Role of tendon vibration in multijoint reflex coupling in the hemiparetic arm post stroke

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ROLE OF TENDON VIBRATION IN MULTIJOINT REFLEX COUPLING IN THE HEMIPARETIC ARM POST STROKE

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ABSTRACT
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Post stroke hemiparesis causes reflex coupling in multiple muscles of the arm, leading to atypical movements that hamper motor control. In particular, people post-stroke can become unstable while holding the arm at the end of a planar motion. Recently, we have found that tendon vibration of the wrist flexors improves the stability of the arm during a hold task. The objective of the current study was to identify the effects of vibration applied to the wrist flexors on the biceps and triceps stretch reflexes, generated using a tendon tapper. In people post-stroke, tendon tap perturbations of the biceps and triceps elicit heteronymous spinal reflexes in muscles of the wrist, elbow and shoulder. We hypothesized that if tendon vibration improved stabilization of the arm through spinal reflex pathways, then heteronymous tendon tap reflexes would be modified by wrist vibration. Ten chronic stroke survivors and 5 age-matched controls participated in this study. Subjects were seated in a high-back chair, force/torque measurements were made from the 6 axis load cells at the elbow and wrist and EMG signals were recorded from 8 muscles. Isometric maximum voluntary contractions (MVCs) were performed for wrist and elbow flexion/extension and shoulder abduction/adduction. The test protocol consisted of 6 active tasks and 3 relaxed conditions in a randomized order, each consisting of 30 taps, with vibration applied during the middle 10 taps. The active tasks consisted of the same task types as the MVCs; however, the subjects maintained their primary force/torque between 10% and 30% of their MVCs. Peak-to-peak amplitude of the reflexes showed negligible changes in amplitude during vibration compared to the non vibration trials. These results showed that tendon vibration did not affect the multi-joint reflex coupling of muscles across the arm. Thus, the effects of tendon vibration as a sensory intervention, as seen in previous studies on arm stability do not appear to occur at the spinal level. These results imply that the effects of vibration on arm stability likely occur in supraspinal structures, suggesting a change in supraspinal sensorimotor integration underlies the effects.

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CHAPTER 1: INTRODUCTION

Each year approximately 795,000 people in the United States are affected by a new or recurrent stroke (Heart disease and stroke statistical update: 2009, 2010). Of these people, approximately 40% suffer from reduced motor function in their arm due to hemiparesis (Parker et al., 1986). Hemiparesis in the arm is associated with disturbed muscle tone, muscular discoordination patterns known as synergies and weakness (Dewald et al., 2001). Enhanced motor recovery of the affected arm post-stroke could substantially improve functional activities of daily life such as eating, dressing and driving. Hence, improvement in motor function of the arm post stroke is a very important factor in rehabilitation and restoration of quality of life.

Augmenting sensory information can improve motor function in the arm post-stroke as evidenced in previous studies (Conrad et al., 2009). Tendon vibration applied at the wrist flexors improves motor control of the arm in the horizontal plane (Conrad et al., 2009). However, the exact mechanism of improved control of movement using sensory augmentation through tendon vibration is not clearly understood. We hypothesize that it could mainly be attributed to two mechanisms. One possible mechanism would involve enhanced integration of sensorimotor information at the supraspinal level. Improvements could be associated with enhanced proprioceptive input to the cortex and improved excitation input to the cerebellum. Both structures play an important role in motor control and the correction of movement error. A second factor that could aid in controlling motion is improved reflexes at the spinal level resulting from enhanced cortical control in regulating reflexes.

The goal of this thesis was to improve our understanding of the neural mechanisms underlying improvements in control of the hemiparetic arm movement using targeted tendon vibration. The approach of this study was to identify the effects of tendon vibration, applied to the wrist flexors, on the biceps and triceps stretch reflexes generated using a motorized tendon tapper.

The following sections outline a number of background topics related to this study. These sections address spinal reflexes, a method to generate them (tendon tapping), neural coupling of reflexes in the arm, sensory-motor dysfunctions post-stroke, and most importantly, previous research depicting improved arm function post-stroke during horizontal planar movement with the use of tendon vibration.

1.1 Stretch reflexes

Reflexes have an important role in molding the motor control of movement. Lengthening of a muscle can lead to its contraction, a phenomenon known as the 'stretch reflex'. A simple reflex pathway, the reflex arc, involves a sensory receptor with its afferent fibers and the motoneurons along with the muscle they are innervating. These reflexes begin at the muscle spindles, which are the sensory receptors for stretch and consist of one group of primary (Ia) afferents and one group of secondary (II) afferents (Matthews, 1972). Ia afferents play a major role in the stretch reflex. They are highly sensitive to stretch, transmit sensory signals from muscle spindles, and synapse with alpha motoneurons in the ventral motor horn of the spinal cord, thereby exciting the motoneuron. This leads to contraction of the homonymous muscle, and is known as the monosynaptic stretch reflex (Kandel et al., 2000; Lance et al., 1965). Reflexes can also be

polysynaptic in nature, involving one or more spinal interneuronal circuits (Kandel et al., 2000).

1.1.1 Tendon tap reflexes

A tendon tap is known to elicit, via a reflex arc, a phasic stretch reflex caused by a sudden stretch of a muscle (Toft et al., 1989). The tendon tap generates spinal reflexes by activating muscle spindles' Ia afferents (Pierrot-Desseilligny et al., 2005). This leads to a synchronous volley in the Ia afferents, which are the fastest conducting fibers arising from the muscle spindles. These afferent nerve fibers in turn generate excitatory post synaptic potentials (EPSPs) in the spinal alpha motoneurons and in interneurons in the anterior horn of different segments of the spinal cord. The spinal cord conducts nerve impulses via the motoneurons to the muscles (efferent pathway) causing them to contract (Dick, 2003). There is a delay of about 20-25 ms for the muscle contraction to occur in response to tendon percussions (Burke et al., 1983).

The muscle contraction or shortening of the homonymous muscle that is elicited by a tendon tap is proportional to the amount of stretch in the muscle and is on the order of few millimeters (Clarke et al., 1973). The contraction increases up to a maximum value as a result of incrementing force through the tendon tap, beyond which it remains constant. This ceiling effect is possibly because the muscle spindles are maximally excited and no further increase can be produced by raising the tendon force further (Stam et al., 1987). Since a tendon tap can effectively elicit stretch reflexes, they are used to study the reflex pathways.

1.1.2 Neural coupling of stretch reflexes

Apart from being monosynaptic, stretch reflexes are also known to have heteronymous pathways and affect synergist muscles involved in performing the same motor task. Muscle spindle Ia afferents have heteronymous monosynaptic connections on motor neurons of distal muscles in cats, monkeys (Illert et al., 1999) and in humans (Illert et al., 1999; Lance et al., 1965). Heteronymous reflex responses to Ia afferent excitation have also been shown in agonist/antagonist muscle pairs of the elbow and shoulder, such as biceps and triceps, or the pectoralis major and posterior deltoid (McClelland et al., 2001).

Heteronymous stretch reflexes, occurring across multiple joints of the arm, account for the coordination of arm movements. In a study conducted by Perreault et al. (2005), 3D ramp and hold perturbations were applied to the whole arm to generate stretch reflex responses and coordination among multiple muscles was observed, since reflexes were generated at shoulder and elbow joints. They also showed that heteronymous stretch reflexes could account for the coupling since there was a delay in excitatory response during lengthening of the triceps (both lateral and long head) (Perreault et al., 2005). This study provides evidence for the occurrence of heteronymous reflex coupling across muscles of the arm in healthy subjects.

Coupling of stretch reflexes throughout the arm is disturbed in people post-stroke. Altered stretch reflexes are observed in the hemiparetic arm, which is contralateral to the lesion site in the brain. Abnormal multijoint muscle coordination patterns have been observed in the hemiparetic arm during isometric contractions. These synergy patterns

consist of coactivations of shoulder adductors with elbow extensors and shoulder abductors with elbow flexors (Dewald et al., 1995). Disturbed coordination between the shoulder and elbow also exists in involuntary stretch reflexes. Altered limb function can be marked by multijoint reflex coupling. (Trumbower et al., 2008) demonstrated abnormal coupling between elbow flexor and shoulder abductors with the limb supported against gravity, indicating the impact of multijoint reflex coupling on arm function. Heteronymous multijoint reflex coupling has also been characterized previously in the arm of stroke survivors by stretching their paretic elbow. Sangani et al., (2007) demonstrated in the afferent connections linking the shoulder, elbow and wrist muscles that both monosynaptic and polysynaptic connections existed. Better knowledge of the reflex coupling patterns in the arm is important to understand the abnormal synergy patterns for improving motor function in patients post-stroke.

1.1.3 Quantitative assessment of stretch reflexes in neurological impairments

Tendon tapping is a widely accepted technique for assessing neuromuscular stretch reflexes (Rosenbaum et al., 1995; Walker et al., 1976). Changes in reflex magnitude provide insights into impairments of the nervous system, fatigue and aging. Tendon taps can be used to evaluate both the functional ability of the affected limb and the efficacy of a drug or therapeutic treatment (Fedotova et al., 1976).

Diseases of the upper motoneuron (neurons originating in motor cortex and carrying information down to the final common pathway) are characterized by hyperactive phasic stretch reflexes. These heightened tendon tap reflexes, accompanied by an increased passive resistance during stretch of a muscle, indicate the occurrence of

spasticity defined as " increased resistance to passive stretch" (Dietz et al., 2007). A hyperactive reflex raises concerns about the integrity of descending pathways such as the corticospinal tract, which influences the excitability of the reflex arc (Walker et al., 1976). These stretch reflexes have short-latencies and magnitudes that correspond to the amount of the muscle tone, which is increased in spasticity (Toft et al., 1989). Exaggerated reflexes occurring in spasticity have been attributed to many factors such as reduced presynaptic Ia inhibition (Fellows et al., 1993), an increase in Ia monosynaptic excitation and changes in input to spinal interneurons (Dick, 2003). Increased motoneuronal excitability has also been suggested to cause heightened reflex responses observed in spasticity (Katz et al., 1989). Conversely, diseases of lower motoneurons (neurons originating in the spinal cord and terminating in the skeletal muscle) are characterized by decreased or absent stretch reflexes. Decreased reflexes usually occur due to a disturbance in the afferent or the efferent pathways of the reflex and are an indication of diseases in the peripheral nervous system (Toft et al., 1989; Walker et al., 1976). These examples show that exaggerated tendon tap reflexes provide a measure for diagnosing and monitoring diseases of the upper and lower motoneuron.

Tendon tap reflexes also provide valuable information on the symmetry of lesions interrupting corticospinal pathways. If hyperactive reflexes are unilateral, then the lesion must be present in the corticospinal tract of the opposite side (Walker et al., 1976). Stretch reflexes also hint towards the position of lesions in the spinal cord. For instance, an absent triceps reflex suggests a lesion in the C6-C7 spinal segmental level (Dick, 2003). Using this knowledge clinicians are able to use tendon tap reflexes as a test of the

neurological system and tendon tap reflexes are valuable in diagnosing the location of disease in the nervous system.

There are a few disadvantages in using tendon tap reflexes for diagnosis. Quantization of stretch reflexes becomes an issue when they are not stringently standardized. A standard perturbation in terms of amplitude and velocity is very important for generating reproducible stretch reflexes (Toft et al., 1989). Controlled perturbations can be produced using programmed electronic hammers. Generating reflexes from flaccid and non-contracting muscles in patient populations is difficult. Also, eliciting stretch reflexes in relaxed healthy individuals is also hard because of the hypoexcitability of the motoneurons (Dick, 2003). However, in some studies, this problem is circumvented by triggering stretch reflexes while individuals generate isometric muscle contractions. Fellows et al., (1993) have suggested that reflexes generated via tendon taps do not provide an indication of excitability in simple reflex pathways. These researchers have suggested using 'muscle stretch' over tendon tapping because tendon taps are not an impetus the nervous system is subjected to in daily life (Fellows et al., 1993). Nevertheless, tendon tapping has continued to be used as a test method despite the fact that similar perturbations are not encountered in daily life. While there are some minor disadvantages, quantification of stretch reflexes generated by a tendon tapper is an invaluable tool for the clinical assessment of stretch reflexes in man.

1.2 Sensory-motor impairments post-stroke

Muscle weakness, defined as the inability to generate adequate levels of force, is a primary cause of motor dsyfunction in hemiparetic patients. Weakness restricts motor rehabilitation post-stroke (Bourbonnais et al., 1989). Clinical studies in the arm have shown that weakness particularly affects wrist and finger flexors, as compared to shoulder and elbow muscles (Colebatch et al., 1989). Numerous physiological changes have been identified which contribute to muscle weakness in patients post-stroke. In a study conducted by McComas et al. (1973), loss in the amount of properly functioning motor units was observed and was attributed to trans-synaptic degeneration of motoneurons (McComas et al., 1973). An irregular recruitment order of motor units (Grimby et al., 1974, Rosenfalck et al., 1980) and alterations in their firing patterns (Rack et al., 1969) could lead to decreased levels of force production in hemiperatic patients. Reduction in force production can also be attributed to changes at the muscle level. Alterations in structural and mechanical properties of the shoulder and elbow muscles also contribute to lower force levels in the muscles. Low electromyographic activity is sometimes observed in the paretic side of hemiparetic subjects in conjunction with increased tone. The increased resistance to stretch (spasticity) in these hemiparetic subjects is due to altered mechanical properties of the shoulder and elbow muscles, rather than heightened stretch reflexes (Bourbonnais et al., 1989). Together these studies suggest that modifications in muscle and motoneuron properties along with the loss of descending drive, particularly via corticospinal tracts leads to reduced force production, resulting in muscle weakness.

Synergies are muscle coactivation patterns arising post-stroke and are a cause of motor dysfunction. In the hemiparetic arm, abnormal coupling of activity between muscles is observed. This leads to an overall discoordination of muscle activity and loss of individuation between joints of the arm. Stereotypical synergies have been observed and classified as the extensor and flexor synergies. The extensor synergy consists of shoulder extension/adduction with wrist flexion and elbow extension. The flexor synergy is comprised of shoulder flexion/abduction with wrist extension and elbow flexion (Brunnstrom, 1970). Atypical muscle activity patterns have also been observed in the upper extremity between shoulder abductors with elbow flexors and shoulder adductors with elbow extensors during isometric tasks (Dewald et al., 1995).

Spasticity is one of the symptoms arising from the upper motoneuron syndrome and is a characteristic of many of the diseases affecting the central nervous system, like stroke. Spasticity, as defined by Lance (1980) is "a motor disorder characterized by a velocity-dependent resistance in tonic stretch reflexes with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex the passive stretch of a limb". Spasticity is usually marked by hypertonia with exaggerated stretch reflexes (Lin et al., 1999). It has been observed that spasticity resulting from stroke results in pain, obstructs motor activity and causes other complications (Collin et al., 1988). Spasticity varies along the limb, being more prominent in the distal muscles as compared to the proximal muscles (Nielsen et al., 1995).

There is also a broad loss in somatosensation post stroke that includes touch, position, stereognosis, pressure, thermal sensation and motor precision (Sullivan et al., 2008). Due to these broad deficits, somatosensory loss has a significant influence on the

diagnosis and results of stroke rehabilitation (Reding et al., 1988; Winward et al., 1999). However, not much has been done to evaluate somatosensory loss as compared to evaluations of deficits in motor control post-stroke. Research has shown that motor impairments are more prominent in people with both sensory and motor loss as compared to sensory loss alone (Reding et al., 1988). Clinicians are now increasingly regarding sensory examination necessary for determining motor dysfunction post-stroke (Winward et al., 1999). The increasing importance of somatosensory assessment has led clinicians to come up with new assessment strategies to evaluate sensory loss. Sensory interventions such as electrical stimulation and tendon vibration have shown promising outcomes in people after stroke (Levin et al., 1992; Shirahashi et al., 2007; Tyson, 2003). The examination of somatosensory loss will aid motor rehabilitation post-stroke.

1.3 Sensory interventions post stroke

Studies have been carried out with sensory interventions aimed at somatosensory systems. Sensory interventions are increasingly being used to show improvements in motor function (Shirahashi et al., 2007) and spasticity (Levin et al., 1992). A range of sensory interventions have been carried out, which include thermal and electrical stimulation, vibration and pneumatic pressure (Sullivan et al., 2008). For instance, tendon vibration, which is relatively less studied, increases Ia afferent firing in the muscle spindles (Brown et al., 1967) causing a vibratory reflex in the homonymous muscle (Burke et al., 1976b). During tendon vibration, the largest number of Ia afferents are activated when a stimulus in the range of 80-100 Hz is used (Roll et al., 1989). Application of tendon vibration in this range leads to a 1:1 firing response in the Ia

afferents. In addition to causing a vibratory reflex, vibration can result in movement illusions consistent with an impression of a steady muscle length change (i.e. position) (Eklund, 1972). Hence, tendon vibration can alter the sense of limb position and cause a vibratory reflex resulting in the contraction of the homonymous muscle as well as simultaneously relaxing the antagonist.

Tendon vibration has been shown to be a powerful tool for improving motor capacity. In people with spasticity, vibration enhances voluntary contractions and simultaneously relaxes the antagonists (Hagbarth et al., 1968). The volitional force of contraction of a muscle increases with the application of tendon vibration (Ribot-Ciscar et al., 2003). Vibration also facilitates fusimotor drive. In decerebrate cats, application of low amplitude vibration to the Achilles tendon excites the primary afferents of the triceps surae and produces a weak facilitation of fusimotor neurons (Trott, 1976). Hence, tendon vibration can be used as a tool for improving motor function in the hemiparetic arm.

1.4 Effects of tendon vibration on planar motion in hemiparesis post-stroke

Sensory interventions, such as tendon vibration, can augment sensory input to the central nervous system (CNS). Tendon vibration, applied to the wrist flexors, improves end point stability in the hemiparetic arm during targeted point to point arm movements on a planar surface (Conrad et al., 2011). The improved arm stability was characterized by reduced electromyography activity from 8 muscles spanning the arm, suggesting an improvement in motor control that doesn't involve co-contraction. Reductions in kinematic parameters were also observed. Stability was quantified by the frequency of

oscillation at the endpoint and the magnitude of the movement, characterized by the 'stability error' – defined by the power of the endpoint trajectory signal. The error frequency during the vibration trials and stability error during the post-vibration trials was significantly reduced (Figure 1-1). There was also a reduction in grip pressure, indicative of better control of muscle activity at the target location. An overall improvement in the stability of the arm was observed at the target positions.

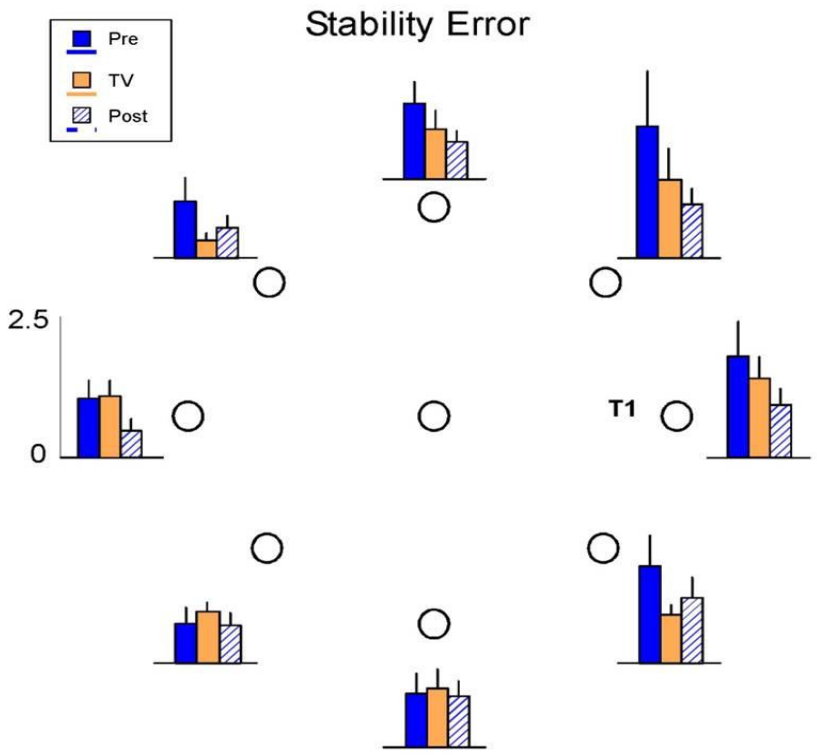

Figure 1-1. Stability Error: Pre-vibration, Vibration, Post vibration. A significant decrease in the post-vibration trials seen at several target locations. Adapted from Conrad, 2011.

Tendon vibration also improves shoulder instability in the hemiparetic arm, at the end of planar arm movements, in the presence of a divergent force field (Conrad et al., 2009). Similar to the center-out task, better command and stabilization of the movements are observed at the target location in the divergent field, reflected by a reduction in the

stability error. An additional interesting finding with the application of vibration in the presence of a divergent force field was reduced muscle activity in healthy subjects along with the stroke subjects (Figure 1-2). This finding suggests that applying wrist tendon vibration can lead to better proprioceptive control of the arm.

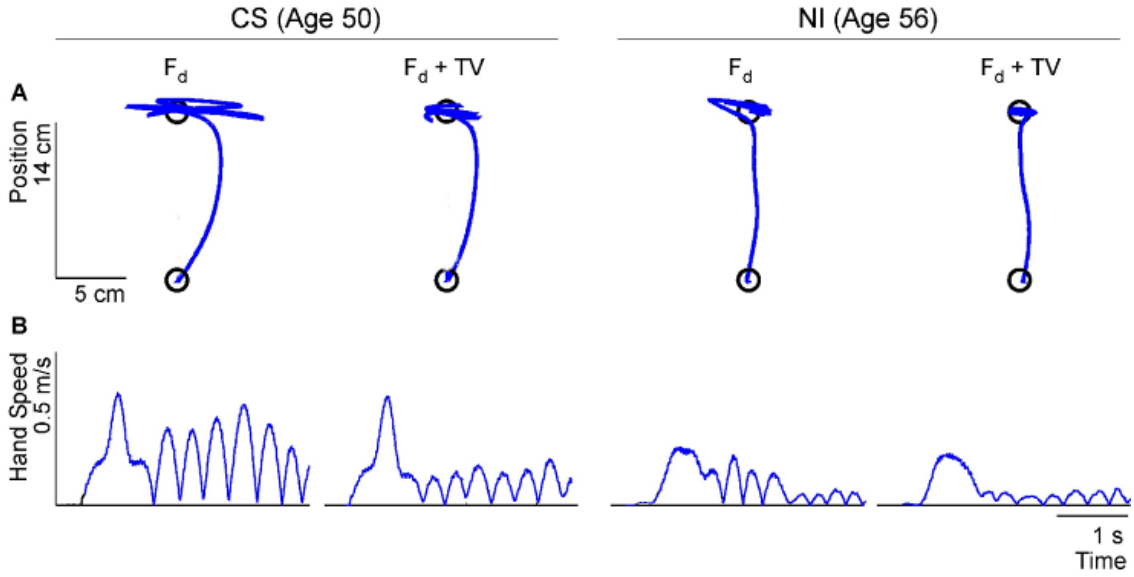

Figure 1-2. Characteristics of movement. Position and velocity plots of a stroke subject and a healthy aged match control subject. Tendon vibration stabilizes arm movement in the presence of a divergent force field at the target. CS-Chronic stroke, NI- Neurologically intact. Adapted from Conrad, 2009.

Tendon vibration also demonstrated an improvement in the stability of the hemiparetic arm during a figure eight tracking task on a planar surface (Conrad et al., 2009). Hand velocities and path lengths decrease while tracking a target with tendon vibration applied to the wrist (Figure 1-3). Dynamic stability error also reduces significantly, similar to results of the application of wrist vibration in a divergent force field and while performing point to point movements. Muscle electromyography (EMG) activity was also lowered while performing the tracking task. Improvements in arm movement during a Figure eight tracking task could suggest that tendon vibration aids in

better feedforward and feedback error control during manual tasks in the hemiparetic arm post-stroke.

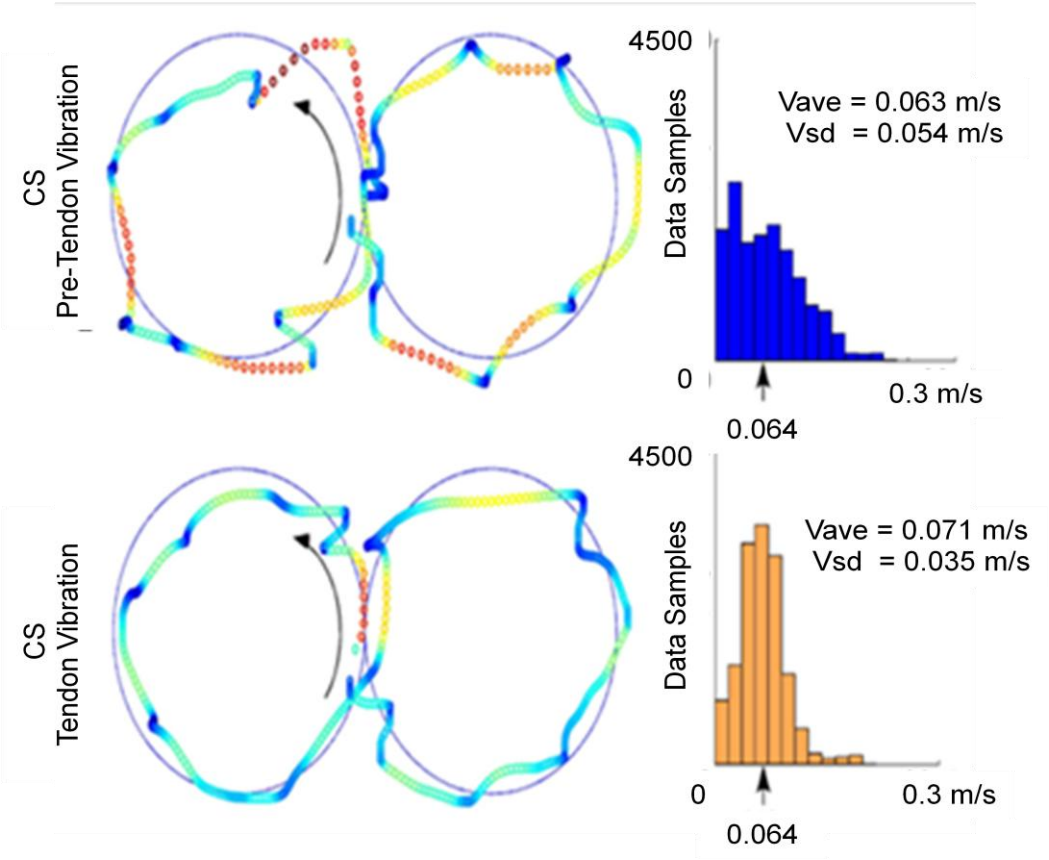

Figure 1-3. Figure eight tracking task during pre-vibration and vibration trials. Segmentation of arm movements improved. Velocity profiles are observed to become more normally distributed around the target. Adapted from Conrad, 2009.

These studies advocate that tendon vibration may be a useful tool for rehabilitative therapy because of its positive effects in stabilizing the arm and improving motor function while performing tasks.

The effects of vibration, in enhancing motor performance might be produced by a change in reflex regulation. This can be assessed by observing the effect of vibration on the stretch reflexes generated at the spinal level. Our approach involved the use of a

tendon tapper as a probe for generating reflexes to identify the effects of tendon vibration at the spinal level.

1.5 Specific aim:

The specific aim of this thesis was to understand whether the mode of action through which tendon vibration operates in enhancing motor stability involves spinal stretch reflexes. For this purpose we applied tendon vibration at the wrist and generated stretch reflexes at the biceps and triceps tendon using an electromagnetic tendon tapper. Quantifiable differences in EMG amplitudes of the stretch reflexes generated by biceps and triceps tendon taps could be suggestive of the effects of tendon vibration applied at the wrist musculature of the affected arm during motor tasks. We hypothesized that if tendon vibration improved stabilization of the arm through spinal reflex pathways, then heteronymous tendon tap reflexes would be modified by wrist vibration.

CHAPTER 2: ROLE OF TENDON VIBRATION IN MULTIJOINT REFLEX COUPLING IN THE HEMIPARETIC ARM POST STROKE

2.1 Introduction

The objective of this study was to identify the effects of tendon vibration (TV) applied to the wrist flexors on the biceps and triceps stretch reflexes, generated using a tendon tapper. Hemiparesis in the arm caused by a stroke leads to sensory dysfunction and causes reflex coupling in multiple muscles of the arm leading to uncharacteristic movements (Dewald et al., 2001). In this study, we assessed whether wrist vibration would condition the reflexes in muscles across multiple joints and we quantified the changes in the magnitude of the reflexes thus generated.

Tendon tap perturbations can elicit multijoint reflex coupling. Multijoint reflex coupling is observed in the muscles of the hemiparetic arm that hampers motor control (Sangani et al., 2007). Tendon tap perturbations of elbow flexors and extensors elicit reflex coupling in multiple muscles of the hemiparetic arm (both proximal and distal) during passive and isometric contractions (Sangani, 2008). Thus, tendon tap perturbations, similar to the perturbations provided by a reflex hammer used by a doctor to test deep tendon reflexes, are an effective way of producing multi-joint reflex coupling.

Tendon vibration can augment sensory information and hence improve motor function in the hemiparetic arm. People post-stroke can become unstable while holding

the arm at the end of a planar motion. Previous studies have shown that sensory interventions such as TV increase stability of the hand in both stable and unstable work environments (Conrad et al., 2009). TV has also been shown to improve the motor response to transcranial magnetic stimulation through activation of muscle spindles, which increase the firing rate of Ia afferents to the CNS (Steyvers et al., 2003). It increases the response of primary muscle spindle endings during isometric voluntary contractions (Burke et al., 1976a) and also during relaxed passive trials (Burke et al., 1976b). TV can thus function as a sensory intervention as it affects proximal muscles of the arm, which are not directly activated using vibration (Conrad et al., 2009).

In this study, we hypothesized that if TV improved stabilization of the arm through spinal reflex pathways in previous studies (Conrad et al., 2009), then heteronymous tendon tap reflexes would be modified by wrist vibration. Chronic stroke and neurologically intact individuals participated in this study and were tested in a relaxed state as well as during simple isometric tasks involving the shoulder, elbow and the wrist. Peak-to-peak EMG amplitudes of the reflexes were quantified and compared during the pre-vibration, vibration and post-vibration trials. The outcome of this study suggests that that the effects of vibration on arm stability as seen in previous studies (Conrad et al., 2009) likely occur in supraspinal structures, possibly reflecting a change in supraspinal sensorimotor integration mechanisms.

2.2 Research methodology and materials

This section outlines the subject sample, research design protocol and setup, the instrumentation involved and data analysis.

2.2.1 Subject sample

The subjects for this study consisted of 10 chronic stroke (CS) survivors with hemiparesis of the arm and 5 age-matched neurologically intact (NI) controls. The age range of the participating stroke survivors was 46-80 yrs (mean age 58.2 yrs), of which there were 4 female and 6 male. The age range of the neurologically intact controls was 54-67 yrs (mean age 60.2), of which there were 2 female and 3 male. The inclusion criteria for the stroke subjects was that the occurrence of stroke > 6 months, at least 21 years of age and hemiparesis in the upper extremity as a result of stroke. Stroke subject exclusion criteria consisted of an occurrence of contractures in the upper limb, other neuromuscular disorders, use of botulinum toxin to reduce spasticity and failure to give informed consent. The study was conducted in agreement with the Helsinki declaration, was approved by the Marquette University's Institutional Review Board (IRB) and was performed after obtaining informed consent from all the participants.

Table 2-1. Subject Information

Subject	**Gender**	**Age**	**Arm Tested** [#]	**Fugl-Meyer** [£]
1s	Female	59	Left	50
2s	Male	57	Left	26
3s	Male	46	Left	-n/a-
4s	Female	63	Left	62
5s	Female	80	Left	38
6s	Male	46	Right	56
7s	Female	61	Right	50
8s	Male	60	Right	57
9s	Male	60	Right	-n/a-
10s	Male	48	Right	31
1c	Male	64	Left	-
2c	Male	66	Left	-
3c	Female	56	Right	-
4c	Female	56	Right	-
5c	Male	59	Right	-

s - Chronic stroke survivors with hemiparesis of the arm
c- Neurological intact controls
- Arm Tested

- Affected arm was tested for chronic stroke subjects
- Dominant arm was tested in neurologically intact controls

£ - Based on Fugl-Meyer Scale (0 – 66; (Fugl-Meyer et al., 1975)

2.2.2 Tendon tapper

The tendon tapper consisted of a linear motor (P Series, LinMot Inc, Delavan, WI). A linear variable differential transformer (LVDT) (Accusens Series 2000 DC-EC, Measurement Specialists, Inc., Hampton, VA) was mechanically coupled to the motor to measure the linear displacement during the tendon taps (Figure 2-1B). The tendon tapper was programmed using LabVIEW (National Instruments, Austin TX). Square waveforms were generated using a function generator (within LabVIEW) at a frequency of 1Hz and amplitude of 1V. The square wave pulse width was varied between 50% – 60% duty cycle (between 50ms – 60ms) across subjects to acquire a clear persistent reflex response at the biceps and triceps tendon. The starting distance between the tip of the tapper and the tendon was maintained at 6 cm (+/- 1 cm) across subjects. The magnitude of the tapper i.e. the distance travelled by the linear motor's shaft to depress the tendon was 15cm. The biceps tendon was depressed with a rubber bumper to obtain reproducible perturbations since the biceps are known to have longer tendons than the triceps (Thilmann et al., 1990).

2.2.3 Tendon vibrator

The tendon vibrator was applied to the wrist flexors during the experimental sessions. The tendon vibrator was placed in Teflon tubing with an outer diameter of 1.5 cm and was applied to the wrist flexor tendons. The vibrator consisted of a semicircular unbalanced mass (4.8776 g and 1.2 cm diameter) attached to a shaft of a motor (1319 TO12SR, Faulhaber Inc., Clearwater, FL) with an encoder (model IE2-400) (Figure 2-

1C). The center of mass of a 2D semicircular unbalanced (eccentric) weight of radius R was at a distance 4R/ (3*pi) from the shaft. Therefore, the center of mass of the eccentric weight (3D semicircle) would be the same (X, Y) coordinate as the 2D semicircle, but the z-coordinate will in the middle of the mass. So the moment arm was the distance from the shaft to the center of mass of the eccentric weight which was 4R/(3*pi) or 0.254 cm. The tendon vibrator was programmed using LabVIEW (National Instruments, Austin TX) through its controller (MCDC 3006S, Faulhaber Inc, Clearwater, FL). The vibrator was programmed to vibrate at a frequency of 90 Hz. This frequency lies in the 70-100 Hz frequency range in which muscle spindles have been found to be most sensitive (Roll et al., 1989).

2.2.4 EMG, Torque and LVDT recordings

Surface EMG recordings were made from 8 muscles namely: pectoralis major (Pect), anterior deltoid (AD), posterior deltoid (PD), biceps (Bic), triceps (Tri), wrist flexors (Wflex), wrist extensors (Wext) and brachioradialis (BRD). The skin was prepared using alcohol and the electrodes (Vermed Medical Inc., Bellows Falls, VT) were placed on the muscle bellies. The pre-amplifiers were taped on the muscles between the EMG electrodes to reduce any motion artifacts that could be generated when the tip of the tapper hit the tendon. The EMG signals were pre-amplified, and then amplified (Bortec AMT-8, Bortec Biomedical Ltd., Calgary, Canada) at 10,000X, low-pass filtered at 500 Hz and then digitized with a SCB-100 acquisition board (National Instruments, Austin TX) at a sampling rate of 1000 samples/s. The force/torque data from two six-axis load cells and position signals obtained from the LVDT were low pass filtered at 250 Hz

with a custom hardware circuit and were also digitized at 1000 samples/s, similar to the EMG signals.

2.2.5 Experimental Setup

The subjects were seated on the chair of a Biodex System 3 (Biodex Medical Systems, Inc., Shirley, NY). A base consisting of two load cells (JR3, Inc., Woodland, California) was attached to the pedestal of the motor. The affected arm was placed on the base such that the elbow and wrist were positioned on the two (six axis) load cells which measured the force/torque in the x, y and z directions. The hand was secured onto a wooden ball mounted on a Panavise base (PanaVise Products Inv.Reno, NV) mounted on the wrist load cell (Figure 2-1A). The arm was fastened tightly onto the base with Velcro straps and foam strips. A shoulder belt was used to strap the trunk of the subject to avoid upper body movement and any possible changes in body position.

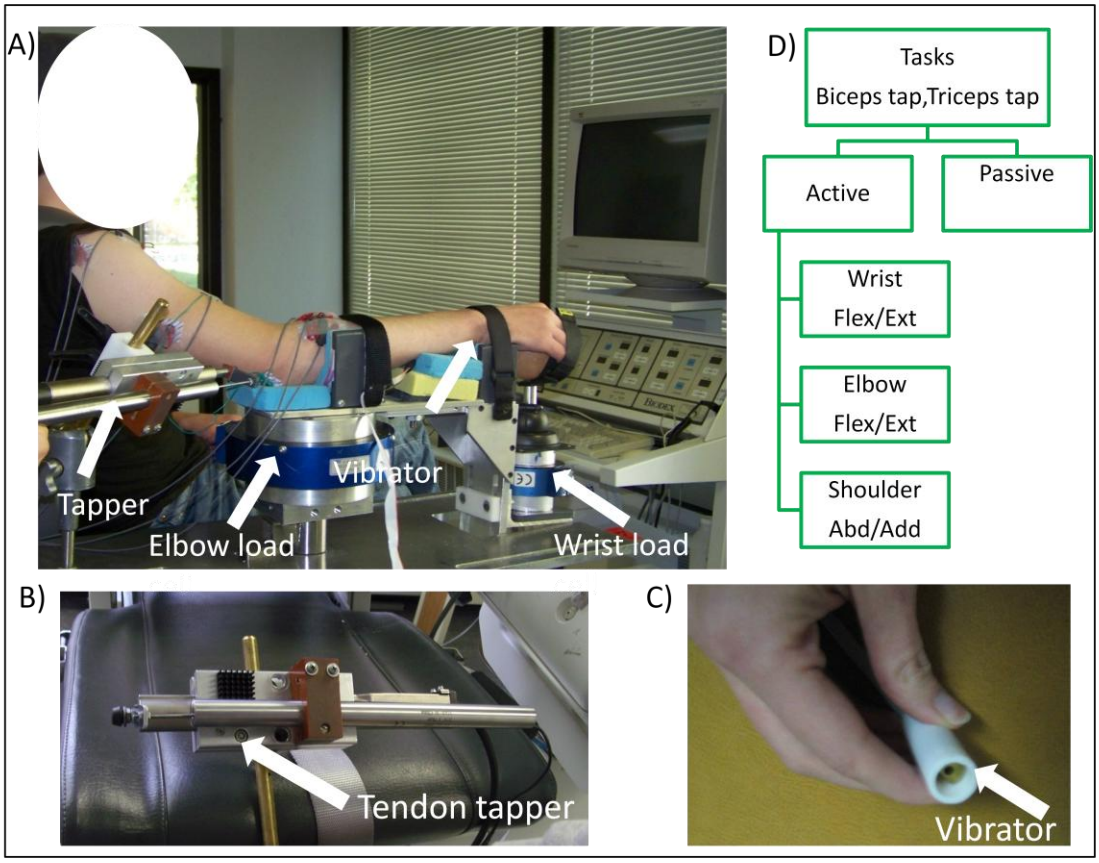

Figure 2-1. Experimental Set-up. A) A subject seated on a Biodex chair. The arm was strapped to the chair and the hand rested on a wooden ball placed on the wrist load cell. Load cells placed under the elbow and wrist measured the force/torque in the x, y and z directions. Electromyograms (EMGs) were recorded from eight muscles of the arm. **B)** An electromagnetic hammer (tendon tapper) was used to generate stretch reflexes at the biceps/triceps tendon. A linear variable differential transformer (LVDT) was mechanically coupled to the linear motor to keep track of its position. **C)** A tendon vibrator was applied to the wrist flexor tendons. It consisted of a DC-motor with encoder with an eccentric mass attached to the motor's shaft, which rotated to produce vibration. **D)** Research protocol. The subjects performed three passive (relaxed) and six active tasks, namely: wrist flexion/extension, elbow flexion/extension and shoulder abduction/adduction. The tasks were in a randomized order and each condition was tested with 30 taps each.

2.2.6 Research protocol

The experimental paradigm was divided into two main parts. In the first part, two blocks of isometric maximal voluntary contractions (MVCs) were performed in wrist flexion/extension, shoulder abduction/adduction, and elbow flexion/extension. Visual feedback of the torque they were activating to perform these tasks was provided on a computer screen. The subjects were asked to sustain their maximum torque for a period of 4-5 seconds while performing these tasks. The MVC data was analyzed using MATLAB (The Mathworks, Natick, MA). The maximum torque generated at each joint in these tasks was used in the second part of the experiment.

The second part of the experiment consisted of 6 active tasks and 3 relaxed conditions. Tendon taps under relaxed conditions were measured in the beginning, middle and at the end of the experimental protocol. The active/isometric tasks consisted of the same task types as the MVCs and were randomized and placed in between the passive tasks (Figure 2-1D). The relaxed trials were performed to check the effects of vibration on the reflexes generated by the tapper when the subject was relaxed. Relaxed conditions were tested to delineate the effects of vibration on the reflexes in the absence of a volitional drive, since the subjects were not performing any tasks. While performing active tasks, the subjects were asked to maintain their primary force/torque between 10% and 30% as displayed to them on a computer screen by two parallel lines. There was a 10ms delay in the torque which was being displayed. Once the subjects could maintain their torque corresponding to the tasks they were performing, tendon tap perturbations were initiated. Each of these tests consisted of three sets of 10 tendon taps. The first set

of 10 taps was with the tapper and no TV, to serve as a baseline recording. The second set of 10 taps was with the combined tapper and vibration to observe the effects of vibration. The third set of 10 taps had only tendon tap perturbations (no TV) to identify any aftereffects of vibration. The vibration was initiated one second prior to the start of the tapper and terminated one second after the tendon tap perturbations. The total duration of the experiment was approximately 2 hours.

2.2.7 Data analysis

The data acquired when the subjects performed MVCs was analyzed to be used during the second part of the experiment (i.e. when the subjects performed active tasks). The force/torque data acquired for each of the tasks performed during the MVC (that is, wrist flexion/extension, shoulder abduction/adduction and elbow flexion/extension) was filtered using a zero-phase, sixth order Butterworth filter. The data were low pass filtered at a cut off frequency of 5 Hz. The baseline was found for the first 1000 points and subtracted from the filtered data to remove the DC offset. The maximum value of the MVC was computed. 10% and 30% of this maximum value was shown to the subjects by a pair of parallel lines on the screen while they performed the active tasks.

The tapper position data, acquired from the LVDT, was used to identify the reflexes generated by the tendon tapper. For each of the active and passive tasks, a threshold value from the LVDT's position data was identified for the pre-vibration, vibration and post-vibration trials. This threshold value was used to identify the start and end points of each tendon tap perturbation. The reflex response in the muscles was identified as the peak-to-peak amplitude, calculated as the difference between the highest

positive and lowest negative peak in the EMG data in the window 100ms after the tapper hit the tendon (Figure 2-2). The EMG data was zero-phase filtered using an 8^{th} order Butterworth filter. The data were bandpass filtered between 10 and 250 Hz and then notch filtered for removing electrical noise at 60 and 120 Hz and noise caused due to vibration at 90 Hz. To guarantee the accuracy of the maximum and the minimum peak, each reflex was examined by naked eye to detect artifacts. A few criteria were used for identifying the reflexes manually. One approach was based on identifying the timing of muscle reflex responses, which have been determined previously based on each muscle's reflex latencies in response to tendon tap perturbations (Sangani, 2008). Artifacts caused by tapper movements were also identified to distinguish between the tapper movement artifacts and the muscle reflex response. Since movement artifacts are fairly constant and the reflex responses vary with each tap due to changes in excitability of the motoneuron pool, this approach involved superimposing the signals for each tap, aligned by the tendon tap commands, to identify the reflex responses. The data for a subject was discarded only if a reflex response in the muscle being tapped was not observed by using the approaches mentioned above to identify reflexes manually.

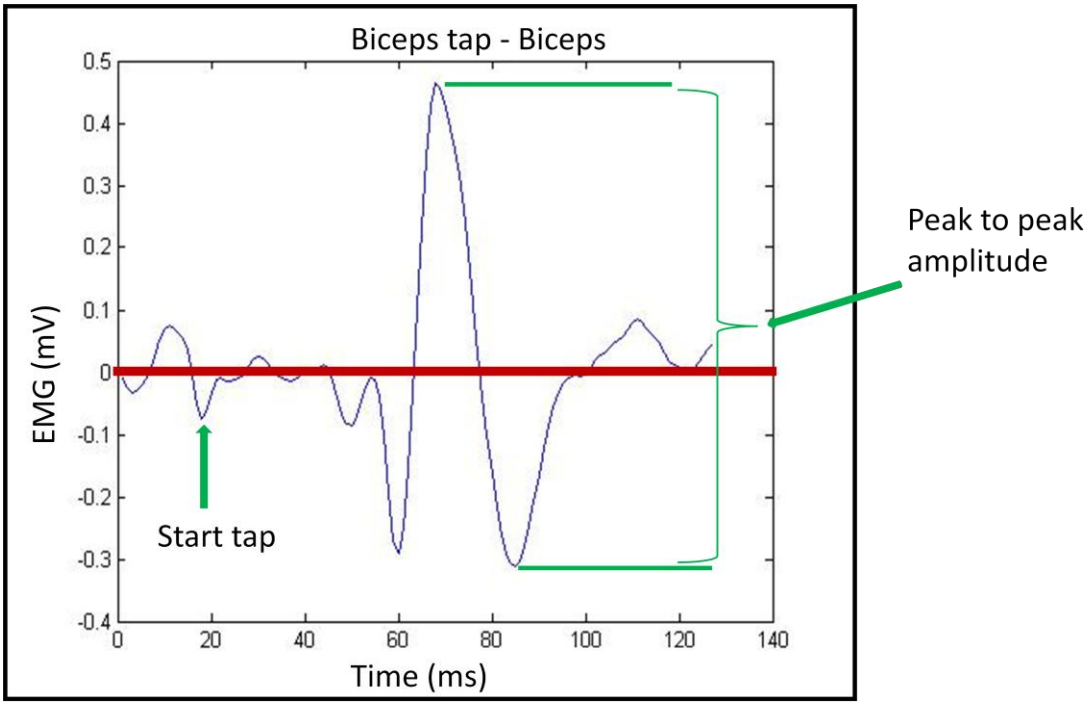

Figure 2-2. Data Analysis. Figure depicts an EMG reflex response in the biceps muscle (homonymous muscle) in response to biceps tendon tap perturbation. The highest positive peak and the lowest negative peak points of the signals were identified and the difference was computed to yield the peak-to-peak amplitude of the tendon tap reflexes.

After the peak-to-peak amplitudes were found for each subject and for each task (active and relaxed) the mean of the peak-to-peak EMG amplitudes was calculated across all trials (pre-vibration, vibration and post-vibration). This mean value (a single value computed across all trials) was then used to normalize the peak-to-peak reflexes for each muscle, for each trial. The normalized peak-to-peak data for each subject were then averaged across each muscle for each of the pre-vibration, vibration and post-vibration trials. A grand total average was then computed across subjects for each trial type. Flowcharts showing the normalization and averaging of the peak-to-peak reflexes of the muscles for active tasks and the relaxed condition are shown in Figures 2-3 and 2-4 respectively.

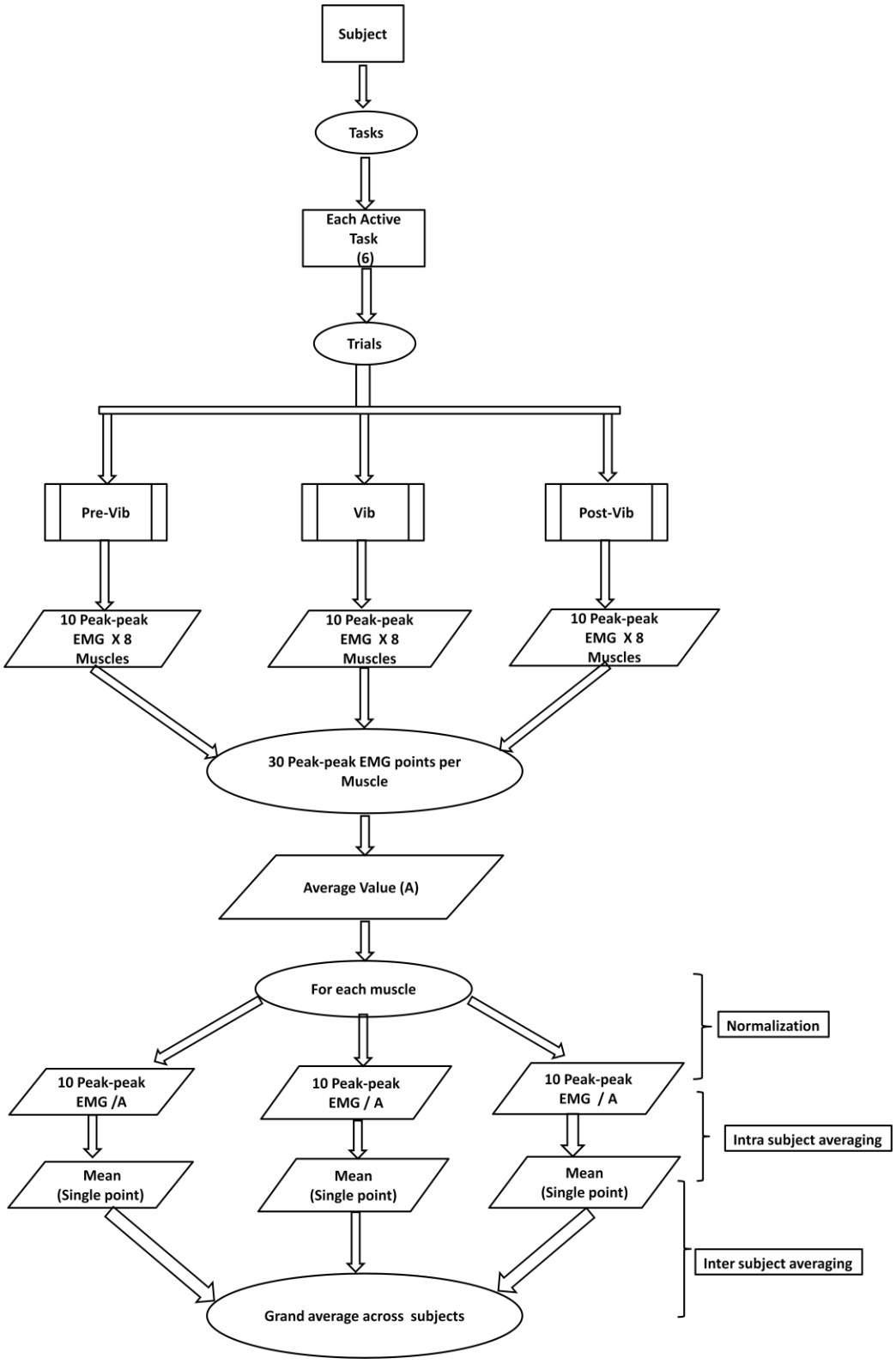

Figure 2-3. Active Data. Flowchart showing the normalization and averaging of peak-to-peak reflexes of the pre-vibration, vibration and post-vibration trials within the active tasks of each subject.

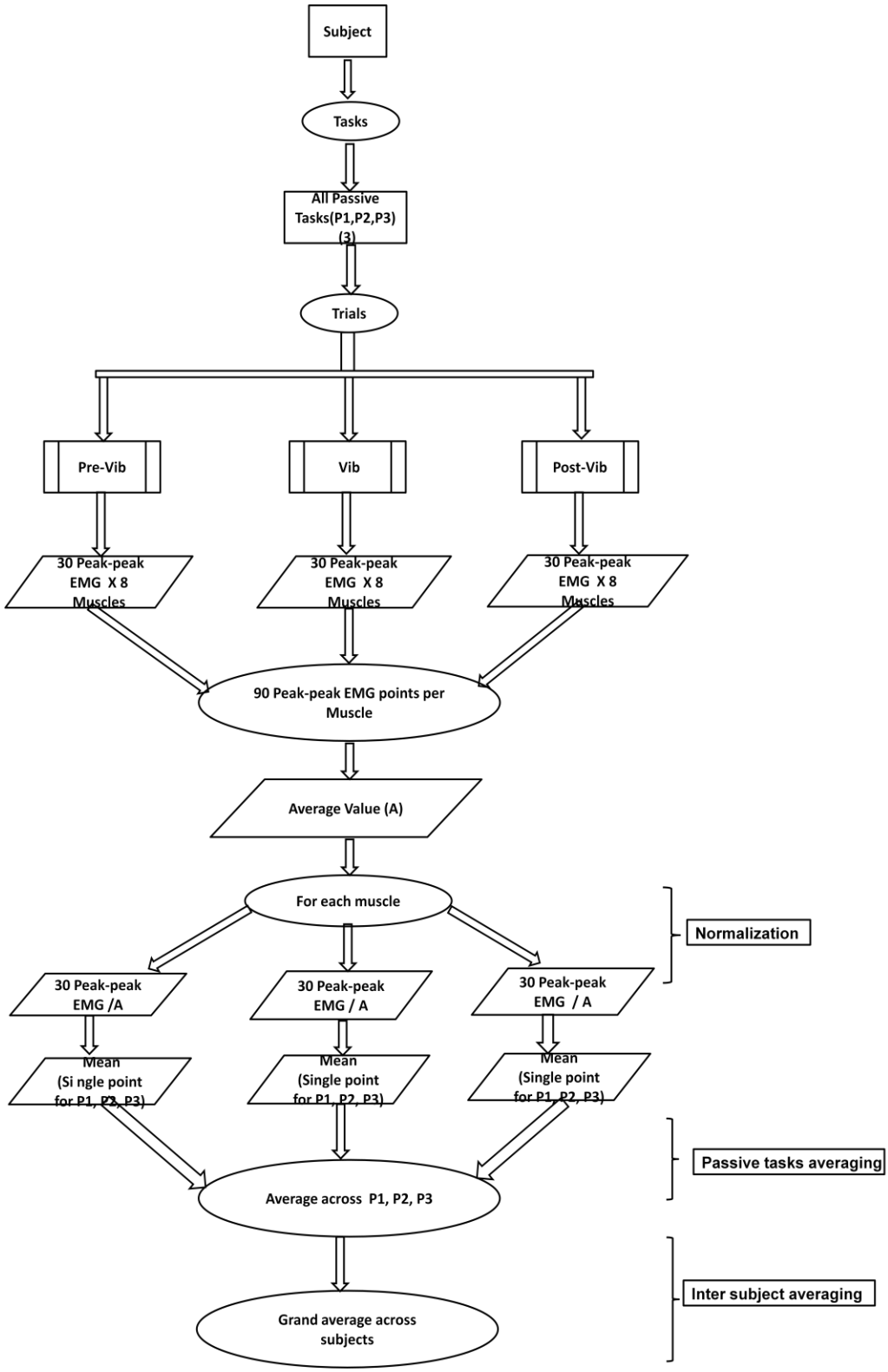

Figure 2-4. Passive Data. Flowchart showing the normalization and averaging of peak to-peak reflexes of the pre-vibration, vibration and post-vibration trials within the relaxed condition of each subject.

2.2.8 Statistical Analysis

Statistical analysis was performed to compare the peak-to-peak EMG amplitudes of the reflexes between the pre-vibration, vibration and post-vibration trials in each of the active and relaxed tasks. Univariate ANOVAs ($\alpha = 0.05$) were run for each muscle in a task (both active and relaxed) between subjects for all the trials (pre-vibration, vibration and post-vibration). The trials were considered to be fixed factors and the subjects as random factors. If significant results were obtained, a Fisher's LSD test was run to compare the means and establish differences between the pre-vibration, vibration and post-vibration trials.

2.3 Results

No significant differences were found in the elbow tendon tap reflexes with wrist tendon vibration. Peak-to-peak amplitude of the reflexes showed negligible differences during vibration and post-vibration trials as compared to the pre-vibration trials. Note that in the analysis for triceps tendon tap perturbations in stroke subjects, the results for only 9 subjects have been used. There was an absence of reflex responses in one subject's homonymous muscle (triceps) during tendon tap perturbations at the triceps.

2.3.1 Relaxed conditions

The normalized and averaged peak-to-peak EMG reflex amplitudes across muscles during relaxed conditions in stroke subjects for both the biceps and triceps tendon taps are shown in Figure 2-5. The relaxed tasks were performed to observe the

effects of tendon vibration on spinal reflexes in the absence of volitional drive. As seen in A.7, A.8, A.9, A.16, A.17, A.18, during biceps and triceps tendon tap no significant differences in reflex amplitudes were observed during the vibration and post-vibration trials as compared to the pre-vibration trials in both the homonymous and heteronymous muscles of the arm. Moreover, the result held true in NI subjects under the same condition (Figure 2-6).

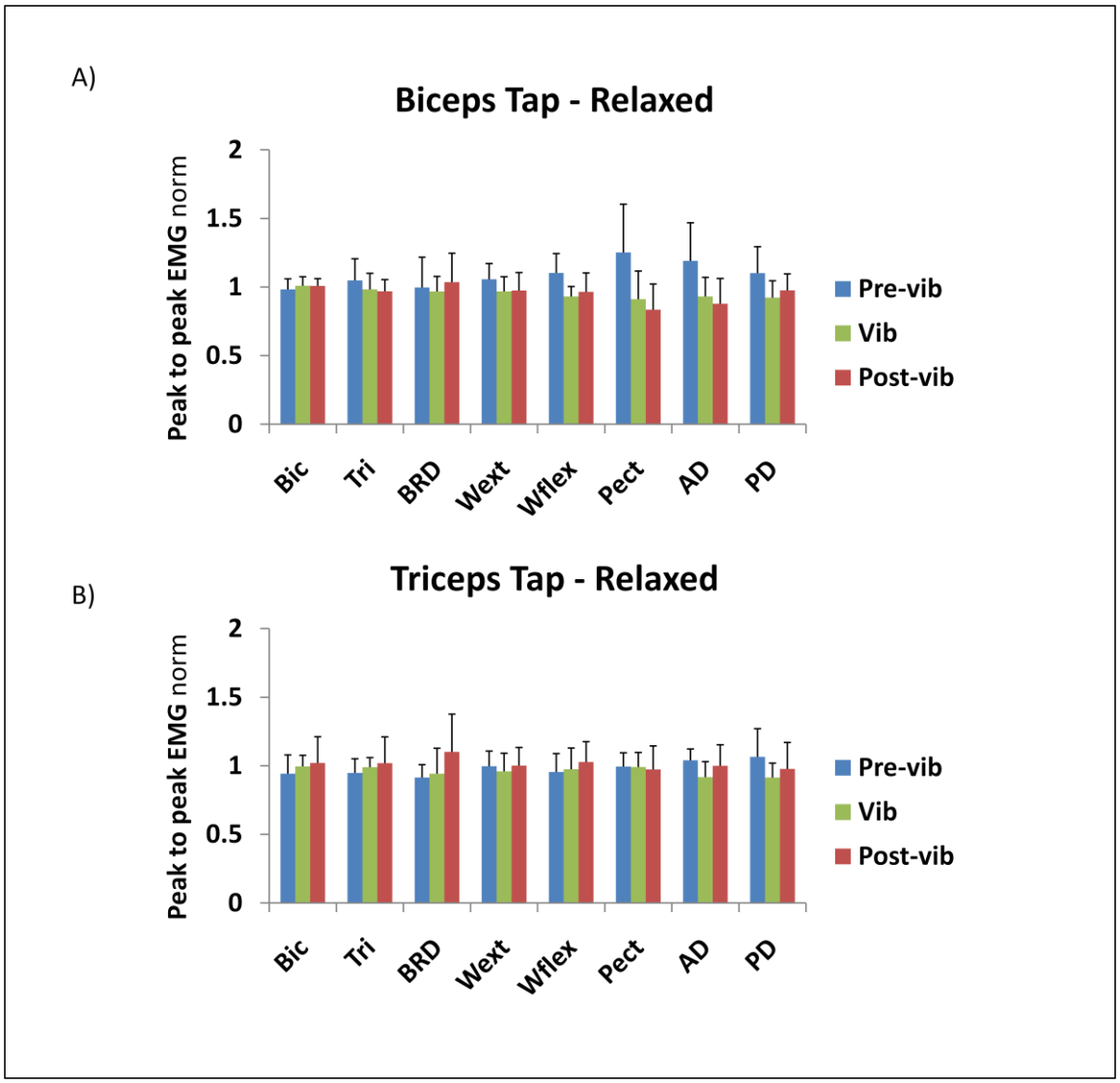

Figure 2-5. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during relaxed conditions in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during relaxed conditions across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during A) biceps and B) triceps tendon tap.

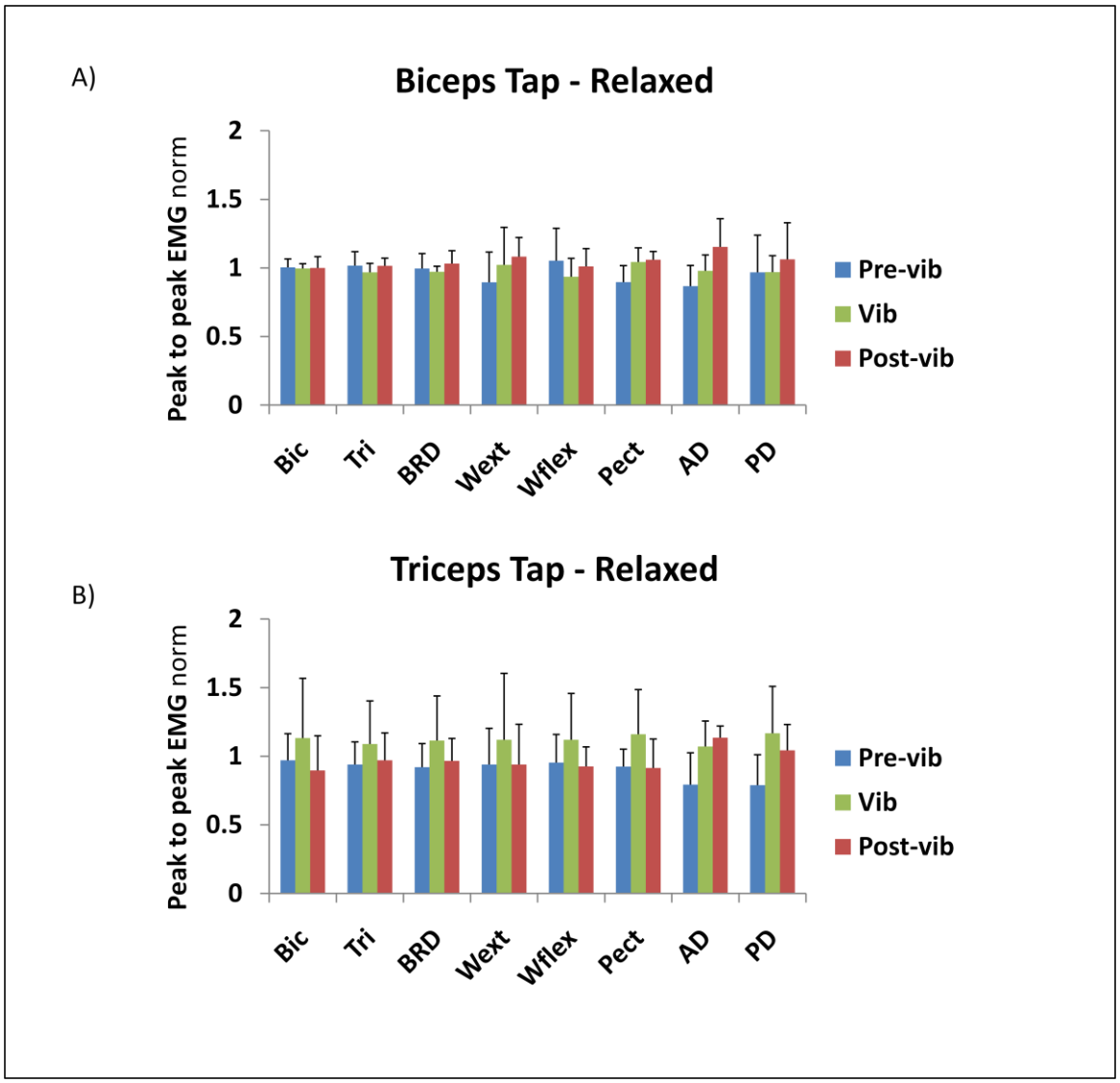

Figure 2-6. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during relaxed conditions in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during relaxed conditions across muscles of the arm in control subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

2.3.2 Active tasks

The normalized and averaged peak-peak EMG reflex amplitudes across active tasks for stroke and control subjects are described in this section. The active tasks involving different muscles of the arm were isometric. These tasks were performed to observe the effects of TV on volitional drive by augmenting the Ia sensory afferents while tapping the biceps and triceps tendons.

2.3.2.1 Elbow flexion/extension tasks

The effects of biceps and triceps tendon tap perturbations on the normalized and averaged peak-to-peak EMG reflex amplitudes of the elbow during isometric tasks (flexion and extension) in stroke subjects are shown in Figure 2-7 and 2-8. No significant difference was observed in the reflex amplitudes of the muscles between the pre-vibration, vibration and post-vibration trials during elbow flexion and extension tasks with vibration (A.1, A.2, A.10 and A.11). In control subjects, vibration also did not cause any effect on the peak-to-peak reflex amplitudes in the isometric tasks involving the elbow across muscles of the arm (Figure 2-9 and 2-10). There was no significant difference between the pre-vibration, vibration and post-vibration trials during both the elbow flexion and extension tasks (A.19, A.20, A.28 and A.29).

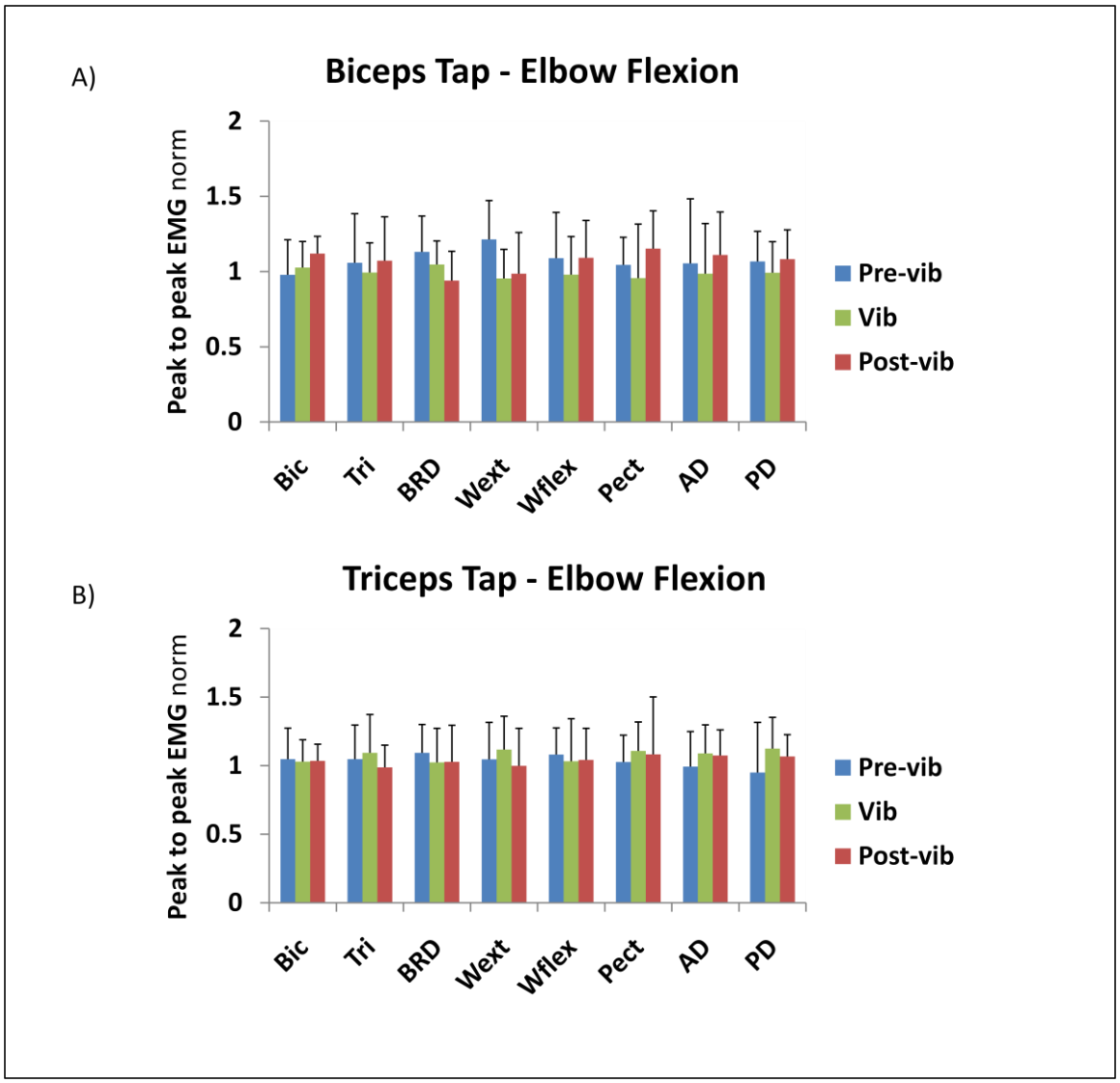

Figure 2-7. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during elbow flexion task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during elbow flexion (A, B) across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

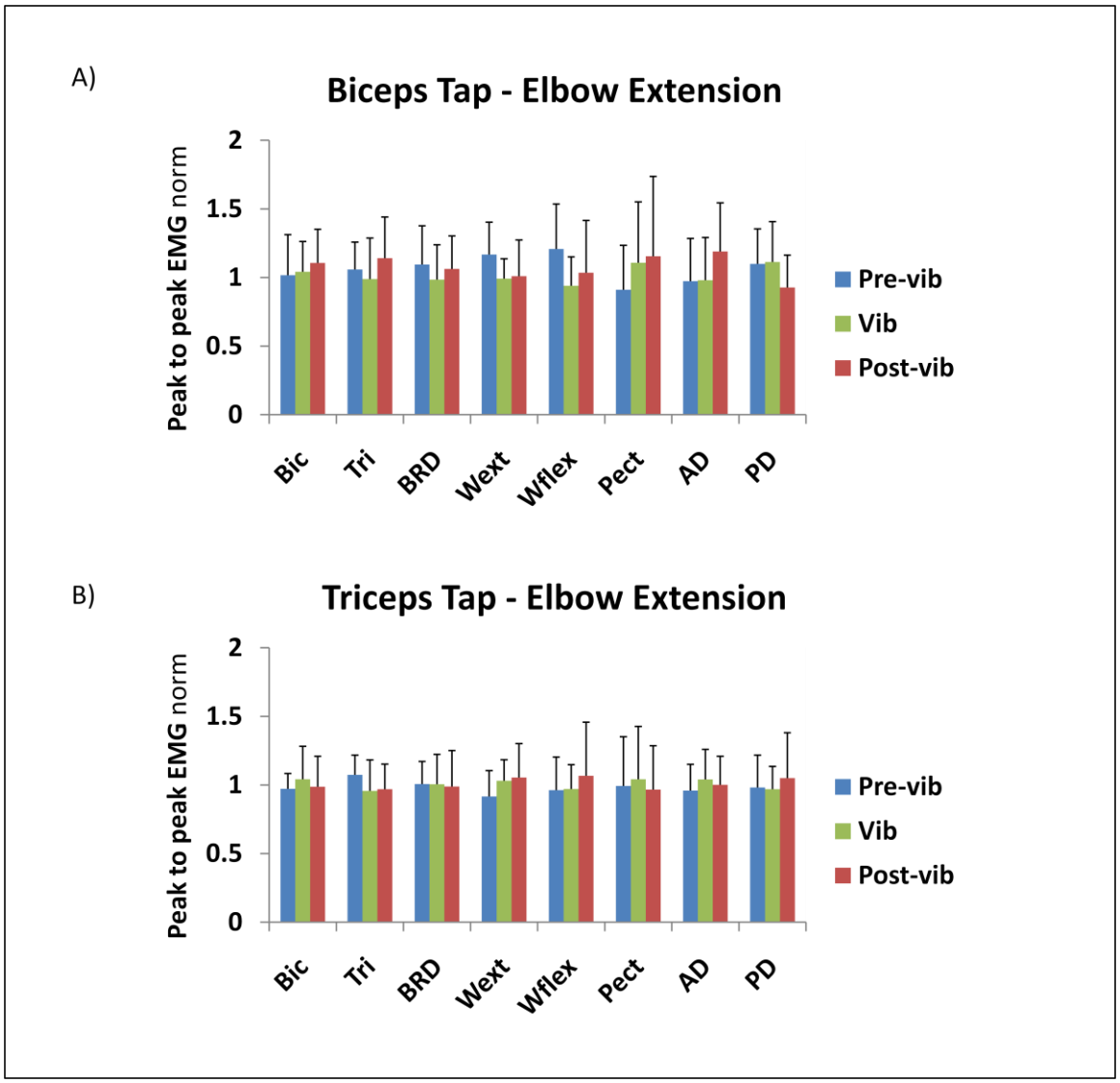

Figure 2-8. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during elbow extension task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during elbow extension (A, B) across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

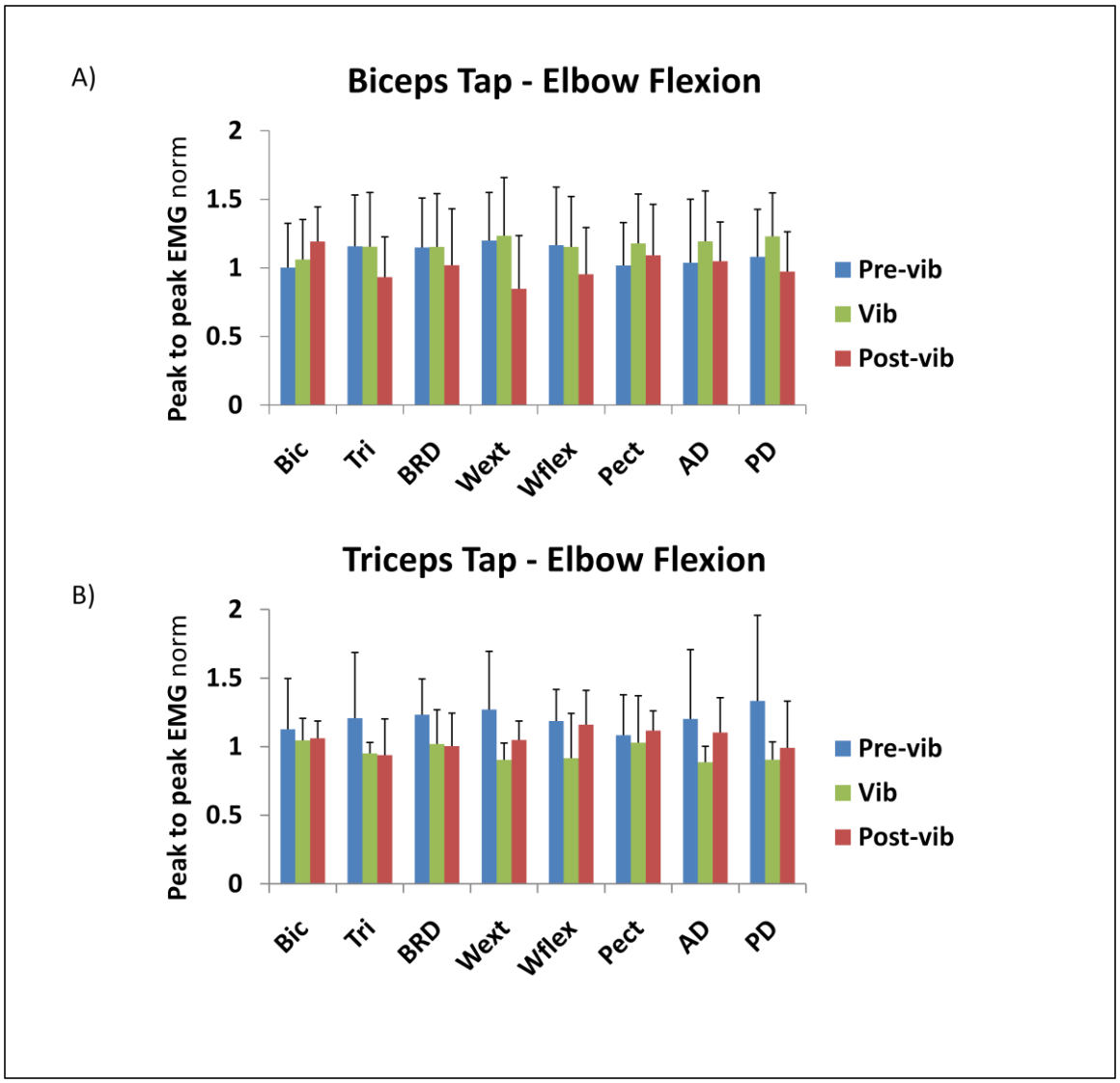

Figure 2-9. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during elbow flexion task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during elbow flexion (A, B) across muscles of the arm in control subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

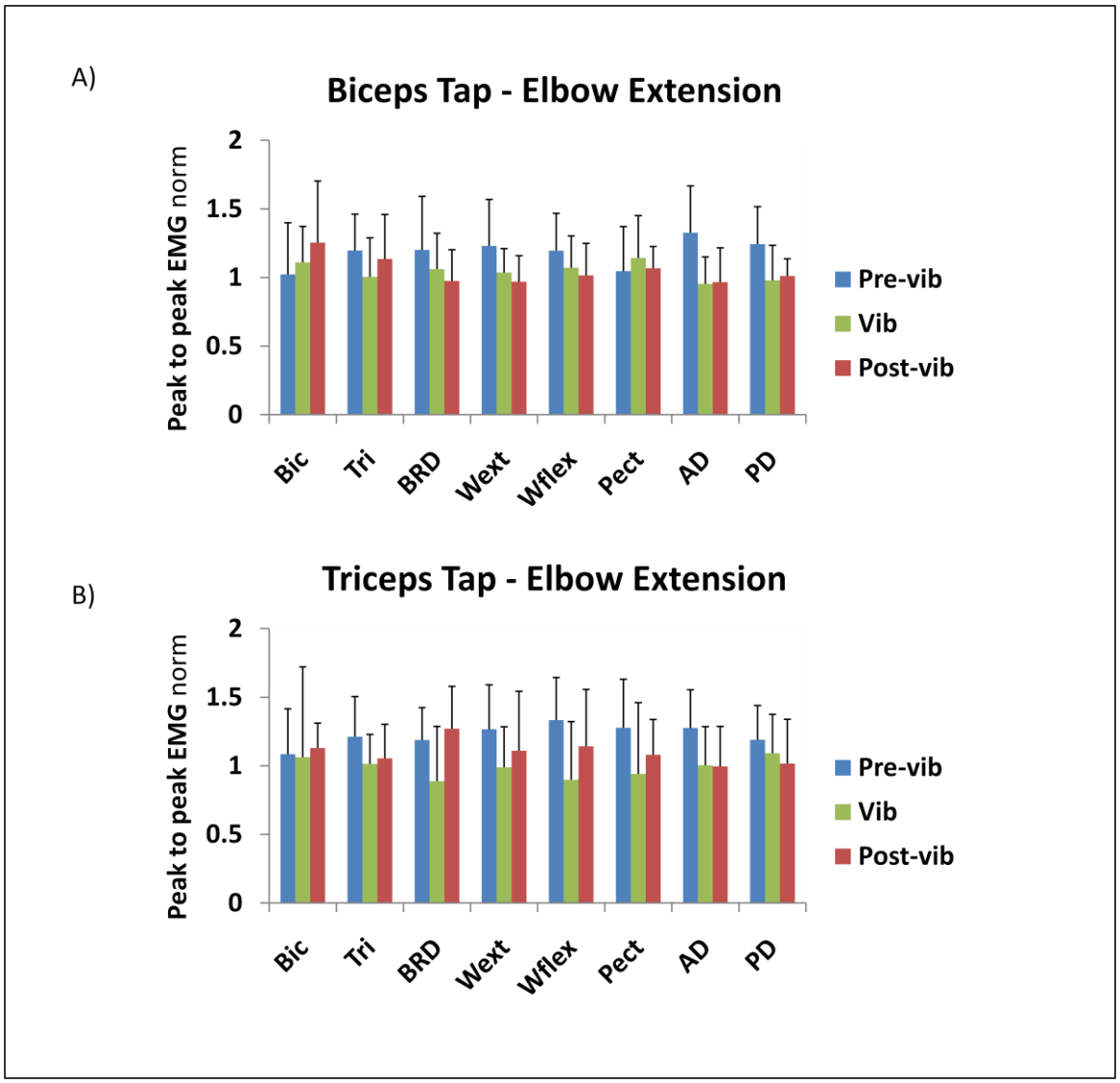

Figure 2-10. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during elbow extension task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during elbow extension (A, B) across muscles of the arm in control subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

2.3.2.2 Wrist flexion/extension tasks

Normalized and averaged peak-to-peak reflex EMG data during active tasks involving the wrist are shown in Figure 2-11, 2-12, 2-13 and 2-14. No significant differences were observed in the peak to magnitude of reflexes in the muscles with TV in the wrist flexion task during the biceps and triceps tendon tap (A.4 and A.13). Additionally, in the wrist extension task (Figure 2-11) no significant differences were observed in the reflex amplitudes during the periods of vibration and post-vibration trials as compared to the pre-vibration trials (A.3 and A.12). In control subjects, Figure 2-12 and 2-13, vibration also did not result in a significant difference in the peak-to-peak reflex amplitudes across the muscles of the arm (A.21, A.22, A.30 and A.31).

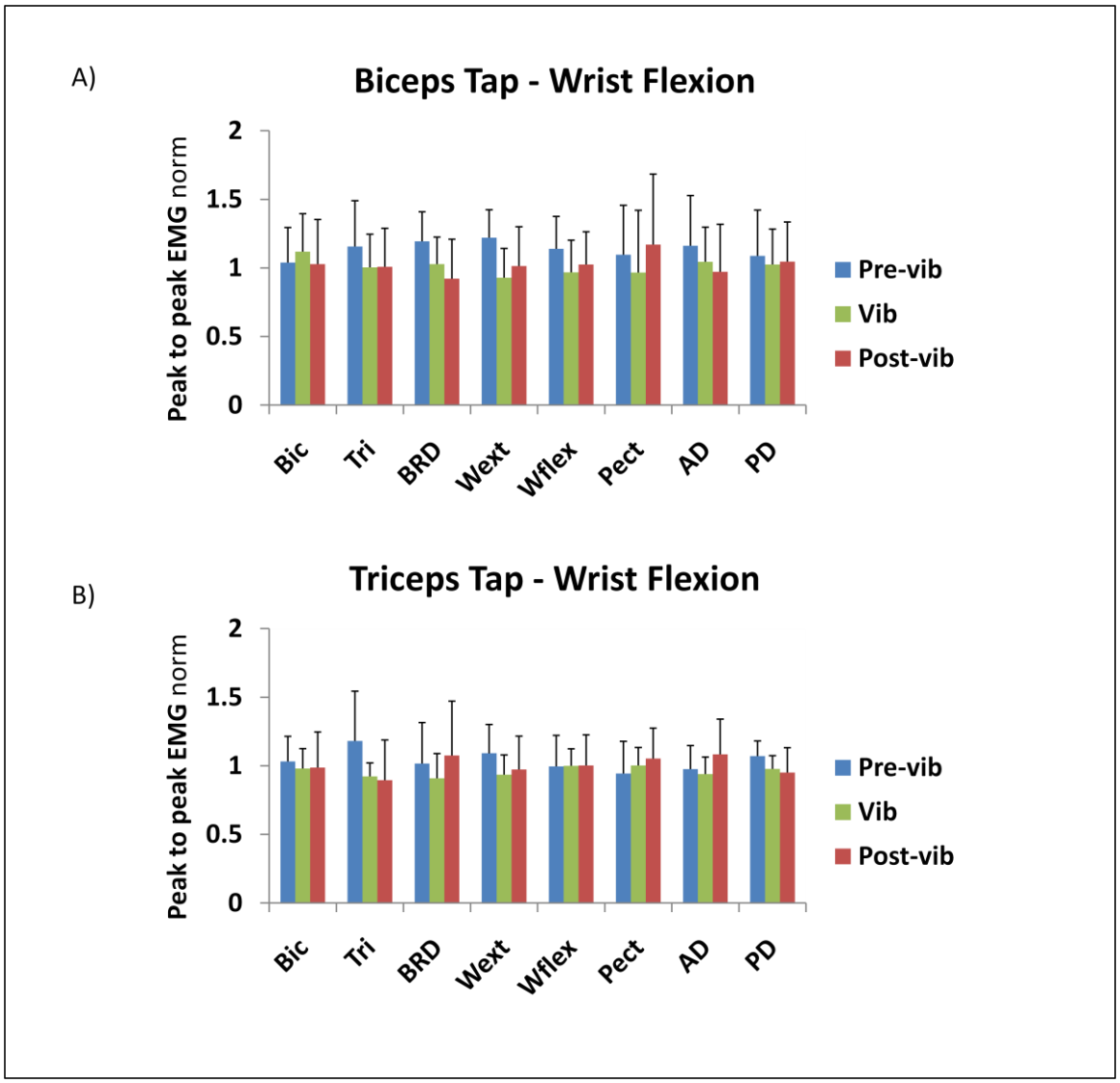

Figure 2-11. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during wrist flexion task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during wrist flexion across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

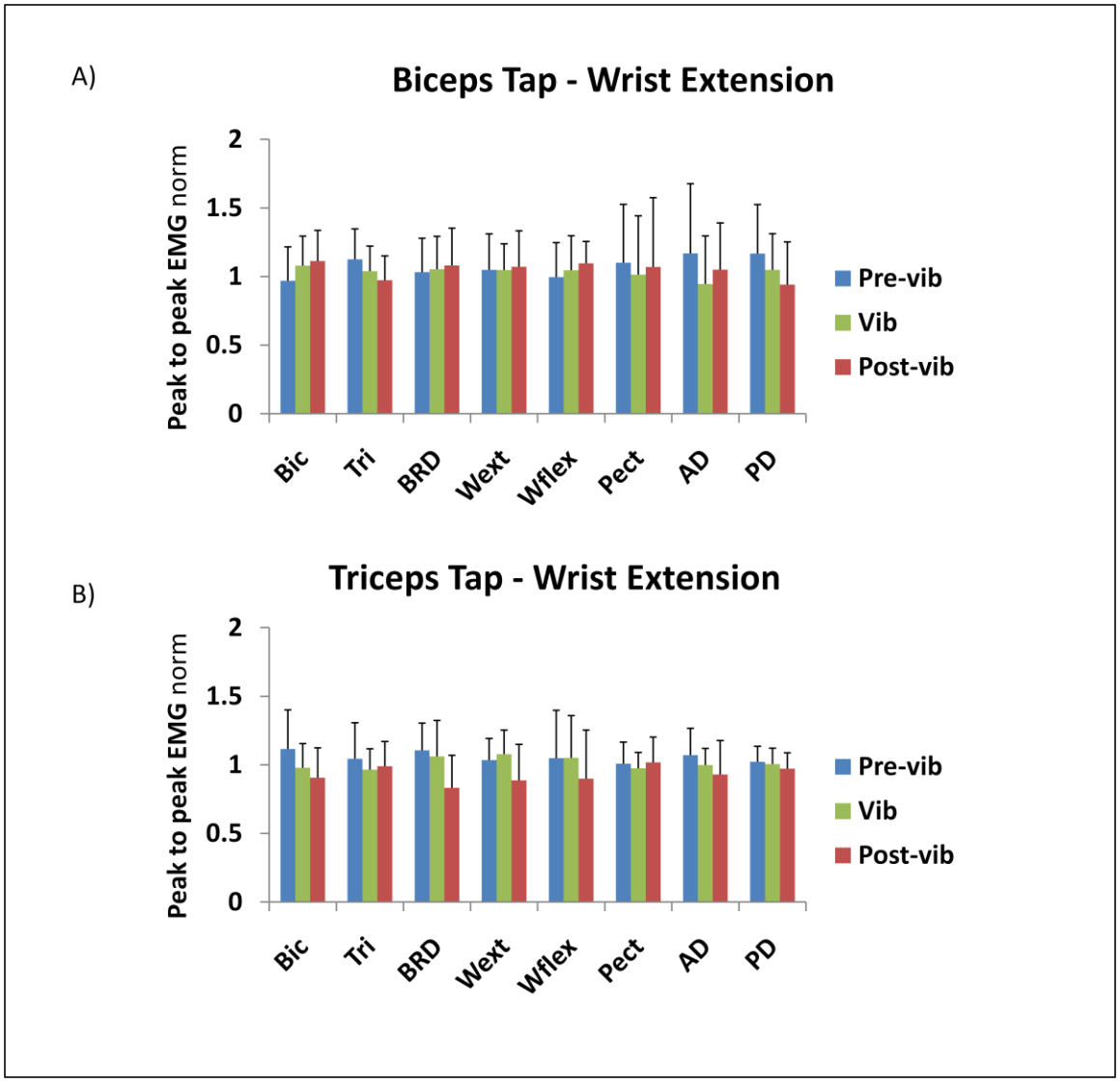

Figure 2-12. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during wrist extension task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during wrist extension across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

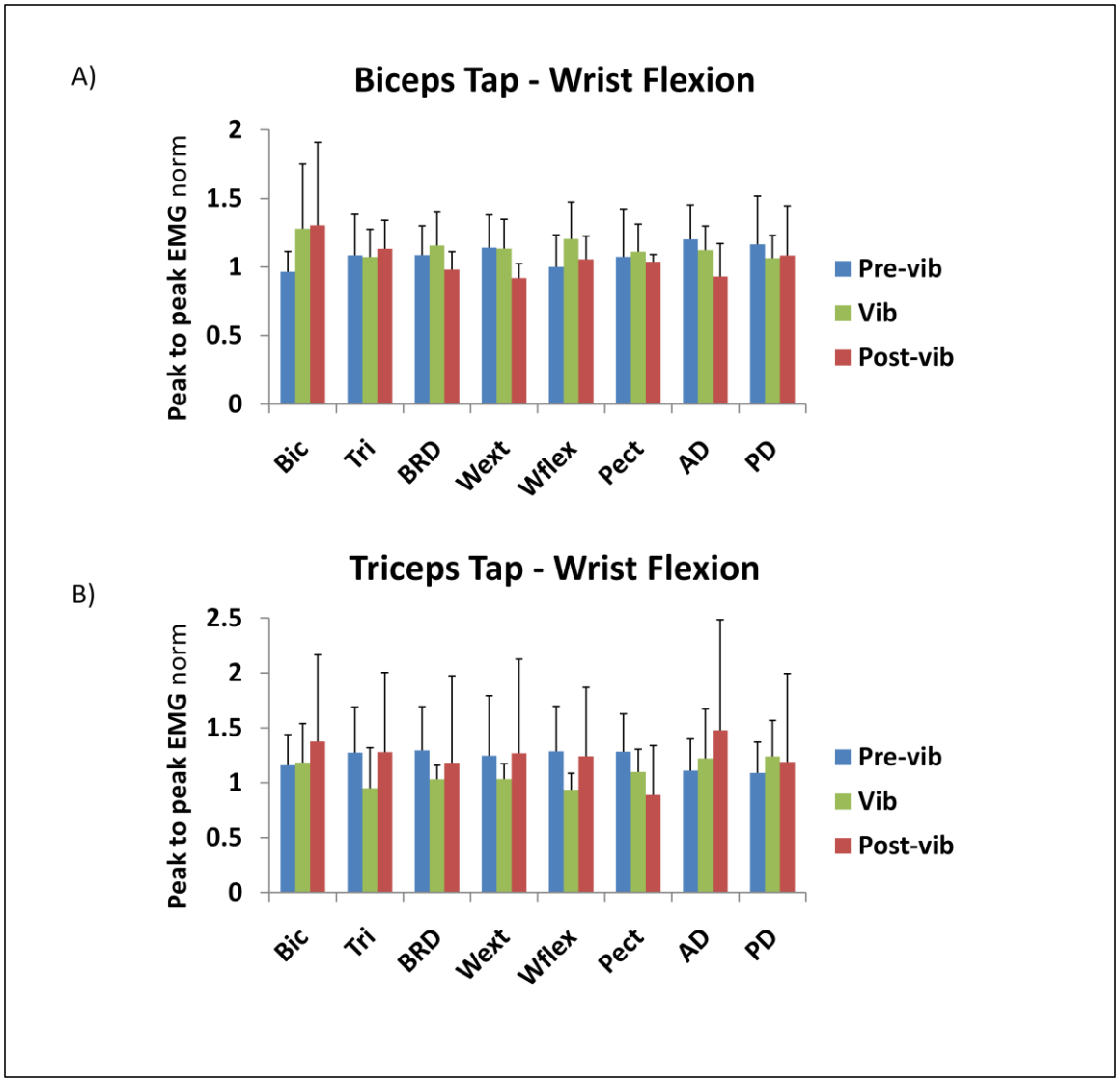

Figure 2-13. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during wrist flexion task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during wrist flexion across muscles of the arm in control subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

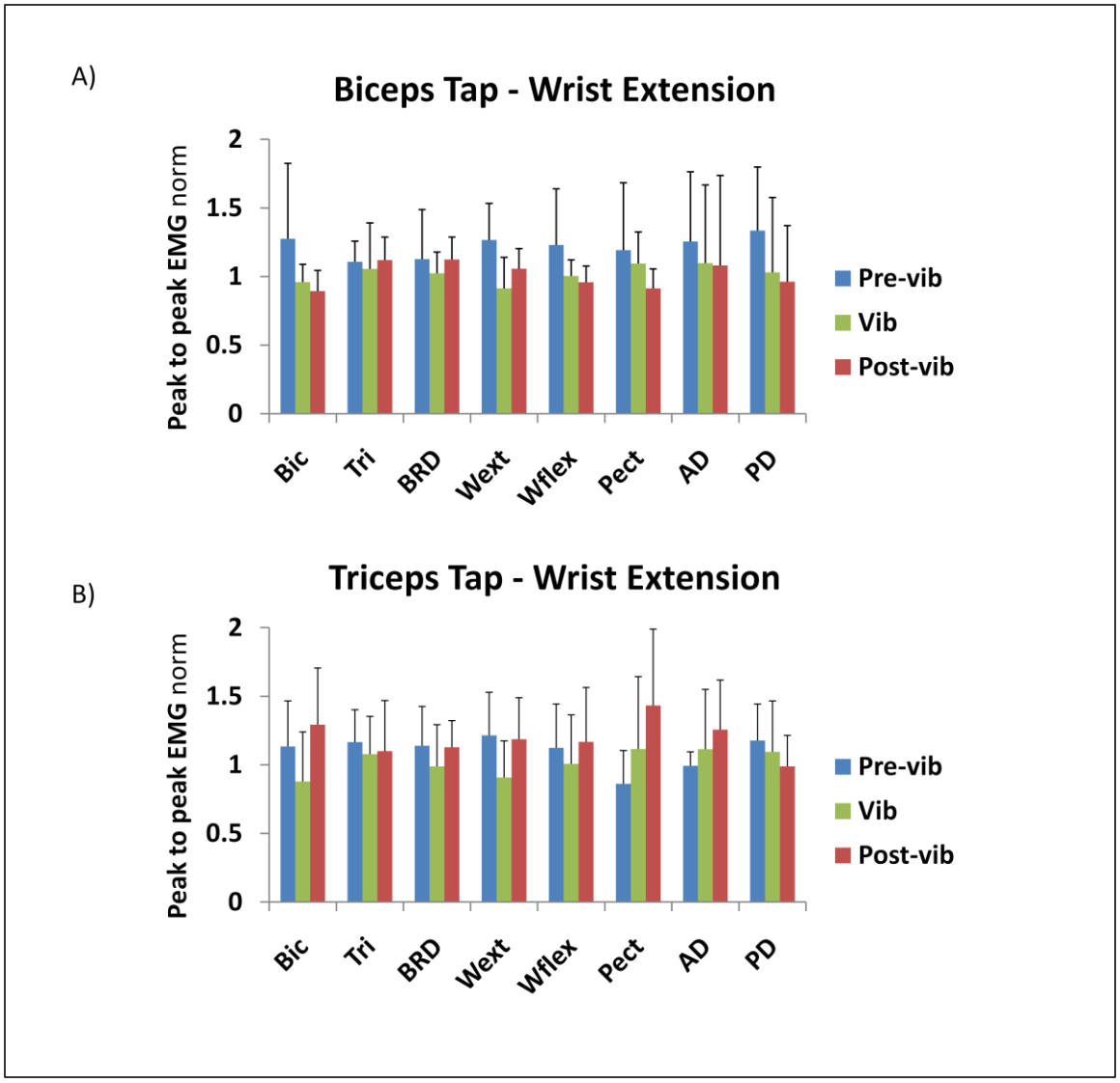

Figure 2-14. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during wrist extension task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during wrist extension across muscles of the arm in control subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

2.3.2.3 Shoulder abduction/adduction tasks

Active tasks involving the shoulder during both the biceps and triceps tap for both stroke and control subjects are shown in Figure 2-15, 2-16 and 2-17, 2-18 respectively. In stroke subjects, Figure 2-15, while tapping the biceps and triceps tendon during the shoulder abduction task, vibration did not result in any significant difference in the reflex amplitudes (A.5 and A.14). In NI controls, Figure 2-17, vibration also did not cause a significant effect on the reflex amplitudes of the muscles as compared to the pre-vibration trials in the shoulder abduction task (A.23 and A.32).

In stroke subjects, while tapping the biceps tendon in the shoulder adduction task (Figure 2-16 A), the peak-to-peak reflex amplitude of the wrist extensors muscles of the vibration trial was significantly higher as compared to the pre-vibration trial as seen in the post-hoc Fisher's LSD test ($p = 0.025$) (A.6). During the triceps tendon tap, in the shoulder adduction task (Figure 2-16 B), the pre-vibration values in the brachioradialis muscle were significantly higher as compared to the vibration trials as observed in the post-hoc tests ($p=0.010$) (A.15). Additionally, by running multiple ANOVAs and considering statistical significance at $p \leq 0.05$, there is a 5% chance to obtaining random statistical significances. Hence, the few instances of statistical significance that were obtained could be due to multiplicity (running multiple ANOVAs). In controls, there was no significant difference between the pre-vibration, vibration and post-vibration trials (Figure 2-18) in the peak-to-peak EMG reflex amplitudes during the shoulder adduction task (A.24 and A.33).

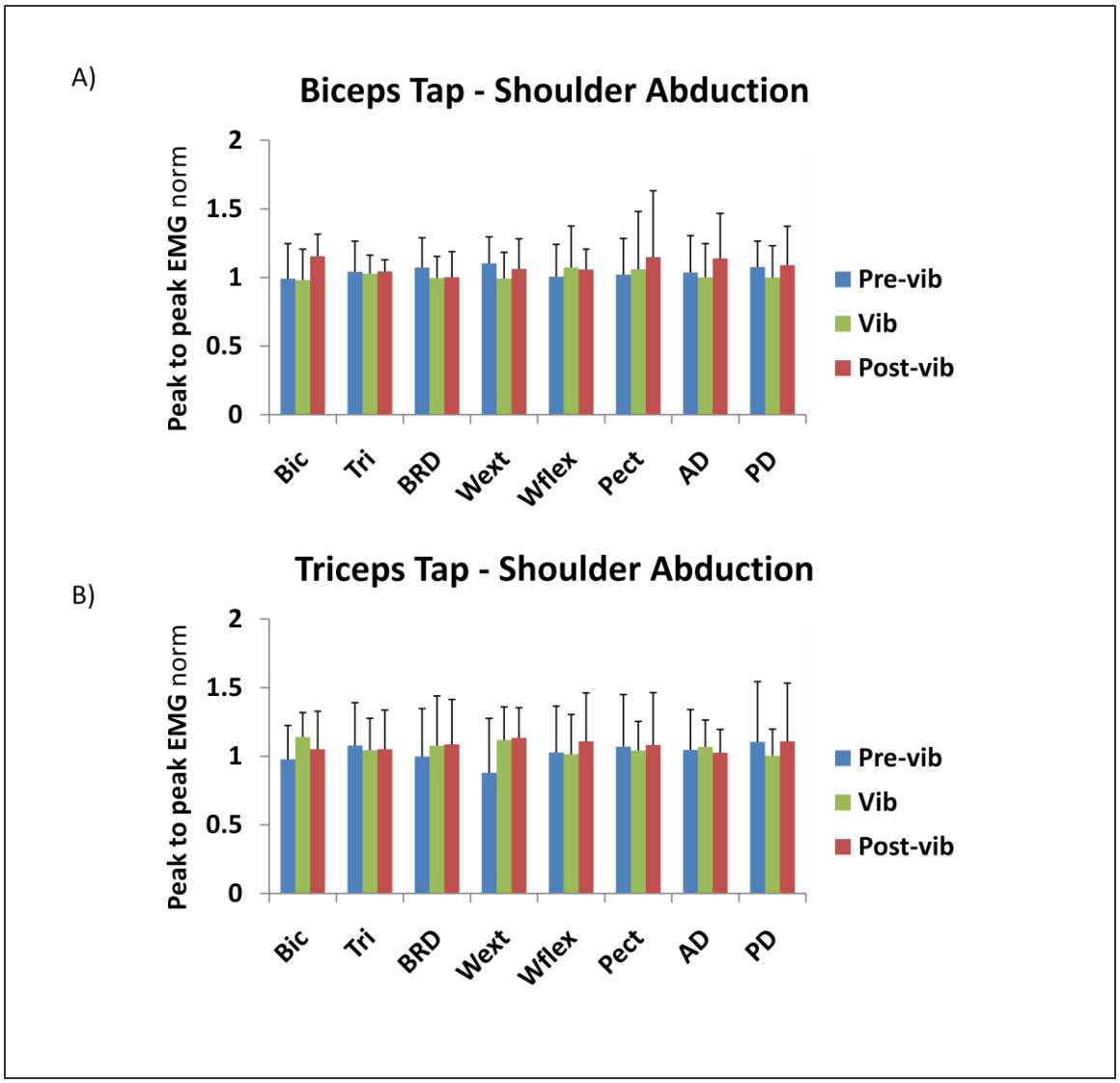

Figure 2-15. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during shoulder abduction task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during shoulder abduction (A, B) across muscles of the arm in stroke subjects. No significant difference observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials in shoulder abduction task during both biceps and triceps tendon tap.

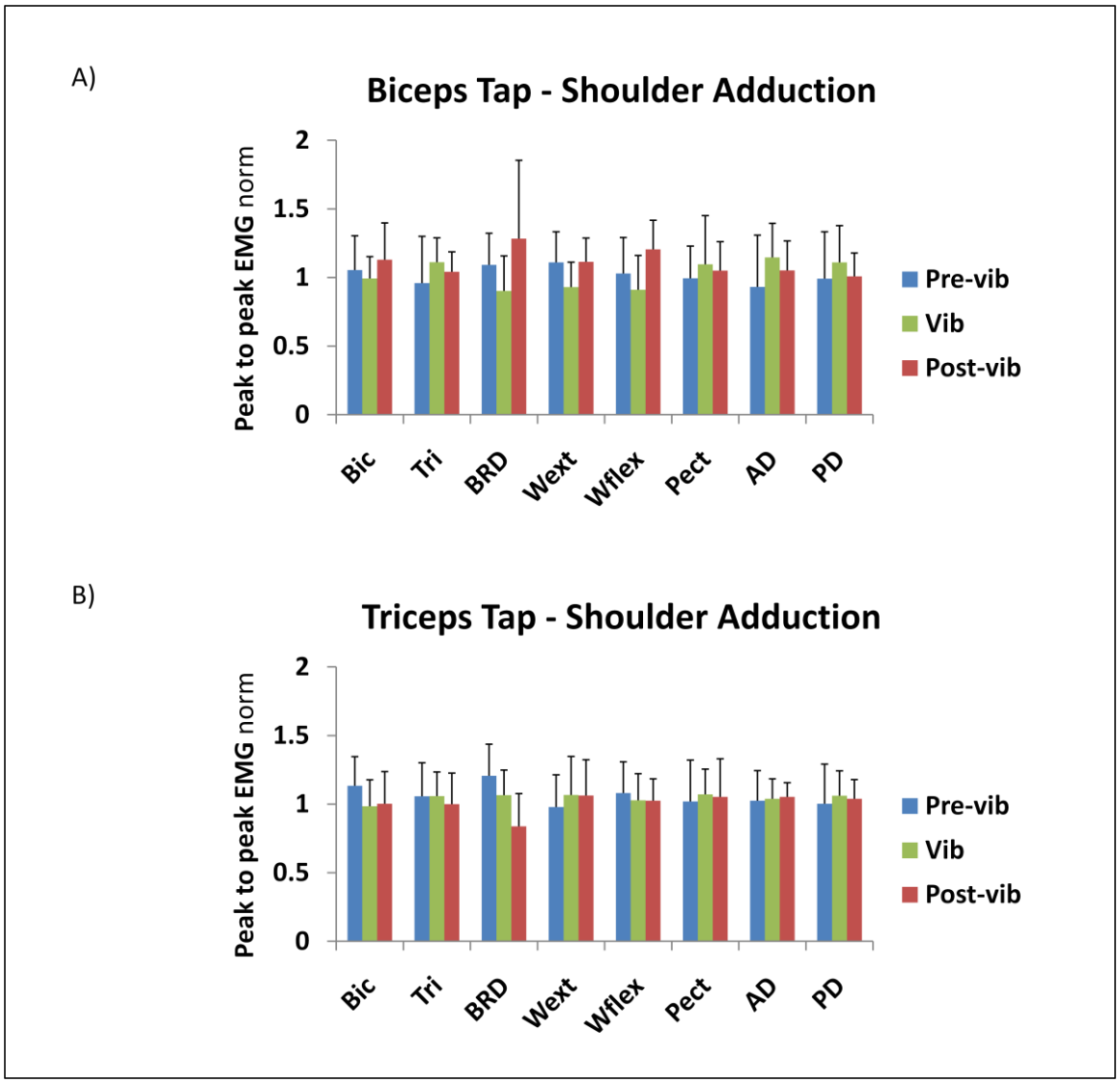

Figure 2-16. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during shoulder adduction task in stroke subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during shoulder adduction (A, B) across muscles of the arm in stroke subjects. During biceps tendon tap, wrist extensors peak-to-peak EMG amplitudes were higher during the vibration trials as compared to the pre-vibration trials. During triceps tendon tap, in the shoulder adduction task, the post-vibration values were significantly lower as compared to the pre-vibration trials. Statistically significant at $p < 0.05$.

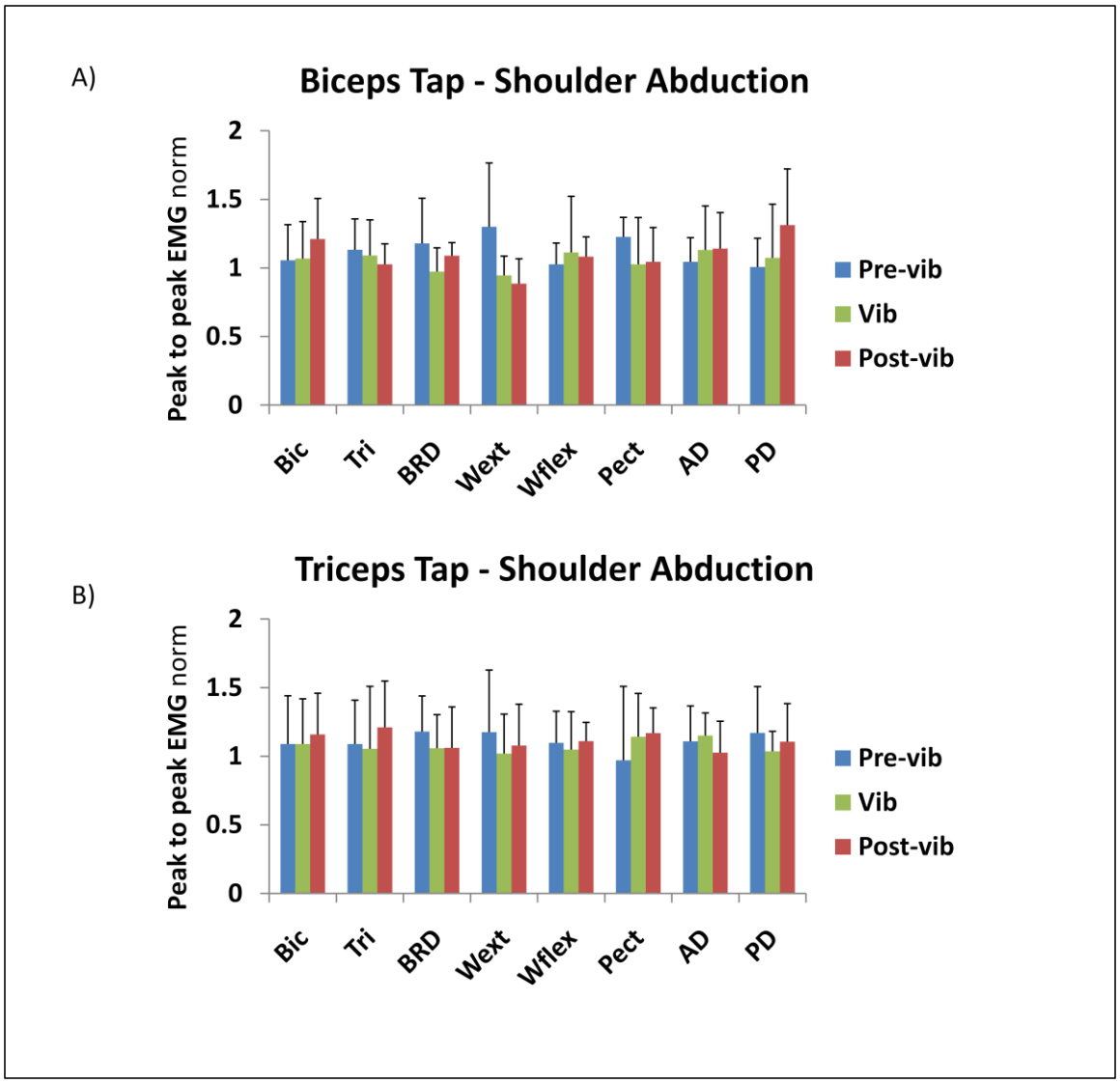

Figure 2-17. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during shoulder abduction task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during shoulder abduction (A, B) task across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

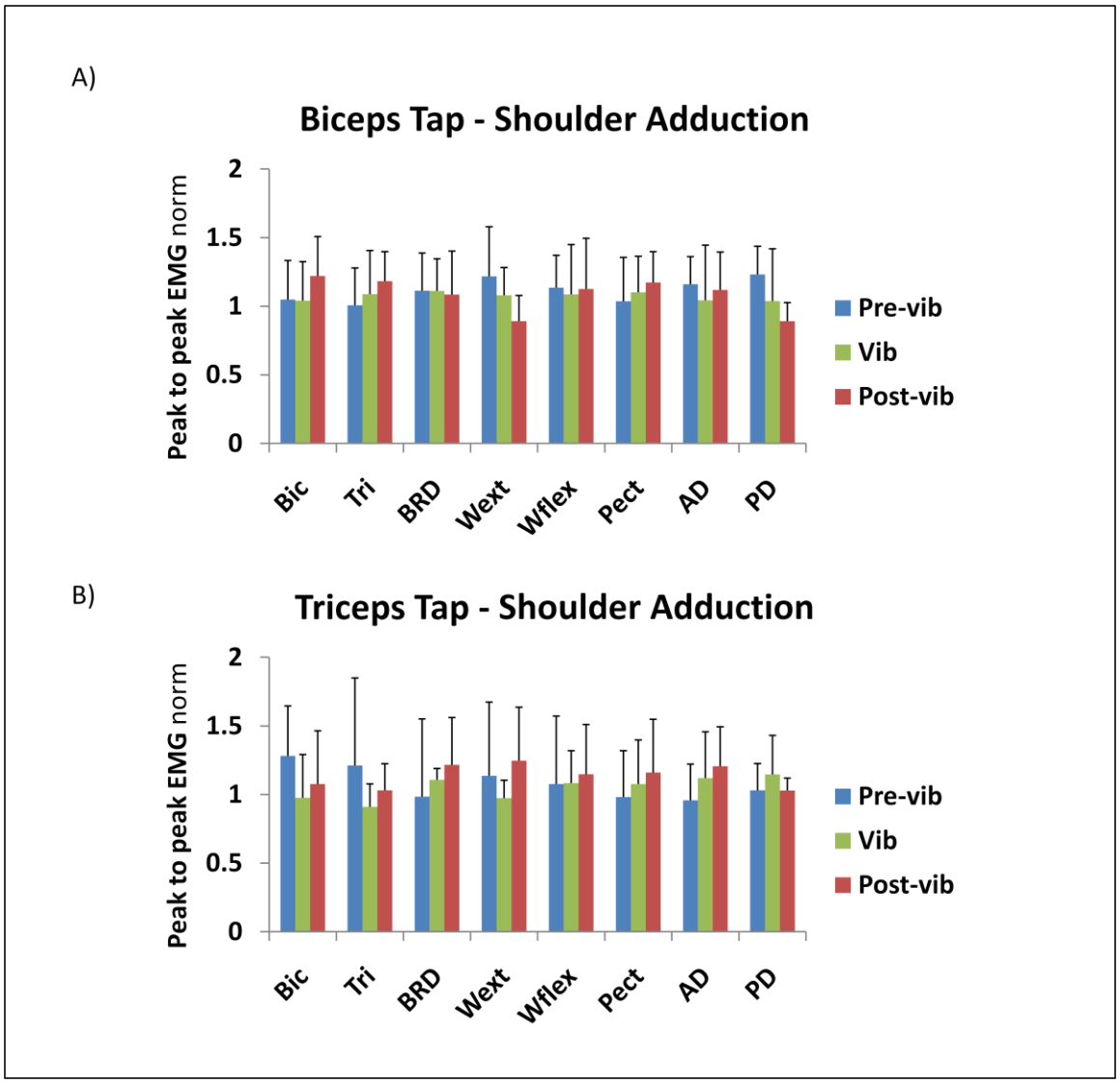

Figure 2-18. Peak-to-peak EMG reflex amplitudes to biceps (A) and triceps (B) tendon tap during shoulder adduction task in control subjects. Normalized and averaged peak-to-peak amplitudes (mean ± SD**)** during shoulder adduction (A, B) task across muscles of the arm in stroke subjects. No significant difference was observed in the reflex amplitudes between the pre-vibration, vibration and post-vibration trials during both biceps and triceps tendon tap.

2.4 Discussion

Our results demonstrated no significant difference in the stretch reflex amplitudes throughout the arm when TV was applied to the wrist flexors. During relaxed as well as the active isometric tasks, the peak-to-peak EMG signals obtained from the vibration and the post-vibration trials did not differ when compared to the pre-vibration trials. In the current analysis, the small sample size coupled with the use of a random effect analysis, could have contributed to the lack of statistical significance between the trials (pre-vibration, vibration, post-vibration). For example, a repeated measures statistical analysis of tendon tapping in relaxed conditions suggests possible interaction effects are between the trials and muscles. On a per muscle basis, in the wrist extensors, wrist flexors and pectoralis major the mean values stayed high during the pre-vibration trials, dropped during vibration and stayed low in the post-vibration trials. Although similar trends may be present in other tasks, the results were not generally significant. This suggests small effects which would require a larger sample size to differentiate between trials.

In this section we will discuss our results in the context of modulating the primary afferent feedback at the spinal level and the possible modes of action of TV at the supraspinal level.

2.4.1 Modulation of Ia afferent feedback at the spinal level in relaxed conditions by Tendon Vibration

In our study, vibration did not cause any effect on the tendon tap in relaxed conditions, which could be partly due to the phase difference between the tendon tapper and vibrator or due to a difference in the discharge rates of Ia afferents with TV. It has

been shown that TV stimulates the primary afferents in the muscle spindles, increasing the sensory input to the spinal cord during relaxed conditions in humans (Burke 1976). Specifically, during passive movement tasks vibration can suppress the monosynaptic reflexes if the tendon tap activation of Ia afferents is 'out of phase' or not in the same phase as that of the vibration cycle (Burke et al., 1976b). This suppression of the monosynaptic reflexes by vibration has been associated with a presynaptic inhibition, affecting Ia afferents- α motoneurons (Latash, 2008). However, our study did not demonstrate suppression of tendon tap reflexes during relaxed conditions with vibration as seen in these previous studies. This may be because our tasks consisted of relaxed trials and not of passive movements. Moreover, there was no specific phase relationship between the tendon tapper and the tendon vibrator in our tasks. Also, the EMG response of a muscle to TV has been observed to have a large initial phasic surge followed by a drop, with a latency consistent with the conduction velocity of Ia afferents (about 10-20 ms for a monosynaptic response) and then again, an increase in muscle activity (Matthews, 1984). However, the increase in muscle activity is less than the initial phasic response, reducing their response to the ongoing vibration (Matthews, 1984). Hence, our results demonstrated no significant difference in the reflex amplitudes during relaxed conditions, perhaps because because of a non-specific phase relation between the tapper and the vibrationrator or due to the varied Ia afferent firing response with vibration.

2.4.2 Modulation of Ia afferent feedback at the spinal level on active tasks by Tendon Vibration

Our results indicate that TV did not cause a significant difference in the peak-to-peak reflex amplitudes during active tasks of the elbow, wrist, and shoulder. This section aims

to discuss the modulation of Ia afferent feedback at the spinal level through TV pertaining to our results.

The results from our study show that the absence of vibratory effects on the elbow flexion/extension task may be attributed to the application of vibration to only the wrist flexors. During elbow flexion/extension isometric tasks in controls and stroke subjects we saw no change in the peak-to-peak reflex amplitudes between the pre-vibration, vibration and post-vibration trials. There is some evidence suggesting that during elbow flexion/extension movements, vibration applied to wrist flexors and extensors or to wrist flexors or extensors along with elbow flexors results in an undershoot towards the extension target (Kasai et al., 1992). These researchers saw no change when vibration was applied to a single muscle. Kasai's et al., (1992) study supports and provides evidence that Ia afferents from the elbow flexors and wrist muscles converge onto the same inhibitory motor neuron in the spine causing an inhibition of the elbow extensors (Cavallari et al., 1989). This is because the firing of Ia afferents from the wrist flexors/extensors will inhibit the triceps motoneurons thus causing an undershoot towards the extension targets. Differences in the reflex amplitudes at the spinal level (via Ia afferents) with TV could have been observed if two muscles converging onto the same interneuron would have been vibrated simultaneously, in place of vibrating a single wrist flexor muscle. Also as seen in previous studies, movement towards a target in a horizontal plane improved with wrist TV (Conrad et al., 2009). Movement towards a target involves simultaneous stretch of the elbow flexors along with the wrist flexors (due to vibration of the wrist flexors). The improvement observed in reaching the target could

be explained due to simultaneous activation of Ia afferents from the wrist and elbow flexors converging onto the same interneuron.

Additionally, we did not observe an effect on the wrist flexion/extension tasks with tendon vibration. An absence of effects of vibration on the active tasks involving the wrist could also be due to vibrating a single muscle. In our study, application of vibration on the wrist flexors did not result in a significant difference on the peak-to-peak reflex amplitudes during the wrist flexion/extension tasks compared to the non-vibration trials. This could be attributed to the specific projections of Ia afferents on the propriospinal neurons. In a study when vibration was applied during alternate wrist flexor-extensor movements on two muscles simultaneously; i.e. either elbow flexor and wrist flexor or elbow extensor and elbow flexor an under-shoot of wrist extensors towards the target position was seen (Kasai et al., 1994). The results from Kasai's et al., 1994 study reinforced the idea that Ia afferents from elbow flexors and elbow extensors leads to inhibition of wrist flexors and extensors respectively (Cavallari et al., 1992). This is due to the firing of Ia afferents from the elbow flexors which would inhibit the wrist flexor motoneurons causing an undershoot towards the wrist extensor targets. Particularly during elbow movement, afferents from the elbow can cause reflexes at the wrist flexors (Burke et al., 1992). Hence, by vibrating any of these two muscles simultaneously, alternate wrist flexion/extension movements could be generated (Kasai et al., 1994). This provides a possible reason for not observing an effect in the active tasks involving the wrists with TV applied to only one muscle.

TV of the wrist flexors did not affect the reflex amplitudes of isometric tasks involving the shoulder joint (abduction/adduction). Patients post-stroke can become

unstable while holding the arm at the end of a planar motion. It is known that the shoulder plays a major role in maintaining arm stability and an increase in its instability could lead to movement errors (Conrad et al., 2009). However, in our study involving isometric tasks of the shoulder (abduction/adduction), there was no requirement for sensory motor integration involving proprioceptive afferents, since the tasks being performed were stationary force producing tasks requiring no movement error correction. Isometric tasks performed by subjects produced only the motor command for performing the task. The improvement in movement errors due to enhanced sensory-motor integration in planar movements with wrist vibration could be attributed to spinal mechanisms. Although, this is highly unlikely because the peak-to-peak amplitude of biceps and triceps tendon tap reflexes showed negligible changes during vibration as compared to non vibration trials in the current study. Recent studies have shown that tendon vibration of the wrist flexors improves the stability of the arm during a hold task (Conrad et al., 2009). These observations suggest that wrist TV can augment sensory information regarding movement error of the upper arm, leading to enhanced processing of sensory-motor integration resulting in improved arm stability. The current results in which we observed negligible differences between the trials suggest that the effects of wrist vibration on arm stability involving the shoulder likely occur in supraspinal structures, possibly reflecting a change in supraspinal sensorimotor integration mechanisms.

2.4.3 Supraspinal structures likely involved in enhancing motor outcomes by tendon vibration:

Augmenting sensory input can improve stability of the arm through supraspinal pathways. Our study involved testing the effects of vibration on a spinal pathway. The results did not demonstrate any change in the peak-to-peak EMG reflex magnitudes at the spinal level with TV during relaxed conditions and active tasks. However, TV has been previously shown to improve stability of the arm, especially at the shoulder on a planar surface (Conrad et al., 2009). This effect of wrist TV on arm stability could be attributed to the tasks performed by subjects involving correction of movement errors requiring enhanced processing of sensorimotor information. Sensorimotor processing has a higher probability of occurring at the supraspinal level than at the spinal level (Conrad et al., 2009). In Conrad's study the muscle activity decreased while performing multi-directional arm movements suggesting the involvement of supraspinal structures in improving stability. Spinal structures involved in increased arm stability would have led to increased EMG activity of muscles because of higher recruitment of motoneurons. Additionally, co-contraction of agonist/antagonist pairs was not observed in muscle activation patterns, indicating the involvement of supraspinal structures for improving arm stability. In this section we will review the sensorimotor structures that are probably involved in enhancing sensory motor performance at the supraspinal level through TV (Conrad et al., 2009).

TV has been shown to enhance the cortical and cortico-spinal excitability, leading to improved motor function. Subjects with post-stroke hemiparesis demonstrate hyper-active stretch reflexes (Schmit et al., 1999) and abnormal synergy patterns (Brunnstrom,

1970). Augmenting sensory information via TV could help promote near normal patterns of motor activity in stroke subjects by modulating motoneuronal activity through the corticospinal tracts. TV increases excitation of primary motor cortex through Ia afferents (Steyvers et al., 2003). TV, mediated by Ia afferents, causes excitation of the cortico-spinal drive, demonstrated by a high motor evoked potential (MEP) elicited using transcranial magnetic stimulation (TMS). Another study showed an increase in the cortical (motor and prefrontal) activity of the muscle and in the corticospinal projections with short-term TV increasing Ia afferent activity to the cortex, quantified via MEPs (Rollnik et al., 2001; Smith et al., 2005). These studies show that the cortical areas and the corticospinal projections have sensory motor integration areas which could play a major role in sensorimotor processing, and are likely to be absent at the spinal level. The tasks in our study were designed to study the effect on TV on spinal reflexes and did not involve sensorimotor integration from higher brain structures. Hence, we were unable to observe an effect of TV on spinal, tendon tap reflexes.

Another method by which TV improves motor performance and accounts for better sensorimotor integration is by enhancing the proprioceptive information regarding the limb's movement and position. Proprioceptive information can be enhanced by increased Ia afferent input to higher brain structures such as the motor and sensory cortices using TV (Romaiguere et al., 2003). Activation is seen mostly in areas involving sensorimotor control depicting larger activation when the movement speeds are faster (Romaiguere et al., 2003). Co-vibration of wrist flexors and extensors or to the biceps and triceps muscles at different frequencies can lead to fast and slow illusionary movements (Gilhodes et al., 1986; Romaiguere et al., 2003). These results suggest the

involvement of supraspinal structures, particularly the premotor, sensorimotor and parietal cortices for the processing of proprioceptive signals from the agonist/antagonist muscles via TV (Gilhodes et al., 1986; Romaiguere et al., 2003; Rollnik et al., 2001; Smith et al., 2005; Steyvers et al., 2003). Improved proprioceptive information from Ia afferents to the brain will help in better coordination of movement and superior planning for correction of movement errors.

These studies provide evidence for some of the higher brain structures involved in improving motor outcome and arm stability via increased sensory input by Ia afferents through TV. However, future studies are required to investigate the involvement of precise supraspinal integration mechanisms involved in improved arm stability and motor function by TV.

2.5 Conclusion

These results showed that tendon vibration did not affect the multi-joint reflex coupling of muscles across the arm from tendon tap perturbations. No significant difference was observed with vibration compared to the non-vibration trials in the peak-to-peak reflex amplitudes in muscles during relaxed conditions and during active tasks. Thus, the effects of tendon vibration on arm stability reported previously in Conrad's study (Conrad et al., 2009) do not appear to occur at the spinal level. These results imply that the effects of vibration on arm stability likely occur in supraspinal structures, suggesting a change in supraspinal sensorimotor integration underlies the effects.

CHAPTER 3: FUTURE DIRECTIONS

Our results did not demonstrate any effects of vibration on the peak-to-peak reflex amplitudes across muscles generated with the tendon tapper as a probe. This likely suggests the involvement of sensorimotor processing areas within the supraspinal region for the increase in arm stability seen in previous studies (Conrad et al., 2009). Yet, our study, which involved relaxed and isometric tasks, could not identify the possible sensorimotor integration areas that may be involved in stabilizing the arm with TV. This section elucidates some of the studies that could help us gain a better understanding of the supraspinal structures that may be involved in improving motor function and arm stability with TV.

One approach could be studying the effects of TV with transcranial magnetic stimulation (TMS) on tasks involving fine motor control of the arm. Studies involving fine motor tasks such as pointing to targets on a screen using laser pointers without supporting the subject's arm could be designed. It has been observed that the NI subjects are unstable when they use a laser pointer. People post-stroke are even more unstable due to decreased shoulder stability and heightened stretch reflexes. TMS applied to the sensory-motor integration areas could be used to investigate the effects of TV on the motor evoked potential (MEP) amplitudes recorded via EMG when the subjects perform a laser pointing task. This study could provide us an understanding of the cortical areas involved in sensorimotor processing for correcting movement errors with TV.

Other studies using modalities such as the EEG, MEG and fMRI could aid in understanding the cortical and corticospinal projections involved in improving motor

function of the arm with TV. The reaching and tracking tasks performed by stroke and NI control subjects on a planar surface have shown to improve arm stability, especially at the shoulder (Conrad et al., 2009). Cortical activities during these tasks could be monitored with MEG. While performing these tasks cortical activities could also be monitored using fMRI and EEG. Tasks could be designed using pneumatic motors for generating force perturbations which would not interfere with the EEG and fMRI signals. These studies will provide a better understanding of the changes in activity taking place at the cortical and corticospinal level.

Another potential study could be designed by vibrating two muscles at different joints and simultaneously monitoring corticospinal projections to the motoneurons with fMRI. Studies have shown that vibrating the flexor/extensor muscles at one joint with the flexor muscles at another joint leads to alternating flexion/extension movements (Kasai et al., 1992; Kasai et al., 1994). This would provide us with an enhanced understanding of the mechanisms incorporated by TV in performing active movements by modulating the corticospinal drive.

In summary, the mechanisms integrated by TV for enhanced processing of sensorimotor information to correct movement errors and improve arm stability at the supraspinal level are yet to be understood. A better understanding of the mode of action utilized by TV may guide in establishing it as a rehabilitative therapy for individuals post-stroke.

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APPENDIX A: STATISTICAL ANALYSIS

This appendix provides the p-values obtained by running multiple univariate ANOVAs ($\alpha = 0.05$) for each of the active and relaxed conditions between subjects. The peak-to-peak reflex amplitudes of the muscles between the pre-vibration, vibration and post-vibration trials were compared. A post-hoc (Fisher's LSD) test was also performed to compare and contrast the means obtained between the pre-vibration, vibration and post-vibration trials. The degree of freedom (df1, df2) for stroke and neurologically intact control subjects was (2, 18) and (2, 16) respectively.

A.1: Stroke –Biceps Tendon Tap – Elbow Extension Task

Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.946	.801	.756	.953
Tri	.514	.301	.347	.922
BRD	.230	.093	.295	.497
Wext	.163	.061	.295	.369
Wflex	.129	.051	.161	.537
Pect	.557	.312	.837	.417
AD	.857	.850	.590	.725
PD	.175	.218	.524	.070

A.2: Stroke –Biceps Tendon Tap – Elbow Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.325	.618	.146	.325
Tri	.829	.648	.565	.905
BRD	.593	.510	.657	.276
Wext	.055	.021	.501	.081
Wflex	.344	.861	.184	.243
Pect	.999	.991	.973	.981
AD	.934	.718	.884	.829
PD	.769	.693	.475	.746

A.3: Stroke –Biceps Tendon Tap – Wrist Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.325	.470	.429	.139
Tri	.165	.344	.319	.061
BRD	.742	.531	.946	.489
Wext	.642	.425	.985	.415
Wflex	.530	.801	.411	.287
Pect	.561	.368	.964	.346
AD	.940	.991	.769	.760
PD	.361	.310	.631	.142

A.4: Stroke –Biceps Tendon Tap – Wrist Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.651	.372	.535	.780
Tri	.227	.132	.965	.143
BRD	.058	.153	.291	.019
Wext	.053	.017	.271	.152
Wflex	.370	.165	.458	.499
Pect	.290	.198	.158	.894
AD	.330	.221	.909	.183
PD	.320	.219	.888	.174

A.5: Stroke –Biceps Tendon Tap – Shoulder Abduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.058	.915	.034	.043
Tri	.953	.766	.738	.971
BRD	.486	.662	.453	.241
Wext	.266	.115	.280	.595
Wflex	.797	.524	.628	.877
Pect	.822	.797	.539	.719
AD	.478	.463	.235	.637
PD	.179	.339	.067	.348

A.6: Stroke –Biceps Tendon Tap – Shoulder Adduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.118	.542	.148	.047
Tri	.267	.128	.756	.218
BRD	.078	.345	.027	.166
Wext	.025	.017	.019	.957
Wflex	.089	.150	.032	.425
Pect	.758	.888	.487	.578
AD	.121	.056	.718	.111
PD	.172	.066	.430	.264

A.7: Stroke –Biceps Tendon Tap – Passive 1 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.4	.292	.840	.213
Tri	.847	.578	.858	.705
BRD	.283	.569	.310	.121
Wext	.675	.458	.438	.973
Wflex	.298	.136	.276	.669
Pect	.703	.699	.655	.408
AD	.314	.918	.176	.208
PD	.665	.698	.612	.374

A.8: Stroke –Biceps Tendon Tap – Passive 2 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.310	.234	.811	.157
Tri	.174	.331	.346	.065
BRD	.587	.761	.320	.485
Wext	.761	.735	.693	.466
Wflex	.314	.197	.183	.966
Pect	.142	.076	.878	.102
AD	.244	.150	.984	.145
PD	.297	.145	.239	.765

A.9: Stroke –Biceps Tendon Tap – Passive 3 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.508	.608	.517	.253
Tri	.931	.900	.810	.715
BRD	.215	.176	.758	.103
Wext	.353	.191	.872	.247
Wflex	.061	.023	.468	.096
Pect	.309	.360	.533	134
AD	.690	.695	.643	.397
PD	.787	.584	.531	.936

A.10: Stroke –Triceps Tendon Tap – Elbow Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.583	.759	.318	.483
Tri	.425	.257	.980	.267
BRD	.793	.770	.504	.704
Wext	.323	.550	.363	.141
Wflex	.627	.943	.430	.390
Pect	.785	.821	.652	.501
AD	.644	.366	.775	.532
PD	.722	.427	.703	.675

A.11: Stroke –Triceps Tendon Tap – Elbow Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.513	.438	.265	.725
Tri	.599	.557	.677	.320
BRD	.589	.315	.522	.709
Wext	.705	.773	.416	.596
Wflex	.275	.117	.315	.547
Pect	.771	.714	.728	.477
AD	.227	.160	.871	.120
PD	.690	.397	.710	.630

A.12: Stroke –Triceps Tendon Tap – Wrist Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.274	.418	.411	.113
Tri	.158	.252	.060	.413
BRD	.154	.516	.195	.061
Wext	.119	.968	.071	.077
Wflex	.519	.368	.838	.288
Pect	.638	.489	.837	.372
AD	.880	.734	.883	.627
PD	.996	.987	.935	.947

A.13: Stroke –Triceps Tendon Tap – Wrist Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.584	.691	.531	.311
Tri	.198	.078	.434	.294
BRD	.738	.699	.700	.443
Wext	.315	.134	.423	.462
Wflex	.420	.556	.464	.196
Pect	.399	.304	.802	.207
AD	.340	.926	.193	.224
PD	.327	.168	.817	.245

A.14: Stroke –Triceps Tendon Tap – Shoulder Abduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.214	.130	.129	.995
Tri	.188	.148	.794	.093
BRD	.767	.665	.777	.476
Wext	.158	.117	.851	.083
Wflex	.982	.908	.943	.851
Pect	.739	.445	.657	.746
AD	.465	.358	.800	.246
PD	.479	.293	.303	.984

A.15: Stroke –Triceps Tendon Tap – Shoulder Adduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.116	.043	.420	.191
Tri	.783	.680	.493	.782
BRD	.010	.061	.160	.003
Wext	.596	.643	.578	.316
Wflex	.342	.328	.156	.642
Pect	.799	.673	.515	.817
AD	.953	.935	.829	.766
PD	.982	.857	.969	.888

A.16: Stroke –Triceps Tendon Tap – Passive 1 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.464	.303	.799	.228
Tri	.588	.630	.545	.876
BRD	.417	.214	.524	.582
Wext	.367	.171	.216	.957
Wflex	.887	.839	.622	.761
Pect	.458	.700	.299	.170
AD	.264	.186	.341	.279
PD	.371	.303	.897	.400

A.17: Stroke –Triceps Tendon Tap – Passive 2 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.429	.209	.615	.460
Tri	.107	.118	.034	.001
BRD	.099	.081	.998	.091
Wext	.359	.289	.185	.027
Wflex	.218	.345	.332	.070
Pect	.193	.115	.569	.043
AD	.421	.199	.328	.774
PD	.254	.148	.604	.063

A.18: Stroke –Triceps Tendon Tap – Passive 3 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.409	.189	.603	.435
Tri	.451	.262	.705	.150
BRD	.361	.279	.203	.813
Wext	.559	.312	.273	.903
Wflex	.482	.475	.153	.439
Pect	.418	.327	.973	.358
AD	.059	.027	.503	.112
PD	.214	.096	.633	.236

A.19: Controls –Biceps Tendon Tap – Elbow Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.213	.564	.231	.094
Tri	.210	.126	.132	.976
BRD	.430	.354	.746	.224
Wext	.438	.395	.680	.221
Wflex	.399	.508	.478	.189
Pect	.603	.354	.456	.846
AD	.120	.086	.867	.066
PD	.139	.065	.665	.129

A.20: Controls –Biceps Tendon Tap – Elbow Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.493	.631	.483	.252
Tri	.446	.503	.218	.542
BRD	.544	.507	.288	.670
Wext	.436	.459	.213	.581
Wflex	.488	.555	.246	.541
Pect	.496	.434	.260	.707
AD	.509	.343	.308	.936
PD	.408	.281	.234	.899

A.21: Controls –Biceps Tendon Tap – Wrist Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.398	.273	.903	.228
Tri	.758	.664	.476	.796
BRD	.634	.582	.357	.697
Wext	.017	.006	.091	.104
Wflex	.301	.243	.756	.152
Pect	.347	.903	.197	.236
AD	.994	.924	.937	.987
PD	.597	.431	.891	.360

A.22: Controls –Biceps Tendon Tap – Wrist Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.373	.269	.851	.205
Tri	.950	.778	.978	.799
BRD	.505	.557	.257	.560
Wext	.058	.763	.027	.044
Wflex	.337	.167	.289	.710
Pect	.676	.392	.697	.629
AD	.130	.346	.230	.050
PD	.878	.622	.781	.827

A.23: Controls –Biceps Tendon Tap – Shoulder Abduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.173	.820	.129	.090
Tri	.975	.858	.844	.985
BRD	.056	.028	.049	.728
Wext	.129	.085	.942	.076
Wflex	.677	.397	.587	.750
Pect	.116	.054	.678	.106
AD	.532	.400	.837	.303
PD	.188	.640	.180	.086

A.24: Controls –Biceps Tendon Tap – Shoulder Adduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.397	.882	.224	.278
Tri	.840	.568	.792	.755
BRD	.673	.404	.821	.535
Wext	.378	.604	.381	.181
Wflex	.501	.413	.752	.268
Pect	.378	.920	.254	.219
AD	.676	.392	.701	.626
PD	.402	.369	.665	.199

A.25: Controls –Biceps Tendon Tap – Passive 1 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.297	.542	.332	.134
Tri	.525	.285	.430	.761
BRD	.732	.541	.478	.918
Wext	.640	.469	.738	.301
Wflex	.897	.902	.662	.752
Pect	.207	.085	.377	.333
AD	.344	.775	.271	.178
PD	.591	.858	.441	.348

A.26: Controls –Biceps Tendon Tap – Passive 2 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.516	.265	.605	.528
Tri	.911	.723	.986	.710
BRD	.240	.435	.335	.102
Wext	.170	.516	.201	.072
Wflex	.819	.590	.989	.599
Pect	.171	.254	.411	.069
AD	.311	.361	.521	.140
PD	.109	.291	.230	.041

A.27: Controls –Biceps Tendon Tap – Passive 3 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.311	.141	.530	.356
Tri	.580	.431	.857	.340
BRD	.362	.786	.281	.189
Wext	.557	.457	.766	.308
Wflex	.303	.226	.818	.160
Pect	.634	.776	.365	.525
AD	.936	.752	.767	.985
PD	.701	.518	.903	.446

A.28: Controls –Triceps Tendon Tap – Elbow Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.503	.267	.428	.729
Tri	.336	.240	.858	.184
BRD	.309	.226	.165	.837
Wext	.500	.282	.844	.370
Wflex	.366	.169	.459	.484
Pect	.413	.201	.411	.613
AD	.295	.213	.846	.159
PD	.424	.490	.529	.204

A.29: Controls –Triceps Tendon Tap – Elbow Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.764	.786	.659	.481
Tri	.995	.924	.947	.997
BRD	.320	.179	.887	.221
Wext	.630	.426	.407	.973
Wflex	.227	.155	.127	.895
Pect	.467	.232	.493	.582
AD	.341	.346	.160	.598
PD	.751	.772	.655	.467

A.30: Controls –Triceps Tendon Tap – Wrist Extension Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.302	.462	.133	.396
Tri	.925	.885	.705	.814
BRD	.464	.370	.248	.775
Wext	.287	.335	.128	.520
Wflex	.485	.748	.258	.402
Pect	.358	.757	.288	.183
AD	.332	.361	.559	.153
PD	.666	.773	.588	.386

A.31: Controls –Triceps Tendon Tap – Wrist Flexion Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.752	.498	.558	.925
Tri	.592	.894	.357	.425
BRD	.630	.503	.805	.367
Wext	.824	.684	.850	.554
Wflex	.331	.150	.511	.392
Pect	.389	.589	.402	.186
AD	.499	.389	.274	.798
PD	.541	.356	.339	.971

A.32: Controls –Triceps Tendon Tap – Shoulder Abduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.566	.475	.751	.312
Tri	.601	.355	.857	.450
BRD	.367	.380	.588	.174
Wext	.534	.336	.977	.349
Wflex	.421	.269	.257	.975
Pect	.437	.222	.692	.388
AD	.091	.561	.040	.101
PD	.275	.130	.662	.252

A.33: Controls –Triceps Tendon Tap – Shoulder Adduction Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.381	.222	.923	.255
Tri	.847	.969	.642	.615
BRD	.328	.338	.589	.152
Wext	.427	.959	.277	.258
Wflex	.494	.622	.490	.252
Pect	.535	.822	.409	.302
AD	.380	.637	.364	.184
PD	.468	.263	.855	.340

A.34: Controls –Triceps Tendon Tap – Passive 1 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.209	.154	.110	.832
Tri	.364	.869	.202	.258
BRD	.303	.962	.194	.180
Wext	.575	.368	.983	.378
Wflex	.227	.221	.104	.625
Pect	.413	.227	.843	.302
AD	.038	.991	.025	.025
PD	.099	.744	.083	.049

A.35: Controls –Triceps Tendon Tap – Passive 2 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.405	.260	.246	.968
Tri	.431	.317	.238	.841
BRD	.408	.240	.272	.931
Wext	.428	.254	.284	.938
Wflex	.400	.229	.276	.897
Pect	.405	.243	.263	.955
AD	.407	.214	.773	.323
PD	.391	.194	.349	.684

A.36: Controls –Triceps Tendon Tap – Passive 3 Task				
Muscle	ANOVA (p-value) (Pre-vibration, Vibration, Post-vibration)	Post-Hoc (Fisher's LSD) (p-value)		
		Pre-vibration: Vibration	Vibration: Post-vibration	Pre-vibration: Post-vibration
Bic	.380	.760	.306	.196
Tri	.770	.486	.676	.774
BRD	.324	.706	.279	.159
Wext	.223	.236	.099	.574
Wflex	.344	.399	.158	.523
Pect	.424	.503	.514	.204
AD	.131	.190	.053	.423
PD	.347	.161	.370	.568