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ABSTRACT

THE EFFECT OF MECHANICAL VESTIBULAR STIMULATION ON MUSCLE TONE, SPASTICITY and DYSTONIA IN INDIVIDUALS WITH NEUROLOGICAL IMPAIRMENT

By
Ghaith J. Androwis

In the desire to better understand spasticity mainly in Cerebral Palsy (CP) and to examine vestibular stimulation as a future intervention for .muscle tone reduction, and to be able to describe the change in level of spasticity in subjects with disability and describe interventions effects, a series of experiments are done on children with spasticity. In addition to understanding the otoliths in the vestibular system and their projections, properties and pathways a more important major objective of this work is to validate the changes in otoliths signal caused by vestibular stimulation based on the Equilibrium Point Hypothesis and the inclusion of EMG data in assessing the level of spasticity.

Stimulation to the saccule in the otolith is induced to reduce spasticity. The otoliths are sensitive to acceleration, and detect the direction and magnitude of gravity, as well as transient linear accelerations due to movement. This is a form of a biological accelerometer. The vestibular mechanical stimulation is provided using a vertical stimulation chair that moves up/down at a constant frequency of 2 Hz and amplitude of ~ 7.5 centimeter for time duration of 15 minutes. This form of stimulation targets the saccule organ in the vestibular system, which results in alterations of the descending signals of the vestibular system responsible for setting tone of the antigravity muscles. Electromyography (EMG) is simultaneously recorded from the quadriceps (Vastus Lateralis) and hamstring (Biceps Femoris) muscles along with the PKD test. The

activation of EMG during PKD can be understood in relationship to the flexion and extension of the lower leg. It is interesting that EMG activity for quadriceps is seen at every flexion cycle in the post stimulation data, while on the other hand EMG activity is nearly continuous in the initial cycles of PKD in the pre stimulation. This may be an indication of a change in the activation pattern of EMG from the agonist and antagonist muscles as a result of the vestibular stimulation, which is caused by neural changes in the vestibular descending signal.

Preliminary studies done on subjects without disability comparing NASA jump test pre and post riding a rollercoaster indicate that there are significant differences in the knee and hip angles, which can be explained as a result of the change in muscle tone caused by the exposure to microgravity or cyclic gravity while being on rides. A preliminary study done on a 35 year old subject with CP, showed promising results in reducing spasticity after stimulating the vestibular system using the vestibular stimulation chair. Data from the Pendulum Knee Drop (PKD) test show a significant reduction in the knee stiffness (K), and virtual trajectory (θ_{vt}) that is noticed as a change in the shape of knee trajectory post stimulation when compared to pre stimulation.

The final work presented in this study includes seven subjects with spasticity due to cerebral palsy. The PKD test, along with EMG, is used to assess their level of spasticity. Alterations to the vestibular descending signals while passing through the vestibular nuclei and going down toward the alpha motor neurons command a change in the muscle activation patterns that are responsible for setting the level of spasticity or muscle tone. Furthermore, this effect was found to be retained for at least 15 minutes post stimulation. One subject's data is excluded from the study due to her high initial baseline

measure of muscle tone and spasticity which is determined with the extreme firing of EMG bursts. In all the other six subjects of this study, the knee stiffness and damping parameters show a dramatic decrease post vestibular stimulation, and a smaller change is also noticed in the virtual trajectory (θ_{vt}) specifically in two subjects who have no dystonia. Four of the subjects have dystonic spasticity and the other three do not have dystonia along with spasticity. The vestibular stimulation effect is different between the two groups, and it is found that stimulation do not have the same effect on the level of dystonia as much as it do on spasticity. Subsequent analyses of EMG lead to a potential linkage between the EPH and the muscle reflexes (EMG).

It is important to note that: 1) this work cannot be claimed as a permanent treatment for children with CP, but a combination of the described stimulation along with the proper physical therapy might have a very positive effect on the disorder. 2) Another path that can have a similar impact on the described population is by changing the stimulation duration intensity and providing the stimulation more frequently for at least five consecutive weeks might have a major impact on reducing the level of spasticity in children with CP.

THE EFFECT OF MECHANICAL VESTIBULAR STIMULATION ON MUSCLE TONE AND SPASTICITY IN INDIVIDUALS WITH NEUROLOGICAL IMPAIRMENT

by
Ghaith J. Androwis

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THE EFFECT OF MECHANICAL VESTIBULAR STIMULATION ON MUSCLE TONE AND SPASTICITY IN INDIVIDUALS WITH NEUROLOGICAL IMPAIRMENT

Ghaith J. Androwis

Dr. Richard Foulds, Dissertation Advisor — Date
Associate Professor of Biomedical Engineering, NJIT

Dr. Sergio Adamovich, Committee Member — Date
Associate Professor of Biomedical Engineering, NJIT

Dr. Allan Strongwater, Committee Member — Date
Clinical Professor of Orthopedic Surgery,
Chief, Pediatric Orthopedic Surgery, St. Joseph Hospital- Paterson, NJ

Dr. Jorge Serrador, Committee Member — Date
Associate Professor Pharmacology and physiology,
Associate Director of Research, Rutgers

Dr. Gerard G. Fluet, Committee Member — Date
Assistant Professor of Rehabilitation and Movement Sciences, Rutgers

BIOGRAPHICAL SKETCH

Author: Ghaith J. Androwis

Degree: Doctor of Philosophy

Date: January 2014

Undergraduate and Graduate Education:

- Doctor of Philosophy in Biomedical Engineering,
  New Jersey Institute of Technology and Rutgers -
  The State University of New Jersey, Newark, NJ, 2014

- Master of Science in Biomedical Engineering,
  New Jersey Institute of Technology, Newark, NJ, 2009

- Bachelor of Science in Biomedical Engineering,
  New Jersey Institute of Technology, Newark, NJ, 2009

Major: Biomedical Engineering

Presentations and Publications:

Androwis. G. J., Michael. P. A., Foulds. R. A. (2013), Alterations of Neuromuscular Signals as a Result of Vestibular Stimulation, *Proceedings from NER 2013: The* 6th international IEEE EMBS Neural Engineering Conference.

Androwis. G. J., Foulds. R. A., Strongwater. A., Stone. D. (2013), Quantifying the Effect of Mechanical Vestibular Stimulation on Muscle Tone and Spasticity, *Proceedings from NEBEC 2013: The IEEE 39th Annual Northeast Bioengineering Conference.*

Androwis. G. J, Jewaid. D. I, Foulds. R. A, (2013), The Neural Contribution to Passive Joint Movement in Individuals with Cerebral Palsy, *Proceedings from NEBEC 2013: The IEEE 39th Annual Northeast Bioengineering Conference.*

Androwis. G. J, Simon. D, Foulds. R. A (2012), Assessment of Passive Knee Stiffness and Set Point, *Proceedings from NEBEC 2012: The IEEE 38th Annual Northeast Bioengineering Conference.*

Simon. D, Androwis. G. J, Foulds. R. A (2011) Equilibrium Point Model of Knee Joint Spasticity *Proceedings from NEBEC 2011: The IEEE 37th Annual Northeast Bioengineering Conference.*

Redding, M.J., Androwis. G. J , Foulds. R. A (2009), Artificial Limb for treadmill Locomotors Training with Partial Weight Support, *Proceedings from NEBEC 2009: The IEEE 35th Annual Northeast Bioengineering Conference.*

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LIST OF SYMBOLS

~	Approximately
θ_{vt}	Virtual trajectory assigned by CNS.
θ	Theta (angle measured in radian)
K	Stiffness (knee stiffness), (N-m/rad)
B	Damping (knee damping), (N-m-sec/rad)
PKD	Pendulum Knee Drop Test.
CNS	Central Nerves System.
EMG	Electromyography
CP	Cerebral Palsy
TM_{VT}	Time to the inflection point of the
EPH	Equilibrium Point Hypothesis
(F-M) Scale	Fahn-Marsden Scale
UDRS	Unified Dystonia Rating Scale
GDS	Global Dystonia Rating Scale
Ha	Hip Angle
Ka	Knee Angle
αMN	Alpha motoneurons

CHAPTER 1

INTRODUCTION

1.1 Objectives and Specific Aims

The life-long disability which occurs as a result of brain damage before, during, or right after birth is a description of Cerebral Palsy (CP). This disorder affects about one in every 303 children in the United States [1]. It affects movements, posture, normal muscle growth and muscle lengthening during daily activity. The objectives of this work are to 1) better understand spasticity, 2) develop new methods and techniques to reduce muscle tone in spastic population mainly Cerebral Palsy (CP), 3) to understand vestibular stimulation as a future intervention for spasticity, 4) to be able to describe the level of spasticity in subjects with disability and describe intervention's effects. In addition to understanding the otoliths in the vestibular system and their projections, properties and pathways a more important major objective of this work is to validate the changes due to vestibular stimulation to otoliths based on the Equilibrium Point Hypothesis and the inclusion of EMG data in assessing the level of spasticity. The following are the main aims of this work.

Specific aim I: Vestibular stimulation of subjects without spasticity (5 subjects). Vertical stimulation on five subjects without CP was provided and changes in the PKD test is studied to establish a baseline for any changes that might occur in children with CP. The vestibular stimulation consists of 7.5 cm of vertical oscillation for 15 minutes at 1.5 Hz

Specific aim II: Vestibular stimulation of a disabled subject (1 subject). Vertical stimulation of one subject with CP is done and changes in the PKD test were studied. This component of the study will allow the final refinements of experimental procedures and platform design. The vestibular stimulation consists of 7.5 cm of vertical oscillation for 15 minutes at 1.5 Hz

*Specific aim III and IV: Vestibular stimulation of children with disability (1 subject).*Vertical stimulation of one subject with CP was provided and changes to the PKD test were evaluated. The vestibular stimulation consists of 7.5 cm of vertical oscillation for 15 minutes at different frequencies (1, 2) Hz; in order to confirm a more physiologically matched frequency for vestibular stimulation.

Once a more physiologically matched frequency was determined, vestibular stimulation was provided and changes in PKD test were evaluated. The vestibular stimulation consists of 7.5 cm of vertical oscillation at the more physiologically matched frequency for different time (5, 15, 25) duration in order to confirm the best time duration for vestibular stimulation. The frequency and time duration were determined according to the greatest short term effect.

Specific aim V: Vestibular stimulation of children with disability (7 subjects). Vertical stimulation of 7 subjects with CP was provided and changes to the PKD test were evaluated. The vestibular stimulation consists of 7.5 cm of vertical oscillation for 15 minutes at 2Hz. The purpose of this study is to examine whether there are any significant changes in the muscle tone as a result of providing the mechanical vestibular stimulation

to both groups (Subjects with and without dystonia) and to be able to explain the results for each group, to differentiate between them in terms of the different kinematics noticed in the study. Another important point in this study is to explain the impact of vestibular stimulation on both groups and show the distinction among them.

1.2 Neuromuscular Disability

1.2.1 Background on Spasticity and Dystonia

Spasticity is considered a major disabling condition which alters functional activities. It is defined as increased muscle activity that is dependent on the velocity of the joint. It has also been referred to as velocity-dependent hypersensitivity of reflexes [2]. The range to which this excess muscle activity may interfere with motor control and function is debated in the literature. Spasticity is experienced by a large population of children with neuromotor disabilities such as cerebral palsy (CP). It is estimated that 75% of children with CP experience spasticity [3]. Similar spasticity is seen in the stroke population, but both are different from spasticity caused by spinal cord injury [4].

Dystonia is a disorder of persistent muscle contractions causing limbs to erratically move in the presence of agonist and antagonist co-contractions. Hypertonia might be produced as a result of dystonia whenever contractions are activated prior the presence of an external force to passively move a limb [5, 6]. Children with dystonia commonly have other features, including poor dexterity, and abnormal patterns of muscle activation. Eye movement and oromotor abnormalities are frequently associated, but these features do not distinguish dystonic hypertonia from other causes of hypertonia [7].

The presence of spasticity and dystonia is common in children with CP. Due to dystonia, communication between the relaxation and activation of the agonist and antagonist muscles are altered. Contractions result and individuals with dystonia are unable to voluntarily control it. This has an effect on the posture of the body and its normal movements.

For the purpose to correctly evaluating improvement outcomes caused by interventions, it is important to distinguish and quantify the differences between spasticity and dystonia [8]. Therefore, the goal of this study is to develop a deeper understanding of the roles of spasticity and motor control in limiting the function of children with cerebral palsy, and to explore a possible clinical procedure that reduces spasticity in individuals with neuromotor disabilities.

1.2.2 Common Techniques Used to Assess Spasticity

The most commonly used measurements of spasticity are the Tardieu scale, the Ashworth Scale and the modified Ashworth Scale. The latter is considered the current 'gold standard' in clinical, semi-subjective, assessment of spasticity. This is a test that is administered by a therapist, in which the affected joint of a subject is rotated at a moderate angular velocity. The therapist assigns a ranking to the muscle tone (mechanical resistance) which is recognized in the joint. The table below shows the range of the rankings and the general guidelines for assigning scores.

Table1.1 Ashworth and Modified Ashworth Scales

Score		Ashworth Scale (1964)	Modified Ashworth Scale Bohannon & Smith (1987)
Ashworth	Modified		
0	0	No increase in tone	No increase in muscle tone
1	1	Slight increase in tone giving a catch when the limb was moved in flexion or extension	Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension.
2	1+		Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the reminder (less than half) of the ROM (range of movement).
3	2	More marked increase in tone but limb easily flexed.	More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.
4	3	Considerable increase in tone - passive movement difficult.	Considerable increase in muscle tone passive, movement difficult.
5	4	Limb rigid in flexion or extension.	Affected part(s) rigid in flexion or extension.

Source: Pandyan, A.D., Johnson, G.R., Price, C.I.M., Curless, R.H., Barnes, M.P. and Rodgers, H. A review of the properties and limitations of the Ashworth & modified Ashworth scales as measures of spasticity. Clinical Rehabilitation, 13(5), 373-383. (1999).

The original Ashworth Scale ranked resistance or stiffness on a scale from 0-5. 0 indicates no increase in stiffness above what would be felt in a person without impairment. A score of 5 indicates that the joint is rigid and cannot be moved by the therapist. Values of 1, 2, and 3 represent a pseudo-linear increase in added resistance from no resistance to rigidity. As spasticity is considered by many to be a hypersensitive reflex triggered by velocity through neural activation there is concern that the increased resistance felt by the clinician is the result of higher muscle stiffness that is not directly due to spasticity of neural origin. Resistance could be caused by changes in the material properties of the muscle. Hence, the Modified Ashworth Scale introduced an additional ranking labeled 1+, to indicate that the therapist felt a change in resistance (a catch)

within the movement of the joint. This ‘catch’ is believed to be the reflex or rapid change in muscle tone triggered by reaching a velocity threshold [9].

Another measure of spasticity is the Tardieu Scale which addresses the effects of velocity and resistance to stretch in the assessment of muscle tone. The intensity of the resistance to stretching a muscle, the angle at which the catch is first appreciated, and the differences noted when a muscle is stretched at different velocities are the outcomes of the Tardieu scale [10]. It is believed that moving the limb at different velocities can more easily assess the response of the muscle to stretch since the stretch reflex responds differentially to velocity. The ability to objectify the velocity dependent nature of spasticity distinguishes the Tardieu scale from the Ashworth scales.

The Wartenberg pendulum test (Wartenberg, 1951) is commonly used to assess spasticity by measuring passive knee motion. In its original form, the trajectory of the knee was described in subjective terms. Several decades later Bajd and Vodovnik used the technique to quantify spasticity. This test was used to assess changes of the angular knee trajectory, passive stiffness and damping. The resistance to knee motion that is related to muscular, tendons, and properties of the knee tissue is considered intrinsic resistance. Both of the intrinsic resistance and the neural origin resistance define the passive stiffness and damping of the joint; which might have an impact on the range of motion and angular displacement of the knee. Wartenberg pendulum test involves lifting the limb under examination against gravity to extension and, when relaxed, releasing it, causing it to fall and swing freely. The test requires angular joint movement to be measured over time until the limb comes to rest. Because the test is performed while the limb in a relaxed state, the pendulum test aims to be a measure of passive stretch of the

muscle. Surface EMG is recorded simultaneously from the quadriceps and hamstring muscles [11, 12].

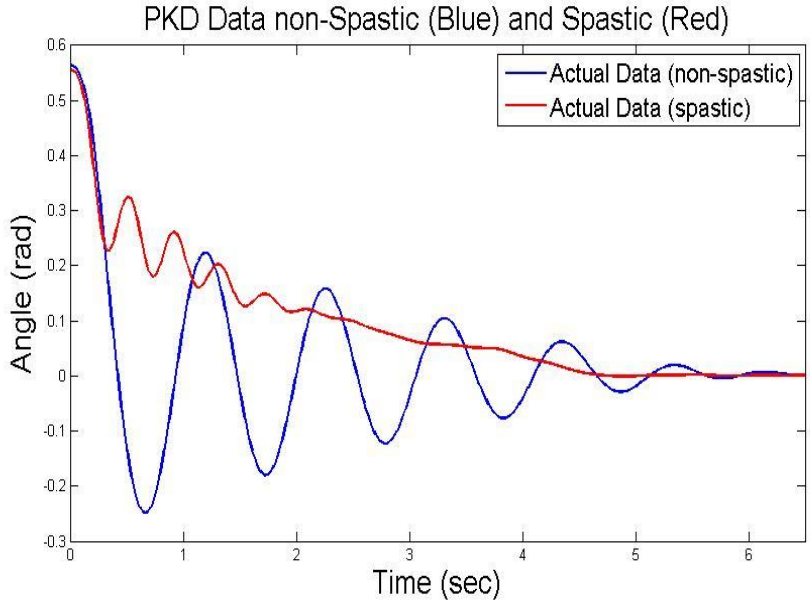


Figure 1.1 PKD for a subject with spasticity (red) and PKD for a subject without spasticity (blue). The red plot represents a classical appearance of an individual with CP. The difference in appearance between the two plots is clear. The amplitude of the oscillation in the red plat is smaller than the blue. The period in the blue plot is greater than it is in the red plot; this is an indication of a smaller stiffness.

1.2.3 Common Techniques used to Assess Dystonia

The severity of dystonia may change depending on the segment of the body which is involved in an activity. This can make the rating scales of the level of dystonia hard to develop. A number of clinical assessments have been developed to evaluate the level of dystonia such as Fahn-Marsden Scale (F-M), Unified Dystonia Rating Scale (UDRS) and a Global Dystonia Rating Scale (GDS). Fahn-Marsden (F-M) rating scale is a widely used assessment of dystonia. This test was first reported in 1985. Two features are included in the F-M rating scale: a movement scale based on the neurological examination and a disability scale based on the patient's opinion of his/her disability in

activities of daily living. The neurological examination evaluates nine body regions: mouth, eyes, speech and swallowing, neck, right arm, left arm, trunk, right leg, and left leg. Scores for each body region are found, and a formula based on a provoking factor, a severity factor and a weighting factor provides scores for the tested body region. The range of provoking factor is from 0 to 4 and is explained as the following: 0 →No dystonia, 1 → Dystonia on particular action, 2 → Dystonia on many actions, 3 → Dystonia on action of a distant body part (overflow) or intermittently at rest, and 4 → Dystonia at rest [13, 14].

The limitation of the F-M rating scale such as lack of ease in administration which relates to separately determining provoking and severity factors were the initiative to introduce other assessments. The alternative to the F-M rating scale is the GDS, and has been shown to be at least as reliable and valid but simpler and easier to apply [14].
Using the GDS scale the severity of dystonia in 14 body areas is rated. This scale has ranges from 0 to 10 and is explained as the following: 0 → no dystonia, 1 → minimal, 5 → moderate and 10 → severe dystonia). The simplicity of the GDS scale does not require modifying ratings or weighting. The sum of all the body areas is the total score, and the maximal total score of the GDS is 140 [15].

The other alternative scale is the UDRS. This technique was developed to address the limitations of the F-M scale regarding the flexibility to report specific sites as the rated areas are smaller and more defined, and shows great promise [14]. This scale includes separate ratings for proximal and distal limbs, and elimination of the subjective patient rating for speech and swallowing which are included in the F-M. 14 body areas are rated including eyes and upper face, lower face, jaw and tongue, larynx, neck, trunk,

shoulder/proximal arm (right and left), distal arm/hand (right and left), proximal leg (right and left), and distal leg/foot (right and left). For each of the 14 body areas assessed, the UDRS has a severity and a duration rating. Each body area has a specific severity rating and ranges from 0 (no dystonia) to 4 (extreme dystonia). Whether dystonia occurs at rest or with action is assessed based on the duration rating which ranges from 0 to 4, it also assess whether dystonia is predominantly at maximal or sub maximal intensity. The total score for the UDRS is the sum of the severity and duration factors. The maximal total score of the UDRS is 112 [15].

Table1.2 Fahn-Marsden Scale (F-M) Scales [15]

Fahn Marsden rating scale

Region	Provoking factor	Severity factor	Weight	Product
Eyes	0–4	×0–4	0.5	0–8
Mouth	0–4	×0–4	0.5	0–8
Speech and swallow	0–4	×0–4	1.0	0–16
Neck	0–4	×0–4	0.5	0–8
Arm (R)	0–4	×0–4	1.0	0–16
Arm (L)	0–4	×0–4	1.0	0–16
Trunk	0–4	×0–4	1.0	0–16
Leg (R)	0–4	×0–4	1.0	0–16
Leg (L)	0–4	×0–4	1.0	0–16
Sum				Max 120

Source: Comella, C. L., Leurgans, S., Wuu, J., Stebbins, G. T., & Chmura, T. (2003). Rating scales for dystonia: a multicenter assessment. Movement disorders,18(3), 303-312.

1.2.4 Traditional Interventions to Spasticity Reduction

Considerable clinical effort continues to be given to reducing the degree of spasticity. Current methods include physical and occupational therapy, mechanical casting, use of pharmaceutical agents such as baclofen, valium, and both neurosurgery and orthopedic surgery. Other techniques including Hyperbaric Oxygen, Hippotherapy and the Maribelle exercise assist system “Bouncing chair” have claimed motor improvements, with no explanation or objective measures offered.

CHAPTER 2

VESTIBULAR SYSTEM SENSORS
DESCRIPTION, PHYSIOLOGY, AND PATHWAYS

In Chapter 2 a description of the vestibular system is discussed along with the physiological components of the system and its main pathways. The vestibular system sensory input and its contribution to muscle tone are also discussed in this chapter. The impact of acoustical pleasure on changing the threshold level of acoustical activation in the otoliths is also discussed as well as the impact of microgravity while in space on changing neural signals in astronauts, and the use of the Hoffmann reflex (H-reflex) as a biomechanical measure in space is briefly explained.

2.1 The Vestibular System Components

The three well known semicircular canals sense rotational acceleration, while the lesser known vestibular sensors, the otoliths (utricle and saccule) detect linear acceleration. In the semicircular canals, the inertia of the fluid causes the fluid to stay the same while the canals are rotated. In the otoliths, there are hair cells and the otoconia -solid CaCO3 crystals- on their distal ends that are fixed in a gelatinous matrix containing. When the head accelerates, the crystals attempt to remain at rest, while the base of the hairs moves with the head, causing the vestibular nerve fibers to have an impulsive activity. Either excitation or inhibition of hair cells is the result of bending of the cilia. Otolith sensitivity to acceleration leads to its ability to detect the direction and magnitude of gravity, as well as detection of any momentary linear acceleration caused by movements [18, 19, 20, 21]. This is a form of a biological accelerometer. The utricle typically senses acceleration in horizontal movement of humans. The saccule senses acceleration in typical vertical

movement, plus gravity. The two sensors are oriented at ~ 90 degrees and both may produce an acceleration vector. These sensations contribute to setting appropriate joint impedance, for example to absorb the impact when falling, while still maintaining stable posture.

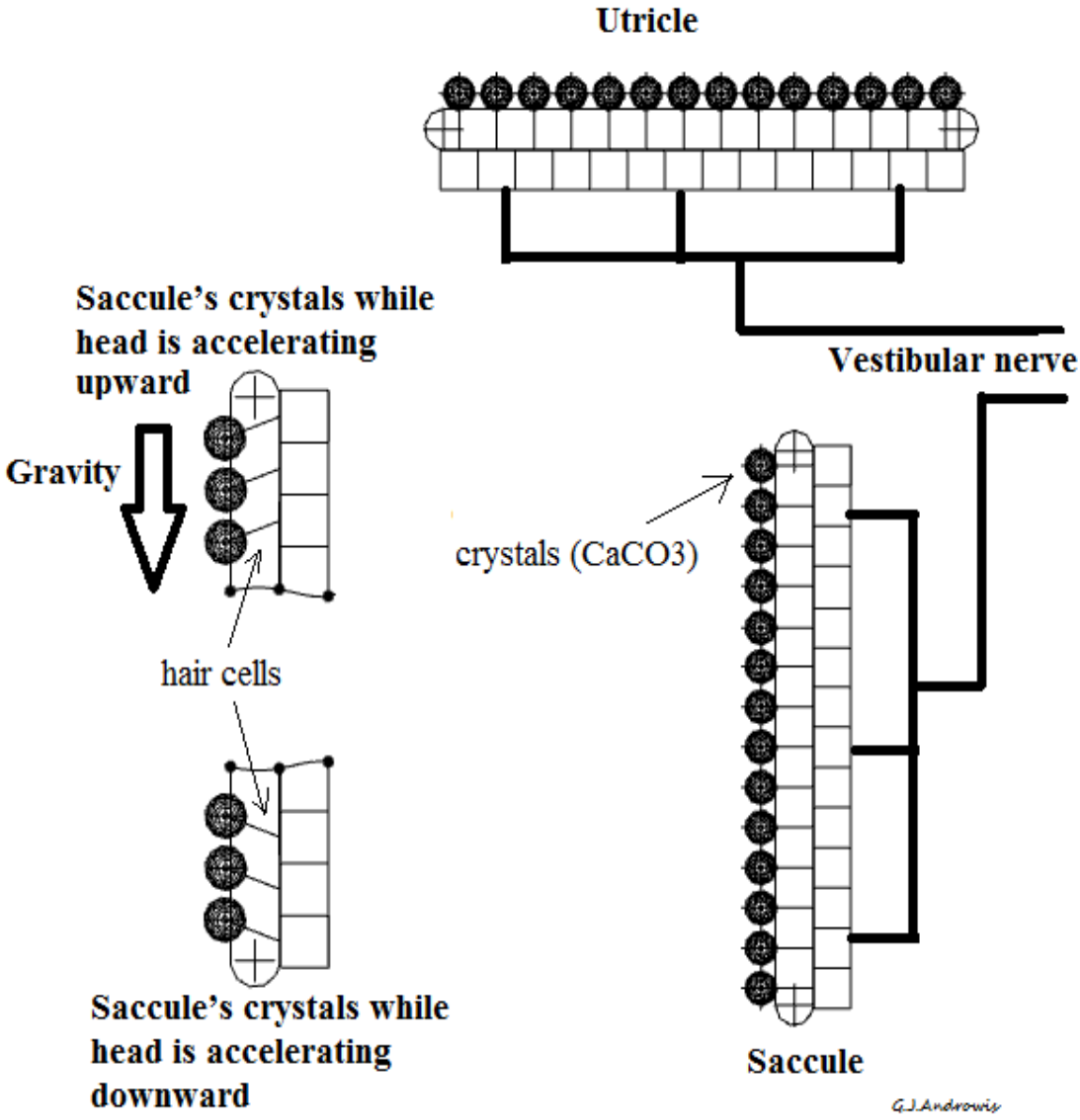


Figure 2.1 Shows the physiological description of the otoliths.
Source: Purves.D, Neuroscience Purves et al 4th Edition, ISBN-10: 0878936971 | ISBN-13: 978-0878936977.

2.2 Vestibular System Pathways and Projections

The neural pathways for the vestibular system synapse through the vestibular ganglion and nerve at the medial, lateral, or inferior vestibular nuclei. Other projections from the vestibular system go directly to the cerebellum as shown in (Figure 2.2). The vestibular nuclei project through the spinal cord to efferent neurons through the medial vestibulo spinal fiber to contribute in setting muscle tone in the upper extremity. Other projections travels though the lateral vestibulo spinal tract and contribute in setting the muscle tone of the lower extrimity by adjusting the vestibular signal received by the flexor and extensor of the antigravity muscles [17]. These projections play a role contributing in setting the muscle tone (Figure 2.2). Transmission of signals can be sent to contralateral, to the abducens nucleus (ABD) for vestibulo-ocular reflex production, to higher brain centers to provide information about spatial orientation, or to the spinal cord motor neurons that produce reflexes to stabilize posture and muscle tone [22].

A study done by Feldman et al. on cats suggested that stimulation to the lateral vestibular nucleus (Deiters') resulted in a decrease of the stretch reflex and this decrease changed the threshold of the stretch reflex. It was also found that the gradient of recruitment of motor units is dependent on the sensitivity in the reflex arcs from muscle afferents, and the primary endings contribute the main autogenetic excitation in the stretch reflex. The gamma bias may change the threshold of muscle spindles endings to a great degree [51]. Another study done by Schilder and others confirm the important relationship between the vestibular system and CNS structures in developing motor skills, integrating postural reflex, and regulating arousal level [48].

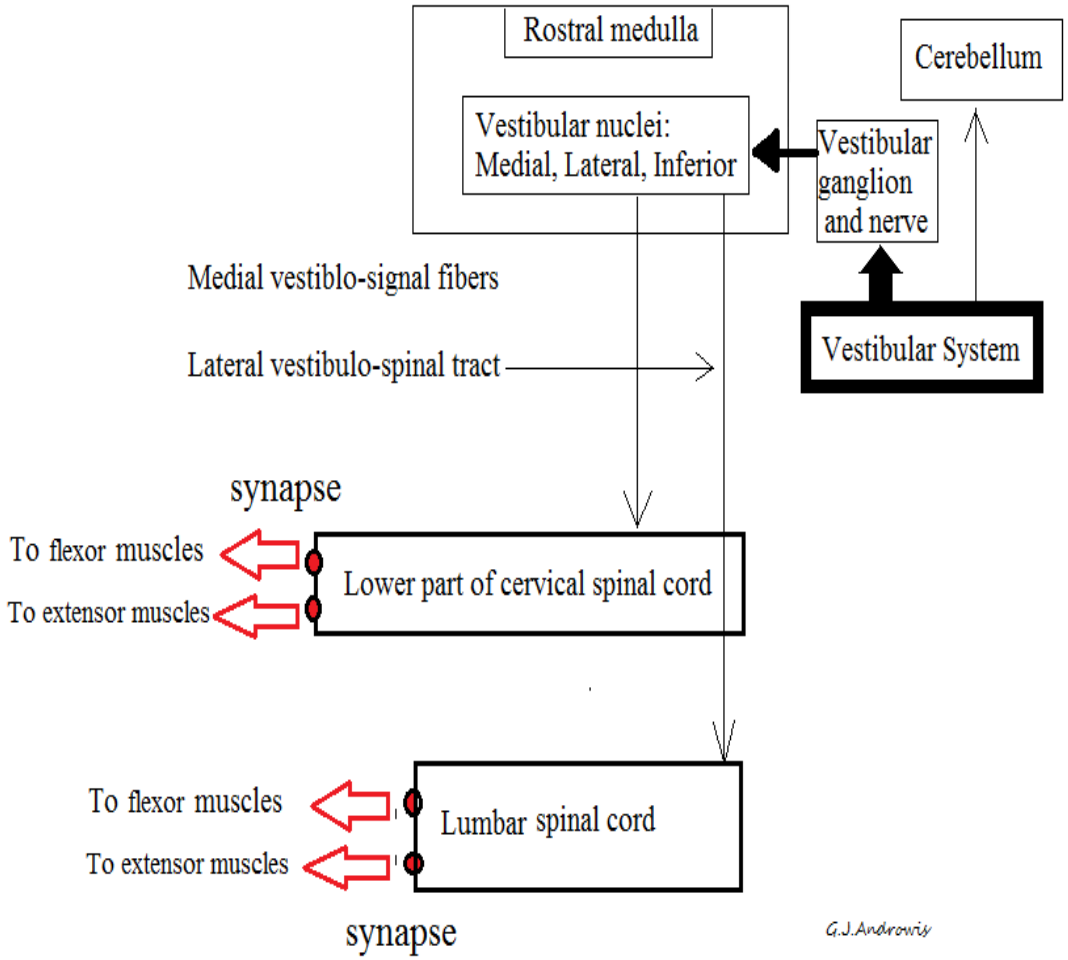


Figure 2.2 The neural pathways of the vestibular system and its contribution and projections to setting the muscle tone.
Source: Timothy C. Hain, MD, accessed on March 03 2013, OTOLITHS. http://www.dizziness-and alance.com/disorders/bppv/otoliths.html.
Wolfe. J M, Kluender K R, Levi. D M, Bartoshuk. L M, Herz . R S, Klatzky R L, Lederman. S J, and Merfeld D M, Sensation & Perception, Third Edition, 2011, Massachusetts ISBN-13: 978-0-87893-572-7.2.3 Acoustical Saccule Pleasure

There is a direct connection between the motor neurons and the nuclear complex. Projections from the otoliths reflecting sensation of linear acceleration reach the vestibulospinal reflex. The lateral vestibulospinal tract originates from the ipsilateral lateral vestibular nucleus where most of its input is coming from the otoliths and the cerebellum. This specific pathway creates antigravity postural motor activity or

protective extension, primarily in the lower extremities, in response to the head position changes that occur with respect to gravity [52].

2.3 Acoustical Saccular Pleasure

Todd et al. suggest that pleasurable sensations caused by acoustically evoked sensations of self-motion may justify a human desire for exposure to loud music [25]. They claim that activation of the vestibular system may be evoked by natural acoustic stimuli above about 90 dB SPL with frequencies between 100 and 300 Hz. The concurrence of these intensity and frequency values with those typically encountered in dance clubs and rock concerts suggests that the threshold for acoustic activation of the saccule could be a physiological basis of the rock and roll threshold [25].

2.4 Astronaut Studies after Weightless Exposure

Similar situations show habituation. For instance studies of astronauts, who have been exposed to microgravity for several days, show that their otoliths and associated neural functions become adapted to zero gravity [26]. Normally the cilia and crystals in the saccule are tilted at an angle due to earth's gravity, but in microgravity (minor gravity) the (hair/crystal) becomes horizontally straight. This stimulates the macula which generates nerve impulses in the vestibular branch of the vestibulocochlear nerve (CNV III) and information is sent to the CNS about the head acceleration. When returning to Earth, inappropriate reflexes are noticed. A reduced muscle tone is also noticed compared to prior to space flight. Readjustments to earth's gravity takes place after a period of several days on Earth. A dynamic model indicates that leg stiffness changes accounted

for the kinematic differences due to adaptation in open-loop modulation of leg impedance [27].

2.5 The Hoffmann Reflex (H-Reflex)

Spindle stretch reflex can be induced mechanically (tendon tap). In addition to this muscle spindle stretch reflex can be induced electrically in a technique called the H-reflex. The difference between the two types is that the muscle stretch occurs then followed by the spinal reflex; in contrast an electric stimulus generates the H-reflex. The same pathway explains for the H-reflex and stretch reflex. The H-reflexes can be measured in any muscle if it is possible to provide stimulation to the peripheral nerve of the tested muscle [53].

The H-reflex is considered as a valuable tool in assessing modulation of monosynaptic reflex activity in the spinal cord as it bypasses the input of the muscle spindle. Therefore, the H-reflex provides an estimate of alpha motoneurons (αMN) excitability when presynaptic inhibition and intrinsic excitability of the αMNs remain constant. This measurement can be used to evaluate the response of the nervous system to musculoskeletal injuries, various neurologic conditions, pain, application of therapeutic modalities, exercise training, and performance of motor tasks. This is very useful in human motor control studies [54].

At the site of the electrical stimulus, signal induces the efferent fibers which results in the M-wave signal, and also induces the afferent (sensory) fibers that projects to the homonymous motoneurons until they reach and synapse on αMNs, then the αMNs generates an action potentials that travels by the efferent until they reach the neuromuscular junction and produces a twitch response in the EMG (the H-reflex). When

the action potentials in the αMNs reach a neuromuscular junction, a synchronized twitch is produced in the muscle [53, 54].

2.6 Mathematical Optimization Model

In order to describe the motion dynamics of knee joints affected and unaffected by spasticity utilizing the PKD test, a mathematical optimization model (Simon, 2010) based on the equation of motion $\{I\theta'' + B(\theta' - \theta'_{vt}) + K(\theta - \theta_{vt}) = mgLsin(\theta)\}$ will be used to help understanding the neural contributions to the clinically observed features of spasticity. As a result of using this optimization model on the PKD test, three variables stiffness K, damping B and virtual trajectory θ_{vt} can be used to describe spasticity. The model is based on the Equilibrium Point Hypothesis (EPH) which stands on the principle that modifications in descending signals to the spinal segmental apparatus may be described as setting threshold values of muscle length for the tonic stretch reflex. When the length of a muscle is below this threshold, the muscle is silent. On the other hand, when the muscle's length is over the threshold, the muscle is activated and the level of activation increases with the difference between the threshold value and the actual muscle length. Muscle shortening or contraction is the outcome of muscle activation, which brings its length closer to the threshold [28].

2.7 Dominant Peak 2 Hz

It has been noted in a number of studies that during walking the vertical acceleration of the head exhibits a dominant peak at 2 Hz. The movement of the trunk upward to a peak is reached while passing over the single supporting foot, and the trunk is at its lowest point during the double-stance [29]. Therefore the vertical movement of the whole body

exhibits a dominant component at the frequency of stepping. Over-ground human walking studies [30] show that the average preferred cadence is close to a step frequency of 2 Hz. The vertical head acceleration is critical in maintaining posture through spinal reflexes in part mediated by the otoliths. Additionally a predilection for a 2-Hz frequency of movement has also been witnessed in music [31]. This idea of 2 Hz can be helpful in determining the frequency of the vestibular stimulation.

CHAPTER 3

MECHANICAL VESTIBULAR STIMULATION AND EQUILIBRUM POINT HYPOTHESIS (EPH)

3.1 Mechanical Vestibular Stimulation

Previous work done by (Fee and Samworth, Fee and Foulds) shows that a dramatic reduction of spasticity-induced disturbance of the passive swing of the lower shank about the knee is caused as a result of using a one-degree of freedom mechanism that accelerates persons with cerebral palsy up and down in the gravitational direction[16, 17]. Evaluation of spasticity pre and post vertical accelerations using the PKD test seen in the following plot:

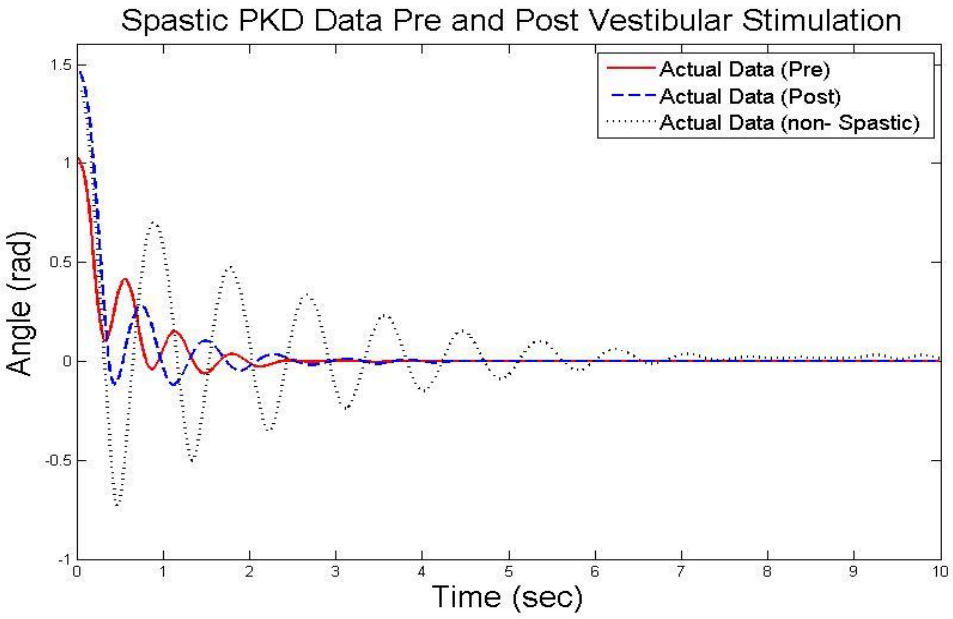


Figure 3.1 PKD for spastic subject pre (red plot) and post (blue plot) vestibular stimulation. The post plot indicates an increased range of motion and a trend toward a trajectory that appears closer to a non-spastic subject.
Source: Fee, J.W. & Foulds, R.A. Neuromuscular Modeling of Spasticity in Cerebral Palsy. IEEE Transactions on Neural Systems and Rehabilitation, 12(1), 55-64. (2004).

3.2 The Equilibrium Point Hypothesis

The main principle that the Equilibrium Point Hypothesis (EPH) is based on is the idea of control depending on threshold for activation of neurons. The EPH offers a structure to analyze both voluntary and involuntary movements. The EPH was first described by Feldman to control a simple single joint system, and then was improved to describe advanced movements as locomotion, and multi-joint movement system [32]. Intentional motor actions are important in the EPH theory. To achieve these actions, electrochemical effects descending from the brain are sent to the motoneurons and transferred to threshold alterations that cause muscle lengths or joint angles changes during the initiation of the involved motoneurons. Therefore, depending on the actual position of body and its threshold (referent or virtual trajectory) location, the CNS is able to identify the location and timing so muscles are activated without the need of detailed descending information [33].

It was concluded in a study done by Asatryan and Feldman [34] that the angle of the threshold (R) or the virtual angle is constant when the set point is not changed and muscles are not activated for a movement. When subjects wish to purposely change their pose, a new R value is generated by the CNS. It was also found in the same study that in order to completely activate a limb (such as the shank) to full extension, the R value may be beyond the upper limit of the biomechanical range of motion for the involved joints (knee). On the other hand to completely relax the muscles, the value of R has to be set outside the lower limit of the biomechanical range of motion of the involved joints. This confirms that the CNS identifies the kinematics, inertial properties of the joint, and

controls the value of R to be within or outside the range of motion of the involved joint [34].

3.3 The Optimization Model used to Analyze PKD Data

The optimization model used in analyzing the collected data in this research is a forward dynamic model that generates kinematic data by using optimized model parameters. Previous work done in our Neuromuscular Engineering lab by [Simon and Foulds (2004) and Swift et al. (2007)] has introduced the idea of a virtual trajectory based pendulum model of knee motion. In the model, the points where the net moment about the knee equals the moment of inertia multiplied by the angular acceleration is 0 ($\sum M = I\theta'' = 0$) represents the equilibrium points of the knee trajectory [35]. Instead of specifying a curve through the points of inflection defining the system's equilibrium trajectory, a forward model is created with an optimized sigmoidal curve to represent the centrally driven virtual trajectory. The presence of both of θ_{vt} and the gravitational loading causes the limb to follow a system equilibrium trajectory [35].

Using this model the limb's movement can be characterized by three parameters; knee stiffness (K), damping (B) and virtual trajectory (θ_{vt}). The optimization model is based on the shank equation of motion [12]:

$$I\theta'' + B\theta' + K\theta = mgLsin(\theta) \quad (3.1)$$

The equation is then modified to include the virtual trajectory term as the following:

$$I\theta'' + B(\theta' - \theta'_{vt}) + K(\theta - \theta_{vt}) = mgLsin(\theta) \quad (3.2)$$

Where I is moment of inertia, L is the distance to center of mass and m is the mass of the lower leg [35].

The model uses Simulink (Mathworks, Inc., Natick, MA) and follows Newton's second law of motion. The moments in equation (3.2) consist of the gravitational moment $mgLsin(\theta)$, stiffness moment $K(\theta- \theta_{vt})$, and damping moment $B(\theta'-\theta'_{vt})$). Using the model with MATLAB optimization toolbox, three parameters are optimized: stiffness (K), damping (B), and virtual trajectory (θ_{vt}). The value that represents the change in θ_{vt} is the time of the inflection point in the sigmoid curve. Shorter sigmoid inflection time indicates a steeper θ_{vt}.

A proportional derivative controller is used in the optimization model [36], and the first parameter to be optimized is selected and assigned the first controller variable. Parameters are changed, and then the level of either increase or decrease in the cost function is evaluated, and finally decides whether to keep or reject the new value [16]. The sum-squared error (SSE) represents the cost function of comparing a sample-by sample of the experimental data and the model output [37]. When the SSE reaches a minimum value determined by user-defined criteria the following variable is evaluated and assigned the controller variable. The model cycles through all the variables sets to reach a minimum SSE value that goes no lower. The minimum SSE value represents a measure of the goodness of fit between the experimental data and the output of the [36].

3.4 Validation of the Optimization Model

To validate the optimization model used to analyze the PKD test data, a Simulink model was created to generate data similar to the PKD test data using known K, B, and θ_{vt} parameters. The Simulink model is explained in the following figure:

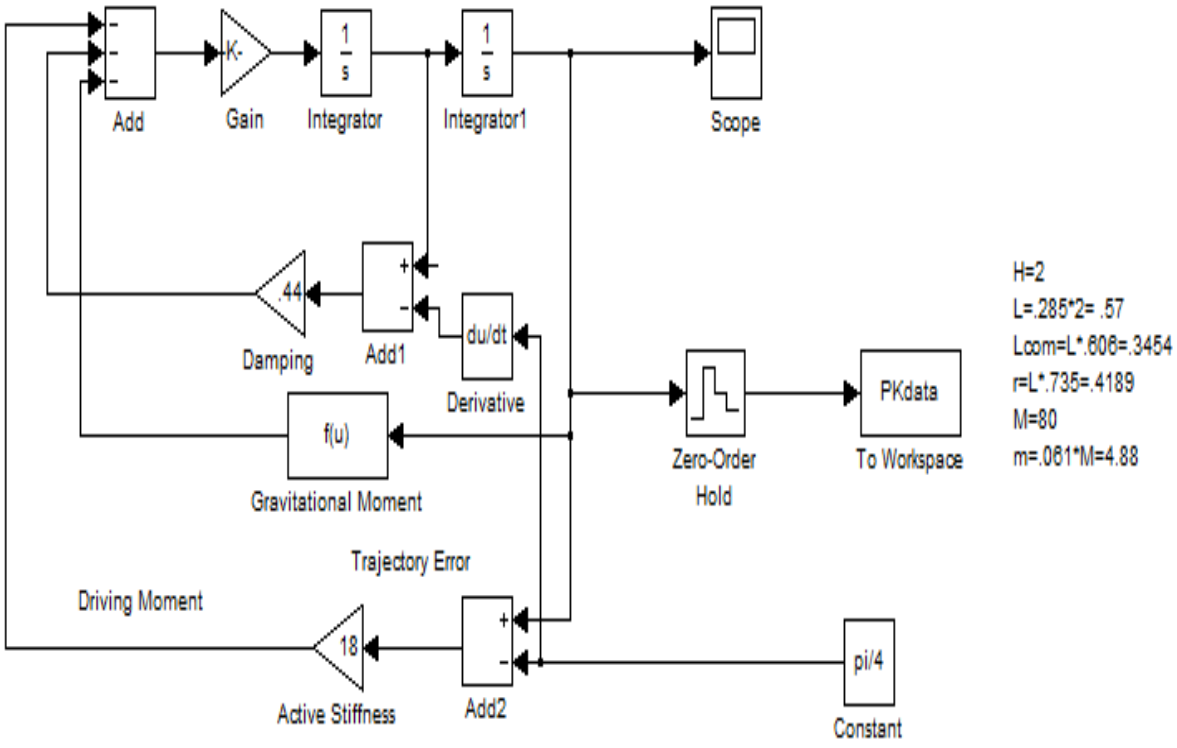


Figure 3.2 Simulink model to generate trajectory similar to the PKD data.

The stiffness, damping and virtual trajectory parameters used in this model to generate the data explained in Figure 3.3 are 18 N.m/rad, 0.44 N.m.sec/rad, and pi/4 rad respectively. In this model a constant value for θ_{vt} was used to validate the robustness of the optimization model to optimize for K, and B in subjects with dystonia (constant θ_{vt}). The trajectory generated from the model is illustrated in the following figure:

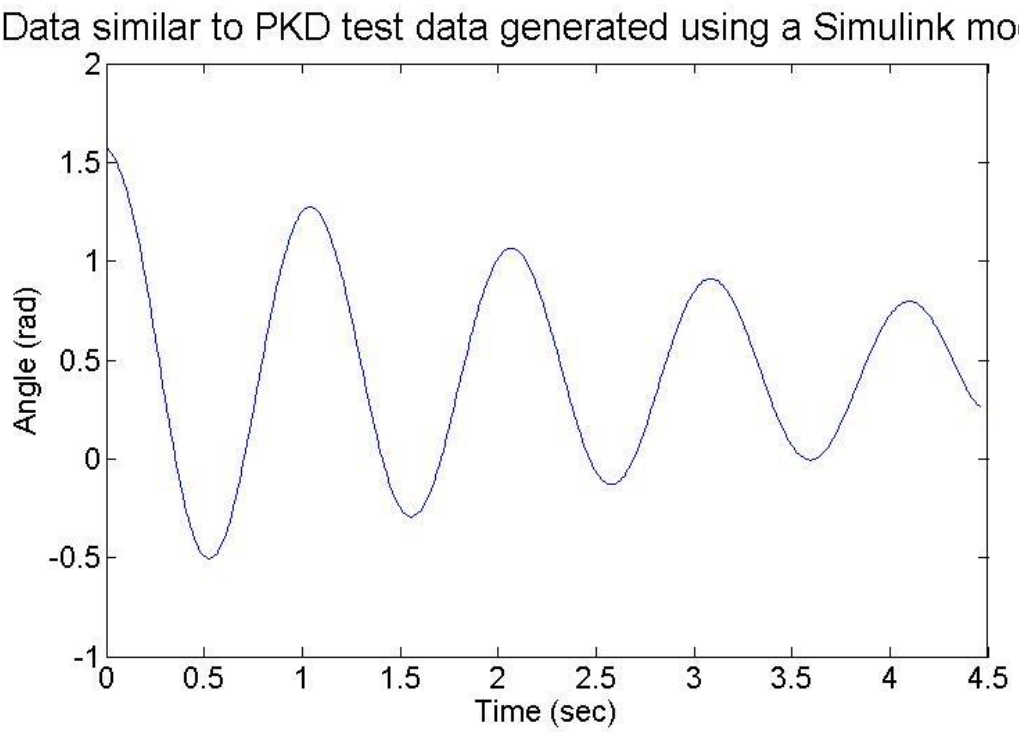


Figure 3.3 PKD data generated using a Simulink model.

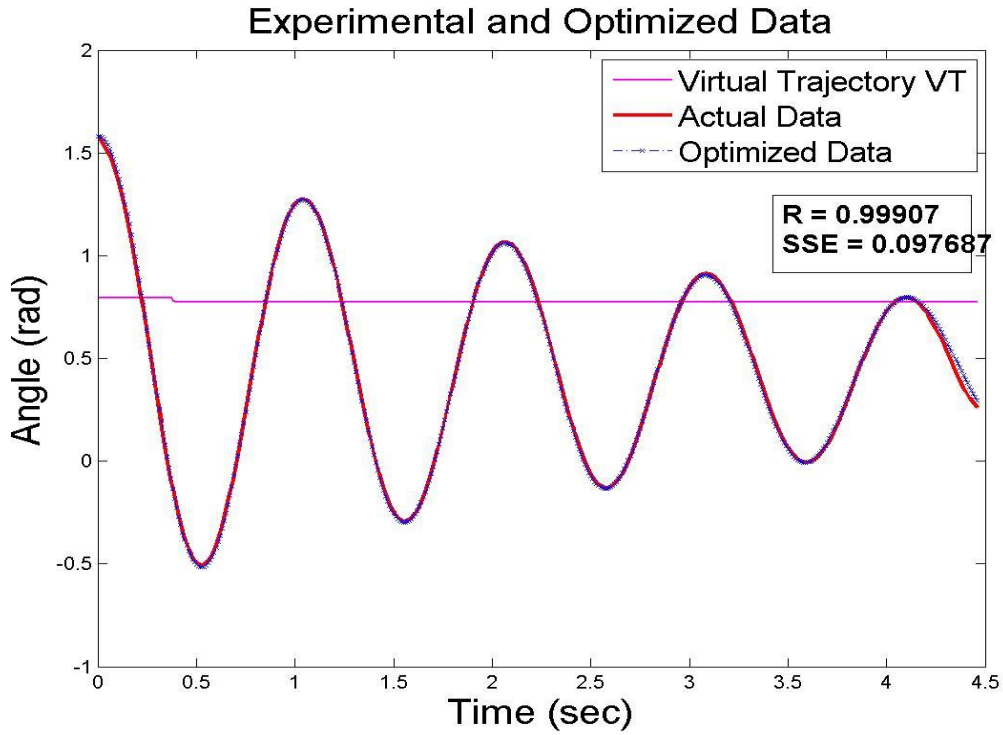


Figure 3.4 The optimized PKD data generated using Simulink model.

The results of the optimization model to analyze for K, B, and θ_{vt} are represented in Figure 3.4. The following table shows the optimized parameters.

Table 3.1 The Optimized Parameters of the Simulink PKD Data

Damping (B) (N-m-sec/rad)	Stiffness (K) (N-m/rad)	(θ_{vt}) Time to inflection (sec)
0.4512	18.2725	0.3795

The optimization model used to analyze the PKD test data in this work uses a sigmoidal curve to represent the centrally driven virtual trajectory (explained previously) [35], this is the cause of the small glitch seen on Figure 3.4 at 0.3795 (sec) which represents the time of the inflection point of θ_{vt}.

CHAPTER 4

SIX FLAGS PILOT STUDY

4.1 Introduction

A variety of studies on the effect of exposure to microgravity and its effect on otolith induced alteration of human performance have been conducted. Exposure to microgravity can cause intensive changes in posture, balance and locomotion. For instance astronauts, after returning from spaceflight, experience changes in their walking [38, 39]. As explained previously the saccule crystals remain oriented horizontally in space then are tilted again when returned to earth. This changes the signals sent by otoliths to CNS and the tone of the antigravity muscles is be altered.

The roller coaster ride works as a mechanical stimulator for the vestibular system, as during the ride subjects experience time when gravity equals ~ zero, which might have a stimulating impact on the vestibular system leading to changing the muscle tone signal. The reflex mechanisms are essential for many pre-programmed motor responses such as those required to stabilize posture after a voluntary jump and therefore astronauts experience disruption in their ability to maintain postural equilibrium when performing such tasks [39].

4.2 Designing the Study

A wonderful opportunity was available to conduct a convenience study, testing the relationship between vestibular stimulation and roller coaster rides at Six Flags. In this study we did not control the rides or the number of subjects, since participants were involved in another study. The hypothesis was to test whether the otoliths are stimulated

by microgravity environments (free fall moments) created by the roller coaster rides. This study was done twice on site, each employing a different method of measurement.

Experiment A: The first measurement method was the Wartenberg Pendulum Knee Drop (PKD) test. (Nine subjects with no history of disability were tested of whom two were not included in the analysis due to their inability to relax while performing the PKD test).

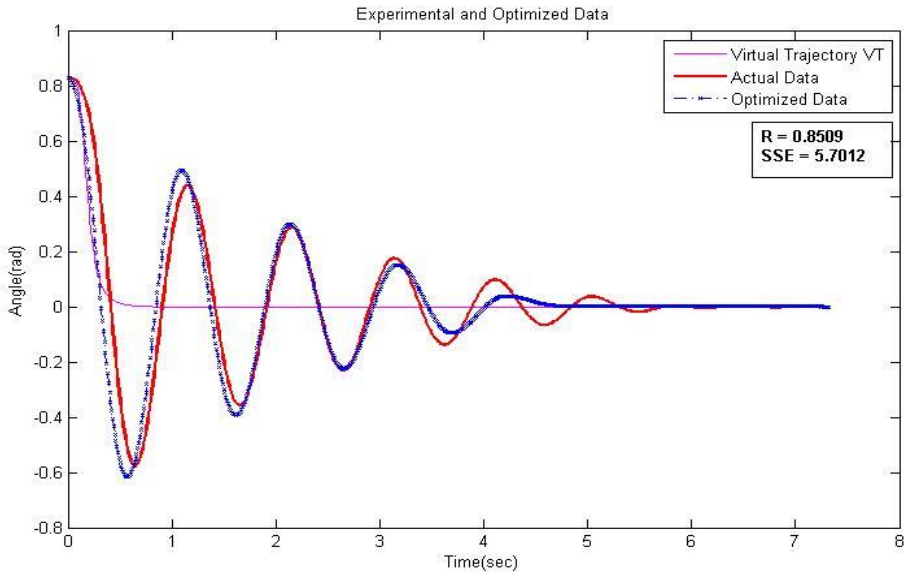
Experiment B: The second measurement method was done using a modified NASA Jump Test. (Eleven subjects with no history of disability were tested).

Experiment A, this experiment has a within subject design, and the independent variable is the muscle tone in the body, dependent variables are the joint stiffness (K), damping (B) and virtual trajectory (θ_{vt}) which are computed in the analysis using our lab's optimization model for analyzing the PKD test data. The initial pre stimulation measurement of each subject represents the control measurement, and the post-ride measurement represents the treatment measurement.

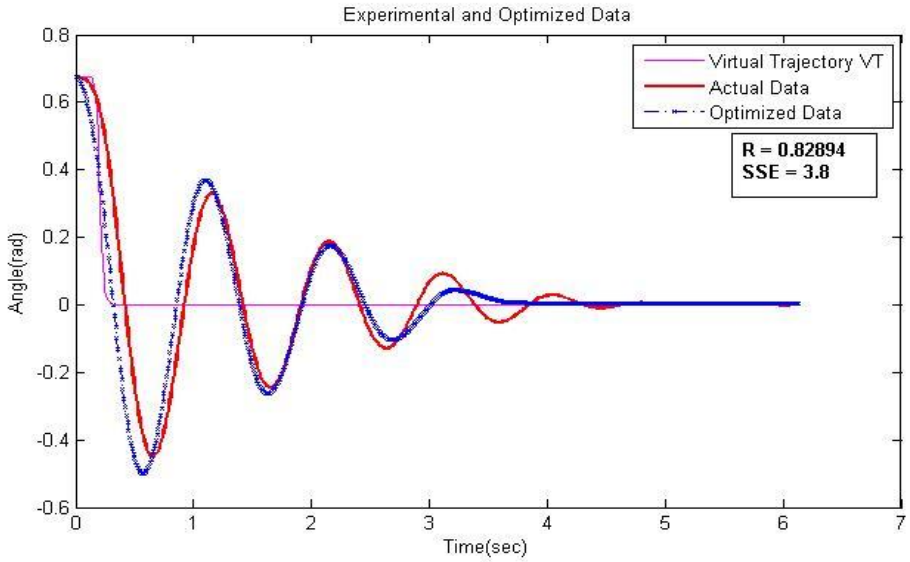
Using this mathematical model gives the ability to optimize values of K, B, and θ_{vt} pre and post rides, and provides the ability to quantify changes in the knee trajectory in terms of these three parameters. θ_{vt} is modeled as a sigmoid function as explained in the previous chapter. The value of time to its inflection point was chosen to represent the shape of the sigmoid. Longer inflection time indicates a shallower sigmoid and therefore a shallower θ_{vt}. Shorter sigmoid inflection time indicates a steeper θ_{vt}.

4.3 Results

The output results of the optimization model for the first subject are represented in the following examples:



Example 4.1 PKD test data of the first subject pre roller coaster ride.



Example 4.2 Example of the PKD test data of the first subject post roller coaster ride.

4.4 Statistical Analysis

To examine whether there is any impact from the roller coaster riders on the muscle tone, a paired t-test design is established to test significant difference between pre and post rides using the PKD test and the results of the statistical analysis are as the following:

Table 4.1 Statistical Analysis Results of the Paired t-test for PKD Test Data

Paired Samples Test			
Optimized Parameter	t	df	Sig. (2-tailed)
Pair 1: Post ride (B) Pre ride (B)	0.977	6	0.366
Pair 2: Post ride (K) Pre ride (K)	0.917	6	0.394
Pair 3: Post ride (θ_{vt}) Pre ride (θ_{vt})	0.902	6	0.377

Paired samples t-test revealed that there is no significant difference on knee damping pre-ride Vs. post-ride (paired-samples t-test, t (6) =0.977, p>0.05).
Paired samples t-test revealed that there is no significant difference on knee stiffness pre-ride Vs. post-ride (paired-samples t-test, t (6) =0.917, p>0.05).
Paired samples t-test revealed that there is no significant difference on virtual trajectory pre-ride Vs. post-ride (paired-samples t-test, t (6) =0.902, p>0.05).

Based on the results above we can conclude that either the rollercoaster ride did not change the muscle tone, or as expected that the ride effect on the muscle tone in the participants with no history of disability might be very small and an assessment such as the PKD test may be unable to capture the change in persons without impairment.

4.5 Six Flags- NASA jump test

Experiment B, in addition to the PKD test, a jump test was used in a subsequent visit to Six Flags. This test is modeled on NASA jump test on astronauts following space flights. Each subject completed a jump off of a 30 cm high block pre and post roller coaster rides. This experiment has a within subject design. The dependent variables are the change in

hip and knee angles upon landing. The initial measurement of each subject pre ride represents the control measurement, and the difference between pre and post ride measure, represent the treatment effect. The hip and knee joint angles of the subjects following a jump off the block before and after the roller coaster ride are calculated and compared.

Hypotheses: The hypothesis is that riding on roller coaster ride expose the subject to cyclic gravitational changes that alter the otoliths signals to CNS, resulting in variations to the muscle tone that causes differences in the following:

I. Change in hip angle between pre and post rides.

II. Change in knee angle between pre and post rides.

To calculate the change in hip and knee angles, three sensors of an Ascension Technologies trakSTAR electromagnetic tracker were fixed to the subject's torso, thigh, and shank. All sensors were put in a housing that was designed in Pro-Engineer and printed using a 3D printer (ABS Plastic), and Velcro was used to strap the sensor-housings and hold them in place as seen in figure 4.3.

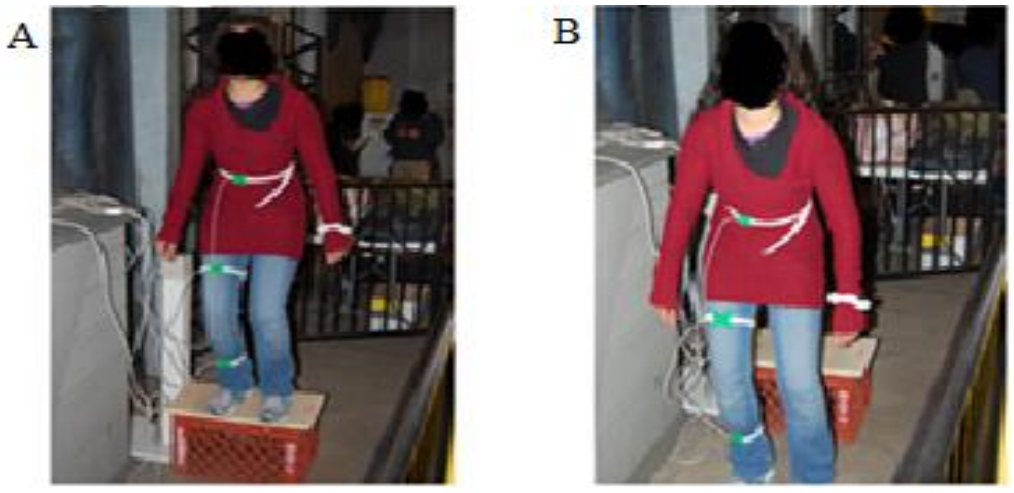


Figure 4.3 (A) One participant while jumping off of the 30 cm block, (B) Subject upon landing. The location of each of the three trackSTAR sensors is included.

Each sensor in the trakSTAR gives 6 degrees of freedom, 3 transitional (X,Y,Z) and 3 rotational (Roll, Pitch, Yaw) of which we are using only the roll angle. The data collected were used to calculate the change in hip and knee joint angles for pre and post rides as seen in the following Figure 4.4:

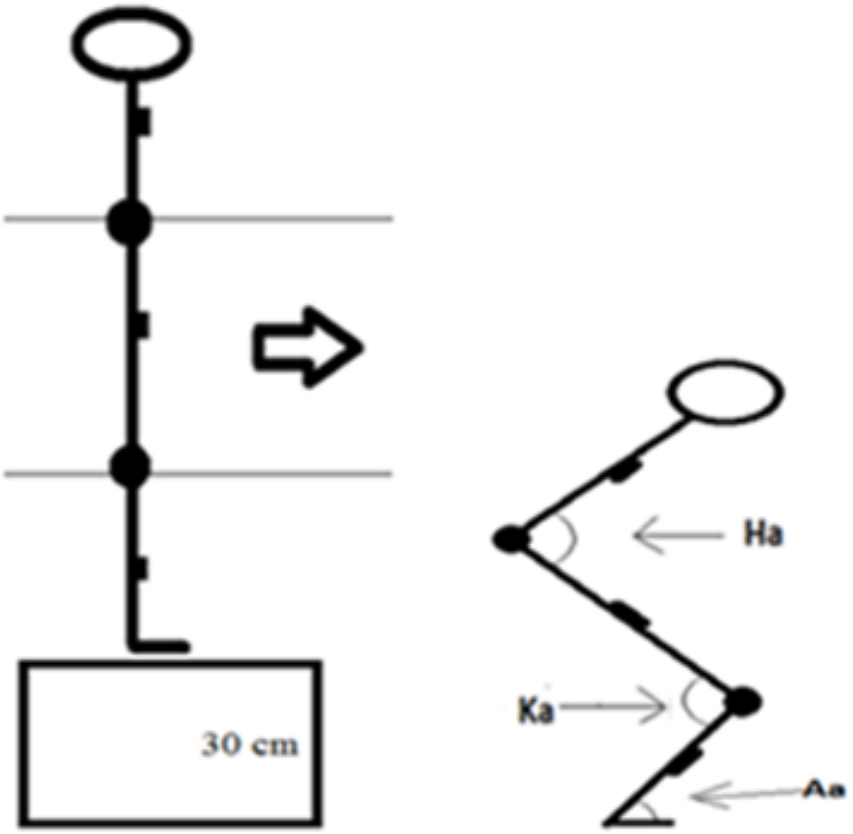


Figure 4.4 The change in knee and hip joint angle as subjects perform the modified NASA jump test. The hip angle (Ha) is calculated by finding the angle between torsos between torso and thigh, the knee angle (Ka) is determined by calculating the angle between the thigh and the shank segments.

4.6 Statistical Analysis on NASA Jump Test

In this experiment a within subject design is used as each subject has repeated measurements. A paired t-test design is established to test significant difference between

pre and post rides for hip and knee joint angles, and the results of the statistical analysis are as the following:

Table 4.2 Statistical Analysis of the Paired t-test for the NASA Jump Test

Paired Samples Test			
Optimized Parameter	t	df	Sig. (2-tailed)
Pair 1: hip_ post ride hip- pre ride	3.981	10	0.003
Pair 2: knee_ post ride knee- pre ride	2.284	10	0.045

The roller coaster ride resulted in a mean reduction of 8.45 (SD 7.04) in the hip angle between pre and post rides, a statistically significant effect (paired-samples t-test, $t(10)=3.981$, $p<0.05$)."
The roller coaster ride resulted in a mean reduction of 3.58 (SD 5.20) in the knee angle between pre and post rides, a statistically significant effect (paired-samples t-test, $t(10)=2.284$, $p<0.05$).

4.7 Conclusion

Cyclic gravity stimulation works as an effector to change the muscle tone in the body, and it was found that stimulating the vestibular system using momentary free fall approximating zero gravity causes a significant difference in muscle tone. It was determined that there is a significant difference between the change in hip angle and the change in knee angle pre and post rides. Therefore the null hypothesis Ho was rejected. There was a mean reduction of 8.45° in the hip angle of post rides, and a mean reduction of 3.58° in the knee angle of post rides. The reduction in the hip and knee angles is an indication of a greater joint compliance in leg segments that can be explained due to reduced muscle tone and stiffness.

The significant difference between the change in hip pre and post rides caused by stimulation to otoliths can be explained by the fact that motions about the waist would permit faster acceleration of the center of mass (COM) and thus faster correction of postural displacements so a change in the hip angle is the easiest way to maintain balance and postural equilibrium while jumping [40, 41, 42]. These findings are consistent with those reported by Bloomberg et al.. The increase in hip strategy is consistent with the evidence that the vestibular system plays a large role in control of the trunk [43, 44]. A better understanding of changes in coordination of joints following vestibular stimulation may aid the efforts to develop training methods for improvement of postural control in CP and vestibular deficient patients.

One possible limitations of this experiment are that subjects had to walk for 10-15 seconds after the ride was over to reach the experiment apparatus. The test was then done. This is due to the necessity of following the park's safety procedures and regulation. This affected the study in a way that it might have made it harder to find a significant difference between treatments. Also it is important to note that subjects may have partially readapted to their altered muscle tone.

CHAPTER 5

RESEARCH DESIGN AND METHODS- VESTIBULAR STIMULATION

5.1 Apparatus

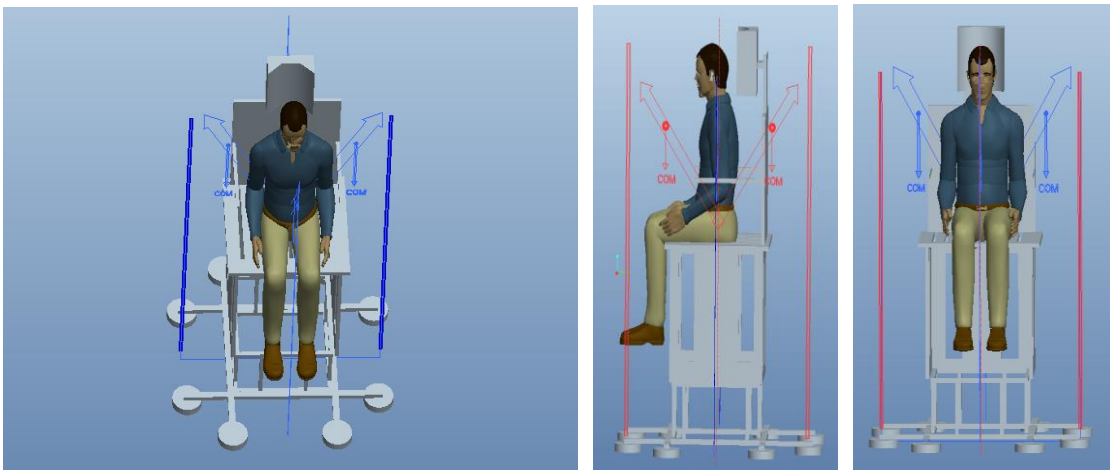
5.1.1 Vestibular Stimulation Chair

The seat of the stimulation chair is a modified Prospect Chair that sits on top of a custom designed actuator that moves the seat up and down. The prospect chair is commonly used in rehabilitation centers and is specifically designed for individuals with disability. This chair is equipped with padded restraints for safety.

The actuator consists of four pneumatic pistons connected to a hospital grade air compressor. The compressor is quiet and minimizes startle reflexes. The amplitude and frequency of movement of the prospect chair is controlled by a laptop PC using a custom MATLAB program to control amplitude and frequency. The maximum vertical travel of the chair (7.5 cm) is mechanically limited in the event of computer failure and the maximum frequency (2.5 Hz) of movement is inherent to pistons and air supply. The pneumatics provided electrical isolation. An emergency shut off switch immediately stops the chair's movement. The chair was regularly examined for mechanical integrity (loose fasteners, pneumatic actuators retention straps…etc.) as well as electrical connections. Preventative maintenance was performed prior to all experiments.

To prevent the chair from tilting, an extension of 12 inches was added to each side of the base, so that if a subject moves to the extremes positions allowed by the prospect chair at the maximum vertical extension, the combining center of mass of the subject and chair will not exceed the boundaries of the chair's base, as seen in Figure 5.1:

A



B

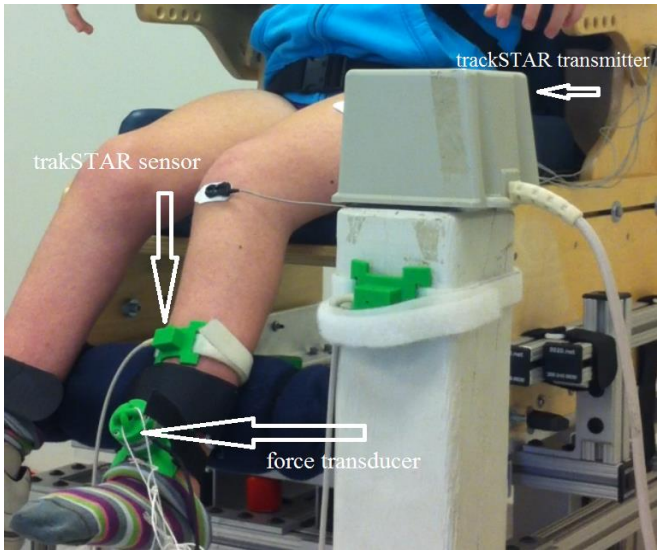


Figure 5.1 (A) The chair design and COM locations while subject is sitting on the chair, (B) The trakSTAR and force trasducer used in PKD test.

5.1.2 Wartenberg Pendulum Knee Drop

After vestibular stimulation, subjects remain seated in the Prospect chair. To sense the shank movement, the apparatus includes an ATI (ATI Industrial Automation) multi-axis force/torque sensor and a trakSTAR (Ascension Technologies) electromagnetic position/rotation sensor. The ATI sensor measures the force required to hold the limb in opposition to gravity. This force measurement can be used to calculate the torque that

results from the initial holding of the limb at or close to full extension of the lower limb prior to its release during the pendulum knee drop test.

The trakSTAR sensor senses the knee joint angle at 100 samples per second, as the limb is released and swings during the test. The ATI (ATI Industrial Automation) multi-axis force/torque sensor senses the force at 100 samples per second until the limb is released and swings freely during the test. Surface EMG from the agonist and antagonist muscles (quadriceps-Vastus Lateralis- and hamstring- Biceps Femoris) was measured by a bioamplifier (Grass-Telefactor) at 1000 samples per second. EMG data were filtered using a band pass filter (fourth-order Butterworth; cut-off frequencies of 35 and 150 Hz) then rectified. The trackSTAR and force transducer data were filtered using a low pass filter with a cut-off frequency of 10 Hz. All the collected data were simultaneously recorded using customized MATLAB code.

5.2 Procedure and Methodology

The following procedure was followed and performed on each subject. Prior to the start of the study, subjects were weighed and measured for total height and leg length. Subjects were seated in the chair with their thighs supported in a horizontal position, and their torso maintained a 105 degree angle (slightly reclining) of position.

The trakSTAR sensor was attached to the shank of the left leg. The ATI force sensor was used to measure the force applied by the experimenter while the shank was lifted, held in position, and then released. Both the ATI and the trakSTAR sensors are mounted on a plastic housing designed in Pro-Engineer and printed in ABS using 3D printer. The shape of the housing casts the shape of the shank. In addition, during the

PKD test, surface EMG from the agonist and antagonist muscles was measured by a bioamplifier.

5.2.1 Assessment Procedure

To perform the assessment, the experimenter administered the PKD test by lifting the left shank against gravity to an extension angle and, when the subject was relaxed, released, causing it to fall and swing freely. The release time is represented by the force transducer frames when holding torque equals zero. The test included angular joint movement measured over time until the limb comes to rest. Because the test was performed while the limb was in a relaxed state, the pendulum measures passive stretch of the muscle. Surface EMG was recorded simultaneously from the quadriceps and hamstring muscles. The absence of EMG activity indicated that the subject is relaxed. EMG also aids in the detection of reflex activity.

5.2.2 Experiment Duration

During vestibular stimulation subjects remained seated in the chair and were stimulated for a 15 minutes trial. Sessions were conducted at the research lab (Lab 670-Fenster hall) and the vestibular laboratory at St. Joseph's Regional Medical Center. Subjects were examined with the PKD tests pre and post vestibular stimulation. The necessity to perform many tests was to evaluate how long the stimulation effect lasts.

The duration of the complete session of data collection lasted for ~2 hours, including attachment of the surface EMG electrodes and the evaluation measurements trials per subject.

5.2.3 Analysis of the PKD Test Data

In order to understand the possible neural contribution to changes in muscle tone, and to describe the motion dynamics of knee joints affected and unaffected by spasticity utilizing the PKD test, data were collected on these same subjects and a mathematical optimization model [45] based on the equation of motion $\{I\theta'' + B(\theta' - \theta_{vt}) + K(\theta - \theta_{vt}) = mgLsin(\theta)\}$ was used to help understand the neural contributions to the clinically observed features of spasticity [46, 47].

Using the mathematical model it is possible to optimize values of K, B, and θ_{vt} pre and post, and to quantify the observed changes in terms of these three parameters used in a paired t-test design to confirm any changes in K, B, and/or θ_{vt}. θ_{vt} is modeled as a sigmoid function. The value of time to the sigmoid inflection point is used as the single variable representing of the shape of the sigmoid. Longer inflection time indicates a shallower sigmoid and therefore a shallower θ_{vt}. Shorter sigmoid inflection time indicates a steeper θ_{vt}.

5.3 Subjects

Specific aim I: This study involved five subjects with no known neurological impairment. Subjects with no disability were recruited via flyers and other announcements. Subject's age ranged between 18-30.

Specific aim II: This included one subject with spasticity, 35 year old male with CP and spasticity.

Specific aims III, IV: These aims were conducted on 7 children with spasticity, ages 4-18, and Ashworth Scale (1-3).

Exclusion criteria for specific aim (III, and IV): subjects younger than 4 or older than 18, Ashworth Scale (4), contractures, subjects who are planning on having surgeries during the duration of the study, and subjects with a known history of motion sickness or seizure. The potential subjects were identified and approved by Dr. Allan Strongwater, M.D. Pediatric Orthopedic Surgeon, St. Joseph Regional Medical Center, Paterson NJ. The subject's medical history was noted with respect to past interventions and therapy.

5.4 Specific Aim I

5.4.1 Overview

Purpose 1 (PKD test): to examine whether there is any significant difference in K, B, and θ_{vt} pre and post stimulation in subjects without disability. Purpose 2 (NASA Jump test): to examine whether there is any significant difference in hip and knee angle pre and post stimulation in subjects without disability that can be interpreted as an indication of a change in the muscle tone.

Vestibular stimulation was conducted on five subjects without disability and changes in the PKD test results were quantified, along with surface EMG pre and post stimulation. The NASA jump test was also administered pre and post stimulation. This component of the study allowed the refinement of experimental procedures and platform design. The vestibular stimulation consisted of 7.5 cm of vertical oscillation for about 15 minutes at 1.5 Hz.

5.4.2 Results

In this experiment, the aim was to test whether the hypothesis that vestibular stimulation has an impact on muscle tones in subjects without disability. The data of the PKD test for all five subjects is examined in a paired t-test design. The plots in Figures 5.2 and 5.3 show the PKD test data of pre and post stimulation.

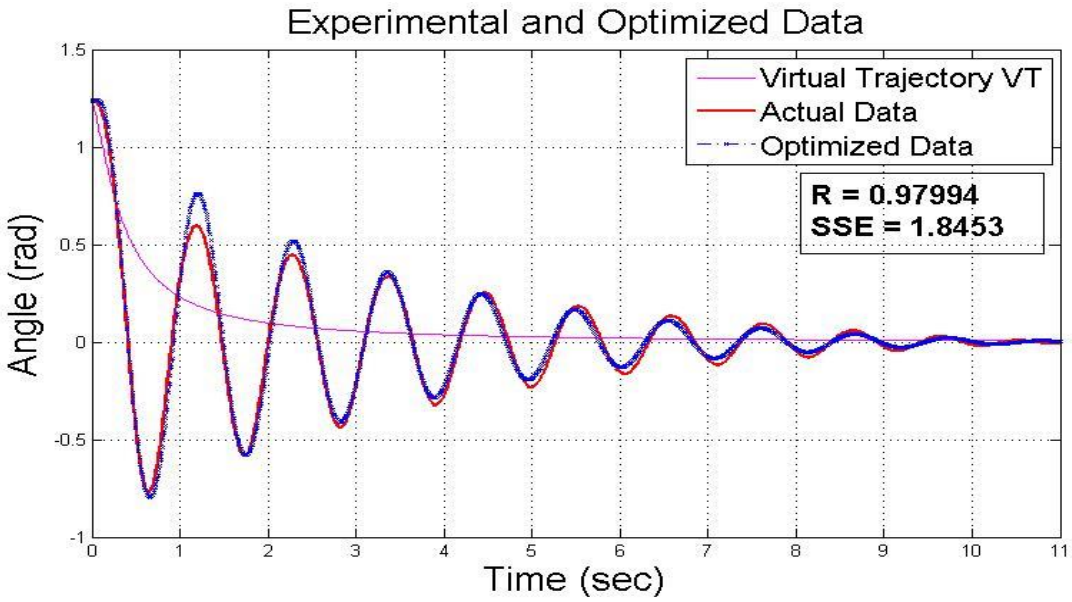


Figure 5.2 The PKD test data pre stimulation of a subject without disability.

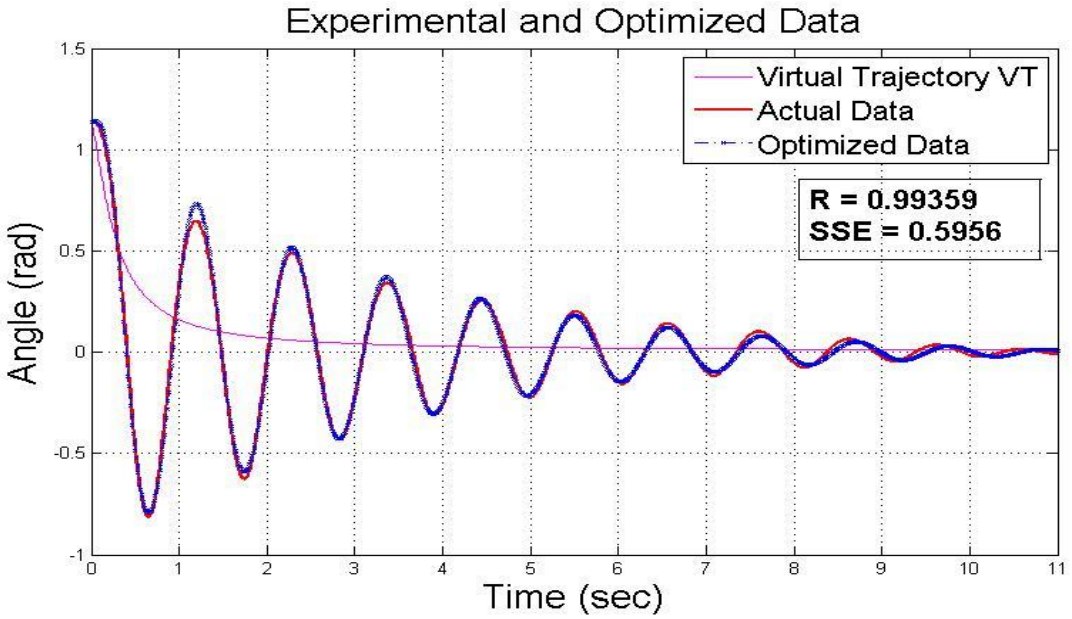


Figure 5.3 The PKD test data post stimulation of a subject without disability.

Table 5.1 Optimized Parameters of the PKD Test Date for Five Participants with no History of Disability

Subject #	Damping (B) (N-m-sec/rad)		Stiffness (K) (N-m/rad)		(θ_{vt}) Time to inflection (Sec)	
#	Pre	Post	Pre	Post	Pre	Post
1	0.18	.17	2.26	2.31	.35	.27
2	0.36	.41	3.77	2.66	.52	.40
3	0.91	.82	5.11	4.31	.63	.57
4	0.16	.18	3.03	2.77	.51	.30
5	0.30	.27	4.48	3.68	.46	.35

The experiment had a within subject design therefore a paired t-test was designed and revealed to the following results.

Table 5.2 Statistical Analysis Results of the Paired t-test for PKD Test Data

Paired Samples Test			
Optimized Parameter	t	df	Sig. (2-tailed)
Pair 1: Post Stimulation (B) Pre Stimulation (B)	0.534	4	0.622
Pair 2: Post Stimulation (K) Pre Stimulation (K)	2.779	4	0.049
Pair 3: Post Stimulation (θ_{vt}) Pre Stimulation (θ_{vt})	4.367	4	0.012

Paired samples t-test revealed that there is no significant difference on knee damping pre-ride Vs. post-ride (paired-samples t-test, t (4) =0.534, p>0.05).
Paired samples t-test revealed that there is significant difference on knee stiffness pre-ride Vs. post-ride (paired-samples t-test, t (4) =2.779, p<0.05).
Paired samples t-test revealed that there is significant difference on virtual trajectory pre-ride Vs. post-ride (paired-samples t-test, t (4) =4.3673, p<0.05).

Participants of this experiment were also evaluated using the NASA jump test, and

Table 5.3 The angular values of knee and hip joints pre and post stimulation.

Table 5.3 The Results of the Calculated Knee and Hip Joint Angel of NASA Jump Test for Five Participants with no History of Disability

Subject #	Hip (rad)		Knee (rad)	
#	Pre	Post	Pre	Post
1	0.548	0.523	1.020	0.834
2	0.645	0.538	1.350	1.119
3	0.441	0.382	0.995	0.821
4	0.722	0.549	1.210	1.169
5	0.788	0.536	1.216	1.204

Table 5.4 Statistical Analysis Results of the Paired t-test for NASA Jump Test Data

Paired Samples Test			
Calculated Angles	t	df	Sig. (2-tailed)
Pair 1: Post Stimulation (Hip) Pre Stimulation (Hip)	3.032	4	0.039
Pair 2: Post Stimulation (Knee) Pre Stimulation (Knee)	2.977	4	0.041

Vestibular stimulation resulted in a mean reduction of 0.123 rad (SD 0.087 rad) in the change of hip angle between pre and post stimulation, a statistically significant effect (paired-samples t-test, $t(4)=3.032$, $p<0.05$).
Vestibular stimulation resulted in a mean reduction of 0.128 rad (SD 0.096 rad) in the change of knee angle between pre and post stimulation, a statistically significant effect (paired-samples t-test, $t(4)=2.97$, $p<0.05$).

5.4.3 Discussion

In this experiment, vestibular stimulation of subjects with no history of disability showed promising impact on changing muscle tone. The results of the PKD test shows that there was no significant differences between the value of B, but it shows a significant difference between the values of K and the time to inflection point in the optimized model. The reduction in last two terms (K, and θ_{vt}) can be linked to a change in the muscle tone in these subjects. This suggests that vestibular stimulation that consisted of 1.5 Hz, 7.5 cm in amplitude for 15 minutes had an effect on changing the stiffness and virtual trajectory. It is important to note that a similar population when tested using PKD test in six flags on roller coaster rides did not show significant differences. This can be explained since roller coaster rides are not as concentrated as the stimulation provided using the chair. The vestibular stimulation chair targets the saccule organ, on the other hand roller coaster rides stimulates others vestibular organs and the stimulation intensity may not be as strong as the stimulation chair. The major question then is: will a similar type of stimulation have an effect on subjects with disability? This will be tested and explained in the following sections.

5.5 Specific Aim II

5.5.1 Overview

The purpose of this aim is to determine whether mechanical vestibular stimulation to the otoliths would result in a change on the muscle tone on subjects with disability by examining the significant differences in K, B, and θ_{vt} and to test whether the hypothesis of stimulating the otoliths organs in the vestibular system has an effect on the muscle

tone/spasticity in individuals with neuromuscular disabilities. In addition to this is the importance of developing a deeper understanding of the process of vertical vestibular stimulation using a mechanical vestibular chair and to establish a clinical procedure that reduces spasticity in individuals with neuromuscular disabilities.

One spastic subject was tested and the muscle tone was evaluated using the PKD test, along with surface EMG. PDK test was administered before and after stimulation. The results of this experiment were also useful to allow final modifications to the research design and experimental procedure that was followed in this experiment, with children with CP (will be discussed in the following sections). The vestibular stimulation consisted of 7.5 cm in amplitude of vertical oscillation for about 15 minutes at 1.5 Hz. pre and post stimulation on individuals with disability.

5.5.2 Methodology and Experimental Procedure

The data analyzed in this aim consist of the time course of the knee's angular displacement of a 35 year old CP male subject. Subject was seated as explained in previous chapters, with the femur stabilized in the horizontal orientation and the lower leg hanging freely as seen in Figure 5.4.

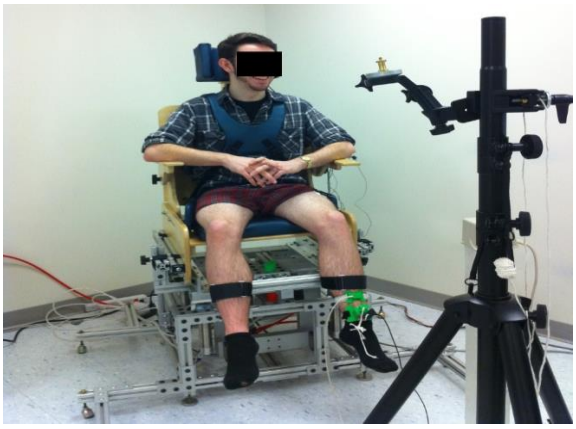


Figure 5.4 The vestibular stimulation apparatus and the PKD test chair.

5.5.3 Results

The data of the PKD test are explained in the following figures. Figure 5.5 represents the data of pre stimulation and Figure 5.6 represents the PKD test post stimulation.

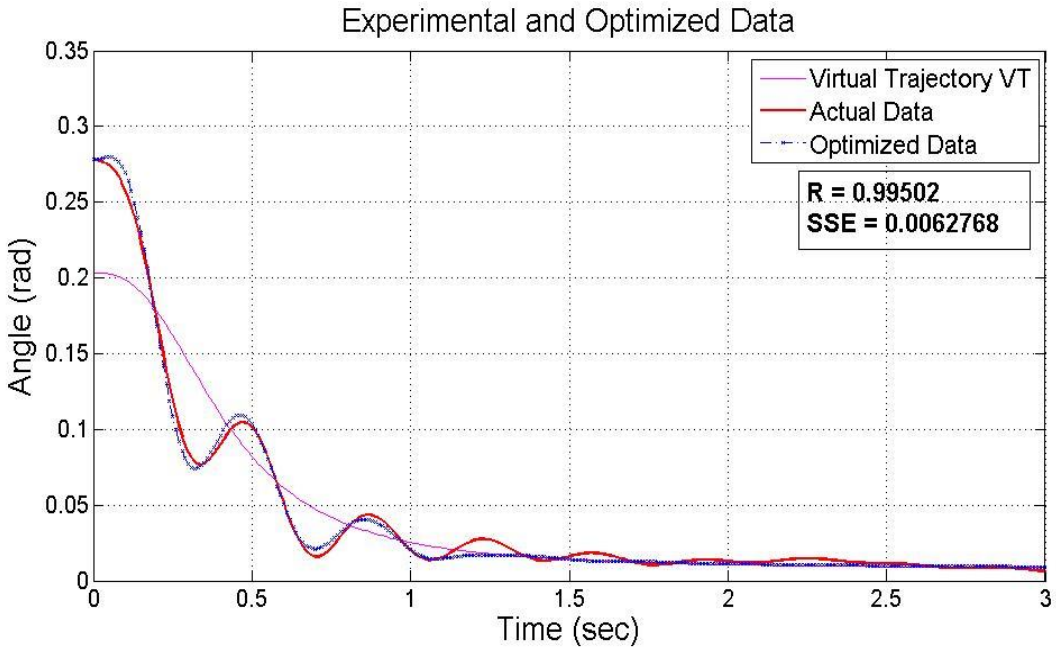


Figure 5.5 PKD test of one subject with CP- spasticity pre vestibular stimulation.

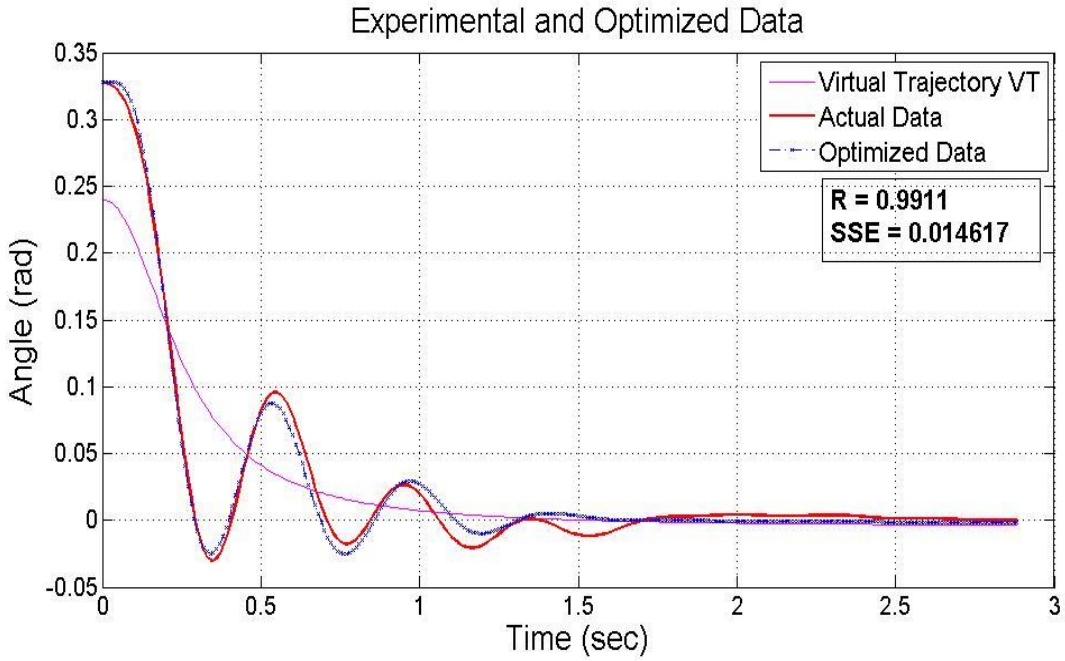


Figure 5.6 PKD test data of one subject with CP- Spasticity post vestibular stimulation.

Comparing pre vs. post vestibular stimulation; Changes in the shank trajectory are visibly obvious. The changes observed in PKD trajectory are similar to those found by Fee et al. (1995), Fee's results explained in page 16. The post stimulation PKD trajectory for this subject has features similar to a trajectory belonging to a person without spasticity (page 37). Using the optimization model, values of K, B, and θ_{vt} pre and post were produced quantifying the observed changes in terms of these three parameters. Again, θ_{vt} is modeled as a sigmoid function, with the value of the time to its inflection point chosen to represent the shape of the sigmoid. Longer inflection time indicates a shallower sigmoid and therefore a shallower θ_{vt}. Shorter sigmoid inflection time indicates a steeper θ_{vt}. It is also noticed that a higher range of motion was gained post stimulation. The following table shows pre and post stimulation parameters:

Table 5.5 Optimized Parameters of the PKD for an Individual with CP- Spasticity

	Damping (B) (N-m-rad/sec)	Stiffness (K) (N-m/rad)	(θ_{vt}) Time to inflection (Sec)
Pre	1.4530	100.2948	0.4155
Post	1.2896	75.8453	0.2586

An estimate of K for a non-disabled person with the same anthropometrics would be between 5-10 N-m/rad. This can be contrasted with the pre value of K which is ~ 10 times higher than expected of a person with spasticity at his age and muscular characteristics. The post stimulation K shows a decrease in the stiffness which confirms that the intervention reduces stiffness. A less dramatic change in damping is also seen.

The most significant change is in the shape of θ_{vt} which is dramatically steeper, approaching the θ_{vt} measured in persons without spasticity.

5.5.4 Discussion

The work in this aim demonstrated a mechanical technique that results in a noticeable difference on the PKD test when administered on a spastic subject. The change in the shank's trajectory can be explained due to changes in the muscle tone at the level of the antigravity muscles caused by stimulating the saccule, which led into a reduction in the sensitivity and increase in the threshold at the level of the alpha motoneurons.

A number of questions need to be answered to have a better understanding about the mechanical vestibular stimulation process. For instance what is the effect's duration after stimulation? What is the ideal Frequency? What is the ideal stimulation duration? Answers for those questions will be covered in the following chapters.

5.6 Specific Aim III and IV

5.6.1 Overview

Mechanical vestibular stimulation is not very well understood. Insufficient research has been done to fully understand its process. It was very important to first be able to know the proper characteristics of mechanical vestibular stimulation needed to impact the otoliths signals. Therefore, vestibular stimulation was provided to 1 child with cerebral palsy and motor changes were evaluated with the PKD test, along with surface EMG. PDK test was conducted before and after stimulation. The vestibular stimulation consisted of 7.5 cm of vertical oscillation at frequencies of 2 Hz and (5, 10, 15) minutes

as well as 1 Hz and (5, 10, 15). Stimulation amplitude was 7.5 cm. Purposes: 1) to determine the most effective frequency for stimulation. 2) To determine the most effective time duration for stimulation. 3) To determine the stimulation effect duration.

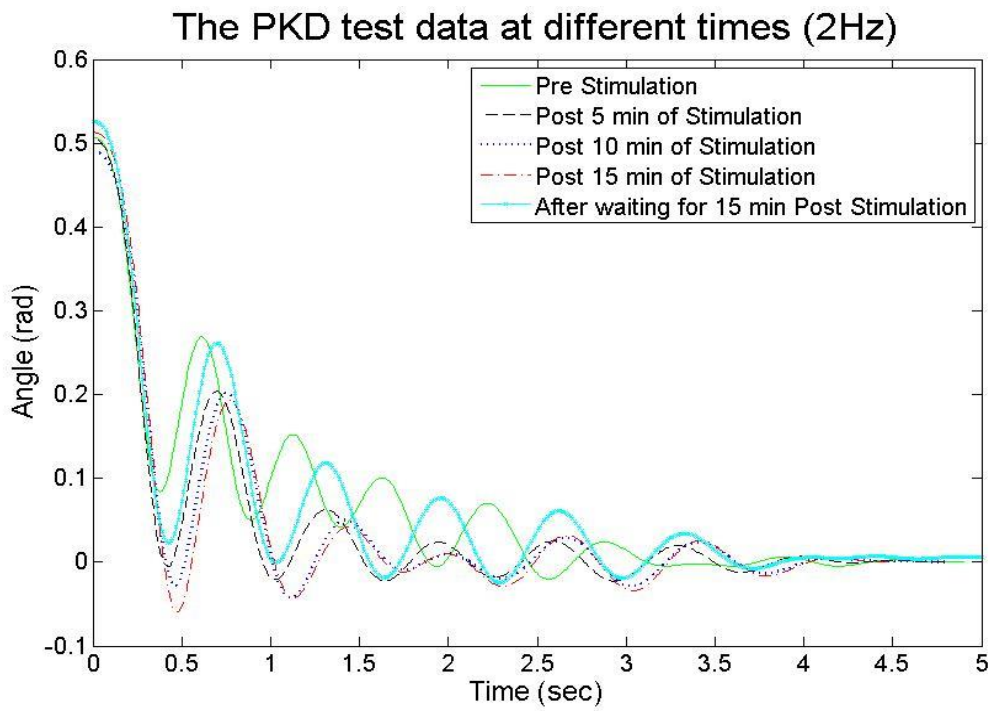
5.6.2 Methodology and Experimental Procedure

In this study, one subject was first examined using the PKD test, and the measure was the pre stimulation evaluation. Subject then was provided with 5 minutes of stimulation at 2 Hz, then tested using the PKD, followed by 5 minutes of 2 Hz stimulation, tested using the PKD, then provided with 5 minutes of 2 Hz of stimulation, then administered the PKD test. One hour later the procedure was repeated but with a stimulation frequency of 1 Hz.

5.6.3 Results

Figure 5.7 shows the optimized parameters following the PKD test at different stimulation duration and different stimulation frequency.

A



B

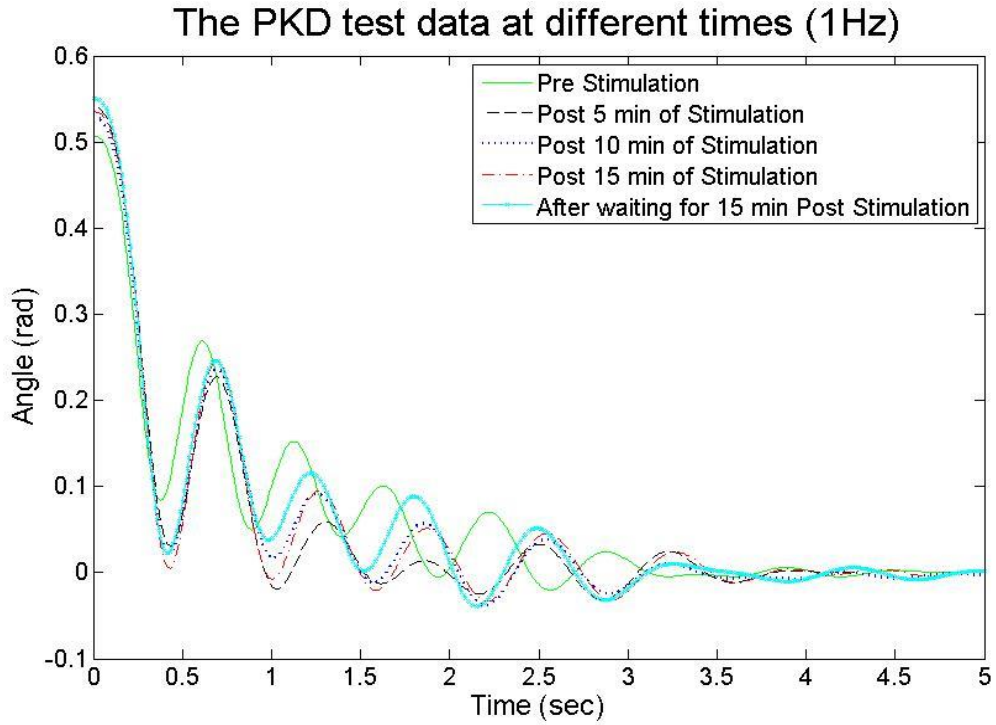


Figure 5.7 Superimposed plot of the PKD test output during different stimulation duration with 2 Hz of stimulation frequency (A) and 1 Hz of stimulation frequency (B).

Table 5.6 Optimized Parameters of the PKD for an Individual with CP- Spasticity at Different Vestibular Stimulation Durations with Frequency of 2 Hz

2 Hz Stimulation				
Stimulation duration	(θ_{vt}) Time to inflection (Sec)	Damping (B) (N-m-rad/sec)	Stiffness (K) (N-m/rad)	Impact ratio
Pre (0 minutes)	0.37043	0.40875	26.72088	0
Post 1(5 minutes)	0.4156	0.27095	16.5626	0.59533
Post 2(10 minutes)	0.40415	0.24683	14.52965	0.76135
Post 3(15 minutes)	0.35835	0.22945	13.05813	0.98257
15 minutes wait after Post	0.44035	0.29333	16.33368	0.48235

Table 5.7 Optimized Parameters of the PKD for an Individual with CP- Spasticity at Different Vestibular Stimulation Durations with Frequency of 1 Hz

1 Hz Stimulation				
Stimulation duration	(θ_{vt}) Time to inflection (Sec)	Damping (B) (N-m-rad/sec)	Stiffness (K) (N-m/rad)	Impact ratio
Post 1(5 minutes)	0.54453	0.21623	19.45731	0.27284
Post 2(10 minutes)	0.44655	0.26775	17.25354	0.49375
Post 3(15 minutes)	0.40035	0.29833	19.01393	0.47779
15 minutes wait after Post	0.49673	0.33292	19.6018	0.11098

The data in this aim suggest that mechanical vestibular stimulation provided with 2 Hz has a greater impact on changing the level of spasticity in an individual with CP. Accumulation of stimulation resulted in a greater positive impact on reducing the muscle tone in both stimulation frequencies (1 and 2) Hz. A stimulation impact ratio value was calculated for each of the different stimulation durations (Equation 5.1). The stimulation impact ratio value is a representation of the change with respect to the

optimized value of pre vestibular stimulation. A higher stimulation impact ratio value is an indication of a greater impact due to stimulation.

$$\text{Impact ratio} = [(K_{pre} - K_{post})/ K_{pre} + (B_{pre} - B_{post})/ B_{pre} + (\theta_{vt\_pre} - \theta_{vt\_post})/ \theta_{vt\_pre}] \quad (5.1)$$

The values of the impact ratio calculated at each stage represent the level of the stimulation impact on the subject. Figure 5.8 shows an illustration of the impact ration over time. It is noticed that the effect of the 2 Hz stimulation has almost diminished after the one hour wait prior to providing the 1 Hz vestibular stimulation as the value of the impact ratio for 5 minutes of 1 Hz stimulation (0.27284) is less than the value representing 15 minutes wait after the final 2 Hz stimulation (0.48235).

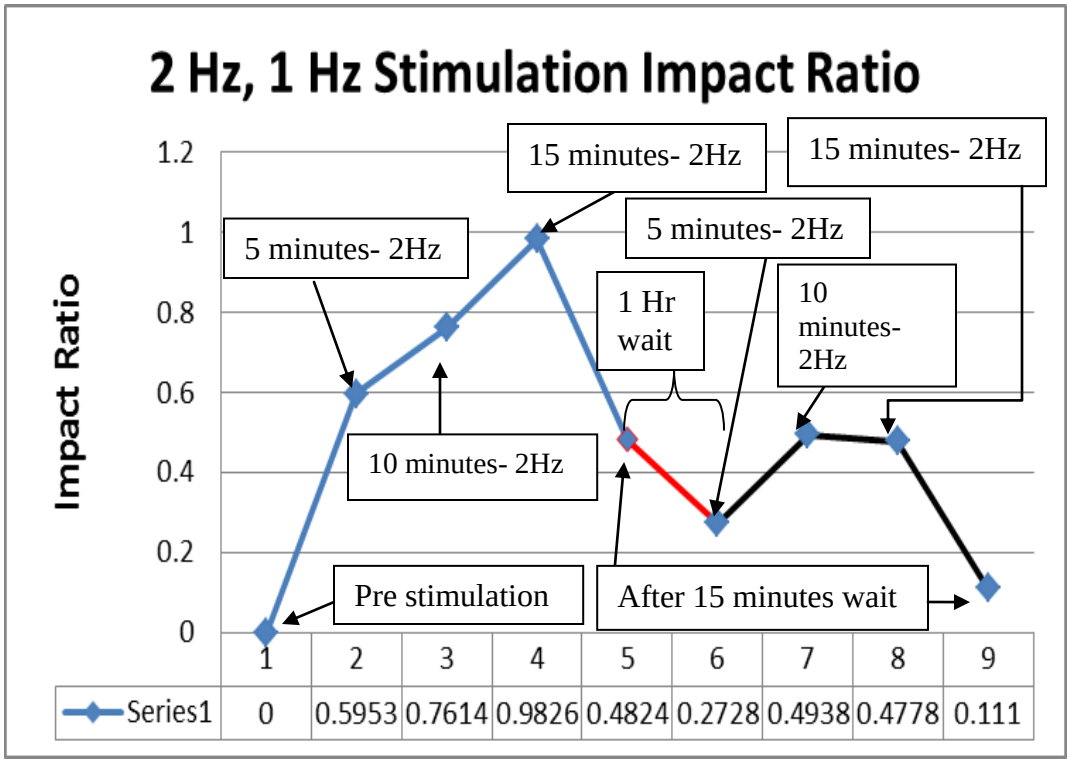


Figure 5.8 The impact ratio of each stage during the experiment.

The appearance of the plots in figures 5.7 can be better understood when considering the optimized parameters shown in Tables 5.5 and 5.6. The value of the impact ratio is largest with 2 Hz and 15 minutes of stimulation and smallest with 1 Hz

and 5 minutes of stimulation. Investigation of the stimulation effect duration was pursued. The PKD test was administered 15 minutes after the final post stimulation. The acceleration provided in the 2 Hz, and 7.5 cm of stimulation can be calculated as explained in the following equation:

$$\text{Acceleration} = \text{Amplitude} * (2* \text{pi} * \text{Frequency})^2 \quad (5.2)$$

⇨ **Acceleration= 11.83152 m/sec^2**

The output of the PKD test shows that 15 minutes after stimulation, the effect starts to fade away and trajectory is on its way back to appearance similar in characteristics to those described for pre stimulation. This suggests that stimulation is retained for at least 15 minutes after stimulation. Another important point is that when comparing the output 15 minutes after final post stimulation for 1 Hz and 2Hz, it is noticed that the stimulation effect retention in the PKD output is greater for 2 Hz than it is for 1 Hz. This might be as result of the greater impact of 2 Hz stimulation. The results in this experiment suggest that a meaningful vestibular stimulation at 2 Hz for 15 minutes can show important effect.

5.7 Specific Aim V

5.7.1 Overview

Better understanding of mechanical vestibular stimulation and its effect on changing the muscle tone in individuals with CP was the motivation of this aim. Seven children with CP (5 females and 2 males) were involved in this study. The PKD test was used to evaluate the degree of subject's spasticity. The vestibular stimulation consisted of vertical oscillation with 7.5 cm in amplitude, frequency of 2 Hz and 15 minute [49].

EMG was simultaneously recorded from the quadriceps (vastus lateralis) and hamstring (biceps femoris) muscles along with the PKD test. The activation of EMG during PKD can be understood in relationship to the flexion and extension of the lower leg. It is interesting that EMG activity for quadriceps is seen at every flexion cycle in the post stimulation data, while on the other hand EMG activity is nearly continuous in the initial cycles of PKD in the pre stimulation. This may be an indication of a change in the activation pattern of EMG from the agonist and antagonist muscles as a result of the vestibular stimulation that caused neural changes in the vestibular descending signal.

Some subject diagnoses included dystonia along with spasticity; this clinical known fact was not revealed prior to the study. This fact added a very interesting flavor to the study in a way that we were able to distinguish between both groups, and it was a must to separate them in order to properly analyze their data. Subjects with CP and dystonia exhibited a different response to the PKD test. Therefore, the purpose of this study is to examine whether there are any significant changes in the muscle tone as a result of providing the mechanical vestibular stimulation to both groups and to be able to explain the results for each group, and to differentiate between them in terms of the different kinematics measured in the study. Another important point in this study is to explain the impact of vestibular stimulation on both groups and show the distinction between them. The data in this study were analyzed using the optimization model. A paired t-test was designed and used to confirm the change in K, B, and/or θ_{vt} for the PKD test.

5.7.2 Methodology and Experimental Procedure

The procedure of this aim is similar to procedures that are explained in previous sections.

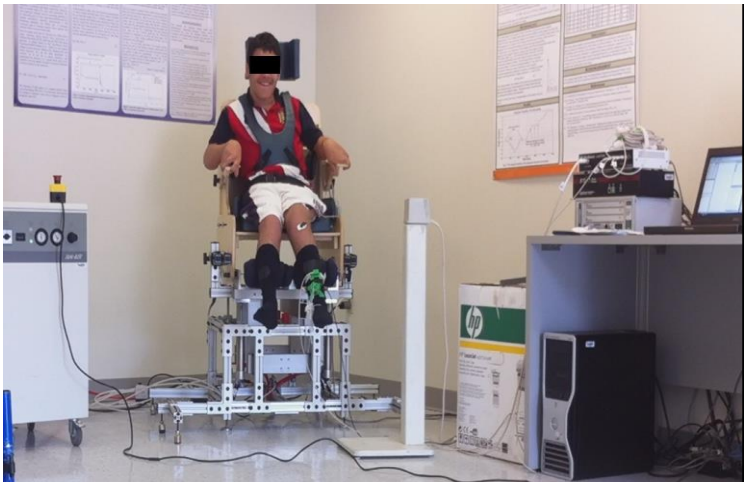


Figure 5.9 A representation of the vestibular stimulation system and the apparatus of the PKD test while a subject with CP is sitting on the chair.

The 15 minute stimulation session started at 1Hz for 30 seconds and increased to the desired 2Hz over the first minute. This was important to subjects with CP so they are allowed to adapt to the stimulation. The procedure of both the pre and post trials of PKD tests was similar and was administered by the same experimenter who lifts the examined limb to a specific angle against gravity then releases it, producing an angular trajectory as the limb falls due to gravity. Participants are described in Table 5.7, and an important inclusion criterion is that they were previously diagnosed with spasticity.

Table 5.8 The Physical Characteristics of the Participants. A Summary of the Anthropometrics for all Seven Participants

Subject #	*Age (Year)*	*Sex*	*Weight (Kg)*	*Height (cm)*	*Shank Length (cm)*
1	12	F	56.7	155	36
2	12	F	64.8	151	35.5
3	14	M	48.9	152	37
4	14	F	33	148	38.5
5	12	M	33	145	33
6	15	F	43	150	34
7	11	F	27	130	34.5

5.7.3 Results

A noticeable change in the appearance of the knee angular position data (pre and post stimulation) is seen in Figures (10-15). This change can be described as a decrease of the frequency of oscillation, an increase in the amplitude of each cycle, and a steeper descent to the resting angle. These indicate decreases in joint stiffness and damping, and a change in θ_{vt}, respectively. The optimized parameters quantify these observations. Table 5.8 summarizes these parameters pre and post vestibular stimulation for all subjects.

Table 5.9 Summaries of the Pre and Post Output Parameters

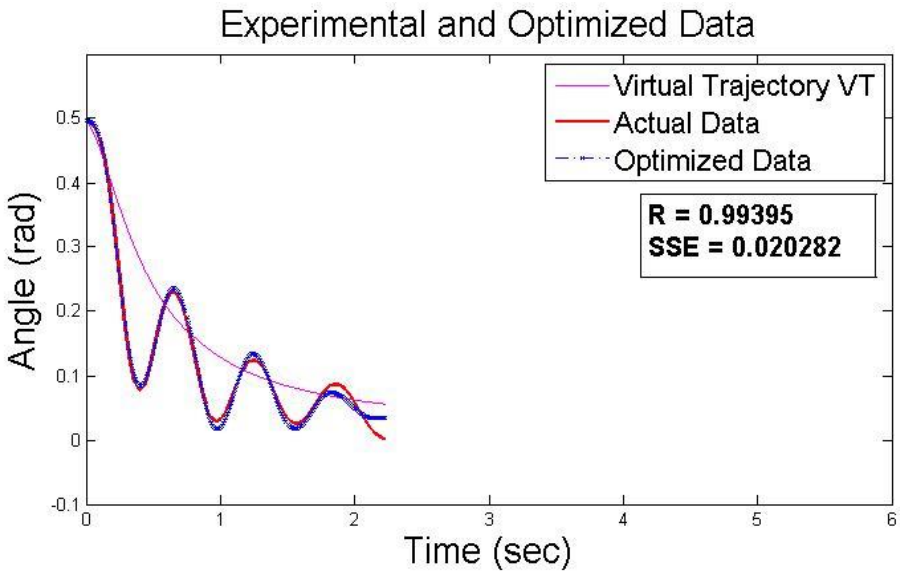
Subject #	(θ_{vt}) Time to inflection (Sec)		Damping (B) (N-m-rad/sec)		Stiffness (K) (N-m/rad)	
	Pre	Post	Pre	Post	Pre	Post
1	0.642186	0.378206	0.179556	0.055682	23.031519	16.614889
2	1.897597	0.842737	0.097578	0.040831	26.736352	16.037422
3	1.461600	0.561023	0.242000	0.042433	38.151500	14.891120
4	0.610485	0.227810	0.173606	0.057877	27.623828	21.168981
5	0.402020	0.184430	0.029886	0.021694	24.236279	17.183511
6	0.650309	0.536653	0.107584	0.057907	31.132244	24.578976
7	Very Stiff_ Subject was unable to relax for the PKD test_ See Figure 5.16 for details and explanation					

Table 5.10 Summaries of the Clinical Assessment of the Participants

Subject #	Classification	GMFS	Modified Ashworth scale left leg- PKD test	
			Quads	Hams
1	Spastic/Dystonic	4	1	3
2	Spastic	3	2	2
3	Spastic	3	2	2
4	Spastic/Dystonic	3	+1	2
5	Dystonic	4	+1	2
6	Spastic/Dystonic	-	2	+2
7	Spastic	-	+1	0

The optimization model used to analyze the data is a sigmoid model (θ_{vt}). The sigmoid inflection time indicated here describes the shape of θ_{vt} and a longer inflection time indicates a shallower sigmoid and therefore a shallower θ_{vt}, in contrast a shorter sigmoid inflection time indicates a steeper θ_{vt}.

A



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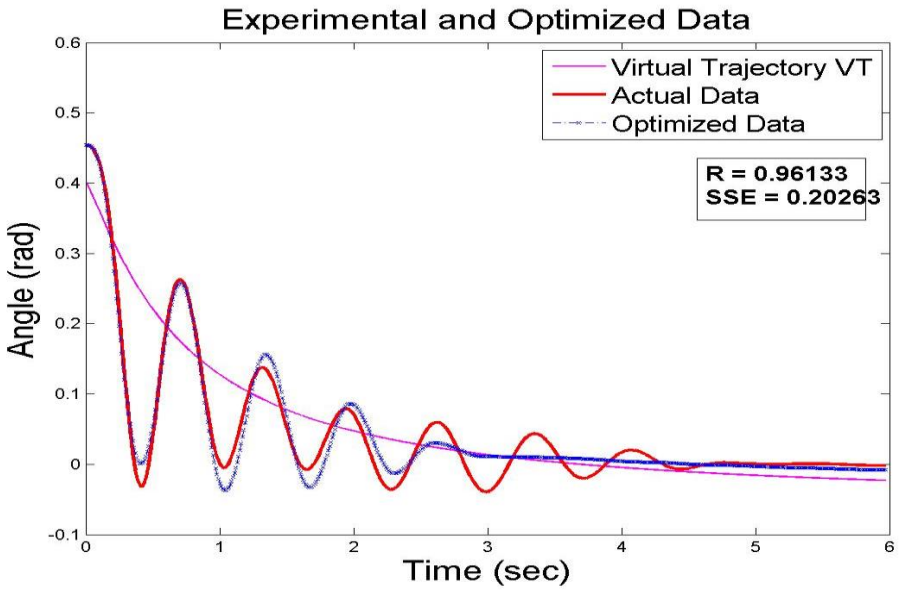
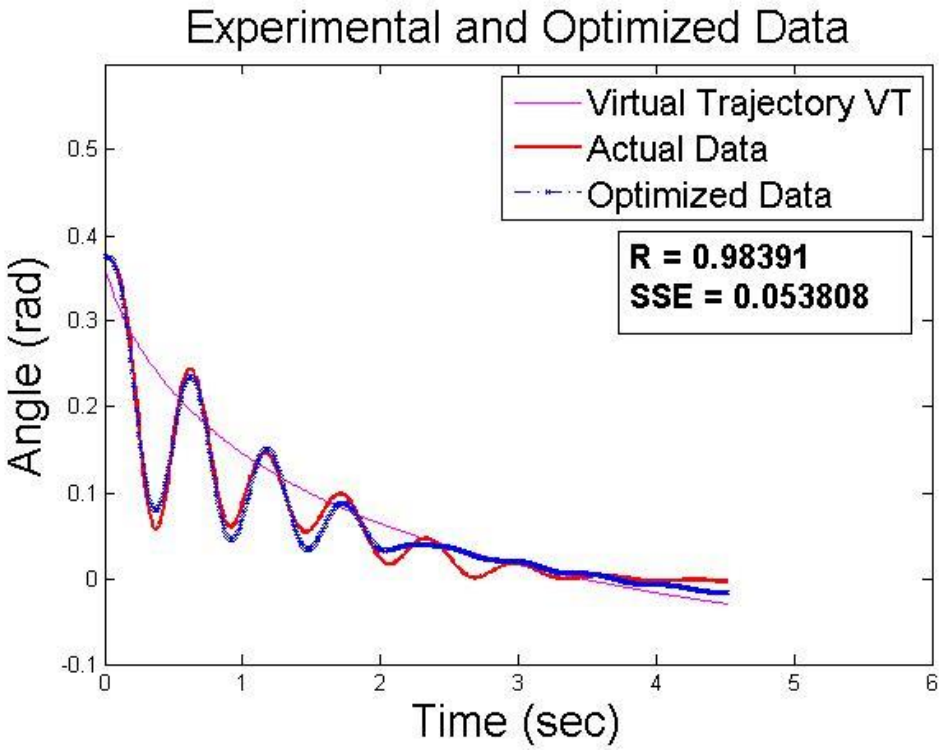


Figure 5.10 The optimuized PKD test data pre (A) and post (B) stimulation for subject 1. This subject is clinically diognosed with dystonia. The PKD test was administered and data in Figur 5.10.A were cliped at about 2 seconds because subject involuntarily moved their shank at that point due to dystonia.

A



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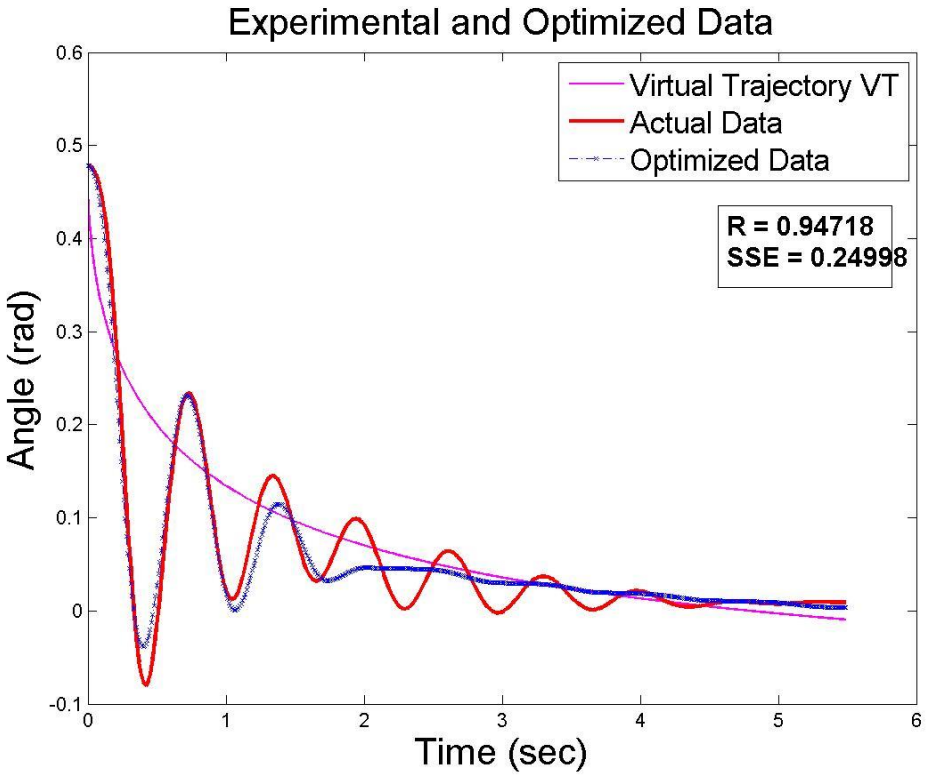
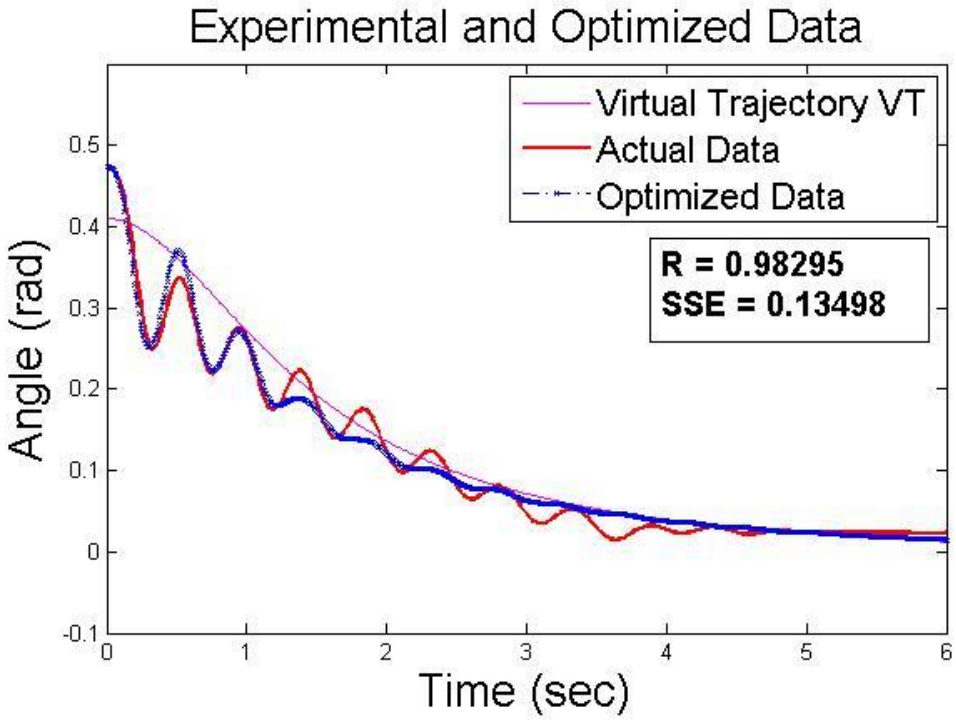


Figure 5.11 The optimized PKD test data pre (A) and post (B) stimulation for subject 2.

A



B

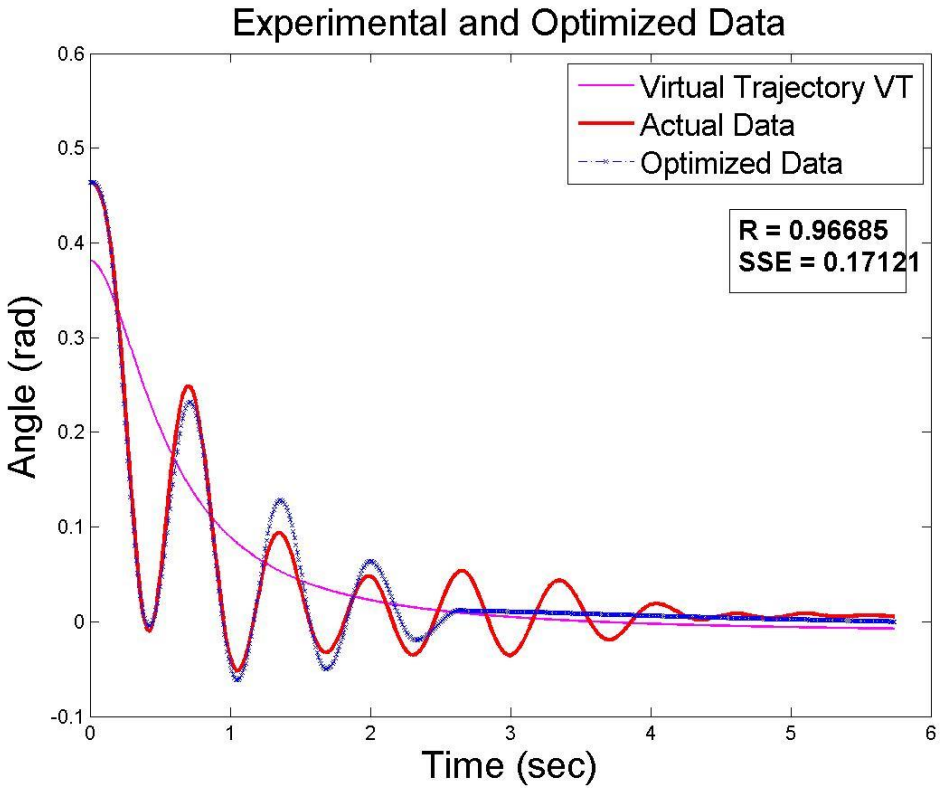
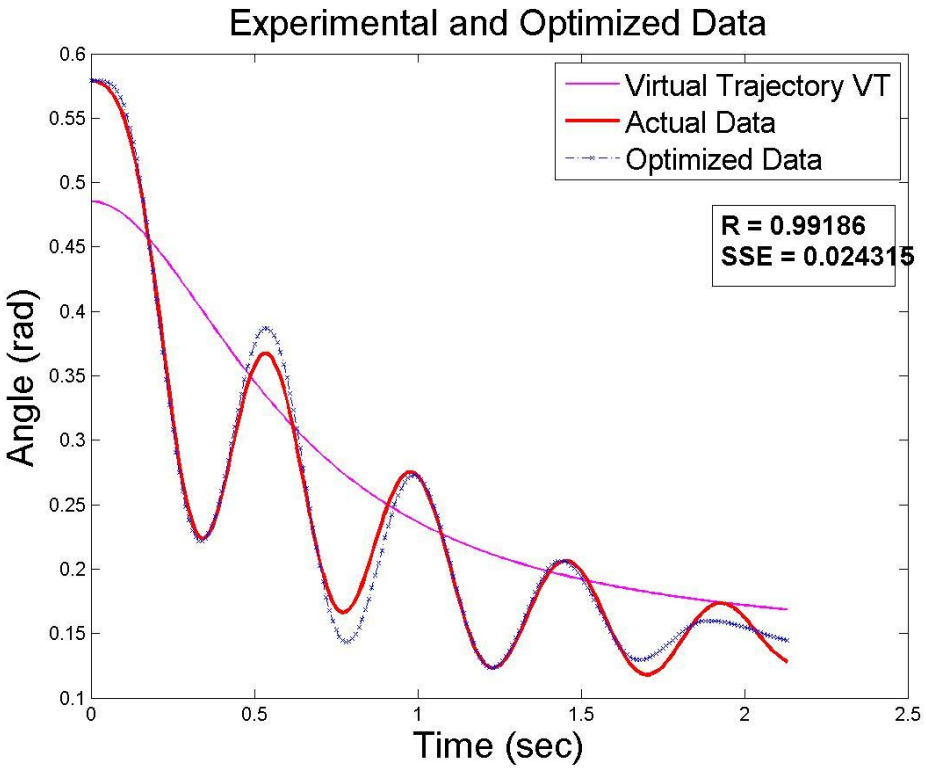


Figure 5.12 The optimized PKD test data pre (A) and post (B) stimulation for subject 3.

A



B

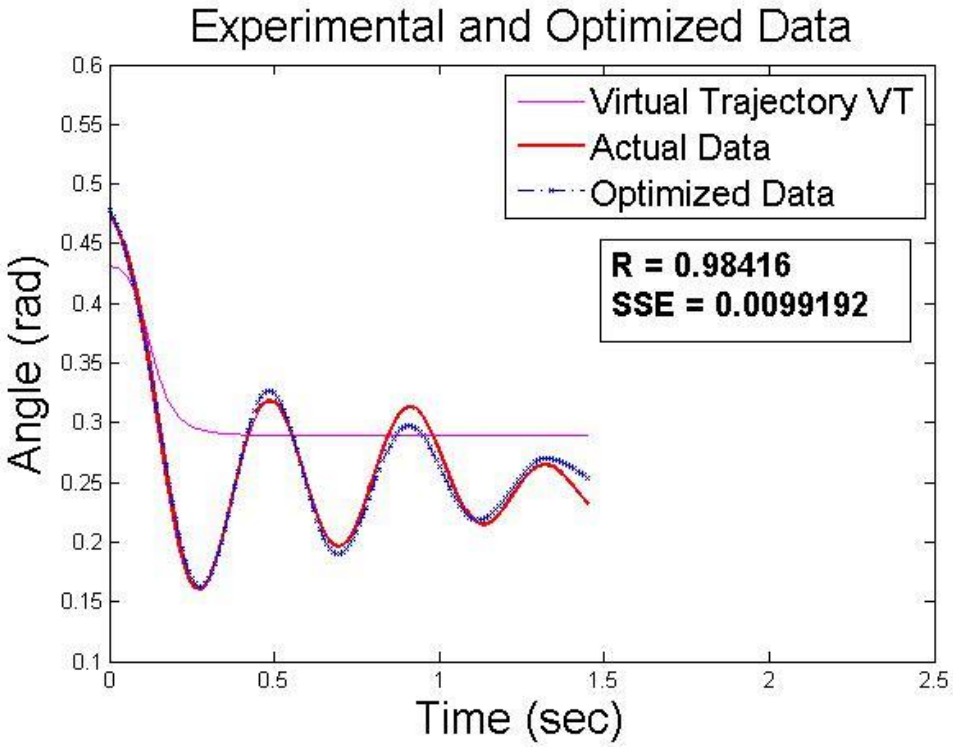
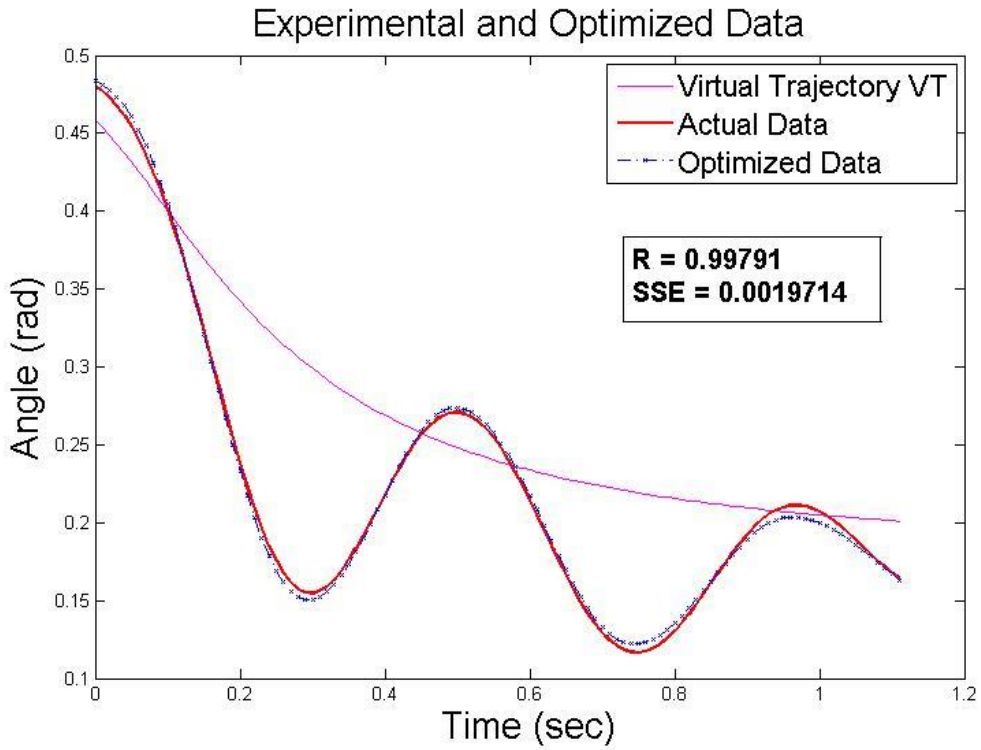


Figure 5.13 The optimized PKD test data pre (A) and post (B) stimulation for subject 4.

A



B

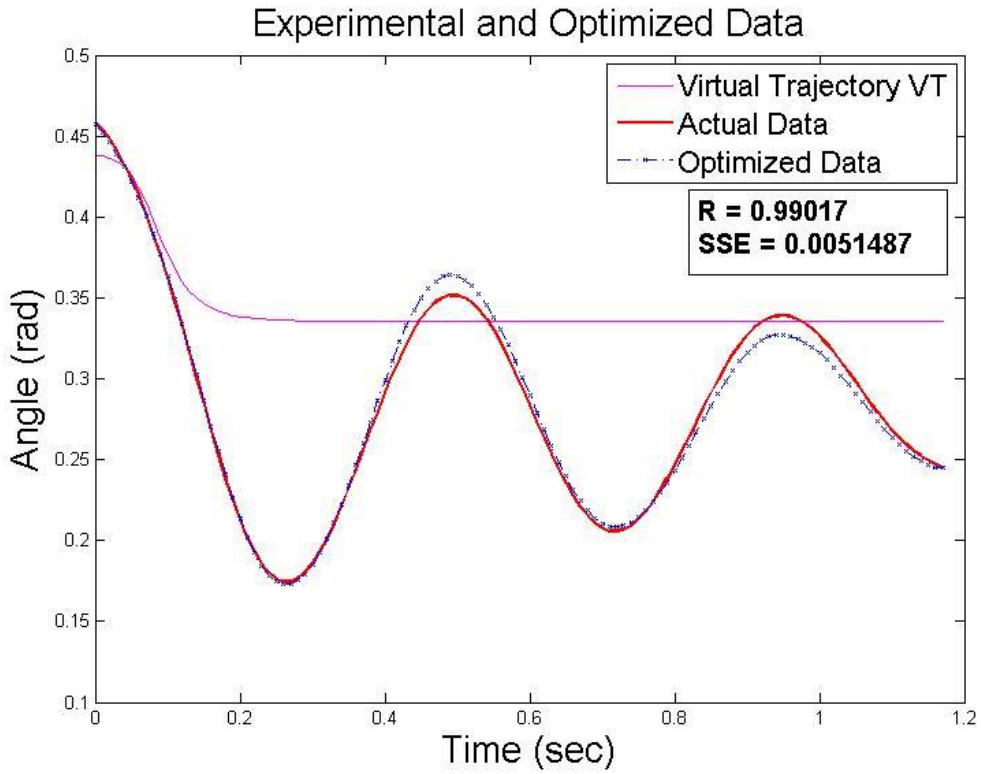
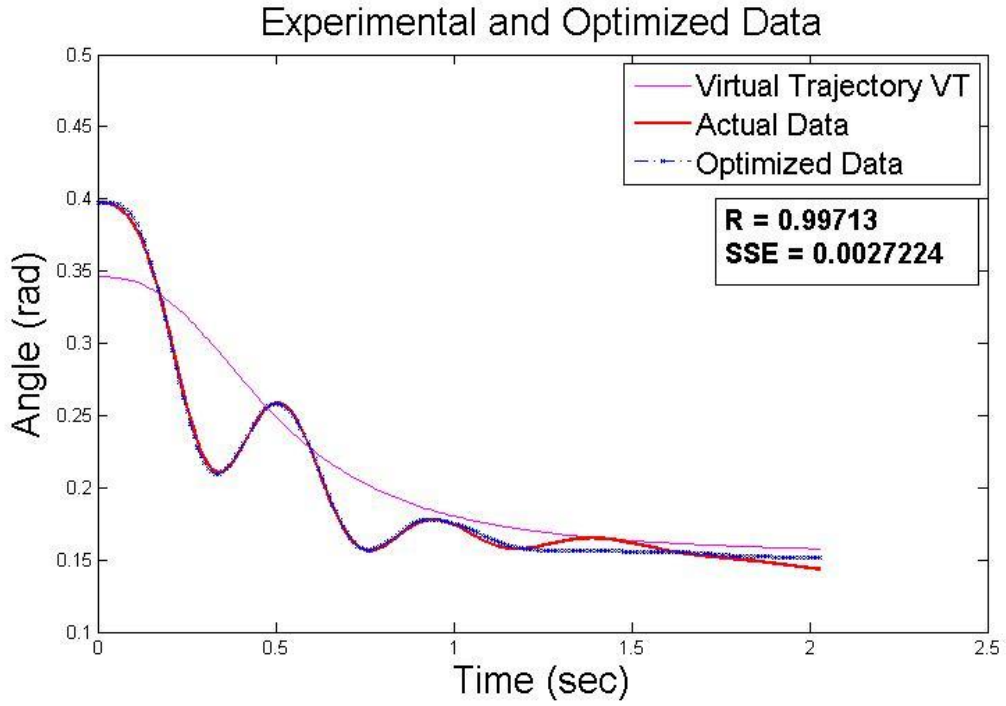


Figure 5.14 The optimized PKD test data pre (A) and post (B) stimulation for subject 5.

A



B

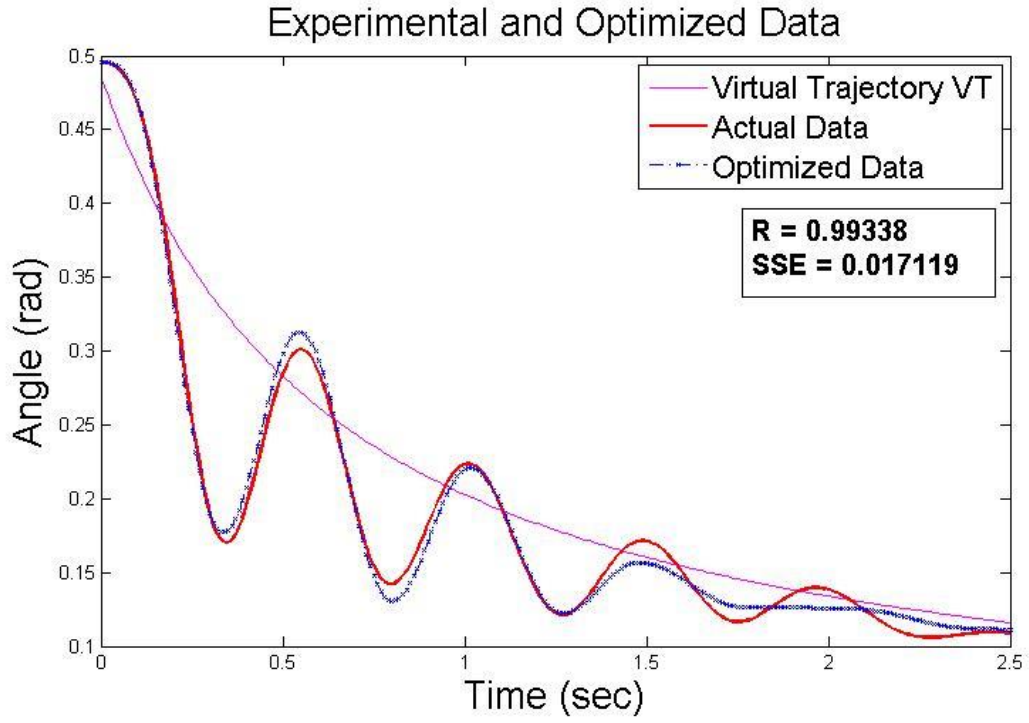


Figure 5.15 The optimized PKD test data pre (A) and post (B) stimulation for subject 6.

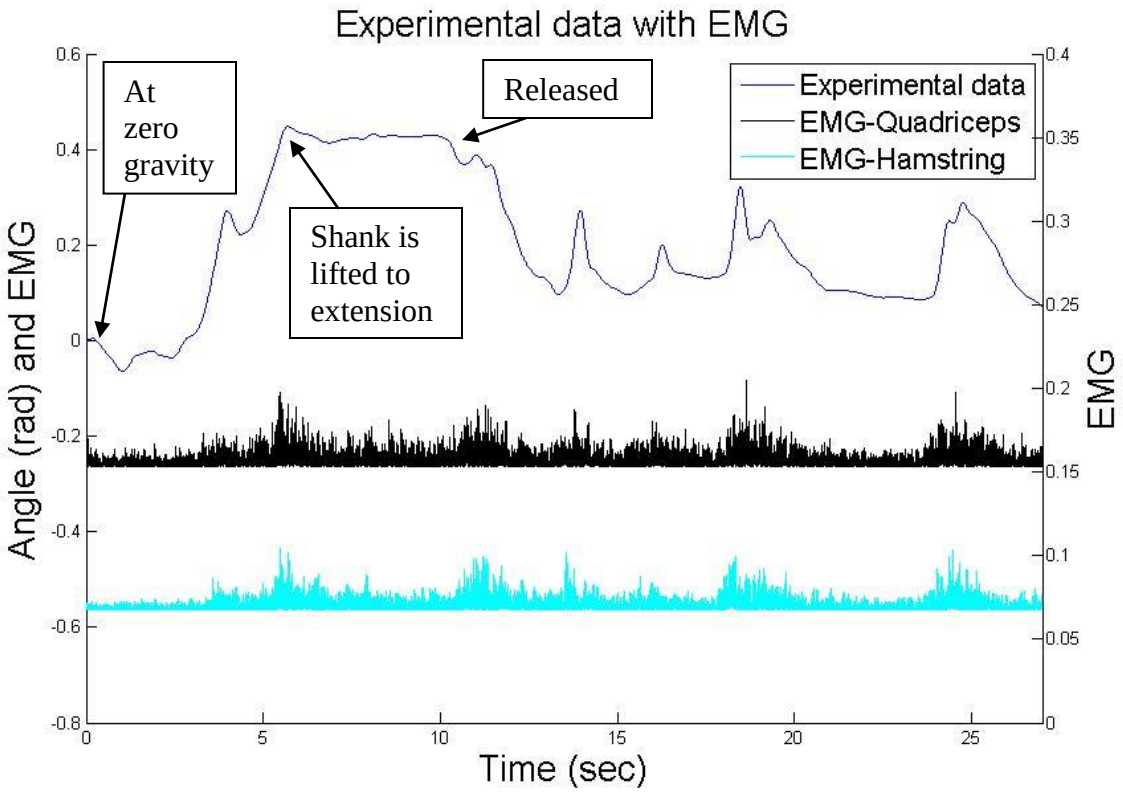


Figure 5.16 The angular position data along with EMG is represented for subject #7.

This subject experienced very high initial base line measure. This is an indication of an extremely high stiffness that subject is unable to control. PKD test was not successfully done on this subject, and the data had to be excluded from the study. EMG data show a continuous EMG bursts and muscle reflexes as soon as the research had an attempt to administer any action on the tested limb. It is also noticed that EMG bursts were synchronized on both the agonist and antagonist muscles (quadriceps and hamstring muscles) which caused a very high stiffness level at the tested limp.

The PKD test data for subjects #1, 4, 5, and 6 have a different kind of behavior due to sudden (muscle reflexes) that occur during the PKD test. This different behavior can be explained by looking at the VT of the data on the 1st half.

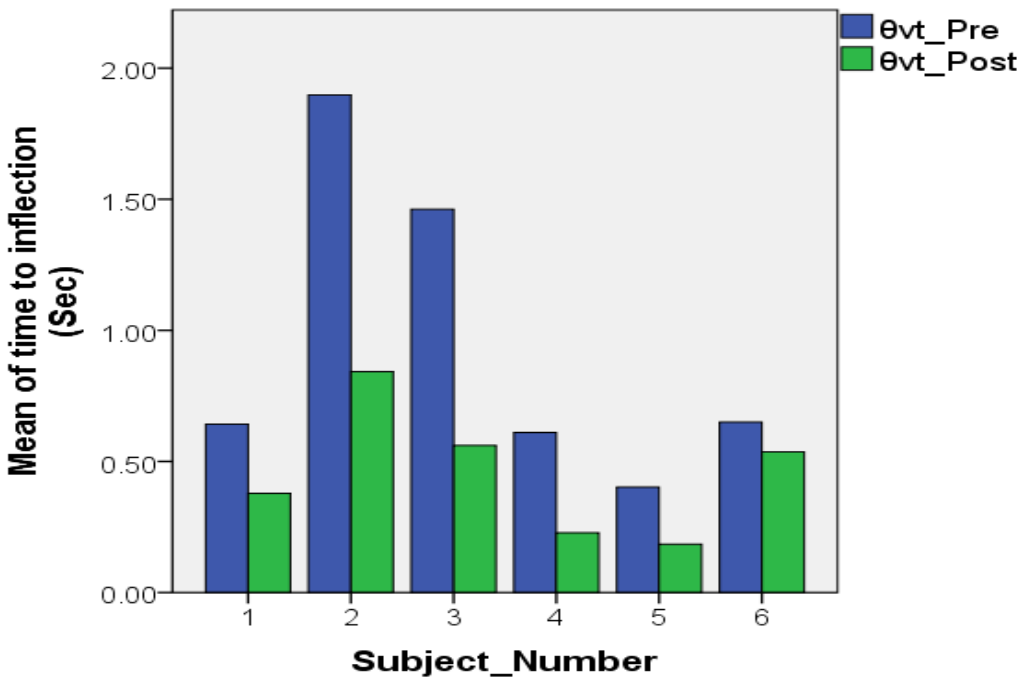


Figure 5.17 The optimized θ_{vt} parameter for the participants.

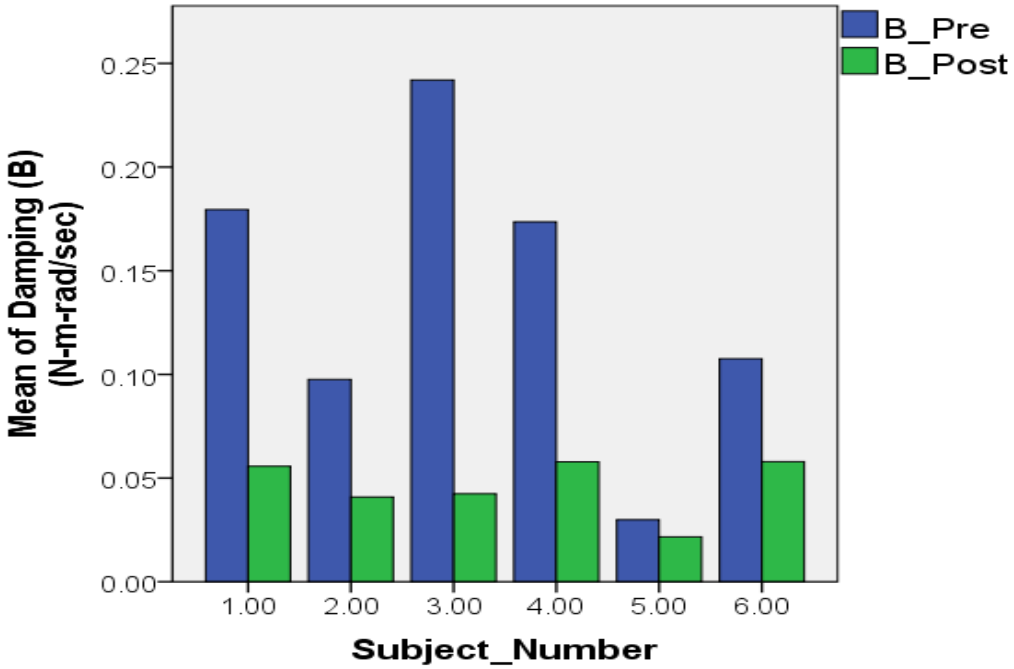


Figure 5.18 The optimized damping (B) parameters for the participants.

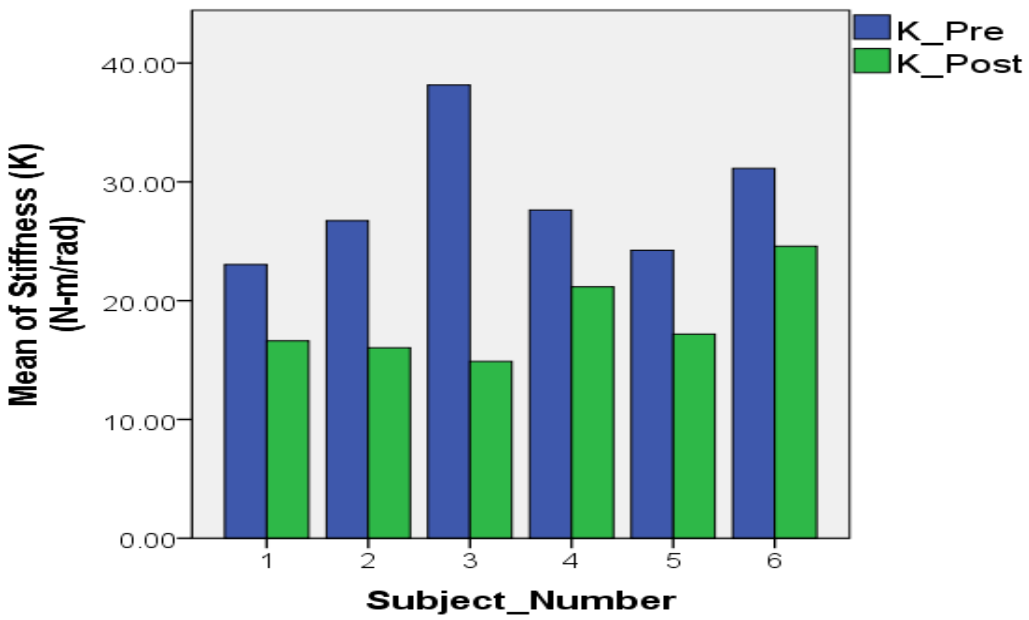


Figure 5.19 The optimized stiffness (K) parameters for the participants.

To better evaluate the changes in the optimized PKD test parameters, and to understand the effect of vestibular stimulation on participants, the data explained in Table 5.8 were analyzed using a paired t-test design. Data from subject #7 were not considered in the study since subject was unable to cooperate in the study procedures. An attempt to administer the PKD test is described in Figure 5.16 showing the angular position of the shank along EMG data collection from quadriceps and hamstring muscle. The statistical analysis results are described in Table 5.9 as the following:

Table 5.11 Statistical Analysis Results of the Paired t-test for PKD Test Data

Paired Samples Test			
Optimized Parameter	t	df	Sig. (2-tailed)
Pair 1: Post Stimulation (B) Pre Stimulation (B)	3.319	5	0.021
Pair 2: Post Stimulation (K) Pre Stimulation (K)	3.701	5	0.014

Paired samples t-test revealed that there is significant difference on knee stiffness pre-ride Vs. post-ride (paired-samples t-test, t (5) =3.319, p<0.05).
Paired samples t-test revealed that there is significant difference on virtual trajectory pre-ride Vs. post-ride (paired-samples t-test, t (5) =3.701, p<0.05).

5.7.4 Discussion

The results show a reduction in post stimulation parameters of stiffness and damping. The optimized parameters of θ_{vt} cannot be considered for a statistical analysis because θ_{vt} in dystonic subjects was controlled by dystonia and was different than θ_{vt} for subjects without dystonia, therefore we cannot run a statistical analysis including data of θ_{vt} from both groups, but can visually compare the appearance change in θ_{vt}. It was also noticed that θ_{vt} for subjects with spasticity only has changed due to vestibular stimulation and became stepper in post stimulation (Figure 5.11 and Figure 5.12). The limited number of subjects without dystonia did not allow running a statistical analysis on θ_{vt} for this group. These results are consistent with the appearance of the knee trajectories. The results here suggest that vestibular stimulation had a higher impact on stiffness and damping than θ_{vt}. It is also important to notice that the PKD test data for some of the subjects had to be clipped in order to get the PKD test analyzed through the optimization model. This was caused due to dystonia (involuntary muscle activation) which was experienced in 4 out of the 7 subjects (subjects: 1, 4, 5, and 6). Subjects 2 and 3 experienced only spasticity and subject 7 has an extremely high initial baseline measure of muscle tone and very high spasticity. Therefore, to understand the differences between subjects, it was important to classify them into two groups. The results found in each group were different. It is suggested that θ_{vt} was not affected by stimulation for subjects with a combination of dystonia and spasticity on the other hand; vestibular stimulation had an impact on subjects with only spasticity. This can be understood by the idea that dystonia controls setting θ_{vt} and therefore dystonia will overcome the CNS attempts to have a correction to θ_{vt}. When this happens, θ_{vt} may become fixed at a certain angular position. This

reinforces the idea that θ_{vt} is somewhat independent of K and B, and not a result of K and B.

Subjects (1, 4, 5 and **6**) do not have the classical shape of PKD test expected from a subject with CP. The shank is nearly vertical prior to administering the PKD test, then shank is pulled against gravity, held, and released. What appears to happen is that the leg oscillates about some constant angle before it actually goes down to zero vertical. The shank also may experience a sudden pull up or a drift downward after the oscillation. In one of the subjects (subject #7) the baseline EMG and the shank resting angle are very high. This happens with subjects who experience a very high level of spasticity. We are able to characterize the different level in spasticity by explaining the difference between the dystonic population vs. non- dystonic. To our knowledge, there has not been any PKD test work done to explain the level of dystonia. Other studies only distinguish them by saying "less affected and more affected" [50] with no further explanation and make no mention of dystonia. It is believed that it is possible to distinguish the two groups using the PKD, and can use our optimized B, K, and θ_{vt} to characterize changes in both spasticity and dystonia.

Based upon the results we can conclude that 15 minutes of vestibular oscillation is sufficient to stimulate the saccule to induce a marked change of neural origin in spasticity. In all seven subjects who participated in this study, the knee stiffness and damping values show a dramatic decrease post vestibular stimulation, a change is also noticed on the parameter describing θ_{vt} in subjects without dystonia. One way the result illustrated in this study can be explained is by the assumption that there is a reduction in the sensitivity of the antigravity muscles and an increase of the reflex threshold resulting

from the mechanical vestibular stimulation. Therefore, alterations to the vestibular descending signals while passing through the vestibular nuclei and going down toward the alpha motor neurons will command a change in the muscle activation patterns that are responsible for setting the level of spasticity or muscle tone. It is important to note that in some of the dystonic subjects it might be possible if they are upset or emotionally feel not very well, the knee would behave completely differently. Subjects also might increase their baseline tone which makes it extremely difficult to engage to perform any type of study.

Previous study conducted by Syczewska et al has shown that the PKD test has no statistical difference in the output of the PKD between trials of the same subject. In the study they examined 21 children with CP [50]. Another study done by Fowler et al has concluded that the PKD is a simple and useful tool for the purpose of assessing the lower extremities of individuals with CP, and that the first swing in the PKD is the most sensitive measure of the level of spasticity [55]. White et al have demonstrated that using PKD to assess spasticity and quantify changes to the level of spasticity is warranted as the data showed high to very high between day test-retest reliability of the thirteen variables calculated from the PKD in both individuals with and without spasticity [56].

A very interesting study conducted by Watt on astronauts in space in an attempt to better understand the effect of microgravity in space on changing the sensitivity of the spinal cord. The hypothesis was that the spinal cord excitability decreases and spinal cord react less to stimuli. To test this hypothesis the H-reflex was used in this study. The results revealed that the H-reflex response decreased significantly while in microgravity with a reduction of 35% in the excitability of spinal cord in weightless. Upon return to earth,

improvements in the H-reflex response were noticed in the day after landing, and full recovery was noticed after ten days [57]. The decrease in the spinal cord excitability has to be compensated by an increase signal projections from the brain. This might allow a greater value to the contribution signal of the otolith in setting the muscle tone in the muscles.

It has been reported in other research studies looking at the effect of muscle vibration that a reduction in spasticity is noticed as a result of the vibration [58, 59]. This might be one of the limitations to our vestibular stimulation. This can be examined by employing the H-reflex measurement to better understand changes in the threshold of the alpha-motoneurons caused as a result of vestibular stimulation and distinguish it from muscle vibration.

In another attempt to gain a deeper understanding about what exactly is happening with the virtual trajectory (θ_{vt}), the first half of the PKD test was further analyzed. Looking at the 1st half of the data and compare it to the 2nd half (with sigmoid shape in the beginning) it is noticed that the θ_{vt} in the 1st half plateaus at the end plateau of the 2nd half of the data (PKD test), therefore it has an effect on the way the shank oscillates about that specific angle (the end plateau in both halves of the data). The end plateau of the 1st half represents a VT for the hamstring, on the other hand during the beginning of the 2nd half of the data (modeled with the PKD) is a representation of a VT for the quadriceps alone. In the plateau seen shortly after releasing the shank, θ_{vt} is a combination of both VTs. The following Figures (5.20- 5.23) illustrate the idea of looking at both halves of the data.

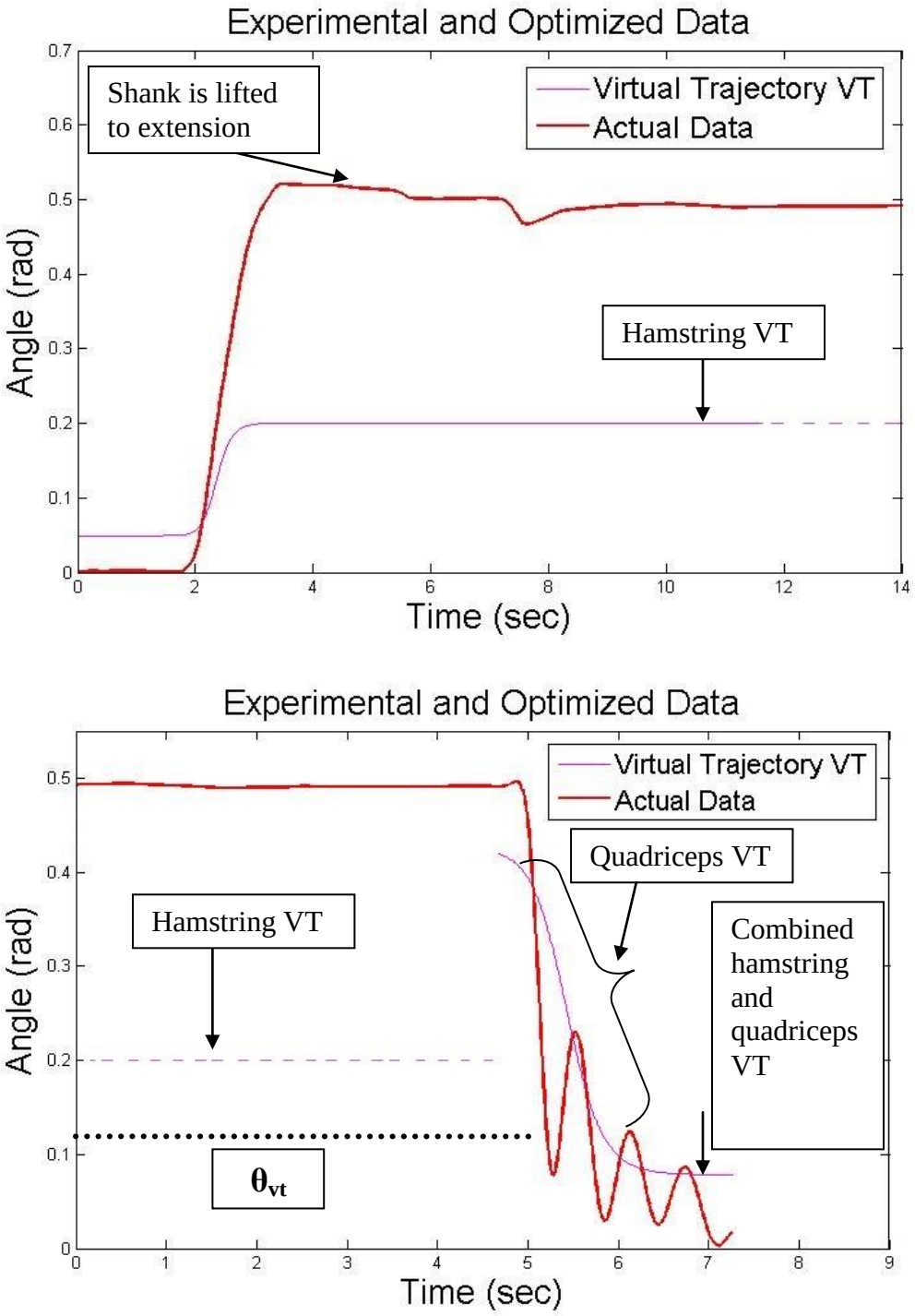


Figure 5.20 The location of θ_{vt} stuck in subjects with dystonia (subject 1).

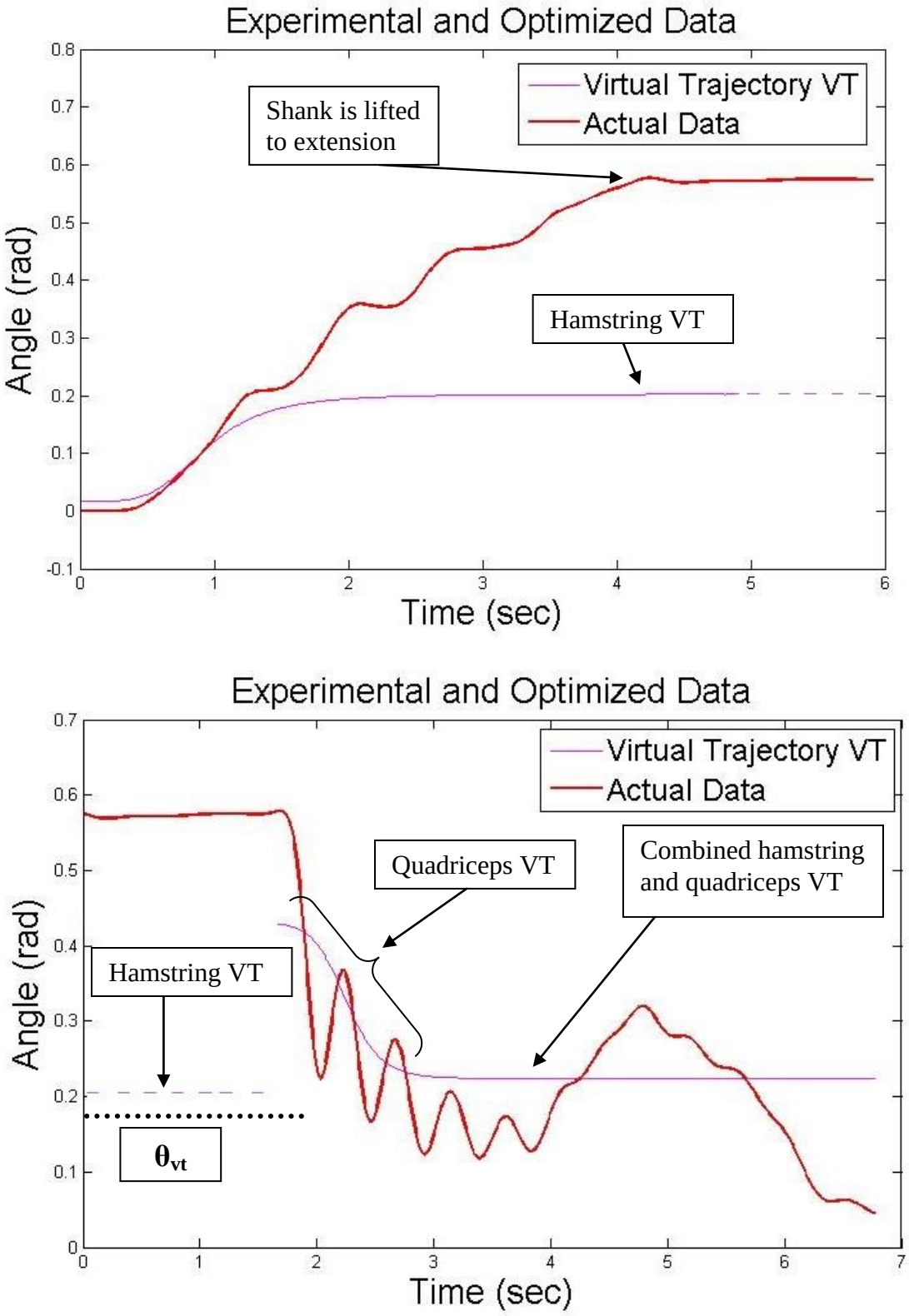


Figure 5.21 The location where θ_{vt} is stuck in subjects with dystonia (subject 4).

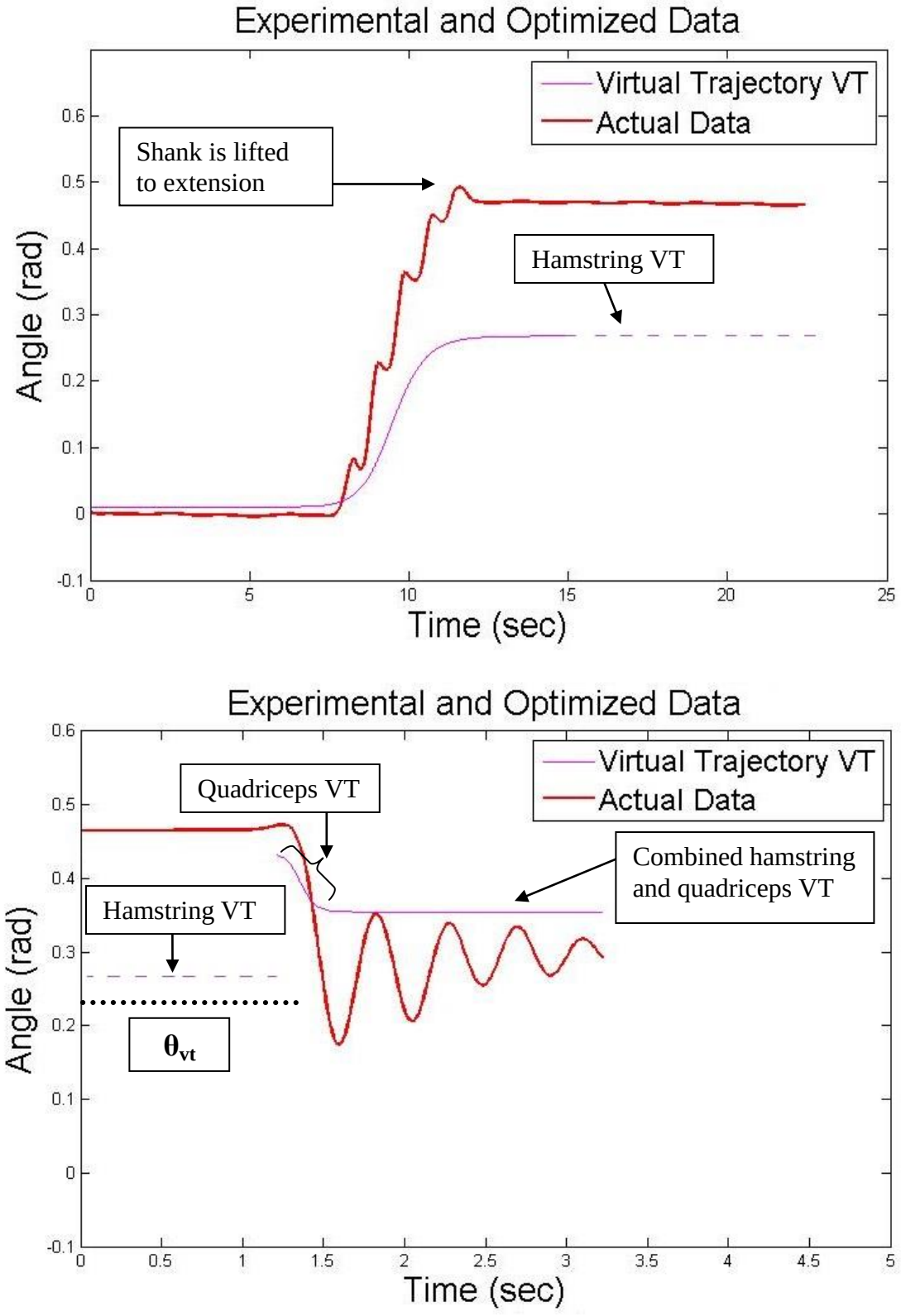


Figure 5.22 The location where θ_{vt} is stuck in subjects with dystonia (subject 5).

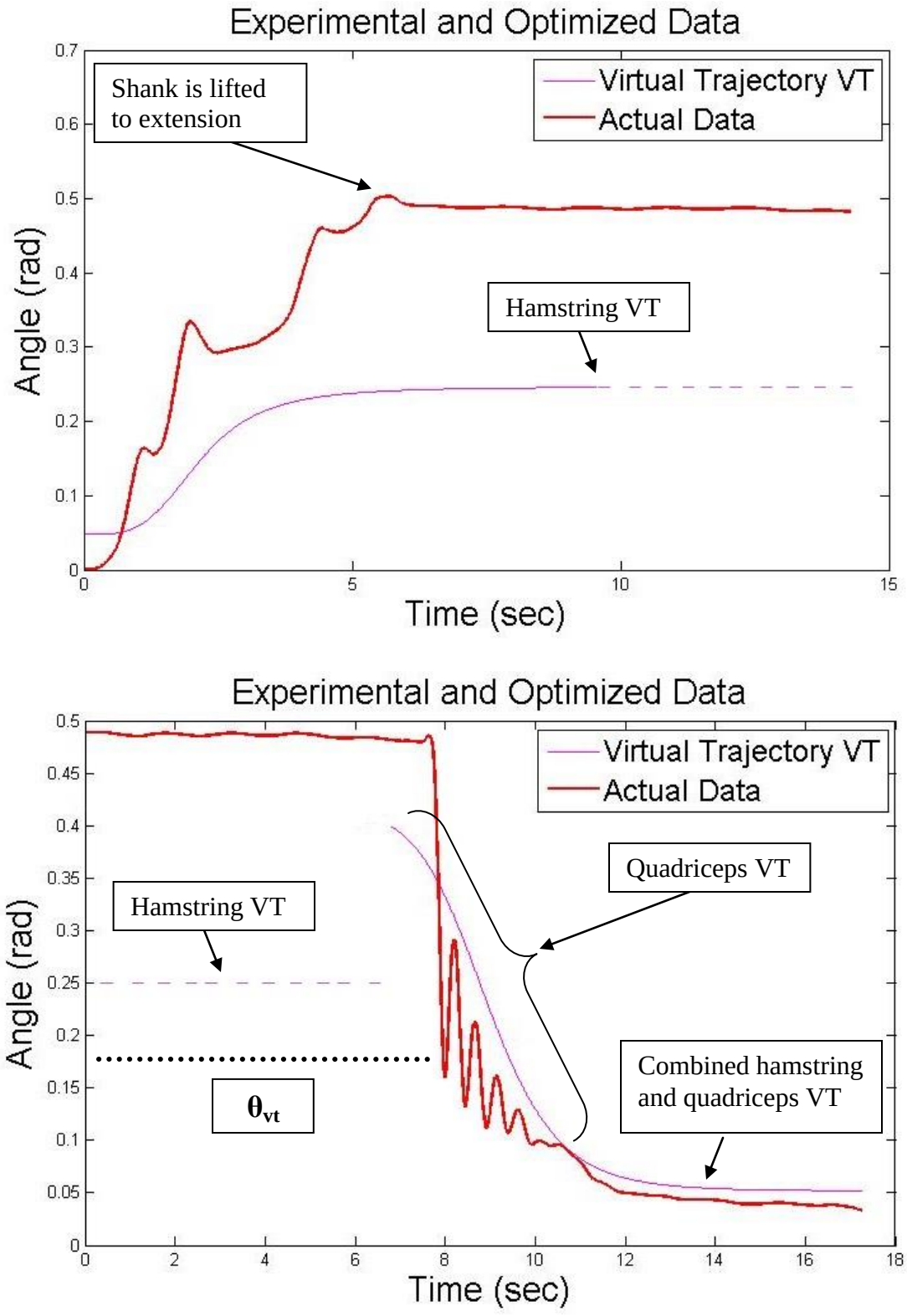


Figure 5.23 The location where θ_{vt} is stuck in subjects with dystonia (subject 6).

The end of the plateau in the 1st half of the data remains as the main angle of oscillation during the PKD. This is an interesting phenomenon as it actually explains the cause of the non-classical appearance of the PKD test where the shank appears to oscillate about an angle greater than zero, then changes over time to reach zero. This is an appearance that is mostly seen in subjects with dystonia.

In order to investigate this phenomenon, an experiment was done to confirm the origin of this unusual behavior of θ_{vt}. This involved one subject with dystonia, and the attempt was to administer the PKD test on the subject with some modifications as the following: Firstly, the PKD test is administered to locate where the shank is oscillating about during the PKD test. Then the experimenter lifted the subject's shank against gravity to almost the maximum extension, holds it in place for ~3 seconds, then slowly lowers the angle at which the shank is held, hold for ~ 3 seconds, then release. Basically during the lifting of the shank, experimenter is triggering the CNS to provide a θ_{vt} that follows the actual position of the limb, but in fact what happens is that the θ_{vt} gets stuck somewhere along the way. To determine this, by lowering the shank down to almost the same location where θ_{vt} is noticed to get stuck (experimenter is allowing for $\theta_{actual} \approx \theta_{vt}$) which should not cause a difference in position or might generate very minimal amount of moment. Then, once the shank is released, shank stays for very short time static then drifts down to its zero vertical. Figure 5.24 describes a representation of this procedure.

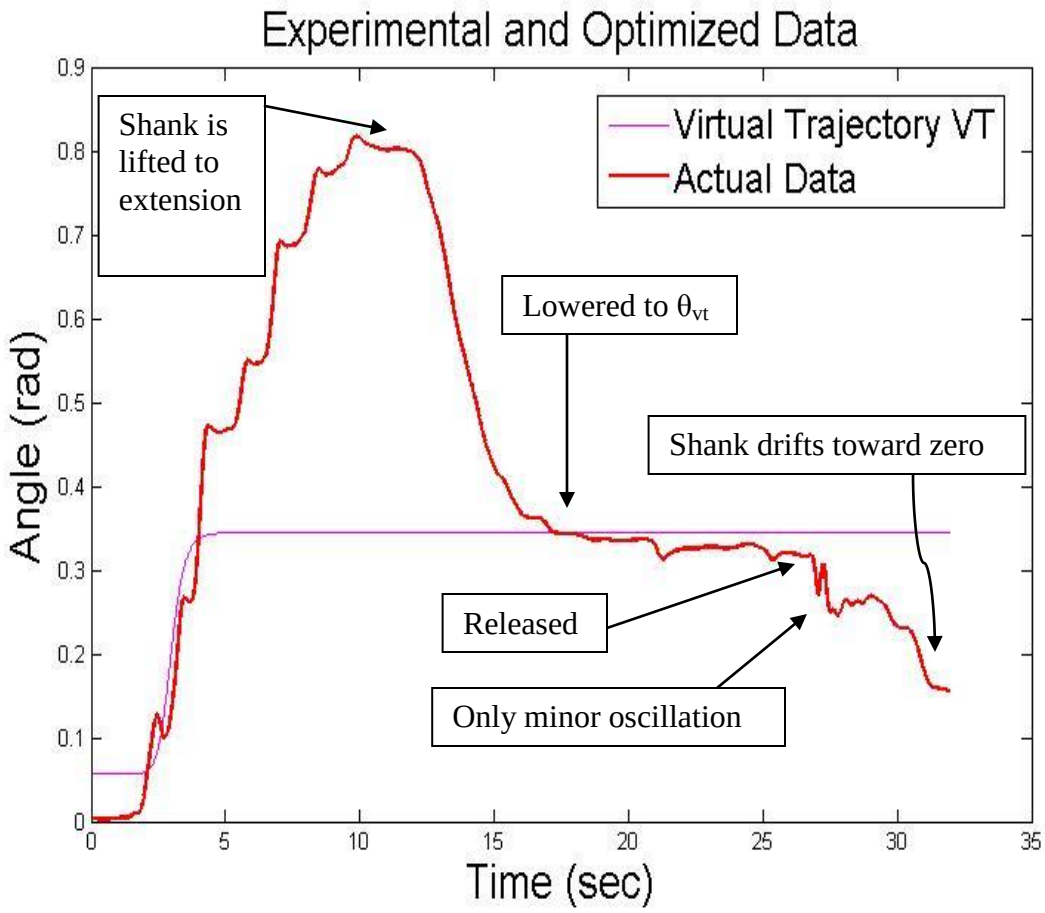


Figure 5.24 The plateau θvt during pull up and PKD in subject with dystonia (subject 6).

In dystonic subjects we can claim that the θ_{vt} gets locked at some angle during the pull up of the shank against gravity and stays there, therefore during the PKD test since the shank's θ_{vt} is actually below the angle that it is held at, the leg, once dropped, oscillates about that locked angle then drifts down to zero vertical. The angle that the shank's θ_{vt} gets locked at can be defined by analyzing the first half of the PKD test data and is described by the horizontal plateau of the shank's θ_{vt} during the pull up and the holding portions before the point of release.

5.7.5 Conclusion

The benefit of this work is not only limited to the fact that we are able to characterize the changes in PKD trajectory post mechanical stimulation, but in addition to that we can distinguish and describe the differences between subjects with and without dystonia. It is also interesting to notice that the two subjects with spasticity and without dystonia provided feedback stating that they felt much relaxed after stimulation. This feeling lasted for several hours (These children are smart enough to distinguish their level and provide a plausible self-assessment). This can be an important entity that has to be measured on participants in future studies by including the Gross Motor Function Measure. The following are the major conclusion points of this study:

- It is the belief that the repeated vertical movement of the vestibular chair at a frequency of 2 Hz for 15 minutes stimulates the otolith to sense falling (in the direction of gravity) and acceleration upward. This stimulation has a beneficial effect for a minimum of 15 minutes. We believe that this repetition introduces a temporary habituation or recalibration to the central nervous system's response to vestibular afferents. The effect is to alter the vestibular contribution to muscle tone. This key point opens a number of interesting ideas that should be considered for future work:

    a. A time window in which we believe will allow a more accurate assessment of existing motor skills and function. This might be important if physical therapy can be done during this period of time. (Future study)

b. Opportunity to offer therapy provided by physical therapists among the period of lower spasticity while the vestibular stimulation effect. (Future study)

c. The effect of stimulation on spasticity in the upper extremity. (Future study)

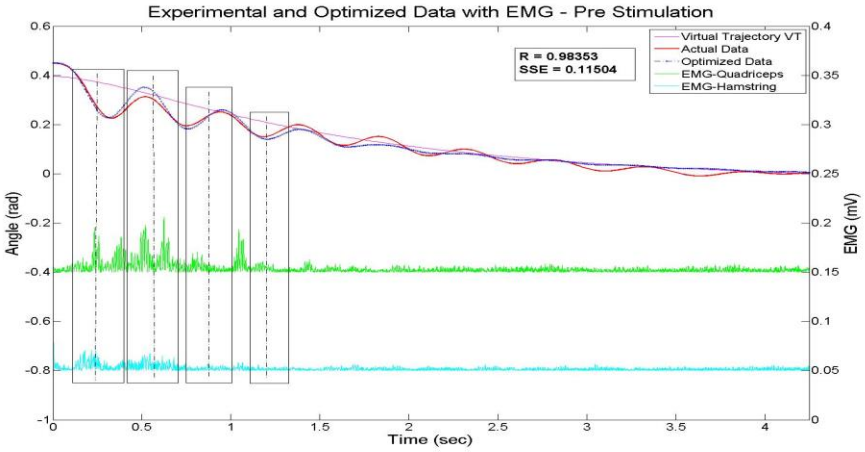
- In all subjects in this study except subject 7, the optimized parameters of stiffness and damping were decreased. θ_{vt} changed due to stimulation in subjects with spasticity only, but saccular stimulation did not impact θ_{vt} for subjects with both spasticity and dystonia.
- The observation of dystonic PKD test shows a different θ_{vt} due to dystonia. θ_{vt} in subjects with dystonia is in fact independent of changes to K, and B. Subjects with dystonia seem to have a limit in θ_{vt}.
- Using the PKD test, we are able to separate individuals with spasticity only from individuals with spasticity and dystonia.
- Using the element of EPH (K,B, and θ_{vt}), we are able to effectively understand passive knee rotation.

It is important to note that: 1) this work cannot be claimed as a permanent treatment for children with CP, but a combination of the described stimulation along with the proper physical therapy might have a very positive effect on the disorder. 2) Another path that can have a similar impact on the described population is by changing the duration intensity and providing the stimulation more frequently for about 5-7 weeks might have a major impact on reducing the level of spasticity in children with CP.

APPENDIX A
PKD, EMG, AND THEIR RELATIONSHIP WITH THE EPH

Subsequent data analysis (on subjects with spasticity without dystonia) with the inclusion of EMG suggested a potential linkage between the EPH and the muscle reflexes (EMG).

A



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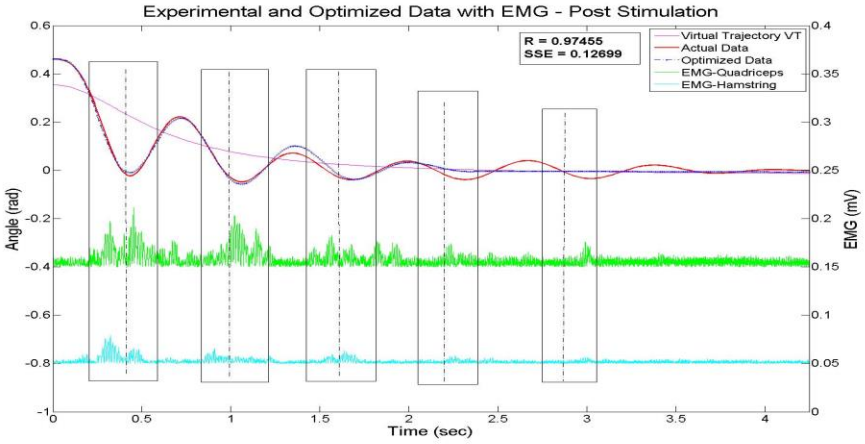

Figure A.1 The experimental and optimized trajectories along with EMG of subject #3 for pre, and post stimulation are illustrated in Figure A.1..A and A.1.B consecutively. EMG- Quadriceps is shifted by 0.15 (mv) and EMG- Hamstring is shifted 0.05(mv) for better illustration of the plots.

In the model the reflex torque is represented by the term K (θ- θ_{vt}). The vertical boxes that are added to Figures A.1.A and A.1.B have left and right boundaries aligned with the intersection of the virtual and actual trajectories where θ= θ_{vt}. The vertical dashed lines in each box are aligned with the point where θ- θ_{vt} is at a maximum for each cycle. An interpretation of this is that the EMG bursts begin and end within the boundaries defined by θ= θ_{vt}, and reach their peak amplitude coincidental with θ- θ_{vt}= max. Interestingly the inflection points of the actual trajectory which correspond to the maximum angular velocity do not coincide with the peak EMG which would be expected if the reflex were entirely velocity dependent. This raises questions about the conventional definition of spasticity. This, along with investigation of stimulation dosage, stimulation effect, and possible functional gains (e.g., gait and balance) are the future aims of this research. Schematics

APPENDIX B
3D DESIGNS AND SCHEMATICS

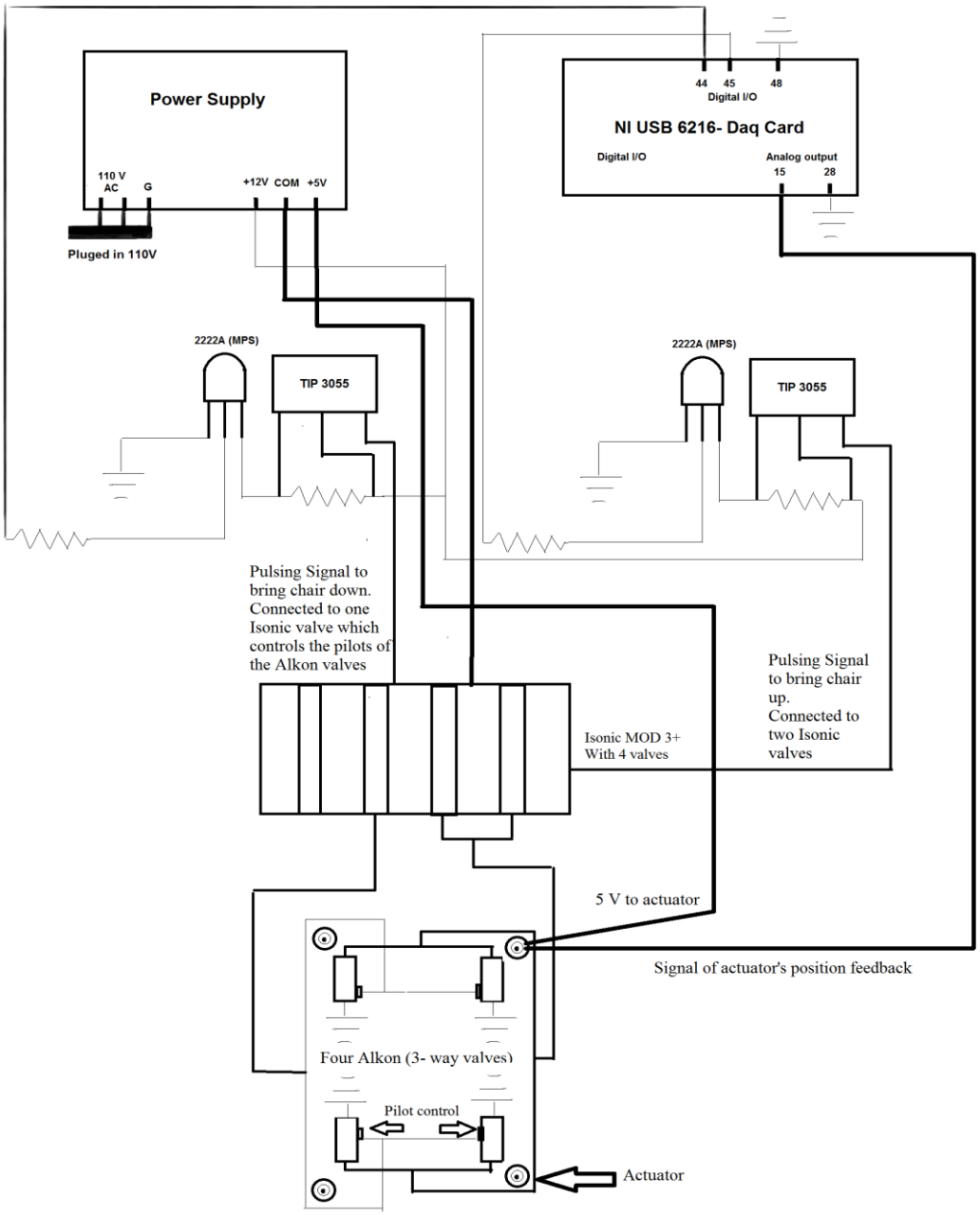

Figure B.1 Schematic of the main electronic controlling the vestibular stimulation chair.

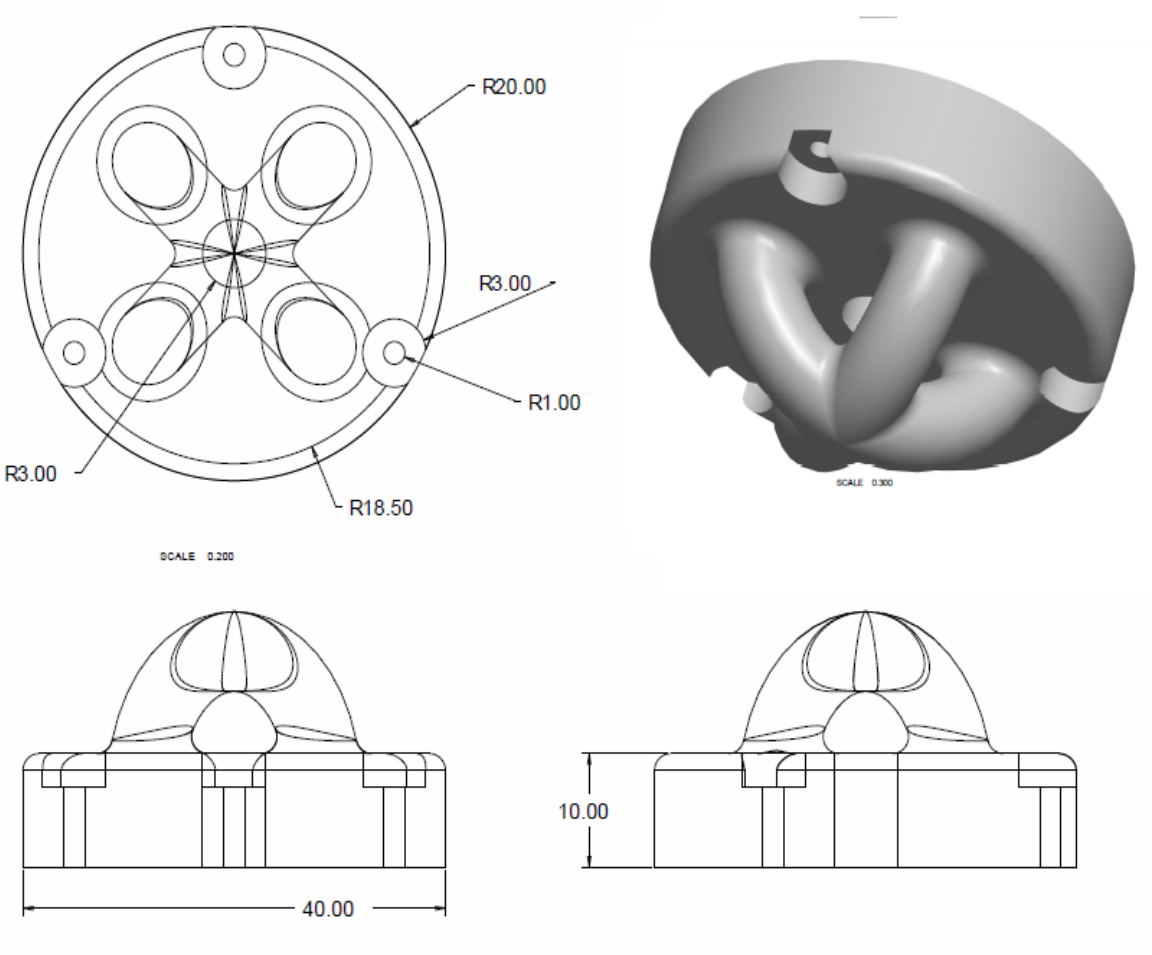

Figure B.2 The top portion of the force transducer attachment. This portion has an access to insert a string, which it can be used to lift the subject's segment. On this part there are three hole that are designed to connect it to the top plate of the force transducer using three screws.

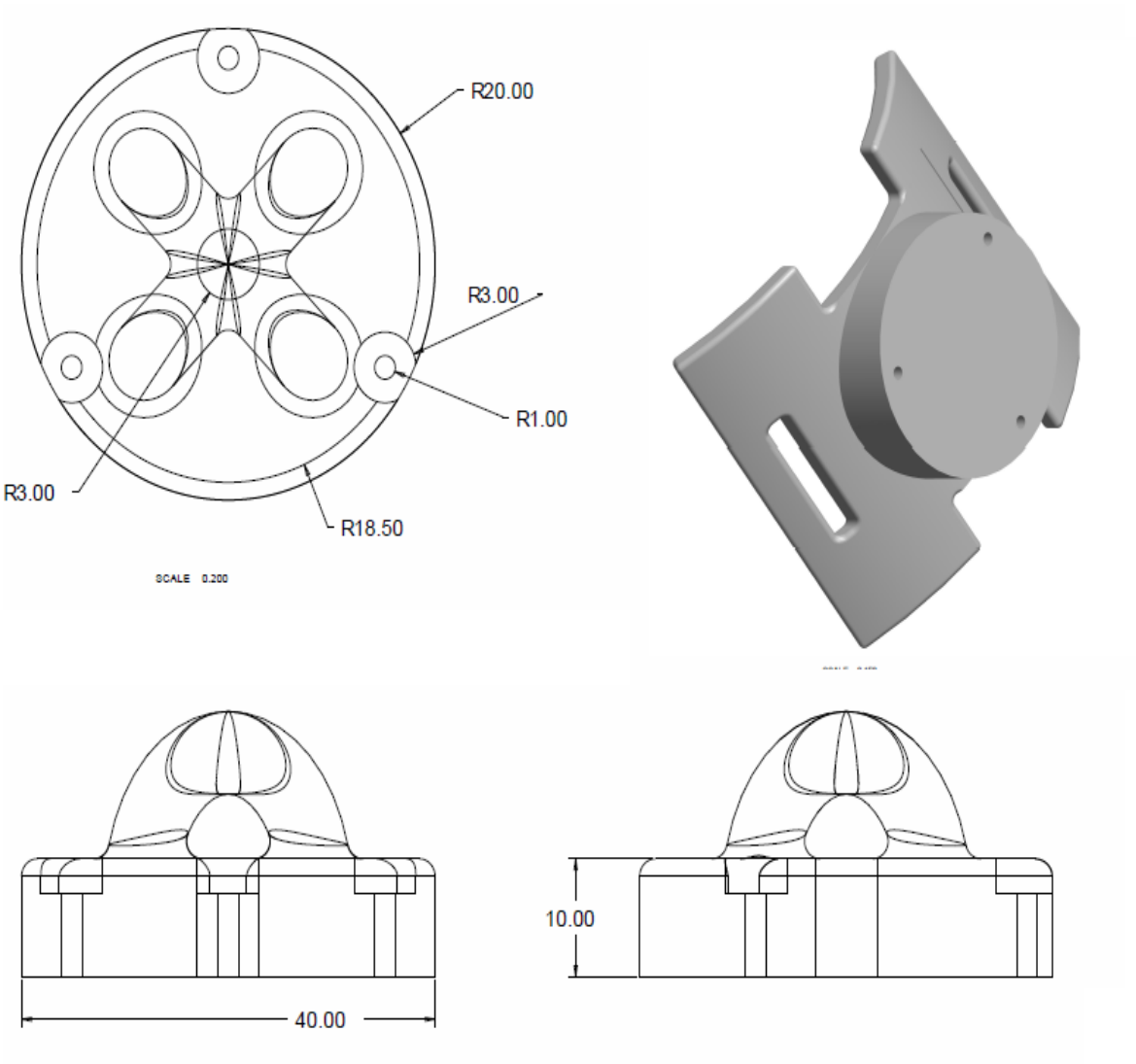

Figure B.3 The bottom portion of the force transducer attachment. The force transducer gets attached to this this portion using three screws.

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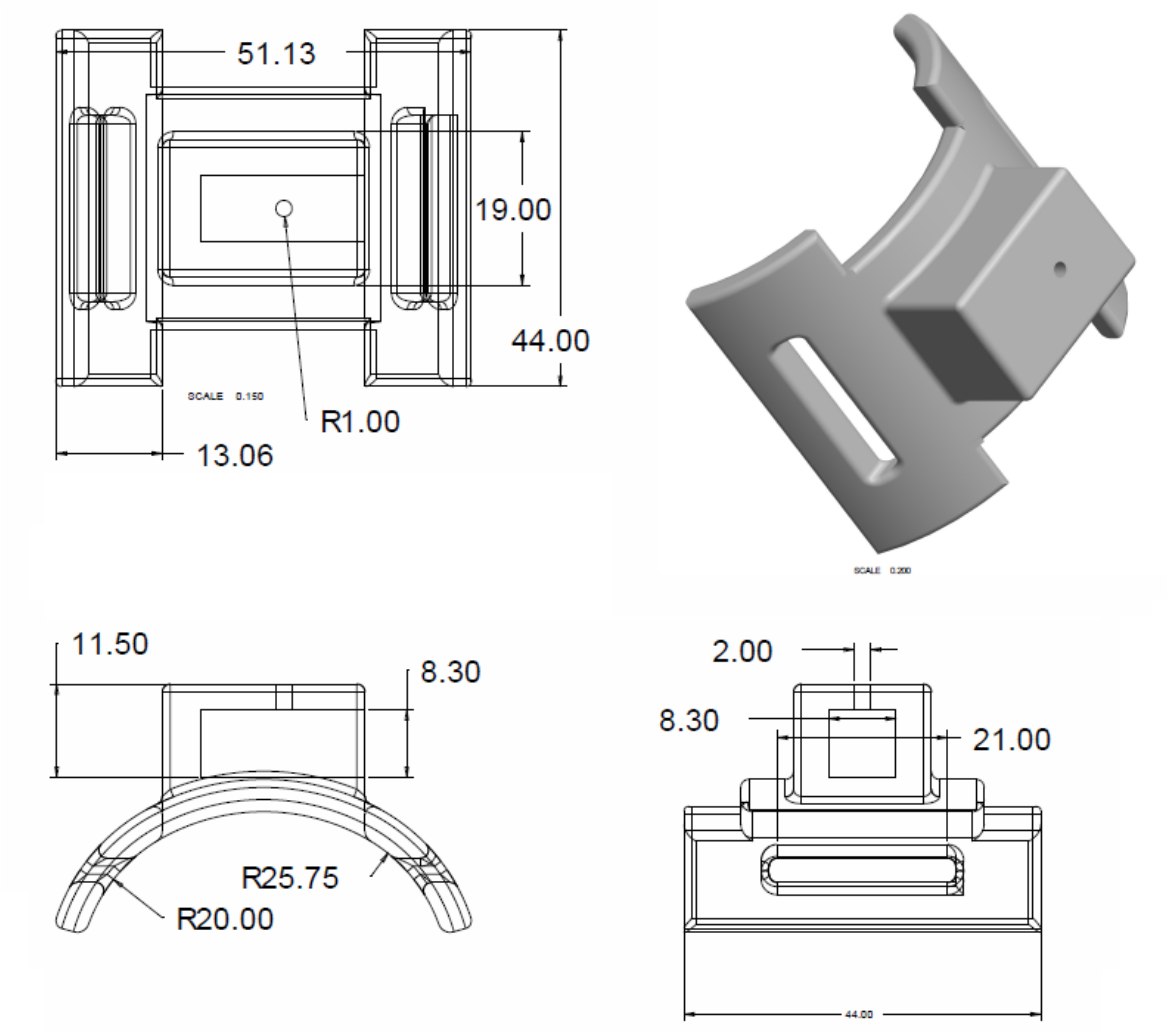

Figure B.4 The housing of the trakSTAR sensor. This portion has an access to insert Velcro straps so it can be easily attached to subjects segment. There is also a small hole on top of the housing to securely hold the sensor in place.

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