The Effects of Injury on the Neuromotor Control of the Shoulder

By

Hannah Simpson

July 2020

Director of Thesis: Dr. Chris Mizelle

Department of Kinesiology

Abstract: The shoulder is one of the most mobile and unstable joints in the body. When the function of the shoulder muscles is altered, and it is without the appropriate neuromotor control, the shoulder can become dysfunctional. It is unknown how previously injured individuals vary in movement patterns or whether their brains change compared to their healthy counterparts. The **purpose** of this study was to compare neuromotor control of the shoulder between individuals with and without a previous shoulder injury. To achieve this, we used an upper extremity task with motion capture to analyze kinematic performance of the shoulder complex and electroencephalography (EEG) to evaluate neural connectivity of the brain. We **hypothesized** that individuals with previous injury to the shoulder would have different kinematic patterns as well as a less direct or evasive way of achieving their goal-oriented trajectory. We also hypothesized that participants with previous shoulder injury to have more diffuse patterns of brain connectivity during performance of the task, as compared to healthy participants. Our **kinematic results** made it evident that healthy and post-injured individuals have different anterior/posterior trunk displacement and hand pathways toward their targets. Our **neurological results** showed significant changes in brain connectivity in post-injured individuals across conditions. RPE scoring increased and decreased in response to an increase and decrease in

weight resistance, but scores were higher in post-injured individuals. Further research is needed to understand how individuals modify movement kinematics in different joints and determine how consistent these changes are across tasks and patterns of brain activation.

The Effects of Injury on the Neuromotor Control of the Shoulder

A Thesis Presented to

The Faculty of the Department of Kinesiology

East Carolina University

In Partial Fulfillment of the Requirements for

The Master of Science in Kinesiology

Biomechanics and Motor Control Concentration

Hannah Simpson July 2020

THE EFFECTS OF INJURY ON THE NUROMOTOR CONTROL OF THE SHOULDER

by

Hannah Simpson

APPROVED BY:

DIRECTOR OF THESIS:

Chris Mizelle, PhD

COMMITTEE MEMBER:

Nicholas Murray, PhD

COMMITTEE MEMBER:

Patrick Rider, MS

CHAIR OF THE DEPARTMENT

OF KINESIOLOGY:

J.K. Yun, PhD

DEAN OF THE GRADUATE SCHOOL:

Paul Gemperline, PhD

Table of Contents

Chapter I: Introduction

The shoulder is one of the most mobile and unstable joints in the body. The superficial muscles and especially the rotator cuff play an important role in stabilization and control of complex courses of motion(1). When the function of the shoulder muscles is altered, and it is without the appropriate neuromotor control, the shoulder can become dysfunctional. This could result in poor performance of athletes during competition and individuals performing daily activities. The functionality of the muscles around the shoulder after injury, and how chronic injury affects neuromotor control strategies, have not been well documented. This study sought to identify the relationship of compensation due to shoulder injury on brain activation (using electroencephalography; EEG), and differences in movement kinematics among healthy and post-injured participants (using 3D motion capture).

Injury to a major joint of the body like the shoulder complex can often result in various alterations in an individual's activities of daily living. Many individuals strive to regain their normal routines as quickly as they can. Active individuals among this population strive especially hard to reclaim their fast-paced lives, yet this quick recovery can cause more harm than good(2). Interest in how the brain and neuromotor system adjust following injury, as well as the deficits in range of motion, has grown over the years. The ability of an injured shoulder to complete the same movements as before the injury becomes of question(3). It is known that the efficacy of muscle activity is dependent upon the optimal alignment of the scapula on the chest wall and the length-tension relationship of the scapular stabilizers and rotator cuff. Therefore, for optimal dynamic control during activity, the scapula stabilizers must activate in a consistent and coordinated fashion(1). This ability to call on and activate various muscles to perform a multitude of tasks involves a deeper look into the neural networks related to brain connectivity.

Brain activity is dependent on the task (i.e., cognitive or physical) in which an individual is engaged. Connections between different areas of the brain can vary in relation to the task an individual is completing as well as the individual's overall health. Not only might individual brain regions respond differently in the case of a chronically injured shoulder, but the patterns of communication between brain regions might also be altered to accommodate compensatory upper extremity behavior(5). However, these patterns of communication have not been explicitly evaluated in the context of chronic shoulder injury or dysfunction.

It is unknown how previously injured individuals vary in movement patterns in relation to their healthy counterparts. It is also of question of how the connectivity of the brain changes between the two groups. Very few studies have been found to relate the intertwining dynamics of neuromotor control and brain connectivity.

Purpose:

The purpose of this study was to compare neuromotor control of the shoulder between individuals with and without a previous shoulder injury. To achieve this, we used an upper extremity task to analyze kinematic performance of the shoulder complex and electroencephalography (EEG) to evaluate neural connectivity of the brain.

Hypothesis:

H1: We hypothesized that individuals with previous injury to the shoulder would have different kinematic patterns than individuals who had never experienced a shoulder injury. We expected participants with previously injured shoulders to have a less direct or evasive way of achieving their goal-oriented trajectory as compared to healthy participants.

H2: We hypothesized that participants with previous injury to the shoulder would have more diffuse patterns of brain connectivity during performance of the task, as compared to healthy participants.

Significance:

Previous studies have identified the effects of an upper extremity injury on either the nervous, neuromotor or behavioral system, but very few studies have addressed the effects with a combination of all three systems. Therefore, we plan to collect electroencephalogram data and motion capture data on both healthy and post injured individuals to identify the differences among populations.

Delimitations:

The following delimitations were identified for this study:

1. All participants had either healthy with no known injuries to the shoulder or with a known self-reported rotator cuff injury.
2. This study was limited to shoulder injuries that occurred on the right shoulder. Left shoulder injuries were excluded from the study.
3. All participants had to complete an injury questionnaire before the completion of the study.

Limitations:

The following limitations were identified for this study:

1. The analysis was limited by the accuracy of the motion capture, and electroencephalogram, as well as by the existing error associated with data collections using a combination of these systems.
2. The synchronization of movement with motion capture system and electroencephalogram may be limited using electromyographic system.
3. The analysis of the upper extremity movement was captured in a three-dimensional space which required a simplification of the human body into four segments.
4. The trial sequences among the three phases remained in the same for each participant.
5. Concussions or any other brain injury were not specified on the self-reported injury questionnaire.
6. The sample size was limited due to the Covid 19 pandemic.

Operational Definitions:

Central Nervous System (CNS): Controls most functions of the body and mind. It consists of two parts: the brain and the spinal cord.

Compensation: The counterbalancing of any defect of structure or function. A mental process that may be either conscious, or more frequently, an unconscious mechanism by which a person attempts to make up for real or imagined physical or psychological deficiencies.

Electroencephalography (EEG): An instrument that measures electrical potentials on the scalp and generates a record of the electrical activity of the brain.

Internal Model: A process that simulates the response of the neuromotor system in order to estimate the outcome of system disturbance.

Mechanoreceptors: A specialized sensory receptive structure found in the skin and articular, ligamentous, muscular, and tendinous tissue about a joint.

Neuromotor Control: coordination of muscular action with the nervous system. Requires precise proprioceptive input from the periphery, along with processing and input from the central nervous system

Proprioception: A sense gained primarily from input of sensory nerve terminals in muscles and tendons (muscle spindles) and the fibrous capsule of joints combined with input from the vestibular apparatus.

Chapter II: Review of Literature

Introduction

It is known that the human body is susceptible to injury as well as adaptable after an injury has occurred. Changes in motor performance along with adaptations post-injury have been well documented in the literature(4). These changes experienced during the execution of a task can be observed on the muscular, nervous and behavioral levels. Alterations of the shoulder complex and movement patterns could be a result of fatigue experienced in the shoulder caused by either a single event (acute) or the accumulation of repetitive stress (chronic)(6). Other deficits that range from atraumatic to traumatic injury also play a critical role in altering shoulder kinematics pathways. The question that then arises is how does injury affect the neuromotor system, nervous system, as well as lead to behavioral adaptations as a whole?

Alterations in Normal Shoulder Function

The rotator cuff is one of the most critical components of shoulder function. It is also important for the successful completion of tasks requiring the ability to position the arm and hand precisely in a space(7). The shoulder is dependent on coordinated, synchronous motion in all joints of the complex to be able to perform with its full mobility(8). The joint complex consists of three degrees of freedom (DOF) that directly correlate with the stability of the shoulder. Injury to this shoulder complex reduces the controlled manifold of the shoulder, reducing stability of the joint(9). Among reduction of stability, injury could be due to various types of tendonitis, impingement syndromes, recurrent subluxations and dislocations, as well as degenerative joint disease(10). As damage occurs to the shoulder, there is an alteration in the normal kinematic and neurological patterns that are typically carried out during movement. These changes in kinematics can affect the distribution of forces on the body, leading to worsening or reoccurring injuries(6).

The function of the shoulder complex relies on many intrinsic and extrinsic factors. Distractive forces seen on the glenohumeral joint during athletic events play a role in increasing tensile forces and static restraints in the shoulder. This distraction of the glenohumeral joint leads to instability as well as to mechanoreceptor damage. After damage occurs in the mechanoreceptors, kinesthetic awareness of the shoulder is inhibited, and the shoulder becomes dysfunctional(4). However, deficits and modifications experienced in upper-limb movements may occur in a variety of ways. One way could consist of alterations in kinematic patterns that may result in injury. A common injury that results in a modification of patterns would be where pain is present, and the body uses compensation to work around that pain. However, another way would be when alterations in the kinematic pattern is what causes the injury and injury due to muscle fatigue is an example of this. Smidt and Mcquade(10) reported that on a gross scale, the synchronicity of motion between the scapula and the Humerus is altered by fatigue of the muscles.

The shoulder complex is the most mobile region in the body and is dependent on the synchronous movement of all of its components to be fully mobile(8). Alterations in movement and behavior patterns, as well as the neural control of the shoulder that results in a shift in muscle activations, play a crucial role in the changing of normal shoulder function. These separate variables intertwine to alter and adapt movements performed by the shoulder. After normal shoulder function is compromised, these variables provide the shoulder with the capacity to complete the fullest extent of mobility as possible, even with limitations present.

Muscular System: A Role in Stability and Proprioceptive Feedback

The overall musculature of the shoulder complex is made up of more than 25 muscles(10). Though the amount of muscle support that the shoulder has surrounding it is great, the shoulder is still intrinsically very unstable. It relies on the integrity of noncontractile structures to provide static stability. Though, not all are included, these structures consist of the glenoid labrum, capsule, capsular ligaments and bony articulation(4). Dynamically, the shoulder is mainly constructed of the rotator cuff, deltoid, biceps brachii, teres major, latissimus dorsi, and pectoralis major muscles. These muscles provide important stabilizing support for the shoulder during movement(11). The dynamic contributions emerge from feedforward and feedback neuromotor control of the muscles crossing the joint. Behind the effectiveness of the dynamic restraints are the biomechanical and physical components of the joint, which contribute to range of motion, muscle strength and endurance(12).

The muscles of the rotator cuff demonstrate very strong direction-specific activity during task-oriented movement pertaining to isometric rotation in the unsupported mid-range abduction of the arm(13). This example of movement leaves the shoulder and arm vulnerable to various loads. The human body must be able to call on various groups of muscles to perform movements during a variety of loads and actions. Multiple command options are offered because of the range of muscle groups present and acting about the joints, and because of the many motor units comprising each muscle(14). However, it is not easy to separate the neuromotor control over motor activities and the neural commands that control the overall motor program. Lephart(12) gives an example of this by describing the execution of throwing a ball. While performing a throw, particular muscle activation sequences occur in the rotator cuff muscles to ensure optimal glenohumeral alignment and compression required for joint stability are provided. The individual throwing the ball is consciously and voluntarily deciding to perform this particular task. However, the involuntary muscles activating during this task are doing so unconsciously and synonymously with the voluntary muscle activations directly related to the characteristics of the task (e.g., speed, direction)(12). Therefore, it is evident that the conscious decision to perform an action and the voluntary and involuntary patterns of neuromotor activation are linked and driven by the central controller.

In relation to the status of the joint and its muscles, proprioceptive information of the shoulder comes into consideration, where there is a specialized form of somatosensation that focuses on the joint movement (e.g., kinesthesia) and joint position(15). Afferent proprioceptive feedback develops from information transmitted by mechanoreceptors to the central nervous system, relaying information about joint position and joint movement (16). Mechanoreceptors are responsible for this proprioceptive feedback causing neuromotor responses which are present in the musculature surrounding and controlling the joint(12,16,17) . Therefore, it is logical to assume that when muscles are injured, they begin to shift in their normal functioning roles, and that proprioceptive feedback is also affected. The function of the muscular system directly affects the feedback to the nervous system and vice versa.

Nervous System: Central Nervous System's Role in Task-Oriented Movement

The direct interaction between the static and dynamic components of functional stability is mediated by the sensorimotor system. According to Riemann and Lephart(12) the sensorimotor system encompasses all of the sensory, motor and cognitive integration and processing components of the CNS involved in maintaining functional joint stability. There have been significant advances in literature in understanding how the CNS adapts arm movements to changes in arm and environment dynamics. The nervous system has multiple ways of assessing its own motor performance. The CNS may adopt a variety of motor command sequences to perform the same task within a given environment(14). Integration of sensory input received from all parts of the body is largely considered to begin at the level of the spinal cord(15). Proprioceptive information from the shoulder and the overall upper limb are conveyed via the spinothalamic tracts and relayed to the somatosensory cortex where it is referred to a central body map allowing the conscious awareness of arm position and movement in space(18). The CNS can control the limbs by commanding an array of stable equilibrium positions aligned along the desired movement trajectory(19).

It is known in the literature that planning, initiation and control of upper extremity movement is a distributed process in the brain(20,21). It is also known that specific locations of activation are especially seen in the sensorimotor cortices of the brain(21–23). The results of a study performed by Nathan et. al(21) suggested that functional, goal-oriented movements like reaching and grasping elicit higher activation states when compared to nonfunctional reachingonly or grasping-only movements. It is stated that the amount of cognitive effort needed to perform the specific movement changes. The higher activation intensities and increased area of activation for goal-oriented reaching and grasping task could reflect the increased effort needed to perform the task as compared to the simple reaching-only or grasping-only task.

As previously shown, the brain varies in its activation levels depending on what movement is occurring as well as the location of the activation in the brain. The parietal lobe, located between the central sulcus and the Calcarine sulcus is highly involved with the processing of proprioceptive and visual information to provide the individual with spatial information of that particular environment or workspace (24). The cerebellum contains a functional organization that suggests that lateral portions of the cerebellum correspond to activity in the more distal parts of the body (e.g., hand, foot)(25). Accordingly, the cerebellum's ability to function in its role of coordinating specifically timed movements, continuous comparisons between movements of different joints in the upper extremity would be needed to assure continued accuracy(26).

According to Hork and Rymer(27) kinematic errors are transduced by both vision of the moving limb and by muscle spindle afferents which appear to signal a combination of both muscle fiber length and velocity. These errors can be a product of a simple error performed by an individual or from an injury resulting in an error. Proprioceptive deficiencies, which exist in individuals with functional deafferentation, create major deficits in movement control(28,29), can result in increased movement variability, the inability to maintain stable hand postures without visual guidance, and a reduced capacity to detect and correct motion errors based on limb movement information after completing a task(30). However, performance of that task requires more than just the central nervous system to initiate and successfully execute the motor command, it is dependent on a compilation of systems working as one.

Neuromotor Control

The nervous system in combination with the muscular system provides the human body with its ability of motor control. The coordination of muscular action with the nervous system is known as neuromotor control. It requires precise proprioceptive input from the periphery, processing and input from the central nervous system (including learned or trained movements). The intertwining of systems involves timing of muscle recruitment as well as muscle contraction states(31). Neuromotor control makes reference to the nervous system's control over muscle activation and its capacity for task performance(12). The role that neuromotor control plays is a critical component in the stability of the shoulder joint. In the perspective of joint stability, neuromotor control can be explained as the unconscious activation of dynamic constraints occurring in preparation for and in response to joint motion. It also has the ability to handle loading for the purpose of maintaining and restoring functional joint stability(12).

The architecture and the high mobility of the shoulder complex predispose nerves to various dynamic or static compressive and/or traction injuries(32). Deficits like fatigue and injury can affect the function of the entire shoulder including both the nervous and muscular systems. This loss in function can stem all the way down from the shoulder's proprioceptive feedback to the CNS. In a healthy normal shoulder, afferent proprioceptive feedback that is integrated in the CNS evokes efferent neuromotor responses as both spinal reflexes and preprogrammed responses significant to functional stability of the should joint complex(11). However, because fatigue inhibits proprioceptive feedback from the shoulder to the CNS, the neuromotor responses may be hindered, leading to instability of the joint and eventually joint injury. If an individual's ability to recognize joint position, especially in positions of susceptibility, is obstructed, they may be prone to injury due to increased mechanical stress placed on both static and dynamic structures responsible for joint stability(11).

Researchers have found that, with training, activation of the appropriate musculature gradually shifted from a delayed error feedback response to a predictive feedforward response(33). This is important in the formation of internal models that help to better predict and control outcome of movements. Restoration of functional stability in the shoulder requires attention from both stabilizing structures that are compromised and the neuromotor responses that are vital to joint stability through a functional rehabilitation program(11). Thus, it is important to note that stabilization of the shoulder is widely dependent on both the muscular and nervous systems to be able to function to its full capacity.

Neural Differences to Changes in Kinematic Patterns

A basic understanding of motor control implies an understanding of what is being controlled and how the control process is being organized in the central nervous system. Normal motor control suggests the ability of the central nervous system to use current and previous information to coordinate effective and efficient movements by transforming neural energy into kinetic energy(34). The cerebellum is an essential part of the neural network involved in adapting goal-directed arm movements (38). When a sensory error is made due to varying problems (i.e., fatigue, habit or injury) an increase in brain activity can be observed(35). A critical feature of adaptation is that it allows individuals to alter their motor commands based on errors from prior movements. Differences in brain activity levels can be observed in many regions of the brain ranging from the parietal lobe to premotor areas, depending on what task or error that may have been performed.

Connectivity between different brain regions is inferred from temporal associations between spatially remote neurophysiological events. One measure of connectivity is a correlation between two simultaneously recorded signals in the frequency domain, called coherence, which can be assessed in humans using EEG(36). Connections between different areas of the brain regions can vary depending on the task an individual may be completing and the healthiness of the individual. Not only might individual brain regions respond differentially in the case of a chronic shoulder injury, but the patterns of communication between brain regions might also be altered to accommodate compensatory upper extremity behavior. It is well known that the parietal and premotor areas share dense connections that facilitate computations related to upper extremity motor function(5) and that these connections are predominantly in the hemisphere contralateral to the moving limb. However, these patterns of communication have not been explicitly evaluated in the context of chronic shoulder injury or dysfunction.

Behavioral Adaptations

Humans learn and adapt from the time they are born well into their adulthood. Throughout a lifetime there are many stages of learning, and each stage happens at different rates and in some cases overlap one another. In the Gentile model(37), the initial stages of learning are defined as the basic movement pattern needed to achieve a goal, as well as being able to identify components of the environment that are important to that task. During this stage, the individuals are encouraged to go through trial and error while actively testing their abilities. The human mind learns and corrects itself through this trial and error process.

The overall behavioral learning system shapes an individual's movement and brain patterns with this adapting process. Motor skill learning consists of two learning processes, explicit and implicit learning(37). When considering explicit learning, the individual concentrates on the attainment of a singular goal, just like in the initial stages of learning(38). The goal-oriented movement is attempted for early success, the performer then is able to develop a sense of a "map" between their body and the conditions of the environment(38). Whenever kinematic movement patterns can be consciously adapted by the performer it is known to be regulated by explicit processes(37). Implicit learning is a form of unconscious, incidental and procedural knowledge acquisition that occur over a gradual period of time(39). An individual will unconsciously merge successive movements, couple simultaneous components and regulate active forces inherent in a particular task(37).

What a system can and cannot learn, the magnitude of generalization, and rate of learning gives researchers clues to the underlying performance architecture(40). The perceptual framework interprets the performance of motor tasks. When initially presented with a request to perform an entirely new movement, individuals look for relationships between previously executed movements and interpolate a reasonable approximation. As individuals practice the movement, they are able to store newly tried motor programs using a new representation based on apparent outcome(41). This ability to store new motor programs when presented with a new or different movement is an example of adaptation.

The concept of adaptation allows not for cancellation of effects in a novel environment, but for maximization of performance in that environment while predicting a re-optimized trajectory(42). In other words, instead of cancelling out a kinematic pattern that may cause pain due to injury, the pattern of the movement is re-routed to cause less pain and/or reduce worsening of the injury. So instead of adaptation being a cancellation process, it may calibrate the brain's prediction of how the body will move and consider the costs correlated with new and different task demands(43). After injury, adaptation is inherently important for rehabilitation by making movements flexible, but it can also be used to ascertain whether some individuals can generate a more normal motor pattern(43,44)**.**

The question that then arises is does adaptation recalibrate the depiction of movement patterns in the brain? According to Kluzik et. al(44), the results suggested that gradual changes during training conditions resulted in smaller trial-to-trial movement errors and are more likely to lead to changes in neural representations of the body's dynamics, with a greater generalization of adaptation across varying conditions(43,44). As individuals practice a novel task, the errors will decrease over time. Repeated adaptation can lead to learning of a new, more permanent motor calibration. Though less understood, this type of learning is likely to be an important method for making long-term improvements in individuals' movement patterns(43). Once a successful pattern is established, the individual is able to distinguish between regulatory (directly influences movement) and non-regulatory (does not directly influence features of the environment) movement properties, and the next stage of learning begins(38).

Conclusion

The human body is known to be an adaptable system, made up of a multitude of structures that work together to create a coordinated functional unit. This system relies on this constant coordination of movements to function properly in its full range of mobility. However, the balance of the body can be easily interrupted by the presence of an injury or a deficit of any type.

Adaptation plays a critical role in assisting the body with coping with changes due to injury. Changes in motor performance along with adaptations after injury have been documented in many ways throughout the literature. The nervous system along with the muscular system have been presented as being crucial components for both functional and mechanical stability. It has also been shown that behavioral adaptation after injury leads to neural adaptation. This phenomenon leads to the hypothesis that when individuals experience a shoulder injury, the way they go about reaching their goal trajectory varies from healthy individuals who have never experienced an injury. The first hypothesis leads to the second hypothesis, which represents the behavioral changes through neural changes in the brain. The individuals who have experienced an injury to the shoulder are hypothesized to have varied activation patterns in the brain during preprocessing of a movement and during the movement itself.

Understanding the neural correlates in the brain that pertains to upper extremity neuromotor control and how they relate to the control of goal-oriented tasks would be beneficial in the development of therapeutic paradigms. This knowledge may also provide insight regarding the mechanisms that facilitate cortical plasticity(21). In conclusion, understanding the innerworkings of each individual system that plays a role in the shoulder's ability to function correctly is critical in knowing how to best reoptimize an individual's kinematics after injury occurs. Each system (nervous, neuromotor and behavioral) affects the shoulder in their own individual ways. However, knowing how they collectively affect the shoulder is important in understanding the overall ability of the shoulder to readapt after injury.

Chapter III: Materials and Methods

Introduction of Study Design:

The purpose of this study was to measure the effects of previous injury on the neuromotor control of the shoulder. Participants were categorized into healthy and injured shoulders groups for this experiment. Following the completion of a questionnaire describing injury or non-injury experienced by the individual, a maximal strength test of the shoulder was performed and used to adjust the load on the participant's wrist during the weighted trial of the study. Full range of motion was observed during three separate trials, and differences between the groups were measured.

Participants:

Ten participants in total were recruited to participate in this study. The participants consisted of two individuals with a self-reported previous injury to the right shoulder experienced between 6 – 12 months prior to participation in the study, and eight individuals with no previous injury to the right shoulder. Participants were a mean age of 22 years old ± 3 years. Participants were right hand dominant; as determined using the Edinburgh Handedness Inventory (See Appendix A). Participants who had previous injuries to their nondominant left shoulder were excluded from the study. Participants were informed of potential risks associated with the EEG and motion capture, and an informed consent was obtained before any measurements were taken (See Appendix B). The protocol and consent form were approved by the East Carolina University and Medical Center Institutional Review Board (UMCIRB).

Equipment and Measurement Protocol:

Maximal Strength Testing

For the maximal strength protocol, participants extended their arm forward as they grasped a handle that was connected to a force transducer (BioPac Systems Inc., Goleta, CA). The force transducer was attached to a platform on which the participant was standing. Participants performed an isometric maximal velocity contraction of shoulder flexion in the sagittal plane, pulling upwards on the handle with their elbow at 180 degrees. They were asked to pull up with maximal effort for three seconds and then rest and they performed this contraction three times. Their maximum strength was calculated from the peak force in which they produced from the three sets. The force measurements calculated through this maximal strength protocol were used to normalize the percentage (i.e., 10 and 15% of max weight) used across the participants.

Electroencephalogram

For EEG preparation, participants were seated in a chair and any hair care products were removed from the hair with an alcohol-saturated cotton pad. The forehead was prepared by wiping the area with a cotton pad and a solution of pumice and Vitamin E, thereby removing any residual oil and dirt from the skin. Then, participants were fitted with a 64-channel EEG cap (Compumedics Neuroscan, Charlotte, NC) to record neural activity using SynampsRT (Compumedics Neuroscan, Charlotte, NC). Once the cap was in place and properly aligned, the scalp under each electrode was prepared by first gently abrading the skin using the wooden end of a standard cotton swab with pumice and Vitamin E to reduce impedance to the electrode, and then inserting a conductive gel with a 16-gauge blunt needle.

Vicon Nexus

For Motion capture preparation, participants were seated in a chair surrounded by a Vicon Nexus system (Vicon, Oxford, UK) including six Vicon Bonita cameras and one Vicon Vero camera collecting at 120 frames per camera per second. Participants were asked to remove any clothing garments that were located on the arms for a more accurate application and reading of the trackers. The Upper Limb Model(45) marker set by Nexus was used in the placement of infrared markers during static calibration. In addition to static calibration markers, rigid body tracking markers were placed on various sites of an individual's upper extremity including hand, forearm, upper arm, and thorax (Figures 1 and 2). Elastic bandages along with Velcro on the marker's skeleton were used to attach markers to the appropriate sites on the participant.

Figure 1: Represents the posterior view of participant set-up and marker placement.

Figure 2: Represents the lateral view of participant set-up and marker placement.

Trials: Sequence Set-up

Each participant performed in conditions designed to test upper extremity motor function. During the first portion of the study, the participant performed a simple static calibration trial, where the individual was in a "motorcycle pose" and hold that position for 3 seconds. The second portion of the study consisted of four trials with each trial consisting of three sequences with a fifteen second rest in between each sequence. The sequences performed evaluated a range of motion as the arm is in extension. A target symbol moved in eighteen different directions to nine boxes. The hand segment was recorded using the “Center of Mass Position” to track where the hand was in space. Brain activity, difficulty level measured by RPE scores between each trial, along with neural and neuromotor activations were recorded. There are four conditions that were presented to the participant (e.g., free of resistance, 10% of maximum resistance, 15% of maximum resistance, and then free of resistance). The first condition consisted of being free of resistance. No additional load was added to the participant’s upper extremity during this condition. The participant went through the simulation sequence one time before the second condition begins.

The second condition that the participant underwent used resistance. The resistant condition consisted of cuff weights (AliMed Inc, Dedham, MA) attached to the participant’s wrist. The load of resistance ranged between 1.36kg-6.8kg. A maximum testing protocol performed before the beginning of the study to get a participant specific maximum weight measurement. A percentage (i.e., 10% and 15%) of the individual’s maximum weight resistance was used as the load for the resistance trials. As before, brain activity, difficulty level, and neuromotor activations were recorded during the trials. Each of participant performed each trial with a resistance as well as no resistance. The arrangement of all four trials remained the same; Trial one consisted of having no weight applied to the participant’s wrist, followed by trial two having 10% of the participant’s maximum weight being applied, then trial three with 15% of their maximum weight, lastly trial four with no weight.

Hand pathway coordinates were taken from the center position of the hand and were recorded from each frame and followed throw space. Displacement of the trunk was calculated by the max distance in which the trunk moved forward/backward as well as laterally, both subtracted from the minimum distance moved in both directions. Elbow angular position was also calculated by taking the max degree in which the elbow moved subtracted from the minimum degree.

Rate of Perceived Exertion/Questionnaire

The rate of perceived exertion (RPE) was used to evaluate the level of difficulty or easiness of the task between each trial. RPE assisted in understanding what the participants experienced during each trial (See Appendix C). The scale also provided information on if the task was too demanding for the participant to complete. Along with RPE, an individual's personal history with sport or injury may have some impact on the effect of movement and neuromotor compensation. As such, prior to leaving the laboratory, participants completed a questionnaire related to their self-reported injury history. This questionnaire disclosed no sensitive information (See Appendix D).

Data Analysis

The trajectories of the arm in the horizontal plane were measured using motion capture. Targets were presented to individuals and the infrared markers located on the participants were measured in a three-dimensional space. In general, a measure was selected to quantify the performance of the movements executed by each participant, and to assess differences in kinematics between group and trials. These measures were taken over the set of fifty-four movements that comprised each trial. Motion capture data were analyzed using Visual 3D (V3D) software (C-Motion Inc., Germantown, MD). Displacement analysis was calculated for trunk displacement, Elbow Flexion and right-hand pathways using V3D and Excel (Microsoft Inc., Redmond, WA). The motion capture marker set was filtered through a 4_{th} bidirectional order low pass Butterworth filter with a cutoff frequency of 6Hz. Specifically, for EEG, the cross spectrum was derived from the frequency domain and calculated for all channel pairs. The cross spectrum was then used to calculate corrected imaginary coherence(46) between all channel pairs to estimate patterns of interregional neural communication. Nonparametric permutation statistics were used to determine statistical differences ($p < 0.05$, 1000 permutations). No assumption could have been made about the underlying distribution of the data, thus a nonparametric permutation statistical approach, based on the FieldTrip toolbox(47), was taken. At the individual participant level, corrected imaginary coherence data were used to create a null statistical distribution, or a distribution that would be true if there was no dependence on specific channel pairs in the actual distribution of connectivity estimates. This was accomplished by randomly permuting electrode labels through 1000 permutations. A Fisher's Z-statistic map was then calculated as:

Zmap = (true connectivity – permuted connectivity mean) / std (permuted connectivity)

and was used to threshold the true connectivity estimates.

Chapter IV: Results

During the extent of the data collection, 5 of the 10 participants successfully completed the motion capture as well as the EEG portions of the study. All participants successfully completed the study using EEG. There were significant differences observed in the EEG data (Alpha band, Low and High Beta bands, and Theta band) among participant groups. In the motor control literature, Alpha, Beta and Theta frequency bands are most commonly used in the study of neural activations(48,49). Kinematic data that was observed by analyzing differences among healthy and post-injured individuals: thorax displacement, elbow angular position and right-hand trajectory. The third sequence of each trial was used to analyze each kinematic component.

EEG Data

Alpha Band (8 to 12 Hz)

The results of the EEG data showed significant differences in alpha wave connectivity between participant groups (i.e., healthy and post-injured). An increase in the distribution of alpha band connectivity was observed from healthy participants (Figure 3) to post-injured participants (Figure 4). Trial 4 reveals an even greater difference in alpha band connectivity between healthy participants (Figure 5) and post-injured participants (Figure 4). It was observed that from Trial 1 to Trial 4 (Figures 4 and 5) post-injured participants' alpha band connectivity deviated from dominating in the posterior portion of the left hemisphere to spreading across into other brain regions. The healthy participants' alpha band connectivity remained remotely unchanged from trial 1 to trial 4, shifting marginally to other regions.

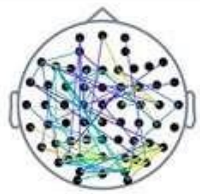

Figure 3.
Represents the cross spectral Alpha waves observed during **Trial 1** of the **Healthy** Participants

Figure 4.
Represents the cross spectral Alpha waves observed during **Trial 1** of the **Post-injured** Participants

Figure 5.
Represents the cross spectral Alpha waves observed during **Trial 4** of the **Healthy** Participants

Figure 6.
Represents the cross spectral Alpha waves observed during **Trial 4** of the **Post-injured** Participants

Beta Bands (12 to 38 Hz)

Significant differences were observed in the connectivity for both low (12-15Hz) and high (22-38Hz) beta bands between the participant groups. As seen in Figure 7, the low beta analysis revealed that post-injured participants demonstrated a dense connectivity centrally of the brain throughout the extent of the trials whereas the healthy participants after trial 1 began to transfer the area of the densest connectivity to the parietal region of the brain. The connectivity of the post-injured participants shifted from a proportionally balanced volume of horizontal, vertical and diagonal connections (Trials 1-3) to predominantly vertical connections between frontal and parietal lobes (Trial 4). Figure 6 demonstrates the significant differences observed among high beta band between participant groups. The high beta analysis revealed that post-injured participants possessed a greater distribution of significant connectivity during the entirety of the four trials in comparison to the healthy participants. The results showed significant connectivity concentrating more in the left frontal hemisphere for healthy participants, whereas post-injured participants varied across the cerebral cortex.

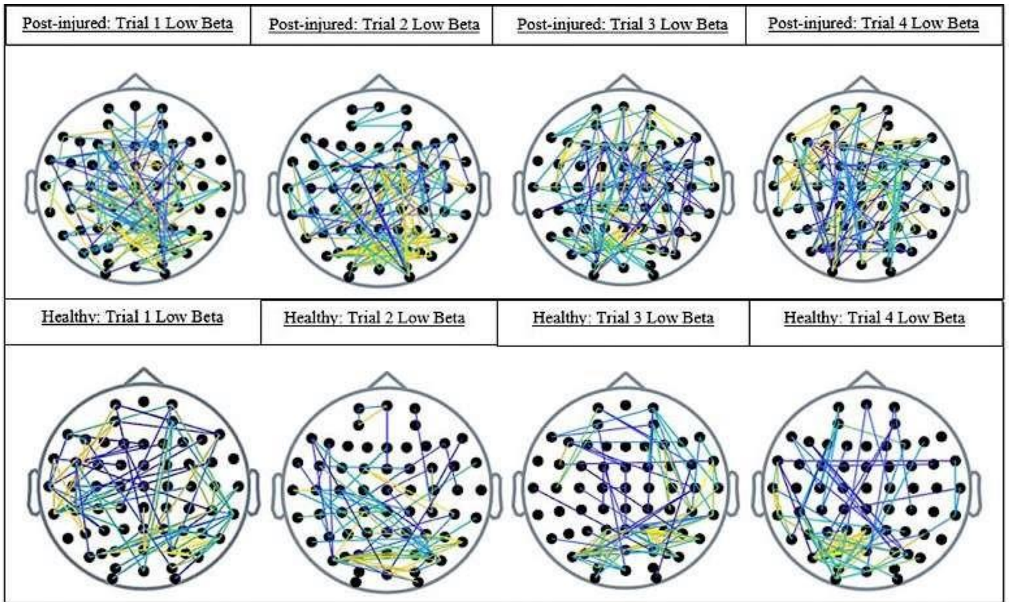

Figure 7. Represents the cross-spectrum connectivity of Low Beta waves ($p \leq .05$) observed for post-injured and healthy participants from Trial 1 to Trial 4.

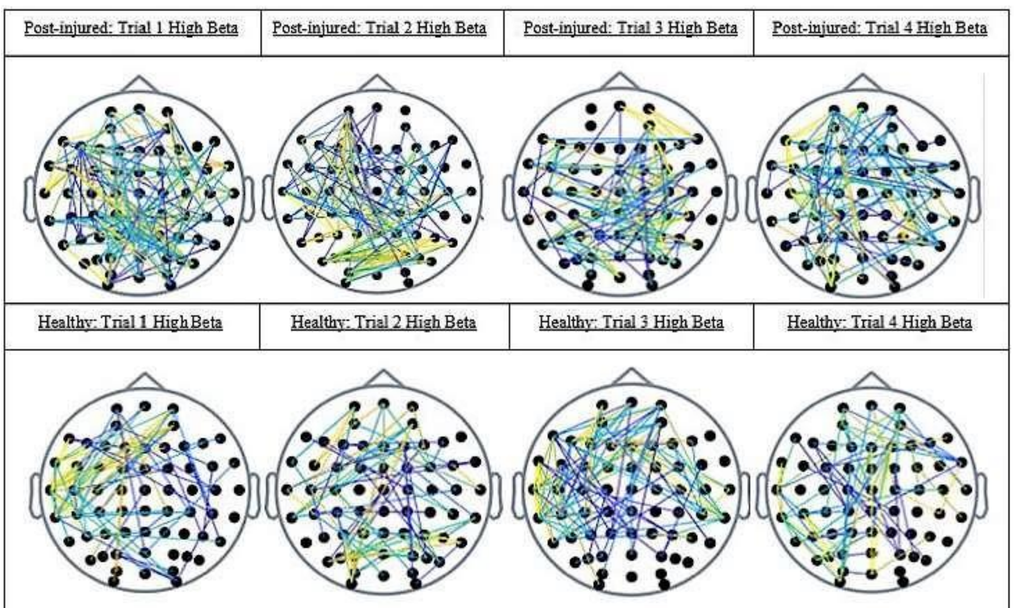

Figure 8. Represents the cross-spectrum connectivity of High Beta waves ($p \leq .05$) observed for post-injured and healthy participants from Trial 1 to Trial 4.

Theta Band (3 to 8 Hz)

Significant differences in theta band connectivity between participant groups were observed during the length of all four trials (Figure 9). Considerable differences were shown in the central most area of the brain. Post-injured participants' connectivity remained centrally located for all trials. The healthy participants' connectivity throughout the trials was considerably scattered to multiple brain regions, with a shift to the left posterior area of the brain in Trial 3. The quantity of connections observed is greatly increased in the post-injured theta band analysis in comparison to the healthy participants.

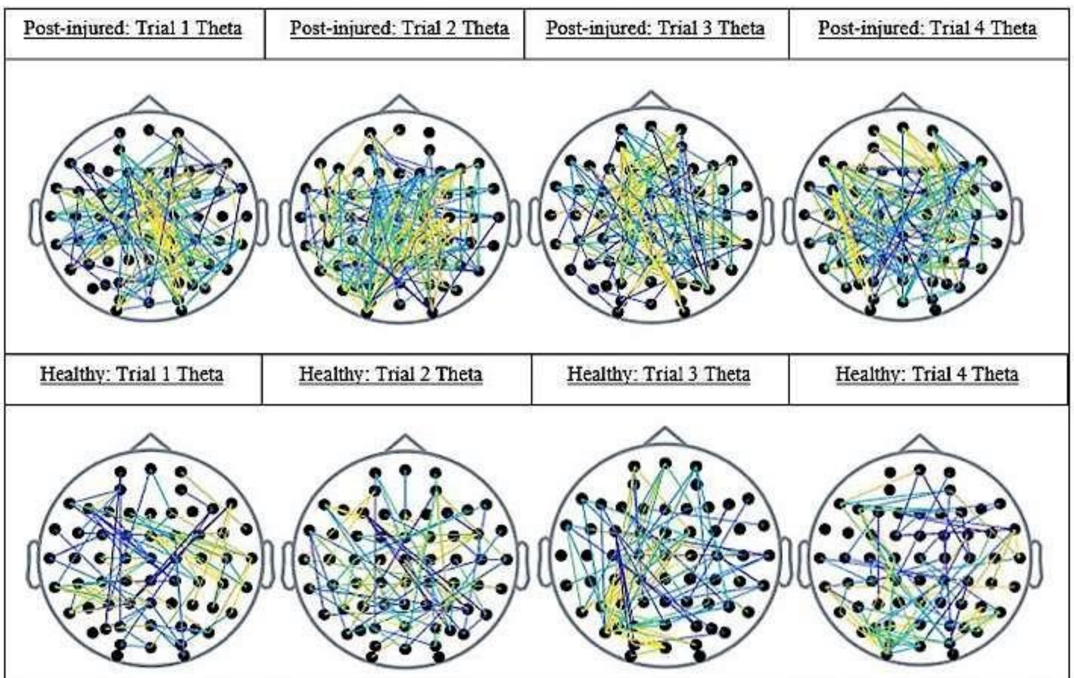

Figure 9. Represents the cross-spectrum connectivity of Theta waves ($p \leq .05$) observed for post-injured and healthy participants from Trial 1 to Trial 4.

Kinematic Data

Right Hand Trajectory Pathways

The motion capture showed slight differences in the right-hand pathways between participant groups. Similar variations of pathways were executed by both participant groups. The average pathway that the right hand of the healthy participants remained closely to a straight line to the nine targets (Figures 10-13). A similar trend was observed with the post-injured participants. Each trial displayed a similar resemblance. Trial 3 (15% of max weight), showed the biggest difference among participant groups (Figure 12).

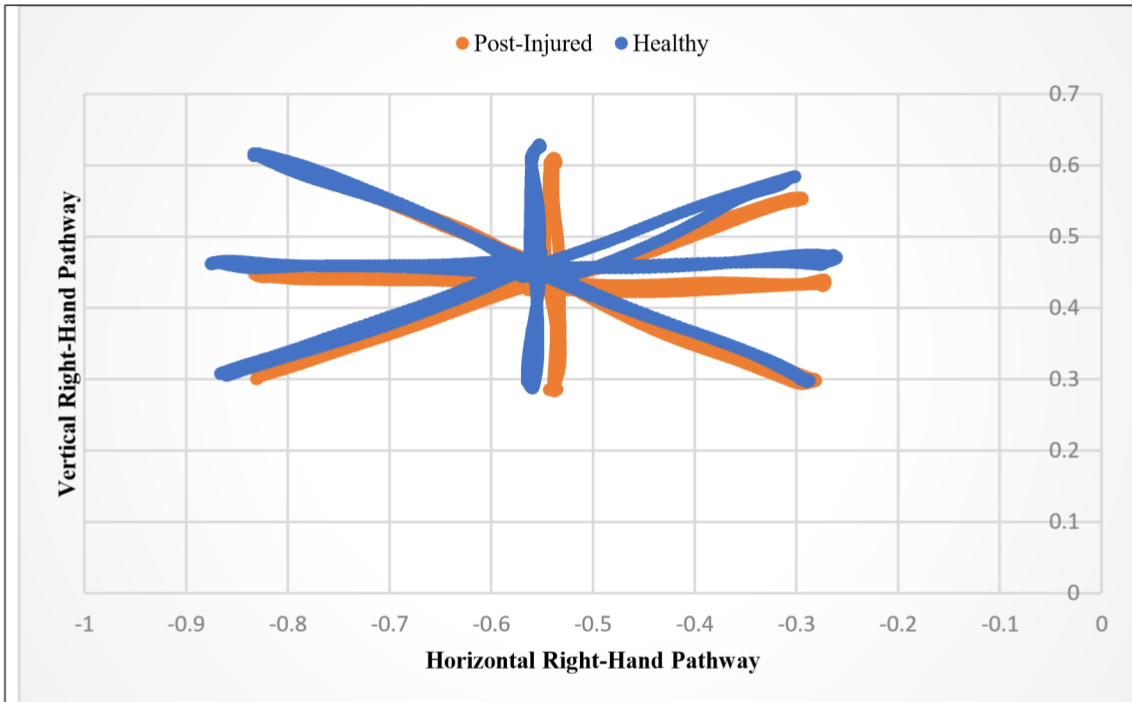

Figure 10. Represents the right-hand pathways executed by both participant groups to the nine targets during the third sequence of Trial 1 (No Resistance).

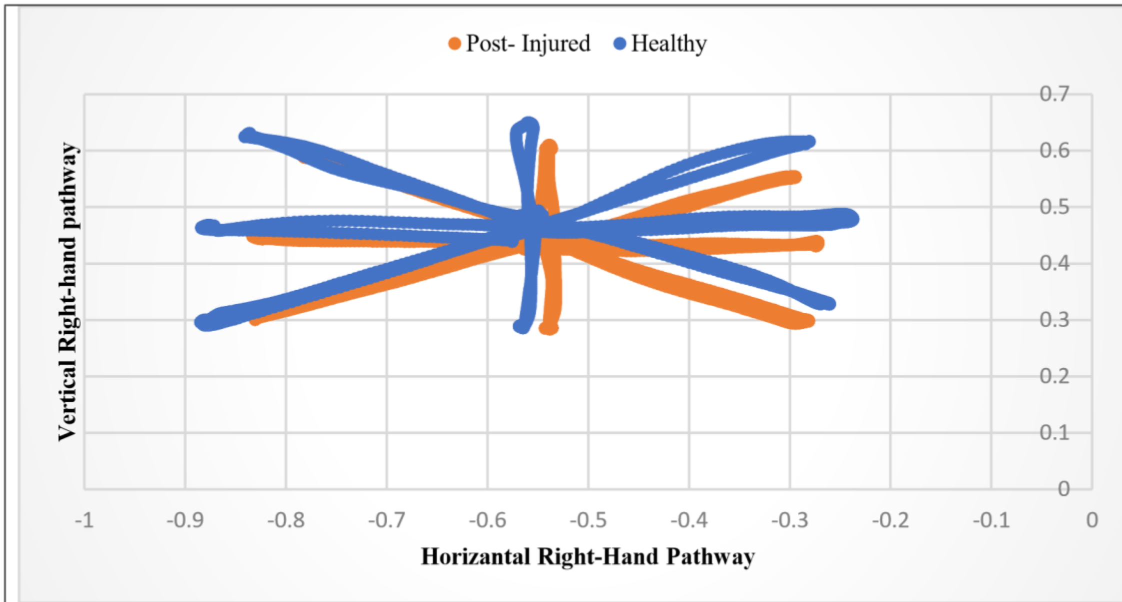

Figure 11. Represents the right-hand pathways to the nine targets executed by healthy and post-injured participants during the third sequence of Trial 2 (10% of Max).

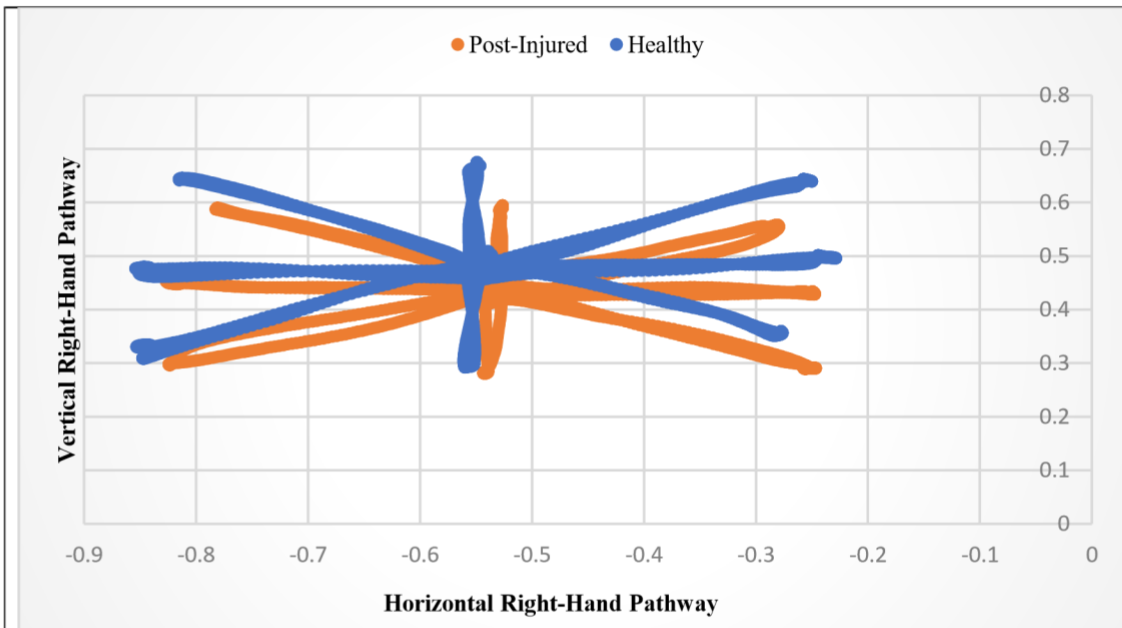

Figure 12. Represents the right-hand pathways to the nine targets executed by healthy and post-injured participants during the third sequence of Trial 3 (15% of max).

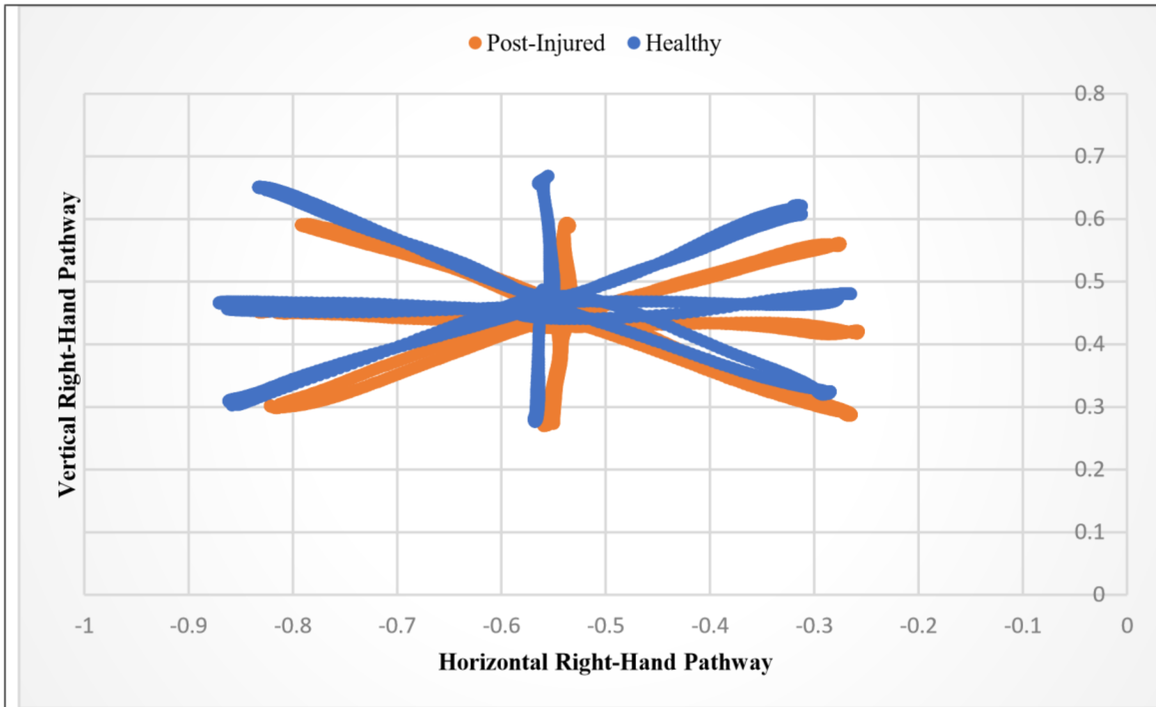

Figure 13. Represents the right-hand pathways to the nine targets executed by healthy and post-injured participants during the third sequence of Trial 4 (No Resistance).

Trunk Displacement

Differences in trunk displacement were observed in both lateral and anterior/posterior movement during the course of the four trials. The results of the lateral displacement (Figure 14) followed a similar pattern between participant groups, with post-injured showing a slightly higher change in lateral displacement. Anterior/Posterior displacement provided a greater difference among participant groups (Figure 15). Healthy participants displayed a horizontal linear relationship between trials. Post-injured participants had a steep linear increase from Trial 1 to Trial 3 (no resistance to 15% of max resistance), with a decrease in trunk anterior/posterior displacement during Trial 4 (no resistance).

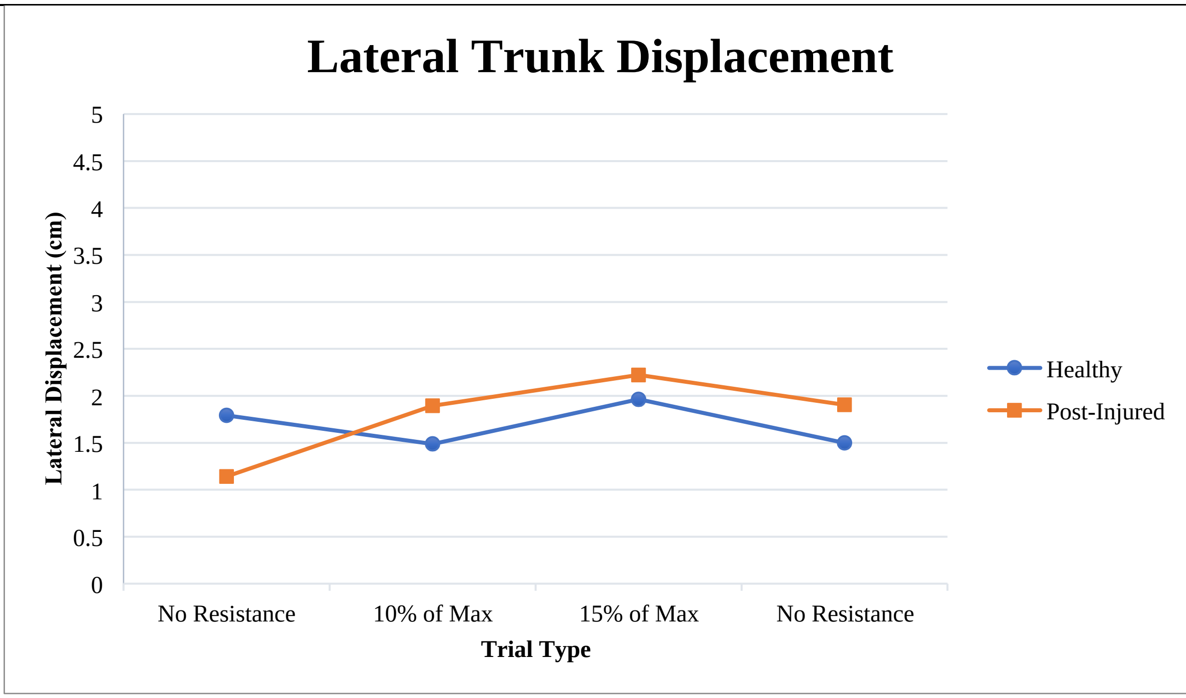

Figure 14. Represents the lateral displacement (cm) of the trunk during the third sequence of each of the four trials.

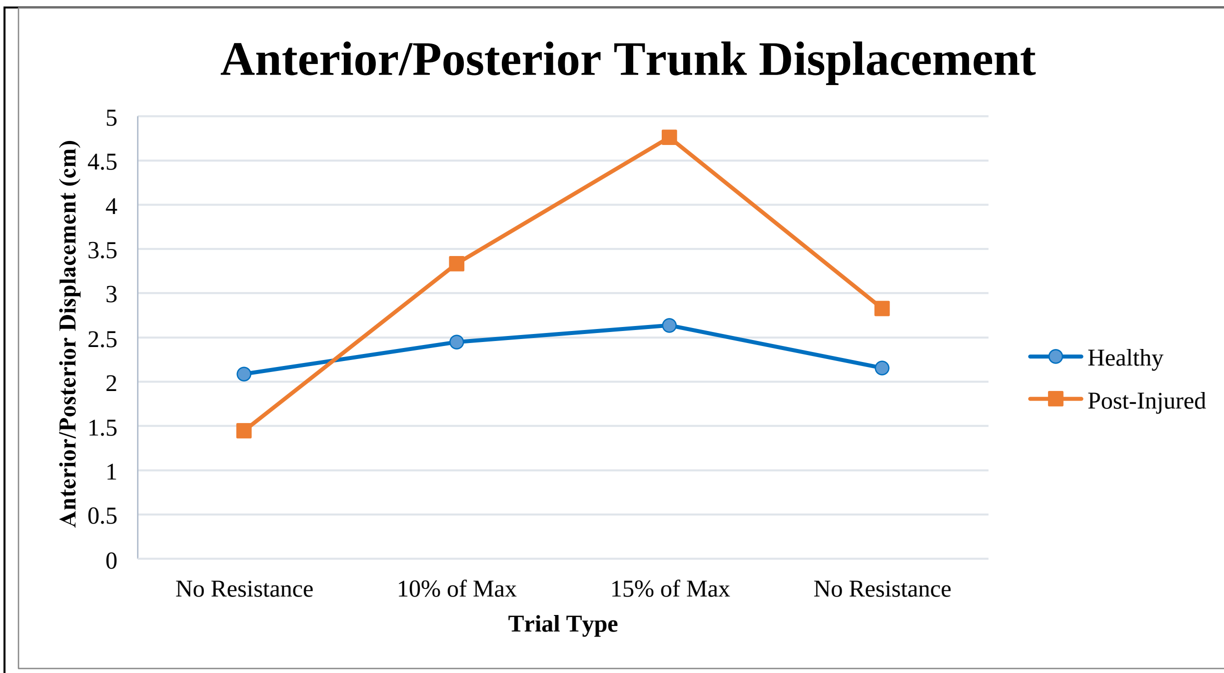

Figure 15. Represents the anterior and posterior displacement (cm) of the trunk during the third sequence of each of the four trials.

Elbow Flexion

Differences in elbow flexion were observed between participant groups as well as trials. Slight changes were seen in the degrees of the elbow between groups as seen in Figure 16. Both groups decreased in angular position from Trial 1 to Trial 2 (No resistance to 10% of max). This decrease was followed by post-injured group increasing in angular position during Trial 3 (15% of max), from approximately 10º to approximately 14º. While the healthy group remained remotely unchanged from Trial 2 to Trial 3. Trial 4 (no resistance), showed a mirror effect to Trial 1 with a slight decrease in degrees change of the elbow position.

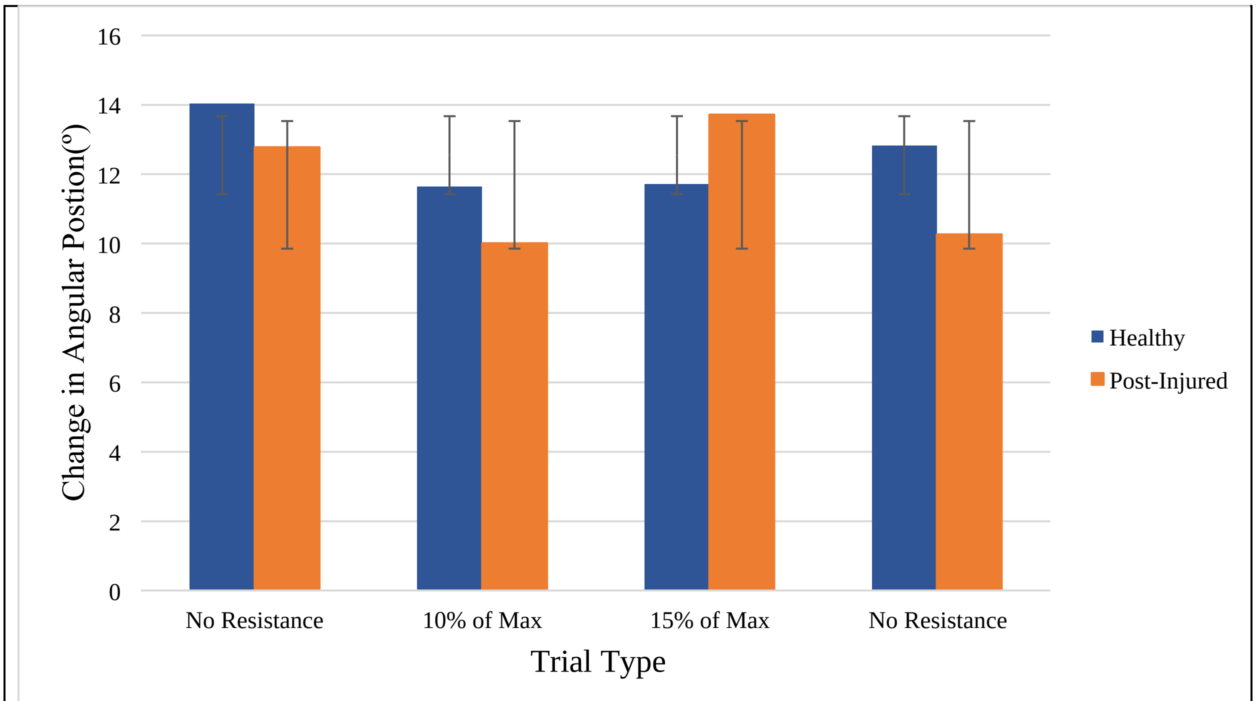

Figure 16. Represents the differences in elbow angle (with standard deviation) observed during the four trials by healthy and post-injured participants

Borg CR-10 Rate of Perceived Exertion

Healthy participants verbally reported lower RPE rating averaging about 1.82 (scoring: easy) across the four trials. Post-injured participants reported a higher RPE rating averaging approximately 4.38 (scoring: sort of hard-hard) . The biggest increase in scoring for both groups was seen during Trial 3, as observed in Figure 17.

	Participant Type	Average Rating	Borg CR-10 Scale Descriptor
Trial 1	Healthy	1.13	Really Easy
	Post- injured	2.5	Easy-Moderate
Trial 2	Healthy	1.88	Easy
	Post- injured	5	Hard
Trial 3	Healthy	2.75	Moderate
	Post- injured	6.5	Really Hard
Trial 4	Healthy	1.5	Really Easy-Easy
	Post- injured	3.5	Moderate-Somewhat Hard

Figure 17. Represents the averaged perceived exertion scores of both participant groups.

Chapter V: Discussion

This study compared the differential effects of past shoulder injury on movement patterns and brainwave connectivity during a repetitive reaching target task. The relative mechanics of the right upper extremity limb of both groups did not differ, though variation in trunk displacement was evident. The higher ratings of perceived exertion as the number of trials increased, demonstrated the task getting harder as weight was added to the individuals' arms. It was observed that with a higher force demanding task, cross-spectral connectivity in the brain significantly increased for individuals with a pervious injury.

Changes in Kinematics

The hypothesis that individuals with previous injury to the shoulder would have different kinematic patterns than individuals who had never experienced a shoulder injury was supported by this study. The second part of the hypothesis which stated that participants with previously injured shoulders would have a less direct or evasive way of achieving their goal-oriented trajectory was partially supported. Anterior and posterior trunk displacement was greater for previously injured individuals, however lateral trunk displacement, elbow angular position and hand pathways were similar with healthy individuals.

Trunk displacement, both anterior/posterior and lateral, and elbow angular position were analyzed due to their role in upper extremity mobility. It was observed that both groups demonstrated an increase in trunk lean in relation to a slight decrease in change of elbow angular position. This observation is consistent with previous studies which found an increase in elbow angle, and trunk lean as fatigue set in as a compensatory factor(6). Though fatigue was not necessarily a measured component in this study, perceived exertion and difficulty level was. It may be said that as an individual completed the trials, there was an increase in difficulty for both participant groups. Therefore, changes in mechanics may have been used for compensation in increasing weight and difficulty. One noticeable relationship among both kinematic components mentioned (specifically during 15% of max resistance) was that during the trials with a heavier weight, an individual may increase greatly in trunk movement, with also a slight increase in elbow angular position as well. Though the elbow angular position fluctuated among all trials without an obvious trend between participant groups.

Hand trajectory pathways remained relatively the same among participant groups, though it can be observed that as load was added pathways shortened for the post-injured individuals. Throughout the trials, trunk displacement was evident as well as change in elbow angular position. Overall, all participants had the same nine targets in which the right hand was intended to follow. Compensation was not seen as much in the pathways executed, but more in the varying mechanics more proximal to the shoulder (i.e., trunk displacement and elbow angular position).

Changes in Brain Connectivity

The hypothesis that participants with previous injury to the shoulder would have more diffuse patterns of brain connectivity during performance of the task, as compared to healthy participants was supported. Each frequency band (Alpha, Beta, and Theta bands) analyzed provided significant differences between the participant groups. The most significant evidence being observed in Alpha band and Theta band activity.

As stated in previous studies, the alpha band is observed primarily in posterior regions of the brain, as well as laterally. Alpha activity is also higher in amplitude on an individual's dominant side(50). It is known that the alpha band takes an important role of motor activity and motor imagery as well as visual tasks(51). It was observed that from the first trial of no resistance to the fourth trial of no resistance, post-injured participants' demonstrated an alpha band connectivity that deviated from dominating in the posterior portion of the left hemisphere to spreading across into other brain regions. This may suggest that as an individual's effort begins to heighten, the brain must call on more areas in order to complete the same task at the same level of difficulty. This observation is consistent with previous studies(52, 53). The healthy participants' alpha band connectivity remained relatively unchanged from trial 1 to trial 4, shifting slightly to other regions. It can be implied that unlike post-injured individuals, the healthy group did not need to rely on a greater distribution of brain areas in order to perform the same task.

High Beta activity was evaluated for its role in distal limb control, especially over the sensorimotor strip, and the low beta representation has been shown in previous studies to demonstrate the clearest distinctions between the limbs over widespread brain areas, particularly the lateral premotor cortex (54). Beta bands are typically seen during active thinking, focus as well as while being highly alert, and, like alpha band activity, beta bands play an important role of motor activity and motor imagery(50, 51). The high beta analysis revealed that post-injured participants possessed a greater distribution of significant connectivity during the entirety of the four trials in comparison to the healthy participants. The results of high beta activity suggest that despite the difficulty level of the task, the task itself required greater high beta activity for post-injured individuals. Healthy participants had greater connectivity that concentrated more in the left frontal hemisphere, which is related to planning and concentration. Whereas, post-injured participants varied in increased connectivity across the cerebral cortex relying on several regions of the brain. The greater high beta activity may be associated with higher complexed thoughts, integration of new experiences and higher anxiety(50). The low beta analysis revealed that post-injured participants demonstrated a dense connectivity centrally throughout the extent of the trials, which may be associated with the motor cortex region. According to previous literature, low beta activity is associated mostly with quiet, focused, introverted concentration(55). It can be assumed that post-injured individuals required higher concentration across trials to complete the same tasks as their healthy counterparts. Overall, higher levels of both high and low beta activity were evident in the execution of the goal oriented task for the post-injured individuals.

Theta band activity was observed for its role in carrying substantial information about movement initiation and execution(56). Theta band also is responsible for spatial recognition and cognitive as well as visual tasks. Theta bands activations is thought to originate from the anterior cingulate and mainly appears when one is performing a task requiring focused concentration, and its amplitude increases with the task load(57). Considerable differences in theta band connectivity between participant groups were shown in the central regions of the brain. Post-injured participants' connectivity remained centrally located and dense for all trials, especially the third trial. These results are consistent with previous literature, where increased difficulty in a task resulted in increase in theta band activity(58). Unlike the post-injured individuals, the healthy participants' connectivity throughout the trials was considerably distributed across multiple brain regions.

Changes in Borg C-10 Rate of Perceived Exertion

Higher perceived exertion was observed with an increase in weight throughout the trials. An increase in anterior/posterior trunk displacement was seen with an increase in exertion. Moreover, increased changes in brain connectivity were seen with higher perceived task difficulty. Also, individuals were more likely to adjust their mechanics with increased perceived exertion.

Kinematic Data, EEG Data and RPE Scores

Overall, mechanically speaking, healthy and post-injured individuals showed moderate differences in kinematic patterns. Exception being the anterior/posterior trunk displacement being greater as well as differences in hand pathways for the previously injured shoulders. However, the central nervous system has shown to take on significant changes in patterns after an individual has experienced a shoulder injury. Previous research has provided evidence to support both of these observations(6,52,53). RPE scores also increased and decreased with task demands.

Chapter VI: Conclusion

In conclusion, this study identified significant differences in alpha, low/high beta, and theta band connectivity in individuals with previous shoulder injury, as well as differences in anterior/posterior trunk displacement and hand pathways while performing a repetitive, goaloriented upper extremity task. In contrast, after compensatory factors were observed in the lateral trunk displacement, hand pathways remained relatively unchanged among participants throughout the course of the trials. Kinematic variability increased at proximal joints (i.e. trunk, elbow angular position), but not as extreme distally (hand pathway) after changes in resistance was applied. Functional connectivity increased in post-injured individuals, relying on greater areas of the brain unlike their healthy counterpart. These findings agree with previous research during repetitive reaching tasks, and provide some validity to the idea that injury/fatigue adaptations are governed by not just kinematic principles, but by a higher level hierarchical organization of the central nervous system. Furthermore, these results underscore the importance of considering how neurologically different an individual who has experienced a shoulder injury may be, rather than an exclusive focus on just kinematics. If the neurological aspect is considered, maybe rehabilitation processes can be better understood and executed to better treat the individual in the future. Further research is needed to understand how individuals modify movement kinematics across different joints and determine how consistent these changes are across tasks and brain connectivity.

Bibliography

1. Blache Y, Begon M, Michaud B, Desmoulins L, Allard P, Maso FD. Muscle function in glenohumeral joint stability during lifting task. PLoS One. 2017;12(12):1–15.
2. Kraemer W, Denegar C, Flanagan S. Recovery from injury in sport: Considerations in the transition from medical care to performance care. Sports Health. 2009;1(5):392–5.
3. Hsu CJ, Meierbachtol A, George SZ, Chmielewski TL. Fear of Reinjury in Athletes: Implications for Rehabilitation. Sports Health. 2017;9(2):162–7.
4. George j.Davies, Dickoit-Hoitman S. Neuromuscular Testing and Rehabilitation of the Shoulder Complex. Jt Struct Funct A Compr Anal. 1993;231–70.
5. Dancause N, Barbay S, Frost SB, Plautz EJ, Stowe AM, Friel KM. Ipsilateral connections of the ventral premotor cortex in a NewWorld primate. 2008;495(4):374–90.
6. Cowley JC, Gates DH. Proximal and distal muscle fatigue differentially affect movement coordination. PLoS One. 2017;12(2):1–17.
7. Hawkes DH, Alizadehkhaiyat O, Kemp GJ, Fisher AC, Roebuck MM, Frostick SP. Shoulder muscle activation and coordination in patients with a massive rotator cuff tear: An electromyographic study. 30, Journal of Orthopaedic Research. 2012. 1140–6.
8. Terry GC, Chopp TM. Functional Anatomy of the Shoulder. J Athl Train. 2000;35(3):248–55. : https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1323385/
9. Scholz J, Gregor S. The uncontrolled manifold concept: identifying control variables for a functional task. Exp Brain Res. 1999;289–306.
10. Smidt GL, Mcquade K. Scapulothoracic Muscle Fatigue Associated with Alterations in Shoulder Elevation. J Orthop Sport Phys Ther. 1998;28(2).
11. Joseph B. Myers, MA, ATC and Scott M. Lephart, PhD A. The role of the sensorimotor system in the athletic shoulder. J Athl Train. 2000;172(3):351–63.
12. Lephart BLRSM. The Sensorimotor System , Part I : The Role Functional Joint Stability. 2014;(June).
13. Boettcher CE, Cathers I, Ginn KA. The role of shoulder muscles is task specific. J Sci Med Sport. 2010;13(6):651–6.: http://dx.doi.org/10.1016/j.jsams.2010.03.008
14. Scheidt RA, Reinkensmeyer DJ, Conditt MA, Rymer WZ, Mussa-Ivaldi FA. Persistence of Motor Adaptation During Constrained, Multi-Joint, Arm Movements. J Neurophysiol. 2000;84(2):853–62. : http://www.physiology.org/doi/10.1152/jn.2000.84.2.853
15. Lephart SM, Warner JJP, Borsa PA, Fu FH. Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. J Shoulder Elb Surg 1994;3(6):371–80. : http://dx.doi.org/10.1016/S1058-2746(09)80022-0
16. Joseph B. Myers, MA, ATC; Kevin M. Guskiewicz, PhD A, Robert A. Schneider, MS, PT, ATC; William E. Prentice, PhD, ATC P. Proprioception and Neuromuscular Control of the Shoulder After Muscle Fatigue. 1999;34(4):362–7.

17. Grigg P. Peripheral Neural Mechanisms in Proprioception. Hum Kinet. 1994;

18. Damien Bachasson, PT, PhD, Anshuman Singh, MD, Sameer Shah, PhD, John G. Lane, MD, and Samuel R. Ward, PT P. The Role of the Peripheral and Central Nervous Systems in Rotator Cuff Disease. 2015;(8):1322–35.

19. Bizzi E, December R. Posture control and Trajectory Formation During Arm Movement. J Neurosci. 1984;4:2738–44.

20. Tanji J, Shima K. Role for supplementary motor area cells in planning several movements ahead. 371, Nature. 1994. 413–6.

21. Nathan DE, Prost RW, Guastello SJ, Jeutter and DC, Reynolds NC. Investigating the neural correlates of goal-oriented upper extremity movements. NeuroRehabilitation. 2012 12;31(4):421–8. : http://jproxy.lib.ecu.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=ccm&AN=104390764&site=ehost-live&scope=site

22. Biernaskie J, Corbett D. Enriched rehabilitative training promotes improved forelimb motor function and enhanced dendritic growth after focal ischemic injury. J Neurosci. 2001;21(14):5272–80.

23. Plautz EJ, Milliken GW, Nudo RJ. Effects of repetitive motor training on movement representations in adult squirrel monkeys: Role of use versus learning. Neurobiol Learn Mem. 2000;74(1):27–55.

24. Connolly JD, Andersen RA, Goodale MA. FMRI evidence for a parietal reach region in the human brain. Exp Brain Res. 2003;153(2):140–5.

25. Nitschke MF, Kleinschmidt A, Wessel K, Frahm J. Somatotopic motor representation in the human anterior cerebellum. A high-resolution functional MRI study. Brain. 1996;119(3):1023–9.

26. Synofzik M, Lindner A, Thier P. The Cerebellum Updates Predictions about the Visual Consequences of One's Behavior. Curr Biol. 2008;18(11):814–8.

27. Houk JC, Rymer ZW. Neural control of muscle length and tension. Handb Physiol Nerv Syst Mot Control. 2011;257–323.

28. Ghez C, Sainburg R. Proprioceptive control of interjoint coordination. Can J Physiol Pharmacol. 1995;73(2):273–84 http://www.nrcresearchpress.com/doi/10.1139/y95-038

29. Barden JM, Balyk R, Raso VJ, Moreau M, Bagnall K. Dynamic Upper Limb Proprioception in Multidirectional Shoulder Instability. Clin Orthop Relat Res. 2004;(420):181–9.

30. Ghez C, Gordon J, Ghilardi MF. Impairments of reaching movements in patients without proprioception . II . Effects of visual information on accuracy. 2013;73(1):361–72.

31. Mcgowan C, Hyytiäinen H. Muscular and neuromotor control and learning in the athletic horse. Comp Exerc Physiol. 2017;6(16);13:1–10.

32. Tapadia M, Mozaffar T, Gupta R. Compressive Neuropathies of the Upper Extremity: Update on Pathophysiology, Classification, and Electrodiagnostic Findings. J Hand Surg

Am. 2010;35(4):668–77: http://dx.doi.org/10.1016/j.jhsa.2010.01.007

33. Thoroughman KA, Shadmehr R. Electromyographic Correlates of Learning an Internal Model of Reaching Movements. J Neurosci. 1999;19(19):8573–88. : http://www.jneurosci.org/lookup/doi/10.1523/JNEUROSCI.19-19-08573.1999

34. Horak FB. Assumptions Underlying Motor Control for Neurologic Reabilitation. 1991;11–27.

35. Yu CX, Ji TT, Song H, Li B, Han Q, Li L. Abnormality of spontaneous brain activities in patients with chronic neck and shoulder pain: A resting-state fMRI study. J Int Med Res. 2017;45(1):182–92.

36. Tarokh L, Carskadon MA, Achermann P. Developmental changes in brain connectivity assessed using the sleep EEG. Neuroscience 2010 年;171(2):622–34. : http://dx.doi.org/10.1016/j.neuroscience.2010.08.071

37. Gentile AM. Movement science: Implicit and explicit processes during acquisition of functional skills. Scand J Occup Ther. 1998;5(1):7–16.

38. Muratori LM, Lamberg EM, Quinn L, Duff S V. Applying principles of motor learning and control to upper extremity rehabilitation Lisa. 2013;26(2):94–103.

39. Yang J, Li P. Brain Networks of Explicit and Implicit Learning. PLoS One. 2012;7(8).

40. Atkeson CG. Learning ARM Kinematics and Dynamics. Annu Rev Neurosci 1989; 12(1):157–83.: http://www.annualreviews.org/doi/10.1146/annurev.ne.12.030189.001105

41. Loeb. Finding common ground between robotics and physiology. 1983;203–4.

42. Jun Izawa1, Tushar Rane1, Opher Donchin2 and RS. Motor adaptation as a process of reoptimization. JNeuroscience. 2008;

43. Bastian AJ. Understanding sensorimotor adaptation and learning for rehabilitation. Curr Opin Neurol. 2008;21(6):628–33.

44. Kluzik J, Diedrichsen J, Shadmehr R, Bastian AJ. Reach Adaptation: What Determines Whether We Learn an Internal Model of the Tool or Adapt the Model of Our Arm? J Neurophysiol. 2008;100(3):1455–64.: http://jn.physiology.org/cgi/doi/10.1152/jn.90334.2008

45. Systems VM. Vicon Upper Limb Model. 2007;(July):1–18.

46. Ewald A, Marzetti L, Zappasodi F, Meinecke FC, Nolte G. Estimating true brain connectivity from EEG/MEG data invariant to linear and static transformations in sensor space. Neuroimage 2012;60(1):476–88: http://dx.doi.org/10.1016/j.neuroimage .2011.11.084

47. Oostenveld R, Fries P, Maris E, Schoffelen JM. FieldTrip: Open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. Comput Intell Neurosci. 2011;2011.

48. Lim S, Yeo M, Yoon G. Comparison between concentration and immersion based on EEG analysis. Sensors (Switzerland). 2019;19(7):1–13.

49. Von Stein A, Sarnthein J. Different frequencies for different scales of cortical integration: From local gamma to long range alpha/theta synchronization. Int J Psychophysiol. 2000;38(3):301–13.

50. Roohi-Azizi, M., Azimi, L., Heysieattalab, S., & Aamidfar, M. (2017). Changes of the brain's bioelectrical activity in cognition, consciousness, and some mental disorders. Medical journal of the Islamic Republic of Iran, 31, 53. https://doi.org/10.14196/mjiri.31.53

51. Ketenci, S., & Kayikcioglu, T. (2019). Investigation of Theta Rhythm Effect in Detection of Finger Movement. Journal of experimental neuroscience, 13, 1179069519828737. https://doi.org/10.1177/1179069519828737

52. Haller, S., Cunningham, G., Laedermann, A., Hofmeister, J., Van De Ville, D., Lovblad, K. O., & Hoffmeyer, P. (2014). Shoulder apprehension impacts large-scale functional brain networks. AJNR. American journal of neuroradiology, 35(4), 691–697. https://doi.org/10.3174/ajnr.A3738

53. Jiang, Z., Wang, X.-F., Kisiel-Sajewicz, K., Yan, J. H., & Yue, G. H. (2012). Strengthened functional connectivity in the brain during muscle fatigue. *NeuroImage*, *60*(1), 728–737. https://doi.org/https://doi.org/10.1016/j.neuroimage.2011.12.013

54. Wheaton, L. A., Carpenter, M., Mizelle, J. C., & Forrester, L. (2008). Preparatory band specific premotor cortical activity differentiates upper and lower extremity movement. Experimental brain research, 184(1), 121–126. https://doi.org/10.1007/s00221-007-1160-4

55. Abhang, P., Gawali, B., & Mehrotra, S. (2016). *Introduction to EEG- and Speech-Based Emotion Recognition Book.*

56. Ketenci, S., & Kayikcioglu, T. (2019). Investigation of Theta Rhythm Effect in Detection of Finger Movement. Journal of experimental neuroscience, 13, 1179069519828737. https://doi.org/10.1177/1179069519828737

57. ANI. (2019). Understanding brain waves. Neurofeedback Alliance. http://neurofeedbackalliance.org/understanding-brain-waves/

58. Tsujimoto, T., Shimazu, H., & Isomura, Y. (2006). Direct recording of theta oscillations in primate prefrontal and anterior cingulate cortices. Journal of neurophysiology, 95(5), 2987– 3000. https://doi.org/10.1152/jn.00730.2005

List of Appendices

Appendix A

EAST CAROLINA UNIVERSITY
University & Medical Center Institutional Review Board
4N-64 Brody Medical Sciences Building· Mail Stop 682
600 Moye Boulevard · Greenville, NC 27834
Office **252-744-2914** · Fax **252-744-2284** ·
www.ecu.edu/ORIC/irb

Notification of Initial Approval: Expedited

From:	Biomedical IRB
To:	Chris Mizelle
CC:	Chris Mizelle
Date:	2/28/2018
Re:	UMCIRB 18-000350 Neuromuscular Control of the Shoulder

I am pleased to inform you that your Expedited Application was approved. Approval of the study and any consent form(s) is for the period of 2/28/2018 to 2/27/2019. The research study is eligible for review under expedited category #4,7. The Chairperson (or designee) deemed this study no more than minimal risk.

Changes to this approved research may not be initiated without UMCIRB review except when necessary to eliminate an apparent immediate hazard to the participant. All unanticipated problems involving risks to participants and others must be promptly reported to the UMCIRB. The investigator must submit a continuing review/closure application to the UMCIRB prior to the date of study expiration. The Investigator must adhere to all reporting requirements for this study.

Approved consent documents with the IRB approval date stamped on the document should be used to consent participants (consent documents with the IRB approval date stamp are found under the Documents tab in the study workspace).

The approval includes the following items:

Name	Description
Edinburgh Handedness Inventory	Surveys and Questionnaires
Neuromuscular Control of the Shoulder	Study Protocol or Grant Application
Neuromuscular Control of the Shoulder Consent form	Consent Forms
Neuromuscular Control of the Shoulder- Questionnaire	Surveys and Questionnaires
Neuromuscular Control of the Shoulder Recruitment Flyer	Recruitment Documents/Scripts

The Chairperson (or designee) does not have a potential for conflict of interest on this study.

Appendix B

Edinburgh Handedness Inventory

The Edinburgh Handedness Inventory was used as a measurement scale to assess the dominance of the participants right or left handedness in everyday activities. For an individual to qualify for this study, the participant could not be ambidextrous or left-handed.

Edinburgh Handedness Inventory[1]

Your Initials:____________

Please indicate with a check (✓) your preference in using your left or right hand in the following tasks.

Where the preference is so strong you would never use the other hand, unless absolutely forced to, put two checks (✓✓).

If you are indifferent, put one check in each column (✓ | ✓).

Some of the activities require both hands. In these cases, the part of the task or object for which hand preference is wanted is indicated in parentheses.

Task / Object	Left Hand	Right Hand
1. Writing		
2. Drawing		
3. Throwing		
4. Scissors		
5. Toothbrush		
6. Knife (without fork)		
7. Spoon		
8. Broom (upper hand)		
9. Striking a Match (match)		
10. Opening a Box (lid)		
Total checks:	LH =	RH =
Cumulative Total	CT = LH + RH =	
Difference	D = RH – LH =	
Result	R = (D / CT) × 100 =	
Interpretation: (Left Handed: R < -40) (Ambidextrous: -40 ≤ R ≤ +40) (Right Handed: R > +40)		

[1] Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh inventory. *Neuropsychololgia*, *9*, 97-113.

Appendix C

Informed Consent Form to Participate in Research

Participants were informed of potential risks associated with the EEG and motion capture, and an informed consent was obtained before any measurements were taken. The protocol and consent form were approved by the East Carolina University Institutional Review Board.

East Carolina University

Informed Consent to Participate in Research
Information to consider before taking part in research that has no more than minimal risk.

Title of Research Study: The Effects of Injury on the Neuromotor Control of the Shoulder

Principal Investigator: J.C. (Chris) Mizelle, Ph.D.
Institution, Department or Division: East Carolina University / Department of Kinesiology
Address: 170B Minges Coliseum
Telephone #: 252-328-9271

Researchers at East Carolina University (ECU) study issues related to society, health problems, environmental problems, behavior problems and the human condition. To do this, we need the help of volunteers who are willing to take part in research.

Why am I being invited to take part in this research?

You are being invited to take part in this research because you are a healthy volunteer who meets our inclusion and exclusion criteria. The decision to take part in this research is yours to make. By doing this research, we hope to learn how the brain processes tool use in daily tasks, and how aging might affect these processes.

If you volunteer to take part in this research, you will be one of about 30 people to do so.

Are there reasons I should not take part in this research?
You should not volunteer for this study if you are not between the ages of 18-35, or if you have any previous history of serious neurological or upper extremity neuromotor illness or injury that results in pain during a continuous lifting motion of the shoulder.

What other choices do I have if I do not take part in this research?
You can choose not to participate. Your participation is strictly voluntary.

Where is the research going to take place and how long will it last?
The research will be conducted at East Carolina University, in the laboratory of Dr. Mizelle (170 Minges Coliseum). You will need to come to 170A Minges Coliseum one time during the study. The total amount of time you will be asked to volunteer for this study is approximately 2 hours over one day.

What will I be asked to do?
You will be asked to do the following: participate in a research study designed to help us understand the effects of Rotator cuff tear injury on neuromotor control and range of motion of the shoulder complex. Please read through the section related to your participation below. Please ask any questions you may have about your participation.

If you decide to participate in this study, you will come to the Sensory-Motor Control Lab (room 170A in Minges Coliseum, East Carolina University) for a single visit lasting approximately 2 hours. You will be asked to perform various movements and contractions with your shoulder and arm while brain signals are being recorded

Consent Version # or Date: 12.5.2017

with Electroencephalography (EEG). You will also have sensors placed on your muscle to measure how active they are while you are producing force Electromyography (EMG). Lastly, you will have reflectors attached to your arm and shoulder to measure the range of motion during each task. There will be two questionnaires that must be complete before leaving lab pertaining to the self-reported shoulder injury history.

What might I experience if I take part in the research?
EEG is a non-invasive test. However, participants may experience some discomforts, which are common to the EEG procedures as a result of taking part in this study. There may be mild discomfort and adhesive residue apparent upon the removal of certain EEG sensors. There may be temporary alteration of hairstyle after removal of the EEG cap and some subjects may experience minimal hair loss. Impression marks from the EEG cap and sensors will also be present after the research study, but will resolve shortly after the research study is completed.

Producing movements away from normal motions during the range of motion tasks may occasionally lead to muscle soreness. This is common in many activities, and will resolve within 24-48 hours after the research study is completed.

EMG is also a non-invasive measurement. However, there may be mild discomfort and adhesive residue apparent upon the removal of the EMG sensors.

Motion capturing monitors require the use of reflectors. The reflectors will be applied using adhesive tape and elastic bandages. There may be a mild discomfort during application and removal of the bandage.

Will I be paid for taking part in this research?
We are unable to pay you for the time you volunteer.

Will it cost me to take part in this research?
It will not cost you any money to be part of the research.

Who will know that I took part in this research and learn personal information about me?
ECU and the people and organizations listed below may know that you took part in this research and may see information about you that is normally kept private. With your permission, these people may use your private information to do this research:

- Any agency of the federal, state, or local government that regulates human research. This includes the Department of Health and Human Services (DHHS), the North Carolina Department of Health, and the Office for Human Research Protections.
- The University & Medical Center Institutional Review Board (UMCIRB) and its staff have responsibility for overseeing your welfare during this research and may need to see research records that identify you.

How will you keep the information you collect about me secure? How long will you keep it?
All personally identifiable information, such as this form, will be kept in a locked file cabinet within Dr. Mizelle's office, thus providing two levels of security. You will be assigned a participant number that will be used to track performance, and a hard copy of the key to breaking the subject codes and related identifiers will be held under lock and key Dr. Mizelle's secured office space. Your name and participant number will not be identified in any subsequent report or publication. All electronic data (EEG, EMG, force) will be fully de-identified, and will be kept for storage on an encripted hard drive, also inside Dr. Mizelle's locked office. All research records and/or identifiers will be securely maintained for 5 years past the completion of the study, and will then be securely destroyed. It is also possible that your de-identified data may be used in future research without anyone knowing it is information from you.

What if I decide I don't want to continue in this research?
You can stop at any time after it has already started. There will be no consequences if you stop and you will not be criticized. You will not lose any benefits that you normally receive.

Who should I contact if I have questions?
The people conducting this study will be able to answer any questions concerning this research, now or in the future. You may contact the Principal Investigator at 252-328-9271 (days, between 9:00 am and 5:00 pm).

If you have questions about your rights as someone taking part in research, you may call the Office of Research Integrity & Compliance (ORIC) at phone number 252-744-2914 (days, 8:00 am-5:00 pm). If you would like to report a complaint or concern about this research study, you may call the Director of the ORIC, at 252-744-1971.

I have decided I want to take part in this research. What should I do now?
The person obtaining informed consent will ask you to read the following and if you agree, you should sign this form:

- I have read (or had read to me) all of the above information.
- I have had an opportunity to ask questions about things in this research I did not understand and have received satisfactory answers.
- I know that I can stop taking part in this study at any time.
- By signing this informed consent form, I am not giving up any of my rights.
- I have been given a copy of this consent document, and it is mine to keep.

____

Participant's Name (PRINT) **Signature** **Date**

Person Obtaining Informed Consent: I have conducted the initial informed consent process. I have orally reviewed the contents of the consent document with the person who has signed above, and answered all of the person's questions about the research.

____

Person Obtaining Consent (PRINT) **Signature** **Date**

Appendix D

Rate of Perceived Exertion

The rate of perceived exertion (RPE) was used to evaluate the level of difficulty or easiness of the task between each trial. The Borg C-10 RPE scale assisted in understanding what the participants experienced during each trial. The scale also provided information on if the task was too demanding for the participant to complete.

1-10 Borg Scale of Perceived Exertion	
0	Rest
1	Really Easy
2	Easy
3	Moderate
4	Sort of Hard
5	Hard
6	
7	Really Hard
8	
9	Really, Really Hard
10	Maximal

Appendix E

Neuromotor Control of the Shoulder Questionnaire

Each participant was required to complete a questionnaire before the completion of the study. The questionnaire was used to get background information on each participant. This information was used to understand pain level, post-injury, and length of time in which the individual may have been injured.

Neuromotor Control of the Shoulder Questionnaire

1. Have you ever experienced a shoulder injury? Yes____ No____
2. If yes: Which shoulder did this occur to? Right____ Left_____
3. How recent was the injury? ______________________________
4. Do you experience any discomfort while performing certain activities? Yes____ No____
5. If yes: what activities?

   ____

   ____
6. What type of discomfort (if any) do you feel during activities (i.e. fatigue, stiffness, soreness)?

   ____

   ____