oral baclofen does not cross the blood-brain barrier effectively, therefore its abilities to decrease spasticity are limited.\textsuperscript{228-232}

**Benzodiazepine**

Benzodiazepine is another commonly used drug for management of spasticity. Benzodiazepine works by enhancing the inhibition of pre and postsynaptic activity by increasing the infinity of endogenous GABA receptors. In clinical trials benzodiazepine has demonstrated a decrease in spasticity similar to that of intrathecal baclofen, which will be discussed later in this section. Unfortunately the use of benzodiazepine is limited due to adverse side affects,
which include habituation, sedation, fatigue, and agitations. Due to the side effects of benzodiazepine, physicians prescribe dosages in low levels as an adjunctive medication to the primary drug regimen.\textsuperscript{228,229,233}

Clonidine

Clonidine is similar to Tizanidine in that they are both alpha2-adrenergic agonists, and therefore also selectively binds and stimulates alpha2 receptors within the spinal cord. Clonidine is commonly used to control increased blood pressure due to its ability to inhibit sympathetic nervous system activity. Preliminary studies have found that clonidine does contribute to mild decreases in spasticity, but researchers are unsure why the decrease occurs. Clonidine is also unique in its ability to be administered via a transdermal patch placed on the skin allowing for a time release of the medication. Medical researchers hope that by using a transdermal patch the management of this drug will become more functional for the patient in everyday life and may eliminate the use of oral medication for spasticity.\textsuperscript{228,229,234,235}

Dantrolene

Dantrolene is a drug, which alters muscle function at the cellular level and does not affect the CNS. Dantrolene directly effects the skeletal muscle cells by impairing the release of calcium from the sarcoplasmic reticulum within the muscle cell during excitation. In response to a normal action potential, calcium is released from the sarcoplasmic reticulum storage sites, which initiates myofilament cross-bridging and subsequent muscle contraction. The most common side effect of dantrolene is generalized muscle weakness; therefore
Dantrolene may be counter productive in a rehabilitation setting while trying to facilitate functional movements. This drug may also cause severe hepatotoxicity with the effects being higher in women and in men over 35 years of age. Other less serious side effects which may occur are drowsiness, dizziness, nausea and diarrhea.\textsuperscript{228,229, 238}

**Phenothiazine**

Phenothiazine is commonly used to treat anti-psychotic conditions like Schizophrenia but has demonstrated secondary effects in decreasing spasticity. The decrease in spasticity is likely due to the drug’s properties in blocking alpha-adrenergic receptor sites in the CNS. They reduce gamma motor neuron excitability while antagonizing the postsynaptic actions of dopamine. Unfortunately their use is limited by sedation and the development of extrapyramidal side effects such as tardive dykinesia.\textsuperscript{228,229}

**Tizanidine**

Tizanidine is a newer drug recently released by the FDA used to decrease spasticity and is a commonly prescribed medication for patients diagnosed with multiple sclerosis and spinal cord injuries. Studies have also reported mild decreases in spasticity among patients with closed head injuries, stroke and cerebral palsy. Due to its chemical structure as a alpha2-adrenergic agonist, tizanidine is able to bind to both imidazoline receptors as well as alpha2-adrenergic receptors within the spinal cord. In binding to these sites, tizanidine is able to decrease the polysynaptic reflex activity by reducing the amount of excitatory neurotransmitters released from the presynaptic site. Another feature
of tizanidine is its ability to inhibit the release of Substance P from the nocioceptive sensory afferent neurons. Common side effects of tizanidine are sedation, dry mouth, asthenia, visual hallucinations and dizziness. Orthostatic hypotension and elevated liver enzymes have also been identified in patients medicated with tizanidine.^[228, 229, 237-239]

The use of oral medications to manage spasticity has been thoroughly investigated by the manufacturer and also in independent studies. In using a variety of randomized control studies oral medications are proven to be a viable options in controlling spasticity in children with CP.

**Intrathecal Medications**

**Baclofen Pump**

Sometimes patients do not respond to oral medications for the management of spasticity due to side effects or other problems. Researchers have reported that approximately thirty percent of patients using oral medications are not able to manage their spasticity, or endure complications, and side effects.^[240] Another option for these patients would be the use of baclofen through an intrathecal pump surgically placed within the abdominal cavity though continuous infusion of baclofen. Injected baclofen has the same properties as oral baclofen previously mentioned. Researchers believe that the reason for the increased number of positive outcomes seen in patients using the baclofen pump in the management of spasticity is due to inability for oral baclofen penetrate the blood-brain barrier. When baclofen is administered though intrathecal methods, the medication does not have to pass the blood-brain barrier, but instead directly inhibits the
superficial presynaptic and postsynaptic receptors of the spinal cord responsible for tone in the upper and lower limbs.\textsuperscript{241}

To counter the limitations of orally administered baclofen, a new method of delivery was developed. The means by which researchers successful introduced therapeutic amounts of baclofen across the blood brain barrier is by delivering the medication directly into the cerebrospinal fluid via a catheter. This is newer modality known as either Intrathecal Baclofen (ITB) or Continuous Intrathecal Baclofen Infusion (CIBI).\textsuperscript{241, 242} In 1996 the FDA approved the use of CIBI in patients with spastic CP. The baclofen is delivered pass the dura surrounding the spinal cord and directly into the surround CSF via a catheter, which is connected to a computerized pump.\textsuperscript{241, 242} The pump is about the size of a hockey puck and is surgically placed under the skin around the pelvic region. Implantation of the baclofen pump is not identified as a permanent procedure and if complication occur the pump may be quickly removed. Battery maintenance and baclofen refills may both be accomplished simply from the surface of the skin.\textsuperscript{241, 242}

Reported advantages for the use of intrathecal baclofen include the reduction of spasticity, improvement of function, reversibility if desired outcome is not attained, and the ability to easily regulate dosage levels. Disadvantages in using intrathecal medications are the increased risk of infections of the surgical site and also in the CSF, mechanical malfunction of the pump or catheter line, leakage of CSF into the abdominal cavity, medical consequences of overdose and cost.\textsuperscript{241, 242}
Before the baclofen pump is surgically placed the patient must successfully complete a pilot test in which baclofen is injected in the subarachnoid space via a lumbar puncture. To complete this pilot test, the patients hip adductors and flexors, knee flexors and ankle plantarflexors are evaluated bilaterally using the Ashworth scale for spasticity and Spasm Scale for muscle spasms. Once the evaluation is completed a fifty-mcg dose of baclofen is injected intrathecally via a lumbar puncture. Normal response to the drug occurs in about thirty to sixty minutes and last four to eight hours. Once the effect of the baclofen is felt by the patient a re-evaluation identical to the initial evaluation of lower extremity spasticity and spasms is performed at intervals of one, two, four and eight hours to identify any changes which may occur. 242, 243

Using the evaluation protocol just outlined on adults with spinal spasticity and children with CP, Armstrong et al. in eighteen of twenty-three subjects, and Albert et al. in sixteen of nineteen subjects, reported a clinically significant decrease in the amount of lower extremity spasticity. Due to the pilot test being only one injection the effects do not last. 244, 245

Studies also have investigated the effect a surgically placed intrathecal pump has on adults presenting with spinal spasticity. Penn et al. studied the effects of the Baclofen through intrathecal injection on twenty patients with spinal spasticity due to spinal cord injury of multiple sclerosis. 246 In six months, results showed a decrease in muscle tone in all twenty subjects for an average of 4.0 to 1.2 as assessed by the Ashworth scale and a decrease in muscle spasms in eighteen of nineteen an average of 3.3 to 0.4 patients as assessed by spasm frequency. In
a twenty-four month follow up muscle tone was sustained at an average of one as assessed by the Ashworth scale and spasms were reduced to a level which did not interfere with daily function. 246

In another study Penn et al. reported on a thirty-month longitudinal study in which sixty-two adult spinal cord injury patients were continuously evaluated for changes in spasticity after receiving a Baclofen pump. 247 A marked decrease in two points in spasticity, as assessed by the Ashworth scale, was seen in all but two of the subjects. After the thirty month initial study, eighty-four of the pumps continued to work successfully for another five years without failure. In both studies Penn et al concluded that intrathecal Baclofen is an effective long-term treatment for spasticity. 246,247

Researchers also have investigated the effect the baclofen pump had on spasticity of cerebral origin. 248 Albright et al. investigated thirty-seven patients both adult and children with spasticity of cerebral origin. At six and twelve months after receiving the intrathecal baclofen pump, marked decreases in tone were noted along with increases in lower extremity range of motion and upper extremity function. Penn reported that a correlation between improved outcomes and the increased dosage levels existed. Researchers concluded that due to the ability to regulate intrathecal baclofen, spasticity at the cerebral origin might be effectively managed. 248

Another study by Armstrong et al. further investigated the effectiveness of intrathecal Baclofen in children who present with marked spasticity of cerebral origin. 249 Nineteen children with a medical diagnosis of either brain injury or
spastic CP were followed for a period of one to five years. Armstrong reported favorable results in the decrease of spasticity as assessed by the Ashworth scale. Complications with the pumps were very significant in this study with over half of the subject experiencing problems with the performance of the pump. Conclusions from this study demonstrated improvements for children with spasticity of cerebral origin but also that this treatment can result in significant complications due to malfunctions of the mechanical pump. 249

Within all the patient groups, in which the intrathecal baclofen pump is used, a variety of serious complications occurred. Overdose problems due to programming mistakes in the pump has lead to lethargy, heavy sedation and major cardiac and respiratory complications to the extent of requiring ventilator assistance. 241, 242 Infections initially were very problematic and normally required removal of the pump, but the use of prophylactic antibiotics before and after surgery has nearly eliminated any risk of infection. 250 Catheter complications such as blockage or kinks as well as cerebral spinal fluid leakage are also common problems with intrathecal baclofen pumps. 241 Removal of the pump due to complications is not that common. 250 Albright et al. reported that in forty-eight months of use only six pumps had to be removed due to malfunction and Armstrong et al reported that only two out of fifty-three pumps had to be removed in a five year time frame. 248, 249

Within the literature numerous studies exist which investigate the efficacy and safety of intrathecal baclofen. 251-257 Unfortunately, for most of these studies primarily focused on adult SCI, TBI and MS patients and provided limited support
for intrathecal baclofen use in pediatric treatment of CP. These studies do however, identify that intrathecal baclofen is a viable treatment tool to manage spasticity. Therefore, more randomized controlled studies are needed to further investigate the usage of baclofen and the intrathecal pump to manage spasticity in children with cerebral palsy.

Nerve Blocks

Phenol Blocks

In children with CP, the presence of focal spasticity may limit their functional activities. An example would be persistent spasticity only in the hamstrings or gastrocnemius muscle, creating deviations in a child’s gait. In these situations physicians may elect to treat the spasticity at the local level through nerve blocks instead of through oral medications, which affect the entire CNS.

The most commonly used nerve-blocking agent is phenol. Phenol has unique chemical properties which produce a local anesthetic affect and destroy the axons of the motor nerves while leaving the endonurial tube intact and fully functional. Due to the fact that phenol nerve blocks are only temporary physicians are unable to establish a specific time line in which distal growth of the damaged nerve ending may occur and spasticity begins to limit function.

The administration of phenol can occur in a variety of different conditions. One method of administration is through a direct injection into the nerve trunks. Complications with this injection can occur because the trunk is part of the mixed nerve, consisting of both motor and sensory components. After the injection, a decrease in tone does occur, but secondary effects include painful
paresthesias due to phenol also damaging the sensory nerves in the trunk. In performing a closed motor branch block the paresthesias is commonly felt in the previously described technique avoided. Closed motor branch blocks are more common in upper extremities due to the use of a neurostimulator to ensure that only the motor nerve is being injected.

Research has demonstrated that the longest lasting effects and most predictable outcomes occur when an open motor block is performed. Open motor blocks require surgical isolation of the motor nerve branch in a sterile environment. This procedure has demonstrated decreased levels in spasticity lasting up to eight months. The only difficulty in open blocks is the required surgical isolation and increased risk of infection. Intramuscular motor point blocks are a non-surgical procedure in which phenol is injected directly into specific motor points within the spastic muscle. A needlepoint stimulator is used prior to injection to specifically identify the location of the motor point to ensure accuracy of the injection. The time duration of decreased spasticity is less then an open block but the advantages are no surgical procedure is required and multiple motor points maybe injected during a significantly shorter time frame.

In reviewing the literature, only two studies investigate the effectiveness of phenol nerve blocks in relation to children with CP. Neither one of these studies were controlled, nor were they able to account for many variations in measurements. Conclusions of both studies did identify that phenol nerve blocks did decrease focal spasticity in children with spasticity allowing for improvement

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of function. Limitations in the amount of research are likely due to the increased use of Botox injections in the clinical setting versus block nerves.

Botulinum Toxin

Botulinum toxin or Botox has recently been used as a newer method to control muscle spasticity. Botulinum toxin A is a neurotoxin, which is produced by the gram-positive Clostridium botulinum, which acts on the target muscles to decrease spasticity. The toxin causes muscle weakness and reduced spasticity by inhibiting the release of acetylcholine and by hindering its release from the vesicles.264,265

After the initial injection, the clinical effect is not noticed for twenty-four to seventy-two hours. Clinical effects of the injection average two to six months after the administration of the toxin. Subsequent injections are not recommended any less than twelve weeks due to likeliness of immune system response to the toxin. The return of original symptoms occurs due the increased release of acetylcholine from the vesicles following the injection of the skeletal muscle.265,266

EMG activity allows for the physician to accurately identify which muscle should be injected. Common advantages in using Botox are that the effects of the injections are temporary therefore reversible if medically required, clinical evidence identifies improvements in functional activities, and is less expensive when compared to some of the surgical and other therapeutic options.267-268

A study by Feblings et al investigated the efficacy of Botox injection in a randomized controlled study of 29 children with CP between the ages of two and
five. The inclusion criteria into this study were limited to children with spastic hemiplegia. Botox injections were given only into the spastic upper extremity. The children were evaluated for improved function using Quality of Upper Extremity Skills Test (Quest) and Pediatric Evaluation of Disability Inventory (PEDI). The authors of this study concluded that Botox injections, when used with children with moderate spasticity, are an effective treatment options in improving upper extremity function.

The use of Botox also has been investigated for improvements of energy expenditure during exercise and ambulation in children with spastic CP. Massin et al evaluated 15 children with spastic CP to determine improvement in their walking endurance as identified by oxygen uptake and time to exhaustion. Significant improvements in time to exhaustions and oxygen uptake occurred, therefore allowing the authors to conclude that the use of Botox is a viable treatment tool to improve energy cost in children with spastic CP.

Another use for Botox in children with CP is to decrease post-operative pain following muscle release surgery in spastic lower extremities. Barwood et al investigated the effects of Botox as an analgesic in a randomized controlled study. The authors believed that following muscle release surgery a significant amount of pain occurs from secondary muscle spasms. Using Botox prior to the surgical procedure may eliminate part of the postoperative pain the child may be feeling. The results of the study supported that Botox injections in the target muscle prior to surgical releases will decrease the amount of pain the child and the duration of recovery in a hospital setting.
Numerous other controlled studies also have investigated the efficacy of Botox injections for the management of children with CP.\textsuperscript{272-276} Even with an extensive amount of favorable outcomes identified in the current literature, such as improvements in functional activities, strength, and gait, some researchers question the results. The key is that the importance of decreased spasticity is identified in the carry over to a child’s functional activities; therefore improvements in function can be indirectly associated with decreased focal spasticity.

Surgical Procedures

Selective Dorsal Rhizotomy

One of the few neurological surgical procedures currently being used for the management of cerebral palsy is selective dorsal rhizotomy (SDR). SDR is a surgical procedure used to reduce spasticity in affected muscle groups by decreasing the afferent input primary to the CNS from the muscle spindles and golgi tendon organ. Increased spasticity in a lower extremity muscle group is identified by the inability of the cerebral cortex to control descending inhibitions to the lower motor neuron and target muscle. Therefore, by selectively severing dorsal rootlets within the spinal nerve levels of L1 – S2 a reduction in the amount of afferent stimulation to the damaged portion of the cerebral cortex will occur. This decreased afferent input can help provide adequate inhibition to the descending tracts and possibly decrease the spasticity.\textsuperscript{277,278}

When the SDR was first performed, the child must undergo a complete lumbar laminectomy, which is the surgical removal of the bony arches of one or
more vertebrae. Surgeons have now started to perform a less complicated procedure called a laminotomy, which is only the removal of the lamina of a vertebra instead of an entire arch.\(^{279,280}\) This newest adjustment to the SDR procedure of L\(_1\) – L\(_5\) is performed to provide full exposure of the dura covering of the posterior spinal cord. With the posterior cord exposed, access to the dorsal roots of L\(_1\) – S\(_2\) is easily attained. From each root a separation of twelve or more individual nerve rootlets is performed and then identified by their possible association with spasticity within the lower extremity.\(^{277}\) This determination of which rootlet is associated with spasticity is based on abnormal electromyography results upon stimulation of a specific nerve rootlet.\(^{281}\) It is estimated that an average of forty to fifty percent of the dorsal nerve roots at each level are severed during the procedure. A shortcoming of the procedure is identifying the correct afferent nerve rootlet. Researchers have found that a poor correlation exists between nerve rootlet electromyography and actual spasticity in the extremity muscle group. The SDR procedure usually takes about six to seven hours to successfully complete so as to ensure the identity of the correct afferent nerve and to sever the proper rootlets.\(^{282}\)

Through past experiences surgeons have determined that the ideal candidate for SDR is an ambulatory child with a medical diagnosis of spastic diplegia between the ages of three and eight years of age. Marked extremity spasticity, along with good muscle strength and trunk balance, is also required for the procedure. The child must also demonstrate adequate intelligence, strong motivation and an ability to successfully participate in physical therapy (PT) and
occupational therapy (OT) programs post surgically. These last requirements are very important secondary to the need for rehabilitation services after the procedure. Contraindications for SDR include ataxia, dystonia, muscle weakness and severe fixed joint contractures. Common complications identified after the procedure are marked lower extremity limb weakness, a decreased sensation especially to touch and temperature changes and decreased proprioception in the limbs. Most of these complications present as severe but normally are temporary in nature, especially sensation changes. Spinal deformities are also noted such as spondylolysis, spondylolisthesis and increased lumbar lordosis. Surgeons have reported that the number of spinal deformities has decreased slightly and is partially due to the procedure being changed from a laminectomy to a laminotomy.

Numerous research studies have investigated the SDR and the efficacy of the procedure. Buckon et al. identified that SDR does produce a marked decrease in spasticity of the affected limb due to a change of one to two points on the Ashworth Scale, as well as an improvement in overall range of motion within the lower extremities. Boscarino et al. identified a variety of improvements in children one year after undergoing SDR. Using three-dimensional gait analysis increases in hip and knee extension during stance, knee flexions during swing and ankle dorsiflexion during the entire gait cycle were noted. Similar improvements of children who have underwent SDR ten years previously were also identified by Subramanian et al using three-dimensional gait analysis.
A variety of research studies examined changes in the functional abilities in children after having undergone SDR. Peacock et al. investigated twenty-two children with spastic diplegia at six and twelve months after having undergone SDR. In using the Pediatric Evaluation Disability Inventory (PEDI) clinically significant improvements were seen in gross motor skills, standing balance, walking and upper extremity activities. Therefore the researchers concluded that SDR was an effective surgical procedure for the children with spastic CP. Dudgen et al. also investigated changes in children with CP after undergoing SDR. Twenty-nine children diagnosed with either spastic diplegia or spastic quadriplegia underwent SDR and then postoperative physical and occupational therapy. At six and twelve months postoperatively, upper-limb movement, self care, and functional mobility also were evaluated using the PEDI. Significant clinical changes were identified using the PEDI in children with spastic diplegia but only in functional mobility and self care. No significant changes were identified in upper-extremity reaching or coordination tasks after the SDR. Spastic quadriplegics had no changes in any categories as assessed by the PEDI.

Between Peacock and Dudgeon both groups of spastic diplegic children demonstrated clinically significant improvements, but the spastic quadriplegics demonstrated no significant improvements. The lack of improvement seen in the spastic quadriplegic could be in relation to the SDR procedure itself. SDR specifically severs lower extremity afferent nerve rootlets; therefore the likelihood of seeing improvements in a spastic diplegic is far greater than in a spastic
quadriplegic. Another consideration is the amount and intensity of PT and OT may be different. Peacock's physical therapy plan required more hours per day for a longer duration of rehabilitation then Dudgeon's group.\textsuperscript{289, 290}

In comparing functional improvements seen in SDR one critical component that must be investigated is the importance of physical rehabilitation after the procedure. As seen in Peacock and Dudgeon's studies, physical therapy was part of the rehabilitation plan but was not investigated in relation to its importance to functional outcomes after SDR. Three studies have been performed which investigate SDR and its relationship to PT and OT received by children with CP.

The first study by McLaughlin et al. is a randomized clinical trial, which compared children with spastic diplegia who received SDR with physical and occupational therapy to those children who just received the identical therapy.\textsuperscript{291} Forty-three children were randomly assigned to the SDR plus PT group or just the PT group with thirty-eight completing the 24-month study. Both groups underwent identical PT programs except for the initial six weeks of inpatient therapy received by the SDR group. The number of subjects per group was twenty-one and seventeen respectively. Data was collected using the Gross Motor Functional Measure (GMFM) at the initiation of the study and after, six, twelve and twenty-four months. Results of the GMFM were 7.0 percent improvement for the SDR plus PT and 7.4 percent improvement for just the PT group with a P value of 0.94 meaning no significant change occurred between the two treatment groups. McLaughlin concluded that the SDR with PT does not produce improvements as measured by the GMFM and therefore, SDR may not
be the most viable option for a child with CP especially when considering possible surgical complications and the child's initial functional condition. A structured PT program will likely produce the same functional outcomes while limiting risks factors to the child.  

Another study is by Steinbok et al. The setup of this study was very similar to that of McLaughin et al. Thirty children were randomly selected, fifteen into each group between SDR and PT and just PT. Each group was followed for only nine months and initial data was collected using the GMFM at the start of the trial and at the end of the trial nine months later. In this study there were statistically significant improvements in the SDR and PT group in comparison to just the PT group. At nine months, results for the SDR and PT group showed an improvement of 11.3 percent and 5.2 percent improvement in just the PT group. The researchers concluded the marked increase in the improvement seen in the SDR group is unexplainable and therefore not a clear indicator to the validity of SDR.  

The final study by Wright et al was also designed to compare changes in motor function of children with spastic CP one year following SDR procedure in comparison to just PT. For this study twenty-four children were randomly assigned to each treatment group, twelve children per group. The GMFM was administered initially, at six months and at twelve months. At one year the results of the GMFM demonstrated marked improvements similar to that of Steinbok et al. Wright reported an improvement in the SDR plus PT group of 12.1 percent and in the PT group an improvement of only 4.1 percent. Again the
Wright et al. concluded that SDR plus PT leads to marked improvements in functional abilities as identified by GMFM in comparison to a child receiving just PT. These three studies all have been almost identical in setup, but yet two of the three showed statistically significant improvements in children with spastic diplegic CP when assessed by the GMFM to compare SDR and PT to just PT. One of the conclusions could be the initial level of disability of each group of children. Case studies have shown that children with spastic diplegic CP, who are more involved, tend to show better post surgical outcome after SDR.

Another possible reason for the marked improvements in Steinbok and Wright’s studies may be due to the number of rootlets severed during the SDR. Steinbok and Wright both reported that an average of forty-five to fifty percent of the rootlets were severed during the SDR procedures on their subjects in comparison to the twenty-six percent severed in McLaughlin’s subjects.

Another goal for SDR is to reduce orthopedic deformities, which are commonly seen in spastic CP. Chiconie et al. examined the effect of heal cord, adductor and hamstring releases on 178 children with spastic CP using the Kaplan-Meijer evaluation tool. Groups of children between the ages of 2 to 4 and 5 to 19 were evaluated. The 2 to 4 age group showed a marked decrease in the amount of orthopedic surgeries required versus the 5 to 19 age group; therefore, suggesting the younger the SDR is performed, the greater likelihood of fewer orthopedic deformities which require surgery. Much more research is needed to establish the validity of this assumption.
This section discussed the SDR surgical procedure and its place in the management of spasticity in CP. A variety of research articles also have investigated different preoperative and postoperative aspects of the SDR procedure. The evidence through this research suggests that the SDR is a very viable tool in the treatment of spastic CP.

Orthopedic Procedures

The use of orthopedic surgical procedures is still a medical intervention for children with CP. Complications such as joint contractures, bone and joint deformities and scoliosis hinder basic function of a child such as sitting and walking. Surgical management of these complications includes soft tissue releases and transfers, and the correction of bony deformities caused by spastic muscles. Common outcome goals following orthopedic surgery include improvements in function, comfort in activities of daily living, positioning and prevention of future deformities and contractures. This section will discuss primary orthopedic surgical procedures being used for the management of spasticity in children with CP. These surgical interventions will be investigated for effectiveness and validity as each procedure relates specifically to the hip, knee and ankle.

Orthopedic surgeons have categorized the many different types of orthopedic surgical procedures being used to manage spasticity in children with CP into soft tissue procedures and bony reconstruction. Tenotomy is a surgical procedure in which the orthopedic surgeons completely release a severely spastic muscle from its origin or insertion. The tendon is surgically relocated into an anatomical
position in which the spasticity of the muscle does not hinder the function of the effected extremity. Muscle transfers are another soft tissue procedure very similar to a tenotomy. The difference being the tendon is transferred to another insertion site, allowing the spasticity to assist in functional movement. Instead of severing or relocating the spastic muscle, orthopedic surgeons may elect to lengthen the muscle belly, which may produce physiological responses evident in decreased spasticity as well as improve function at a specific joint. If soft tissue procedures such as the ones previously mentioned, are not indicated or do not provide successful outcomes, surgical interventions such as osteotomies, bone fusions and joint replacements may be required to attained desired outcomes.

Current literature, which discusses orthopedic procedures in relation to CP, categorizes the information either by surgical procedure, such as the one previously mentioned, or by anatomical location including the hip, knee and ankle. This discussion will categorize orthopedic surgical procedures by the specific anatomical location.

Due to the complexity of the hip and pelvis, numerous orthopedic procedures are used. These procedures include muscle transfers of the iliopsoas, adductors and proximal hamstrings, adductor tenotomies, pelvic and femoral osteotomies and resections, arthrodesis and arthroplasty. Fewer orthopedic procedures are used at the knee and ankle then at the pelvis and hip. Common procedures at the knee include hamstring lengthening and a muscle transfer of the rectus femoris to assist in knee flexion. At the ankle orthopedic procedures used include the lengthening of the gastrocnemius, Achilles tendon, peroneus brevis
and a combination of lengthening and/or transferring of either the anterior or posterior tibialis muscle groups.

**Hip and Pelvis**

The hip is an intricate component in an individual's functional ability to ambulate and maintain a sitting position. In spastic CP, complications at the hip can cause significant loss of function for a child. Surgical orthopedic interventions for hip dislocations, subluxation and pelvic asymmetry in children with CP can be classified into three groups: tenotomies, myotomies and muscle-tendon transfers, arthroplasty and arthrodosis, and osteotomies. Each of the three groups will be investigated individually within this section. Soft tissue procedures such as tenotomies, myotomies and muscle transfers are commonly practiced as surgical options for children with CP.

The most common type of orthopedic condition at the hip is subluxation, which progresses to dislocation and is caused primarily by spasticity of hip and pelvic musculature. In child either CP muscle imbalance or joint contractures are the bases for lateral migration of the hips. This condition presents as mild pain and discomfort while in a sitting position. Progressive lateral migration of the hip starts to produce more extensive gait deviations and if left untreated may lead to dislocation. Researchers have estimated it takes about six years from the time of initial hip migration until dislocation occurs in a child with CP. Due to numerous factors an exact timeline is not clinically used to make in any medical conclusions. Other causes of hip dislocation, in children with CP, include acetabular dysplasia, fetal femoral geometry and flexion-adduction.
contractures. The primary surgical procedures, which will be discussed, include soft tissue procedures, joint replacement and osteotomies. Extensive amounts of research describing different aspects of soft tissue procedures are available throughout the literature. As mentioned previously, orthopedic surgical procedures have been used for many years to correct different orthopedic conditions. Issues which trouble orthopedic surgeons are, what, if any, specific clinical finds can be used as an indicator for performing any soft tissue procedure at the hip. Furthermore, what measures can be predictors for outcomes identified with soft tissue orthopedic procedures. A study by Cornell et al investigated if the migration index (MI) could assist in predicting future hip subluxations or dislocation following adductor tenotomies. Cornell's findings initially support changes in the MI of a child's hip could be a predictor of outcomes but warns that more research needs to be performed to account for all the variables. In a similar study Miller et al and Abel et al both concluded in separate studies the MI is a strong predictor for soft tissue surgical outcomes in controlled environments. Both of these authors also express the controlled environment of this study cannot account for many different variables.

In reviewing different studies, which have investigated and reported outcomes of different soft tissue orthopedic procedures for hip subluxation and dislocations in the treatment of CP, both favorable and unfavorable outcomes have been reported. Banks et al reported of forty-five children who underwent adductor myotomies, thirty percent of the surgical hips became clinically unstable within a year. Samilson et al in another study of forty children, who underwent
adductor tentononies reported that over thirty-five of the surgical hips became dislocated within two years following the procedure. Sherk et al reviewed charts of forty-five children, who had undergone different soft tissues procedures for subluxation or complete dislocation of the hips, and concluded that single soft tissue procedures alone are not successful. Surgical procedures and diagnostic instrumentation has improved significantly since most of these studies were published and therefore, may support an argument to the increased number of surgical failures.

More specifically, soft tissue procedures have been singled out for their lack of success when used by themselves. Recently, both Scott et al and Tucker et al investigated adductor soft tissue procedures in long term follow-up studies. Using gait analysis, Scott investigated thirty-three subjects after undergoing adductor transfers at an average of 9.6 years after the procedure. Functional improvements were identified by ninety-four percent of the patients. Significant abnormalities in pelvic obliquity were identified in eighty-five percent of the children, which led to a thirty-six percent increase in unilateral hip subluxation. Turker et al also concluded similar findings to that of Scott when performing a retrospective study of adductor tenotomies in ambulatory and non-ambulatory children with CP. Turker identified that following 113 children who underwent ninety-three adductor tenotomies, fifty-eight percent of the children continued to demonstrate increased subluxation as identified through MI values. The important aspect in reviewing the negative outcome of soft tissue procedures is to note that each one was a single technique. Using combinations of soft tissue
procedures have tended to demonstrate significant improvements in function following surgery.

Significantly more favorable responses were identified in the literature, primarily when combinations of surgical soft tissue procedures were used. A study by Uematsu et al investigated the effectiveness of an iliopsoas transfer in relation to hip subluxations in children with CP. Uematsu reported unfavorable results when only the iliopsoas transfer was performed. Favorable results were reported when combined with pelvic or femoral osteotomies, therefore supporting that single soft tissue procedure might not be indicated. In another study Sharrard et al reported that out of twenty-five iliopsoas transfers coupled with adductor released in children with subluxed hip only on joint became unstable. Joint instability was caused by detachment of iliopsoas in which surgery corrected the problem. Onimus et al reported significantly favorable responses in decreased hip migration when iliopsoas transfers and adductor tenotomies were combined. Onimus also reported that children under the age of four had the best results and recommend this procedure to be performed on children between two and three years of age.

Aronson et al investigated forty-two children with spastic CP who underwent only a posterior adductor transfer or a combination of the transfer and the iliopsoas transfer. After an average of 5.7 years following the procedure, both groups of children demonstrated improvements in gait and hip range of motion, but a greater amount of improvements were observed when both procedures were performed. In a study by Cottalorda et al investigated fifty-seven hip in
thirty children who had undergone either an adductor tenotomies alone or in combination with anterior obturator neurectomy. Results of this study also supported the use adductor tenotomies in combination to anterior obturator neurectomies but cautioned of secondary peripheral nerve complications. In using a soft tissue lengthening procedure, Deluca et al investigated the efficacy in other soft tissue procedures in children with spastic CP when combined with psoas lengthening in the prevention of pelvic tilt. Psoas lengthening in combination with medial and lateral hamstring lengthening were the only surgical combinations, which reduced excessive anterior pelvic tilt.

In reviewing the literature, numerous authors caution against performing soft tissue procedures on a unilateral hip. Gage et al and Silver et al both urge soft tissue releases should be avoided because of the abnormal biomechanical stress on the opposite hip. Abel et al also reports other complications which may arise due to soft tissue procedures. The key problem identified by Abel was over-lengthening which may result in reduced muscle force generation, new lower extremity deformities, crouch or stiff knee gait and over activity of antagonist muscle to the surgically lengthened muscles.

Total hip arthroplasty (THA) is another surgical procedure used to correct orthopedic deformities in individuals with spastic CP. Degenerative hip disease is most commonly diagnosed in non-ambulatory individuals with CP. The primary functional goals for THA include improvements in sitting time and tolerance, caregiver satisfaction, and decreased pain. Many surgeons will seek
other surgical and treatment options before THA because of the limited longevity of hip joint replacement.\textsuperscript{222}

Two different studies investigated the longevity of THA. A study by Buly et al investigated the longevity of THA in eighteen patients with nineteen THAs performed and reported a ninety-five percent success at ten years for dislocation and eighty-six percent rate for surgical revision.\textsuperscript{223} Weber et al reports that patient under the age of forty a reasonable longevity of the prosthetic can be expected.\textsuperscript{224} Another complication with THA is the risk of post surgical dislocation. THA protocols vary between surgeons and are established to help decrease the change of hip after surgery is still susceptible to dislocation.\textsuperscript{224}

In reviewing the literature, numerous studies identify favorable outcomes when THA is used to increase function and decrease pain in individuals with CP. McCarthy et al reported that in thirty-four patients, fifty-six surgical hips, at a two-year checkup, following replacement, improvements of function were sustained with no secondary surgical complications occurring.\textsuperscript{225} In another study Root et al reported that thirteen of fifteen patients receiving unilateral THA were pain-free six weeks following surgery.\textsuperscript{226} At five years post-replacement all of the patients were still pain free and subjective reports by caregivers identified a significantly higher functional level then before surgery. Consistent reports of THA performed on children with CP are decrease in pain, increased range of motion, increased caregiver satisfaction and decreased pelvic migration.\textsuperscript{226}

Other procedures used in the reduction of dislocated hips are femoral and pelvic osteotomies. Hip instability is caused by the excessive activity of spastic
muscles, which cause deformities in the hip joint such as excessive femoral anteversion or varus rotation and pelvic asymmetry. Osteotomies are surgical procedures used to correct bone deformities at the pelvis and femoral head to correct abnormal alignment in the lower extremities. Unlike soft tissue procedures, femoral and pelvic osteotomies involve significant amounts of bone reconstruction to attain the desired outcome. Possible complications in osteotomies include decubitus ulcers due to post-surgical casting, fractures at the surgical site and infections.\textsuperscript{327,328}

Significant research supports femoral neck osteotomies as a surgical treatment for subluxed or dislocated hips. Femoral osteotomies are normally performed with secondary soft tissue procedures such as hamstring or adductor releases. Atar et al identified through retrospective surgical reports that only nine of forty-four hips, which received femoral head osteotomies and muscle releases dislocated and required further surgery.\textsuperscript{329} Atar also reported that in sixty-four surgical hips where innominate and femoral head osteotomies were performed with muscle releases, no hip instability was identified.\textsuperscript{329} Settcererri et al also reported favorable results when femoral osteotomies without soft tissue procedures were performed in non-ambulatory children with spastic quadriplegic CP.\textsuperscript{330}

In surgical osteotomies different types of procedures require specific internal hardware components or external fixtures commonly used to correct long bone deformities. Moens et al reported on twenty-four femoral osteotomies, which used a modified Ilizarov frame instead of internal hardware.\textsuperscript{331} Advantages in
using the Ilizarov frame to support the osteotomy included minimal scarring, early ambulation, and good hip rotational control. 331 Most recently Hau et al investigated two leading internal hardware devices used in femoral osteotomies. 332 Favorable outcomes such as improvements in gait and joint stability were attained using both devices as implants for femoral osteotomies with a minimal amount of complications and surgical revisions. 332

In the pelvis and acetabulum numerous types of surgical osteotomies exist. In the surgical treatment of pelvic and hip abnormalities, seen in children with CP, the literature tends to identify the Chiari osteotomies as the primary choice of orthopedic surgeons. 333 The Chiari is relatively simple in comparison to the Steel, Southerland and Eppright, which are significantly more complicated and require more recovery time. Two studies by Osterkamp et al and Graham et al both investigated the effectiveness of the Chiari procedure. 333, 334 Favorable outcomes were identified in both studies identified by significant improvements in gait and other functional improvements. 333, 334

In a study by Pope et al, comparisons were made between three different pelvic osteotomies: Steel, Chiari, and Salter. Pope did identify, through retrospective review of surgical records, the increased amount of surgical and rehabilitation requirements in the Steel and Salter osteotomies. 335 The study concluded that all three types provided improvements in hip stability in patients with CP, but recommended the Chiari for older children. 335 Recently Brunner, who also investigated pelvic and femoral osteotomies and which would produce the most favorable outcomes, conducted a study and found results that apposed
those of Pope. Overall the most favorable osteotomies identified by Brunner were the Pemberton, a modified version of the Chiari procedure. Brunner provided evidence demonstrating that when any of the previously mentioned osteotomies were performed with an iliopsoas muscle transfer, improvements in gait and functional activity occurred.

Knee

Limitations in knee flexion, a common deformity is often associated with spasticity and/or shortened hamstrings or contractions of the quadriceps and hamstrings. Limitations in knee flexion also may be caused by secondary problems identified in hip and pelvic abnormalities. Persistent knee flexion can lead to such common problems as knee joint contractures and shortening of the sciatic nerve. The more commonly used surgical procedures to correct knee flexion abnormalities are hamstring lengthening or releases, rectus femoris transfers or releases or a combination.

Due to the more simplistic design of the knee joint in comparison to the pelvis and ankle some general indications for orthopedic intervention have been established. These include persistent knee flexion of twenty to thirty degrees knee flexion during stance phase, a popliteal angle greater than forty degrees, and hamstring over-activity with persistent knee flexion in terminal swing. Common surgical goals for children with knee flexion deformities include decreasing the amount of inefficiency due to a crouch gait pattern, increasing stride length, decrease compensatory ankle equinus and hip flexion, and
improving sitting balance and posture while decrease likeliness of hip dislocations.  

The most commonly discussed procedures in the literature are hamstring lengthening combined with rectus femoris transfers. These combinations of surgical procedures are commonly used to correct knee flexion abnormalities caused by increased lower extremity spasticity as presented in spastic CP. Thometz et al, in a study, marked limitations in sagittal knee plane flexion and increased co-contraction activity of the hamstrings and quadriceps were noted. Gage et al identified that lengthening the hamstrings usually improved knee extension during stance, but limitations still existed in knee flexion during swing. 

In a control study Gage identified that improvements in knee flexion occurred when hamstring and rectus femoris transfers were used in combination. Based on biomechanical considerations, a study by Ounpuu et al questioned the effect of rectus femoris transfers in relation to one location of surgical attachment. In evaluating different insertion sites in seventy-eight children diagnosed with spastic CP, who underwent the rectus femoris transfer surgery, Ounpuu concluded that different insertion locations did not provide any statically improved function.  

Strengthening is critical to maintain the new range, which has been gained through muscle lengthening and transfers. Damiano et al investigated the relationship of strength and its effect on gait function following hamstring lengthening. In a controlled study Damiano determined that although hamstring surgery produced immediate improvements in knee range of motion and gait
dynamics, strength changes were not evident until later during the recovery period.  

In children with stiff-knee gait patterns caused by spastic CP, Miller et al attempted to predict preoperative outcomes based on the EMG activity of the rectus femoris prior to transfer surgery. The studied demonstrated significant improvements in functional gait and active range of motion within the previous stiff knee, but most importantly identified that preoperative EMG activity is very useful in predicting surgical outcomes.

Another procedure used in moderate to severe cases of knee flexion deformity is tenotomies of the distal end of the hamstring or rectus femoris. Most orthopedic surgeons tend to use tenotomies as a final option in children with chronic knee flexion deformities. Within the literature few studies address the efficacy of either hamstring or rectus femoris muscle releases.

Damron et al performed a retrospective analysis of children with spastic CP who underwent hamstring tenotomies. One hundred and seventeen children were followed after receiving hamstring tenotomies for an average of 3.4 years. Evidence of significant improvements in knee range of motion occurring in under one year, with sustained improvements for up to four years lead Damron to conclude that hamstring tenotomies are a viable option in correcting knee flexion deformities. In two other studies researchers investigated the effectiveness between rectus femoris transfers and releases. Sutherland et al identified improvements during swing phase in peak knee flexion of 9.1 degrees in children who underwent rectus femoris releases and 16.2 degrees in children who

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underwent rectus femoris transfers. Sutherland concluded that sufficient evidence existed to support using rectus femoris transfers instead of total release to improve knee flexion deformities seen in children with spastic CP. Ounpuu et al also investigated the different effects which rectus femoris transfers and releases have on knee flexion abnormalities in children with spastic CP. Ounpuu study identified similar results as Sutherland’s in that rectus femoris transfers produce the most clinically significant improvement in swing phase, then when the rectus femoris is released.

Ankle and Foot

At the ankle and foot common orthopedic conditions can be grouped into equinus and varus deformities and pes valgus. The most common of these conditions are the equinus deformities of the foot. Equinus deformities at the foot in children with CP are primarily caused by muscle imbalance between the plantar and dorsi flexor muscle groups. A child with an equinus deformity commonly presents as a toe-walker. More severe cases of equinus deformities may significantly hinder functional tasks such as transfers. The most commonly used orthopedic surgical technique to reduce the amount of equinus seen in a child with CP is Achilles tendon lengthening or Z-lengthening. Another surgical intervention is the Vulpius procedure, which involves incisions into the fascia of the gastrocnemius instead of the Achilles.

Achilles lengthening is a procedure that has been the mainstay treatment of equinus deformities commonly seen in children with CP. Numerous studies conclude that lengthening the Achilles tendon produces favorable outcomes.
In a study by Darmon et al lengthening of the Achilles tendon to correct equinus deformities resulted in significant (p<0.0001) initial gain in dorsiflexion when compared to the baseline. The improvements initially identified in ankle dorsiflexion, as a result of the surgical lengthening, maintained statistically significant for seven years postoperatively. In another study by Etnyre et al pre and postoperative evaluation of EMG activity, lower extremity range of motion and dynamic ankle motion resulted in significant improvements in all three variables.

Other studies have identified there also is a change of reoccurrence of an equinus deformity after Achilles lengthening. Sala et al recalled twenty-seven children ranging between two to nine years following Achilles lengthening to correct equinus deformity, and after evaluation of the children, the reoccurrence rate of equinus deformities was identified as twenty-two percent. Sala evaluated numerous clinical characteristics and concluded that increasing hamstring contractures was the primary factor influencing recurrence of equinus deformity. Orthopedic surgeons also are challenged by the exact age of a child, which will provide the best outcome for the procedure. In a retrospective review, Rattey et al evaluated the reoccurrence of equinus deformity in hemiplegic and diplegic children who underwent Achilles lengthening. Rattey identified that children, six years of age or greater, and in both categorizes at the time of the procedure had a significantly less chance of equinus deformity reoccurrence than children under the age of six.
Significant concerns have been expressed in literature about the over-lengthening of the gastrocnemius and soleus complex in the achilles lengthening procedure. A newer intervention called the Vulpius procedure is being used as a surgical option for the correction of equinus deformity. The Vulpius differs from the Achilles procedure in that part of the fascia surrounding the gastrocnemies is severed instead of part of the Achilles tendon. In a comparison study by Yngve et al, the Achilles lengthening and the Vulpius procedure were evaluated using gait analysis. Both surgical procedures showed overall moderate improvements in gait parameters and efficiency. The Vulpius group consistently demonstrated abnormal mid-stance work while the achilles lengthening group demonstrated a lack of push off in preparation for swing.

Pes valgus is another orthopedic condition occurring at the foot but is not as common as an equinus deformity. Eversion, plantar flexion and abduction of the forefoot produce a prominence of the talus, characterized as pes valgus. This deformity is surgically corrected by reducing the sub-talar joint and forefoot to a neutral position while the ankle is plantar flexed. Orthopedic surgeons have identified three contributing factors, which may lead to the pes valgus deformity. First is a change in the axial rotation of the sub-talar joint, which is being caused by spastic peroneal muscles. Second is that a pes valgus deformity may be caused by contractures of the gastrocnemius and soleus muscle groups, which result in plantar flexion of the calcaneous. Lastly is by a continuous abnormal fetal developmental, medial deviation of the talus.
Four orthopedic procedures are identified in the literature to correct pes valgus deformities. These procedures include the Grice extra-articular sub-talar arthrodesis, staple arthroeriesis, triple arthrodesis and Grice-Schede procedure. All four of these procedures are based on surgical arthodesis of the ankle with different variations in each procedure based on the deformity of the ankle and foot. 363-367

In reviewing the literature, very few articles currently address the efficacy and validity of the previously mentioned procedures. Of the results reported, only fair outcomes are identified in the different procedures. Most of the studies also suggest that the chances of future surgeries to correct further deformities are likely. Also, some studies agree that performing pes valgus correction surgery might lead to further complications due to growth factors and the complexity of the ankle. Most studies agree that surgical consideration for correction of pes valgus deformity should not be performed until after ten years of age. 363-367

Varus deformities are the least common orthopedic condition seen in children with CP. Much like the equinus, the varus deformity is caused by an imbalance of weak peroneal muscles and spastic anterior or posterior tibial muscles. The most common surgical procedures used to correct varus deformities include the lengthening or splitting of and the transfer of either the anterior or poster tibialis muscle. Within the literature a substantial amount of evidence exists, which supports the use of anterior and posterior tibialis transfers. 362, 366-370

In the anterior tibial transfer procedures, a secondary lengthening of the posterior tibial tendon occurs to create symmetry of the surgical ankle. Results
of a 6.2-year follow-up study by Barnes et al identified that in twenty-two surgical procedures where split anterior tibialis procedures were performed, along with posterior tibial lengthening, eighteen of twenty patients had significant improvement in gait.\textsuperscript{371} Vogt et al investigated long term results of children receiving only the split anterior tibial transfer surgery, and also identified significant improvements in functional gait patterns and a decreased need to use orthotic devices.\textsuperscript{372} Vogt identified that more research needs to be conducted to determine if lengthening the posterior tibialis is always necessary in split tibialis anterior transfers.\textsuperscript{372}

Another surgical option for varus deformities at the foot is split posterior tibialis transfer. This procedure differs from the anterior tibialis transfer in that no lengthening of the opposing muscles are necessary. Studies by Kagaya et al and Muller et al both reported marked improvements of equines foot deformities, which were being caused by spasticity and also in functional ambulation.\textsuperscript{373, 374} Kagaya also concluded the split posterior tibialis transfer procedure could be a surgical consideration in a child diagnosed with athetoid CP.\textsuperscript{373}

Similar to neurological surgical procedures, orthopedic surgical procedures are very common in the management of spasticity in CP. The research discussed in this section does support the efficacy and validity of orthopedic surgery as viable tool in the treatment of spastic CP.
Conventional Rehabilitation Services

This section of this literature review will discuss treatment techniques commonly used in rehabilitative medicine. These treatment techniques are the more commonly used evidence based treatments for children with CP. The discussion will begin with a review of motor control and some of the more common theories of motor control. Next a connection will be made between motor control theories and current common neurofacilitation treatment techniques used in children with CP such as neurodevelopmental treatment and sensory integration. This section will then conclude by reviewing conventional treatment options not directly linked to motor control theories such as strength and exercise training, serial casting, electrical stimulation and orthotics.

Motor Control Theories

The next section will discuss therapy based rehabilitation treatment for children with CP. Some of these therapeutic treatments parallel different theories of motor control. Therefore, to fully understand these treatments to include components of the Adeli treatment a review of different theories of motor control is required.

Motor control is defined as the ability of the CNS to regulate and or direct the musculoskeletal system in purposeful acts. Motor control involves such mechanisms as sensory stimulation and motor functions. There are many different theories of motor control but for purposes of this case report only five will be discussed. These theories include reflex, hierarchical, motor programming, systems, and dynamics action theories.
Reflex Theory

One of the first proposed theories on motor control was the reflex theory. Originally, researchers believed that reflexes worked together and or in sequence to perform functional movement. The reflex theory is based on a stimulus, which provokes a response in a target organ such as skeletal muscle. This stimulus-provoked response in turn can lead to many other reflex responses in a chain effect. Stimulus of a receptor, such as a stretched muscle spindle, passes through the CNS and promotes a response such as a muscle contraction. Therefore, according to this theory all movements require some form of sensory stimulus within a closed loop system to generate movements.\textsuperscript{375-378}

Current understanding of the CNS questions the reflex theory for a couple of reasons. First, movement can be initiated without sensory input. The reflex theory cannot explain how functional movement occurs without sensory stimulation. Second, cognitive development and motor learning is not considered in this theory. Through practice an individual can learn how to perform functional movements without sensory input such as the reflex theory supports.\textsuperscript{375-378}

Even though the tenets of reflex theory of motor control are not fully applicable as a stand-alone treatment, it is important to discuss the clinical implications to the theory. Initially the therapist would focus their evaluation on what reflexes were either present or absent based on movement behavior and how they effected movement of a patient. An example would be developmental reflexes in young child. The treatment would then focus on retraining motor control in functional tasks based on enhancing or reducing the effect of specific
reflex activity. By altering reflex activity more favorable functional outcomes may occur.\textsuperscript{375-378}

Hierarchical Theory

The hierarchical theory of motor control is characterized by a top-down structural organization. The basis of this theory is that the CNS is organized so that the higher centers of the brain control and influence the lower centers in a rigid vertical hierarchy. Also, in a hierarchical theory of motor control lines of control do not cross nor is there a bottom up means of control. Due to this lack of bottom up control and ability to provided feedback to the CNS, the hierarchical theory is viewed as an open loop system.\textsuperscript{375,376,379,380}

There are a couple of limitations to the hierarchical theory. First, is that the hierarchical model is too simple and rigid when compared to the vast amount of tasks controlled by the CNS. Also, this theory does not address reflexes within a normal CNS such as a knee jerk reflex. Another limitation is the unexplained role of sensory input into the CNS. It is theorized that once the movement has been learned it is can be performed without the need for peripheral feedback. But if every possible movement an individual may make is stored centrally within the higher functioning centers of the CNS what is the use of sensory feedback?

The clinical implications for the hierarchical model include the foundation of neurofacilitation treatment approaches. Such treatments include Rood’s approach to neuromuscular dysfunction, neurodevelopmental therapy (NDT), Brunnstrom’s stroke rehabilitation and proprioceptive neuromuscular facilitation (PNF). Treatment programs based on the hierarchical theory of motor control
focus primarily on inhibiting abnormal reflexes while promoting righting and equilibrium reactions of the patient. It is believed that an increase of function may occur by altering reflex activity.\textsuperscript{375,376,379,380}

The more current views of motor control have shifted the focus from the concept of the CNS being reactive and instead have begun investigating what causes a specific action. One of the newer theories of motor control to explore the physiology of movement is the motor program theory. The concept of this theory is based around a central motor pattern or program, which is stored within the CNS. What separates the previously discussed theories of motor control to the motor program theory is that movement can be activated either through stimulus input or by a stored program.\textsuperscript{375,376,379,380}

**Motor Program Theory**

The motor program theory is based on two different levels of central programming. The higher-level programs within this theory are known as a general motor program or GMP. The GMP program controls abstract movement within the higher levels of the CNS. This control of movement is similar to the hierarchical theory except the motor program theory allows for sensory input and therefore is not strictly a top down model. The central pattern generator or CPG is the second level of movement control based on the motor control theory. Unlike the abstract controls of the GMP, the CPG controls very specific aspects of movement such as walking. Within the last decade an extensive amount of research has been published which investigates the theory of CPGs.\textsuperscript{383-389} The
CPGs within the CNS are similar to the wiring of a circuit board with the only difference being that the CPGs are modifiable by sensory input.\textsuperscript{375,376,379,380}

Also within the motor program theory of motor control two separate secondary theories exist which are identified as closed and open loops. The closed loop theory of motor programming suggests that all movements are controlled by sensory feedback into the CNS. The sensory feedback will allow continued movement and also make any corrections to the movement as needed. Major limitations to the closed loop theory include how movement is initiated if sensory feedback is always required. Another limitation is the explanation of how fast reactionary moves occur when sensory feedback is unable to meet the requirements of speed.\textsuperscript{375,376,379,380}

The open loop theory of motor programming provides a more acceptable explanation for movement based on the understanding of the CNS and motor control. The open loop theory states that centrally generated programs are stored within the CNS until the initiation of movement occurs. To initiate movement sensory feedback is not required. Once movement is initiated then sensory feedback can allow any modification of a specific movement to occur. Therefore, the open loop theory allows for initial movement while any corrective input from the CNS involves components of the closed loop theory.\textsuperscript{375,376,379,380}

Numerous clinical implications results from the motor program theory of motor control. The motor program theory allows a therapist to establish a treatment program, which focuses on functional movements such as reaching for a glass of water versus activities to just move muscle groups. From a clinical perspective
the motor program theory also promotes the importance of practice in order to develop or fine-tune damage within the CNS and therefore also increase the capability for feed-forward control. 375,376,379,380

Systems Theory

The systems theory of motor control identifies the body as a mechanical system, which can be subjected to internal and external forces. The system theory also proposes that the responsibility of movement is not isolated to one component of the CNS but instead movement is shared amongst many interacting systems such as internal and external forces acting on the body. 375,376,390,391

An important component of the systems theory is the understanding that multiple degrees of freedom occur within each individual movement. Due to the multitude of possible movements it is likely that overlapping degrees of freedom may occur. In order to eliminate redundant degrees of freedom and simplify movement, the systems theory hypothesized that a hierarchical control exists within the overall system. By grouping degrees of freedom into synergies the CNS is able to more easily manage the movements of the body. This will simplify the controls of movement and could likely increase functional movement.

The clinical implications with the systems theory of motor control are two-fold. First, when conducting an initial evaluation of a patient with CNS dysfunction it is important to identify and assess the body as a mechanical system that can be influenced by both internal and external forces. This is to ensure that all the systems, which help promote movement, are also properly assessed. Second, is
that treatment needs to be based on promoting active motor control in order to improve functional activities of an individual. An example would be again reaching for a glass of water. When setting up a treatment based on the systems theory the functional activity is picking up the glass. The other considerations would include the size of the glass, amount of fluid in the glass and use of the glass such as to drink from or just move. The system model accounts for all of these variables in relation to function. The use of functional training supports the systems theory, which is to identify and include all the systems of the entire body into treatment. 375,376,390,391

Dynamic Action Theory

The dynamic action theory of motor control suggests that movement emerges from cooperation among many subsystems such as environmental and behavioral aspects within a task specific context. The ability for the CNS to self-organize is a basic principle of the dynamic system model. Self-organization allows individual parts of a system to collectively work together in an effective manner in order to complete the task. Unlike the system theory of motor control, the dynamic system theory does not support the concept that a hierarchical program is required to initiate or control a given task. 375,392-394

Another unique perspective to the dynamic system is how discrete changes in movement occur. The dynamic system theorizes that a change in movement occurs when one or more of the components of the system undergo a critical change. When the system undergoes a critical change, which regulates the behavior of the entire system, it is identified as a control parameter. By
identifying control parameters, the dynamic action theory focuses on physical explanations contributing to movement instead of commands from CNS. 375,392-394

One of the major limitations to the dynamic action theory is one may perceive that the CNS has a limited role in movement when compared to the physical system of an individual and the environment. It is important to understand the dynamic action theory is based on the CNS contributions to movement but the interface of many different systems with the CNS. 375,392-394

Within the dynamic action theory one of the key clinical implications is how movement is emergent from different systems. What this means is that through the self-organization of input from multiple components dynamic movements emerge allowing changes in movement to occur. By understanding the basics physical and dynamic properties of the body, one could develop a treatment plan to help regain motor control and improve functional activities. 375,392-394

Neurological Rehabilitation Models

Within the literature numerous theories of motor control exist. Clinicians have used these scientific theories of motor control and developed them into different types of neurological rehabilitation treatments. Gordon et al has identified that grouped motor control theories and clinical practices parallel each other. 398 Gordon grouped the motor control theories into three main models; reflex, hierarchical or systems. 398 From these three motor control models three neurological rehabilitation models have been developed in parallel to the reflect different components of the motor control theory. The neurological rehabilitation models are muscle reeducation, neurofacilitation and contemporary task
oriented. Only the neurofacilitation and contemporary task oriented are
commonly used and will be the focus of discussion in this section.\textsuperscript{395-397}

The neurofacilitation approach of rehabilitation is based primarily on the reflex
and hierarchical models of motor control. Some of the more commonly used
neurofacilitation based treatments include, neurodevelopmental training (NDT),
Brunnstrom's approach, Rood approach to neuromuscular dysfunction,
proprioceptive neuromuscular facilitation (PNF) and sensory integration (SI). For
children with CP the two most common neurofacilitation based treatments are
NDT and SI. Both of these treatments will be discussed in further detail in the
next section.\textsuperscript{395-398}

A newer approach of neurological rehabilitation is the task-oriented model.
This treatment option is based on components of all three grouped motor control
models. The task-oriented model identifies that movement emerges from many
systems, which affect the body. Unlike neurofacilitation approach, the task-
oriented approach focuses on identification of functional task versus just
movement patterns. Another key component of task-oriented approach is to
teach motor problem solving skills and effective compensation in functional tasks.
Neurofacilitation and task-oriented are both commonly used treatment options in
neurological rehabilitation. For many years though NDT and SI have been the
foundation of treatment for children with CP. By incorporating concepts of
neurofacilitation into the task-orientated model greater improvements may
occurring in the functional activity of a child with CP.\textsuperscript{395-398}
Neurodevelopmental Training

Neurodevelopmental training or NDT was developed in the 1940's by Dr. and Mrs. Bobath to help promote gross motor skills, balance and quality of movement in patients with movement disorders. The treatment techniques of NDT were initially designed and are commonly used in the treatment of children with CP. The NDT theory of treatment is based on the premise that abnormal movements result from neurological deficits within the CNS. Numerous factors can contribute to these abnormal movement patterns such as primitive reflexes, abnormal muscle tone, poorly developed righting and equilibrium responses, sensory deficits and muscle strength.

The NDT treatment framework is centered on the ability of a therapist to promote and or facilitate normal movement patterns in a patient with known CNS damage. By promoting normal movements in individuals with CNS damage, compensatory movement patterns will not develop or progress and therefore hinder functional movement. This is especially important in children due to the limited amount of motor learning, which has occurred. 225, 400-404

NDT treatment uses specific handling techniques directed towards the components of movement that are causing the impaired motor performance. Commonly impaired movements that NDT was designed to treat include postural alignment and stability, mobility skills, weight bearing, weight shifting and balance. The foundation of NDT treatment is based on a couple key points. First, is that treatment of abnormal movements focuses on a combination of facilitation and inhibition techniques based on sensory input by proprioceptive...
cues. Second, is that normal movement patterns are guided through key points on the patient's body. This specific handling will also allow the therapist to inhibit any abnormal movement patterns. Third, is that NDT treatment promotes movement from a proximal position in order to gain stability of a distal component. Common treatment goals for NDT include the reduction and normalization of muscle tone, the integration of postural reactions the facilitation of active adaptive posture and movement.

Although NDT treatment is widely used and accepted by clinicians as a viable and effective treatment for neurological patient, little outcomes evidence exists within the literature due to the challenge of recording objective changes. The studies that do exist focus towards both lower and upper extremity changes in functional performance. In a study by Fetters et al, eight children with spastic CP were treated using both NDT and practice techniques for five days to evaluate any changes in reaching abilities to include kinematic variables such as time, velocity and acceleration. Fetters concluded that with each treatment being used individual treatment goals were likely not to be met. But if the two techniques were combined a favorable attainment of goals may be achieved. This study was severely limited due to the number of patients, duration of treatment time and the standardized level of care.

In a study by Jonsdottir et al the effectiveness of NDT was evaluated by assessing improvements in postural control. The subjects were eight children diagnosed with spastic CP. Similar to the previous study, the children participated in treatments, which consisted of both NDT and practice. Changes
in the children’s posture were assessed using videography and kinematic analysis. The data demonstrated significant improvements in postural control in the NDT treatment group when compared to practice and also in functional reach activities therefore supporting the use of NDT in the treatment of CP.  

Another study by Adams et al investigated gait changes in children with spastic CP following NDT treatment. The study was a quasi-experimental design of forty ambulatory children diagnosed with spastic CP underwent a six-week NDT treatment program. The children were evaluated before and after the treatment on gait characteristics that included step length, foot angle cadence and velocity. The results demonstrated that significant improvements occurred in step length, foot angle and velocity and therefore, lead Adams to conclude that NDT was an effective treatment to improve functional ambulation. The major limitation to this study is the lack of a control group.  

Evidence also exists within the literature that does not support the efficacy of NDT treatment. Law et al used a combination of NDT and serial casting to evaluate improvement in hand function and upper extremity usage in a child with spastic CP. In a randomized crossover designed study NDT and serial casting was compared to an intensive regular OT treatment program. The study identified that there was no significant changes in outcomes between the two treatment groups. Therefore, concluding that NDT and serial casting may not be beneficial in reducing upper extremity spasticity to improve function.  

In reviewing the literature the evidence supports that the use of NDT is effective in the treatment of children with spastic CP. The literature indirectly
suggests that the best treatment option is to incorporate the basic handling techniques of NDT with functional activities. Example would be to facilitate postural control using NDT handling techniques while having a patient reach. This allows for proximal stabilization of the trunk with a functional task such as reaching. The treatment can incorporate continuous practice to ensure mastery of the specific skill.

**Sensory Integration**

Sensory integration or SI is a treatment which focuses on sensory impact and how afferent input to the CNS may alter a child's motivation, attention, movement and social / emotional well being. Treatment principles of SI include using different controlled sensory input to encourage appropriate adaptive responses. SI also provides encouragement for the child to use intrinsic motivation and to promote appropriate and purposeful behaviors. The primary therapeutic use for SI is for the treatment of children with learning disorders, attention deficits and autism but also has demonstrated positive outcomes in children with CP.\(^{408}\)

Most children with CP experience difficulty in the adequate processing of sensory input as the stimulus relates to their surrounding environment. This inability to process sensory input hinders appropriate motor outcomes needed for functional activity. The treatment outcomes are most favorable when SI is not used as a single therapy but in combination with a secondary motor based therapy like NDT. Some pediatric therapists, both physical and occupational therapists, use a combined aspect of both SI and NDT principles in treating children with CP. By combining NDT and SI principles therapists are given more
treatment options to attain specific goals and to improve functional outcomes of children with CP.\textsuperscript{408}

Only a few studies within the literature investigate the efficacy of SI as a primary treatment for children with CP. In a study by Bumin et al, 41 children diagnosed with spastic CP were randomized into three treatment groups.\textsuperscript{409} Children within the first two treatment groups received sensory-perceptual-motor training either in a individual or a group setting. The third group was a control group, which consisted of the children continuing with their current home program. Children in all three treatment groups were evaluated before and after receiving treatment using the Ayres Southern California Sensory Integration Test and Physical Ability Test. Bumin reported the results of this study demonstrated that children treated using the sensory-perceptual-motor scored significantly higher when compared to control.\textsuperscript{409}

Other studies pertaining to SI within the literature are very similar in design and results.\textsuperscript{410-411} Similar to NDT, SI treatment lacks controlled studies that investigate the efficacy of treatment in children with CP. The lack of SI research is likely due to the significant amount of variables, subjectivity of the results and the difficulty in designing a study to specifically measure therapeutic outcomes.\textsuperscript{408}

Exercise and Strengthening

Therapeutic exercise is an important component in the development of a treatment plan for a child with CP. For many decades strengthening exercises have not been incorporated into treatment programs for children with CP due to
limited functional gains. Unfortunately, these limited gains in children with CP were being compared to patients with polio. Currently, a significant amount of evidence has been published within the literature that not only identifies that children with CP have muscle weakness but also that therapeutic exercises helped to improve gait and motor function.

In developing the exercise component of a treatment plan, activities need to be specific for strength and selectivity deficits as identified though the initial evaluation and also directed towards short and long term goals for the child. Three common deficits are the basis for exercise training in children with CP and include strength, endurance, and coordination. A key component to increasing strength is to progress the child from gravity eliminated positions to working against gravity. The disassociation and differentiation of opposite limbs, joints and muscle are also important in developing strength, control and coordination.

The literature investigates a variety of different aspects of strength training in relation to motor control, improved gait and energy demands. Different means of evaluating functional improvement include three-dimensional gait analysis; energy expenditure index (EEI) and gross motor functional measure (GMFM). Parker et al disagreed with the use of GMFM to evaluate changes in strength and the correlation to functional outcomes. In a control study Parker investigated the use of the GMFM and determined that it is not specifically designed to measure changes in anaerobic power in relation to exercise training in children with CP. Three-dimensional gait analysis and EEI have been
used to assess improvements in gait function which correlate to decreased energy demands of the child. Gage has identified that the reduction of co-spicity, restoration of stance phase stability at the hip and knee, and the elimination of foot drag will decrease the amount of energy used by a child with CP. In using three-dimensional analysis, changes in the biomechanics of a child's gait can be monitored. EEI is another easily used test, which will provide researchers with information specifically related to changes in energy usage.

The literature discusses the relationship between spastic lower extremity strength and its carry over to improvements in gait. Researchers have identified that in a child with CP, the amount of energy required for functional ambulation is double or triple the normal requirements. Therefore, the importance of strength training to improve functional gait in a child with CP is essential. In a study by Damiano et al major lower extremity muscle groups in children with CP were specifically targeted for strengthening based on gait deviations caused by weakness. The study concluded that strength training in children with CP is effective in increasing muscle strength and also to improve gait deviations. Two other studies by Damiano also investigated how strength training in children with CP affected gait deviations and functional abilities. Both studies produced favorable outcomes and recommend that exercise training may play a critical component in improving common gait deviations as seen in children with CP. All three studies did stress that in the treatment of children with CP strengthening alone is not as beneficial then if combined with other types of physical therapy such as mobility and gait training.
Different studies have also investigated if exercise training or rehabilitation therapy has caused a change in the endurance on children with CP. In a study by Rothman et al significant improvements were reported in the vital capacity of children with CP. Rothman stated that children with limited mobility are less likely to experience pulmonary complication due to lack of exercise. A controlled study by Hutzler et al compared improvements in vital lung capacity in children with CP who participated in a convention physical therapy program and a water exercise program. Results of the study concluded the significant improvements occurred in the swimming class in comparison to the conventional physical therapy program.

Throughout the literature, studies support that strengthening targeted muscles of a child with CP will improve functional activities such as gait. Strength training will also decrease the amount of energy needed for functional activity. The literature may not agree on a specific type of exercise training for a child with CP but did agree that exercise training is an important component in the overall treatment plan.

Serial Casting

Serial Casting is another treatment technique used to decrease spasticity and therefore, improve functional activity of the lower extremity. The purpose of serial casting is to place the extremity into either an extended or flexed position providing an elongated stretch directly to the spastic muscles. Clinicians believe that the prolonged stretch provided by the casts provides a proprioceptive input into the CNS therefore inhibiting the spasticity. Serial casting also increases the...
length of the muscle while decreasing the sensitivity of the muscle to stretch. The construction of the casts is either plaster or fiberglass. The casting materials are available in a variety of colors especially for the younger children. Typical applications are a straight leg cast for limitations in knee extension due to tight hamstrings, ankle for excessive plantar flexion due to tight gastrocnemius muscle, or a combination of both. Application of the casts is extremely labor intensive and requires a minimum of two people. For a child who is already non-ambulatory adding casts to their lower extremities makes caring for them even more of a challenge due to the limitation of the casts. The child normally remains in the casts for five to seven days then the casts are removed for a couple of days and then the process is repeated. The repetitive casting occurs for three to four complete casting cycles.

Within the literature only a minimal amount of research is available which investigates the efficacy of serial casting in an controlled environment. In a retrospective study, Cottalorda et al investigated changes in toe walking children with spastic CP. In reviewing charts of 20 children with excessive plantarflexion whom underwent serial casting the authors of this study found that significant improvements of dorsiflexion had occurred. The authors concluded that serial casting is an efficacious alternative to a surgical procedure especially in younger children. Another study by Brouwer et al also investigated the effectiveness of serial casting in children with spastic CP when compared to idiopathic toe-walkers. The study consisted of 16 children, eight per group for
6 weeks of serial casting. Brouwer reported that moderate improvements were noted in both groups in increased active and passive dorsiflexion. No change occurred in gait velocity and stride length in either group. Brouwer concluded that serial casting produced positive outcomes in both treatment groups but that idiopathic toe walkers may have a longer carryover than children with CP. 433

Numerous other studies exist within the literature, which compare serial casting to other treatments such as Botox injections. 434-435 Even though positive outcomes are identified for all of the interventions, the most important issue is that of ease and comfort for the patient and family. Serial casting is a viable treatment option for children with spastic CP but is normally used secondarily to other treatment options.

Orthotics

The primary use of a lower extremity orthoses is to provide stability and control of the limb during ambulation and other functional activities. Factors to consider in assessing the need for an orthoses are available range, foot alignment, flexibility and voluntary control. In situations when an orthosis is used on a joint with contractures, the tendency of the child is to use compensatory actions to perform functional activities. These compensations will continue to cause hypermobility at the joints, which are compensating while the contractured joint remains hypomobile. 436-439

The uses of orthosis in children with CP are primarily the ankle-foot-orthosis or AFO. AFOs can be classified into three different types: Dynamic AFO (DAFO), Molded AFO (MAFO) and the Articulating AFO (AAFO). DAFOs are built with a
specially molded footplate in the bottom of the orthosis. Areas under the toes and arch of the foot are built up to position the sub-talar and mid-tarsal joints into a neutral position. This neutral position provides subsequent stability and support for the foot. The neutral position also helps to decrease abnormal tone in the foot while improving biomechanical alignment through the ankle, knee, hip, and pelvis. 436-439

MAFOs or solid AFOs are a rigid designed orthosis. Indications for the use of a MAFO in children with CP include a correctable equinus position, a nonstructural genu recurvatum during stance phase and pes valgus secondary to increased tone in the gastrocnemius. MAFO is most commonly used to decrease ankle plantar-flexion during the stance phase of ambulation. Straps across the front of the brace controls the excessive plantarfexion therefore keeping the foot in proper position. 436-439

The AAFO or hinged AFO is an orthosis with a built in hinge at the ankle. The use of an AAFO is indicated when a child with CP is able to control dorsiflexion but is unable to control plantarflexion. Biomechanical changes unique to the AAFO include increased ankle movement and increased lower extremity segmental symmetry during gait. Functionally the AAFO allow a child more mobility and control while ambulating and at play. Newer designed AAFO also allows for adjustments in PF stops to be made by the therapist with relative easy and accuracy. The AAFO can also be used as a night splint for children with excessively tight gastrocnemius muscles. By adjusting the AAFO to increase dorsiflexion a sustained stretch of the tight muscle occurs. 436-439
In reviewing the literature numerous studies investigated the validity of a variety of orthoses as treatment options for children with spastic CP. The results of these studies are predominantly favorable from improvements in range of motion to changes in a variety of gait parameters. Even though the literature demonstrates the efficacy of orthoses in children with CP the decision to use such a treatment lies within the rehabilitation team.

**Electrical Stimulation**

Currently there are two different treatment options discussed within the literature, which investigate electrical stimulation as a treatment option for children with CP. The two forms of electrical stimulation include therapeutic electrical stimulation or TES and neuromuscular electrical stimulation or NMES. This section will define treatment protocols for each type of electrical stimulation and discuss current research within the literature.

TES is a method by which low intensity electrical stimulation is given at nighttime. A Canadian physician, Dr. Pape in 1985, originally developed the TES technique in hopes of promoting the growth activity of antagonist muscle groups. TES differs from NMES in that it employs low intensity stimulation just above threshold and therefore muscle contractions do not occur. While asleep skeletal muscles are repaired and stimulated to regenerate by the release of trophic hormones which are carried in the blood stream. The proposed mechanism of action for TES is based on using electrical stimulation to increase blood flow to the antagonist spastic muscle therefore increasing muscle growth.
Within the literature there is a limited amount of research that investigates TES as a treatment option for children with CP. In a study by Sommerfelt et al. 12 children between the ages of 5 and 12 completed a 24-month study in which TES was applied to the antagonist muscles of the spastic lower extremities during a standardized physical therapy program. The children were randomized into two groups of six. Each group received TES either in the first or last 12 months of the study. All the children were assessed on changes in motor function based on GMFM and also by subjective assessments of the parents and healthcare providers. Objectively there were no significant changes identified in any of the children, however 11 of the 12 parents did report noticing considerable improvements in functional activities around the house following the TES treatment.

In a study by Steinbok et al. TES was used in children with spastic CP one year after SDR to determine if any functional improvements may occur. The primary outcome of the study was to measure any changes in the GMFM score after undergoing a one-year nighttime TES treatment. Following the one-year treatment timeframe statistically significant improvements in GMFM were reported in children who received the TES when compared to placebo. The study also identified that a high level of compliance with limited complications occurred with the TES treatment protocol. Steinbok concluded that the use of TES in conjunctions with SDR is a viable treatment options for children with spastic CP.
The second use of electrical stimulation is NMES. The use of NMES in children with CP has been investigated more extensively than TES. There are two different means by which NMES can be used in the treatment of children with CP. The first and more commonly used method is to stimulate the antagonist muscles or nerves. By stimulating the antagonist muscle or nerve it is proposed that an inhibitory effect occurs through the interneurons of the spinal cord and therefore reduces spasticity within the agonist muscle group. The second method uses intensified NMES directly on the spastic muscle. The mechanism of action for this form of stimulation is unknown but clinicians believe that when intense NMES is used on spastic muscles without interruptions excessive fatigue is produced. The excessive fatigue will then cause the reduction of spasticity due to the inadequate ability of the skeletal muscle to contract.

Within the literature, only two studies within the literature discuss NMES in children with CP in which the peroneal nerve was stimulated. A study by Gracanin et al. used NMES to the peroneal nerve in 120 children diagnosed with CP and identified improvements in movement patterns at both the hip and knee. In another study by Riso et al. NMES was used to stimulate the peroneal nerve of seven children diagnoses with CP. Even through a few minor improvements in ankle ROM occur, the result of Riso study did not show as favorable results compared to the outcomes of Gracanin.

Within the literature there are also studies, which investigate using NMES to stimulate the muscles of ankle dorsiflexion. In a study by Dubowitz et al. NMES
was applied to the tibialis anterior of two children with CP one to three hours a day. \(^{464}\) Dubowitz reported an increase in number and quality of voluntary contractions occurring within the stimulated muscle groups. Subjectively the parents reported improvements in functional activities and ambulation. \(^{464}\) In another study by Atwater NMES was used in conjunction with an NDT therapy program. \(^{465}\) Upper and lower extremities were evaluated for improvements in ROM and functional activity such as reaching and gait. Atwater reported that only minimal improvements occurred in three of the children's UE reaching and that no significant improvements occurred in gait and LE ROM. \(^{465}\)

In a study by Leyendeker et al a comparison was made between NDT and NDT plus NMES in 20 children with CP. \(^{466}\) The groups were divided into 10 children each; with one group receiving NDT and the second group receiving NDT plus NMES. Initially Leyendeker reported that functional improvements were identifiable in the NDT and NMES treatment group. Unfortunately once the study concluded there were only minimal improvements in both treatment groups. \(^{466}\)

Another method in which NMES has been used is in the stimulation of both the spastic and the antagonist muscle groups. A Case study by Carmick et al reported on three children diagnosed with CP that were treated using NMES in combination with dynamic-systems, task oriented model of motor learning. \(^{467}\) The most significant improvement occurred when both the gastrocnemius and the tibialis anterior were stimulated during ambulation. When the tibialis anterior alone was stimulated the children routinely began initial contact with a flat foot
and poor active ankle dorsiflexion. Once stimulation occurred during the gait cycle the children demonstrated an increase in active dorsiflexion and adequate heel strike occurred. \(^{467}\)

Comeaux et al also investigated the use of dual electrical stimulation to the ankle in a study of fourteen children with CP. \(^{468}\) The children underwent four four-week training phases that consisted of a pre and post treatment exercise program, gastrocnemius stimulation and gastrocnemius and tibialis anterior stimulation. During each of the treatment phases of the study video recordings were taken of each child and ankle dorsiflexion were measured. Comeaux reported that significant improvements in ankle dorsiflexion occurred in both stimulation groups but no difference occurred in improvement of ankle dorsiflexion between single and dual stimulation. \(^{468}\)

Current evidence within the literature supports the use of different forms of electrical stimulations in children with CP. What is lacking in the literature is any consistency on a standard treatment protocol such as indication for treatment and specific setting for stimulation units.

Non-Conventional Treatments

This section of the literature review will also discuss treatment techniques, which are not so commonly used in the treatment of children with CP. These treatment techniques tend to have a more limited amount of information compared to the previous section.
Hyperbaric Oxygen Treatment

Hyperbaric oxygen treatment or HBOT is a newer experimental treatment being used to manage CP. HBOT has been used in the medical community since 1879 as a method of anesthesia for surgical patients. In 1976 the FDA approved the use of pressurized oxygen for the treatment of many chronic conditions such as gas emboli and decompression illness. Recently medical researchers have identified a possible potential in HBOT in the treatment of children with CP. 469-473

The theory behind why hyperbaric treatment works in children with CP is still unclear. Researchers are hypothesizing that due to the lack of oxygen to a specific region of the CNS, the damaged nervous tissue may become dormant as a means of a protection response. This dormant activity if it is occurring as hypothesized, is being reversed by the increased oxygen tension being produced by HBOT. The increased oxygen tension acts as a signal to the damaged nervous tissues to resume normal function. Stimulation of the dormant nervous tissue may include a continuation of developmental growth, re-myelination and modeling of pathways within the CNS, all of which could provide the increase function being seen in children following HBOT. 469-473

Oxygen under pressure increases the number of oxygen molecules entering the blood stream than the amount entering at sea level. While breathing pure oxygen, at sea level, all the hemoglobin is already filled to capacity. The remaining molecules of oxygen are transported in the blood stream via the plasma. Plasma does not have the ability that hemoglobin does to stabilize
oxygen; therefore, increasing oxygen tension levels forces direct absorption of oxygen to the tissue. This forced absorption therefore, increases the amount of oxygen and stimulates healing.\textsuperscript{469-473}

Researchers are using Single Photon Emission Computed Tomography or SPECT to investigate HBOT and its effects on children with CP. SPECT is a newer imaging technology used to observe metabolic activity of the brain. The patient is injected with a small amount of radiological material and scanned once the radiological material has circulated throughout the body. SPECT scans can identify changes of blood perfusion within the CNS, therefore, providing information about possible changes in the metabolic activity of the CNS following HBOT.\textsuperscript{468-473}

A pilot study by Montgomery et al is the only current research study investigating the effects of HBOT on children with CP. Twenty-five children diagnosed with spastic diplegia CP received twenty, sixty-minute HBOT treatments at 1.75 atmospheres of pressure. Moderate improvements were identified in the GMFM for gross skill, the Jebsen for fine motor and the Modified Ashworth for spasticity. Subjective reports by a parental questionnaire also identified improvement following HBOT. Limitations to this study include the lack of randomization and a control group.\textsuperscript{474}

Adeli Treatment Program

The information contained in this section comes primarily from verbal interviews and observations. Within the United States only a few health care providers have been allowed to observe the Adeli treatment at Euro-med of
Poland. Numerous interviews pertaining to the Adeli treatment were conducted with Dr. Edward Dabrowski, Medical Director of the Physical Medicine and Rehabilitation Center at Children's Hospital of Michigan, Dr. Steven Siconolfi, Associate Dean of Research and Graduate Studies at Wayne State University School of Medicine, and Gretchen Backer Senior Physical Therapist and Director of Children's Hospital of Michigan's Motion Analysis Laboratory. These medical professionals have traveled to Poland and observed the treatment numerous times and therefore, are referred to as subject matter experts. Information pertaining to the history of the original suit was obtained through interviews with Dr. Steven Siconolfi. His experience with both the original suit and the current Adeli suit is extensive. Dr. Siconolfi was a scientist at National Aeronautics and Space Administration (NASA) for eleven years, specializing in the physiological affects of the human body in weightlessness and led the investigation into the original penguin suit.

Observations by myself, Troy Lase, were conducted at North Oakland Medical Center (NOMC) in Pontiac Michigan where the Adeli treatment is administered excluding the use of the actual suit. Three treatments were observed to gather information pertaining to the secondary modalities and exercise components of the Adeli program.

History

In the early 1960's both the United States and Russia began research to determine the influence of weightlessness on the human body during space flight. After spending time in a weightlessness environment, both United States
astronauts and Russian cosmonauts exhibited subtle changes in the circulatory, respiratory and musculo-skeletal systems of the body. Medical examinations of the astronauts and cosmonauts identified minor physiological changes in the decrease of bone mineral density and an increase in muscle atrophy.

In 1971, the Soviet Union designed the first anti-gravitational suit known as the Penguin suit. The primary goal of this suit was to deter the negative effects of weightlessness that astronauts and cosmonauts were experiencing in space flight. The original design of the suit was a system of elastic rubber bands, which mirrored gross flexor and extensor muscle groups. By using the elastic bands to provide forces throughout the body, scientists attempted to reproduce the effects of gravity while in a weightlessness environment.

Many studies by both US and Russian space agencies were not successful in identifying any evidence that the suit simulated gravity and altered the amount of bone mineral density and muscle atrophy that occurred while in space. In the late 1980's, the decision was made to suspend research on the suit due to lack of evidence.

In 1991, scientists from the Russian Academy of Science decided to incorporate the space suit into rehabilitation of children with CP. Initial reports were favorable and therefore the Pediatric Institute of the Russian Academy of Science created the current suit being used for the treatment of CP known as the LK-92 or the Adeli suit.

Using the Adeli suit, clinicians in Poland developed what is currently known as the Adeli Treatment. This treatment consists of functional training and exercise
while in the Adeli suit as well as a strengthening program, balance and selective movement training and a variety of therapeutic activities to include swimming. Each of the individual components of the Adeli Treatment will be investigated in the following sections.

Program Admission

Initially family members contact either the primary treatment center in Poland or admission sponsors for the treatment center within the United States. A pre-admission packet consisting of a current physical, the child’s current functioning level and pertinent medical history is submitted for medical review to the treatment center to determine the child’s eligibility for the program. A non-refundable processing fee is also required prior to any considerations being made. Treatment dates are established once the initial acceptance to the treatment program is established. Thirty days prior to the start date of the Adeli treatment program, the child’s primary care provider will perform a physical examination to ensure that no complications have occurred between initial acceptance and the actual treatment.

Facility

Currently Euro-Med of Mielno Poland is the only treatment center in the world offering the Adeli treatment to include the actual suit. Within Russia, a modified version of the Adeli treatment program with the Adeli suit is available but only to Russian children. Families traveling from the United States must fly into Warsaw, Poland where transportation is provided for them to the treatment center. Most European families travel by train or vehicle directly to the treatment center.
center. The facility consists of a handicap accessible living center for the child and one family member.

Treatment Protocol

As mentioned earlier in this section, the Adeli treatment is administered for twenty-six to twenty-eight days, six days a week, approximately four hours per day. The daily treatment protocol can be categorized into four specific smaller treatment programs. These four smaller treatment programs are: functional training while wearing the Adeli suit, suspended standing activities to promote strength and balance, lower extremity strength and selectivity training and tone management. Treatment progresses in the following order: tone management, Adeli suit treatment, standing balance training, and general strengthening. Each of the four smaller components, as previously mentioned, will be discussed in the order in which they occur while a child is part of the rehabilitation program.

Adeli Suit Design

The Adeli suit is a modified version of the original penguin suit used by the Russians during space flight. The primary difference between the two suits is that the elastic band system in the original suit is incorporated into a flame resistant flight suit providing protection for the cosmonauts in case of a fire. The Adeli suit consists of five major components: a pelvic girdle and corset, upper trunk and shoulder brace, knee braces, shoes and a cap (optional). During treatment each child the entire suit except for the cap. According to Dr. Dabrowski the cap is rarely used in the Adeli treatment. The entire system is
connected through approximately $\frac{3}{4}$ inch elastic cords. These cords originate from the corset and progress distally toward either the head or feet.

Adjustments to the cords can be made in order to increase or decrease tension at the trunk, therefore facilitating proper postural alignment of the child. No specific protocol exists which determines the amount of tension being applied per band in relation to an individual diagnosis, progression of treatment or any other variable. Also there is no system to measure if the amount of tension being placed into the cords is equal either side of the body’s midline. (Figure 1)

**Staff Requirements**

In the Adeli treatment program two physical therapists are the primary providers of therapeutic service for each child. In children requiring more physical assistance a third therapist may assist in treatment.

**Treatment Program Outline**

The Adeli treatment program is twenty-six to twenty-eight days long in duration. Each child undergoes an initial physical examination by the team of physical therapists assigned to that child. The results of the examination are used to assist in establishing the treatment program specific for each child. The Adeli
treatment is administered six days a week with one day of rest. Each day
treatment consists of approximately four hours of therapy that are distributed
throughout the day. The treatment includes gait and transfer training in the Adeli
suit, balance training, strength training and tone management techniques. Each
of these specific treatments will be discussed in further detail later in this section.

Tone Management

Three techniques are used at the start of each treatment session to help
decrease the amount of spasticity in a child. These techniques include the use of
hot packs, acupressure and magnetic beds. All three of these technique lack any
scientific evidence as a viable treatment in tone reduction. The specific use of
each one of these techniques is based primarily on the therapist’s discretion and
previous experiences with a specific patient.

The extended use of hot packs is proposed to decrease the amount of
spasticity in an affected limb is common in preparation before the Adeli
treatment. When questioning the staff at NOMC I was told that the hot packs
decrease the excitability in gamma motor efferent nerves and therefore muscle
spasticity tends to decrease. In searching the literature there is no scientific
evidence to support the use of hot packs for an extended amount of time to
reduce spasticity.

Acupressure is another technique used to help decrease spasticity in an
extremity. Acupressure is a treatment technique similar to the Chinese technique
of acupuncture. Acupressure techniques use the same meridian placement
points as acupuncture but instead of using needles, direct finger pressure is
applied to a specific target location within the patient limbs. By applying pressure to this specific location, clinicians believe it to help decrease the amount of spasticity within either upper or lower extremities. Protocols at both Euro-Med and NOMC recommend that pressure be applied to a specific point for one to five minutes. Once the entire treatment is complete, passive and active range of motion is performed. At Euro-Med range of motion activities are not performed following acupressure; instead, treatment progresses directly to the Adeli suit. In reviewing the literature there is no scientific evidence that investigates acupressure as a treatment to decrease spasticity in any patient populations.

The last technique used by the rehabilitation center is the use of magnetic beds. There is no scientific evidence which discusses electrical fields produced by magnets or their effects on different physiological functions within the human body especially in reducing spasticity.

Adeli Suit Treatment

The primary treatment modality, which involves more then half of the overall treatment time, is the actual use of the Adeli suit. The use of handling techniques, similar to NDT, provides facilitation of active movements for a child while in the Adeli suit. Physical therapists at the rehabilitation center report that the underling basis of the Adeli suit is that it provides proprioceptive feedback to the CNS; therefore handling techniques are a useful adjunct in assisting in active facilitation while performing different treatment activities.

Different functional activities incorporated into the treatment include transfer training from the floor, mat table, chair and standing positions, sitting and
standing balance activities, and pre-gait activities. Common pieces of therapeutic equipment used in this part of the treatment include therapeutic balls, parallel bars, floor and tablemats, pull-up ladders and full-length standing mirrors. All the activities within the suit are directed by the deficits noted in the initial examination and progression of the deficits throughout treatment.

Physical therapists who have observed the actual treatment sessions state that while a child is in the suit, therapists conducting the treatment tend to perform NDT handling technique improperly. The main issue addressed was that regularly improper hand placement occurred during treatment which likely led to the poor facilitation of movement. One therapist questioned if any neurofacilitation could even properly occur because of constraints of the Adeli suit. The bases for this conclusion are that when utilizing proper facilitation techniques, for example at the pelvis, the pelvic girdle of the Adeli suit may actually be countering the facilitation technique. All the therapists agreed that by not using proper handling techniques the therapist at Euro-med were likely just assisting the child move their body versus facilitating proper movement through NDT techniques.

Standing Balance Training

Another piece of equipment used in the Adeli treatment is a modified version of a cage like device, which surrounds the treatment area on three sides and overhead. The box is made of steel rods welded together to make eight-inch squares around the entire structure. The physical dimensions of the structure...
are approximately ten feet by ten feet wide and eight feet high. The floor of the area is covered with a foam floor mat for the child’s protection.

This equipment is used in a variety of standing balance-training activities and functional strengthening. The child stands in the middle of the area with a pelvic belt similar to a weightlifter’s belt. From all four corners of the cage one-inch adjustable elastic bands are secured to four attachment sites on the pelvic belt. Gretchen Baker stated that she believed the external support provided by the belt and the elastic band provide proprioceptive feedback to the CNS and also facilitate proper alignment of the child. For children with marked increases in adductor spasticity, another pair of elastic bands may be placed on the lower extremity to position the limbs in a good standing position.

Once the child is positioned in the cage with all the bands attached properly, the treatment therapist uses handling techniques to facilitate different balance activities and transfer training in kneeling and half kneeling. The bands within the cage help to support the child as well as challenge their balance. Limitations to this piece of equipment are based on overextending the bands, which could produce a snapback response and endanger the child in the device. Another limitation is that the elastic cords may not be equal in tension and therefore give an inadequate position for the child. Adjustments to a child’s height and physical size are easily made because of the cage design and different size elastic cords are available. (Figures 2 and 3)
Strength Training

The last component of the treatment is strength training which uses the same cage structure as described in the previous section. An exercise table is moved into the area for an exercise platform for the child. The child is partially suspended in either a prone, supine, or side-lying position depending on the muscle group being exercised. Multiple pulley systems are placed around the cage with cuff like attachments being connected to different locations of the child's lower extremity. Sand bag weights are placed on the end of non-elastic cords to provide resistance for the exercising limb. Straps also are placed in different positions on the lower extremity, which are not being exercised in order to stabilize the child and promote selectivity of the exercising limb. Another strap is also placed across the pelvis, which stabilizes the trunk while doing the exercises. (Figures 4 and 5)
Current Research

Currently only one study exists within the literature which discuss the Adeli suit as a treatment option for children with CP. This study was conducted in the former Soviet Union and originally published in Russian. Dr. Semenova at the Russian Academy of Science Research Institute of Pediatrics conducted the first study focused on the use of Adeli suit as a treatment option for children with CP. Initial conclusions for this study seemed promising. Unfortunately many design and reporting issues leave this study to be very questionable in its validity. Numerous examples of the poor design of this study are clearly evident after reading the material.

One of the key problems with this study is the lack of format. Most studies are setup with an introduction, methods of the study, results of the study and a discussion of the finding. In the study by Semenova, all four of the previously mentioned sections were combined without any order. Another problem with this study is the lack of any control groups and randomization of subjects within any part of this study. Children participating in this study did not participate in the
Euromed Adeli Treatment. According to this study all the children participated in a treatment program which only included the Adeli suit. Within this study there was no discussion about what the author used in the Adeli suit treatment. This study did report on functional improvements once the children completed treatment in the Adeli suit. Within the study the variables investigated included posture control, walking, speech production and self-care. Again throughout this study there were no operation definitions to evaluation criteria of the each variable. The data for this information within the study did not even have a value system on the Y-axis of the graph. The reader is left to guess what 0 to 90 means. Another surprising aspect in the graph was that in every category the effect of the Adeli suit was doubled in comparison to before receiving treatment in the Adeli suit.

Another component of this study was to analyze cortical EEG activity in thirty-two children with different types of cerebral palsy. EEG percent activities were measured both before and after being treated in the Adeli suit. Again this component of the study lacked any randomization or control groups. The results of the cortical EEG data discussed identified that a delay in cortical electrogenesis occurred in the children but this information is not consistent when compared to the graph of the same data. 36

Even though the author of this study reports that significant positive outcomes were identified numerous limitations were present which makes results very questionable. In general a major challenge is the language and communication barrier. In reading this study one could quickly come to the conclusion that a lot
of information was lost in the translation from Russian to English. Unfortunately, this is common in any language translation. This study was also hard to follow in that different aspects of the paper were not clearly defined. An example of this is that methods of developing and completing the study were never explained or discussed. The article never commented on what types of formal tests were used to establish the efficacy. The study lacked randomization or a control arm, and statistical analysis was not discussed therefore again making the result very questionable. Also, the results, discussions, and conclusions were compiled together, making it hard to understand the critical points and conclusions. Different and/or new medical terminology also posed some challenges in trying to understand the research. Researchers are always eager to either duplicate or modify an existing study to prove the validity of the project. This study would be impossible to reproduce based on a poor description of its design.

Theoretical Framework

In this section the theoretical framework of the Adeli treatment will only address a possible theory to how the actual Adeli suit may decrease spasticity in a child with spastic CP. The modalities associated with the Adeli treatment have been discussed in detail within previous sections of this case report and therefore will not be addressed in this section.

Within the literature there is no discussion of a theoretical framework or mechanism of action for how or why the Adeli suit possibly causes a decrease in spasticity in children with CP. The proposed theory presented in this case report by the author is based on personal experiences, experiences of clinicians who
have witnessed the treatment and the current understanding of neurophysiology and motor control.

In developing a possible theory to how the Adeli suit works there are three major components, which contribute to the theoretic construct of the Adeli suit. By combining the current understanding of these three components and how they may possibly interact with each other, a general theoretical model of the Adeli suit can be proposed. First, is the understanding of the motor program theory of motor control, more specifically the actions of GMPs and CPGs. This theory of motor control will be the foundation for the proposed theory of the Adeli suit. Second, is the current understanding of neurophysiology. More specifically is the understanding of how joint receptors receive stimuli when joints are compressed for sustained periods of time.

Third, is the structural design of the Adeli suit. During the development of the Adeli suit for rehabilitation treatment what were the designers expecting to accomplish with the position of the elastic bands across the body. Again throughout the literature there is no discussion of any possible mechanism of action to how or why the elastic bands work to decrease spasticity. All three of these components will be discussed individually on how the contribute to the theoretical concept of the Adeli suit.

The motor program theory of motor control is the most important component of the proposed theory of the Adeli suit. Other theories of motor control may also play a small role in understand how the Adeli suit works but it is the motor program theory, which will be the focus for this theory. The motor program
theory identifies that movement is initiated and or controlled through a variety of motor programs located within the CNS. As discussed earlier these programs are called GMPs and CPGs. What is not clearly discussed within the literature is the level of maturation of the programs. Meaning that when a child is born are the GMPs and CPGs of the brain and spinal cord in a very basic form of development and non-modifiable or do the programs evolve and become more complex as the child grows due to sensory input. As evidence suggests different types of sensory input into a child is critical for development therefore supporting that GMPs and CPGs are modifiable. But when looking at the CNS of a child with spastic CP, these motor programs may have varying levels of damage. Therefore if the motor programs are modifiable even when damaged, an increased amount of proprioceptive input, similar to the sustained compression forces such as what the Adeli suit produces, may modify the motor programs and cause a decrease in spasticity. As discussed earlier numerous treatments exist which utilize sensory input to decrease spasticity and improve function. But more importantly is how does the Adeli suit provide this proprioceptive feedback into the CNS?

This is where the second component of the Adeli theory, understanding joint receptors is critical in developing a theory to why the Adeli suit works. Within each joint numerous mechanoreceptors provide continuous sensory feedback into the CNS in reference to movement and position of the joint. Four major types of joint receptors have been identified in the literature. They include Golgi-type endings, Golgi-Massoni corpuscles, Ruffini corpuscles and free nerve endings. These joint receptors are located throughout all the joints of the body.
Joint receptors are unique in that they are slow adapting, that is they never fully adapt and therefore provide continuous sensory feedback into the CNS. This continuous feedback into the CNS is possibly how the Adeli suit provides the increase proprioceptive feedback into the CNS. ⁴⁷⁹-⁴⁸⁸

The feedback to the CNS provided by the joint receptors is also a key component to the theory of the Adeli suit. The importance lies in how the elastic bands of the Adeli suit compresses numerous key joint throughout the trunk and lower extremity.

The third component of the Adeli theoretical framework is the design of the actual suit. As discussed earlier the Adeli suit consists of a variety of elastic cords, which according to promotional information provided by Euromed the bands are designed to mimic the gross flexor and extensor muscle groups of the body. But how are the bands actually providing proprioceptive feedback into the CNS. When questioned, clinicians from NOMC who had extensive training using the Adeli suit at Euromed could not specifically answer how the elastic bands provided proprioceptive feedback to the CNS. The clinicians at NOMC stated that just wearing the Adeli suit while doing functional activities provided proprioceptive feedback to the CNS, which limited spasticity and increased function. Unfortunately this answer again supports why more extensive research needs to be conducted on the Adeli suit and the actual treatment.

When looking closely at the Adeli suit, each group of elastic bands cross major joints such as the knee and hip. Also the torso vest has bands, which provide direct compression into the spinal column by pulling downward on
shoulder pads. By tightening the bands to any degree an increased amount of compression occurs into the joint. This increase pressure is likely to stimulate the corresponding joint receptors and therefore provide the increase proprioceptive feedback into the CNS.

Joint compression is a technique, which has been used in children with spastic CP prior to the Adeli suit. Clinicians have used a neoprene compression suit, similar to a wet suit, for children with spastic CP. The suit is identified as a stabilizing pressure input orthosis or SPIO. The SPIO is similar to the Adeli suit in that it provides compression to the trunk and pelvic regions of the child. The difference between the SPIO and Adeli suit is the pressure applied by the Adeli suit is adjustable and also directed where as the SPIO applies a given pressure from multiple directions. Also the SPIO does not have any lower extremity components like the Adeli suit. Clinicians who have worked with the SPIO have identified subjective functional improvement in children. Unfortunately within the literature this is the only article that discussed the use of a compression garment for children with CP.

Now that all three components have been discussed a general theory can be proposed to how the Adeli suit may decrease spasticity in a child with CP. The design of the Adeli suit provides compression of key joints such as the knees, hips and spinal column and sometimes the ankles depending on the suit configuration. The increased compression caused by the elastic bands crossing the joints stimulates the receptors within the joints themselves. The proprioceptive stimulus carried to the CNS for processing is unique due to the
limited amount of excessive compression the body experiences during regular functional activities. Granted, during ambulation and other functional activities compression of the joints does occur but not at a moderately high sustained level such as provided by the Adeli suit. Therefore the sustained compression provided by the Adeli suit across major joints may provide a unique elevated sustained proprioceptive input which allows otherwise damaged but modifiable GMPs to inhibit the amount of spasticity occurring in the lower extremities.

Several limitations exist within this theory of how the Adeli suit works. This proposed theory of the Adeli suit has been developed in a generalized form. Currently there are far too many unknowns about the Adeli suit and it affects on the human body to make real specific assumptions to how it decreases spasticity. Also this theory does not investigate in great detail the neurological aspects of how the proprioceptive input caused by increased compression of the joints might be processed in the CNS. Another limitation is any theory based on the Adeli suit would be very challenging to prove in a research mode due to the complexity of the CNS. Instead investigators can rely on current and future understandings of neurophysiology and motor control to develop a more complex theory for the means by which the Adeli suit works.

Summary Of The Literature

Through this extensive literature review it is evident that even though a significant number of viable treatment options exist for children with CP there is still a need for another. The basis for such a conclusion is that even though medical research has developed numerous treatment options for children with
CP there is still no single medical management for CP. The likelihood of a single treatment for children with CP may never occur but by focusing on new treatment ideas and by establishing questions to be answered through ongoing research there will always be a chance for such a breakthrough. This is where the importance of the Adeli treatment research plays a key role. By conducting this research project and future research projects the validity and efficacy of the Adeli treatment may be determined. Long term this research may either provide the medical profession with another viable treatment option for children with CP or help medical researchers to develop newer and even more efficacious treatments for children with CP.
CHAPTER 3

METHODOLOGY

Study Design

This study is a case report design consisting of a retrospective evaluation of a subject's gait before and after participating in the Adeli treatment. Due to the case report design, inclusion and exclusion criteria are not relevant to this project.

Subject

The subject was a 12-year-old male with a medical diagnosis of spastic diplegic cerebral palsy. Prior to the Adeli treatment the subject was a community ambulator using a wheeled walker and bilateral dynamic ankle-foot-orthotics. Prior to the Adeli treatment the child participated in a variety of both school and hospital based therapy programs. The details of these programs were not available at the time of this report. Past medical history includes a selective dorsal rhizotomy at four years of age. Only minimal changes were reported following the dorsal rhizotomy. The subject was not taking any medications to decrease spasticity during the time of the evaluation or during Adeli treatment.

The subject participated in the Adeli treatment program for 28 days at Euro-Med in Mielno Poland. The subject received four to five hours of active treatment per day, which did not include rest periods between treatments. The activities of the Adeli treatment were outlined in the previous chapter. In Table 1 the daily amount of time per treatment activity is outlined. The overall treatment time line
does not include recreational activities such as swimming, which the subject participated in on a voluntary status.

<table>
<thead>
<tr>
<th>Table 1. Treatment Protocol Times</th>
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<tbody>
<tr>
<td><strong>Type of Therapy</strong></td>
</tr>
<tr>
<td>Hot Packs/ Magnetic Beds</td>
</tr>
<tr>
<td>Strength Training</td>
</tr>
<tr>
<td>Acupressure</td>
</tr>
<tr>
<td>Standing Balance</td>
</tr>
<tr>
<td>Adeli Suit Training</td>
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</table>

**Instrumentation**

**Cameras**

Movements of the lower extremities during gait were recorded using an Elite four-camera optoelectronic system. Each of the camera’s outer lens is fitted with light emitting diodes or LED’s. These LED’s emit infra red light rays towards the body-fixed targets. Once the infrared rays hit the target, they are reflected back to the camera lens. The targets themselves are wooden spheres covered by 3M Scotchlite Brand High Grain 7610 retroreflective tape. Data were collected at 100 Hz. By having each camera collect the reflected signals, a two-dimensional image is generated of the specific movement being recorded. Data output collected from each of the cameras is sent to a video processor so corresponding frames are processed simultaneously. In order to generate a three-dimensional image marker position, data from at least two cameras are needed. This process is known as direct linear transformation (DLT) and will be discussed later in this.
The cameras were placed within the four corners of the laboratory facing towards the center of the room. A calibration of the camera system was performed before initiation of the test. Calibration provides orientation of the cameras in relationship to the working volume and relative position to each other; calibration also creates the laboratory coordinate system.

Video images also were collected in both the sagittal and frontal planes while the subject ambulated for observational gait analysis. Video images were collected using two Panasonic X20 digital video cameras, and with the aid of a Panasonic Digital Effects Generator, both sagittal and frontal planes could be viewed simultaneously on the same television screen. These images were used in conjunction with the kinematic data to analyze the subject's gait pattern.

Validity and Reliability

The validity of this case report is based the BTS Elite motion analysis system, marker placement, goniometric measurements and manual muscle testing. The accuracy of the BTS Elite system is well documented in numerous studies. The most accurate being in a study by Dan et al. where the recorded linear accuracy of the BTS Elite system was recorded to be 0.67 millimeters. In reference to the reliability of this study the only issue to be considered is marker placement on the subject. A skilled physical therapist performed the marker placement in the pre and post treatment. In a study by Selfe and Carson et al., lower extremity marker placements were identified as being very accurate amongst clinicians with training in gait analysis and marker place. This supports the likelihood that the marker placement in both the pre- and post treatment...
evaluations were relatively accurate. Numerous studies have also been published which support the validity and reliability of goniometric measurement, as well as manual muscle testing as viable options of measurement when used properly by trained individuals. 498-497

Procedures

Calibration

A critical component in collecting kinematic data is to establish an operational working volume. The working volume provides the camera system with a specifically defined area to track movements. By placing retro-reflective targets at known X, Y and Z coordinates within the center of the working volume, the cameras can identify their position in space. The physical size of the working volume is a generalized representation of the subject's stride length and shoulder height. Upon defining the working volume, calibration allows for each of the four cameras to define their own position in space through direct linear transformation. In order to determine three-dimensional coordinates of a specific location in space, the known position of each camera is required.

Direct Linear Transformation

Direct linear transformation (DLT) is a mathematical algorithm used to accurately identify the three-dimensional position of a specific target in space. Single cameras are only capable of viewing any object in two dimensions; therefore, to identify a three-dimensional location in space, two separate cameras must be synchronized to the same point. The identification of three-dimensional locations in space is accomplished by having two separate cameras...
receive their own specific infrared reflection from the individual target. Each camera produces its own specific two-dimensional position of the target's location and then synchronizes the image of the target with a second camera. The synchronized two-dimensional images are combined through DLT to develop a three-dimensional position relative to the working volume of the laboratory. Creating a vector from one camera to the two-dimensional image of the target and then projecting this vector into three-dimensional space allows for the exact location of the target to be determined. The second camera completes the same process and is used to calculate the target's location. The three-dimensional location of the target is calculated at the intersection of the projected two-dimensional vectors.

Clinical Examination

A clinical examination was performed by a licensed physical therapist prior to collecting data. The clinical examination was used to determine the impairment in the subject's lower extremities and trunk. The physical examination consisted of posture observations, manual muscle testing of strength, measurement of available range of motion at different joints, leg length, and Modified Ashworth for spasticity.

Test Preparation

The subject wore shorts, which revealed pelvic and lower extremity bony landmarks for target placement. Targets were placed on the subject's skin using two-sided 3M hypoallergenic adhesive tape in the following areas: bilateral ASIS's, spinous process of S2 (midpoint between bilateral PSIS's), thigh wand on
lateral mid thigh, lateral condyle of femur, tibial tuberosity, distal shank of tibia, distal posterior shank of tibia, calcaneus, lateral foot posterior to the fifth metatarsal and medial foot posterior of the first metatarsal head.

Testing Protocol

Once the reflective targets and EMG pads were in place, the subject, prior to collecting data, was able to walk through the calibrated volume to become accustomed to the equipment. Once the subject felt comfortable data collection began. Before the walking trials, the subject stood in the center of the working volume so that a standing file could be recorded. A standing file allows for the identification of additional target locations including the medial femoral condyle and medial and lateral malleoli. The standing file data allow for the calculation of a target position relative to their dynamic local coordinate systems. The standing file data are used to calculate knee and ankle joint centers. Walking data were collected barefoot. Physical assistance was required due to interference produced by the subject's walker. A total of six walking trials per extremity and condition were collected on the subject. Of the six walking trials, the one that best represented the subject's gait pattern per extremity was used for the data analysis.

Data

Data Processing

Following data collection, further processing was required to convert the data into meaningful graphs detailing the kinematics of the subject's gait pattern. Three-dimensional target coordinates were determined through DLT by
combining two different two-dimensional target position vectors in a process called tracking. Tracking involved manual identification of all corresponding targets from at least two different cameras. Once tracking was completed, a linear interpolation algorithm was used to substitute missing data points due to targets being momentarily obstructed. The interpolated data were then converted into a standard computer language format (ASCII) for later use in custom software to calculate joint angles. To process kinematic data, local coordinate systems were calculated. The coordinate systems were aligned with these systems using three non-collinear targets attached to respective body segments. Two target positions were used to first create an anatomical axis, while the third target made up an anatomical plane. The joint coordinate system is a non-orthogonal system fixed to a joint used to determine the orientation of one segment relative to another, described as joint angles.

Data Analysis

The data analysis was based on a three-step process consisting of evaluating data collected from clinical examination, temporal and spatial parameters and three-dimensional kinematics both prior to and following the Adeli treatment. The first step was to operationally define the four requirements of gait and the specific critical events involved with each, as reported by Gage and Perry. The critical events unique to this case report consisted of sagittal plane movements of the hip, knee and ankle. All of the critical events except at the hip have been identified specifically by Perry. Measurement of hip movement is based on the Motion Analysis Center’s coordinate system that utilizes the thigh
pelvis coordinate system. The operational definition of the four requirements and the specific critical events are outlined in Table 2.

<table>
<thead>
<tr>
<th>Requirement Of Gait</th>
<th>Joint</th>
<th>Required Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stability of the weight bearing limb throughout stance</td>
<td>Ankle</td>
<td>Ankle progress from neutral to 5 degrees of plantarflexion during mid-stance phase of gait.</td>
</tr>
<tr>
<td>phase</td>
<td>Knee</td>
<td>Extension from 15 degrees of flexion to nearly neutral in mid-stance phase of gait.</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>Hip extends from 35 degrees of flexion to 10 degrees in mid-stance phase of gait.</td>
</tr>
<tr>
<td>Clearance of the non-weight-bearing foot during swing</td>
<td>Ankle</td>
<td>Ankle progress from 20 to 10 degrees of plantarflexion during initial swing phase of gait.</td>
</tr>
<tr>
<td>phase</td>
<td>Knee</td>
<td>Rapid knee flexion to 40 degrees during pre-swing phase of gait.</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>15 degrees of hip flexion is achieved during initial swing phase of gait.</td>
</tr>
<tr>
<td>Pre-positioning of the advancing foot during terminal</td>
<td>Ankle</td>
<td>Ankle is to maintain a neutral position throughout swing phase of gait.</td>
</tr>
<tr>
<td>swing</td>
<td>Knee</td>
<td>Mid-swing rapid extension of 25 degrees. Terminal swing phase of gait.</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>Mid-swing rapid extension of 25 degrees. Terminal swing full knee extension.</td>
</tr>
<tr>
<td></td>
<td>Hip</td>
<td>Initial swing 15 degrees of flexion. Mid-swing achieve and maintain 25 degrees of flexion.</td>
</tr>
</tbody>
</table>

The second step was to identify seven subphases of gait for both pre- and post analysis allowing for an accurate assessment of the critical events based on the individual subphases of gait. The method to calculate the individual subphase is as follows. Toe off percentages for both the right and left lower extremities were generated through three-dimensional kinematic analysis and are represented graphically by either a solid or dashed vertical line within each of
the graphs. For a normal subject spends approximately 62% of the gait cycle in stance phase and 38% in swing phase, and a normal stance phase is 12% loading response, 19% mid-stance, 19% terminal stance and 12% pre-swing. Within a normal subject, the swing phase is 13% initial swing, 12% mid-swing and 13% terminal swing. Utilizing a calculation by Groth et al., the percent of each subphase of gait was determined. The equation used to determine the subject’s gait cycle subphase percentages is as follows: 

$SSP = SPP \times \frac{NSP}{NPP}$

(SSP=subject subphase percentage; NSP=normal subphase percentage; SPP=subject phase percentage; and NPP=normal phase percentage). For example, to calculate the gait cycle subphase percentage of loading response in a subject whose stance time is 69%, loading response percentage = $69\% \times \frac{12\%}{62\%}$. The subject therefore spends about 14% of the gait cycle in loading response compared to the normal amount of 12%.

The third step was for the evaluator to determine if any changes occurred between pre and post treatment analysis. To achieve this determination each critical event of gait was identified by a specific joint movement within a given subphase of gait. As discussed in the second step, the specific subphases were determined based on the data collected in pre and post treatment analysis. The subphase or subphases unique to a specific critical event are shaded in gray. Also, along the horizontal axis of the graph each of the individual subphases are identified.

At this point the evaluator looked at the gray area, which represents a specific subphase unique to the subject’s gait and determined if the critical event did or
did not occur based on the criteria outlined in the operational definitions. Refer to Figure 6 as an example of the kinematic graph.

**Figure 6. Kinematic Graph Example**

If the critical event did occur in either pre- or post treatment analysis, the evaluator recorded a "yes" meaning successful completion of that critical event. If the critical event did not occur in either pre- or post treatment analysis, the evaluator recorded a "no", meaning the unsuccessful completion of that critical event. Therefore, for each critical event per limb the evaluated provided a score for both the pre- and post treatment analysis.
CHAPTER 4
RESULTS

Observation of Gait

The subject's gait can be characterized by a flexion and extension synergy, which was maximized to allow for functional ambulation. The subject also demonstrated mild scissoring with significant hip instability, possibly contributing to poor balance while ambulating during pre- and post test analysis. Foot drag did not occur while the subject ambulated. The subject required hands on assistance during the gait evaluation.

Temporal and Spatial Gait Parameters

In pretreatment analysis, when compared to normalized data, the subject demonstrated a moderate to marked increase in double support and percent time in stance. Also, in pretreatment analysis, step and stride length were moderately decreased when compared to normal. The subject's walking velocity also demonstrated a marked decrease when compared to normal. Average walking velocity in pretreatment analysis was 25.7 cm/sec and post treatment analysis was 27.8 cm/sec. In post treatment analysis, single and double limb support were notably worse following the Adeli treatment. Other temporal and spatial parameters had non-remarkable changes. Energy expenditure was not assessed as a factor in either pre- or post test analysis. The results of pretreatment temporal and spatial parameters are located in Appendix B and post treatment results are located in Appendix C.
In pretreatment analysis the left lower extremity spent seventy-seven percent of the cycle in stance phase and twenty-three percent in swing phase. In post-treatment analysis of the left lower extremity, seventy-eight percent of the cycle was in stance phase and twenty-two percent was in swing phase. In both pre- and post treatment analysis, the right lower extremity spent an equal amount of time in both stance and swing phases of the cycle. The right lower extremity spent eight-six percent of the gait cycle in stance phase and fourteen percent in swing phase. The sub-phase percentage breakdown for both pre- and post treatment analysis is outlined in Appendix D.

Clinical Examination

The following information is based on clinical examination data collected prior to the three-dimensional gait analysis in both pre- and post treatment settings. This section will identify key clinical findings, which are relevant for the future discussion of measured results. Due to similar identical clinical finding of both pre- and post test examination, the results of clinical examination will be reported together. The complete pre- and post clinical examination findings are located in Appendices E & F respectively.

At both hips, mild to moderate limitations were noted in hip extension range of motion in both pre- and post clinical examinations. Mild to moderate tightness of the hamstrings and rectus femoris also were identified in both pre- and post clinical exams with the left being greater than the right. Moderate tightness also was noted bilaterally in the gastrocnemius and soleus muscle groups with right being greater than left. Marked bilateral weakness also was identified in hip flexors and extensors, hamstrings, rectus femoris, and triceps surae. Moderate to severe
spasticity was identified bilaterally in the hamstrings, rectus femoris, and triceps surae.

Requirement One

The first requirement of gait is the stability of the weight bearing limb throughout the mid-stance phase of gait. At the ankle, the critical event for this requirement is the movement of the ankle from neutral to five degrees of dorsiflexion during mid-stance. Refer to Figure 7 for the subject's kinematic data at the ankle for pre- and post treatment analysis.

In pretreatment analysis, the left ankle began mid-stance at about ten degrees of dorsiflexion and progressed to about two degrees of dorsiflexion at the end of mid-stance. In post treatment analysis, the left ankle began mid-stance at about three degrees of plantar flexion. The left ankle continued to plantarflex about seven degrees approximately halfway through mid-stance phase before dorsiflexing to neutral at the end of the phase. In neither pre- or post treatment analysis did the left ankle achieve the critical event of a neutral position to five degrees of dorsiflexion during mid-stance.

The right ankle in pre- and post treatment analysis began and maintained a neutral position to one degree of dorsiflexion throughout most of the mid-stance phase of gait. During the end of mid-stance, in pretreatment analysis, the right ankle demonstrated two to three degrees of dorsiflexion while in post treatment only about one to two degrees of dorsiflexion were demonstrated. In neither pre- or post treatment analysis did the right ankle achieve the critical event of neutral to five degrees of dorsiflexion during mid-stance.

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For the first requirement of gait, the critical event at the knee is extension from fifteen degrees of flexion to a nearly neutral position during mid-stance. Refer to Figure 8 for the subject's kinematic graphs at the knee for pre- and post treatment analysis. In the pretreatment analysis, the left knee began mid-stance at about thirty degrees of flexion and demonstrated rapid extension to about ten degrees by the end of the sub-phase. In post treatment analysis, the left knee began mid-stance at about fifteen degrees of flexion and quickly extended to only ten degrees of flexion. The left knee maintained approximately ten degrees of flexion throughout mid-stance. In both pre- and post treatment analysis the left knee did achieve the critical event of knee extension from fifteen degrees of flexion to nearly a neutral position during mid-stance.

In the pretreatment analysis, the right knee began mid-stance at about fifteen degrees of flexion and with moderate progression achieved a neutral position in the mid-point of the sub-phase. Throughout the remainder of mid-stance the right knee maintained neutral position. In post treatment the right knee also began mid-stance at about fifteen degrees of flexion. In post treatment analysis, the right knee did not extend as rapidly as demonstrated in pretreatment analysis. Also in post treatment
analysis the right knee was able to achieve and maintain one to two degrees of flexion throughout a moderate amount of mid-stance. In pretreatment analysis the right knee did not achieve the critical event of knee extension from fifteen degrees of flexion to nearly a neutral position during mid-stance. However, in post treatment analysis the right knee did achieve the critical event of knee extension from fifteen degrees of flexion to a nearly neutral position during mid-stance.

Figure 8. Requirement One, Critical Event Two

For the first requirement of gait, the critical event at the hip is extension from thirty-five degrees of flexion to ten degrees of flexion in mid-stance. Refer to Figure 9 for the subject’s kinematic graphs at the hip for pre- and post treatment analysis. In pretreatment analysis, the left hip began mid-stance flexed at thirty degrees and slowly extended to only twenty degrees at the end of mid-stance. In post treatment analysis, the left hip began mid-stance close to forty degrees of flexion but slowly extended to only about twenty-five degrees of flexion at the end of mid-stance. In neither pre- nor post treatment analysis did the left hip did achieve the critical event of hip extension from twenty degrees of flexion to neutral in mid-stance.

In pretreatment analysis, the right hip began mid-stance at approximately twenty-eight to thirty degrees of hip flexion and rapidly extended to about ten degrees at
the end of stance phase. In post treatment analysis the right hip began mid-stance at roughly thirty-five degrees of flexion but slowly extended to about twenty degrees of flexion by the end of the sub-phase. In pretreatment analysis the right hip did achieve the critical event of hip extension from thirty-five degrees of flexion to ten degrees of flexion during mid-stance. However, in post treatment analysis, the right hip did not achieve this critical event at the hip.

**Figure 9. Requirement One, Critical Event Three**

<table>
<thead>
<tr>
<th>Requirement Two</th>
</tr>
</thead>
<tbody>
<tr>
<td>The second requirement of gait is the clearance of the non-weight-bearing foot during the initial swing phase of gait. At the ankle, the critical event is progression from twenty degrees to ten degrees of plantar flexion during initial swing phase. Refer to Figure 10 for the subject’s kinematic graphs at the ankle for pre- and post treatment analysis.</td>
</tr>
</tbody>
</table>
| In pretreatment analysis, the left ankle began initial swing in a neutral position. During the short initial swing phase the left ankle rapidly dorsiflexed to nearly ten degrees. In post treatment analysis the left ankle again began initial swing in a neutral position. The left ankle than flexed rapidly but only achieved about four to
six degrees of dorsiflexion by the end of the phase. In neither pre- nor post treatment analysis did the left ankle achieve the critical event of twenty to ten degrees of plantar flexion during initial swing phase.

In pretreatment analysis, the right ankle also began initial swing in a neutral position but, unlike the left ankle, was only able to achieve two to three degrees of dorsiflexion by the end of the cycle. In post treatment analysis, the right ankle began initial swing at about five degrees of plantarflexion and rapidly achieved approximately two degrees of dorsiflexion by the end of the phase. In pre- and post treatment analysis, the right ankle did not achieve the critical event of twenty to ten degrees of plantar flexion during initial swing phase.

Figure 10. Requirement Two, Critical Event One

For the second requirement of gait, the critical event at the knee is rapid flexion of forty degrees during preswing phase of the gait cycle. Refer to Figure 11 for the subject’s kinematic graphs at the knee for pre- and post treatment analysis. In pretreatment analysis, the left knee began preswing at about thirty-eight degrees of flexion. The left knee gradually flexed throughout preswing to about sixty-five degrees of knee flexion at the end of this subphase. In post treatment analysis, the left knee began preswing phase of gait at about thirty degrees of flexion. The left
knee also demonstrated gradual flexion throughout preswing but was only able to achieve approximately fifty degrees of flexion at the end of preswing. In neither pre- nor post treatment analysis did the left knee achieve the critical event of rapid knee flexion to forty degrees during preswing phase of the gait cycle.

In pretreatment analysis, the right knee eventually began preswing at about ten degrees of knee flexion. The right knee demonstrated rapid knee flexion throughout the preswing phase of gait, completing the phase at almost sixty degrees of flexion. The post treatment analysis was nearly identical to pretreatment analysis. The right knee in post treatment analysis also demonstrated rapid knee flexion throughout the preswing ending the phase also at almost sixty degrees of flexion. In both pre- and post treatment analysis the right knee did successfully achieve the critical event of rapid knee flexion to forty degrees during preswing phase of the gait cycle.

**Figure 11. Requirement Two, Critical Event Two**

For the second requirement of gait the critical event at hip is that flexion is achieved from five degrees to twenty degrees during initial swing. Refer to Figure 12 for the subject's kinematic graphs at the hip for pre- and post treatment analysis. In pretreatment analysis, the left hip began initial swing at thirty degrees of flexion. The left hip then rapidly flexed to about fifty-five degrees of flexion by the end of
initial swing. In post treatment analysis, the left hip began initial swing closer to twenty-five degrees. Similar to pretreatment analysis, the left hip demonstrated rapid hip flexion to about forty-five degrees of flexion. In neither pre- nor post treatment analysis did the left hip achieve the critical event of hip flexion from five degrees to twenty degrees during initial swing phase.

In pretreatment analysis, the right hip began initial swing close to thirty degrees of flexion. Rapid flexion of the right hip was demonstrated throughout the initial swing phase but, due to such a short phase, the hip achieved approximately forty-three degrees of flexion. The post treatment analysis is nearly identical to pretreatment results to include the rapid hip flexion throughout the initial swing phase of gait. In neither pre- nor post treatment analysis did the right hip achieve the critical event of hip flexion from five degrees to twenty degrees during initial swing phase.

**Figure 12. Requirement Two, Critical Event Three**

**Requirement Three**

The third requirement of gait is the pre-positioning of the advancing foot during mid and terminal swing. At the ankle, the critical event is the ability to maintain a neutral position during terminal swing. In pretreatment analysis, the left
ankle began terminal swing with about twelve to thirteen degrees of dorsiflexion. The left ankle plantar flexed about two to three degrees finishing terminal swing at ten degrees of dorsiflexion. In post treatment analysis, the left ankle began terminal swing with about 10 degrees of dorsiflexion. The left ankle then rapidly plantar flexed to a neutral position at the end of terminal swing. In neither pre- nor post treatment analysis did the left ankle achieve the critical event of maintaining a neutral position during terminal swing. Refer to Figure 13 for the subject's kinematic graphs at the ankle for pre- and post treatment analysis.

In pretreatment analysis, the right ankle began terminal swing with only two to three degrees of dorsiflexion. The right ankle then dorsiflexed to neutral during a very short terminal swing phase. Similar to the pretreatment analysis, the right ankle in post treatment analysis began terminal swing with about one to two degrees of dorsiflexion. The right ankle then demonstrated about three to four degrees of plantarflexion to about two degrees by the end of terminal swing. In neither pre- nor post treatment analysis did the right ankle achieve the critical event of maintaining a neutral position during terminal swing.

Figure 13. Requirement Three, Critical Event One
For the third requirement of gait, the critical event at the knee is rapid extension of twenty-five degrees during mid-swing and complete extension during terminal swing. Refer to Figure 14 for the subject's kinematic graphs at the knee for pre- and post treatment analysis. In pretreatment analysis, the left knee began mid-sw ing with approximately seventy-five degrees of flexion. The left knee then demonstrated a moderately rapid extension to about forty-five degrees of flexion at the end of terminal swing. In post treatment analysis, the left knee began mid-swing with about sixty-five degrees of knee flexion demonstrated a moderately rapid extension similar to pre-treatment analysis ending terminal swing at about forty-five degrees of flexion. In neither pre- nor post treatment analysis did the left knee achieve the critical event of rapid extension of twenty-five degrees during mid-swing and complete extension during terminal swing.

In pretreatment analysis, the right knee began mid-swing with sixty degrees of flexion. The right knee then demonstrated rapid extension to nearly forty degrees of flexion at the end of terminal swing. The results of the post treatment analysis were nearly identical throughout mid- and terminal swing as described in pretreatment analysis. In neither pre- nor post treatment analysis did the left knee achieve the critical event of rapid extension of twenty-five degrees during mid-swing and complete extension during terminal swing.
Requirement Four

The fourth requirement of gait is adequate step length of the advancing limb. At the knee the critical event is rapid extension of twenty-five degrees during mid-swing and complete extension during terminal swing. Refer to Figure 14 for the subject’s kinematic graphs at the knee for pre- and post treatment analysis. The results of pre- and post treatment analysis of both the right and left knee are identical to the third requirement of gait and therefore the results will not be reported again.

For the fourth requirement of gait, the critical event at the hip is fifteen degrees of flexion during initial swing and to achieve twenty-five degrees of flexion during mid-swing. Refer to Figure 15 for the subject’s kinematic graphs at the hip for pre- and post treatment analysis. In pretreatment analysis, the left hip began initial swing at approximately thirty-five degrees of flexion. Throughout initial and mid-swing the left hip rapidly flexed to greater than sixty degrees of hip flexion at the end of mid-swing. In post treatment analysis, the left hip began initial swing slightly more extended at about thirty degrees of flexion but also rapidly flexed to a position of greater than sixty degrees at the end of mid-swing.
During mid-swing, the left hip flexed to greater than sixty degrees at the end of this phase. In neither pre- nor post treatment analysis did the left hip achieve the critical event of fifteen degrees of flexion during initial swing or achieve twenty-five degrees of flexion during mid-swing.

In pretreatment analysis, the right hip began initial swing in approximately twenty-two degrees of hip flexion. Throughout initial and mid-swing the right hip rapidly flexed to approximately forty degrees. In post treatment analysis, the right hip began initial swing in the identical position as in the pretreatment analysis. During mid-swing the right hip flexed to about fifty degrees at the end of the phase. In neither pre- nor post treatment analysis did the right hip achieve the critical event of fifteen degrees of flexion during initial swing or achieve twenty-five degrees of flexion during mid-swing.

Figure 15. Requirement Four, Critical Event Two
CHAPTER 5
DISCUSSION

Requirement One

The first requirement of gait is the stability of the weight-bearing foot throughout the mid-stance phase of gait. Movement at the ankle during mid-stance phase of gait is the most critical event for this requirement. To assist the body in momentum, the tibia must pass over the ankle creating what is called the second ankle rocker. A functional ankle rocker is identified by the ankle dorsiflexing from a near neutral position to almost ten degrees during mid-stance phase of gait.

In neither pre- nor post treatment analysis, did the left ankle demonstrate adequate dorsiflexion for a second ankle rocker to occur. The most likely reason for the lack of dorsiflexion in the left ankle during mid-stance was due to the presence of an extensor synergy. This conclusion is supported by the amount of rapid knee extension occurring in the same timeframe. Post treatment analysis of the left ankle did demonstrate the possibility of mild improvement in motor control during mid-stance, but the ankle still did not demonstrate the amount of dorsiflexion needed for the ankle rocker to occur. Other factors, which may be contributing to the lack of dorsiflexion being seen at the left ankle during mid-stance, include increased spasticity of the triceps surae and a five to ten degrees of dorsiflexion, which were both identified in pre- and post treatment clinical examinations.
In neither pre- nor post treatment analysis did the right ankle demonstrate adequate dorsiflexion for a second ankle rocker to occur. The right ankle did not demonstrate an extensor synergy pattern as seen in the left ankle. Instead in both pre- and post treatment analysis, the right ankle maintained a neutral position throughout mid-stance. The lack of motion at the right ankle is likely due to moderate spasticity of the triceps surae that was identified in the clinical examination.

The importance of knee extension during mid-stance is to provide stability for the limb as it advances over the ankle. Initially, stability of the knee in mid-stance is accomplished by the activation of the quadriceps. Stability of the knee throughout the remainder of mid-stance is then accomplished by an extension torque created at the knee controlled by knee ligaments, coupled with calf muscle activity. Therefore, if adequate knee extension is not achieved during mid-phase, the stability of that limb may be limited.

In both pre- and post treatment analysis, the left knee demonstrated an adequate amount of extension to ensure at least minimal stability of the left lower extremity during mid-stance. In pre-treatment analysis, the left knee began mid-stance with an excessive amount of flexion due to a flat foot initial contact. Once contact was made during the loading response the left knee then rapidly extended to a near neutral position at the end of mid-stance. As discussed previously, this rapid extension is likely due to an extensor synergy pattern, which was also identified in the left ankle. Clinical examination data also
identified spastic rectus femoris, which also could be contributing to the rapid knee extension during mid-stance.

In post treatment analysis, flexion of the left knee at initial contact was not as excessive as in the pretreatment analysis. The left knee rapidly extended during loading response allowing the fifteen degrees of flexion at the beginning of mid-stance. However, throughout mid-stance the left knee demonstrated only minimal extension, but enough to likely create an external flexor moment at the knee. Again, an extensor synergy is likely causing part of the extension seen especially during loading response. Since the subject demonstrated mild flexion throughout mid-stance, it is likely that limited motor control combined with tight hamstrings may be causing the inadequate extension needed for increased stability. In the left lower extremity, moderate spasticity of the hamstrings was identified in both pre- and post treatment clinical examination.

In pretreatment analysis, the right knee demonstrated an excessive amount of extension but with poor control to ensure at least minimal stability of the right lower extremity during mid-stance. However, in post treatment analysis the right knee did demonstrate more control during extension in order to provide minimal stability of the right lower extremity. The right knee in both pre- and post treatment analysis demonstrated a rapid excessive amount of extension through the loading response until the beginning of mid-stance. In pretreatment analysis, however, the right knee continued rapidly towards neutral in a likely uncontrolled movement. The right knee in pretreatment analysis maintained this locked neutral position throughout mid-stance. A probable reason for the poor control of
the right knee in pretreatment analysis is likely due to an primitive extensor synergy combined with poor motor control. In post treatment analysis, improved control of the right knee occurred during extension. Throughout mid-stance in the post treatment analysis the right knee did demonstrate more control of extension. More controlled extension could possibly be due to either improved motor control or decreased tightness in hamstrings and was identified in the clinical examination.

At the hip, the critical event is hip extension occurring during mid-stance. Extension of the hip is related to pelvic and trunk stability for both the stance limb and contralateral advancing limb. In both pre- and post treatment analysis, the left hip did not demonstrate sufficient hip extension to ensure stability of the pelvis. The most likely reason for the lack of hip extension in mid-stance is marked weakness of hip extensors, which was identified in the clinical examination. Mild spasticity noted in the clinical examination of the hip flexors likely had very little affect on the limitation of hip extension.

The right hip in pretreatment analysis did demonstrate an adequate amount of hip extension to ensure pelvic stability during mid-stance. However, in post treatment analysis the right hip demonstrated less extension then in pretreatment analysis and therefore, did not meet the requirements of the critical event. Similar to the left hip, the right hip extensors demonstrated marked weakness in both pre- and post treatment analysis. The right ankle in both pre- and post treatment analysis maintained a neutral position and therefore, allowed an increased amount of hip extension to occur. The reasons for the difference
between the amount of right hip extension pre- and post treatment is unclear, since muscle strength, range of motion and spasticity did not change.

**Requirement Two**

The second requirement of gait is the clearance of the non-weight bearing foot during the swing phase of gait. The critical event for this requirement includes a coupled action of ankle plantarflexion, knee and hip flexion to ensure the advancing foot does not contact the floor during swing limb advancement. The combination of these three critical events during swing phase of gait in a normal person creates a movement pattern to ensure clearance of the advancing limb. This same general movement in an individual with CP represents a primitive flexor synergy movement pattern driven by the CNS to also ensure clearance of the advancing limb.

In both the right and left lower extremities, a primitive flexion synergy pattern was evident. During initial swing, the left ankle in both pre- and post treatment analysis demonstrated only a minimal amount of dorsiflexion in a very small initial swing phase. This finding may be partially due to a functionally non-existent plantarflexion during preswing caused by marked weakness in the triceps surae as reported in clinical examination. Even though only a minimal amount of dorsiflexion did occur in both pre- and post treatment analysis, it was enough to ensure that the toe of the foot did not make contact during limb advancement.

The right ankle in pre- and post treatment analysis demonstrated only a minimal amount of dorsiflexion in comparison to the left ankle, as well as a significantly shorter initial swing phase. Similar to the right ankle there was no
evidence of a functional toe off prior to initiation of swing. The likely cause of the limited amount of dorsiflexion is due to poor motor control coupled with moderate triceps surae spasticity, as identified in the clinical examination.

Normally, rapid knee flexion during preswing is the critical event needed to ensure the clearance of the advancing foot. In both pre- and post treatment analysis, the left knee did not demonstrate rapid flexion during preswing, which is noted graphically by a gradual increase in the slope line. Since knee flexion is part of a functional synergy, the limitation of rapid flexion is likely related to rectus femoris spasticity.

However, in the later phase of preswing the right knee in both pre- and post treatment analysis did demonstrate rapid flexion to ensure clearance of the foot. During the first third of preswing, the right knee initially demonstrated only a few degrees of flexion which would not have been enough to ensure adequate clearance of the advancing foot. However, the right knee then rapidly flexed throughout the remainder of the phase to ensure clearance of the advancing foot. This slow initiation of the rapid knee flexion is likely caused by rectus femoris spasticity as noted in the clinical examination. The flexor synergy also is likely to be contributing to the rapid flexion especially since a marked amount of right hip flexion occurred in the same time frame.

An adequate amount of hip flexion during initial swing is critical to help ensure the clearance of the advancing limb. The critical event at the hip is to achieve and maintain thirty degrees of flexion during initial swing, allowing the knee to extend in preparation for initial contact. In both pre- and post treatment analysis,
the left hip demonstrated an excessive amount of hip flexion throughout a very short initial swing phase. Again, this rapid hip flexion is likely due to a primitive flexor synergy. However, the hip flexion was not controlled and maintained at thirty degrees for the knee to properly extend. Therefore, the hip did not achieve the critical event for this requirement of gait. Poor selective motor control could possibly be contributing to the inability of the left hip to achieve and maintain thirty degrees of flexion. Throughout the remainder of swing, the left hip continues to demonstrate greater than sixty degrees of flexion.

In both pre- and post treatment analysis, the right hip demonstrated an excessive and rapid amount of hip flexion throughout a very short initial swing phase, but the hip was unable to maintain this position. The increased amount of hip flexion was likely compensatory for the limited amount of rapid knee flexion. Also, poor motor control coupled with the flexor synergy ensures foot clearance. Since the right hip was unable to maintain a controlled flexed position, the right hip in pre- or post treatment analysis did not achieve this critical event.

Requirement Three

The third requirement of gait is proper positioning of the advancing foot during terminal swing. Positioning of the foot in neutral and full knee extension is critical to ensure heel first contact in order to correctly produce the heel rocker action that occurs during loading response. Also, the proper positioning of the foot for initial contact helps to eliminate possible instability and balance while transitioning from swing to stance phase of gait. Critical events at the ankle,
knee and hip must occur to ensure proper positioning of the foot in preparation for initial contact.

In neither pre- nor post treatment analysis did the left ankle maintain a neutral position throughout terminal swing phase of gait. The likely cause of the inability for the left ankle to achieve and maintain a neutral position is due primarily to poor motor control. Throughout swing phase, the flexor and extensor synergy patterns are likely controlling most of the movements in the lower extremities. Therefore, for the ankle to accomplish this critical event, the subject would have to selectivity move the ankle out of the synergy pattern and maintain a neutral position. Since this did not occur, one can assume that the proper motor control on the distal joints was inadequate. This conclusion is supported by knee extension and ankle plantarflexion occurring during terminal swing, both components of an extensor synergy pattern. Also contributing to the inability to maintain a neutral position is plantar flexion spasticity and weak dorsiflexors, which were both identified in the clinical examination.

Similar to the left, the right ankle in neither pre- nor post treatment analysis maintained a neutral position throughout terminal swing phase of gait. The right ankle did demonstrate a limited amount of dorsiflexion at the beginning of a very short terminal swing. Again, movement of the right ankle into plantarflexion in terminal swing is likely caused by the presence of an extensor synergy, similar to the left ankle. The limitation in the amount of movement during terminal swing in comparison to the left ankle is probably caused by an increase in plantarflexor spasticity combined with moderately weak dorsiflexors. Even though the right
ankle did pass neutral at the end of terminal swing, it does not position the foot properly for initial contact.

At the knee, the critical event which must occur to help ensure the proper positioning of the foot during initial contact is the rapid extension to a neutral position through mid and terminal swing phase of gait. In either pre- nor post treatment analysis did the left knee extend to less then forty degrees at the end of terminal swing in preparation for initial contact. Again, the presence of a primitive flexor synergy is likely creating the excessive knee flexion during mid and terminal swing. However, spastic hamstrings could also be contributing to the inability of the left knee to achieve a neutral position. This inability to achieve a neutral position during terminal swing does not allow the foot to be positioned properly for initial contact.

The right knee also demonstrated in pre- and post treatment analysis an inability to extend to a neutral position in preparation for initial contact. Mid- and terminal swing in the right knee were significantly shorter than in the left knee and therefore, limiting the amount of time needed for adequate extension. Again, similar to the left, movement of the right knee in mid- and terminal swing is likely being initiated by a primitive flexor synergy, which does not allow for the adequate position of the foot for initial contact.

**Requirement Four**

The fourth requirement of gait is the adequate step length of the advancing limb. The importance of adequate step length is twofold. The first issue is to ensure balance while ambulating. An individual with a shortened step length also
will have a decreased base of support and is therefore, likely to demonstrate problems in balance while ambulating creating issues of patient safety. The second issue is the change in temporal and spatial parameters, which occur and cause an increase in the amount of energy used during functional activities such as ambulation.

The critical event at the knee for the fourth requirement of gait is identical to the previous requirement of proper foot positioning for initial contact. In this requirement, the extension of the knee ensures that an adequate step length does occur. Therefore, the inability for the knee to properly extend dramatically alters the temporal and spatial parameters causing an increased amount of energy expenditure. Even though energy expenditure was not evaluated in this case report, one can assume that due to the significantly shortened step length the energy expenditure of the subject to ambulate would be increased.

The critical event at the hip for the fourth requirement of gait is to achieve and maintain thirty-five degrees of flexion during initial and mid-swing. The importance of this critical event is to provide a flexed position of the upper leg to allow for the extension of the knee during swing. Even if the knee is able to completely extend during swing phase, without adequate hip flexion the step length will be extremely limited.

In both pre- and post treatment analysis, the left hip rapidly exceeded thirty-five degrees of flexion during initial and mid-swing ending at over sixty degrees of flexion. However, in both cases, the left hip did not maintain a flexed position. Instead the left hip demonstrated rapid flexion throughout initial and mid-swing
phases of gait and therefore, did not achieve the critical event at the hip. The likely reason for the excessive hip flexion during swing phase is due to a primitive flexor synergy pattern, which was present throughout the swing phase.

The right hip, in both pre- and post treatment analysis, also demonstrated a mild amount of hip flexion during initial and mid swing but never maintained a flexed position. In pretreatment analysis, the amount of hip flexion was close to the acceptable range identified by the critical event possibly because of improved selective control over a primitive flexor synergy. But due to a shorter than normal swing phase the left hip in pretreatment analysis was unable to demonstrate the ability to maintain a flexed position and therefore did not meet the requirements of the critical event for this requirement. In post treatment analysis, the right hip exceeded fifty degrees of hip flexion during initial and mid swing. Again the likely reason for the excessive and rapid hip flexion is due to the presence of a primitive flexor synergy pattern.

Summary of Findings

In post treatment gait analysis of the subject following the Adeli treatment only requirements one and two successfully achieved any of the criteria outlined in Chapter 3. The first successful achievement was at the knee in requirement one. The right knee in pretreatment analysis was unable to extend to a near neutral position prior to the Adeli treatment. However, following the Adeli treatment the right knee demonstrated the ability to extend to a nearly neutral position. The left knee in both pre- and post treatment analysis also demonstrated the ability to extend to a near neutral position during mid-stance. The second successful
achievement was the right knee in requirement two. The right knee in both pre- and post treatment analysis successfully achieved the critical event of rapid knee flexion to forty degrees during preswing phase of the gait cycle. Overall, in comparison to pre- and post treatment analysis of the subject following the Adeli treatment, there was no significant changes noted at the ankle, knee, or hip, based on the four requirements of gait outlined in Chapter 3.

**Clinical Implications**

There are three key clinical implications related to this case report and the Adeli treatment. The first is the lack of evidence supporting or describing the Adeli treatment as a viable treatment option for children with CP. As discussed in Chapter 3 the study by Semenova is the only evidence-based article within the literature. Therefore, this case report will add to a limited body of information, which currently exists pertaining to the Adeli treatment.

The second clinical implication is that the results of this case report do not demonstrate the effectiveness of the Adeli treatment as a viable treatment for children with CP. No significant changes occurred in subject's gait following the Adeli treatment based on data collected by computerized gait analysis. However, this is a single case report and illustrates the need for further studies to identify any possible benefits of the Adeli treatment.

Lastly is the lack of a clearly defined treatment protocol to include operational definitions for the individual treatment components of the Adeli treatment. As discussed previously, the treatment protocol established for Euro-med lacks scientific foundation. Furthermore, there are not clear operational definitions for
the numerous treatment components incorporated with the Adeli treatment. Without operationally defined treatment components, the validity and effectiveness of the Adeli treatment is hard to investigate the system thoroughly. Furthermore, without operational definitions of the different treatment components, researchers may have challenges supporting the validity of individual components. Therefore, one may question if the lack of effectiveness as seen in this case report would improve if the clinicians at Euro-Med utilized scientifically proven tone inhibitory techniques or gait-training activities with proven efficacy in combination with the Adeli suit? If a scientifically based treatment protocol with clearly identified operational definitions was utilized at Euro-Med the chance of improvement in the child's gait following the Adeli treatment could possibly have existed in this case report.

**Future Research**

The primary focus for future research is to investigate the active components of the Adeli treatment. As mentioned in Chapter Three, the Adeli suit in combination with numerous components which make up the Adeli treatment. There are two questions for future investigators to ask when evaluating the validity of the Adeli suit. First, do the components of the Adeli treatment actually accomplish what they are designed to do? An example is the technique utilized in the Adeli treatment to inhibit spasticity. The inhibitory techniques discussed in Chapter Two are not supported within the literature. Therefore, one must question the validity of the inhibitory techniques used in the Adeli treatment. Secondly, when the treatment components of the Adeli suit are combined, do
they function as a single treatment unit towards the same task or is there a possibility that the components are offsetting the effectiveness of each other? By conducting multiple case reports and randomized controlled studies, investigators can further evaluate the active components of the Adeli treatment to analyze the validity of the Adeli treatment.

Limitations

Limitations to this study include the following considerations. First, this is a retrospective case report with only one subject and no control. Since there is only one subject, any conclusions about the validity of the Adeli treatment cannot be made. Also, since this was a retrospective report the investigator had no control over the initiation of treatment. Secondly, no functionally based measures with proven validity and reliability were used to determine if functional improvements occurred following the Adeli treatment. Utilizing a functional measurement tool such as the PEDI could provide even more functional quantified data than what has been reported in this case report. Thirdly, only gait analyses of movements in the sagittal plane were evaluated for this case report. This limits the ability of the investigator to evaluate key movements in other planes. For example, the subject demonstrated weak bilateral hip abduction. Therefore, a trendelenburg gait pattern present in visual observations could not be identified or evaluated due to the lack of frontal view. Lastly, in neither pre- nor post- treatment analyses were energy expenditures evaluated.
Conclusions

The purpose of this case report was to identify if any changes occurred following the Adeli treatment in a child with spastic CP. Based on results of the data gathered by three-dimensional gait analysis and clinical examinations, the following conclusion were made: no measurable changes were evident in the four requirements of gait following the Adeli treatment based on the biomechanical analysis of a child's gait kinematics. Also, no measurable change occurred in temporal or spatial gait parameters or improvements of lower extremity impairments based on clinical examination following the Adeli treatment.

Based on the results of this case report, clinicians should take into consideration the lack of scientific evidence surrounding effectiveness of the Adeli treatment for a child with CP when advising parents who are inquiring about this costly and time intensive treatment option. Further research is required to support or further dispute the efficacy of the Adeli treatment as a viable option for children with CP.
Reference List


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<table>
<thead>
<tr>
<th>No.</th>
<th>Author(s)</th>
<th>Title</th>
<th>Journal</th>
<th>Year</th>
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<tr>
<td>37.</td>
<td>Semenava, K</td>
<td>Basis for a method of dynamic proprioceptive correction in the restorative treatment of patients with residual-stage infantile cerebral palsy</td>
<td>Neuroscience and Behavioral Physiology</td>
<td>1997;96:639-643</td>
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446. Pape KE. Therapeutic electrical stimulation the past, the present, the future. NDTA Network. 1996; July/August: 1-7.


466. Leyendecker CH. Electrical stimulation therapy and its effect on the general activity of motor impaired children with CP: a comparative study of Bobath
therapy in combination with the Hufschmidt electrical stimulation unit. European Journal of Rehabilitation. 1986;110;150-159.


Appendix A

Patient Consent Form

Mary Free Bed Hospital & Rehabilitation Center
Grand Valley State University
CENTER FOR HUMAN KINETIC STUDIES

I Sharon K. Appel do hereby authorize the full release of photographs taken of Randall McCombs. I give permission to the staff of Mary Free Bed Center For Human Kinetic Studies to use of the aforementioned pictures for scientific publications and presentations at public scientific meetings at the local and national level. In signing this release I understand that facial exposure of Randall is possible during the previously mentioned events and fully approve of this type of personal exposure. I also give Mary Free Beds Center For Human Kinetics studies full control of these pictures and allow them to use discretion in authorizing there use only in the previously mentioned fashion of which I have authorized.

Sharon K. Appel (mother)  Date

Witness  Date

2020 Raybrook S.E., Suite 101, Grand Rapids, Michigan 49546
Telephone (616) 954-2318 • Fax (616) 954-2475

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Appendix B

Pretreatment Analysis of Temporal and Spatial Parameters

<table>
<thead>
<tr>
<th>TEMPORAL AND SPATIAL GAIT PARAMETERS (Pre-RX)</th>
<th>LEFT SIDE</th>
<th>RIGHT SIDE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1ST DOUBLE SUPPORT (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SINGLE LIMB STANCE (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2ND DOUBLE SUPPORT (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STANCE (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SWING (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GAIT CYCLE TIME (SEC)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STEP LENGTH (CM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STRIDE LENGTH (CM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STEP WIDTH (CM)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CADENCE (STEPS/MI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VELOCITY (CM/SEC)</td>
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</table>

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## Appendix C

Post treatment Analysis of Temporal and Spatial Parameters

### TEMPORAL AND SPATIAL GAIT PARAMETERS (Post-RX)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Left Side</th>
<th>Right Side</th>
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<tbody>
<tr>
<td>1ST DOUBLE SUPPORT (%)</td>
<td>21.3</td>
<td>29.5</td>
</tr>
<tr>
<td>SINGLE LIMB STANCE (%)</td>
<td>24.3</td>
<td>26.5</td>
</tr>
<tr>
<td>2ND DOUBLE SUPPORT (%)</td>
<td>32.9</td>
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<tr>
<td>STANCE (%)</td>
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<td>86.7</td>
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<tr>
<td>SWING (%)</td>
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<td>GAIT CYCLE TIME (SEC)</td>
<td>2.49</td>
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<td>STEP LENGTH (CM)</td>
<td>31.9</td>
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<td>STRIDE LENGTH (CM)</td>
<td>61.3</td>
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<tr>
<td>STEP WIDTH (CM)</td>
<td>26.3</td>
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<td>CADENCE (STEPS/MIN)</td>
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<td>27.1</td>
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Appendix D

Gait Cycle Sub-phase Breakdown

Pre and Post Treatment Right Lower Extremity
Stance 86% Swing 14%

<table>
<thead>
<tr>
<th></th>
<th>LR</th>
<th>MSt</th>
<th>TSt</th>
<th>PSw</th>
<th>Isw</th>
<th>MSw</th>
<th>TSw</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Subphase Percent</td>
<td>12</td>
<td>19</td>
<td>19</td>
<td>12</td>
<td>13</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>Subject Subphase Percent</td>
<td>17</td>
<td>26</td>
<td>26</td>
<td>17</td>
<td>5</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Percent of the Gait cycle at which the Normal Subphase Ends.</td>
<td>12</td>
<td>31</td>
<td>50</td>
<td>62</td>
<td>75</td>
<td>87</td>
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<tr>
<td>Percentage of the Gait Cycle at which the Subject Subphase Ends</td>
<td>17</td>
<td>43</td>
<td>69</td>
<td>86</td>
<td>91</td>
<td>95</td>
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</table>

Pretreatment Left Lower Extremity
Stance 77% Swing 23%

<table>
<thead>
<tr>
<th></th>
<th>LR</th>
<th>MSt</th>
<th>TSt</th>
<th>PSw</th>
<th>Isw</th>
<th>MSw</th>
<th>TSw</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal Subphase Percent</td>
<td>12</td>
<td>19</td>
<td>19</td>
<td>12</td>
<td>13</td>
<td>12</td>
<td>13</td>
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<tr>
<td>Subject Subphase Percent</td>
<td>14.5</td>
<td>24</td>
<td>24</td>
<td>14.5</td>
<td>8</td>
<td>7</td>
<td>8</td>
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<td>Percent of the Gait cycle at which the Normal Subphase Ends.</td>
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<td>31</td>
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<td>62</td>
<td>75</td>
<td>87</td>
<td>100</td>
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<tr>
<td>Percentage of the Gait Cycle at which the Subject Subphase Ends</td>
<td>14</td>
<td>38</td>
<td>62</td>
<td>77</td>
<td>84</td>
<td>91</td>
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Post Treatment Left Lower Extremity
Stance 78% Swing 22%

<table>
<thead>
<tr>
<th></th>
<th>LR</th>
<th>MSt</th>
<th>TSt</th>
<th>PSw</th>
<th>Isw</th>
<th>MSw</th>
<th>TSw</th>
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</thead>
<tbody>
<tr>
<td>Normal Subphase Percent</td>
<td>12</td>
<td>19</td>
<td>19</td>
<td>12</td>
<td>13</td>
<td>12</td>
<td>13</td>
</tr>
<tr>
<td>Subject Subphase Percent</td>
<td>15</td>
<td>24</td>
<td>24</td>
<td>15</td>
<td>7.5</td>
<td>7</td>
<td>7.5</td>
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<tr>
<td>Percent of the Gait cycle at which the Normal Subphase Ends.</td>
<td>12</td>
<td>31</td>
<td>50</td>
<td>62</td>
<td>75</td>
<td>87</td>
<td>100</td>
</tr>
<tr>
<td>Percentage of the Gait Cycle at which the Subject Subphase Ends</td>
<td>15</td>
<td>39</td>
<td>63</td>
<td>78</td>
<td>85.5</td>
<td>92.5</td>
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## Appendix E

### Pretreatment Clinical Examination

<table>
<thead>
<tr>
<th>Patient Name:</th>
<th>Chart Number:</th>
<th>Test Date: 4.23.98 (Pre-treatment)</th>
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<tr>
<td></td>
<td></td>
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</tbody>
</table>

### HIPS

<table>
<thead>
<tr>
<th>Motion</th>
<th>Selectivity/Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>L</td>
</tr>
<tr>
<td>Flexion(120-135)</td>
<td>Normal</td>
</tr>
<tr>
<td>Extension</td>
<td>Knee@0 (10-30)</td>
</tr>
<tr>
<td>Knee@90</td>
<td>Normal</td>
</tr>
<tr>
<td>Thomas test(0)</td>
<td>10: 20 RF</td>
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</tbody>
</table>

### Abduction

<table>
<thead>
<tr>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hips@0 (30-50)</td>
<td>15</td>
</tr>
<tr>
<td>Hips@45</td>
<td>50</td>
</tr>
<tr>
<td>Adduct@10-30</td>
<td>Normal</td>
</tr>
<tr>
<td>Int. Rot (30-45)</td>
<td>90</td>
</tr>
<tr>
<td>Ext. Rot (30-45)</td>
<td>40</td>
</tr>
<tr>
<td>Antever (15-25sil)</td>
<td>52</td>
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</tbody>
</table>

### KNEE

<table>
<thead>
<tr>
<th>Motion</th>
<th>Selectivity/Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>L</td>
</tr>
<tr>
<td>Flex.sup.(135-160)</td>
<td>Normal</td>
</tr>
<tr>
<td>Flex.prox(135)</td>
<td>Normal</td>
</tr>
<tr>
<td>Extension (0)</td>
<td>Unilateral(0-30)</td>
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<tr>
<td>Bilateral(0-20)</td>
<td>10</td>
</tr>
<tr>
<td>SLR (70-90)</td>
<td>70</td>
</tr>
<tr>
<td>F/T Ang (15ER)</td>
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### Ankle

<table>
<thead>
<tr>
<th>Motion</th>
<th>Selectivity/Strength</th>
</tr>
</thead>
<tbody>
<tr>
<td>R</td>
<td>L</td>
</tr>
<tr>
<td>Dorsiflex (Knee@0)</td>
<td>0</td>
</tr>
<tr>
<td>Plantarflex(45-50)</td>
<td>Normal</td>
</tr>
<tr>
<td>Inversion(35-50)</td>
<td>15</td>
</tr>
<tr>
<td>Eversion(15-30)</td>
<td>40</td>
</tr>
</tbody>
</table>

### Comments:
- Right more forefoot dorsiflexion than hindfoot.
- Moderate hallux valgus bilaterally. 1 cm leg length discrepancy (L > R).

### Foot Position

<table>
<thead>
<tr>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>WTBAB</td>
<td>Hind.</td>
</tr>
<tr>
<td>Midft. Mark Plan</td>
<td>Mark Plan</td>
</tr>
<tr>
<td>Foreft. Mod. Abd</td>
<td>Mod. Abd</td>
</tr>
<tr>
<td>Plant pos Mod. ER</td>
<td>Mod. ER</td>
</tr>
<tr>
<td>NWB</td>
<td>Hind.</td>
</tr>
<tr>
<td>Correct</td>
<td>-</td>
</tr>
<tr>
<td>Midft. Mark Plan</td>
<td>Mark Plan</td>
</tr>
<tr>
<td>Correct</td>
<td>No</td>
</tr>
<tr>
<td>Foreft. Mod var</td>
<td>Mod Var</td>
</tr>
<tr>
<td>Correct</td>
<td>Yes</td>
</tr>
</tbody>
</table>

### Spasticity

<table>
<thead>
<tr>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hip Flex.</td>
<td>2</td>
</tr>
<tr>
<td>Adductors</td>
<td>3</td>
</tr>
<tr>
<td>Hamsliga</td>
<td>4</td>
</tr>
<tr>
<td>Dunc-Ely</td>
<td>4</td>
</tr>
<tr>
<td>Plant.If</td>
<td>4</td>
</tr>
<tr>
<td>Clonus</td>
<td>1/1</td>
</tr>
<tr>
<td>Post. Tib</td>
<td>1</td>
</tr>
<tr>
<td>(+) Confus</td>
<td>Yes</td>
</tr>
</tbody>
</table>

### KEY: Spasticity
- 0 - Hypotonic(floppy)
- 1 - Normal
- 2 - Mild(min.resist.)
- 3 - Moderate(pass.mov't)
- 4 - Severe(diff.pass.mov't)
- 5 - Extreme(right)

### KEY: Selectivity
- 0 - only patterned mov't
- 1 - partially isol. mov't
- 2 - complete isol. mov't

### KEY: Strength
- 0 - Flaccid
- 1 - Trace
- 2 - Poor
- 3 - Fair
- 4 - Good
- 5 - Normal

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### Appendix F

**Post treatment Clinical Examination**

#### CLINICAL EXAMINATION RESULTS

<table>
<thead>
<tr>
<th>Patient Name:</th>
<th>Chart Number:</th>
<th>Test Date: 6/12/95 (Post-treatment)</th>
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</table>

<table>
<thead>
<tr>
<th>Motion</th>
<th>Selectivity/Strength</th>
<th>R</th>
<th>L</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>HIPS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flexion (120-135)</td>
<td>Normal</td>
<td>Normal</td>
<td>1/3+</td>
<td>1/3+</td>
<td></td>
</tr>
<tr>
<td>Extension</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee@90 (19-30)</td>
<td>10</td>
<td>9</td>
<td>1/2</td>
<td>1/2</td>
<td></td>
</tr>
<tr>
<td>Knee@90</td>
<td>10</td>
<td>9</td>
<td>Unable</td>
<td>Unable</td>
<td></td>
</tr>
<tr>
<td>Thomas test (9)</td>
<td>0</td>
<td>Mild Rep.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

| Abduction |                      |   |   |   |   |
| Hips@80 (30-50) | 20 | 20 | 1/2 | 1/2 |
| Hips@45 | 45 | 45 |       |       |
| Adduct (10-30) | Normal | Normal | 1/3 | 1/3 |
| Ext. Rot (30-45) | 80 | 80 |       |       |
| Int. Rot (30-45) | 20 | 20 |       |       |
| Antever (15-25%°) | 50 | 50 |       |       |

| KNEE |                      |   |   |   |   |
| Flex sup (135-160) | Normal | Normal | 1/3+ | 1/3+ |
| Flex, prone (<135) | Normal | Normal | 1/4+ | 1/4+ |
| Extension (0) | Normal | Normal |       |       |
| Popliteal angle |                      |   |   |   |   |
| Lateral (0-30) | 40 | 50 |       |       |
| Medial (0-20) | 30 | 30 |       |       |
| IR (70-90) | 50 | 50 |       |       |
| K/T Ang (=15°) | 5 | 5 |       |       |

| Ankle |                      |   |   |   |   |
| Dorsiflexion |                       |   |   |   |   |
| Knee@90 (20-30) | 5 | 10 | 1/3 | 1/3 |
| Knee@90(20-30) | 10 | 13 | 1/3 | 1/3 |
| Plantarflex (45-50) | Normal | Normal | 1/2 | 1/2 |
| Inversion (35-50) | 20 | 15 | 1/1 | 1/2 |
| Eversion (35-50) | 30 | 30 | 1/1 | 1/1 |

#### Foot Position

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>WTBear</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HInd</td>
<td>Neutral</td>
<td>Normal</td>
</tr>
<tr>
<td>MInd</td>
<td>Mark Plan</td>
<td>Mark Plan</td>
</tr>
<tr>
<td>FInd</td>
<td>Mod. Abd.</td>
<td>Mod. Abd.</td>
</tr>
<tr>
<td>Foot pos</td>
<td>Mid ER</td>
<td>Mid ER</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
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</thead>
<tbody>
<tr>
<td>NWB</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HInd</td>
<td>Neutral</td>
<td>Neutral</td>
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<tr>
<td>MInd</td>
<td>Mark Plan</td>
<td>Mark Plan</td>
</tr>
<tr>
<td>CInd</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

#### Spasticity

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
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</thead>
<tbody>
<tr>
<td>Hip Flex.</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Adductors</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Hamstrings</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Dors-Ext</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Plant. Fl.</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Crosse</td>
<td>(variable)</td>
<td>1</td>
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<tr>
<td>Post. Tib.</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>(*) Cords.</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

#### KEY: Spasticity

- 0: Hypotonic/Hyperplastic
- 1: Normal
- 2: Mild (min resist)
- 3: Moderate (pass. mov’t)
- 4: Severe (diff pass. mov’t)
- 5: Extreme (rigid)

#### KEY: Selectivity

- 0: only patterned mov’t
- 1: partially isolated mov’t
- 2: complete isolated mov’t

#### KEY: Strength

- 0: Flaccid
- 1: Trace
- 2: Poor
- 3: Fair
- 4: Good
- 5: Normal

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