FORCE MODULATION DEFICITS IN CHRONIC STROKE: GRIP FORMATION & GRIP RELEASE PHASES

By

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To my Parents and Sister

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LIST OF ABBREVIATIONS

ANCOVA	Analysis of Covariance
ANOVA	Analysis of Variance
BBT	Box and Block Test
CE	Constant Error
F	Female
FMA	Fugl-Meyer Assessment
MAS	Modified Ashworth Scale
H	Hemorrhagic
I	Ischemic
L	Left
M	Male
MMSE	Mini Mental State Examination
MVC	Maximal Voluntary Contraction
N	Newton
O	Age-Matched Older Adults
R	Right
RMSE	Root Mean Square Error
SA	Stroke Affected Hand
SD	Standard Deviation
SU	Stroke Less Affected Hand
Y	Younger Adults

Abstract of Thesis Presented to the Graduate School
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The aim of the present study was to elucidate force modulation deficits in various grip phases in chronic stroke and to develop an algorithm to quantify stair-stepping phenomenon. Nine chronic stroke participants (age = 66.39 ± 9.84 years), nine age-matched healthy adults (age = 66.38 ± 8.31 years), and 10 young adults (age = 22.65 ± 2.94 years) performed a submaximal isometric power grip tracking task. The task consisted of ramp up and down at 5% MVC/s, 10% MVC/s and 20% MVC/s rates. The peak force was set at 35% MVC for all rate trials. Analyses of root mean square error, standard deviation, number of steps, and number of steps with larger step widths revealed three critical findings 1) stroke leads to force modulation deficits for the grip release phase of both hands (affected and less-affected), 2) developed novel approach in quantifying stair-stepping phenomenon while explore possible mechanisms responsible for force modulation deficits in various grip phases, and 3) high functioning stroke survivors showed greater deficits in the grip release phase, whereas low functioning stroke survivors showed greater deficits in grip formation phase. Collectively, these findings carry significant implications for stroke rehabilitation by differentiating low functioning and high functioning stroke individuals with impairments in

different grip phases. Further, potential mechanisms that account for the impairments in the grip phases were discussed and these increased our understanding of stair-stepping phenomenon in aging and stroke.

CHAPTER 1
INTRODUCTION

Stroke and Upper Extremity Function Post-Stroke

Stroke is a neurological deficit characterized by rapidly developing signs of focal or global disturbance of cerebral functions (World Health Organization, 1978). More than 795,000 people in United States suffer from a new or recurrent stroke each year imposing immense rehabilitation cost demands on the economy, amounting to $ 68.9 billion in 2009 (Lloyd-Jones, et al., 2009). Motor deficits post stroke are highly conspicuous on the side contralateral to the most affected hemisphere. These deficits arise because of multifactorial reasons, including weakness of specific groups of muscles (Colebatch & Gandevia, 1989; Patten, Lexell, & Brown, 2004), abnormal muscle tone or hypertonia (Bruke, 1988; Katz, Rovai, Brait, & Rymer, 1992; Lance, 1980; Wiesendanger, 1990), abnormal muscle synergies (Bobath, 1990; Brunnstrom, 1970; Twitchell, 1951) and loss of coordination (Levin, 1996).

Most survivors after initial stroke insult regain their walking ability, but about 30 to 66% of them are not able to use the upper extremity (G. Kwakkel, Kollen, B.J., Wagenaar, R.C., 1999; Sunderland, Tinson, Bradley, & Hewer, 1989; Wade, Langton-Hewer, Wood, Skilbeck, & Ismail, 1983). Similar findings were reported by Gowland and colleagues in that 69% of stroke survivors that were admitted to a rehabilitation unit recover from motor deficits had mild to severe upper extremity disability (Gowland, deBruin, Basmajian, Plews, & Burcea, 1992). Of this population only 14%-16% with initial upper extremity hemiparesis recover complete or near complete motor functions (Hendricks, van Limbeek, Geurts, & Zwarts, 2002). In addition, the recovery process of

upper extremity motor deficits is often slower than the lower extremity motor deficits (G. Kwakkel, Wagenaar, Kollen, & Lankhorst, 1996; Wagenaar, 1990).

A simple task of grasping and holding a glass of water requires fine motor coordination and control of the entire upper extremity muscles, including shoulder girdle, elbow, and hand muscles (Ghez, 2000). A loss of hand function accounts for about 90% loss of upper extremity function (Hume, 1990). Moreover, a hand recovery post stroke often plateaus in about one year leaving impaired daily living activities such as feeding, dressing, holding delicate objects (Andrews, Brocklehurst, Richards, & Laycock, 1981; Ford & Katz, 1966; Jorgensen, et al., 1999), writing and sensation (Fearnhead, 1999; Tubiana, 1981). After 18 months post stroke, 45% of participants had limited hand functions (Welmer, Holmqvist, & Sommerfeld, 2008). Upper arm functions are less impaired than hand functions post stroke (Saladin, 1996; Twitchell, 1951), however, loss of hand functions result in marked disruption in performance of fine and gross motor skills such as reaching, grasping, and manipulating objects, which requires motor planning, control, skill, and coordination.

Grasping/Gripping Function and Stroke

Grasping is one of the most important and complex motor skills. Jeannerod first described two phases of grasping: 1) transport phase for reaching the object by hand activating proximal muscles and 2) grasp phase during which distal muscles of hand make contact with the object for making force adjustments (manipulation) (Jeannerod, 1984, 1986). While lifting an object during the grasp phase, force is exerted by prime movers (agonists) of the hand and modulations occur according to the load force and load characteristics (Johansson, 1998; Johansson & Cole, 1992, 1994; Johansson & Westling, 1988). Frequently, a primary goal of grasping is to maintain a stable grip of an

object by manipulating finger forces (MacKenzie, 1994). In stroke survivors, the neuromuscular system involved in grasping is adversely affected due to disruption of some percentage of the corticospinal system (Hepp-Reymond & Wiesendanger, 1972; Pineiro, et al., 2000; Stinear, et al., 2007; Wenzelburger, et al., 2005), thus, resulting in impaired upper extremity functions (Fugl-Meyer, Jaasko, Leyman, Olsson, & Steglind, 1975; Hermsdorfer, Hagl, Nowak, & Marquardt, 2003). The corticospinal system is distributed in a proximal to distal gradient to the cervical spinal cord with greater motoneuron pools for the distal hand than the proximal arm (Clough, 1968; Colebatch, Rothwell, Day, Thompson, & Marsden, 1990; Fetz & Cheney, 1980; Palmer & Ashby, 1992; Porter, 1993). Therefore, in chronic stroke, insult to this system leads to more severe hemiparesis of the distal muscles than the proximal muscles of the upper extremity (Colebatch & Gandevia, 1989). However, recent findings showed that in acute stroke participants, loss of hand function was caused by loss of movements of many upper extremity segments. These data suggest that the acute phase is an exception to the proximal to distal gradient of motor deficits (Beebe & Lang, 2008). Despite these contrasting anatomical structural and functional differences, gripping and manipulating objects requires movement control of all segments of upper extremity to modulate goal-directed amounts of grip force to complete tasks (Beebe & Lang, 2008; Lang & Beebe, 2007).

Gripping an object is a component of grasping, that includes interaction of hand with the object for manipulation skills (Prodoehl, Corcos, & Vaillancourt, 2009) and can be divided into four phases: 1) opening of the hand, 2) positioning and closing of the fingers and thumb to stabilize an object, 3) exerting force to grasp an object, and 4)

releasing the object by the opening hand (Landsmeer, 1962; Magee, 2007). To understand these phases clearly, we used the following three terminologies in this paper: 1) Grip Formation Phase – positioning and closing of the fingers and thumb to grasp an object; 2) Sustained Grip Phase – exerting force to stabilize an object; and 3) Grip Release Phase – both opening of hand and releasing the object. An incapability of opening the hand leads to impaired formation and release phases. The importance of the release phase of grasp in upper extremity function has been defined in normal healthy children (Lewis, Duff, & Gordon, 2002), children with cerebral palsy (Eliasson & Gordon, 2000) and older adults with stroke (Hines, 1995; Perdan, et al., 2008). However, current grip assessment methods based on these stages are subjective and lack quantifiable measures. Moreover, the literature based on quantifiable measures lacks a definitive understanding how grip phases are differentially altered in healthy subjects and a neurologically impaired stroke population.

Force Control in Aging and Stroke

Force control is the ability of muscles to produce force steadily, accurately, and temporally matched to the target goal (Patten, 2000). Force control is termed as steadiness when referred to isometric force regulation (R. M. Enoka, 1997). The functional significance of steadiness of force involves the ability of an individual to produce precise force and to perform required movement trajectories. To accomplish daily living activities like walking, manipulating objects, writing, and playing sports require control of muscle forces at different levels. These various ranges of forces can be produced by modulating firing properties and the recruitment order of motor units (Freund, Budingen, & Dietz, 1975). Modulations within the properties of motor units can occur in two different strategies: a) rate coding or making adjustments of motor unit

firing frequency and b) recruiting motor units in an orderly manner with increased force levels (Clamann, 1993). Recruitment of motor units and their modulation in firing rates are used in combination normally to produce muscle forces. A relative contribution of either strategy depends on the level of force and the muscles required to accomplish task (Floeter, 2003). Deficits in force control in an elderly population is substantially documented in aging literature, and is often reported as increased force variability, slowness of movement and decreased accuracy of movement compromising execution of motor performance (Doherty, Vandervoort, Taylor, & Brown, 1993; R. M. Enoka, Christou, E.A., Hunter, S.K., Kornatz. W.K., Semmler, J.G., Taylor, A.M., Tracy, B.L., 2003; Galganski, Fuglevand, & Enoka, 1993; Keen, 1994; Laidlaw, Bilodeau, & Enoka, 2000). These deficits in force control may arise because of age-related changes at the motor unit, structural changes such as loss of motor neurons (McComas, Galea, & de Bruin, 1993), increased motor unit innervations ratios, or increased motor unit forces (Doherty & Brown, 1997). In addition to these structural changes, aging leads to disturbances in neural mechanisms causing reduced motor unit discharge rates (Connelly, Rice, Roos, & Vandervoort, 1999), altered recruitment and derecruitment patterns of motor units (Spiegel, Stratton, Burke, Glendinning, & Enoka, 1996), and increased motor unit discharge variability (Laidlaw, et al., 2000). Aging literature also suggests that lengthening contractions (eccentric) are more variable than shortening contractions (concentric) (Burke, Hagbarth, & Lofstedt, 1978; Christou & Carlton, 2002a, 2002b; Fang, Siemionow, Sahgal, Xiong, & Yue, 2001). Further, aging literature suggests that older adults produce a stair-stepping phenomenon especially during force

release phase of an isometric ramp contractions, during movement transitions and also during lengthening contractions (Patten, 2000).

However, quantitative differences in force control and stair-stepping phenomenon during formation and release phases of an isometric contraction in young and older adults has yet to be explored extensively. Moreover, there is no evidence on stair-stepping patterns in force control of chronic stroke participants. Thus, we developed a novel approach to quantify stair-stepping phenomenon. Additionally, aging leads to reduction in rate of force development, which might arise from a) physiological changes like muscle atrophy and slowness of muscle contractile properties (Hook, Sriramoju, & Larsson, 2001; Larsson, Li, & Frontera, 1997) or b) alterations in neural control of muscles (Barry, Riek, & Carson, 2005; Barry, Warman, & Carson, 2005; Darling, Cooke, & Brown, 1989; Morgan, et al., 1994; Seidler, Alberts, & Stelmach, 2002). These force control differences across various rates of grip formation and release during isometric contraction has not been studied in any population. Therefore, we looked at isometric grip phases at different rates of force production in normal, aging, and stroke populations.

Stroke leads to increased tone in the flexor muscles along with weakness of an extensor group of muscles of elbow, wrist and fingers (Trombly, 2007; Twitchell, 1951). This increased tone of finger musculature commonly causes impaired motor control of the voluntary opening of hand (Cruz, Waldinger, & Kamper, 2005; Hines, 1995; Kamper & Rymer, 2001; Popovic, 2005). Stroke participants with sensorimotor deficits produced excessive force while lifting, holding and moving hand-held objects (Blennerhassett, Carey, & Matyas, 2006; Hermsdorfer & Mai, 1996; Nowak, Hermsdorfer, & Topka, 2003;

Wenzelburger, et al., 2005) and also to prevent slipping of an object during precision grip (Hermsdorfer, et al., 2003). Excessive grip force production is a compensatory strategy adopted by stroke participants to deal with their sensorimotor deficits (Johansson & Westling, 1984). Additionally, stroke participants with a subcortical lesion in the left cerebral hemisphere show deficits in scaling the peak load and grip force rates suggesting the presence of motor planning deficits (Raghavan, Krakauer, & Gordon, 2006). Analysis of aperture path ratios in stroke survivors with severe hemiparesis has shown deficits in opening the fingers accurately when approaching an object to be grasped (Lang, et al., 2005). Similar results were reported by Wenzelburg and associates in that movement time was increased during terminal reaching and grasping phases in reach-to-grasp and grasp-to-lift tasks (Wenzelburger, et al., 2005). In addition, previous studies have described movement initiation and termination problems in various upper and lower extremity motor activities (Chae, et al., 2006; Chae, Yang, Park, & Labatia, 2002a; Howes & Boller, 1975; R. D. Jones, Donaldson, & Parkin, 1989). Recently, Soe and colleagues showed that grip initiation and termination was delayed on affected hand with greatest deficits found for the grip termination phase (Seo, Rymer, & Kamper, 2009). This study examined delays in grip initiation and termination phases in a maximal voluntary contraction task. Because of the timing nature of the experimental protocol, force control deficits during these phases were not explored at submaximal everyday activity force levels, which might have given better insight regarding these phases. Moreover, stroke participants even after successfully reaching grip target force level, have problems maintaining constant force while performing sustained isometric grip task (Blennerhassett, et al., 2006; Hermsdorfer &

Mai, 1996). Thus, the current stroke literature shows some evidence of deficits in various grip phases in terms of either delayed grip initiation or termination or deficits in force control during sustained phase, which requires better understanding using force control paradigms.

In particular, evidence concerning force control in grip release phase is completely non-existent. Thus, the development of quantifiable outcome measures evaluating grip phases in stroke survivors will contribute to a better understanding of the capabilities required to execute various grip phases. Moreover, quantifying the gripping phases involved in tracking motor impairments will provide sound empirical evidence for designing rehabilitation protocols to improve grip function post-stroke. Therefore, this study tests how force control is impaired across different power grip phases in chronic stroke participants.

In clinical settings, physical and occupational therapists typically assesses range of motion of fingers and wrist, maximal voluntary grip strength and hand dexterity by various methods (Fugl-Meyer, et al., 1975; Innes, 1999; Jebsen, Taylor, Trieschmann, Trotter, & Howard, 1969; Wolf, et al., 2001). However, these standard measures based on subjective evaluation give only partial information of hand functionality (Marx, Bombardier, & Wright, 1999; McPhee, 1987). Frequently, daily living activities require submaximal force control of fingers; therefore, assessment of maximal voluntary grip force explains only a small part of the hand functionality (Marshall & Armstrong, 2004; McPhee, 1987). Additionally, fine dexterous hand activities require dynamic control of grasping movements due to task constraints and movement of the object in space (Flanagan & Wing, 1993; Wing, 1996). A force tracking task allows understanding of

force control during various phases of grip, i.e., grip formation and grip release. For example, the ability to lift an object (grip formation) followed by accurately placing an object on a surface (grip release) can be studied using force tracking systems. Past research has shown that grip control in participants with neuromuscular disease, showed larger tracking errors in comparison to healthy subjects in a precision grip (Kurillo, Zupan, & Bajd, 2004). Recently, investigators have shown that grip functions are altered with age (Kurillo, Bajd, & Tercelj, 2004; Lindberg, Ody, Feydy, & Maier, 2009; Sosnoff & Newell, 2006; Vaillancourt & Newell, 2003; Voelcker-Rehage & Alberts, 2005). Additionally, brain damaged participants showed improvements in grip force control after feedback based tracking training (Kriz, Hermsdorfer, Marquardt, & Mai, 1995). Similarly, chronic stroke subjects reduced tracking error when a force tracking system was used to rehabilitate their hand functions (Kurillo, Gregoric, Goljar, & Bajd, 2005; Perdan, et al., 2008).

In summary, evidence supporting tracking systems is accumulating as a rehabilitation task assisting stroke participants. Thus, the current force tracking system used to study grip phases across different populations will provide valuable quantitative measures.

Aim, Hypothesis and Significance

Aim

The aim of this study is to quantify force control capabilities and deficits using a force tracking system in chronic stroke, age-matched elderly and college participants during a power grip using kinetic measures of force control.

Hypothesis

The primary hypothesis is that in power grip chronic stroke subjects will display greater force control deficits (root mean square error) during the grip release phase than during the grip formation phase. To control for a potential aging effect, force control data was collected and compared during power grip formation and release phase in healthy young and elderly adults. In addition, relative to healthy elderly and young adults, our secondary hypothesis predicted that stroke will show 1) increased variability of force control (standard deviation) during the release phase, 2) greater number of steps during the release phase, and 3) larger error (root mean square error), variability (standard deviation) and number of steps in grip release phases with higher force rate levels.

Significance

The significance of this study is that quantifying different phases of grip in three populations at three different force rates will help rehabilitation professionals to translate findings to clinical settings for both evaluation and intervention purposes. In addition, further quantifying the stair-stepping phenomenon across grip formation and release phases and populations will provide information about the potential mechanisms underlying this phenomenon.

CHAPTER 2
METHODS

Participants

Participants in the present study were nine chronic stroke (age = 66.39 ± 9.84 years), nine age-matched healthy participants (age = 66.38 ± 8.31 years), and ten healthy young adults (age = 22.65 ± 2.94 years). Two stroke participants were not able to meet inclusion criteria and were not included in our nine stroke participants. Stroke participants were recruited from North Central Florida stroke population, age-matched controls from Living Well, and healthy college students from the University of Florida. Admission criteria for stroke participants, age-matched healthy controls, and college students are summarized in Table 2-1.

For chronic stroke participants, functional ability of the affected upper arm was assessed using the upper extremity subset of the Fugl-Meyer Motor Function Assessment (FMA) before the force tracking protocol (Fugl-Meyer, et al., 1975). The Box and Block test was used to evaluate upper extremity motor function (Mathiowetz, Volland, Kashman, & Weber, 1985). In addition, muscle tone at the wrist and finger joints was assessed in sitting position by the Modified Ashworth Scale. Wrist range of motion was measured with a universal goniometer and finger-thumb range of movements with finger goniometer. Demographic characteristics and clinical data of stroke participants are reported in Table 2-2. All procedures in the study were approved by University of Florida's Institutional Review Board. All participants provided written informed consent prior to participation in the study.

Clinical Assessment Tools

Modified Ashworth Scale

The Modified Ashworth Scale is a clinical test used for the neurologic assessment of muscle tone. Assessment involves application of rapid passive stretch to specific group of muscles by moving limb rapidly through the full range of movement. On a 6 point ordinal scale grades, a minimum score of zero represents no increase in muscle tone and a maximum score of four represents a rigid affected part (severe hypertonia - spasticity).

Box and Block Test

Box and block test is a standardized, timed, quick, simple and inexpensive test for assessing upper extremity manual dexterity and gross motor function. Test involves transferring of individual wooden blocks (2.54 cm cube) within a partitioned box using the unimpaired hand first in 60 s and followed by the impaired hand. Performance for each hand is the number of blocks transferred successfully in 60 s.

Upper Extremity subset of the Fugl-Meyer Motor Function Assessment

FMA is a stroke-specific, performance based impairment index used to assess motor recovery, balance, sensation, and joint positioning and range of movement. Each item is scored on a three-point scale: 0 = cannot perform; 1 = can perform partially; 2 = fully performed. Score of upper extremity subset of FMA ranges from 0 to 66 with score of 66 represents nearly normal function.

Mini Mental State Examination

MMSE (Folstein, Folstein, & McHugh, 1975) is a tool to assess the cognitive mental status. Eleven questions measure five areas of cognition: orientation,

registration, attention and calculation, recall, and language. Maximum score is 30 and a score of 23 or lower indicates cognitive impairment.

Instrumentation

The power grip force measuring device were designed using four force transducers (2 MLP-50; range 50 lbs and 2 MLP-200; range 200 lbs). The transducers (Transducer Techniques, 4.16 × 1.27 × 1.90 cm, 0.1% sensitivity) were embedded in cushioned wooden platforms. The shape and size of the force measuring units were developed based on objects used in daily living activities (e.g., holding a cup). The power grip force customized design allowed assessment of functional gripping forces. The output from each force transducer was amplified using a 15LT Grass Technologies Physio-data Amplifier System (Astro-Med Inc.) with an excitation voltage of 10V and a gain of 200. The analog signal from each force transducer was digitized by a 16-bit analog-to-digital converter (A/D; NI cDAQ-9172 + NI-9215, National Instruments) and sampled at 100 Hz. The least amount of force detectable by the A/D converter was 0.00016 N. The force trace was displayed on a computer screen (1024 × 768 resolutions, 100 Hz refresh rate) with a visual gain of 15 pixels/N. Trial onset, trial offset, and visual stimulus presentation (tracking task) were controlled by a custom Lab VIEW program (8.1; National Instruments).

Experimental Protocol

Participant's Position

Participants were seated in a chair with back support in front of computer screen (17 inches) at 1 m distance. A standardized upper extremity testing position was defined by 5° to 10° of shoulder forward flexion with 10° to 15° abduction, elbow in a 90˚ flexion, forearm in mid-prone position supported on the table and wrist in slight extension to

augment maximal performance of power grip. Participants were instructed to maintain consistent posture during the power grip task and were not allowed to use any compensatory movements (e.g., changing arm or forearm orientation to alter power grip force).

Maximal Voluntary Contraction (MVC)

Prior to starting the experimental protocol, all participants completed a MVC for each hand independently in accordance with a previously established and accepted method (Morrison, 1998; Vaillancourt, Slifkin, & Newell, 2002). Participants were instructed "on hearing an auditory beep to squeeze the power grip device as hard as possible followed by relaxation on the second beep". MVC of each 6 s trial was determined as the average of 10 greatest force samples. Three trials of MVC were performed with 60 s rest between each trial to avoid fatigue (Bigland-Ritchie, Johansson, Lippold, Smith, & Woods, 1983). The computed mean value of three MVC trials was used to calculate each participant's 35% peak force level for further experimental protocol (Coombes, Gamble, Cauraugh, & Janelle, 2008; Vaillancourt & Newell, 2003). Additionally, three MVC trials were recorded as previously stated after completion of the experiment to examine the level of fatigue (Christou, Poston, Enoka, & Enoka, 2007; Ryan, Beck, et al., 2008; Ryan, Cramer, Egan, Hartman, & Herda, 2008).

Force Tracking Task

Following MVC measurement and calculations, a submaximal white stationary force tracing trajectory with peak force of 35% MVC was displayed on the computer screen. This represented trial onset. Participants were instructed to track the stationary force trace as accurately as possible by modulating appropriate power grip forces.

Figure 1-1A shows the actual representation of the trial. Real-time feedback was provided with a blue trace that plotted the force produced by participants. A trial involved rest at 1% MVC for 1 s followed by gradual modulation of power grip force from 1% MVC to 35% MVC peak force, maintaining 35% MVC contraction for 3 s and then gradually returning to baseline (1% MVC). Ramp up and ramp down rates were manipulated at 5% MVC/s, 10% MVC/s and 20% MVC/s to determine the effect of force rate change on force control. Durations of ramp up and ramp down during each trial were 6.6 s, 3.3 s, and 1.7 s for 5% MVC/s, 10% MVC/s and 20% MVC/s trials respectively. Characteristics of a single force trace are described in figure 1-1B.

Procedure

Before proceeding with the experimental protocol, participants signed the informed consent. Once participants were seated comfortably in the required testing position, the experimenter explained entire experimental procedure to participants. Participants then completed three MVC trials as described previously. Following MVC trials, participants performed 15 practice trials to get accustomed to the novel force control task and temporal pattern of the force trajectory along with visual feedback. Additional feedback was provided at the end of each practice trial with the display of root mean square error (RMSE), (i.e., difference between the target force trace and the participants performed force trajectory in blue line). RMSE feedback was not given during the experimental trials. After completion of practice session, 10 experimental trials for each hand at different force rate levels were performed. Hence, each participant completed a total of 30 trials for each hand with 15 s rest interval between trials and 5 min between two hand conditions to avoid fatigue. Force rate conditions were randomized to avoid a test order effect. Hand conditions were fixed in the order of less affected hand practice

block, more affected practice block, less affected experiment block and more affected experiment block. Similarly, young controls and age-matched elderly controls performed dominant hand blocks first during practice and experimental trials. This paradigm was used to enhance learning of the novel task for the more affected hand in stroke and non-dominant hand for controls. After completion of experimental trials with both hands, three more MVC trials were measured. The experimenter remained in the testing room for entire duration to ensure that the participants performed the force tracking task as instructed without compensatory movements.

Data Reduction

Force signals were digitally filtered at a cut off frequency of 20 Hz using a fourth-order Butterworth filter. Raw force-time series was broken down into five phases based on the algorithm (Figure 2-2): 1) phase 1 - onset of trial till onset of ramp up phase (point A to B); 2) grip formation phase - onset of ramp up phase to the peak of the trial (point B to C); 3) sustained grip phase - peak of the trial to onset of ramp down phase (point C to D); 4) grip release phase - onset of ramp down phase to baseline (point D to E); and 5) phase 2 - baseline to end of trial (point E to F). Three force variables, Root Mean Square Error (RMSE), Standard Deviation (SD), and Constant Error (CE) were calculated for grip formation phase, constant grip phase, and grip release phase for each trial. Phases 1 and 2 were not included in the analysis. These two phases were included in the trial to ensure that participants completed entire duration of grip formation and release phases accurately. Table 2-3 shows numbers of trials removed from the analysis as participants were not able to complete trial successfully. Calculations for root mean square error, standard deviation and constant error were done online using a custom built Lab VIEW program and number of steps was

quantified using Microsoft Excel. Each measure is reported as the mean score for each grip phase of the 10 trials at each force rate condition. Measures for each trial are calculated using the following methods.

Root Mean Square Error (RMSE)

Performance error was defined by RMSE. RMSE was calculated as the square of the vertical distance between the target force and amount of force produced. The following formula was used to calculate RMSE for each grip phase.

$$RMSE = \left[\frac{1}{N-1}\sum_{i=1}^{N}(x_i - t)^2\right]^{1/2}$$

Where t = target force, x_i = i^{th} force sample and N = number of samples

Standard Deviation (SD)

The linear trend in the force signal was removed and the amount of total force variability for each grip phase within each trial was determined by standard deviation.

Stair Stepping Phenomenon

During grip formation, there is a sequential increase in force production while during grip release; there is a continuous decrease in force at each required force rate. However, these two phases can intermittently be presented with steps, where there is no change in force. Such duration of constant force production is defined as step. This constant force production duration can range from 20 ms to more than 1 s.

Hence, to quantify the stair-stepping phenomenon in tracking, we used the following algorithm to define step width based on rate of force production for both grip phases.

Defining step width for different rate of change of force: Force data were sampled at 100 Hz: 100 samples in 1 second; 1 sample = 10 millisecond.

5% MVC/s Rate – Change of 5% MVC force requires 100 samples

Therefore, a change of 1% MVC force requires 100 samples × 1% MVC / 5 % MVC = 20 samples

If 10 or more consecutive force samples increased (grip formation) / decreased (grip release) less than 2% of the first force sample in the 10 or more successive force samples, then a step was defined. In other words, if the force trace remained constant (increased / decreased less than 2%) for more than half of the duration required to change force of 1% MVC (more than 100 ms) at 5% MVC/s rate, then it was considered as a step.

Similarly, for 10% MVC/s rate, 10 samples are required to change 1% MVC force. Therefore, if the force trace increased / decreased less than 2% for more than 50 ms, then it was defined as a step.

At 20% MVC/s rate, 5 samples are required to change 1% MVC force. The above criterion defined a step to be 25 ms (i.e., 2.5 samples). However, the precision of our analysis program was not able to capture 2.5 samples. Thus, we adopted conservative approach by using 3 samples. Hence, if more than 30 ms of force trace remained constant (increased / decreased less than 2%), then a step occurred.

Stair-Stepping with Larger Step Widths

Larger step widths were defined if the duration of constant step was five times or more than the criterion duration for step. Hence, for 5% MVC/s rate, larger step widths was define as 5 × 100 ms (criterion duration for step) = 500 ms and above. Similarly, larger step widths were 250 ms or above and 150 ms or above for 10% MVC/s and 20% MVC/s rates.

Statistical Analysis

To examine differences for root mean square error, standard deviation, total number of steps and steps with greater step widths at different rates of force production, separate two-way ANOVAs were conducted; 3 Groups (stroke, older adults, and young) × 3 Grip Phases (grip formation, sustained phase, and grip release) with repeated measures on grip phases. In the previous analysis for total number of steps and steps with larger step widths, only grip formation and grip release phases were used as we didn't expect any steps during sustained grip phase. Separate analyses were conducted for each rate and for each hand condition. In addition, three-way ANOVAs were conducted, 3 Groups (stroke, older adults, and young) × 2 Hand (affected/non-dominant hand, and less affected/dominant hand) × 2 Sessions (pre MVC, and Post MVC) to delineate differences among groups in testing the fatigue effect on pre MVC (before experiment) and post MVC (after experiment). Further, to minimize the effect of fatigue and to test motor learning effect, we divided 10 trials at each rate into two trial blocks: a) 1^{st} block – first five trials and b) 2^{nd} block – last five trials. Analysis was conducted using three-way repeated measures ANOVA, 3 Groups (stroke, older adults, and young) × 2 Trial Blocks (1^{st} block, and 2^{nd} trial block) × 3 Grip Phases (grip formation, sustained force, and grip release). Post hoc analysis included Tukey's HSD test.

Further, when the sphericity assumption was violated, we adopted the Greenhouse-Geisser correction method (Greenhouse, 1959). Additionally, independent *t*-tests for each grip phase were used to test if low functioning stroke participants performed task differently than high functioning participants. *t*-tests were preferred for secondary analysis due to small sample size of low and high functioning stroke participants. To test the relationship between the number of steps and steps with larger

widths to stroke severity, a regression analysis was conducted. This analysis included disease severity (FMA, MAS, and BBT) as independent variables and total number of steps and steps with larger step widths as dependent variables. The alpha level for all statistical procedures was set at $P \leq 0.05$.

Table 2-1. Inclusion criteria

Stroke participants:

1) Single, unilateral, focal cerebrovascular accident
2) At least 6 months post stroke
3) Minimum of 15˚ of wrist extension
4) Minimum of 10˚ of active thumb abduction and opposition
5) Actively able to open fingers
6) Modified Ashworth Scale Score ≤ 3
7) Absence of other neurological deficits such as proprioceptive and sensory deficits of upper extremity

Stroke participants, age-matched healthy participants and college students

1) No cognitive impairment (Mini Mental State Examination Score ≥ 23)
2) Absence of neurological deficits
3) No marked visual or hearing deficits
4) No severe medical problems such as shoulder pain or any other orthopedic condition affecting the upper extremity.

Table 2-2. Stroke participants clinical characteristics

Subject No.	Age (Years)	Sex	Dominant Limb	Stroke Type	Affected Hemisphere	Disease Duration (Months)	FMA	MAS	BBT
1	78.42	M	R	I	L	123	30	2	20
2	65.00	M	L	H	R	155	34	3	18
3	59.92	M	R	I	R	18	50	1	40
4	70.50	F	R	I	L	74	61	0	70
5	71.00	M	L	H	R	26	32	3	25
6	63.08	M	R	I	L	25	59	0	52
7	76.92	M	R	I	R	15	55	0	23
8	56.17	M	R	I	R	15	22	3	1
9	56.42	M	R	N/A	R	148	16	3	1

M – Male, F – Female, L – Left, R – Right, I – Ischemic, H – Hemorrhagic
FMA – Upper Extremity Fugl Meyer Assessment
MAS – Modified Ashworth Scale (Average of Wrist and Long Finger Flexors)
BBT – Box and Block Test

Table 2-3. Total number of trials removed from analysis

Groups	5 % MVC/s	10% MVC/s	20% MVC/s
Young	4 (200)	8 (200)	10 (200)
Age-Matched	6 (180)	7 (180)	30 (180)
Stroke	4 (180)	7 (180)	24 (180)

Number in parenthesis indicate total number of trials collapsed across hand conditions

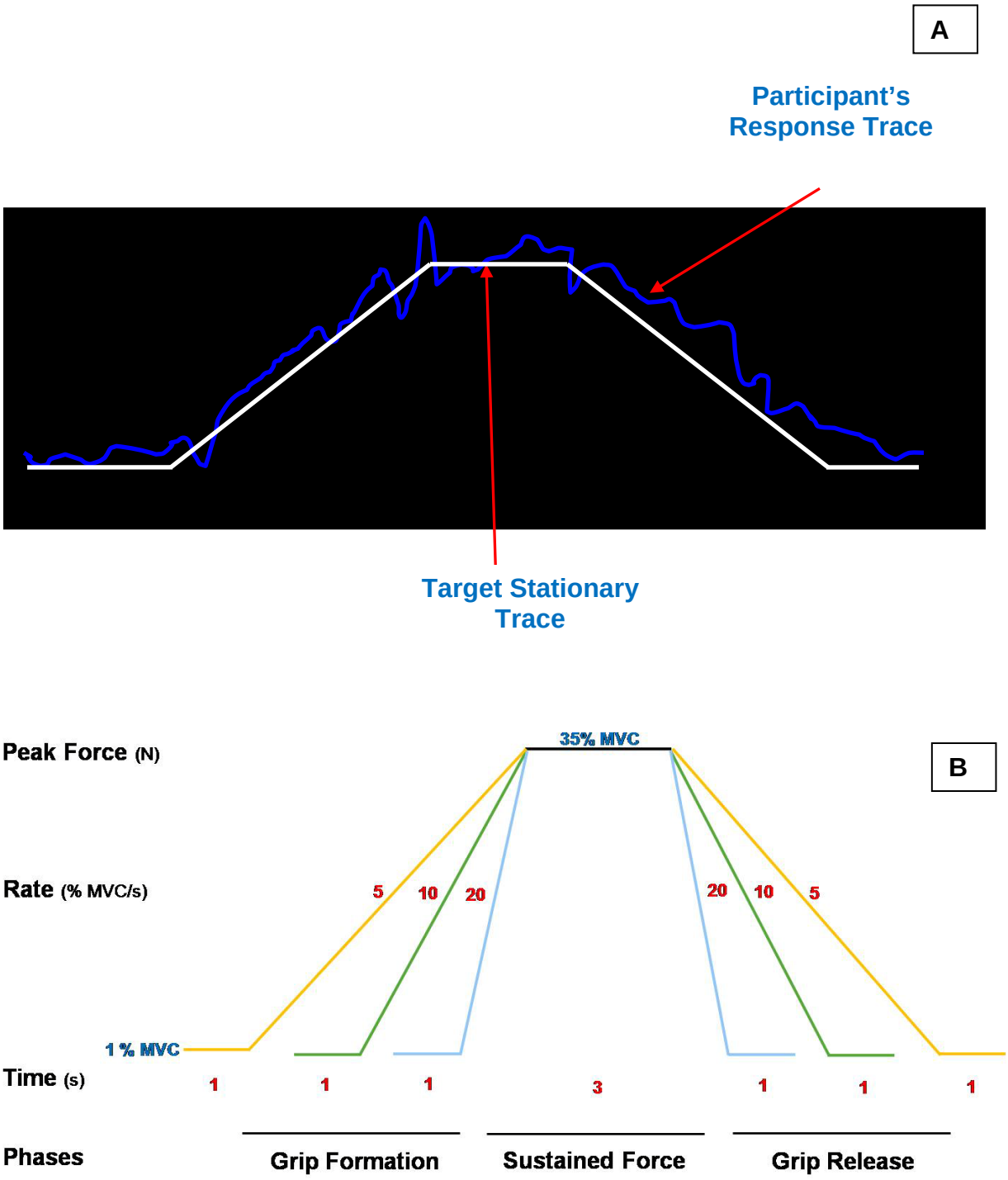


Figure 2-1. Tracking task A) Presentation on the screen during the experiment. B) Characteristics of a tracking task

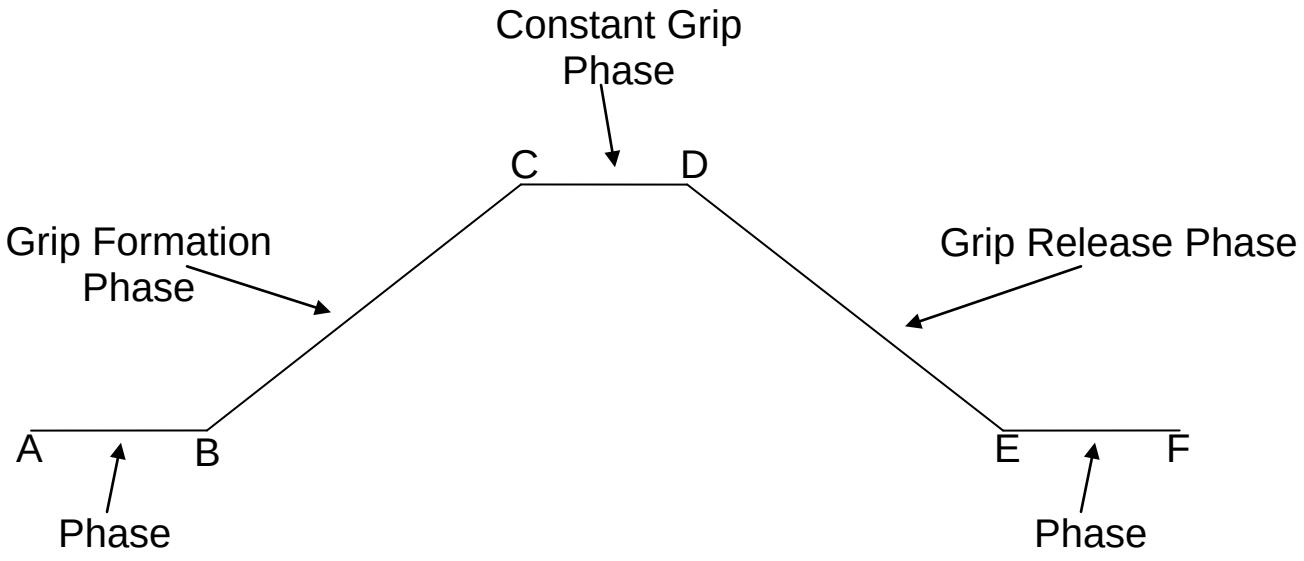


Figure 2-2. Grip phases in a tracking trajectory

CHAPTER 3
RESULTS

Maximal Voluntary Contraction Force and Fatigue

Three-way ANOVA revealed no differences in MVC force before beginning the experimental protocol across groups ($P > 0.40$). Also, pre MVC and post MVC revealed no significant differences (Figure 3-1). However, pre MVC of stroke group on affected hand (less affected hand) varied from 65.48N to 459.08N (244.17N – 508.33N), similarly non-dominant hand (dominant hand) of age-matched ranged from 164.44N – 451.72N (159.65N – 486.78N) and young adults from 143.96N – 498.91N (149.87N – 573.17N). Hence, we used analysis of covariance (ANCOVA) for all outcome measures to account for high amount of variability in pre MVC scores within subjects and these values were used as the covariate. In addition, trial block analysis for each outcome measure (first 5 trials – 1^{st} trial block and last 5 trials – 2^{nd} trial block) also showed no differences. Based on our results, we concluded that fatigue was not the contributing factor for the differences in all outcome measures with minimal to no motor learning effect.

Root Mean Square Error

Affected Hand of Stroke and Non-dominant Hand of Age-Matched and Young Adults

5% MVC/s rate (Figure 3-2)

The 3 × 3 (Group × Grip Phases) mixed design ANCOVA with repeated measures on grip phases, revealed a main effect of group ($F(2,24) = 11.955$; $P < 0.001$) and grip phases ($F(2,48) = 5.455$; $P = 0.007$). Across grip phases, stroke participants produced greatest error (5.576 ± 0.368) compared to age-matched old (3.573 ± 0.371) and young adults (3.245 ± 0.338). Also, grip release phase produced greatest error (5.095 ± 0.307)

than grip formation (3.825 ± 0.165) and sustained force (3.475 ± 0.263). We also found significant Group × Grip Phases interaction ($F(4,48) = 3.770$; $P = 0.020$). Follow up analysis revealed that, stroke group showed greater tracking error on grip release phase compared to age-matched and young adults. In addition, stroke participants produced greatest tracking deficits on grip release phase compared to grip formation and sustained phases. To delineate differences between grip phases, bias score analysis (Grip Release – Grip Formation) showed that stroke participants produced greatest error on grip release phase compared to young ($P = 0.011$) and age-matched adults ($P = 0.05$) (Figure 3-3).

10% MVC/s rate

As shown in Figure 3-4, the 3 × 3 (Group × Grip Phases) mixed design ANCOVA, showed a main effect of group ($F(2,24) = 12.368$; $P < 0.001$) and grip phases ($F(2,48) = 5.699$; $P = 0.010$). Stroke participants showed greatest error (7.022 ± 0.429) compared to age-matched old (4.939 ± 0.431) and young adults (4.176 ± 0.393). In addition, tracking performance was more impaired on grip release phase (7.107 ± 0.462) compared to grip formation (5.301 ± 0.210) and sustained force (3.730 ± 0.232) phases. Significant Group × Grip Phases interaction ($F(4,48) = 3.472$; $P = 0.022$) revealed that, stroke group reported greatest error on grip formation and release phases compared to age-matched and young adults. Stroke participants showed greater impairment on grip release phase compared to grip formation and sustained phases. Bias score analysis (Grip Release – Grip Formation) showed no differences among groups.

20% MVC/s rate (Figure 3-5)

In line with 5% MVC/s and 10% MVC/s rates, a significant two-way interaction was found ($F(4,48) = 4.575$; $P = 0.007$) for 20% MVC/s rate. Stroke participants showed

greatest error on grip formation and release phases compared to age-matched and young adults. Also, stroke participants were more impaired on grip release phase compared to grip formation and sustained force phases. As shown in figure 3-3, bias score analysis was significant (P = 0.020) with stroke participants producing greater error on grip release phase than young adults.

Less Affected Hand of Stroke and Dominant Hand of Age-Matched and Young Adults

At 5% MVC/s rate (Figure 3-6), consistent with affected/non-dominant hand results, we found significant group main effect, $F(2,24) = 3.712$; $P = 0.039$ and Group × Grip Phases interaction ($F(4,48) = 2.571$; $P = 0.050$). Follow-up analysis revealed that stroke participants significantly produced greater error on grip release phase than young adults. Within stroke group analysis revealed greater error scores on grip release phase compared to grip formation and sustained force phases.

For 10% MVC/s (Figure 3-7) and 20% MVC/s (Figure 3-8) rates, we found significant effects of group, grip phases and Group × Grip Phases interaction. Interaction finding represented greater error on grip release phase in stroke group compared to young adults. In addition, for both rates within stroke group analysis showed similar results as seen for 5% MVC/s rate.

Hence, we conclude that irrespective of rate, stroke participant's show tracking error differences on grip release phase even on less affected hand compared to young adults.

Low Functioning versus High Functioning Stroke Participants

As shown in figure 3-9, Independent t-test analysis between low functioning and high functioning stroke participants revealed that high functioning participants showed

significant reduction in error on grip formation phase at 5% MVC/s and 10% MVC/s rates. However, there was no significant difference on grip release phase at all rates. These results suggested that high functioning stroke participants improve on grip formation but on grip release phase there is no significant improvement compared to low functioning individuals.

Standard Deviation

Affected Hand of Stroke and Non-Dominant Hand of Age-Matched and Young Adults (Table 3-1)

The variability outcome measure showed consistent results with our tracking performance further strengthening our results. The 3 × 3 (Group × Grip Phases) mixed design ANCOVA. At 5% MVC.s rate, we found main effects of group ($F(2,24) = 6.485$; $P = 0.006$) and grip phases ($F(2,48) = 14.530$; $P < 0.001$). Post hoc analysis showed that grip release phase (4.699 ± 0.227) was most variable and sustained force phase (2.375 ± 0.218) was least variable. Intermediate variability was found for grip formation phase (4.224 ± 0.163). In addition, stroke participants showed greater variability on sustained and grip release phase compared to young adults. Finally, strong trend was seen with age-matched adults on grip release phase.

Additionally at 10% MVC/s and 20% MVC/s rates, comparable results were evident for main effects as seen for 5% MVC/s rate. Further at 10% MVC/s rate, stroke group produced greater variability than healthy young adults on sustained and grip release phases. At 20% MVC/s rate, we also reported high amount of variability in stroke group compared to young adults but only on sustained grip phase.

Less Affected Hand of Stroke and Dominant Hand of Age-Matched and Young Adults

Table 3-2 reveals consistent findings with affected hand results, stroke participants showed greater variability compared to young adults on grip release phase at 5% MVC/s (P = 0.028) and 10% MVC/s rates (P = 0.010). Further, at 20% MVC/s rate, we found significant differences between stroke and young groups for sustained grip phase. Therefore, we concluded that stroke participant's exhibits greater variability on grip release phase compared to younger adults at 5% MVC/s and 10% MVC/s rates even with less affected hand.

Total Number of Steps

At 5% MVC/s rate, the 3 × 2 (Group × Grip Phases) mixed design ANCOVA with repeated measures on grip phases, showed strong trend of grip phases ($F(1,24) = 3.478$; $P = 0.074$). Collapsed across other conditions, there were fewer steps on grip release phase (14.744 ± 0.507) compared to grip formation phase (16.231 ± 0.555). As shown in figure 3-10, stroke participants on more affected hand produced fewer steps on grip release phase compared to healthy age-matched and young adults. In addition, stroke group also showed less number of steps compared to age-matched and young adults on grip formation phase.

Similar findings were obtained at 10% MVC/s (Figure 3-11) and 20% MVC/s (Figure 3-12) rates for all groups when participants performed tracking task with their affected hand (stroke) and non-dominant hand (elderly and young adults).

Table 3-3 shows noticeable differences in number of steps during tracking task with the less affected hand (stroke) and dominant hand (elderly and young adults).

Thus, across all rates, stroke participants produced fewer steps on both grip formation and release phases when they performed tracking task with either hands.

Low Functioning versus High Functioning Stroke Participants

For the secondary analysis on the number of steps based on severity, one low functioning stroke participant was excluded for grip formation (P9) and grip release (P1) because of extreme scores. The analysis indicated that both low functioning and high functioning stroke individuals showed that on grip formation phase stroke participants produced fewer steps compared to high functioning participants at 5% MVC/s ($P = 0.097$), 10% MVC/s ($P = 0.040$) and 20% MVC/s ($P = 0.008$). Contrasting findings were observed for the grip release phase. The low functioning group accumulated a higher number of steps in comparison to the high functioning group at all rates (Figure 3-13).

Correlation with Disease Severity

A linear regression analysis was computed to assess the relationship between disease severity (as measured with FMA, MAS and BBT) and the total number of steps produced during the tracking task. There was a positive and significant correlation between FMA and the total number of steps produced on grip formation phase at 5% MVC/s ($r^2 = 0.499$, $n = 8$, $P = 0.050$), 10% MVC/s ($r^2 = 0.642$, $n = 8$, $P = 0.017$), and 20% MVC/s ($r^2 = 0.775$, $n = 8$, $P = 0.004$). Similarly, we found negative correlation between FMA and the total number of steps on grip release phase with $r^2 = 0.569$, $n = 8$, $P = 0.030$ at 5% MVC/s rate and $r^2 = 0.422$, $n = 8$, $P = 0.081$ at 10% MVC/s rate. A similar trend was seen at 20% MVC/s rate but, was not reached significance. The scatterplots summarizes the results (Figures 3-14 A, B, and C). Overall, across all rates, there was positive correlation between the two variables on grip formation and contrast was seen for grip release phase. Increase in functional capacity of stroke participants

resulted into greater number of steps on grip formation and reduced number of steps on grip release phase. Similar relationships were evident on total number of steps and other clinical severity measures (MAS and BBT).

Stair Steps with Larger Step Widths

At 5% MVC/s rate, we found main effects of group ($F(2,24) = 12.356$; $P < 0.001$) and a strong trend of grip phases ($F(1,24) = 4.041$; $P = 0.056$) for steps with greater steps widths. In addition, the grip release phase had greater number steps of larger step width than the grip formation phase. Finally, comparisons between groups and grip phases revealed that stroke participants showed more number of steps with larger step widths compared to age-matched and young adults on grip formation and release phases. In addition, stroke participants showed greater number of steps on grip release phase compared to grip formation phase (Figure 3-15).

Similar significant patterns remained the same across 10% MVC/s (Figure 3-16) and 20% MVC/s (Figure 3-17) rates. Further, similar results were reported for the unaffected hand as shown in figures 3-18, 3-19, and 3-20.

Low Functioning versus High Functioning Stroke Participants

One low functioning stroke participant was again excluded from the analysis for grip formation (P9) and grip release (P1) done for number of steps analysis because of extreme scores. Independent *t*-tests showed that low functioning participants produced a higher number of steps with larger step widths on grip formation compared to high functioning participants at 5% MVC/s ($P = 0.099$), 10% MVC/s ($P = 0.046$) and 20% MVC/s ($P = 0.087$). However, the low functioning group showed less number of steps with larger step widths on grip release phase compared to high functioning group but failed to reach significance level across all rates (Figure 3.-21).

Correlation with Disease Severity

Linear regression analysis on total number of steps with greater step widths is in line with above results of low functioning versus high functioning stroke participants. There was a negative correlation between clinical severity measures (FMA and BBT) and the total number of steps with larger step widths on grip formation phase at all rates. Similarly, we found positive correlation between MAS and the total number of steps with larger step widths across all rates. No significant correlation was found between total number of steps with larger step widths and severity measures on grip release phase. The scatterplots summarizes the results correlation with FMA (Figures 3-22A, B, and C).

Table 3-1. Standard deviation across groups and rates of affected hand of stroke and non-dominant hand of controls

Group	5% MVC/s			10% MVC/s			20%MVC/s		
	Formation	Sustained	Release	Formation	Sustained	Release	Formation	Sustained	Release
Young	3.85 (0.27)	1.86 (0.37)	3.83 (0.38)	6.35 (0.22)	2.16 (0.29)	6.19 (0.39)	9.11 (0.45)	2.66 (0.39)	9.37 (0.56)
Age-Matched	4.02 (0.30)	1.98 (0.40)	4.36 (0.42)	6.74 (0.24)	2.43 (0.31)	6.78 (0.42)	11.31 (0.50)	2.87 (0.43)	11.14 (0.62)
Stroke	4.80 (0.30)	3.28 (0.40)*	5.91 (0.41)*	7.18 (0.24)	3.33 (0.31)	7.81 (0.42)†	10.40 (0.49)	4.36 (0.43)†	11.07 (0.61)

* - stroke is different from young and age-matched controls
† - stroke is different from young controls

Table 3-2. Standard deviation across groups and rates of less-affected hand of stroke and dominant hand of controls

Group	5% MVC/s			10% MVC/s			20%MVC/s		
	Formation	Sustained	Release	Formation	Sustained	Release	Formation	Sustained	Release
Young	4.28 (0.80)	1.89 (0.24)	4.40 (0.47)	7.29 (0.30)	2.17 (0.26)	7.03 (0.44)	10.98 (0.57)	2.88 (0.39)	11.36 (0.66)
Age-Matched	4.632 (0.29)	2.32 (0.25)	5.28 (0.49)	7.27 (0.31)	2.62 (0.27)	7.63 (0.46)	11.66 (0.59)	3.87 (0.40)	11.75 (0.68)
Stroke	4.69 (0.29)	2.54 (0.25)	6.33 (0.49)†	7.33 (0.31)	3.10 (0.28)	9.15 (0.46)*	10.97 (0.59)	4.71 (0.40)†	12.78 (0.69)

* - stroke is different from young and age-matched controls
† - stroke is different from young controls

Table 3-3. Mean number of steps as a function of group, grip phases and rates of less affected of stroke and dominant hand of elderly and young adults

Groups	5% MVC/s		10% MVC/s		20% MVC/s	
	Formation	Release	Formation	Release	Formation	Release
Stroke	20.10 (0.67)	15.71 (0.86)†	20.43 (0.95)	14.71 (1.00)*†	16.47 (0.76)	13.70 (1.17)*
Age-Matched	18.52 (0.67)	16.54 (0.85)	20.98 (0.95)	16.81 (1.00)	16.37 (0.76)	15.74 (1.16)
Young	17.63 (0.65)	15.72 (0.82)	21.29 (0.99)	19.99 (0.95)	18.56 (0.73)	20.64 (1.12)

* - stroke is different from young controls
† - grip formation is different from grip release

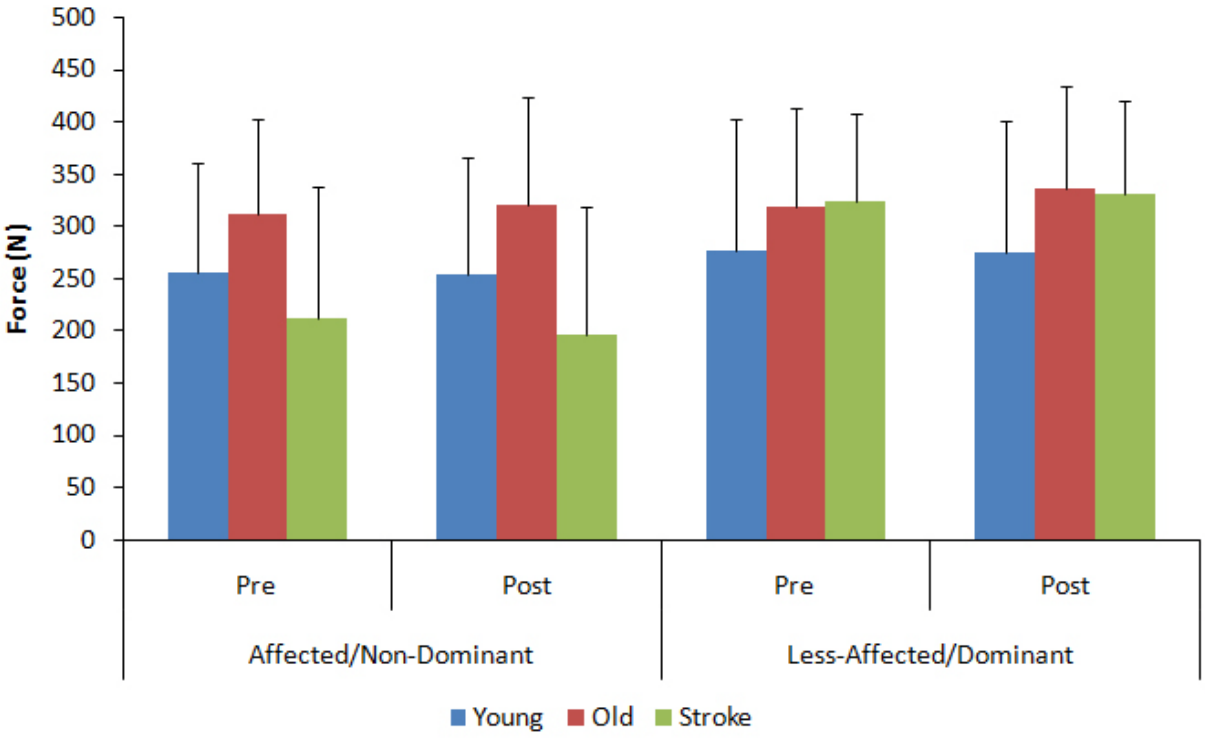


Figure 3-1. Pre MVC and post MVC across groups and hands

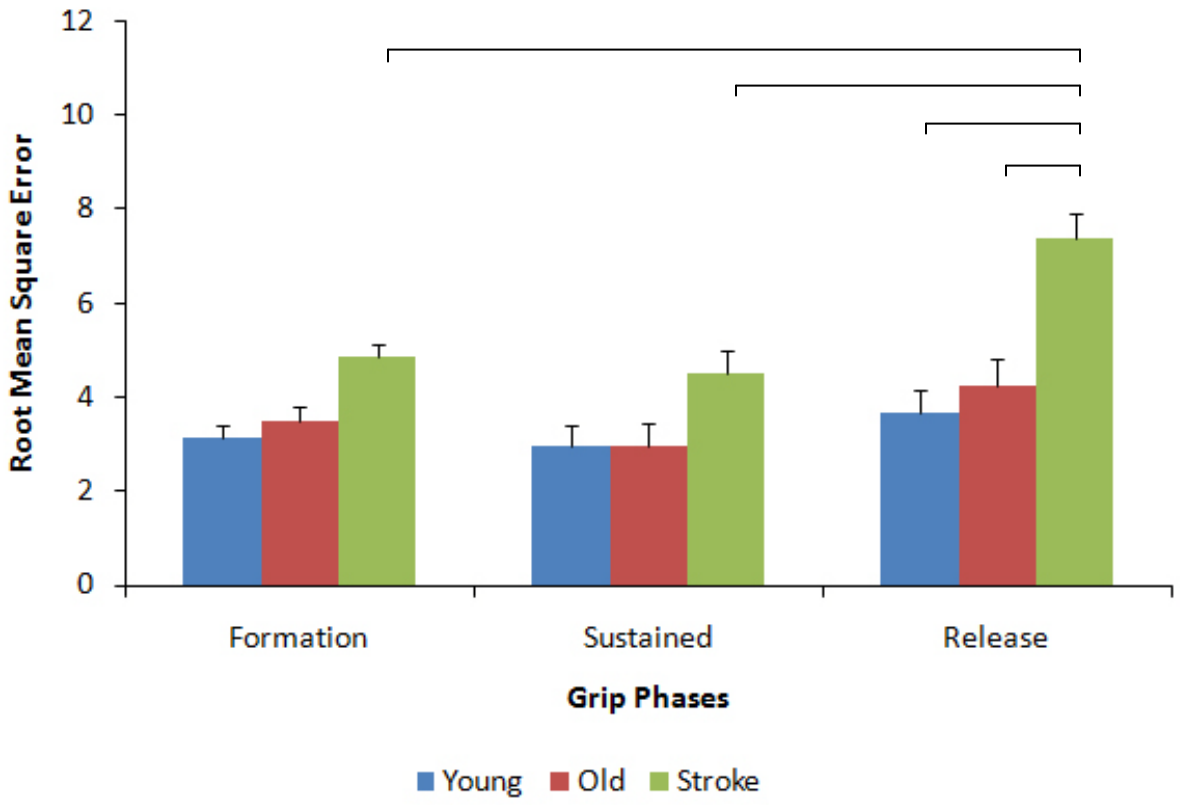


Figure 3-2. Root mean square error across groups at 5% MVC/s rate of affected hand of stroke group and non-dominant hand of controls

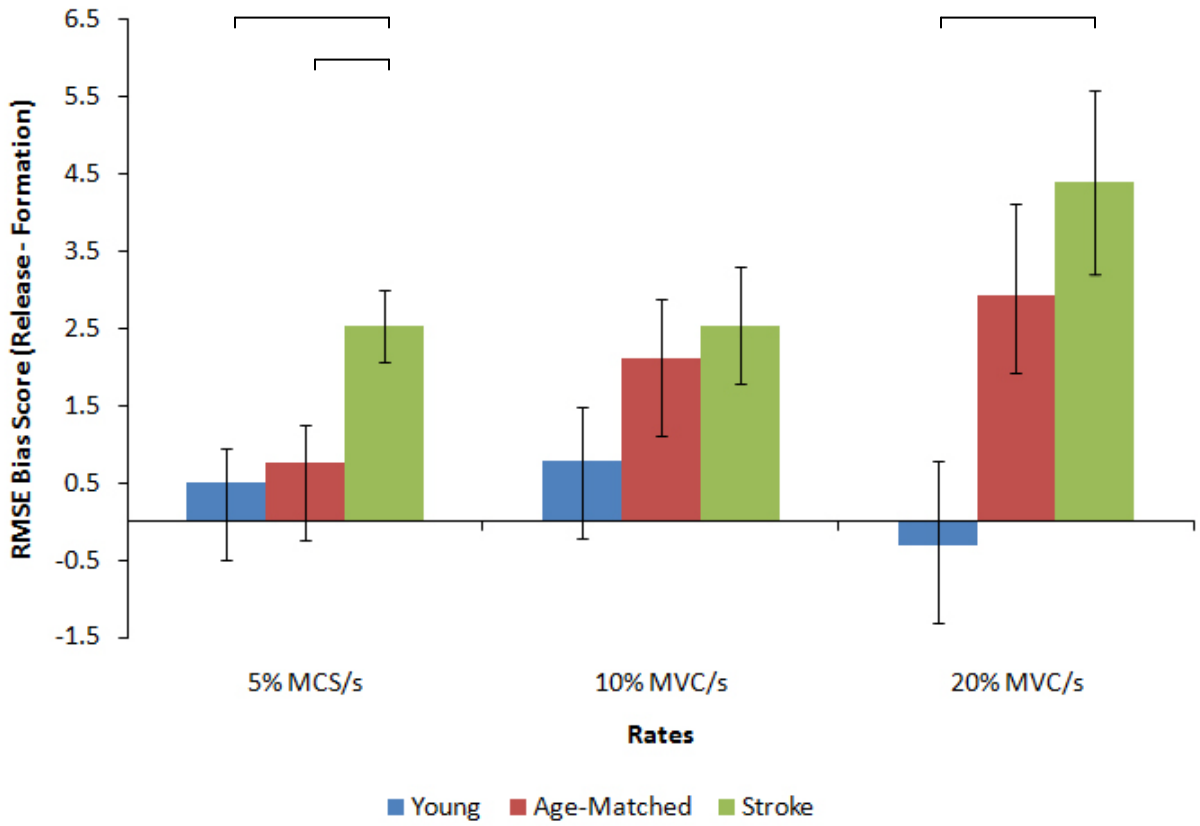


Figure 3-3. Root mean square error bias score across groups and rates of affected hand of stroke group and non-dominant hand of controls

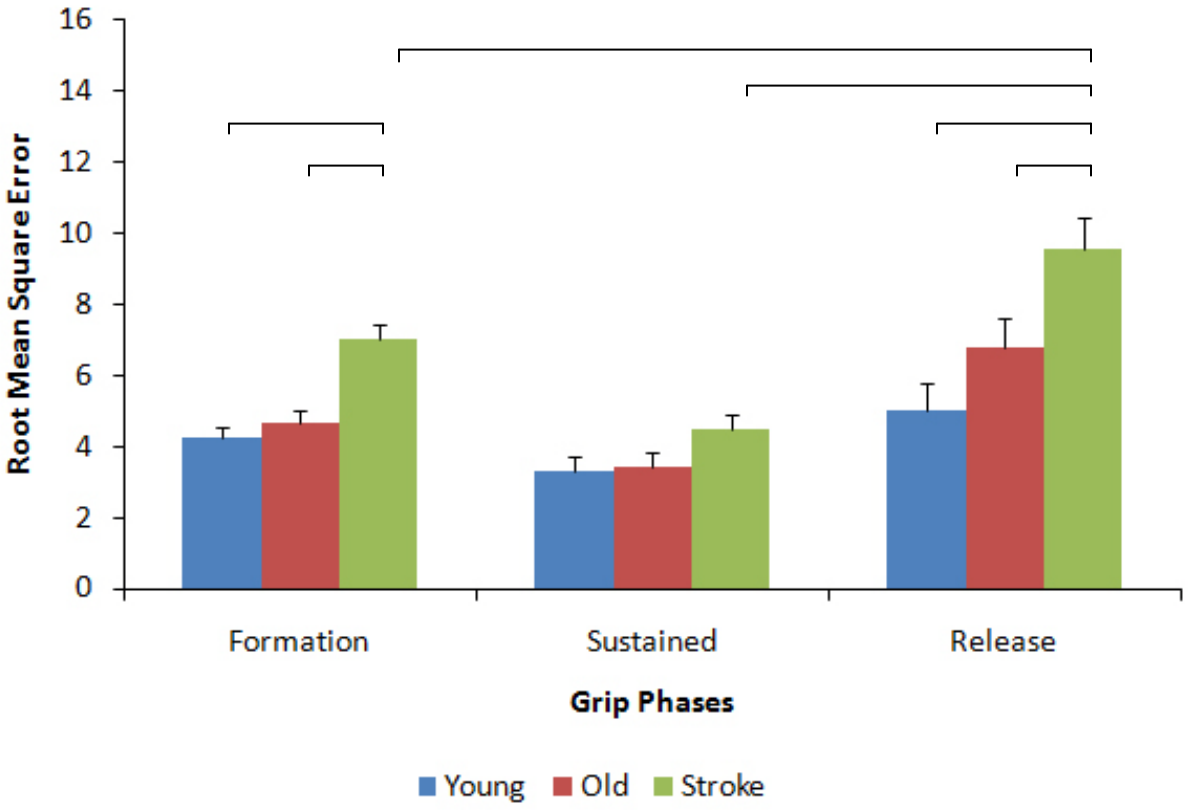


Figure 3-4. Root mean square error across groups at 10% MVC/s rate of affected hand of stroke group and non-dominant hand of controls

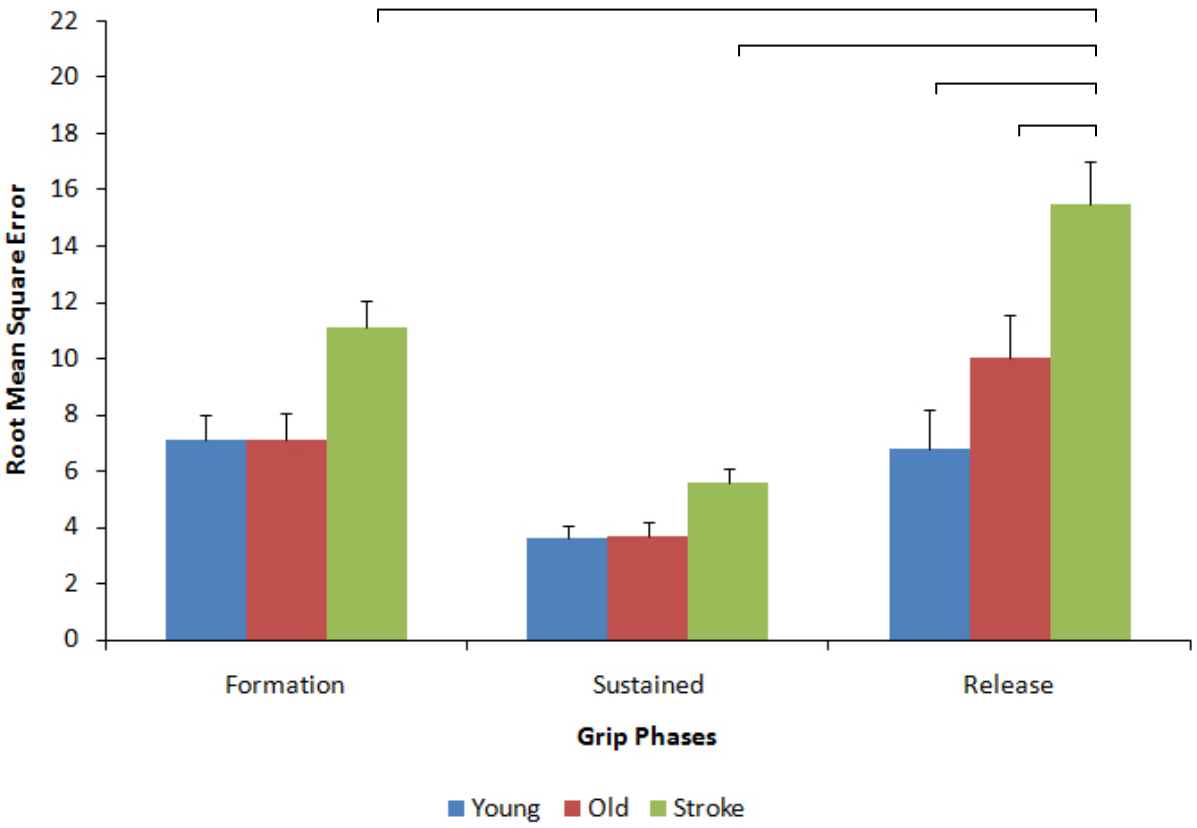


Figure 3-5. 20% MVC/s Rate – Root mean square error across groups of affected hand of stroke group and non-dominant hand of controls

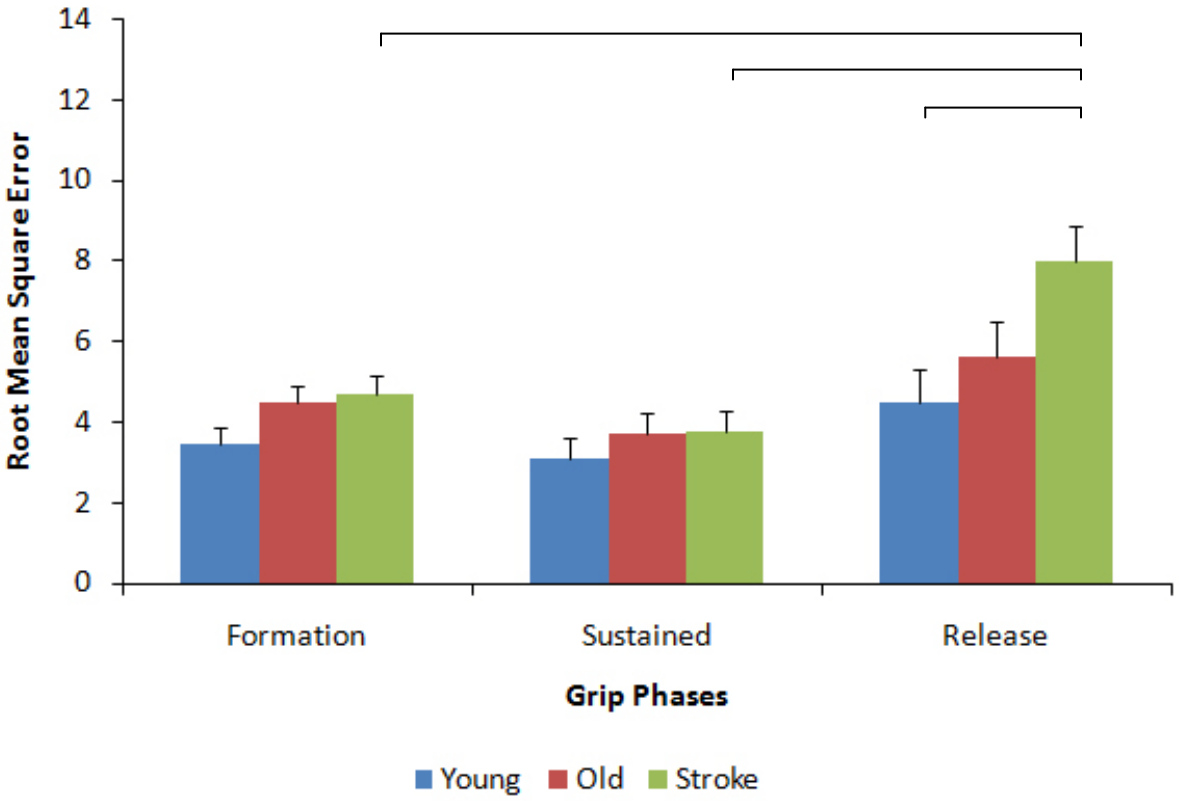


Figure 3-6. Root mean square error at 5% MVC/s Rate of less-affected hand of stroke group and dominant hand of controls

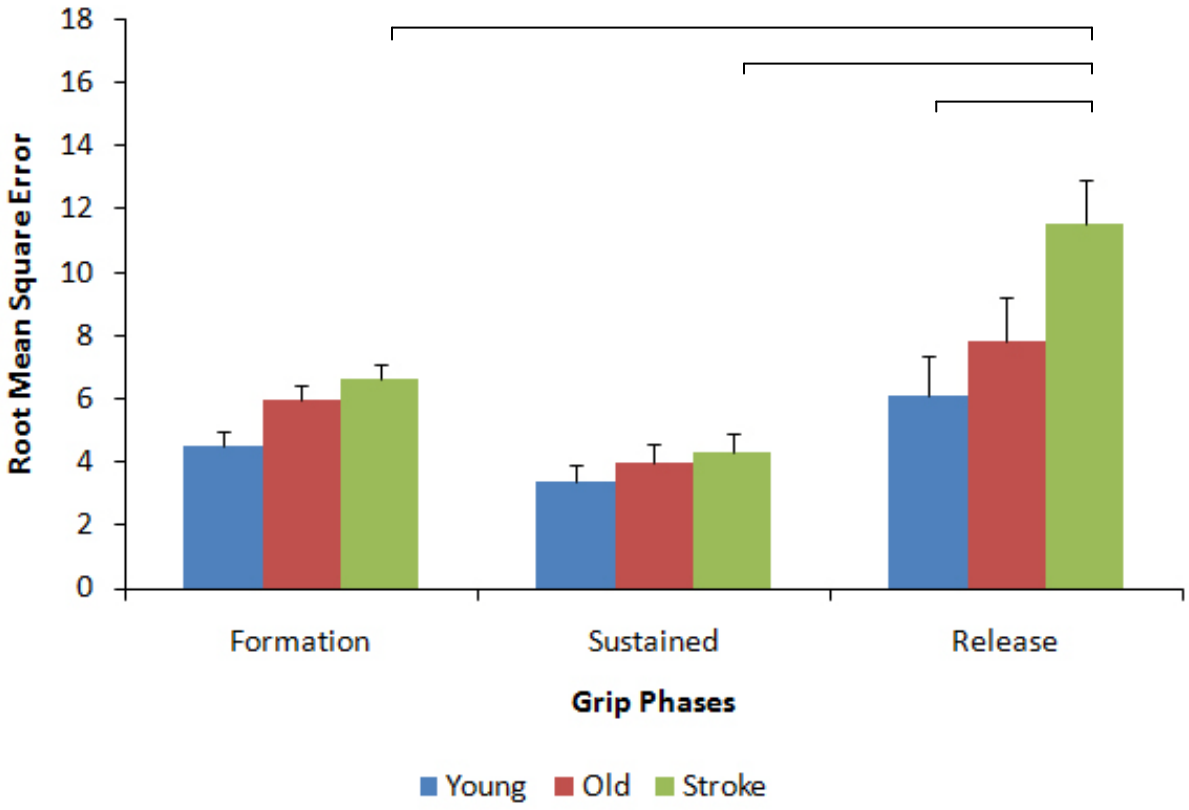


Figure 3-7. Root mean square error at 10% MVC/s rate of less-affected hand of stroke group and dominant hand of controls

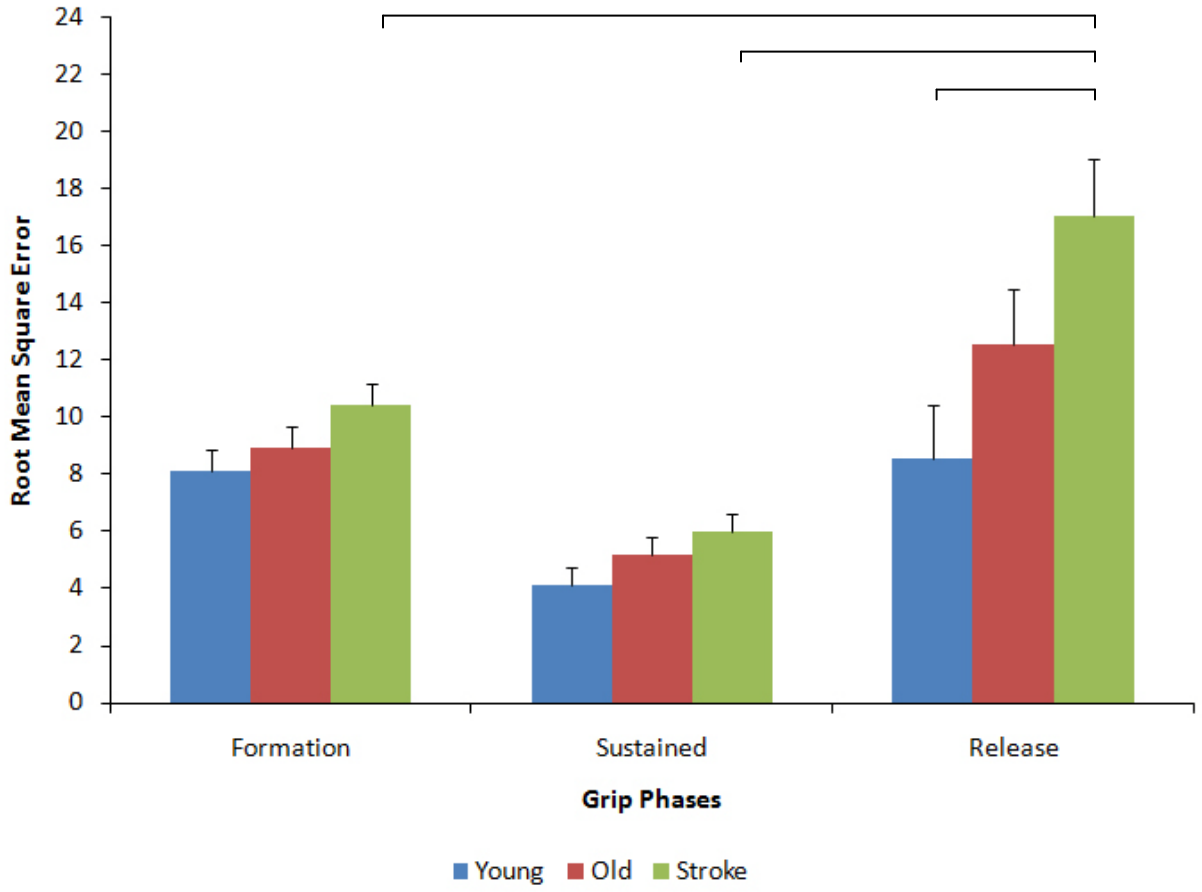


Figure 3-8. Root mean square error at 20% MVC/s rate of less-affected hand of stroke group and dominant hand of controls

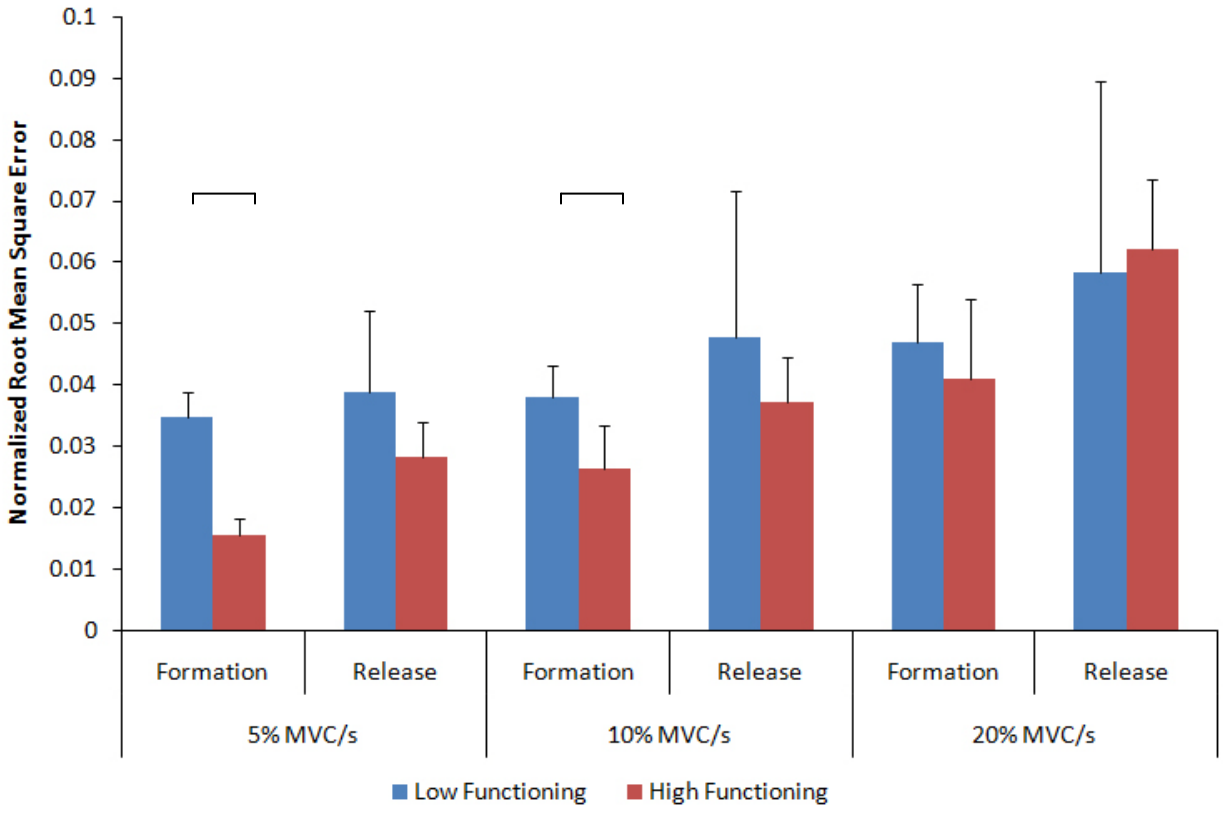


Figure 3-9. Normalized root mean square error across rates of affected hand of low functioning versus high functioning stroke participants

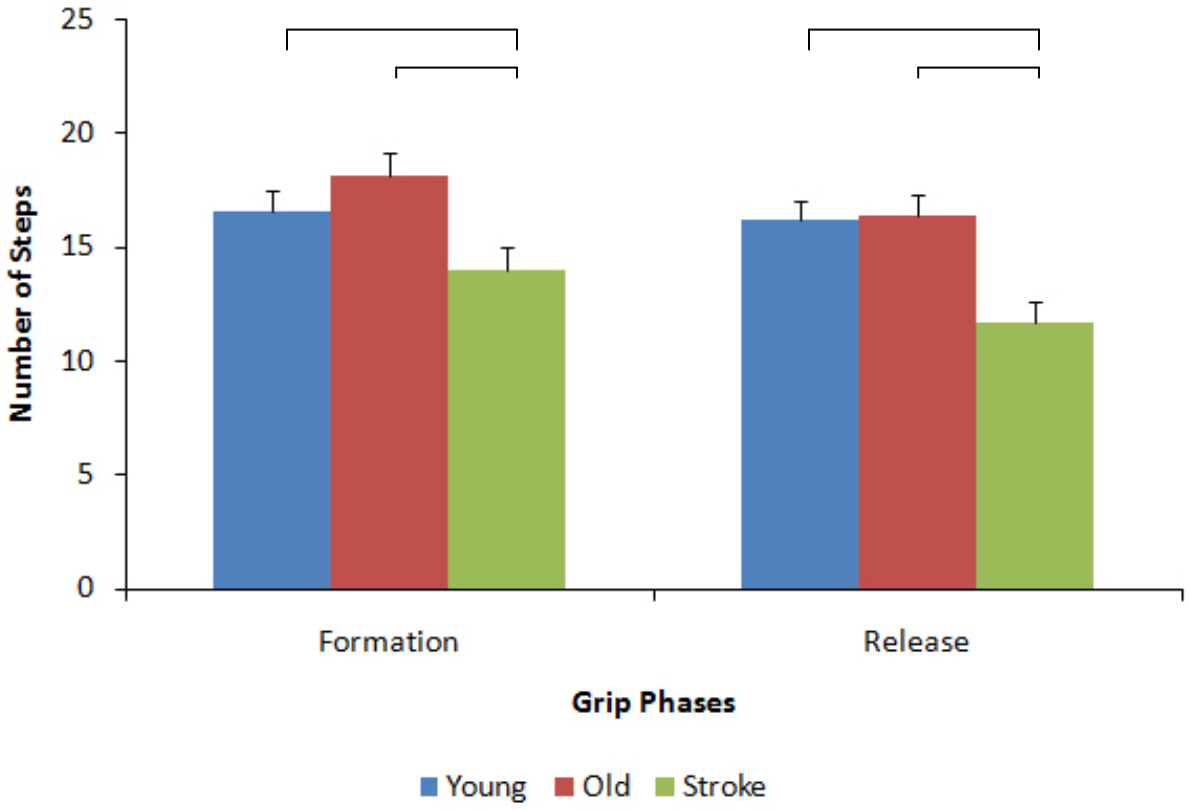


Figure 3-10. Total number of steps at 5% MVC/s rate of affected hand of stroke group and non-dominant hand of controls

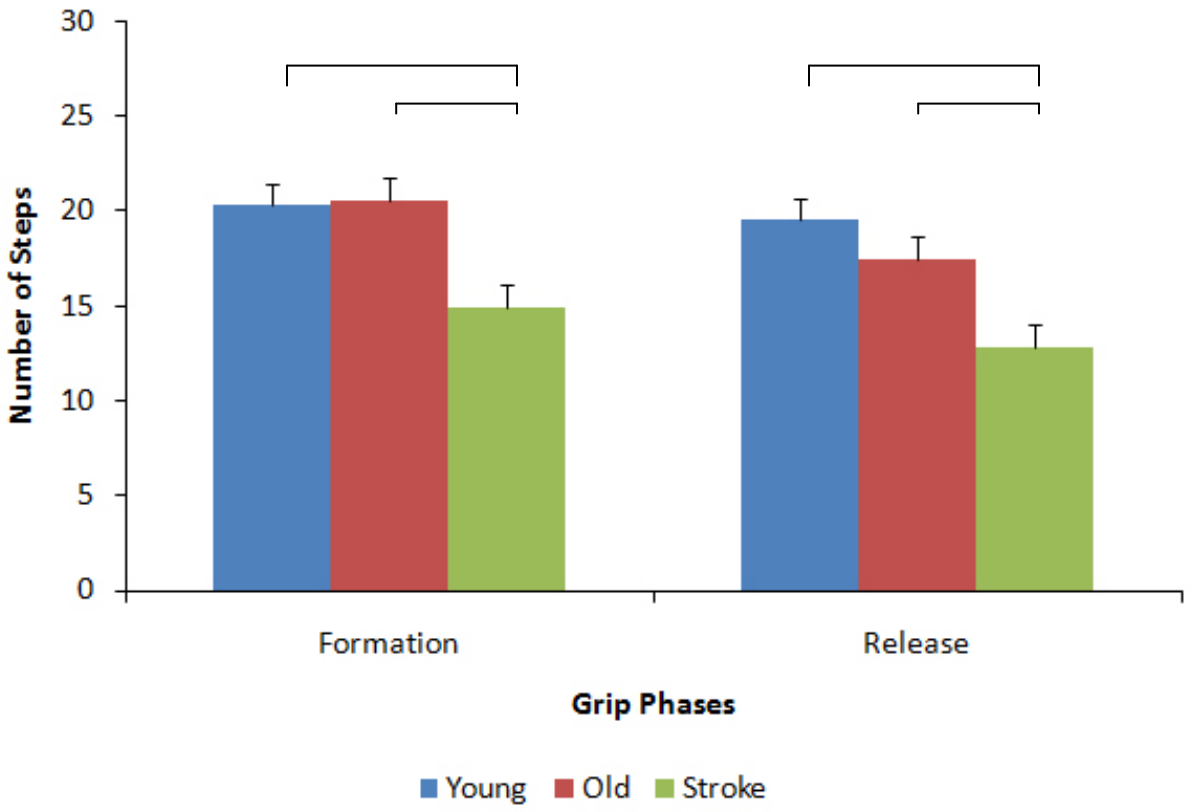


Figure 3-11. Total number of steps at 10% MVC/s rate of affected hand of stroke group and non-dominant hand of controls

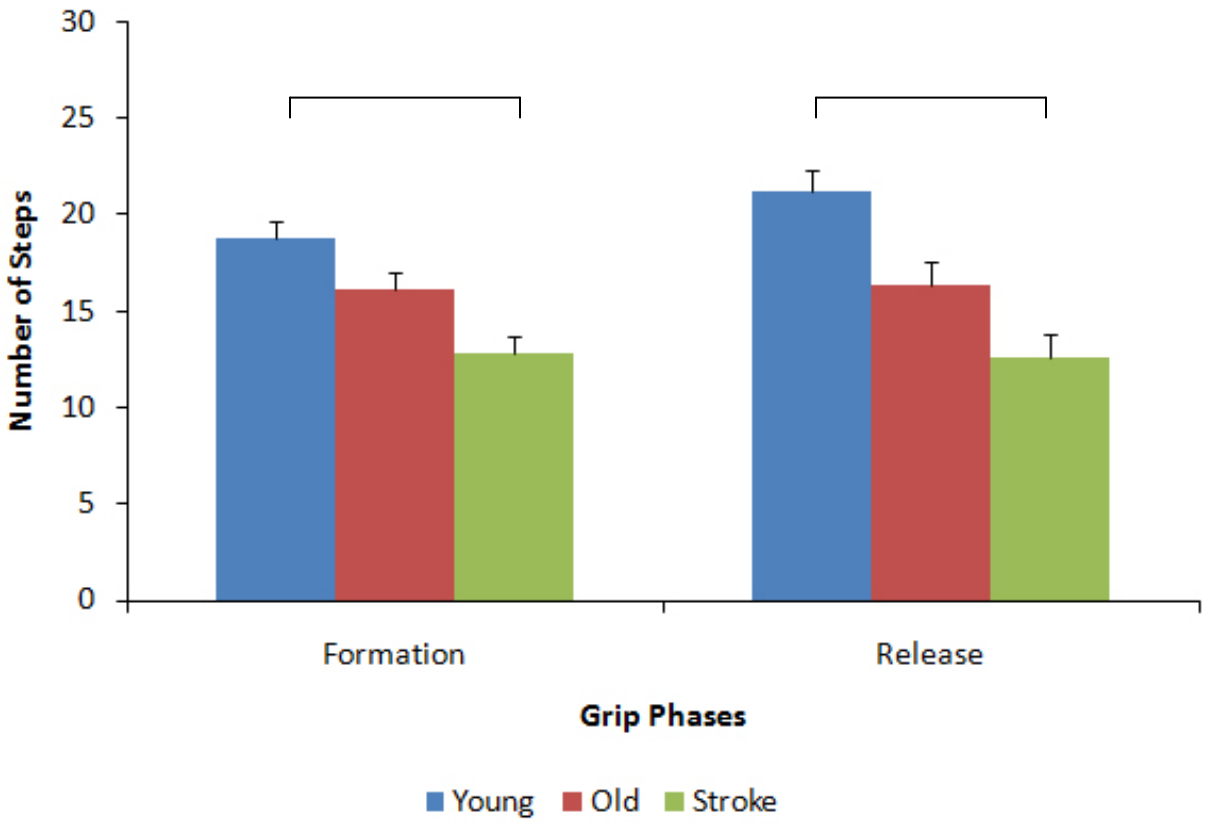


Figure 3-12. Total number of steps at 20% MVC/s rate of affected hand of stroke group and non-dominant hand of controls

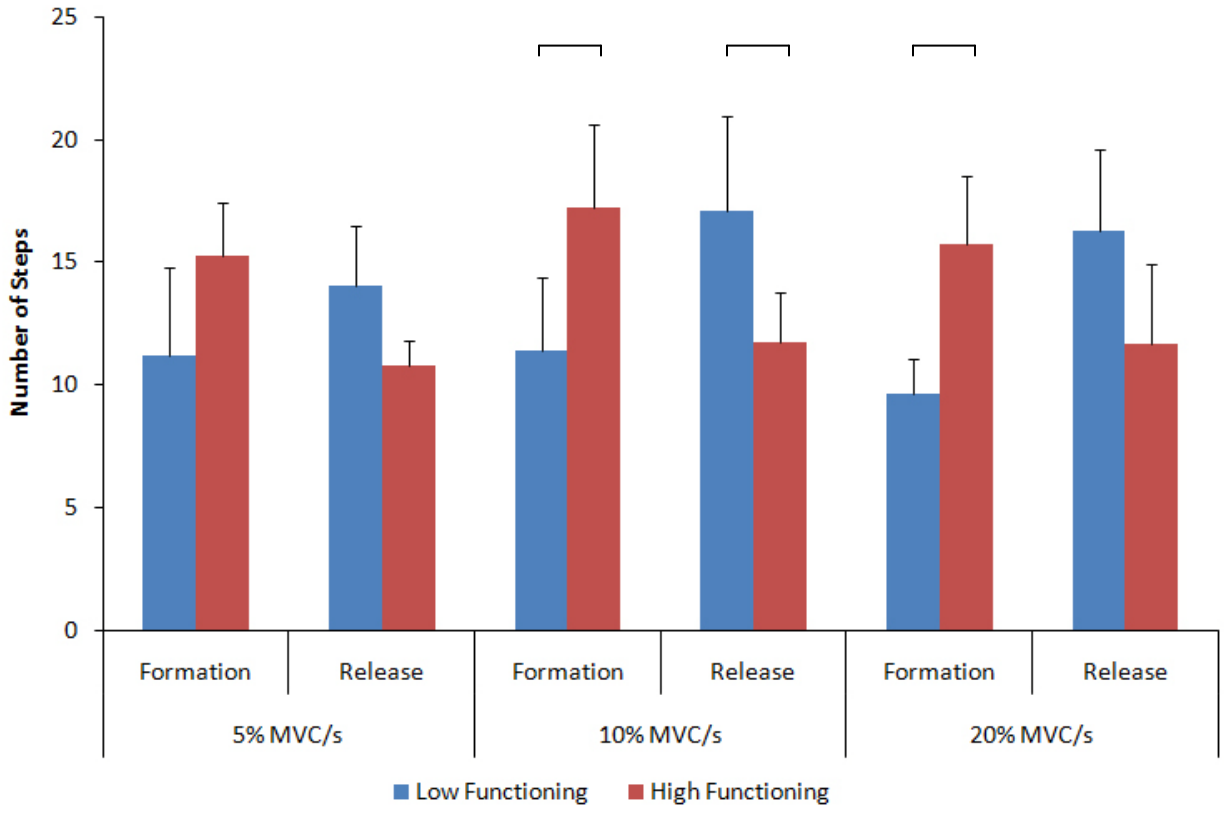


Figure 3-13. Total number of steps across rates of affected hand of low functioning versus high functioning stroke participants

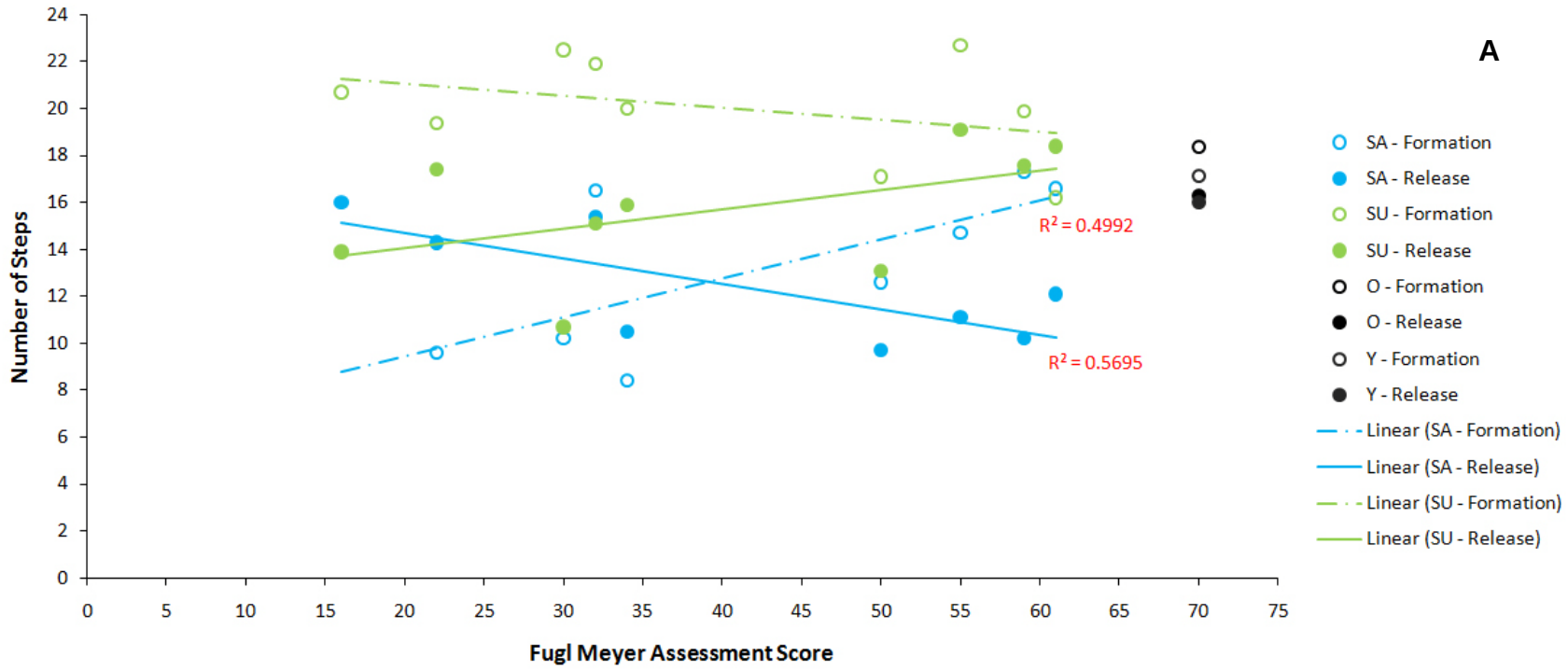
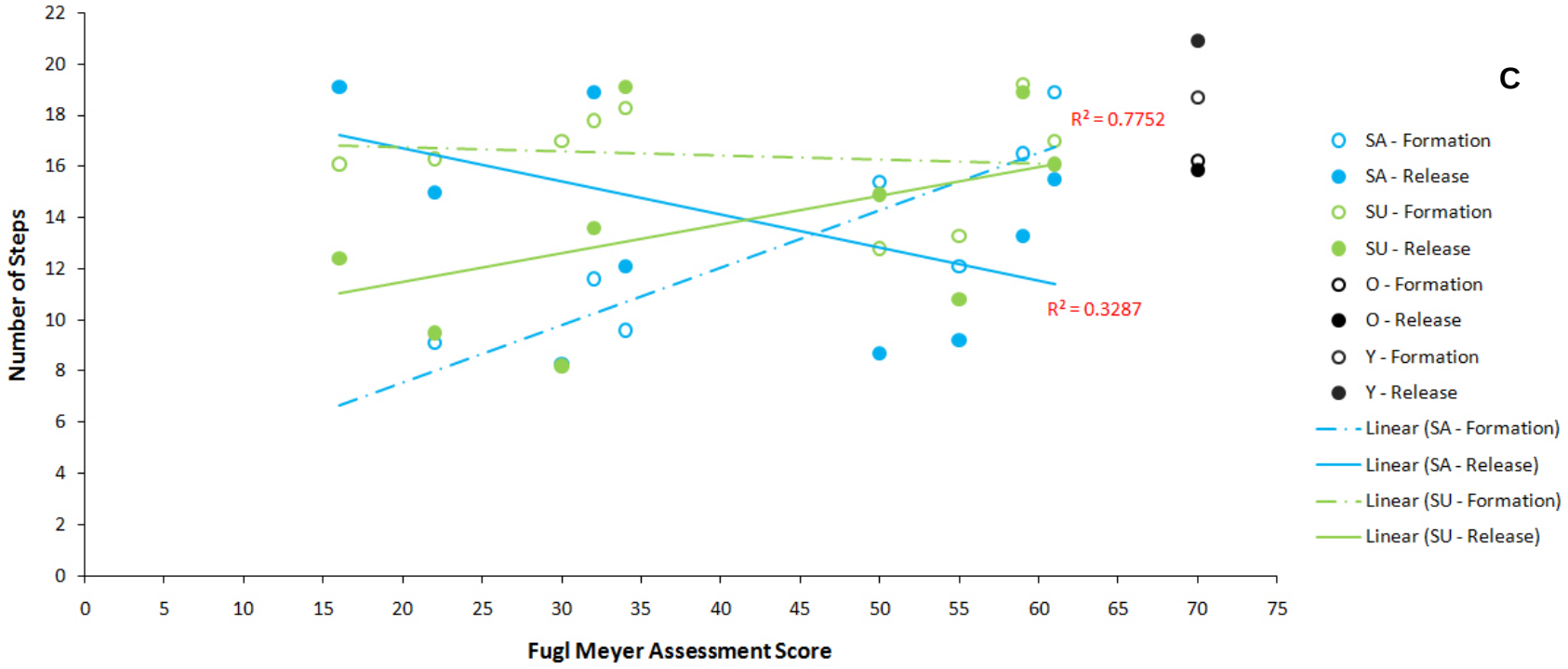


Figure 3-14. Correlation of total number of steps with Fugl Meyer Assessment scores of stroke group at A) 5% MVC/s Rate. B) 10% MVC/s Rate. C) 20% MVC/s Rate.

Figure 3-14. Continued



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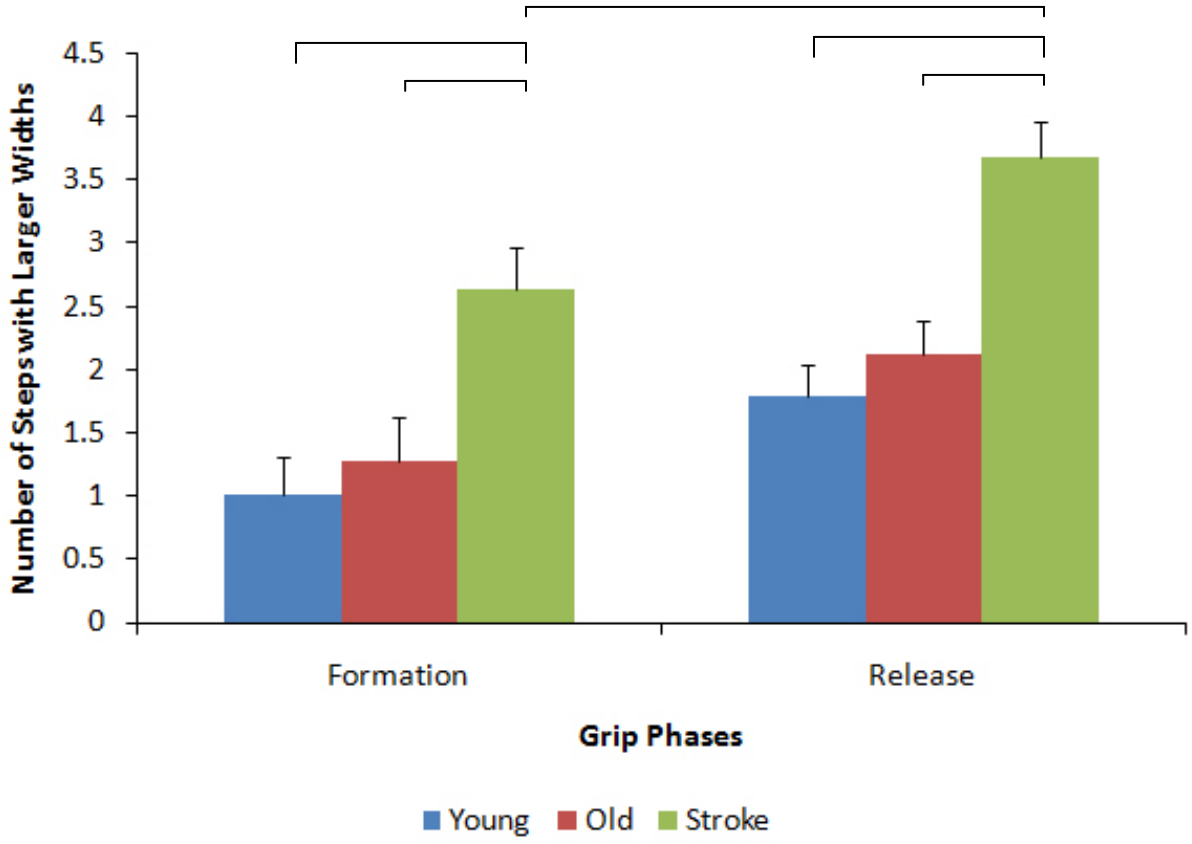

Figure 3-15. Number of steps with larger step widths at 5% MVC/s rate of affected hand of stroke and non-dominant of controls

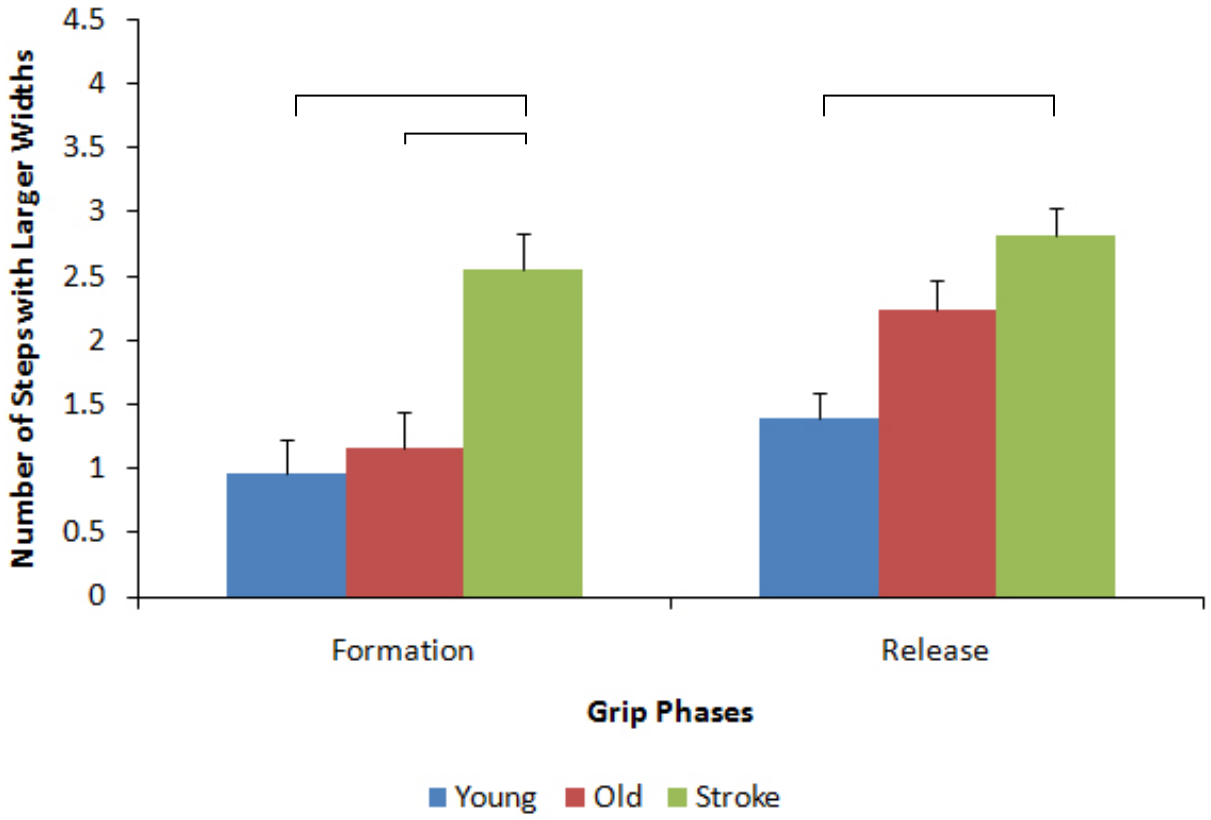

Figure 3-16. Number of steps with larger step widths at 10% MVC/s rate of affected hand of stroke and non-dominant of controls

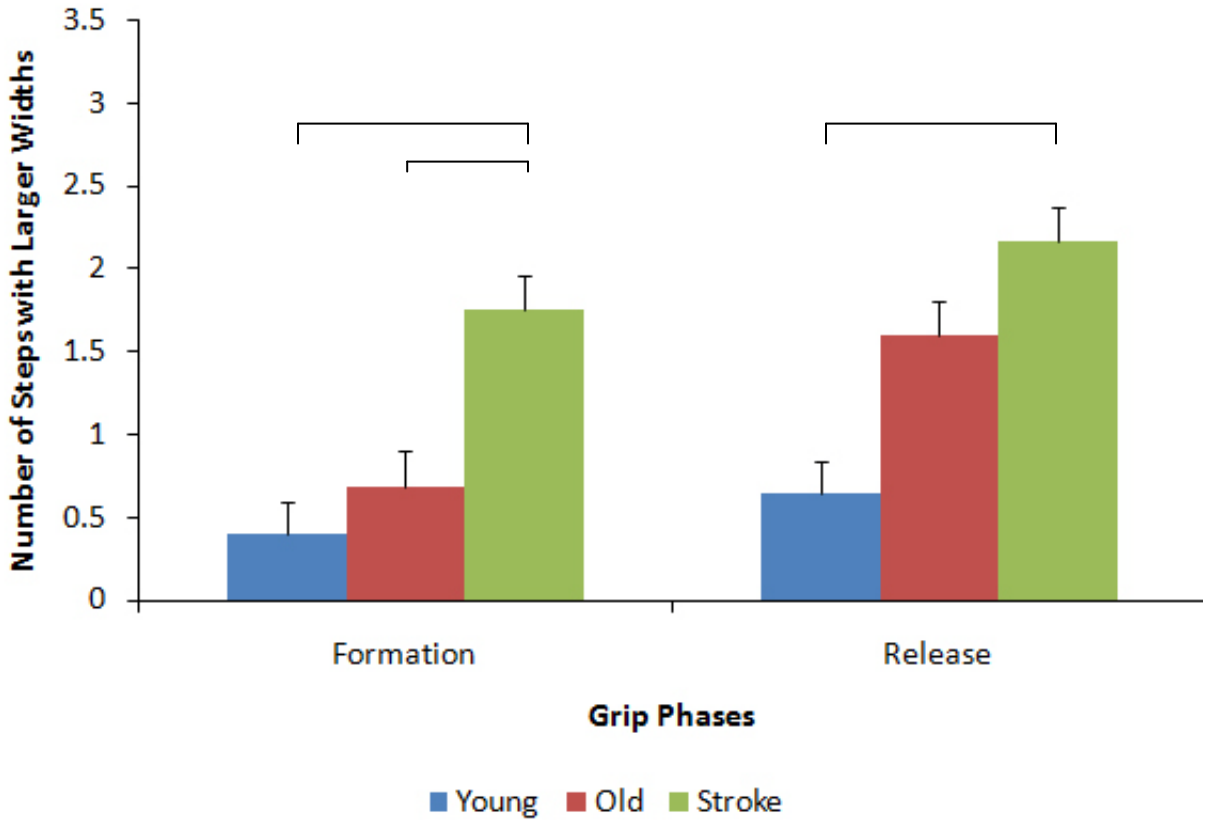

Figure 3-17. Number of steps with larger step widths at 20% MVC/s rate of affected hand of stroke and non-dominant of controls

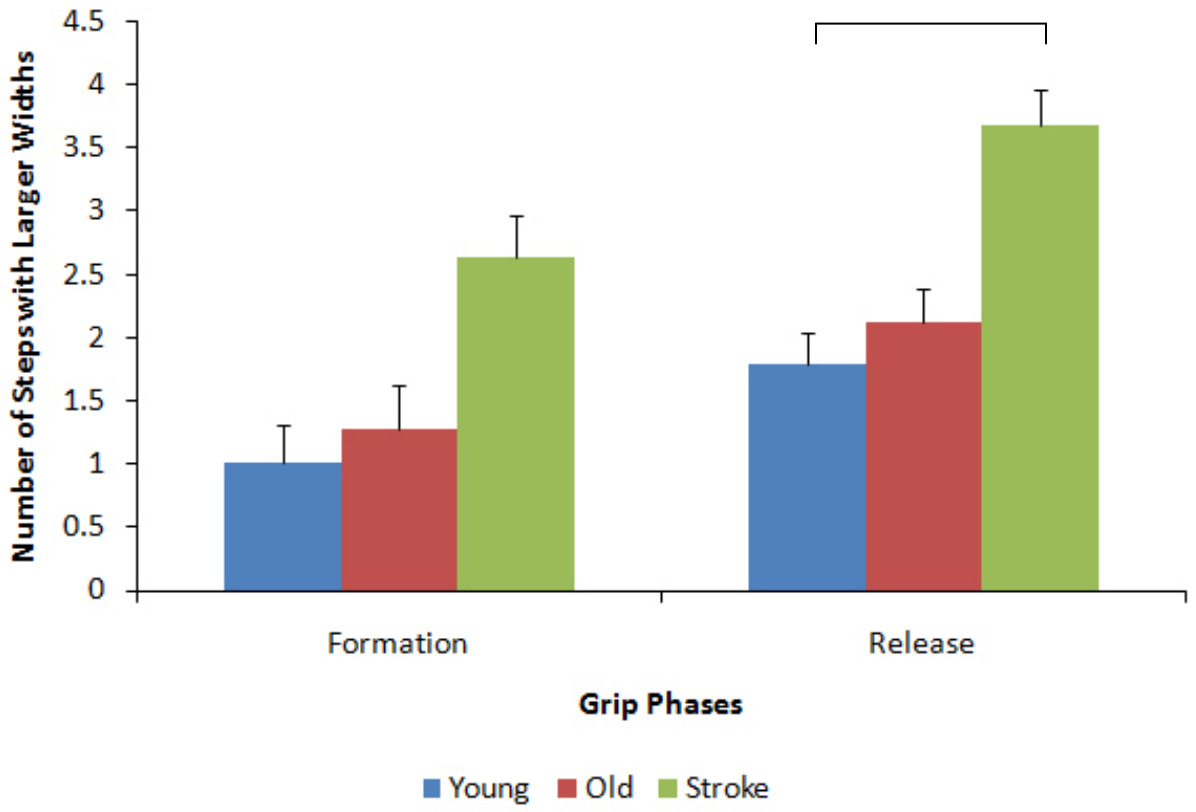

Figure 3-18. Number of steps with larger step widths at 5% MVC/s rate of less-affected hand of stroke and dominant of controls

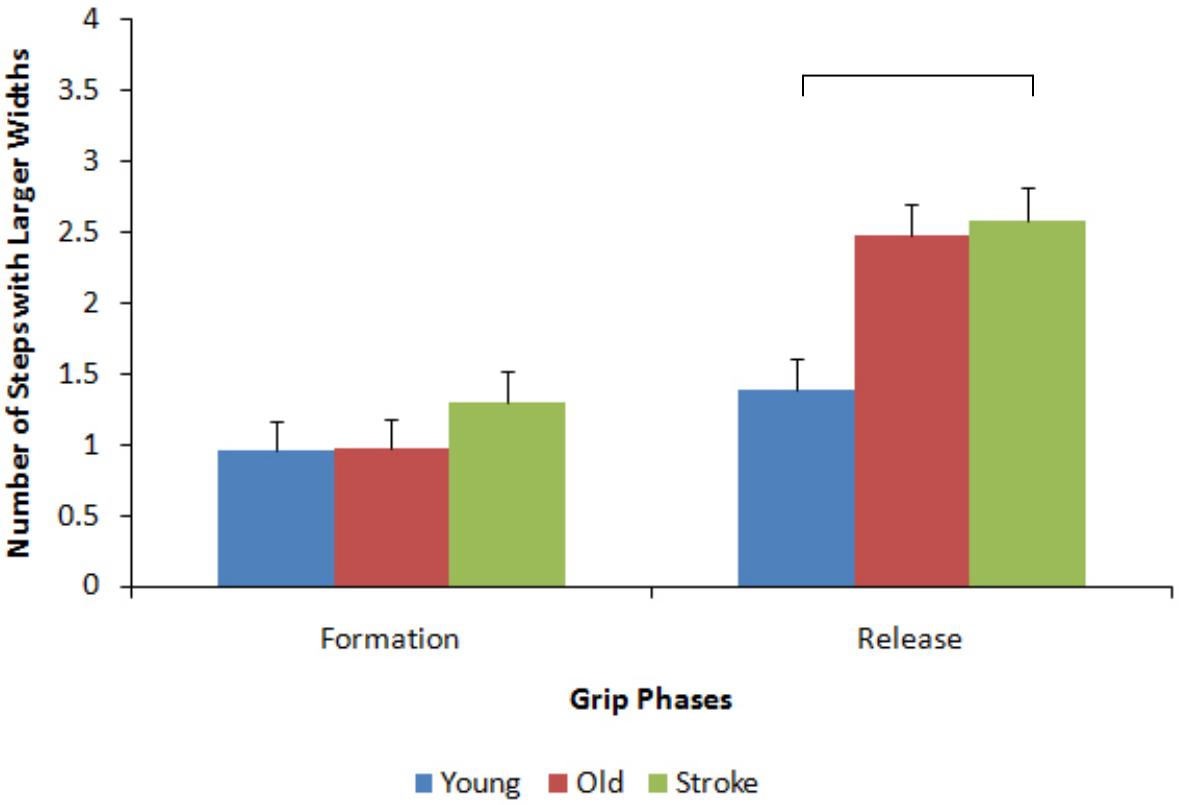

Figure 3-19. Number of steps with larger step widths at 10% MVC/s rate of less-affected hand of stroke and dominant of controls

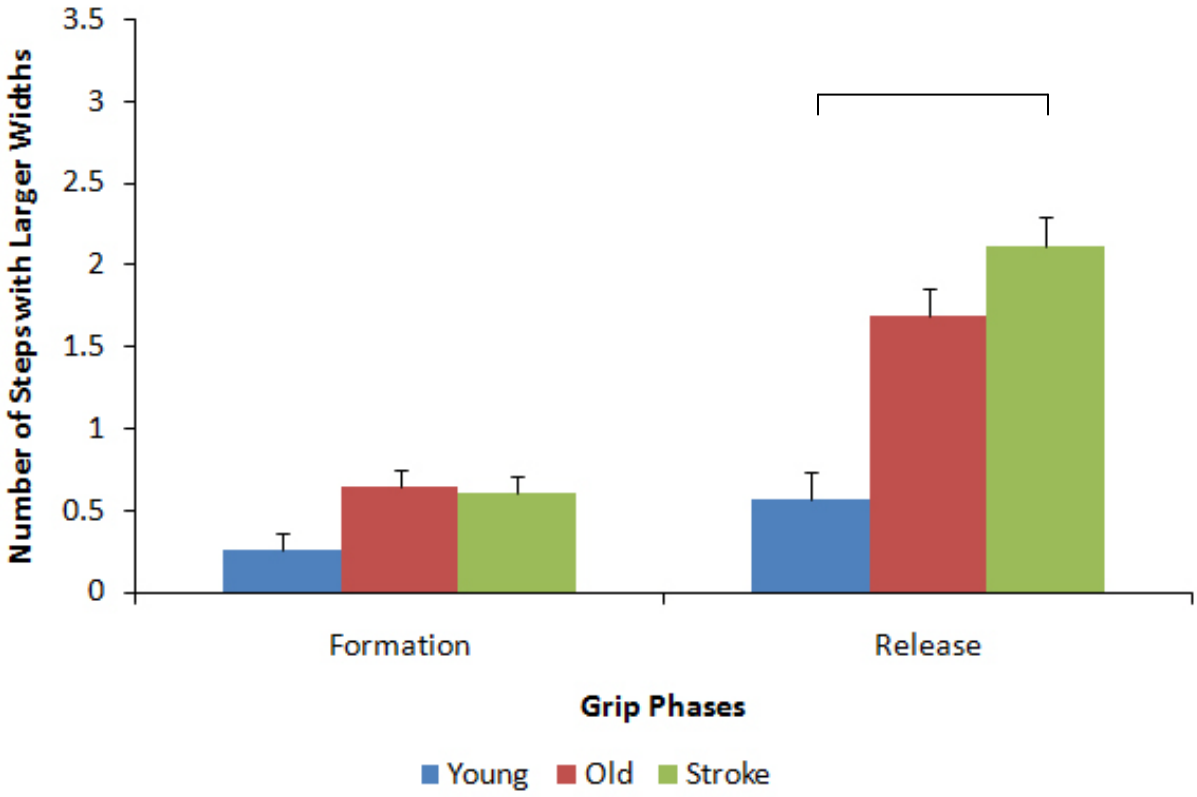

Figure 3-20. Number of steps with larger step widths at 20% MVC/s rate of less-affected hand of stroke and dominant of controls

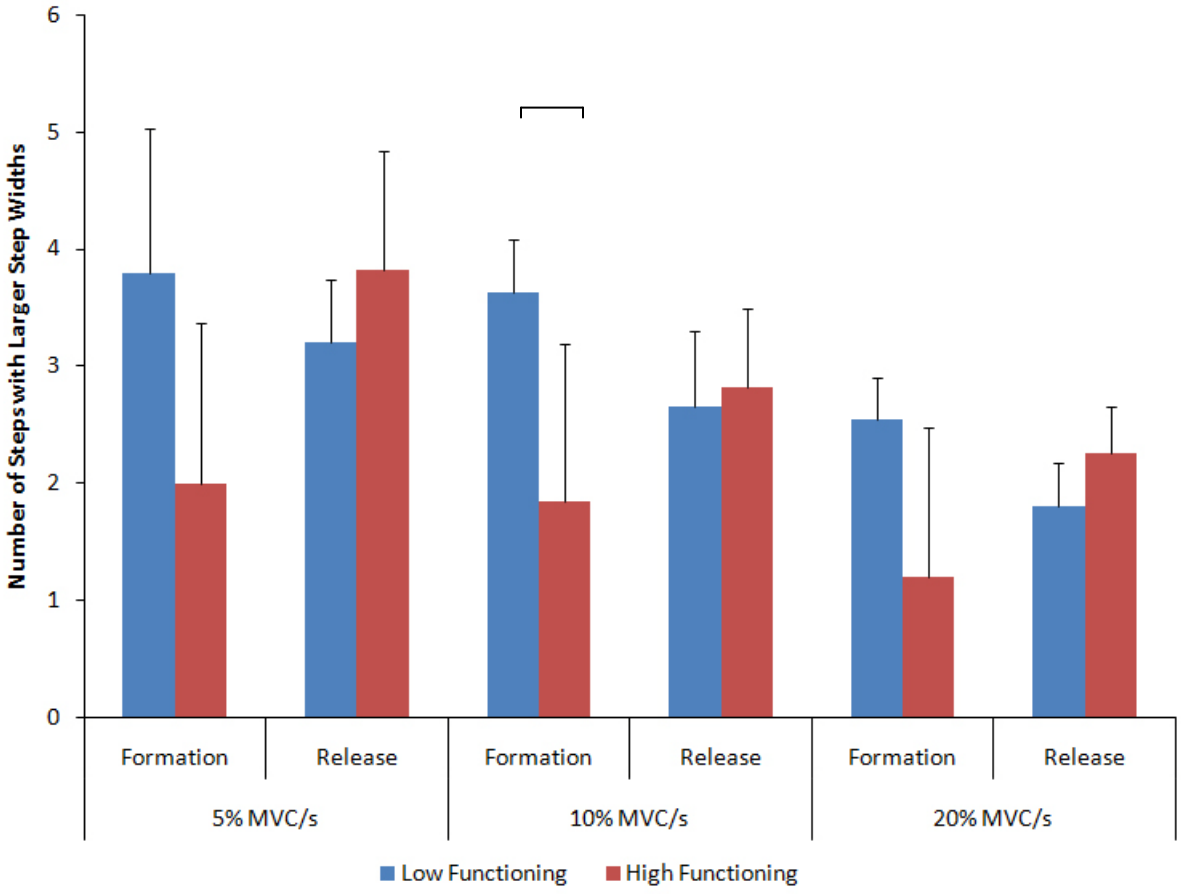

Figure 3-21. Number of steps with larger step widths across rates of affected hand of low functioning versus high functioning stroke participants

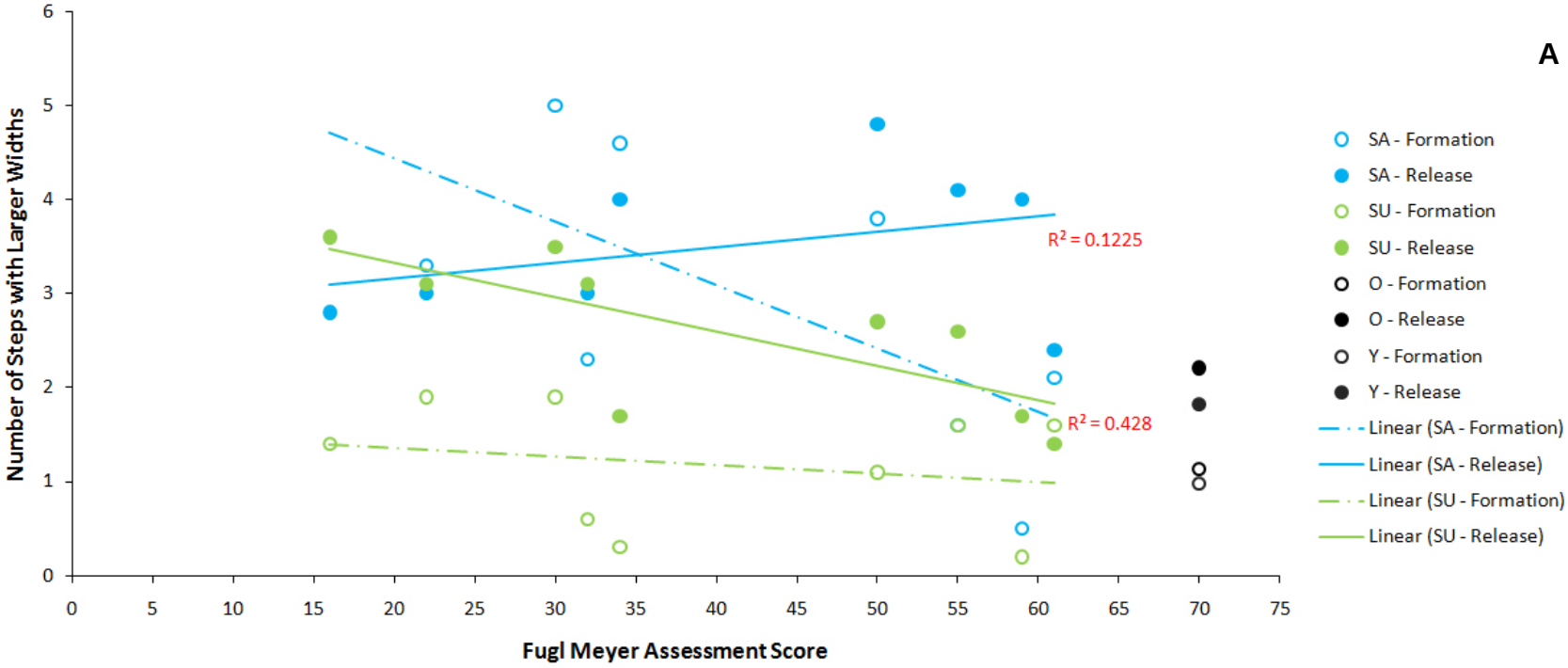

Figure 3-22. Correlation of total number of steps with larger step widths with Fugl Meyer Assessment Scores of stroke group at A) 5% MVC/s Rate. B) 10% MVC/s Rate. C) 20% MVC/s Rate.

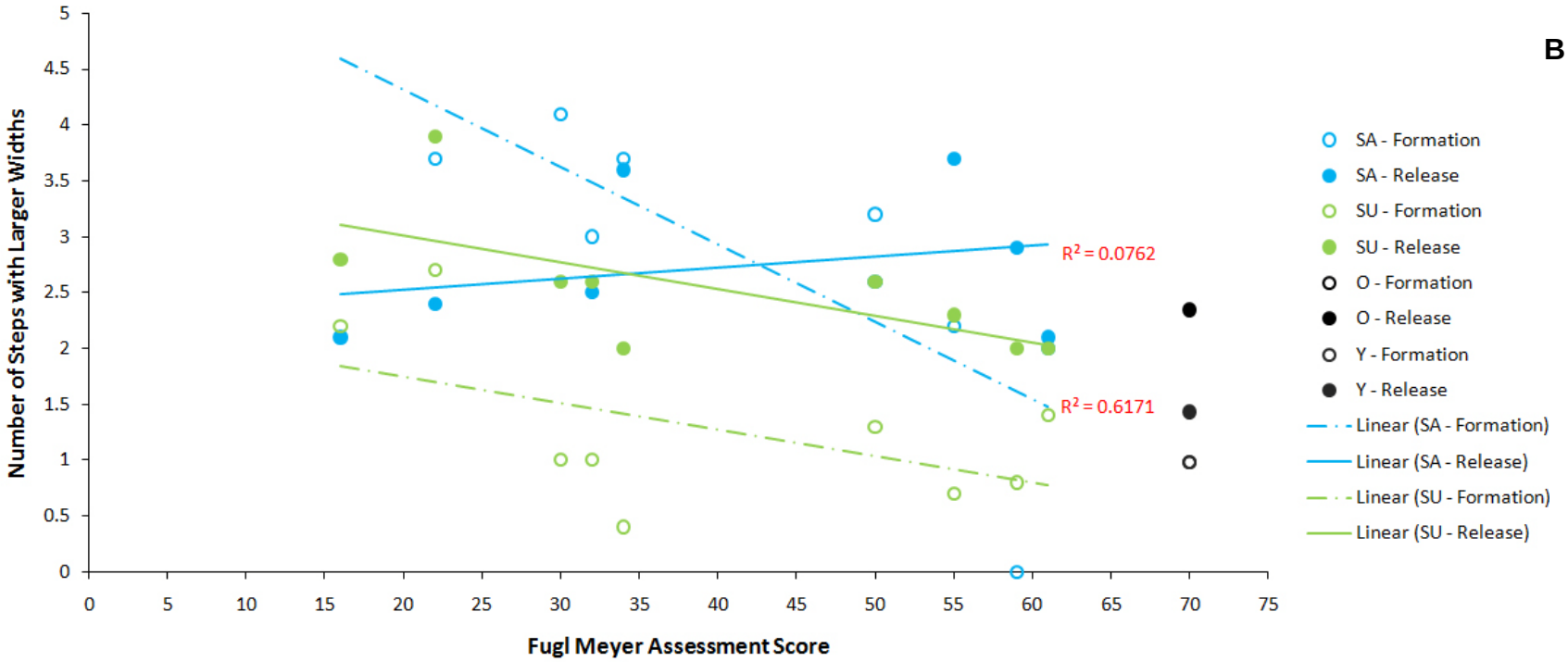

Figure 3-22. Continued

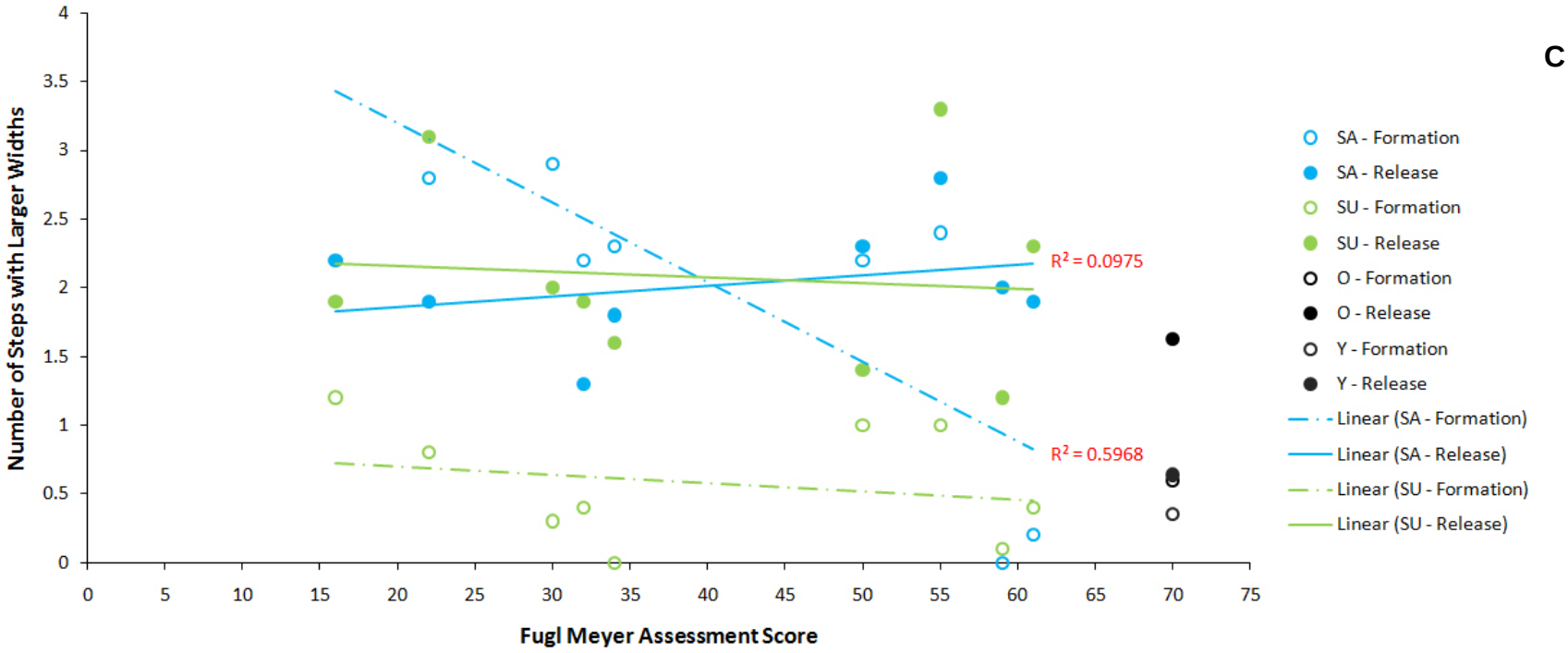

Figure 3-22. Continued

CHAPTER 4
DISCUSSION

The primary finding of the present study supports our hypothesis of tracking performance that chronic stroke participants produce greater force control deficits as measured with root mean square error on the affected hand during the grip release phase than the grip formation and sustained grip phases. Moreover across groups, the root mean square error results revealed that stroke participants have greater tracking deficits on both grip formation and grip release phases compared to young and age-matched healthy adults. Further, chronic stroke survivors are more variable on affected hand than the control groups on sustained and release phases. Similar findings were found for less affected hand. Therefore, we conclude that stroke leads to force control deficits in both hands during both grip phases with greater deficits seen on the release phase.

To examine a stair-stepping phenomenon, we developed a novel approach to quantify the grip release phase. Applying the algorithm to both grip formation and grip release phases indicated stair-stepping evidence. Across all groups there are a greater number of steps during grip formation phase than grip release phase. This finding contradicts our hypothesis and unpublished reports on stepping phenomenon. A discrepancy in our results from the aging literature may account for our methodology adopted for defining step widths based on the rate of force. To understand this novel finding, we investigated steps with larger step widths and found that the grip release phase produced stair-stepping phenomenon which was consistent with the aging literature on visual inspection. Therefore, our results of a higher number of steps with larger steps widths further explains, the visually inspected stair-stepping phenomenon.

In addition, stroke participants had the least number of steps and larger steps compared to healthy elderly and young adults.

The above root mean square error and standard deviation results of tracking performance and number of steps with different step widths can be attributed to five potential mechanisms. First, as it was a tracking task, stroke participants might have problem learning the new task causing greater performance deficits in terms of accuracy and number of steps. Second, stroke participants might not be able to modulate the firing of agonist (flexor) and antagonist (extensor) muscles leading to co-contraction of these muscles. We know that, the power grip task requires some amount of co-contraction to stabilize wrist joint. Further, initial phase of learning a new motor task causes co-contraction. Thus, co-contraction may be one of the potential mechanisms that can explain our findings.

Third, there might be a mismatch between motor unit recruitment (grip formation phase) or de-recruitment (grip release phase) patterns and motor unit firing leading to stair-stepping phenomenon and declined performance. Fourth, visual and perceptual deficits after stroke might have account for some of the deficits observed in force control as the protocol challenged visuomotor system continuously. Fifth, these findings might be because of involvement of higher centers which might have caused motor planning or activation and inhibition deficits. In addition, fatigue might have contributed to reduced performance during grip phases but our secondary analysis on pre MVC-post MVC and trial block disproves fatigue as the potential factor. Moreover, this study was not designed to define or identify the exact mechanism involved in grip formation or grip release phases. Our focus in this first study was to investigate grip phases and rates of

force production for three different types of participants as well as quantify stair-stepping and step widths. We tentatively conclude that combination of these mechanisms lead to greater deficits in grip phases in stroke participants. Hence, further research is required to validate the novel approach to quantifying stair-stepping phenomenon and understand underlying mechanisms.

Motor Learning Deficits after Stroke

Motor learning involves skill acquisition, motor adaptation, and decision making to accomplish goal-directed tasks successfully. Motor learning studies have focused on the effects of various motor learning approaches after stroke (Butefisch, Hummelsheim, Denzler, & Mauritz, 1995; Hanlon, 1996; Platz, Denzler, Kaden, & Mauritz, 1994; Sunderland, et al., 1992). There have been few studies on motor learning deficits (Krakauer, 2006; Takahashi & Reinkensmeyer, 2003; Winstein, Merians, & Sullivan, 1999). Takahashi and colleagues reported that stroke participants have impaired adaptation capabilities on the affected side, attributed to slowness of movement because of weakness and not due to motor learning deficits (Takahashi & Reinkensmeyer, 2003). Based on these motor learning and stroke studies, we conducted a secondary analysis on trial blocks (first 5 trials – 1st trial block and last 5 trials – 2nd trial block) to test if motor learning contributed in gripping deficits. We found no differences across two trial blocks for all grip phases across all rates. Our findings are in line with a recent study which reported that motor learning is preserved post stroke and is related to proprioceptive deficits (Vidoni & Boyd, 2009). None of our stroke participants reported any proprioceptive deficits as measured by proprioceptive component of FMA. In addition, the current procedure consisted of 15 practice trials (five trials at each rate) for each hand, which might have allowed for initial learning of

the novel task. Further, our method tested hand conditions in a blocked order: a) less affected hand practice block, b) affected hand practice block, c) less affected hand experiment block and d) affected hand experiment block. Hence, based on our secondary analysis, experimental procedures, and motor learning literature, we conclude that our findings of deficits on grip phases after stroke are most likely not due to motor learning deficits. However, the present secondary analysis had only five trials per block, so further studies explicitly testing motor learning during grip phases is required before making definitive conclusions.

Co-Contraction of Agonist and Antagonist Muscles

Current findings of tracking performance and number of steps might be attributed to co-contraction phenomenon; activation of the agonist and antagonist muscles, simultaneously. Co-contraction has been reported during early stages of learning a novel skilled task (Franklin, Osu, Burdet, Kawato, & Milner, 2003; Smith, 1981; Thoroughman & Shadmehr, 1999). Also, isometric power grip task itself produces some level of co-contraction among forearm and wrist musculatures to provide stabilization (Basmajian, 1978; Long, 1970; Rasch, 1993). Thus, some level of co-contraction was expected because of the novel power grip task. This might have been accounted with young and age-matched healthy adults as the baseline groups along with practice session. However, Kamper and colleagues reported abnormal EMG activity patterns in the stroke group during isometric flexion and extension torques about the metacarpophalangeal joints of all four fingers (Kamper & Rymer, 2001). In addition, they found decreased EMG activity of extensor muscles of fingers (Kamper & Rymer, 2001) as reported in the wrist, elbow and shoulder muscles (Chae, Yang, Park, & Labatia, 2002b; Dewald, Pope, Given, Buchanan, & Rymer, 1995; el-Abd, Ibrahim, & Dietz,

1993; Gowland, et al., 1992; Hammond, et al., 1988; Hu, et al., 2007; Stoeckmann, Sullivan, & Scheidt, 2009). Comparing these findings with our results might explain possible mechanism for impaired grip phases after stroke. Abnormal co-contraction patterns of forearm flexors and extensors in the stroke group might have caused greater number of steps but in turn decreased number of steps with larger step widths on grip formation phase. One possible reason for this might be increased abnormal activation of extensor muscles with normal flexor activation pattern of forearm during grip formation (predominantly flexion task), which might have caused a greater number of steps with smaller step widths. However, opposite results were found for the grip release phase. A greater number of steps with larger steps widths were found perhaps because of increased flexor activation with diminished extensor activity during the grip release phase (extension task). This explanation needs additional evidence confirming the role of co-contraction during various grip phases. Studies with electromyography and H-reflex techniques combined with kinetic data will advance our understanding.

Motor Unit Recruitment and Derecruitment Patterns

In addition to the above mentioned mechanisms, deficits in motor unit recruitment and derecruitment patterns in stroke participants may have influenced our findings of the total number of steps seen during the tracking task. Control of motor units in normal and aging populations has been studied extensively but, advanced understanding of various motor unit mechanisms in neurological populations like stroke is limited. Stroke leads to changes in the motor unit physiological properties (Young & Mayer, 1982) such as degeneration of motor units in type II (fast twitch) muscle fibers (Brooke & Engel, 1969; Dattola, et al., 1993; Dietz, Ketelsen, Berger, & Quintern, 1986) because of disuse atrophy and these changes may start as early as 2 weeks post-stroke (Hara,

Masakado, & Chino, 2004). Recent surface EMG study of stroke groups showed that recruitment threshold was smaller on affected hand compared to less affected hand suggesting increased activity of low threshold motor units due to degeneration of high threshold motor units (Kallenberg & Hermens, 2009).In addition, according to orderly recruitment of the motor unit principle (size principle), low threshold motor units will be recruited first followed by activation of high threshold motor units. Further, reverse will occur while deactivation of motor units, high threshold motor units will be derecruited first followed by low threshold motor units (Henneman, Somjen, & Carpenter, 1965a, 1965b). Also, orderly recruitment of motor units leads to smooth generation of force which is proportional to the level of force required to recruit the motor unit (Henneman & Olson, 1965; Zajac & Faden, 1985). These two phenomena acting together might explain our findings of total number of steps. During the grip formation phase, the low threshold motor units might have been activated after stroke leading to a smooth increment in force producing greater number of steps with smaller step widths. However, during the release phase, as high threshold motor units are degenerated, we see a greater number of steps with larger step widths causing intermittent type of relaxation of force. The present study did not test this mechanism, therefore, future studies are required to confirm if differences in grip phases is attributed to abnormal motor recruitment or derecruitment patterns.

Visual Perceptual Deficits Post-Stroke

Stroke leads to many visual problems such as low vision, visual field loss, ocular motility disorders and visual perceptual disorders (Dutton, 2003; Falke, et al., 1991; Gilhotra, Mitchell, Healey, Cumming, & Currie, 2002; S. A. Jones & Shinton, 2006; Lotery, et al., 2000; Stone, Halligan, & Greenwood, 1993; Sunderland, Wade, &

Langton Hewer, 1987). A recent multi-center study conducted at 14 acute trust hospitals, revealed that 92% of stroke participants had visual impairment out of 323 stroke participants suspected of having visual deficits (Rowe, et al., 2009). However, stroke participants in the present study didn't report any visual perceptual disturbances on verbal questioning; but no clinical assessment tool was used to confirm visual perceptual deficits. Therefore, we were not able to certainly alleviate a possible contribution of visual perceptual deficits on our tracking results.

Lesion Location

Deficits in grip formation and release phases in stroke group found in this study might be because of differences in brain areas activated during these two phases. Spraker and colleagues using fMRI in normal right-handed young adults reported that during grip formation, left primary motor cortex and bilateral caudate nucleus had greater activity than during grip relaxation. Further, they found that during controlled grip release, there was greater activation of right dorsolateral prefrontal cortex and bilateral anterior cingulate cortex (Spraker, Corcos, & Vaillancourt, 2009). These differential activation patterns during grip phases might have caused differences in the two grip phases of the stroke group because of involvement of these areas. In addition, visually guided grasping movement is controlled by parieto-frontal circuits (Grol, et al., 2007). Also, posterior parietal cortex is involved in spatial perception (Rizzolatti & Matelli, 2003; Ungerleider, 1982), vision-for-action (Milner, 1995; Rizzolatti & Matelli, 2003) and visuospatial attention (Corbetta & Shulman, 2002; Malhotra, Coulthard, & Husain, 2009). Thus, damage to any of these circuits might have lead to different performance deficits which might have influenced our results. However, specific lesion location data was not available from all stroke participants in our study; hence studies exploring these

different circuits in stroke participants are required in future to better understand grip deficits.

Less-Affected Hand Motor Deficits and Stroke

In the stroke literature, there is growing evidence in support of unilateral brain damage leading to significant motor deficits on the less-unaffected hand (Carey, Baxter, & Di Fabio, 1998; Colebatch & Gandevia, 1989; Desrosiers, Bourbonnais, Bravo, Roy, & Guay, 1996; Fisk & Goodale, 1988; Haaland & Harrington, 1989; Halaney & Carey, 1989; Hermsdorfer, Laimgruber, Kerkhoff, Mai, & Goldenberg, 1999; Pohl & Winstein, 1999; Winstein & Pohl, 1995). With this current study, we extended our understanding of the less affected hand motor function deficits to different grip phases. We found consistent results across all rates that our stroke participants showed greatest deficits on grip release phase compared to young adults. Thus, we concluded that stroke leads to bilateral motor deficits. Hence, rehabilitation protocols should emphasize bilateral arm use to improve motor deficits on both hands (Cauraugh, In Press; McCombe Waller & Whitall, 2008).

Low Functioning versus High Functioning Stroke Participants

Our correlation analysis of the total number of steps with clinical measures (FMA, MAS and BBT) suggests three major findings: a) total number of steps on less affected hand on both grip formation and release phases approaches normal values (young and age-matched group means) as the severity of motor deficits reduces, b) similarly, on grip formation phase, as the disease severity improved stroke participants reached normative values, and c) on grip release phase, stroke participants behaved differently as the severity of stroke improved. High functioning participants showed less number of steps compared to low functioning stroke participants, young, and age-matched healthy

adults. Combining all three results, we conclude that on less affected hand low functioning stroke participants have greater impairment on both grip formation and release phases but high functioning participants have normal function. In addition, low functioning participants have impaired grip formation function but high functioning individuals display higher capabilities. However, on grip release phase, low functioning stroke group have near normal function but, release phase shows lesser number of steps in high functioning stroke survivors. Figure 4-1, represents one trial for an individual participant force trace at 5% MVC/s rate from all groups, which explain above mentioned novel findings.

Emerging stroke evidence suggests that weakness of agonist muscles post stroke leads to compromised motor function (R.W. Bohannon, 1989; R. W. Bohannon, 2007; Canning, Ada, & O'Dwyer, 2000; Nadeau, Arsenault, Gravel, & Bourbonnais, 1999; Patten, et al., 2004). Post stroke muscle weakness may arise because of a) loss of muscle mass or motor units (Brooke & Engel, 1969; Dattola, et al., 1993; Dietz, et al., 1986), b) force-velocity relationship of muscles, and c) capacity of nervous control to activate motor units (Chae, et al., 2002a, 2002b; Kallenberg & Hermens, 2009; Patten, et al., 2004). Our low functioning stroke participants also showed weakness with MVC of 123.16N ± 54.48N compared to our high functioning group (MVC – 322.03N ± 97.68N). Thus, muscle weakness or activation deficits evident from stroke literature and our data lead to impaired grip formation phase in low functioning stroke group and near normal grip formation function in high functioning group. Further, we found that grip release phase was impaired more in high functioning group compared to low functioning stroke participants. Deficits in the grip release phase in high functioning individuals

might be because of lack of inhibitory control from higher centers. However, further investigations are required with larger sample size to confirm these novel findings, which will help us answer a few of the most awaited questions in rehabilitation concerning type of intervention for wide variety of stroke population.

In conclusion, this study developed a novel approach to quantify stair-stepping phenomenon in grip formation and release phases during an isometric force tracking task. As discussed above, evidence suggested possible mechanisms. Along with the correlational data of stroke clinical assessment scales, we have speculated possible mechanisms which might be involved in gripping function deficits as supported by our findings. Moreover, combing the low- and high-functioning preliminary evidence with gripping deficits post-stroke and correlation findings, rehabilitation professionals might be able to design treatment protocols based on level of severity to improve hand function post stroke.

Summary

The present study establishes that stroke leads to force modulation deficits for grip release phase of both hands (affected and less-affected). Further, we developed a novel approach to quantify stair-stepping phenomenon to explore possible mechanisms responsible for force modulation deficits in various grip phases. Lastly, this study differentiates stroke population based on the severity of the disease. High functioning stroke survivors showed greater deficits in the grip release phase, which might be because of abnormal derecruitment or inhibition of motor unit patterns. However, force modulation during the grip formation phase was more impaired in low functioning stroke individuals, perhaps because of lack of recruitment or activation of motor units. To further confirm these novel findings and explore possible mechanisms involved in these

force modulation deficits in grip phases post-stroke additional studies are required, which are in preparation for future studies.

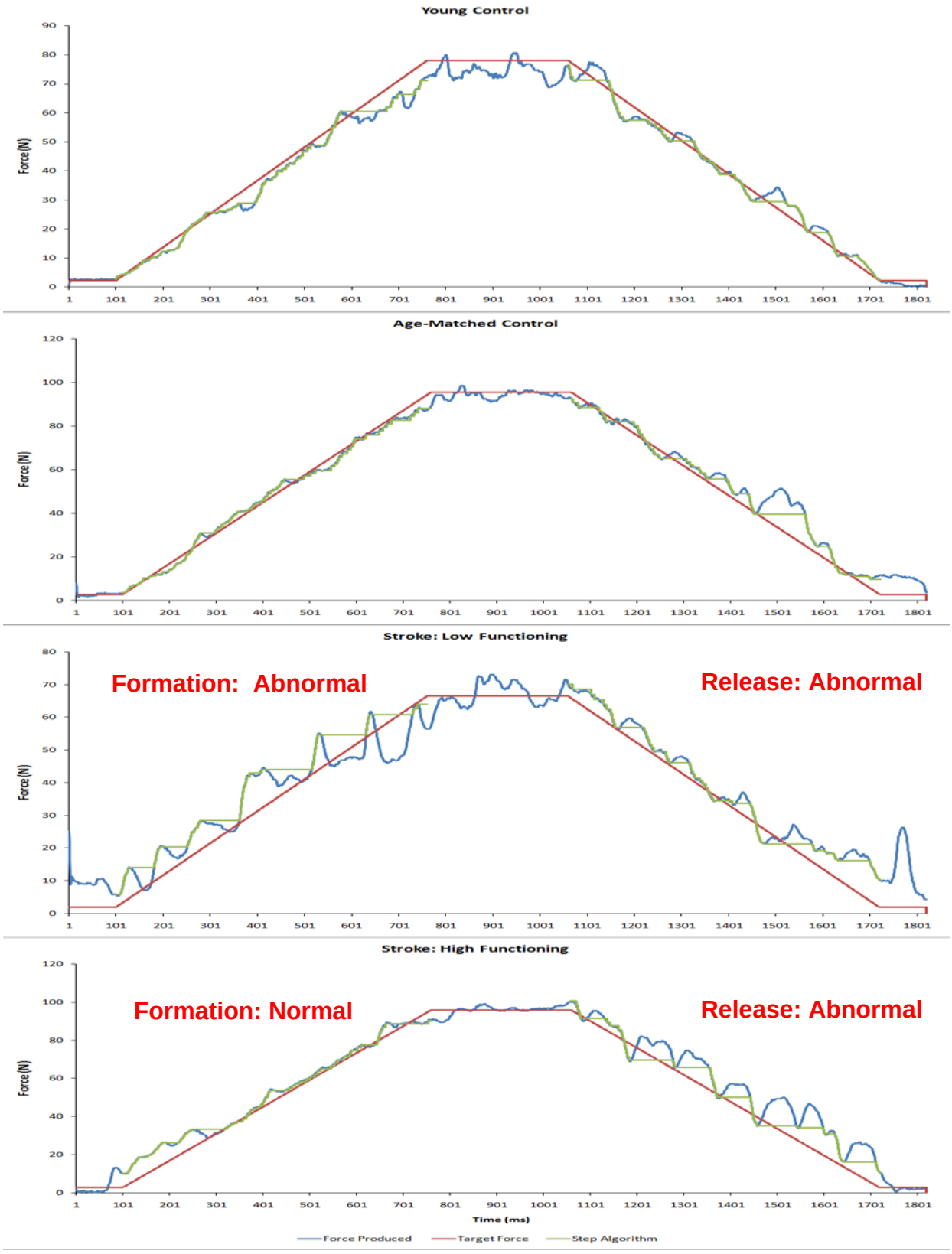

Figure 4-1. Sample force trace of one participant from each group at 5% MVC/s rate

APPENDIX A
FUGL MEYER MOTOR AND SENSORY SCALE

Participant ID
Date
Affected Arm –

A. Shoulder /Elbow/ Forearm

1. Reflexes

	No reflex elicited	Reflex elicited
a. Biceps or finger flexors		
b. Triceps		

2. Flexor Synergy (forearm pronated and crossed over contralateral thigh, bring affected arm to ipsilateral ear)

	Cannot perform at all	Perform partly	Perform faultlessly
a. Retraction			
b. Elevation			
c. Abduction			
d. External Rotation			
e. Elbow Flexion			
f. Forearm Supination			

3. Extensor Synergy (forearm supinated and resting on ipsilateral thigh, bring hand to contralateral knee

	Cannot perform at all	Perform partly	Perform faultlessly
a. Adduction and Internal rotation			
b. Elbow extension			
c. Forearm supination			

4. Mixed Synergy Patterns

	Cannot perform at all	Perform partly	Perform faultlessly
a. Hand to lumbar spine			
	Synergy components begin with onset of movement	Synergy components begin later in movement	No Synergy components
b. Shoulder flexion to 90°, elbow at 0°, forearm neutral or pronated			
c. Forearm pronation – supination, shoulder at 0° elbow flesed at 90°			

5. Isolated Movements without Synergy

	Synergy components begin with onset of movement	Synergy components begin later in movement	No Synergy components
a. Shoulder adduction to 90°, elbow at 0°, forearm pronated			
c. Shoulder flexion 90 to 150°, elbow at0 °, forearm neutral			
	Cannot perform at all	Perform partly	Perform faultlessly
c. Forearm pronation - supination, shoulder flexion 30° elbow at 0°			

6. Normal Reflex Activity (complete only if all tasks in question 5 were performed faultlessly):

	2 or 3 reflexes are markedly Hyperactive	1 reflex is hyperactive or 2 are lively	No more than 1 reflex is lively. None are hyperactive
Biceps, Finger Flexion , Triceps			

B. Wrist (all tests with forearm pronated) :

	No volitional movement	Wrist flexion/extension through partial range	Controlled movement through full range
1. Wrist Flexion/extension with elbow at 90°, shoulder at 0°			
2. Wrist Flexion/extension with elbow at 90°, shoulder at 30°			
	Unable to extend wrist to 15°	Wrist Extension to 15° unable to take resistance	Able to maintain wrist extension to 15° against minimal resistance
3. Wrist stability with elbow at 90°, shoulder at 0°			
3. Wrist stability with elbow at 90°, shoulder at 30°			
	Unable	Incomplete or uncontrolled motion	Complete controlled motion
5. Wrist circumduction with elbow at 90°, shoulder at 0°			

C. Hand

	Cannot perform at all	Perform partly	Perform faultlessly
1. Fingers mass flexion			
2. Fingers mass extension			
	Unable to perform	Performed weakly without resistance	Performed with great resistance
3. Pip-DIP hook grasp: MP joints extended and Pips +DIPs are flexed			
4. Thumb adduction with paper : all other joints at 0°			
	Unable	Able to hold	Hold firmly with tug
5. Pincer grasp with pencil			
6. Cylinder grasp with small can			
7. Spherical grasp with tennis ball			

D. Coordination / Speed (Finger to Nose 5 X with eye closed)

	Marked Tremor or dysmetria	Slight tremor or dysmetria	No tremor or dysmetria
1. Tremor			
2. Dysmetria			
	$\geq$ 6 seconds differ b/w hands	2-5 seconds differ b/w hands	< 2 seconds differ b/w hands
3. Time			

Time (sec) Less-affected Arm:
Time (sec) Affected Arm:

E. Sensation Scale

		Anesthesia	Hyperesthesia or Dyesthesia	Normal
Upper Arm	Affected			
	Unaffected			
Palm of Hand	Affected			
	Unaffected			

F. Proprioception Scale:

	No Sensation	¾ answers Correct	Normal
1. Shoulder			
2. Elbow			
3. Wrist			
4. Thumb			

Upper extremity Fugl – Meyer Score

APPENDIX B
MODIFIED ASHWORTH SCALE

Participant ID –
Date –
Affected Arm –

Left / Right	Muscle under stretch	Score

The modified Ashworth scale

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

APPENDIX C
DEMOGRAPHICS

Subject ID_____

Date_____

1) Date of Birth:

2) Sex:

3) Dominant Limb:

4) Affected Side (Left or Right):

5) Date of first Cerebro-Vascular Accident:

6) Type of Stroke (Ischemic /Hemorrhage):

7) # of incidence of Stroke (Date of last incidence if more than 1):

8) Medication:

9) Any other condition:

10) Mini mental state Examination score:

11) Box and Block:

12) Modified Ashworth Score:

13) Fugl Meyer (Upper extremity):

14) Contact Address and email:

15) Phone

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BIOGRAPHICAL SKETCH

Sagar Naik was born in Navsari, a small town in the state of Gujarat, India. He received the Bachelor of Science in Physical Therapy from The Sarvajanik College of Physiotherapy, Surat, India in 2007. During the same year, he started the Master of Science program at the Applied Physiology and Kinesiology Department of University of Florida, Gainesville. Sagar received his Master of Science from the University of Florida in the fall of 2009 with concentration in motor learning and control under the guidance of Dr. James Cauraugh. He intends to continue pursuing research in stroke rehabilitation.