

Obstacle avoidance behaviour during reaching after stroke

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STATEMENT OF AUTHORSHIP

I, Melanie Christine Baniña, certify that I am the primary author of this thesis. I claim full responsibility for the content and style of the text included herein.

CONTRIBUTION OF COLLABORATORS

All chapters were prepared by Melanie C. Baniña under the supervision of Dr. Mindy F. Levin. Study design, data collection and analyses were completed by Ms. Baniña under the supervision of Dr. Levin. Subject recruitment and data collection assistance were performed by Rhona Guberek and Ruth Dannenbaum. The virtual environment used for the experiment was developed by Christian Beaudoin, assisted by Ms. Baniña. All Matlab codes used for data analyses were written by Dr. Valeri Goussev. Statistical advice was given by Gevorg Chilingarian. French translation of the abstract was completed by Dr. Andréanne Blanchette and Maxime Robert. Institutional affiliation was provided by Dr. Levin. Funding was provided by the School of Physical and Occupational Therapy (Richard and Edith Strauss Doctoral Fellowship, 2008-2010) and by Dr. Levin (CIHR grant, 2010-2014).

STATEMENT OF ORIGINALITY

This thesis contains no material that has been published elsewhere, except where specific references are provided. The study and results presented in Chapters 3, 4 and 5 represent original contributions to knowledge in the field of sensorimotor control of the upper limb after stroke. Specifically, this thesis is concerned with the measurement of deficits in higher-order sensorimotor function, such as obstacle avoidance during reaching, in individuals who are well-recovered post-stroke. Chapter 3 presents the research methodology of this project. Chapter 4 presents results from this project. Chapter 5 presents a discussion about the results of this project and how the results contribute original material to the field of sensorimotor control of the upper limb after stroke.

All data presented in this thesis were collected at the Feil & Oberfeld Research Centre, a site of CRIR (Centre de recherche interdisciplinaire en réadaptation du Montréal métropolitain). The centre is situated at the Jewish Rehabilitation Hospital (Laval, QC), which is affiliated with McGill University. The protocols used in this study have been approved by the Research Ethics Board of CRIR.

ABSTRACT

The ability of a person to perform activities of daily living depends in great part on being able to reach with their arms and interact with objects in the environment. After a stroke, arm motor control is usually disrupted and results in arm paresis. The level of arm impairment varies widely among stroke survivors, but 40-78% of post-stroke individuals recover a good amount of arm function. Clinical evaluation of these individuals demonstrates that their joint ranges of motion, strength, flexibility, sensation and proprioception fall within normal physiological limits. Based on their high level of recovery, these individuals should not have any major activity limitations, yet several studies have shown that well-recovered post-stroke individuals do not use their arm to the expected amount in everyday life activities. Decreased use may be associated with undetected impairments identified only when individuals attempt higher-order motor tasks requiring complex coordination and quick changes in movement. One higher-order motor task, avoiding obstacles while reaching, commonly occurs in everyday environments but is not routinely assessed by clinical scales.

The goals of this thesis were to examine how well-recovered post-stroke and healthy individuals avoid obstacles during reaching and relate obstacle avoidance reaching performance with clinical measures of functional arm use. There were three main objectives of this thesis. The first objective was to identify the obstacle avoidance behaviours used in the control and stroke groups. The second objective was to determine if clinical assessments of arm function and arm use in activities of daily living related to task success. The final objective was to determine if changes in obstacle avoidance ability and clinical measures occurred in the stroke group one year after the initial visit.

A cross-sectional study of a volunteer sample of well-recovered post-stroke individuals (n=17) and healthy controls (n=12) was conducted. Participants performed a reaching task in a virtual environment in which they reached into a refrigerator and touched a juice bottle. In a test block of 60 trials, a sliding refrigerator door would randomly close from the left or right side. Obstacle avoidance ability during reaching was compared between groups. Overall success rates, task performance and movement quality variables were recorded.

We analyzed the reaching behaviour of both groups while avoiding the door ipsilateral to the affected arm. The stroke group was less successful and had a reduced margin of error compared to controls. The stroke group mainly used trunk rotation to avoid the door, while the control group used trunk and elbow flexion. No relationships were found between task success rates and clinical arm function assessments in the stroke group. However, larger margins of error in the stroke group were related to higher Box and Blocks Test scores, which was a measure of manual dexterity. When 10 of the post-stroke individuals participated in a second visit, comparison of the performance in each visit revealed no differences in reaching strategies.

Results of this thesis suggest that in well-recovered post-stroke individuals, residual movement deficits can be revealed when performing a challenging motor task. The potential of using challenging tasks to identify higher-order motor control impairments should be considered when assessing motor recovery in well-recovered post-stroke individuals.

ABRÉGÉ

La capacité d'une personne à effectuer des activités de la vie quotidienne dépend en grande partie de sa capacité à effectuer un mouvement d'atteinte avec ses bras et à interagir avec des objets dans son environnement. Suite à un accident vasculaire cérébral (AVC), le contrôle moteur du bras est affecté et résulte à une parésie du bras. Bien que la sévérité des déficiences au niveau du bras varie chez les individus avec un AVC, 40 à 78% de ceux-ci récupéreront une bonne fonction. L'évaluation clinique de ces individus démontre que leurs amplitudes articulaires, leur force, leur souplesse, leur sensation et leur proprioception se situent dans les valeurs physiologiques normales. Considérant leur niveau de récupération, ces individus ne devraient pas présenter de limitation importante dans leurs activités. Cependant, des études ont démontré que malgré une bonne récupération suite à l'AVC, ces personnes n'utilisent pas leur bras dans leurs activités tel que souhaité. Cette diminution d'utilisation pourrait être associée à des déficiences détectables seulement lors de tâches motrices de haut niveau nécessitant une coordination complexe et des changements rapides dans les mouvements. Une tâche motrice de haut niveau, qui consiste à éviter des obstacles lors d'un mouvement d'atteinte, se produit souvent dans notre environnement de tous les jours, mais est rarement évaluée.

Les objectifs de cette thèse sont d'examiner comment les individus ayant bien récupéré suite à un AVC et des individus en santé évitent des obstacles durant des tâches d'atteintes et d'associer leurs performances avec des mesures cliniques évaluant les fonctions du bras. Il y avait 3 objectifs principaux à cette thèse. Le premier objectif consistait à identifier les stratégies utilisées pour éviter un obstacle. Le deuxième objectif était de déterminer si l'endroit de la lésion, ainsi que la fonction et l'utilisation du bras lors des activités de la vie quotidienne étaient reliés au succès de la tâche. Le dernier objectif était de déterminer s'il y avait eu des changements au niveau de la capacité à éviter un obstacle et des mesures cliniques chez les participants ayant subi un AVC, un an après leur visite initiale.

L'étude d'un groupe d'individus volontaires avec un AVC (n=17) et d'un groupe contrôle de participants sains (n=12) a été effectuée. Les participants ont effectués une tâche d'atteinte dans un environnement virtuel dans lequel il devait atteindre un réfrigérateur et toucher une boîte de jus. La porte du réfrigérateur se fermait aléatoirement du côté droit ou gauche pour 60 essais. La capacité à éviter un obstacle lors d'un mouvement d'atteinte dans un environnement virtuel a été

comparée entre les groupes. Le taux de succès, la performance et la qualité du mouvement ont été enregistrés.

Nous avons analysé les mouvements d'atteintes des deux groupes lorsque les individus évitaient la porte. Le groupe d'AVC a moins bien réussi et plus petite marge d'erreur comparativement aux contrôles. Le groupe d'AVC utilisait la rotation du tronc afin d'éviter la porte, tandis que le groupe contrôle utilisait la flexion du tronc et du coude. Aucun lien entre la réussite de la tâche et des fonctions des membres supérieurs n'a été trouvé pour le groupe d'AVC. Cependant, des larges marges d'erreurs dans le groupe d'AVC ont été liées avec un résultat élevé du test Box and Block, qui mesure la dextérité manuelle. Lorsque dix des individus post-AVC se sont présentés à la deuxième visite, la stratégie de mouvement d'atteinte était similaire que la première visite.

Les résultats de cette thèse suggèrent que les individus ayant bien récupéré suite à un AVC présentent des déficits moteurs résiduels lorsqu'ils doivent effectuer une tâche motrice complexe. Le potentiel d'utilisation des tâches complexes pour identifier les déficiences motrices de haut niveau devrait être considéré lors de l'évaluation de la récupération motrice des personnes ayant eu un AVC.

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LIST OF ABBREVIATIONS

3D VE – three-dimensional virtual environment
ADL – activities of daily living
BBT – Box and Block Test
CMA – cingulate motor area
CMSA – Chedoke-McMaster Stroke Assessment
CSS – Composite Spasticity Scale
DP – divergence point
EPV – endpoint peak velocity
ETL – endpoint trajectory length
ETPV – endpoint time to peak velocity
fMRI – functional magnetic resonance imaging
FMA – Fugl-Meyer Assessment
GDS – Geriatric Depression Scale
IC – index of curvature
IPL – inferior parietal lobule
LBT – Line Bisection Test
M1 – primary motor cortex
MoCA – Montreal Cognitive Assessment
MAL – Motor Activity Log
MAL-SES – Motor Activity Log Self-Efficacy Scale
PE – physical environment
PMd – dorsal premotor cortex
PMv – ventral premotor cortex
PPC – posterior parietal cortex
RPS – Reaching Performance Scale for Stroke
S1 – primary somatosensory cortex
S2 – secondary somatosensory cortex
SMA – supplementary motor area
SPL – superior parietal lobule
WMFT - Wolf Motor Function Test

ORGANIZATION OF THESIS

This thesis is the traditional, monograph format, and is prepared according to the McGill Graduate and Postdoctoral Studies guidelines of thesis preparation.

Chapter 1

Chapter 1 is a literature review composed of 5 sections.

Chapter 2

Chapter 2 outlines the rationale for the thesis, including a synopsis of the literature review followed by the objectives and hypotheses.

Chapter 3

Chapter 3 is a general methods section.

Chapters 4

Chapter 4 contains a summary of the study results.

Chapters 5

Chapter 5 is a discussion of the results with respect to the ideas presented in the literature review (Chapter 2).

Chapter 6

Chapter 6 presents the clinical implications, future directions, limitations and conclusions from the results of the thesis.

References

This section contains a compiled list of references for all chapters.

Appendices

Clinical assessments and the study consent form are included.

The ability of a person to participate in activities of daily living depends in great part on their ability to coordinate trunk, arm and hand movements in order to perform effective interaction of the upper limb with objects in the environment. Performance of everyday activities appears fairly simple and automatic for people with healthy sensorimotor systems. However, this apparent ease and simplicity of movement control does not reflect the fact that controlling reaching movement requires the coordination of a complex network of physiological and psychological processes. Through extensive investigation of reaching in animal and human models, some processes identified include perception of the environment; intention, planning and execution of action; and online proprioceptive and visual control to fine tune the precision and accuracy of the final movement. The term “online” refers to the ability to modify movements as they are occurring. Healthy individuals coordinate cognitive and sensorimotor processes with ease, but injury of the nervous system can upset the balance of control.

One common cause of neural injury affecting the upper limb is stroke. A stroke is the death of brain tissue caused by an occlusion or rupture of a blood vessel supplying the brain (Mackay & Mensah, 2004). Approximately 75.5% of acute stroke patients experienced upper limb hemiparesis or weakness and lack of control of movements on one side of the body (Rathore et al., 2002). In the chronic stage (>6 months) post-stroke, upper limb hemiparesis is still present in approximately 50-75% of survivors (Jørgensen et al., 1995; Mayo et al., 1999; Mayo et al., 2002; Kwakkel et al., 2003). Although individuals may regain some functional use of the upper limb, movements often remain clumsy and slow (Broeks et al., 1999; Brodal 1973; Gandevia 1982; Rode et al., 1996). When a stroke causes hemiparesis, individuals experience a limitation of their abilities to independently perform activities of daily living. However, a large portion of individuals recover well after their stroke (“mild stroke”; Go et al., 2014). Individuals with mild stroke impairments may achieve almost complete recovery, based on clinical motor performance and sensorimotor function measures. Indeed, 40-78% of individuals who have had a stroke have no or mild neurological deficits (Jørgensen et al., 1995, Mayo et al., 1999). Based on their high level of recovery, these individuals should not have any major activity limitations, yet several studies have shown that well-recovered individuals do not use their arm to the expected amount

in everyday life activities (Platz & Denzler, 2002; Carlsson et al., 2004; Edwards et al., 2006; Carlsson et al., 2009; Rand & Eng, 2012).

The disconnect between the “near-normal” clinical scores and the reduced arm use in daily activities indicates that clinical testing is not detecting the level of impairment found in well-recovered post-stroke individuals. Some studies found mildly impaired individuals were just as successful as healthy controls in completing the experimental tasks (Platz et al., 2001; Alt Murphy et al., 2011). However, the same studies detected that tasks were completed using abnormal compensatory upper limb and trunk movement patterns instead of recovering movement patterns observed in healthy subjects (Levin et al., 2009). It has been repeatedly demonstrated that the use of compensatory movement patterns allows the accomplishment of motor tasks despite motor limitations (Levin et al., 2002). However, simply observing performance outcome does not provide any insight into the motor control limitations.

In well-recovered individuals, basic sensorimotor upper limb function is within normal physiological limits in terms of joint ranges of motion, strength, flexibility, sensation and proprioception (Go et al., 2014). However, standard clinical scales likely do not detect impairments of higher-order motor control. Examples of higher-order sensorimotor functions include temporal-spatial coordination between different body segments (e.g. trunk and arm), and the ability to correct body movements when obstacles or perturbations occur in the environment. The operational definition of higher-order motor control used for this thesis is the ability to make rapid online corrections with the arm when avoiding obstacles while reaching. Higher-order motor control does not imply direct measure of cellular activity in the brain. To identify the underlying motor deficits contributing to the lack of arm-use in well-recovered individuals, it is necessary to study movements requiring a higher level of motor control. The ability to quantify higher-order motor control impairments may then enhance upper limb rehabilitation techniques and improve arm function of well-recovered post-stroke individuals.

In the literature review, the following topics will be reviewed:

1. Behavioural characteristics of reaching and obstacle avoidance in healthy individuals
2. Neural control of visually guided reaching
3. Stroke pathology and the development and persistence of upper limb deficits after stroke

4. Behavioural and neurophysiological changes of reaching after stroke, particularly in individuals who have mild stroke impairments
5. Gaps in the clinical measurement of higher-order motor control.

CHAPTER 1: LITERATURE REVIEW

1.1 REACHING IN HEALTHY INDIVIDUALS

Reaching can be described as the ability to coordinate the arm and hand in a purposeful aimed movement. The ability to reach and interact with the surrounding environment is an important component in a wide variety of everyday tasks such as self-grooming, food preparation, housecleaning and performing work tasks. Reaching has been described using kinematic characteristics of endpoint path and velocity; joint angles, angular velocities; arm muscle activity and their sequences of activity; temporal and spatial coordination among the joints; contributions of the rest of the body to reaching; and the relationships amongst these characteristics. Section 1.1 provides a description of reaching behaviour.

1.1.1 BEHAVIOURAL DESCRIPTION OF REACHING IN HEALTHY INDIVIDUALS

Reaching behaviour has been studied extensively and described using multiple characteristics such as hand trajectory formation, velocity and acceleration profiles, and temporal and spatial relationships between movements of multiple joints and muscles.

Previous studies of upper limb reaching movements highlighted some typical behaviour. When comparing multiple trials of a hand movement from a start position to a specific target in front of a person, the movement path of the hand was relatively straight and consistent across trials (Morasso 1981; Soechting & Lacquanti, 1981). Also, the hand velocity profiles were smooth, unimodal, symmetrical, bell-shaped curves (Soechting & Lacquanti, 1981; Abend et al., 1982). When subjects reached to different targets with different speeds, the velocity profiles were compared by normalizing speed and distance across trials. The resulting velocity profiles of the endpoint were also unimodal, symmetrical, bell-shaped curves and similar across the different targets (Atkenson & Hollerbach, 1985). When accuracy was not the primary goal of the reaching movement, peak velocity occurred at the midpoint of the trajectory and acceleration and deceleration times were equal in length. For movements requiring accuracy, deceleration times lengthened since more time was required for the aiming phase of movement (Soechting 1984). In addition to the acceleration/deceleration change in time course, final acquisition of the target

occurred after the rapid initial movement in a secondary refining movement close to the target (Beggs & Howarth, 1972; Carlton 1980; Atkeson & Hollerbach, 1985; Flanagan et al., 1993). An important explanation of the relationship between accuracy and speed was initially developed by Paul Fitts (1954). His model predicted that the time required to move rapidly to a target was a function of the size of the target and distance to the target. The results from the reaching accuracy studies supported the model – movement time increased with the need for accuracy.

Another characteristic of aimed arm movements was that the profiles of endpoint peak velocities and accelerations/decelerations kept their features appropriate for the specific reaching task but were scaled to the movement distance (Gordon et al., 1994). If subjects were instructed to use a curved hand path, the resulting velocity profile was multi-peaked. The more curved the path, the more peaks that occurred in the hand velocity profiles (Abend et al., 1982). In the multi-peaked profiles, the lowest velocity between peaks corresponded to the time at which the curvature was maximal (Abend et al., 1982). In summary, unrestricted reaching movements had straight endpoint movement paths; smooth, unimodal bell-shaped angular velocity profiles of the endpoint across different movement speeds or durations; and peak velocities of the endpoint occurring in the middle of the movement trajectory. Restricting different aspects of reaching revealed other invariant characteristics of the endpoint: curved hand paths had multi-peaked velocity profiles and the deceleration phase of a forward reach lengthened when accuracy was explicitly required for the endpoint to reach a target.

Another group of variables studied in reaching paradigms were the angular velocities and trajectories of the shoulder and elbow. Each of the joint angular velocities changed according to the target location and distance from the individual, but the shoulder and elbow angular velocity changes were tightly coupled. First, the ratio of the elbow to shoulder joint excursions was equal to the ratio of their respective angular velocities (Soechting & Lacquanti, 1981; Abend et al., 1982). For example, when the change in shoulder angle was greater than the change in elbow angle (such as when reaching for a target just above the head level directly in line with the shoulder), the maximal velocity at the shoulder was also greater than the maximal velocity of the elbow. Also, when the angular excursions were equal between the joints, the maximal joint velocities were equal (Soechting & Lacquanti, 1981). The relationship between the shoulder and the elbow was maintained even when wrist position was varied at the end of the movement

(Lacquaniti & Soechting, 1982). The second aspect of the velocity relationship concerned time to peak angular velocity of each joint. Even though the magnitudes of the maximal shoulder and elbow velocities varied depending on the target location, the joint velocities reached their peak at the same time (Soechting & Lacquaniti, 1981). In other words, greater changes in angular velocities in one joint were completed in the same amount of time as smaller changes in angular velocities of the other joint. For the third aspect of the angular velocity relationship, shoulder and elbow angles changed equally in the deceleration phase of the reaches to all target locations (Soechting & Lacquaniti, 1981). Therefore, shoulder and elbow angular velocities were tightly coupled during upper limb aimed movements but scaled according to the target distance from the individual.

Another important characteristic identified in aimed pointing movements in three-dimensional space is motor equivalence (Bernstein, 1967; Berkinblit et al., 1986; Feldman & Levin, 1995). For example, the upper limb has many degrees of freedom of movement, meaning that the shoulder, elbow and wrist could each have a very large number of postures during the reach movement. There would then be the possibility of a large number of interjoint coordination combinations and movement trajectories over multiple trials, but the endpoint would consistently arrive at the target. A similar situation exists for the muscles of the arm. Moving the hand forward along a set trajectory could hypothetically be completed with an ideal order of muscle activation. However, since there are many possible shoulder, elbow and wrist postures during the reach movement, it follows that muscle activity during reaching can also vary greatly in magnitude and direction. The multitude of ways that one motor task can be performed is an example of the concept of redundancy. In terms of motor control, redundancy can be viewed either as having multiple movement plans from which to choose in order to complete a motor action, or having access to a wide range of degrees of freedom within which to perform a motor action.

To summarize, unrestricted aimed reaching movements have smooth, straight endpoint trajectories that finish accurately on the intended target but can have great variability in the segment and joint positions as well as muscle activity of the arm (Bernstein 1967; Scholz & Schöner, 1999; Krishnamoorthy et al., 2007). Although the biomechanics of reaching movements is well understood, the mechanisms by which the movement adjustments occur is an

area that needs to be further developed. In order to study movement adaptations, some commonly used paradigms such as target shifts and obstacle avoidance will be discussed in the next section.

1.1.2 MOVEMENT CORRECTION IN REACHING OF HEALTHY INDIVIDUALS

Real-world environments are often cluttered and unpredictable. Take for example a work desk cluttered with paper, a computer station, a water bottle and a full coffee mug. A person must be able to control their arm and hand movement so that they can reach for the water bottle without hitting and spilling the coffee mug contents onto other objects. Furthermore, if a cat were to suddenly jump onto the desk and obstruct the hand path as the person was reaching for the bottle, the person would have to quickly change their hand path to avoid hitting the cat and still successfully pick up the water bottle. Another example of an unpredictable task would be a novice catcher trying to catch the curve ball of an experienced baseball pitcher. When the pitch is initially thrown, the catcher may assume that the ball is travelling straight to them and thus prepare the catching position of their hand and arm. When the ball suddenly curves, they must quickly adjust their catching position to account for the new ball trajectory. The previous examples illustrate that healthy individuals are able to make smooth, quick adjustments to their upper limb movements when their original arm movements are challenged. Performing quick movement corrections is an indication of intact higher-order motor function (Bernstein 1967).

What is meant by higher-order motor function? Fundamental elements of motor function include the abilities to produce muscle force and rotations about a joint. Basic motor function involves combining motor function elements to move the body. For example, flexing the elbow requires a person to contract the flexor muscles of the arm to apply forces on the forearm and result in rotating the elbow joint through its available range of motion. Therefore, higher-order motor function can be defined as the ability to combine basic motor functions in complex actions with the intention of executing and modifying purposeful movements.

Higher-order motor function of the upper limb involves not only the act of reaching for targets in the surrounding environment, but also making adjustments to the reaching/pointing movements.

As will be described in more detail below, reaching movement correction involves several processes (Goodale 2010; Prablanc et al., 2003; Sabes 2000). The first process involves vision and perception. A person has to perceive the location and size of the target and identify any obstacles in the way. The second process involves planning the movement to take into account the physical properties of the target and the obstacles, as well as to optimize key performance variables of the task, e.g. speed or accuracy requirements. The third process involves monitoring the progress of the movement, e.g. feedback. In the event that an online correction needs to be made, a second movement needs to be planned in order to successfully reach the target.

Each sensorimotor process has delays. It takes 85-150ms for visual information to reach the motor cortex, and then it takes the signal from the motor cortex 10-30ms to reach the arm and hand muscles (Lamme & Roelfsema, 2000; Salenius et al., 1997). The movement has not yet started at this point because it has been reported that there is anywhere from 30-100ms delay between onset of muscle activity and mechanical force production (Corser 1974; Inman et al., 1952; Milner-Brown & Stein, 1975; Ralston et al., 1976). Based on these delays, a movement correction based on visual feedback would take 125-280ms to begin. However, measurements of movement corrections in target shift paradigms have found that corrections occurred as early as 110ms after the visual cue, so feedback could not be the only mechanism for movement correction (Brenner & Smeets, 1997; Day & Lyon, 2000; Prablanc & Martin, 1992; Soechting & Lacquaniti, 1983). Furthermore, movement correction could also not simply be a reflex reaction because the correction would be expected to begin within 70ms of the cue to correct (Crago et al., 1976; Newell & Houk, 1983). For example, in a study of target displacement, subjects were seated in front of a screen where a red target would be displayed. When the target appeared, subjects were instructed to hit the target with a rod as rapidly and as accurately as possible. The researchers measured the reaction and movement times. The average reaction time to start moving after the target appeared was 400ms. Once the subjects started moving their arms, they took a further 250ms to reach the target. In some trials, the target shifted to a new position 34ms after movement initiation. Importantly, overall movement time for perturbed trials were not different from non-perturbed trials. Analysis of the lateral movement of the rod tip revealed that movement adjustment occurred at an average of 110ms after the target moved (Brenner & Smeets, 1997).

Other studies with target displacements occurring shortly after movement initiation suggested that corrections of the reaching movement could be incorporated into the original movement plan. This was evidenced by smooth endpoint trajectories and velocity profiles that did not reveal a distinct secondary movement correction (Goodale et al., 1986; Prablanc & Martin, 1992). When a trajectory correction occurred but the velocity profiles did not reflect a stop of the first movement followed by a distinct secondary movement, the correction was considered an online correction. However, when the time interval increased between presentation of the initial target and the target shift, discrete secondary movements were detectable. Results from target shift paradigms revealed that the corrective movements were either an interruption of the first movement to start a secondary, corrected movement, or one or more corrective sub-movements superimposed on the original movement resulting in the appearance of multiple secondary movements (Flash & Henis, 1991; Soechting & Lacquaniti, 1983; Georgopoulos et al., 1981).

While movement corrections are needed when targets change position, movement corrections also occur when obstacles are in the vicinity of the target. Are the movement corrections to shifting targets and obstacle avoidance similar? Aivar and colleagues (2008) studied the time of movement corrections when either the target moved or when obstacles flanking the target moved. Subjects sat in front of a graphic tablet and interacted with it using a stylus. A small dot representing start position was presented on the right side of the screen. When the subjects placed the stylus on the start position, a target and two obstacles appeared on the left side. The obstacles flanked the target on either side and subjects were required to slide the stylus from right to left across the tablet to touch the target while avoiding the obstacles. The target and obstacles were static in 40% of the trials. In the remaining trials, the target or the obstacles jumped left or right on the screen giving the impression that the opening had moved 2cm. The authors measured the time of the earliest response to the movement. When the target moved, the first response to the change occurred at about 120ms. When the obstacles moved, the earliest reported movement adjustments occurred around 150ms. The authors suggested that the increase in reaction time was the result of having more complexity in the obstacle avoidance task. The goal of the obstacle avoidance task was to avoid hitting the obstacles when reaching and then successfully touching the target, while the target shift task only required accurate aiming to the

target. Therefore, it appeared that the corrections needed in the obstacle shift condition were more complex than the corrections for target shift.

When objects actually blocked the target object, the responses were dependent on the characteristics of the obstacles. First, the hand mid-trajectory curvature maximum occurred just as the obstacle was cleared and the whole trajectory was structured so that there was a minimal clearance distance between the hand and obstacle (Abend et al., 1982; Dean & Brüwer, 1994; Sabes et al., 1998). Second, it was found that location and size of the obstacles affected the movement behaviour (Saling et al., 1998; Mon-Williams et al., 2001; Chapman & Goodale, 2008). Saling and colleagues (1998) examined the changes in arm kinematics in the presence of an obstacle that varied in height. In the control condition, subjects reached across a table to grasp a dowel and lift it. The experimental conditions involved reaching over a low or high obstacle and grasping the dowel. Reaching over an obstacle significantly increased the hand transport time due to the longer path taken to the dowel, but further analysis revealed that the time to peak velocity and peak deceleration occurred at earlier points in time during transport when compared to an unobstructed reach. The authors suggested that the earlier peak velocity and peak deceleration indicated a modification by the system to allow more time for the final part of the hand transport phase (deceleration phase). When obstacles flanked the target, movement time of the hand increased to the target and the hand trajectories altered from the straight-line path to the target, with greater deviations for obstacles that were placed closer to the target compared to objects placed further from the target (Castiello 1996; Howard & Tipper, 1997; Jackson et al., 1995; Tresilian 1998; Mon-Williams et al., 2001, Chapman & Goodale, 2008).

To summarize, avoiding obstacles is a complicated procedure requiring a high level of control over the coordination of upper limb aimed movements. In the obstacle avoidance literature, the behaviour is described with endpoint performance variables such as endpoint trajectories, acceleration/deceleration times, and the clearance around different kinds of obstacles. However, relatively little is known about the movement quality (e.g. joint kinematics, coordination) of obstacle avoidance behaviour. In addition, neural control of visually guided reaching and obstacle avoidance behaviour characteristics is still not fully understood. The next section presents studies on brain areas implicated in the control of visually guided reaching.

1.2 NEURAL CONTROL OF VISUALLY GUIDED REACHING

The neurophysiology that underlies visually guided reaching is complex because many brain areas are implicated in the production of a reaching movement. To start a reaching movement, a person has to perceive the environment and task. To do this, the peripheral nervous system sends visual and somatosensory information about the environment, position of the body and upper limb to the higher-level sensory processing areas found in the occipital, temporal and parietal cortices (Kandel 2012). The processed sensory information is then sent to ventral premotor cortex (PMv) dorsal premotor cortex (PMd), supplementary motor area (SMA) and cingulate motor area (CMA), which are associated with the selection, planning, and preparation of movements. The CMA may also be involved in adjusting motor behaviour by monitoring the emotional state, motivation and possible reward information (Iwata et al., 2013; Shima & Tanji, 1998). Connections from PMv, PMd, SMA and CMA to the primary motor cortex (M1) suggest communication with M1 (Kandel 2012). The M1 then has connections to the spinal cord and motor neurons (Kalaska 2009).

It is important to note that while the preceding description has illustrated the process of movement production in a sequential fashion, there is also abundant parallel distributed processing occurring between many brain areas. Subcortical areas and the cerebellum have different roles in planning, coordinating and modifying movements (Houk & Wise, 1995; Kandel 2012). The cerebellum has been associated with the planning, coordination and adaptation of movements according to the input that it receives from the spinal cord and other areas of the brain (Thach 1998; Manto et al., 2012). The cerebellum does not have a direct connection to the descending motor system, but rather directs its output to the thalamus. The basal ganglia are a group of grey matter nuclei found deep in the subcortical area of the brain that also influence the descending motor systems (Blumenfeld 2010). Depending on whether the basal ganglia send inhibitory or excitatory transmitters to the thalamus, it has an influence of inhibiting or allowing action potentials from upper motor neurons (Alexander 1994). The thalamus projects to the M1, SMA, PMd, PMv and parietal cortex to regulate their function. The result is an intricately developed network used to produce purposeful movement. It follows then that injury to any part of the nervous system disrupts this information flow. To understand the neuromuscular deficits after a stroke, a description of each brain area's normal function is necessary.

Neuroimaging and behavioural studies have highlighted frontal cortex regions associated with planning and execution of motor behaviours. In addition, the parietal cortex contributes to the planning and control of movement by integrating and transforming sensory information and sending this information to the motor areas. In the following section, the current hypotheses of frontal and parietal cortex involvement in visually guided reaching, based on the most recent evidence from anatomical, physiological, and behavioural studies of the human and primate brains will be reviewed.

1.2.1 THE FRONTAL LOBE

Early work from Penfield and Boldrey (1937) showed that stimulation of M1 caused motor activity, with the muscles of the whole body being represented in a predictable map along the precentral gyrus. More recent intracortical microstimulation studies of M1 revealed that stimulation of approximately 90% of M1 neurons evoke simple movements, such as those restricted to one joint (Geyer et al., 2000). When M1 neuronal discharges have been observed, discharge has been related to movement direction and velocity (Georgopoulos et al., 1982; Schwartz 1994). Stimulation of PMd, PMv and SMA also evoke movements, but the characteristics of these movements differ from movements evoked during M1 stimulation, in terms of amplitude and timing. The CMA is thought to be responsible for movement adjustments based on the consequences of the movement (Iwata et al., 2013). A more detailed explanation of each motor area follows.

The PMd and PMv are found in the lateral portion of frontal lobe, anterior to M1. They receive input from the sensory areas, the prefrontal cortex, and the motor nuclei of the thalamus (Kandel 2012; Matelli & Luppino, 1996). PMd and PMv influence motor activity through connections with M1, brainstem, and spinal cord (Augustine 2008). The dorsal and ventral premotor areas are generally involved in many aspects of motor control: selecting and planning of motor actions; and preparing and executing movements (Geyer et al., 2000; Purves et al., 2001).

The PMv has multiple roles (Rizzolatti 2002). First, it may be responsible for assessing an object's physical properties and location so that: (1) the hand can be properly shaped to interact

with that object; (2) the arm and hand can be properly oriented and transported for the required movement direction; and (3) the head and eyes can be oriented to locate the object (Binkofski & Buccino, 2006; Fogassi et al., 2001; Kakei et al., 2001). Hoshi and Tanji (2007) suggested that PMv participated in direct sensorimotor processing – its output to achieve an action directly matched the information received about the goal such as matching the movement of the hand with the location of a target. The authors hypothesized that sensory input about the three-dimensional characteristics of an object led to motor output consisting of motor commands for hand configuration. The second role of the PMv neurons involves the ability to understand an observed action. Neurons become active during specific goal-directed hand actions such as grasping as well when the subject is observing the same specific goal-directed hand actions performed by others (Gallese et al., 1996; Rizzolatti et al., 1996).

The activity of PMd neurons is similar to M1 in that they are correlated with the same movement parameters (e.g. direction of movement). What makes them different from M1 neurons is the time at which the activations occur. In observations of the PMd neuronal activation, monkeys were trained to perform goal-directed movements, but they were required to wait for a set delay period before actually performing the movement. PMd neurons appear to program the intention to move rather than to directly command initiation of movement, as evidenced by increased neuronal activation in the PMd before movement initiation (Godschalk et al., 1985; Riehle & Requin, 1989; Crammond & Kalaska, 1996, 2000). Furthermore, the PMd may be responsible for planning more complex interactions than the PMv, as evidenced by the indirect sensorimotor processing ability of the PMd. The PMd receives multiple sensory inputs consisting of information independent from each other, e.g. target location or arm function. It is suggested that the PMd then retrieves instructions on how to interact with each input, integrates the interactions and outputs a plan of action that incorporates the requirements of information from all the sensory inputs (Hoshi & Tanji, 2007).

The SMA may also be involved in movement selection. In humans, this area initially becomes activated during movement preparation and specifically when performing motor sequences from memory as well as during imagined movements (Augustine 2008; Kandel 2012). Stimulation of neurons in this area evokes a greater number of complex movements when compared to M1 (60% simple movements versus 30% complex movements) (Geyer et al., 2000). The SMA can

be further subdivided into the pre-SMA (rostral portion of the SMA) and SMA proper (caudal portion of the SMA). Tanji (1996) suggested that the SMA has wide influences over multiple aspects of motor behaviour. Its functions cannot be simply dichotomized (e.g. complex versus simple tasks, internal versus external motor initiation, proximal versus distal movements) because activation studies have shown that the SMA is active through the range of these dichotomies. However, differences may exist between the two subdivisions. The pre-SMA becomes more activated than the SMA with complex motor tasks and new motor sequence acquisition (Tanji 1996).

In the macaque monkey, the CMA is found within the cingulate cortex (Amiez & Petrides, 2014). The monkey CMA has been explored using intracortical microstimulation. It can be divided into 3 different somatotopically organized parts. The rostral cingulate motor area (CMAR) is located on both the ventral and dorsal banks of the cingulate gyrus. The CMAR contains representations of the face, hand and leg. The caudal cingulate motor area (CMAc) is divided into the ventral caudal (CMAv) and dorsal caudal (CMAd) areas. The CMAv contains a hand and a leg representation, while the CMAd contains two arm representations and one leg representation (Luppino et al., 1991; Dum & Strick, 1993). The CMAR may be involved in higher-order cognitive aspects of movements such as motor selection and error detection and the CMAc is more directly involved in movement execution (summarized in Takada et al., 2001). Some studies have provided evidence on the existence of human cingulate motor areas (Grafton et al., 1993; Arienzo et al., 2006; Amiez & Petrides, 2014). These studies also suggest that the function of the human cingulate motor area is similar to that of the monkey.

1.2.2 THE PARIETAL LOBE

The parietal lobe can be divided into the following regions: primary somatosensory cortex (S1), secondary somatosensory cortex (S2), and the posterior parietal cortex (PPC). This region receives and processes sensory information from the body and other areas of the brain (Kandel 2012). However, it is not only a passive receiver of sensory input. Functional mapping of the parietal and frontal cortices indicate a bi-directional network of communication between the two

areas (Kandel 2012). Information processed in the parietal cortex contributes information for movement production and provides sensory feedback during ongoing action (Desmurget et al., 1999; Mulliken et al., 2008).

1.2.2.1 THE ROLE OF THE SOMATOSENSORY CORTEX IN VISUALLY GUIDED REACHING

Throughout the process of performing a reach, sensory information is used to orient, plan and modify the arm movement. Initially, sensory information about the body's location, the arm's location, and the body's joint positions may be used to orient the person's arm to the goal. Mapping studies of the S1 have shown that there are multiple sensory representations of the body in the S1, corresponding to each of the cytoarchitecturally different regions of the S1 (Kaas et al., 1979; Disbrow et al., 2000). This increases the amount of information that the S1 can extract from an incoming somatosensory signal. The S1 neurons become active with sensory stimulation and its various regions categorize the signal components according to the type and intensity of the stimulus. In a collection of studies by Kalaska and colleagues (Cohen et al., 1994; Prud'Homme et al., 1994; Prud'Homme & Kalaska, 1994) the activity of neurons in the S1 with tactile receptive fields located on the arms of monkeys were isolated and measured. These cells are activated by skin contact with external surfaces as well as by mechanical deformation of the skin during movement (Cohen et al., 1994). The authors found that in addition to reacting to the type of stimulation on a certain area of the arm, these neurons also showed varying activity with different movement directions and postures of the arm. After the S1 processes the sensory information, it sends output to the S2, which further processes the sensory information to understand the relevance of the information to the individual's current situation. These processes may include sensorimotor integration, learning, and memory (Chen et al., 2008).

Corradi-Dell'Acqua and colleagues (2009) hypothesized that S2 codes for body schema, defined as an egocentric body representation of segment positions in space and time. In this study, subjects were asked to either imagine moving their own arm to match the orientation of an arm displayed on a screen (motor strategy) or to imagine rotating the screen arm until it was in an anatomically correct position relative to a picture of a human body without arms (visual

strategy). Neuronal activity in S2 was shown to increase when subjects used the motor strategy, suggesting that S2 plays a role in specification of the body representation and potentially also in movement planning (Corradi-Dell'Acqua et al., 2009). Thus, evidence suggests that somatosensory cortex codes not only for spatial representation of sensory stimuli on the body, but also proprioceptive information about the location of the body in space. This information may then be integrated into the creation of the movement plan.

1.2.2.2 THE ROLE OF THE PPC IN VISUALLY GUIDED REACHING

The superior and inferior parietal lobes together make up the posterior portion of the parietal cortex. In humans, the superior parietal lobule (SPL) corresponds to Brodmann's areas 5 and 7 (Grafton et al., 1996) while the inferior parietal lobule (IPL) corresponds to Brodmann's areas 7, 39, and 40 (Sakata et al., 1995; Augustine 2008). The PPC takes part in the control of movement and is now considered to be a part of the motor system (Fogassi & Luppino, 2005). Its contribution involves receiving input from other sensory areas (visual, auditory, somatosensory, limbic and vestibular) and then acting as a tertiary sensory association region. The additional processing of sensory information suggests that it has a role in spatial attention, spatial awareness, and transformation of sensory information into behavioural plans (Andersen & Buneo, 2002; Augustine 2008).

There have been contrasting roles suggested for the PPC. First, it was thought to be a higher-order somatosensory association area. This role is based on the observation that single units of area 5 receive converging inputs from multiple areas of SI. Therefore, a potential neural network exists in which multiple sensory inputs can be combined to create representations of body positions and movements (Jones & Powell, 1970; Jones et al., 1978; Pearson & Powell, 1985; Duffy & Burchfiel, 1971; Sakata et al., 1973). However, other studies found that some cells in the SPL did not have receptive fields on the body, but rather became active during specific movements. One study by Mountcastle and colleagues (1975) found that some SPL cells responded to object-directed movements involving arm projection and hand manipulation. They hypothesized that SPL was involved in the selection of appropriate motor responses before the

frontal motor areas. The debate over which of these roles is the true nature of SPL function has evolved into a third view: the SPL may actually be involved in the sensorimotor guidance of motor behaviour by compiling the different sensory inputs and transforming this information into standard coordinates used to plan movements. One common expectation from all three hypotheses is that activity of SPL neurons should be individually modulated by specific movement parameters. These parameters can include movement direction, movement velocity, and limb posture. In a study by Kalsaka and colleagues (1990) monkeys were trained to move a robot arm attached to a torque motor (manipulandum) between 8 different targets while compensating for various loads. Although the loads caused large changes in muscle activity, hand paths and joint angles were not altered when comparing among all load conditions. From neuronal activity measurements, the authors suggested that large changes in M1 activity corresponded with the changes in muscle activity (i.e. forces and torques) and that the consistently unaltered activity in area 5 during all load conditions corresponded with specific invariant spatial parameters (e.g. hand paths and joint angles). These observations of posterior parietal activity during motor activity have led to a number of hypotheses regarding the exact nature of how the PPC contributes to upper limb movement.

1.2.3 VISUOMOTOR TRANSFORMATIONS AND REACHING

Discussion about the contribution of visual information to guide reaching leads to two opposing views. One model of the influence of visual information on movement was proposed by Goodale & Milner (1992), where they suggested that visual information was used in two ways, perception and action, based on the evidence that two visual cortical pathways existed (vision for perception/vision for action model). One pathway travelled from the occipital to the temporal cortex (ventral stream) and the other from the occipital to the parietal cortex (dorsal stream; Goodale & Milner, 1992; Milner & Goodale, 2008). The ventral stream was hypothesized to be the pathway where visual information was transmitted to temporal brain areas implicated in the perception of the environment and the creation of allocentric representations of the characteristics and spatial relations of objects and events in the visual world. The representations would be used to identify goals and select appropriate motor actions, supported by the visual

information sent through the dorsal stream to the parietal cortex. The information in the dorsal stream registered moment-to-moment information about a goal in egocentric coordinates and would then become involved in the process of transforming visual information into motor commands so that the effector's movements could be correctly initiated and precisely controlled in real-time. The dorsal stream would also take into account the presence of other non-target objects and transmit information used to adjust movement parameters and avoid collisions.

Some evidence for the different function of each stream comes from studies of patient D.F., who had bilateral lesions in the ventral stream affecting the object recognition areas (James et al., 2003). When presented with rectangular blocks of various dimensions, D.F. could not report on the object characteristics or describe appropriate grasping postures for the blocks. However, when instructed to pick up the same object, D.F. accurately and appropriately reached and grasped for it (Milner et al., 1991). Furthermore, D.F. had excellent accuracy when pointing to targets in real time, but her pointing became highly inaccurate when a delay was placed between the stimulus and response (Milner et al., 1999). This highlights the possibility that the intact dorsal visuomotor pathway was still able to guide movements and that the perception of the target normally registered by the ventral pathway was no longer available for this patient (Milner & Goodale, 2008). More evidence for the differences in dorsal and ventral stream roles comes from an fMRI (functional magnetic resonance imaging) study that examined dorsal and ventral stream responses to images of faces, objects that could be manipulated such as tools, and scrambled images (Fang & He, 2005). Interocular suppression was used to make the stimuli seem invisible to conscious perception, thus hypothetically taking away stimulation to the ventral stream. The dorsal stream showed activation when subjects were presented with the objects that could be manipulated and had no activation with faces and scrambled images. Culham and Valyear (2006) suggest that the dorsal stream could have object-selective areas that allow patients such as D.F. to correctly interact with handheld objects even without explicit awareness.

Complementary evidence was found in studies of patients with damage to the dorsal stream. In reaching and grasping tasks, patients were unable to initiate appropriate movement parameters such as reach trajectory and grip aperture (Jakobson et al., 1991; Jeannerod et al., 1994; Milner et al., 2003). They also had an impairment of the online control of their movements, as highlighted in studies by Gréa and colleagues (2002) and Pisella and colleagues (2000). Healthy subjects and

patients with damage to the dorsal stream were instructed to point at visual targets. In 20% of the trials, the targets would unexpectedly change location, either to the left or right of the original position. Subjects were instructed either to stop reaching when the target changed location (Location-Stop) or to correct their movements in response to the target jump (Location-Go). In the Location-Go condition, the healthy group corrected their movements to the new target location, while in the Location-Stop condition they were unable to override the automatic correction. In contrast, patients with dorsal stream damage had no instances of automatic correction in the Location-Stop condition. As well, they showed only slow corrective movements during the Location-Go condition. Under the vision for perception/vision for action model, patients had deficits in the online control of movements. Therefore, the patients depended on conscious perception of the change in location of the target before they could make corrections, resulting in longer movement times and delayed corrective strategies.

This leads to a discussion about the nature of the online control mechanisms in the PPC. Sensory inputs are useful in the planning of movement parameters, but sensory feedback loops are considered too slow to be contributors to rapid, online movement control (Desmurget & Grafton, 2000). It has been suggested that the PPC can rapidly control goal-directed movements because it uses forward modelling to estimate the current and future states of the movement (Desmurget et al., 1999; Mulliken et al., 2008). Activation of PPC neurons is associated with specific types of inputs. For example, their firing rates change proportionally to the occurrence of a specific sensory parameter, such as a joint approaching a specific movement angle. Firing rates associated with sensory feedback can be measured, but there is also a period of time after the registration of sensory feedback when the neurons are still active. Desmurget and colleagues (1999) stated that continued PPC neuronal activity may indicate that they also contribute to the online control of movement.

Studies on the effects of optical illusions on healthy subjects further support the proposed differences between the two streams. For example, the Ebbinghaus illusion consists of two identical discs where one disc is surrounded by circles with smaller circumferences and the other by circles with larger circumferences. Subjects perceived that the disc surrounded by smaller circles is larger than the disc surrounded by larger circles. However, when reaching for the target discs, maximum grip aperture was scaled to the real circumference of the disc regardless of the

illusion (Aglioti et al., 1995). Milner and Goodale (2008) explain that these are examples of the distinction between ventral stream visual processes that guide action selection versus dorsal stream visual processes that oversee motor programming.

Another model of how the PPC provides visuomotor control has been developed based on differences between upper limb movements when they occur either within the central or the peripheral visual fields. Evidence that upper limb movements within these two visual fields are controlled by different cortical networks can be found in patients with optic ataxia. These patients are unable to successfully reach and grasp for objects located in their peripheral field of vision. However, when subjects were allowed to orient their eyes and head towards the objects their reaching and grasping movements parameters fell within normal limits (Clavagnier et al., 2007). Lesions of the parieto-occipital junction are common among optic ataxia patients (Karnath & Perenin, 2005). To ascertain the role of the parieto-occipital junction or any other brain areas in visually guided reaching, fMRI studies of reaching tasks were performed in healthy subjects (Prado et al., 2005). Right-handed reaching within the central field of view resulted in activations of the left and right medial intraparietal sulci and the caudal part of the left dorsal premotor cortex. Reaching within the peripheral field of view activated different regions, namely the dorsal and medial parts of the parieto-occipital junction and the rostral portion of the PMd in both hemispheres. These results, along with the results of studies with optic ataxia patients (Gréa et al., 2002; Pisella et al., 2000), indicate that visually guided reaching may be controlled by two different streams from the PPC depending on whether the target is in central or peripheral vision.

The importance of visual input for motor control has been stressed above. It follows, then, that the ability to recognize and understand actions also contributes to the planning of movements. There is a subset of neurons within the parietal and frontal cortices of humans and macaque monkeys known as ‘mirror neurons’ that respond to the execution of goal-directed actions as well as other action-related events (Rizzolatti & Craighero, 2004) such as passive observation of another individual performing the same goal-directed actions, listening to sounds that represent a given action, or verbal descriptions of action-related events (Hamzei et al., 2003; Kohler et al., 2002; Tettamanti et al., 2005). For example, when subjects observed another individual performing various hand actions, there were increases in the amplitudes of motor evoked

potentials in the subjects' hand muscles (Fadiga et al., 1995). Furthermore, when subjects were asked to perform a specific finger movement in response to different types of stimuli, faster response times were recorded when the cue to move consisted of the observation of another individual performing the required action versus symbolic cues (Brass et al., 2000). It was hypothesized that these faster response times occurred because the neurons activated by the observation of finger movements were mirror neurons. By definition, neurons that were activated in recognition of the movement were the same neurons that were activated when the recognized finger movement was actually being performed by the subject. Thus, this group of neurons anticipated the final goal of the movement execution and made the movement plan accessible almost as soon as the stimulus was presented (Buccino et al., 2004). The mirror neuron system may have a role in understanding, interpreting, and anticipating others' actions, all of which can then be used in the planning of one's interaction within the surrounding environment (Craighero et al., 2007).

In another hypothesis of the nature of the perception/action relationship, processes of perception were not separated from processes of action (Rizzolatti et al., 2006). This view was based on observations of inferior parietal cortex functions. Although traditionally considered a sensory association area, neurons in the IPL were shown to discharge when specific motor acts of the eye, hand, and arm were performed (Mountcastle et al., 1975). For example, some IPL neurons fired during active grasping for a piece of food. However, the activation levels of most of these "grasping" IPL neurons were influenced by the subsequent motor act, either eating the food or placing the morsel in a container. IPL neurons were also organized to activate for whole motor actions (ensembles of motor acts leading to reward or natural conclusion) (Rizzolatti et al., 2006). The IPL also contained mirror neurons that responded to watching grasping movements. The "watching" neurons could also be divided into groups that responded more strongly to subsequent eating or to subsequent placing of the food in a container. The authors concluded that the IPC showed the capacity to contribute to action execution as well as to have specific responses to various actions of other individuals. Rizzolatti and colleagues (2006) concluded that mechanisms of action and perception were anatomically linked, with their mechanisms residing in the same cortical areas.

1.3 DESCRIPTION OF STROKE PATHOLOGY

The following description of stroke pathology is summarized from Blumenfeld (2010) and Umphred (2007). A stroke is the sudden loss of neurological function resulting from the interruption of blood flow to the brain. It is considered a cardiovascular disease of the blood vessels that bring blood towards and within the brain. With an ischemic stroke, a clot restricts blood flow and the brain region to which a vessel supplies blood is deprived of oxygen and nutrients. Ischemic strokes can also occur when there is a systemic hypotension, resulting in general low perfusion to the brain. Normal neuronal function depends on aerobic metabolism, so without oxygen and glucose, neurons switch to anaerobic metabolism and enter into the ischemic cascade. This is a series of biochemical reactions that lead to intracellular excitotoxicity and rapid cell death. As the neurons within the ischemic area die, their toxic contents are released into the interstitial space which irritates the surrounding regions and causes the ischemic cascade to propagate to other neurons (Lo et al., 2003). In a hemorrhagic stroke, a blood vessel ruptures and blood accumulates in or around the brain. The hematoma causes increased intracranial pressure which injures brain tissue. The excess pressure also creates areas of ischemia in brain tissue and an ischemic cascade ensues. The blood in the extravascular areas of the brain also has a toxic effect on the exposed brain tissue that results in additional tissue injury.

In both types of stroke, the tissue surrounding the ischemic area called the penumbra is left structurally intact but becomes dysfunctional due to diaschisis and cerebral edema (Claassen et al., 2002; Feeney & Baron, 1986; O'Brien 1979; Ropper 1984; Seitz et al., 1999). Diaschisis is the abnormal neuronal function of structurally intact brain areas because of loss of input from damaged areas. Axonal projections from the dying areas are lost, causing a direct disruption of communication with neurons in the penumbra and a degradation of synaptic signals further along the neuronal pathways (Lo et al., 2008; O'Brien 1979). The penumbra also becomes dysfunctional because it experiences a reduction in blood flow. Intact collateral blood vessels can supply limited amounts of oxygen and glucose to the penumbra to maintain minimal metabolic function, but not normal neuronal function. If the blood supply continues to be deficient in the penumbral region, neuronal death occurs and infarction extends into the penumbra. Penumbral function is also depressed by cerebral edema. Cytotoxic edema is caused by cellular retention of sodium and water due to dysfunctional sodium/potassium membrane pumps of the brain cells

(Astrup et al., 1981; Lo et al., 2008). Vasogenic edema occurs after the blood brain barrier has been damaged and intravascular proteins and fluid cross to brain tissue, creating an osmotic gradient across which water moves from the blood vessels into the brain extravascular areas (Lo et al., 2008; Ropper 1984).

After the initial ischemic event, an inflammatory response occurs as white blood cells are exposed to the damaged tissue (Furlan et al., 1996). They react by removing not only the dead tissue but also the surrounding damaged, but viable, tissue of the penumbra. To minimize injury, acute treatment of a stroke must carefully perfuse oxygen and nutrients to the penumbra while minimizing the triggering the inflammatory response (Lo et al., 2005).

As the progression of cell death is halted, a complex process of neuronal modifications, termed neuroplasticity, begins in order to recover lost function (Nudo 2013). Structural changes occur at the intracellular, intercellular, and system levels of the nervous system. At the intracellular level, both healthy and surviving neurons undergo genetic, biochemical and organelle modifications in order to adapt to the changing needs of the altered nervous system. Between surviving neurons, the strength and efficiency of synaptic communication is improved through four mechanisms. Post-synaptic membranes that were originally connected to the injured area can become more sensitive to neurotransmitters released from surviving neurons of the same functional system. Pre-existing silent synapses are no longer inhibited and become functional when adjacent competing synapses die. Axonal sprouting can occur in injured neurons allowing them the possibility to regenerate their axons and make appropriate post-synaptic connections. Collateral axonal sprouting can also occur in surrounding healthy neurons that take over the function from the lost neurons (Brown et al., 2007; Brown et al., 2009; Capaday et al., 2013; Carmichael 2006; Dancause et al., 2005; Lo et al., 2003). However, while the presence of changes at the intracellular and intercellular levels induces local normal neuronal function, the changes must be orchestrated at a system level in order to induce recovery of body function (Grefkes & Ward, 2014; Murphy & Corbett, 2009).

Following damage to the cortex, the descending motor system pathways become altered. There are a number of descending tracts that can be affected depending on the area of brain damage. A stroke in the primary motor cortex and other frontal and parietal areas affect the function of the

lateral and anterior corticospinal tracts, leading to dysfunction of contralateral limb movement and bilateral axial and girdle muscles. Damage to the midbrain areas affects function of the extrapyramidal (vestibulospinal, reticulospinal and rubrospinal) tracts. The functions of the vestibulospinal and reticulospinal tracts are head and neck positioning, balance, and automatic posture and gait-related movements (Ellis et al., 2012). The rubrospinal tract may contribute to the control of contralateral limb movement (Lemon 2008). In a study of the development of the corticospinal and rubrospinal tracts in post-natal cats, the rubrospinal tract developed first and established rudimentary motor skills that were then refined by the later-developing corticospinal tract (Williams et al., 2014).

When the functions of the corticospinal tracts are altered, there is evidence that the undamaged extrapyramidal tracts compensate for the lost function. In monkey models, the rubrospinal tract compensates almost completely for corticospinal tract damage. In a study by Lawrence and Kuypers (1968a), bilateral corticospinal tracts of monkeys were lesioned. Initially, hand and arm movements were severely affected. However, after a month there was rapid improvement of all hand and arm function with the exception of finger movements and movement speed. In a second phase of the experiment, lesions were made to the rubrospinal tracts. Hand and arm control was lost and the impairments were permanent (Lawrence & Kuypers, 1968b). More recently, there have been imaging studies of the red nucleus and rubrospinal tract in humans after stroke. A diffusion tensor imaging study was performed in acute stroke patients 8-21 days after a stroke affecting the corticospinal tracts. There was higher fractional anisotropy on diffusion tensor image of the red nucleus in the affected hemisphere, indicating red nucleus remodelling and increased neural activity (Yeo & Jang, 2010). In the chronic stage after a stroke affecting the corticospinal tracts, greater fractional anisotropy in bilateral red nuclei was also found in stroke patients compared to a control group, suggesting that the red nuclei and rubrospinal tracts continue to compensate for lost corticospinal tract function long after the initial stroke (Ruber et al., 2012).

The reticulospinal projections also play a potential role in recovery after stroke (Zaaimi et al., 2012). Similar to the studies of Lawrence and Kuypers (1968a, 1968b), Zaaimi and colleagues (2012) examined a monkey at 6 months post-lesion. The monkey had extensive unilateral damage to the corticospinal tract and had recovered gross motor arm and hand function, but not

fine independent finger movements. Stimulation of the contralateral intact corticospinal tract did not provoke movement, but stimulation of the reticulospinal tract elicited responses in the forearm flexor and intrinsic hand muscles. Zaaimi and colleagues (2012) suggested that the reticulospinal tract contributed to functional recovery after corticospinal tract damage.

During recovery after a stroke, imaging studies indicate that plasticity at the neural network level occurs as cortical remapping and changes in neural activation patterns (Nudo 2013). First, there is evidence of reorganization of the brain's neural connections within the penumbra. Before a stroke, activations of specific motor areas are associated with specific motor actions, so a lesion in a motor area results in predictable motor deficits. Nudo and colleagues (1996) showed that successful recovery of an affected limb's function after intense motor rehabilitation was associated with the neurons in the penumbra taking over function of the lost neurons. There is also evidence that in cerebral strokes the unaffected hemisphere may contribute to recovery of function. Imaging studies of subacute motor recovery reveal simultaneous activation of the sensorimotor areas in the unaffected hemisphere when performing movements with the affected side (Chollet et al., 1991; Murayama et al., 2011; Weiller et al., 1992). This would indicate a contribution of the ipsilateral motor pathways to movement control of affected limbs. However, the nature of the ipsilateral hemisphere contribution is unclear because there have been conflicting data of both its beneficial and detrimental involvement in recovery (Jankowska & Edgley, 2006; Rehme et al., 2010; Summers et al., 2007; Takeuchi et al., 2005; Werhahn et al., 2003; Wu et al., 2011). Network plasticity also occurs in the form of changes in neural activation interactions. In the healthy brain, learning novel complex skills is associated with the formation of new cerebellar-cortical synapses (Shmuelof & Krakauer, 2011; Galea et al., 2011). After a stroke interaction between synapses and formation of new synapses between brain areas may be impaired, so a person may find it difficult to relearn previously automatic sensorimotor tasks.

The neuromuscular impairments that result from stroke can differ according to the brain area affected. Motor control is affected when there is damage to the primary and secondary motor and sensory areas. Damage to the M1 leads to problems with generating normal levels of muscle force (Patten et al., 2004). There are also problems with muscle activation such that individual muscles cannot be recruited and in turn, the joints that are crossed by the affected muscles can no longer be finely controlled (Beer et al., 2000; Levin 1996). Abnormal synergies occur when

multiple muscles cannot be appropriately activated and sequenced for functional tasks (Brunnstrom 1970; Roh et al., 2013; Sethi et al., 2013). Impaired muscle activation also manifests as inappropriate co-activation of agonist and antagonist muscles during functional movement (Dewald et al., 1995). In addition to problems with muscle activation, damage to M1 can also lead to abnormal muscle tone (spasticity). Normal muscle has a certain level of resistance to passive stretch (tonic stretch reflex), but a muscle affected by spasticity has a hyperexcitable stretch reflex such that there is an abnormal, velocity-dependent increase in muscle tone (Calota et al., 2008; Lance 1980).

Injuries to the SMA, PMv and PMd do not produce severe movement deficits, but are involved in deficits in higher-order motor planning (Halsband et al., 1993; Sadato et al., 1997). A few case reports of injury to the CMA indicate that patients can experience akinesia and bilateral disturbances of movement control (Kumral et al., 2002; Stephan et al., 1999). The cingulate cortex is also involved in guiding behaviour according to the person's moral standards, emotional state, and level of empathy (Seitz et al., 2006), so injury to the cingulate areas may alter the drive or emotional control of motor behaviour (Dum & Strick, 1993). In a review by Paus (2001), patients with lesions involving the cingulate cortex showed deficits in spontaneous movement initiation and the presence of alien-hand syndrome (an inability to willfully suppress externally triggered movements of the contralesional hand and arm).

Lesions of the cerebellum and small-vessel infarcts affecting subcortical areas (lacunar infarcts) also cause neuromuscular impairments through the impairment of their intrinsic functions. Cerebellar strokes commonly cause ataxia, which is irregular uncoordinated voluntary movement. The manifestations include delayed reaction time, dysmetria (errors in movement range and direction), difficulty stopping and changing directions of movements, dysdiadochokinesia (inability to sustain regular rhythmic movements). Cerebellar lesions can also cause intention tremors and impaired error correction for subsequent movements (Edlow et al., 2008). Lacunar infarcts also result in region-specific syndromes. Lesions of the basal ganglia are not usually symptomatic, but a lesion specifically of the subthalamic nuclei can result in hemiballismus which is unilateral wild flinging movements of the extremity contralateral to the lesion. Lesions of posterior limb of the internal capsule, ventral pons, corona radiata, cerebral peduncle cause pure motor, dysarthria, or ataxic hemiparesis. Thalamic lacunes result in

contralateral somatosensory deficits (Fisher 1982; Fries et al., 1993). However, functions of the cerebellum and subcortical areas are more commonly disrupted when primary and secondary sensorimotor areas or corticospinal tracts are injured. The cerebellum and subcortical areas become dysfunctional because regulatory input and output to and from these areas are abnormal from the infarcted regions.

Lesions of the primary and secondary motor cortices, subcortical regions (involved in motor planning and learning), and the cerebellum directly result in motor control deficits. Furthermore, lesions of the sensory pathways of the brain can also affect motor control (Blumenfeld 2010). There are two pathways for ascending somatosensory information, namely the dorsal column–medial lemniscal system and the anterolateral spinothalamic tract. Lesions of the DC-ML result in deficits with light touch, kinaesthetic sense. Lesions of the lateral spinothalamic tract cause loss of pain sensibility, thermal discrimination, coarse touch, and kinaesthetic discrimination. Lesions of the primary somatosensory cortex affect the somatosensory feedback about body position in space and body segment interrelationships used to refine subsequent movement. There are also deficits in proprioception, two-point discrimination, stereognosis, and localization of touch occurring on the side contralateral to the lesion location (Connell et al., 2008, Winship & Murphy, 2009). Parietal lesions (Brodmann’s area 5 and 7) result in problems with learning involving processing of multiple modalities (Rawley et al., 2009; Vidoni et al., 2010).

The impairments that result from damage to specific areas of the brain are described above, but impairments after stroke are a result of both central and peripheral changes. There are changes at the muscle level after brain damage. When paresis and spasticity occur after a stroke, there is a decrease in physical activity causing secondary musculoskeletal problems such as atrophy and deconditioning, contractures and osteoporosis (Fredericks & Saladin, 1996).

Sections 1.2 and 1.3 summarized the function of brain areas implicated in the control of reaching as well as the impairments that can result from lesions to specific areas of the brain. It is important to note that although specific motor deficits have been associated with specific lesion locations, it has also been observed that similar presentations of paresis from different patients can be caused by damage to brain areas other than the primary motor cortex, such as the premotor, parietal and striothalamic areas (Kunesch et al., 1995). But it has been shown that

lesion location can be a predictor of upper limb recovery. In a study assessing lesion site as a possible predictor of upper limb recovery, it was found that motor recovery was best predicted at two months post-stroke with a combination of clinical outcome measures and pure subcortical lesion (Feys et al., 2000). Another study that stratified stroke subjects according to cortical, subcortical and mixed lesions found that the probability of recovery of isolated upper limb movement was highest with cortical strokes, followed by subcortical and then mixed lesions (de NAP Shelton & Reding, 2001). This data would suggest that the rehabilitation of subcortical lesions could require a different approach than rehabilitation of cortical lesions. Knowing lesion location early after the stroke could potentially identify more accurately the probability of successful recovery and how a patient would respond to treatment.

1.4 DESCRIPTION OF UPPER LIMB DEFICITS AFTER STROKE

Approximately 75.5% of acute stroke patients experience upper limb paresis (Rathore et al., 2002), and in the chronic stage (> six months post stroke), upper limb paresis is still present in approximately 50-75% of survivors (Jørgensen et al., 1995; Mayo et al., 1999; Kwakkel et al., 2003). Changes in reaching behaviour including prolonged movement times, use of compensatory movement patterns, loss of upper limb and trunk segmental control, impaired inter-joint coordination, less accuracy in final position of the hand, and longer time to change movement strategies (Levin 1996; Cirstea & Levin, 2000; Beer et al., 2000; Levin et al., 2002; Cirstea et al., 2003; Lang & Beebe, 2007; Dancause et al., 2002). Reaching deficits in moderately- to severely-affected post-stroke individuals may be related, in part, to muscle weakness, abnormal movement synergies and spasticity (Bourbonnais et al., 1989; Bobath 1990; Levin et al., 2000). There is also altered neural control of visually guided reaching because the normal communication between different areas of the brain and the PNS is disrupted. For more impaired patients, rehabilitation focuses on regaining strength, range of motion, and techniques to regain independence in activities of daily living (Teasell et al., 2013).

Reaching deficits in well-recovered stroke patients are not as apparent because these patients regain high levels of function in their affected arm and hand and are left with very little to no

neurological deficits (Jørgensen et al., 1995; Mayo et al., 1999). However, follow-up studies of well-recovered stroke patients have revealed that these individuals are not using their arms to the full extent expected of them in activities of daily living and also report feeling less confidence in using their arm (Carlsson et al., 2004; Edwards et al., 2006; Carlsson et al., 2009; Rand & Eng, 2012). Current clinical measures of ability in daily upper limb activities may not be capturing the more subtle impairments of well-recovered individuals. Over time, there is a risk of losing more functional ability because disuse causes further muscular and neurological deterioration.

1.4.1 DEFICITS OF VISUALLY GUIDED REACHING IN HEMIPARETIC INDIVIDUALS AFTER STROKE

Individuals with hemiparesis after a stroke have difficulty making smooth and accurate visually guided movements with the affected arm (Fisk & Goodale, 1988). They have prolonged movement times, altered movement trajectories, and less accuracy in final endpoint position (Levin 1996). Many factors contribute to this reaching deficiency including muscle weakness (Bourbonnais & Noven, 1989), spasticity (Burke 1988; Wiesendanger 1990), decreased shoulder girdle mobility (Cailliet 1980; Ryerson & Levit, 1987), incorrect timing of movement pattern components (Carr & Shepherd, 1987; Archambault et al., 1999), abnormal movement synergies (Bobath 1990), and loss of interjoint coordination (Levin 1996). In the presence of these problems hemiparetic individuals use compensatory movement patterns for reaching (Cirstea & Levin, 2000). These include increased use of shoulder and trunk movements and abnormal interjoint coordination between the trunk, shoulder, and elbow joints (Cirstea et al., 2003; Levin et al., 2002). It is important to identify to what extent compensatory movement patterns are present in post-stroke individuals with well-recovered use of their affected arm. It is difficult to ascertain the level of impairment in well-recovered individuals from studies that report only group means from patients with a wide range of impairment.

Understanding the composition of compensatory movement patterns is useful for understanding their role in reaching ability in post-stroke individuals. Normally when the arm is reaching for a goal, there are theoretically an infinite number of joint combinations from which to choose in order to achieve the goal (Bernstein 1967). The availability of many joint combinations can

allow much flexibility for achieving the goal, but observation of repetitive reaching movements towards the same point in healthy subjects reveals that a straight, smooth trajectory is chosen, and that the variability in hand trajectories and joint positions is small compared to all theoretically possible trajectories and joint positions (Levin 1996).

The concept of kinematic redundancy after stroke has been studied by a few groups. In one study, participants were seated and their arms placed in a double-joint manipulandum that prevented movement of the wrist and allowed movement of the shoulder and elbow (Mihaltchev et al., 2005). The position of the hand (the endpoint of the manipulandum) was represented on a computer screen as a cursor. Participants maintained an initial position by moving the cursor to a target on the computer screen. Maintaining this position required the participants to balance an external load (30% maximal voluntary contraction). When the load was released, participants were instructed not to intervene and to allow the arm to naturally move to a new position. In this way, the researchers were able to delineate the size and location of the referent postures produced by the nervous system for controlling the final positions of the hand for this task. Compared to an age-matched healthy group, hemiparetic individuals had a restricted range of final hand positions indicating a decrease in the use of kinematic redundancy. There was also an increased instability around the final endpoint position.

Another analysis of reaching strategies involves distinguishing between the use of equivalent and non-equivalent joint combinations for producing task-specific movements (Reisman & Scholz, 2003). For example, if a specific hand position is the goal of a reach, different shoulder, elbow, and wrist angles can contribute to the arm configuration to achieve the same final hand position. Thus, the different combinations of joint angles are equivalent for a given final goal. This is called goal-equivalent variance (GEV), while non-equivalent joint combinations result in larger hand path variability, termed non goal-equivalent variance (NGEV). In a study of distinguishing the presence of GEV and NGEV, subjects were seated at a table with a target suspended at shoulder level and at a distance of approximately 90% of functional arm length. Subjects reached for the target quickly and accurately while maintaining a consistent speed over all trials. Hemiparetic subjects maintained the ability to limit NGEV joint combinations, evidence that they preserved their ability to limit endpoint final position errors. However, similar to the findings of Mihaltchev and colleagues (2005), subjects with moderate hemiparesis used fewer

equivalent joint combinations to complete the reach. As well, moderately impaired individuals were unable to vary the coupling of their available joint motions during reaching and pointing. This was consistent with the clinical observation that moderately hemiparetic individuals had joint coupling patterns that were different and limited compared to healthy controls (Reisman & Scholz, 2003). It will be important to identify whether or not deficits in the use of redundancy persist in reaching movements of well-recovered post-stroke individuals. Identifying the similarities or differences in the use of redundancy between individuals with different levels of impairments may contribute to the improvement of rehabilitation methods.

1.4.2 MOVEMENT CORRECTION OF REACHING IN HEMIPARETIC INDIVIDUALS AFTER STROKE

Studies of how individuals learn to adapt movement over multiple trials in the presence of perturbations are numerous for healthy individuals (Section 1.1.2), but relatively fewer studies to date have described this behaviour in post-stroke individuals. Dancause and colleagues (2002) studied the ability of post-stroke subjects to correct elbow position when a load was suddenly introduced or removed during a rapid, goal-directed elbow flexion movement and found that subjects with more severe hemiparesis required more trials before they were able to make accurate elbow movements. Other studies have characterized the ability of hemiparetic individuals compared to control subjects to adapt horizontal reaching movements in order to compensate for unpredictable perturbations. Similar to the previously mentioned study, post-stroke individuals did not adapt to perturbations as easily as control subjects, with more errors in trajectory and regulation of final hand position and the requirement of more trials than controls to master the adaptation (Scheidt & Stoeckmann, 2007). The ability to adapt movement over time was also found to be related to the severity of the arm paresis (Dancause et al., 2002; Scheidt & Stoeckmann, 2007).

The ability to perform online corrections within one trial has also not been well studied in post-stroke individuals. One study measuring the ability of post-stroke individuals to perform a target jump task found that the hemiparetic individuals had larger final hand position errors. In addition, subjects with left hemispheric lesions had larger intersegmental coordination deficits, while subjects with right hemispheric lesions had more difficulty with timing and accuracy of

movement corrections (Schaefer et al., 2012). Thus, in studies of reaching and movement correction, post-stroke individuals compared to controls had altered movement patterns while reaching, such as abnormal upper limb joint coordination and increased trunk movement used to assist the movement of the arm towards the target (Musampa et al., 2007; Dancause et al., 2002). Post-stroke individuals also took longer to learn arm movement correction strategies and depending on lesion location, had deficits in intersegmental coordination or the timing and accuracy of the corrective movements when attempting online corrections (Dancause et al., 2002; Schaefer et al., 2012; Scheidt & Stoeckmann, 2007). It is important to note that the studies mentioned so far tested the higher-order motor ability of stroke subjects in restricted upper limb positions, so the extent to which the reaching impairments are present in three-dimensional movement should be quantified.

1.4.3 OTHER FACTORS CONTRIBUTING TO DECREASED ARM FUNCTION AFTER STROKE

The apparent decrease of use of the arm may also stem from other causes. Two potential influences are pain and visual-perceptual deficits. Relationships have been demonstrated between post-stroke pain, sensorimotor problems and recovery of arm function (Appelros 2006; Roy et al., 1994). In patients with visuospatial neglect, there was a relationship between neglect and performance in activities of daily living (Marsh & Kersel, 1993; Kinsella et al., 1993; see review by Rubio & Van Deusen, 1995).

Other influences include emotional and social difficulties such as psychological morbidity, negative sense of self, reduced social activity, and delayed return to work are common after stroke, but are not usually considered as influencing physical motor recovery (Ellis-Hill et al., 2000; Saeki & Toyonaga, 2010). However, it has been shown that patients with marked depressive symptoms had more neurological impairment (Herrmann et al., 1998). Also, depression, functional independence, and quality of life are strongly related to self-care self-efficacy (Robinson-Smith et al., 2000). Self-efficacy in this context is defined as the confidence that a person has in their ability to perform relevant self-care activities (Lev & Owen, 1996). More broadly, self-efficacy is defined as the belief and confidence that an individual has in their

capabilities to perform a specific behaviour (Bandura 1977, 1986). If their level of self-efficacy is high, then they feel that they can better perform the behaviour. There has been a strong association found between self-efficacy and activities of daily living (Korpershoek et al., 2011). However, the clinical scales used to assess activities of daily living encompass both lower and upper limb tasks. Therefore, not much work has been done exploring the role of self-efficacy with upper limb-specific activities of daily living after stroke.

1.5 UPPER LIMB RECOVERY AND ITS MEASUREMENT

Most of the recovery after stroke occurs in the first 30 days and continues at a lesser degree up to six months after the initial brain insult (Duncan et al., 1994). Since approximately 62% of survivors continue to have upper limb deficits, upper limb (UL) long-term recovery is considered poor (Kwakkel et al., 2003). Recovery of functional upper limb use is very task-specific and requires high intensity practice, but the type and amount of practice currently administered in stroke rehabilitation sessions does not meet this requirement (Rand & Eng, 2012; Lang et al., 2007; Teasell et al., 2013). Thus, longitudinal studies have shown that current therapeutic approaches for the upper limb are not very effective. Furthermore, another explanation for the poor rate of recovery is that conventional therapeutic approaches may not target higher-order motor functional impairments, such as the ability to quickly coordinate movements and perform fast online corrections. However, higher-order motor functional impairments may not be targeted because they are not being measured with current clinical tests. Tests such as the Fugl-Meyer Assessment, Chedoke-McMaster Stroke Assessment, and grip strength do measure body function, but may not have the sensitivity to detect the subtle differences in movement quality expected to occur in well-recovered individuals, i.e. a ceiling effect (Carlsson et al., 2003; Rabadi & Rabadi, 2006).

1.5.1 MEASURING HIGHER-ORDER MOTOR FUNCTION IN THE CLINIC

In order to improve higher-order motor function, the impairments must be accurately recognized and quantified. Currently, there are only a few tests of higher-order motor function of the upper limb and they are mainly assessments for basic coordination such as the Finger-to-Nose and knee tapping tests. The upper limb coordination tests measure the ability to perform discrete, intentional movements, but there are a number of concerns regarding their use. First, the assessments were initially developed for detection of cerebellar or vestibular impairments (Patten 1996). However, coordination can be affected by damage to other components of the central nervous system, such as the basal nuclei, dorsal columns, and sensorimotor cortices (Potvin & Tourtelotte, 1985). Thus, the validity, reliability and responsiveness of coordination assessments have not necessarily been determined for other types of cortical and subcortical damage. The second concern with the widespread use of the current coordination assessments is the lack of standardization for administering the tests (Schmitz 2001; Swaine & Sullivan, 1993). A survey of any textbook for neurological examination yields varying instructions for test administration. The third concern is the limited range of scoring methods. Various outcome measures include time of execution for a set number of cycles, number of cycles within a set timeframe, and simple ranking of the presence of incoordination (“can/cannot complete” or “tremor present/not present”; Swaine & Sullivan, 1992). What is missing from the scoring methods of coordination assessments is the ability to detect and quantify compensatory movement patterns used to successfully complete a coordination test. It is important to measure movement patterns because subtle compensatory movements may be the only indicator of mild impairment in well-recovered post-stroke individuals. Kinematic variables such as shoulder, elbow, wrist ranges of motion, as well as hand, arm, and trunk displacement represent movement quality and have been shown to be valid measures of arm motor impairment after stroke in research laboratory settings (Subramanian et al., 2010).

It is relatively difficult to measure mild compensatory movement patterns in the clinical setting. Robotic technology has been used as a tool to precisely measure upper limb task performance parameters such as movement time, reaction time, hand speed, corrective responses (according to number of peaks in the velocity profile), movement direction and hand path length (Scott 1999; Coderre et al., 2010). However, there are some limitations. First, previous studies have focused

mainly on task performance outcome parameters and do not specifically measure how a movement is performed (Levin et al., 2009). Second, to standardize performance parameters and make results comparable between individuals, assessments that use robotic devices often restrict movement to a single plane. However, arm movements in daily activities occur in three dimensional space and require more complicated arm joint coordination than movement in a single plane. The impairments of well-recovered individuals may not be detected in the relatively simpler movements. Virtual reality platforms have been developed to mimic real-world situations, allow full range of movement and allow a safe medium in which to practice daily tasks (Rizzo & Kim, 2005). Therefore, virtual reality environments can be used as a tool to directly measure performance outcome and movement quality of upper limb daily tasks as well as measure higher-order motor control deficits when performing natural, three-dimensional movements.

1.6 SUMMARY

Previous research suggests that successful reaching requires intricate coordination of the upper limb. In this way, upper limb segments can be moved effectively to interact with a desired object. In situations where obstacles interfere with the reaching path, upper limb movement is altered so that the obstacle is avoided and the goal is still attained. Obstacle avoidance requires quick adjustments of the movement plan, resulting in more complicated coordination of the upper limb and trunk. When a stroke causes damage in brain areas associated with upper limb movement, the loss of coordinated arm control make reaching tasks difficult. Avoiding obstacles while reaching may be even more difficult after a stroke. Obstacle avoidance ability has been studied in individuals post-stroke that have visuospatial neglect, but it has not been tested as a potential measure of residual arm sensorimotor function. Although many stroke survivors regain functional use of their arms, they continue to have persistent deficits in activity performance, participation, and health related quality of life (Tellier & Rochette, 2009; Hare et al., 2006; Carlsson et al., 2009; Edwards et al., 2006; Carlsson et al., 2004). Studies have shown that well-recovered post-stroke individuals have differences between their clinically measured functional recovery of the arm and their actual daily arm use (Rand & Eng, 2012; Michielsen et al., 2012). Poor arm use may be due to impairments in higher-order control of movement or a lack of

confidence in the ability of the affected arm. The person may then enter a detrimental cycle of decreasing arm use and increasing impairment. However, current clinical measures are not sensitive enough to detect impairments of higher-order motor function or the existence and extent of compensatory movement patterns found in well-recovered post-stroke individuals (Subramanian et al., 2010).

CHAPTER 2: STUDY RATIONALE

In order to optimally challenge higher-order motor function in well-recovered post-stroke individuals, a task requiring critical timing is necessary. Such tasks have been described in the literature such as reaching for shifting targets or around moving obstacles. We chose avoiding a moving obstacle because it is a common yet challenging occurrence in everyday activities. A virtual environment was used in order to more precisely control the temporal and spatial parameters of the obstacle avoidance task. Such difficult tasks are expected to be more difficult for people with impairments of higher-order motor function. The extent to which deficits in higher-order arm motor function occurs in well-recovered stroke individuals may only be measured using detailed kinematic analysis of movement quality. Being able to detect higher-order arm motor function deficits will allow clinicians and researchers to better design specific rehabilitation programs.

The objectives of the thesis are formulated in the PICO question format which provides a framework for formulating a research question (Sackett et al., 1997). It identifies the population, intervention, comparison group, and outcome measure of the intervention. For this study, PICO elements are:

P – Individuals with well-recovered upper limb function post-stroke

I – Obstacle avoidance task with the upper limb

C – Healthy individuals

O – Reaching behaviour (kinematic measures and task success rates)

The goals of this thesis were to examine how well-recovered post-stroke and healthy individuals avoid obstacles during reaching and relate obstacle avoidance reaching performance with clinical measures of functional arm use.

Details of all outcome measures and statistical analyses are described in the Methodology chapter.

2.1 OBJECTIVES AND HYPOTHESES OF THE THESIS

OBJECTIVE 1:

Successful obstacle avoidance with the upper limb requires rapid adaptations in the hand reaching path to changing task conditions in the environment. In order to adapt the hand path, rapid changes in coordination between arm joints have to occur. The first objective is to identify the rate of task success and differences in obstacle avoidance behaviour (kinematic strategies) used by well-recovered post-stroke individuals and healthy controls.

HYPOTHESIS 1A:

Post-stroke individuals will have more difficulty in obstacle avoidance behaviour. When reaching, the hand or arm will collide more often with the obstacle compared to healthy individuals.

Primary outcome measure: Task success rate – number of trials in which the obstacle is successfully avoided / total number of trials in experimental block

Statistical analysis: The task success rates for each group will be compared using Student's t-test. Also, a comparison of the proportion of individuals in each group who achieved different levels of success rates was performed.

HYPOTHESIS 1B:

Post-stroke individuals will have a decreased adaptability of joint movement which limits their obstacle avoidance (kinematic) strategies.

Primary outcome measure: Divergence point of endpoint displacement/velocity (phase) plots (see Methodology section 3.6.1 for more explanation)

Secondary outcome measures: Joint angles and endpoint performance variables

Statistical analysis: The divergence points of the endpoint phase plots will be compared for significant difference between the healthy controls and post-stroke individuals using Student's t-test. The joint and endpoint kinematics of the healthy controls and the post-stroke individuals

will be compared for significant differences using repeated-measures analysis of variance (ANOVA).

OBJECTIVE 2:

The second objective is to determine if current clinical assessments of arm function and arm use in activities of daily living relate to success in obstacle avoidance ability.

HYPOTHESIS 2:

Higher clinical scores of arm impairment, activity, and performance in the stroke group will be associated with higher task success rates.

Primary outcome measures: Task success rate

Statistical analysis: Correlations will be performed between task success rates and clinical measures (shoulder strength and range of motion, Composite Spasticity Scale, Chedoke-McMaster Stroke Assessment arm and hand subscales, modified Wolf Motor Function Test, Fugl-Meyer Assessment, Reaching Performance Scale, Motor Activity Log).

OBJECTIVE 3:

Considering the previous literature that demonstrated decreased daily arm use in well-recovered stroke patients, it would be expected that the decreased daily arm use over time would cause further deterioration in the ability to perform a higher-order motor task. The third objective is to determine if obstacle avoidance strategies and clinical measures are altered by time in a well-recovered stroke group.

HYPOTHESIS 3A:

After one year (Visit 2), task success rates will decrease compared to the first visit (Visit 1).

Primary outcome measure: Task success rate

Statistical analysis: The task success rates for each group will be compared using Student's t-test. A comparison of the proportion of individuals in each group who achieved different levels of success rates was then performed.

HYPOTHESIS 3B:

After one year, post-stroke individuals will have a decreased adaptability of joint movement which limits their obstacle avoidance (kinematic) strategies.

Primary outcome measure: Divergence point of endpoint displacement/velocity (phase) plots (see Methodology section 3.6.1 for more explanation)

Secondary outcome measures: Joint angles and endpoint performance variables

Statistical analysis: The divergence points of the endpoint phase plots will be compared for significant difference between the healthy controls and post-stroke individuals using Student's t-test. The joint and endpoint kinematics of the healthy controls and the post-stroke individuals will be compared for significant differences using repeated-measures ANOVA.

HYPOTHESIS 3C:

After one year, further decrease in task success will be reflected in the relationship between lower task success rates and lower clinical measures of arm impairment, activity and participation.

Primary outcome measures: Task success rate

Statistical analysis: Correlations will be performed between task success rates and clinical measures for Visit 2 (shoulder strength and range of motion, Composite Spasticity Scale, Chedoke-McMaster Stroke Assessment arm and hand subscales, modified Wolf Motor Function Test, Fugl-Meyer Assessment, Reaching Performance Scale, Motor Activity Log).

CHAPTER 3: METHODOLOGY

3.1 RESEARCH DESIGN

A cross sectional study was conducted to examine the reaching behaviour of individuals after stroke with well-recovered upper limb strength and range of motion as compared to healthy individuals of similar age range. Data were collected in the Sensorimotor Control and Rehabilitation Laboratory located at the Jewish Rehabilitation Hospital (JRH) site of the Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal (CRIR) in Laval, Quebec. The project received ethics approval from the ethics boards of CRIR and L'Hôpital de réadaptation Villa Medica (HRVM).

3.2 POPULATION AND ELIGIBILITY

The target populations included two subject groups: adults aged 40-70 years with recent first cerebrovascular accident, having good recovery of their upper limb with some limitations in ADL involving upper limb use (“stroke subjects”); and healthy individuals of a similar age range (“control subjects”). Inclusion/exclusion criteria for stroke and control groups are listed in [Table 1](#) and [Table 2](#). Shoulder pain was assessed, but was not an exclusion criterion for the study if the subject could perform the reaching task without pain.

3.3 SAMPLE SIZE ESTIMATION

Sample size calculations for this project were based on pilot data collected in our laboratory. Pilot data collected included n=5 control subjects (right dominant, mean age 61±19.2 years), n=2 stroke subjects (both right dominant, right hemiparesis, ages 70 and 58 years). The outcome variable selected for calculation was endpoint peak velocity. The statistical software GPower3 was used to calculate sample size (Faul et al., 2007). With a power of 80%, between 16 and 46 participants were required to detect a difference using repeated-measures ANOVA. Since our calculation was based on a small sample of subjects, we initially aimed to recruit 46 subjects and then revised the sample size after 11 participants had completed the protocol. With n=4 control subjects and n=7 stroke subjects, a total sample size of 22 was needed to achieve a power of 0.83. A summary of subject recruitment is listed in [Table 3](#).

Table 1: Inclusion criteria for stroke subjects

| Criterion | Evaluation cut-off |
|---|---|
| Medical status | First cerebrovascular accident documented with imaging results and not involving basal ganglia or cerebellum; chronic stage of recovery (> 3 months post-stroke). |
| Age 40-70 years | Motor coordination declines with age, and 70 years of age is a reasonable cut-off at which this decline becomes more apparent (Desrosiers et al., 1995). It is hard to distinguish the effects of age from the effects of the stroke, which may introduce bias. |
| Lesion in dominant hemisphere | Edinburgh Handedness Inventory score: a minimum laterality quotient of +25% and -25% is used to distinguish right and left handedness respectively (Oldfield 1971) |
| Well-recovered arm motor ability | Chedoke-McMaster Stroke Assessment score (impairment): ≥ 5 on the arm scale (Gowland et al., 1993) |
| No visuospatial neglect | Line bisection test. Little is known about the effect of visuospatial neglect on movement production (Schenkenberg et al., 1980). |
| No visual field defect | Evaluated by an optometrist (data obtained from medical chart). |
| Ability to comprehend the instructions for the testing procedures | Preliminary interview and Montreal Cognitive Assessment (MoCA). The MoCA was used to detect mild cognitive impairment such as attention and concentration, executive functions, memory, language, visuoconstructional skills, conceptual thinking, calculation and orientation (Nasreddine et al., 2005). Scores below 26 were indicative of mild cognitive impairment, a condition of exclusion. |
| No depression | A score of $>5/15$ on the short-form Geriatric Depression Scale (GDS) is suggestive of depression, while a score $>10/15$ is almost always indicative of depression (Sheikh & Yesavage, 1986). |
| No additional neurological disorder | Medical chart and preliminary interview. |
| No significant deficit in proprioception | A score of $\geq 75\%$ in the sensation evaluation of the Fugl-Meyer Assessment ($\geq 18/24$, as well as a score of $\geq 12/16$ in the position sense subscale of the sensation evaluation) |

[Table 2](#): Inclusion criteria for control subjects

| | |
|---|---|
| No neurological or musculoskeletal disorder | Preliminary interview. |
| Age 40-70 years | Motor coordination declines with age, and 70 years of age is a reasonable cut-off at which this decline becomes more apparent (Desrosiers et al., 1995). It is hard to distinguish the effects of age from the effects of the stroke, which may introduce bias. |
| No depression | A score of >5/15 on the short-form Geriatric Depression Scale (GDS) is suggestive of depression, while a score >10/15 is almost always indicative of depression (Sheikh & Yesavage, 1986). |

[Table 3](#): Summary of participant recruitment, data collection initiation and data collection completion

| | |
|---|----|
| Stroke Group: | |
| Potential participants contacted | 34 |
| - After initial screening questions, did not meet eligibility criteria | 10 |
| Participants with whom data collection was initiated | 24 |
| - Data collection terminated because participant did not meet eligibility requirements in the clinical evaluation | 5 |
| - Data collection terminated because participant was unable to complete experimental session | 2 |
| - Participants who completed data collection | 17 |
| Healthy Group: | |
| Potential participants contacted | 13 |
| Participants with whom data collection was initiated | 13 |
| - Data collection terminated because participant was unable to complete experimental session | 1 |
| - Participants who completed data collection | 12 |

3.4 RECRUITMENT

Clinical coordinators from the rehabilitation hospital network of CRIR (JRH and l'Institut de réadaptation Gingras-Lindsay-de-Montréal, IRGLM) as well as HRVM helped us to identify potential participants. Healthy individuals were identified from people known by the sample stroke population and from volunteers recruited via approved advertisements in the participating hospitals.

Potential participants were contacted by an associate not directly involved with the project. They were given a brief description of the study and asked if they were willing to be contacted by the

research study coordinator. The research study coordinator contacted willing potential participants to provide information about the study procedures, answer questions about the study, screen for eligibility criteria, and schedule appointments for clinical assessment and data collection. At the first session, participants provided written consent and were aware that they always had the option of withdrawing from the study at any time without giving a reason.

3.5 DATA COLLECTION

Participants were scheduled for two sessions. The first session was a 2.5 hour clinical evaluation to determine the levels of sensorimotor impairment and motor function of the shoulder, arm, and hand. The second session was a 2 hour experimental session in which the participants performed repetitive trials of a reaching and obstacle avoidance task within a three dimensional virtual environment (3D VE). The VE was built with the Computer Assisted Rehabilitation Environment (CAREN) software (Motek BV, Amsterdam) on a PC Windows 2000 operating system. Movements and orientation of the arm and trunk were recorded for 5-10s at 120Hz with a 3D optical tracking system (Optotrak Certus, Northern Digital, Waterloo, Canada) and integrated into the 3D VE in real time.

3.5.1 EVALUATION SESSION

An experienced physical therapist conducted the clinical examinations with the participants. The clinical tests were used for two purposes. One was used to screen for participant eligibility and the second was used to determine clinical status of the participants. In this section (3.5.1), descriptions and psychometric properties are given for each clinical test (Stroke Engine, 2013).

3.5.1.1 SCREENING ASSESSMENTS

The following assessment tools were used to verify participant eligibility: Chedoke-McMaster Stroke Assessment, Line Bisection Test, Montreal Cognitive Assessment, the sensation assessment of the Fugl-Meyer Assessment, Geriatric Depression Scale, and Edinburgh Handedness Inventory. In this section each of the scales will be listed and detailed descriptions will be listed in subsections A) through F) below.

A) CHEDOKE-McMASTER STROKE ASSESSMENT

The Chedoke-McMaster Stroke Assessment (CMSA) arm and hand subscales measure arm and hand neuromusculoskeletal and movement-related impairment (Gowland et al., 2003). The sample population of interest were well-recovered stroke subjects, so only people with scores of 5 or greater in the arm and hand subscales were accepted into the study.

The CMSA has excellent content, criterion and construct validity [Content: A literature review found evidence to establish a theoretical basis underlying the content of the CMSA (Moreland et al., 1993); Criterion: concurrent – correlation of CMSA with Fugl-Meyer Assessment, $r=0.95$, and Functional Independence Measure, $r=0.79$ (Gowland et al., 1993); Construct: convergent – correlation of the shoulder, elbow, forearm, wrist and hand FMA with the arm and hand subscales of the CMSA, $r=0.95$ (Gowland et al., 1993)].

The CMSA has excellent test-retest, intra-rater, and inter-rater reliability [test-retest: ICC=0.96 for the gross motor function index (Gowland et al., 1993); intra-rater: ICC=0.95 for arm and ICC=0.93 for hand (Gowland et al., 1993); inter-rater: ICC=0.88 for arm and ICC=0.93 for hand (Gowland et al., 1993)].

B) LINE BISECTION TEST

The Line Bisection Test (LBT) detects the presence of unilateral spatial neglect (Schenkenberg et al., 1980; Plummer et al., 2003). A series of horizontal lines on one page are presented and the subject indicates the centre of each line with a mark. Neglect is indicated if the bisection mark is not centred and if all uneven bisection marks are displaced toward the side of the brain lesion. Any presence of neglect is a cause for exclusion.

The LBT has excellent construct validity [Construct: convergent – correlations with Albert's Test, $r=0.85$ (Agrell et al., 1997) and Baking tray task, $r=-0.66$ (Bailey et al., 2000); correlations with the Star Cancellation Test, $r=-0.40$ (Marsh & Kersel, 1993) and with mean CT-scan damage, CT-scan damage of temporal lobe, parietal lobe, and occipital lobe ($r = -0.44, -0.59, -0.37$, and -0.42 , respectively; Egelko et al., 1988)].

The LBT is highly sensitive and has excellent test-retest reliability [sensitivity = 76.4% (Bailey et al., 2000); $r=0.84$ to $r=0.93$ (Schenkenberg et al., 1980)].

C) MONTREAL COGNITIVE ASSESSMENT

The Montreal Cognitive Assessment (MoCA) detects mild cognitive impairment such as deficits in attention and concentration, executive functions, memory, language, visuoconstructional skills, conceptual thinking, calculation and orientation (Nasreddine et al., 2005). Scores below 26 are indicative of mild cognitive impairment, a condition of exclusion.

The MoCA has excellent criterion and construct validity [Criterion: concurrent – correlation with the Mini Mental State Examination, $r=0.87$ (Nasreddine et al., 2005); Construct: known groups– the MoCA distinguished between healthy controls and mild cognitively impaired patients (Nasreddine et al., 2005)].

The MoCA has excellent internal consistency and test-retest reliability [internal consistency: Cronbach's $\alpha=0.83$ (Nasreddine et al., 2005); test-retest: $r=0.92$ (Nasreddine et al., 2005)].

D) SENSATION AND JOINT PAIN SCORES ON THE FUGL-MEYER ASSESSMENT

The FMA is a stroke-specific, performance-based impairment measure (Fugl-Meyer et al., 1975; Gladstone et al., 2002). The upper limb sensation and joint pain subsections assess light touch, position sense and joint pain during passive motion of the arm and hand joints. (The psychometric properties are presented in section 3.5.1.2D.)

E) GERIATRIC DEPRESSION SCALE

The Geriatric Depression Scale (GDS) detects the presence of depression, indicated by a score of $>10/15$ on the short-form GDS (Sheikh & Yesavage, 1986).

The GDS has excellent construct validity [Construct: convergent – correlations with the Hamilton Rating Scale for Depression, $r=0.84$ and the Zung Self-Rating Depression Scale, $r=0.83$ (Yesavage & Brink, 1983)].

The GDS has excellent internal consistency and test-retest reliability [internal consistency: $\alpha=0.94$ (Yesavage & Brink, 1983) and $\alpha=0.90$ (Agrell & O'Dehlin, 1989); test-retest reliability: ICC=0.84 (Yesavage & Brink, 1983)].

F) EDINBURGH HANDEDNESS INVENTORY

The Edinburgh Handedness Inventory quantitatively assessed hand dominance by scoring hand preference in 10 activities and calculating a laterality quotient (Oldfield 1971). Negative numbers indicated left hand preference and numbers approaching zero indicated ambidexterity. Stroke subjects were not excluded for left-hand dominance; rather the dominance was noted for the purposes of matching with control subjects.

3.5.1.2 CLINICAL ASSESSMENTS

Evaluations of sensorimotor impairment, activity limitations, and self-efficacy were administered. The clinical evaluations were chosen to quantify impairments and limitations according to the International Classification of Functioning, Disability and Health (WHO, 2014). In this section each of the scales will be listed and detailed descriptions will be listed in subsections A) through I) below. Impairments of neuro-musculoskeletal and movement related functions were assessed by measuring shoulder strength and range of motion, as well as administering the Composite Spasticity Scale (CSS), Fugl-Meyer Assessment (FMA) and Reaching Performance Scale for Stroke (RPS). Shoulder strength and ROM were measured in each arm and compared to the less affected side to detect any residual deficit in the affected arm. Spasticity was assessed using the CSS, which scored biceps tendon jerk, resistance to elbow extension and clonus and then summed the scores to give a total spasticity score (Levin & Hui-Chan, 1992). The FMA evaluated reflexes, voluntary arm movements, finger-to-nose coordination, sensation, kinaesthesia and range of motion in order to score disease severity and describe motor recovery (Fugl-Meyer et al., 1975; Gladstone et al., 2002). The RPS evaluated the amount of compensation used for reaching (Levin et al., 2004). The clinical evaluations for activity limitations and participation restrictions included the Wolf Motor Function Test (WMFT), Box and Block Test (BBT), Motor Activity Log (MAL), and a self-efficacy scale based on the MAL (MAL-SES). The WMFT measured global arm function and quantified residual arm motor ability such as carrying, moving, and handling objects (Wolf et al., 1995). The BBT measured gross manual dexterity and quantified the residual deficit in the affected hand (Cromwell 1976). The MAL measured how much and how well the participant used their arm in everyday life activities after stroke (Taub et al., 1994; Uswatte et al., 2006). A self-

efficacy scale was created by asking subjects how confident they were in completing the 30 tasks listed in the MAL.

A) SHOULDER STRENGTH

The strength of the anterior, middle, and posterior portions of the right and left deltoid muscles were measured using a handheld dynamometer (Nicholas, MMT, Lafayette Instruments – model 01160) in standard muscle testing positions (Hoppenfeld 1976). Subjects were seated in a chair with a back support with their feet fully resting on the floor. Arms rested vertically on the side of the body. The physiotherapist stood behind the subjects and stabilized the scapula while palpating the deltoid muscle. The handheld dynamometer was placed proximal to the elbow – anterior for the anterior deltoid, lateral for the middle deltoid, and posterior for the posterior deltoid. The elbow was flexed to 90° and the physiotherapist performed a break test where they gradually applied resistance to shoulder extension, flexion, and abduction (Bohannon 1988). The measure of strength was the mean value of three attempts for the anterior, middle, and posterior portions of each deltoid muscle measured in kilograms.

B) SHOULDER RANGE OF MOTION

The passive ROM of flexion, extension, horizontal abduction, horizontal adduction, internal rotation, and external rotation of the right and left shoulder joints were measured with a goniometer according to standard clinical procedures (Hoppenfeld 1976). Subjects were positioned supine on a plinth.

C) COMPOSITE SPASTICITY INDEX

The CSI rates spasticity by combining three common clinical measures of spasticity (biceps tendon jerk, resistance to elbow extension and clonus) resulting in a score out of 16 (Levin & Hui-Chan, 1992). Scores ranging from 0-9 indicate mild spasticity, 10-12 indicate moderate spasticity and 13-16 indicate severe spasticity. The CSI is valid (Goulet et al., 1996) and reliable (Levin & Hui-Chan, 1992).

D) FUGL-MEYER UPPER LIMB ASSESSMENT

The FMA is a stroke-specific, performance-based impairment measure (Fugl-Meyer et al., 1975; Gladstone et al., 2002). The upper limb subsection assesses the ability of the subject to make isolated movements within and out of pathological synergy patterns. A maximum score of 66 indicates normal function.

The FMA has excellent construct, content and criterion validity. [Construct: correlation of the upper extremity portion of the FMA with the Action Research Arm Test, $r=0.93$; and with the Box and Block Test, $r=0.92$ (Platz et al., 2005); Content: acceptable fit statistics from a Rasch Analysis (Woodbury et al., 2008), and high coefficients of reproducibility (>0.9) and scalability (>0.7) from a Guttman Scale Analysis suggesting that each motor functioning subscale has a valid, cumulative, and unidimensional Guttman scale (Crow & Harmeling-van der Wel, 2008); Criterion: concurrent validity – correlation between FMA upper extremity subscore and Barthel Index total score 5 weeks post-stroke, $r=0.82$ (Wood-Dauphinee et al., 1990); predictive validity – Spearman correlation between FMA and Barthel Index scores at discharge, $\rho_h = 0.72$, meaning that the FMA was able to predict the Barthel Index scores at discharge (Hsueh et al., 2008)].

The FMA has excellent internal consistency, inter-rater and test-retest reliability [internal consistency: $\alpha=0.94$ to 0.98 (Lin et al., 2004); inter-rater: $ICC=0.99$ for the total motor score, $ICC=0.98$ for passive joint motion/joint pain (Platz et al., 2005); test-retest: $ICC=0.97$ for total motor score, $ICC=0.95$ for passive joint motion/joint pain (Platz et al., 2005)].

E) REACHING PERFORMANCE SCALE

The RPS evaluates upper limb and trunk movement quality while reaching and grasping for an object placed close to and far away from the body of the seated subject. Six components are scored: trunk displacement, movement smoothness, shoulder and elbow movements, quality of prehension and the overall accomplishment of the task. A score of 18/18 is indicative of a smooth reaching movement with no compensatory movements.

The RPS has excellent criterion and construct validity [Criterion: concurrent – correlation between the RPS close target score and the CMSA arm ($r=0.92$) and hand ($r=0.95$) scores;

correlation between the RPS far target score and the CMSA arm ($r=0.90$) and hand ($r=0.93$) scores (Levin et al., 2004); Construct: discriminant – an ANOVA was performed comparing RPS scores of mild, moderate and severely affected post-stroke individuals. All scores were significantly different between group (close target: $F=26.9$, $p<0.01$; far target: $F=25.6$, $p<0.01$; Levin et al., 2004)].

The RPS has had preliminary reliability analysis showing adequate inter-rater reliability [among 3 raters, agreement on individual components of the scale ranged from 43%-93%, where $ICC=0.84(0.58-0.95)$ for the close target and $ICC=0.89(0.69-0.96)$ for the far target; Levin et al., 2004].

F) WOLF MOTOR FUNCTION TEST

The WMFT quantifies arm motor ability through timed and functional tasks (Wolf et al., 2001). The modified version of the WMFT consists of 17 items. The first 6 items are timed functional tasks. Items 7, 8 and 14 measure strength. The remaining 8 items analyze movement quality when completing various tasks (Wolf et al., 2001; Whittall et al., 2006). The items are rated on a 6-point scale where lower scores are indicative of lower functioning levels.

A shortened version of the WMFT was used in the clinical evaluation (Bogard et al., 2009). It was found to be valid and responsive (Wu et al., 2010).

The WMFT has adequate criterion and construct validity [Criterion: concurrent – $r=0.57$ for a correlation between the WMFT and the upper extremity subscale of the FMA (Wolf et al., 2001; Construct: known groups – WMFT scores for individuals without impairment were significantly higher than scores of stroke subjects (Wolf et al., 2001)]].

The WMFT has excellent internal consistency, inter-rater and test-retest reliability [internal consistency: $\alpha=0.98$; inter-rater: $ICC=0.94$; test-retest: $ICC=0.95$ (Nijland et al., 2010)].

G) BOX AND BLOCK TEST

The BBT measures unilateral manual dexterity through a simple transfer task (Cromwell 1976). A wooden box divided into two sections with 150 blocks is placed on a table in front of the

subject. The subject moves the blocks one by one from one section to the other section as quickly as possible, aiming to move as many blocks as possible within 60 seconds. A higher number of transferred blocks indicate better hand dexterity.

The BBT has adequate criterion and construct validity [Criterion: concurrent – $r=-0.55-0.80$ for correlations between the BBT, Action Research Arm Test (ARAT), and Nine-Hole Peg Test; Lin et al., 2010; Construct: convergent/discriminant – BBT scores highly correlated with the Minnesota Rate of Manipulation Test, $r=0.91$ (American Guidance Service 1969), ARAT, $r=0.95$ and FMA upper extremity items, $r=0.92$ (Platz et al., 2005)].

The BBT has excellent inter-rater and test-retest reliability [inter-rater: ICC=0.99 (Platz et al., 2005); test-retest: ICC=0.97 (Desrosiers et al., 1994)].

H) MOTOR ACTIVITY LOG

The MAL is a structured interview in which subjects will rate how well (Quality of Movement, MAL-QOM) and how much (Amount of Use, MAL-AOU) they used the impaired arm in 28 upper limb activities during the preceding week (Taub et al., 1993). The rating scale ranges from 0=never used to 5=same as pre-stroke, and is scored in 0.5 increments. If an activity is not performed it is excluded from the final score. The final score is the sum of the scores of all activities in a subscale (i.e. MAL-QOM or MAL-AOU) divided by the number of activities performed. The final score for each subscale ranges from 0 to 5 where higher values indicate arm usage and quality of movement closer to amounts of pre-stroke arm use.

The MAL has good construct validity [Construct: correlation of the MAL with the Action Research Arm Test, $r=0.63$ (van der Lee et al., 2004)].

The MAL has excellent internal consistency and test-retest reliability [internal consistency: AOU $\alpha=0.88$; QOM $\alpha=0.91$ (van der Lee et al., 2004); test-retest: ICC=0.97 (van der Lee et al., 2004)].

1) MOTOR ACTIVITY LOG – SELF-EFFICACY

The MAL is a valid measure of the amount and quality of arm use after stroke and indicates that higher arm and hand use during ADLs are associated with higher levels of dexterity (van der Lee et al., 2004). The MAL-QOM was also shown to have a higher sensitivity to capture spontaneous arm use than the MAL-AOU in mildly- to moderately-affected post-stroke individuals (van der Lee et al., 2004; Uswatte et al., 2005; Chen et al., 2009). Therefore, the initial impetus to base a new arm self-efficacy questionnaire on the MAL questionnaire was the validity and reliability of the MAL as a measure of arm physical ability. Self-efficacy was measured by applying a self-efficacy response metric to each of the MAL activity items (MAL-SES). The self-efficacy response metric is taken from Bandura's Conceptual Model of Self-Efficacy and Guide for Constructing Self-Efficacy Scales (Bandura 2001). There are various self-efficacy scales developed for stroke (for review see Jones & Riazi, 2011, and Korpershoek et al., 2011), but not specifically for upper limb function. Subjects are asked how confident they are in completing each item on the MAL. The possible responses are presented on a horizontal visual analogue scale, from 0=not at all confident to 10=completely confident. The mean response is reported. The MAL-SES final score ranges from 0 to 10 where higher values indicate higher self-efficacy for the incorporation of arm use in daily activities.

3.5.2 EXPERIMENTAL SESSION

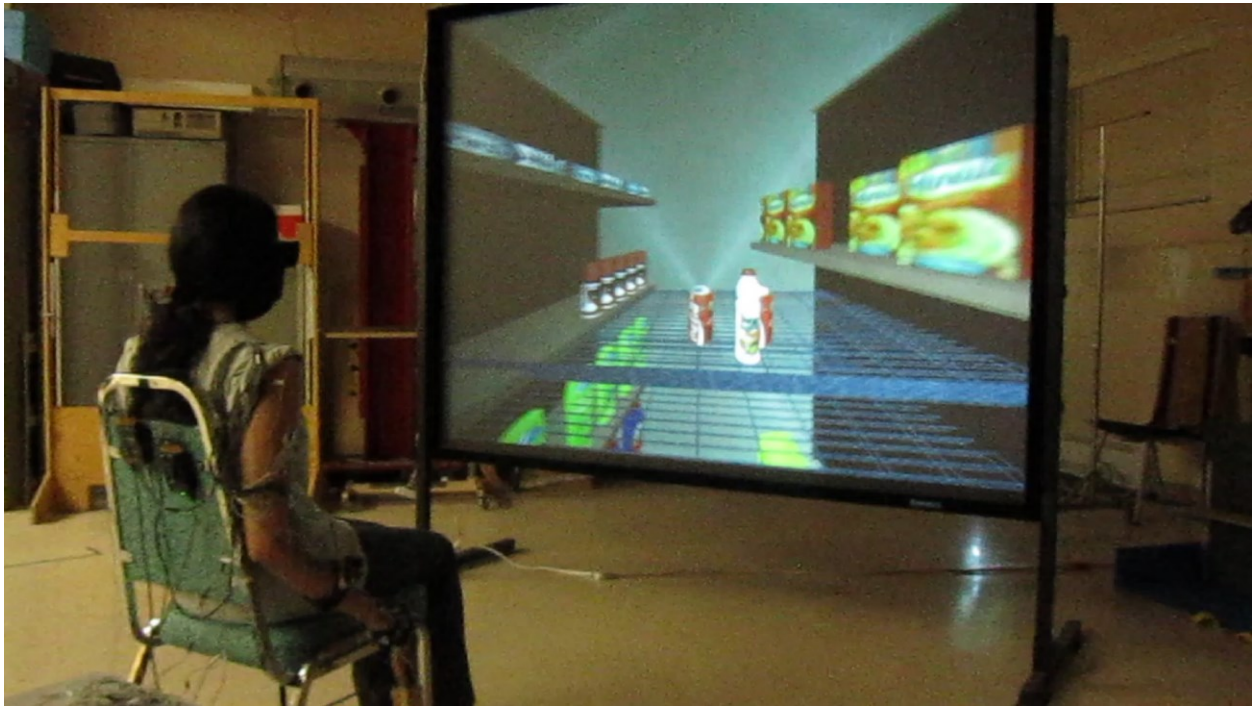
The experimental session was two hours in length (one hour setup, one hour data collection). Subjects performed a task where they rapidly reached for a target object over many trials. In random trials a moving obstacle appeared between the person and the target object while they were in the process of reaching. In order to be successful in the obstructed trial, subjects had to make contact with the target object and not hit the obstacle. A more detailed explanation of the task is given in section 3.5.2.4 below.

3.5.2.1 LAYOUT OF THE PHYSICAL ENVIRONMENT AND SUBJECT POSITION

A standard chair with a backrest and no armrests was placed 1 metre in front of a 2m x 1.5m rear projection screen. The feet of the participants rested flat on the floor and the knee and hip joints

were flexed to 90°. The resting position of the arm to be tested was hanging alongside the body. The upper arm was vertical and the elbow was bent to 30°. The forearm and hand were in a neutral position between supination and pronation. The tip of the third finger resting on a support placed next to the chair in order to standardize the endpoint starting position. The non-tested arm rested comfortably on the lap ([Figure 1](#)).

[Figure 1](#): Picture of the physical set-up of the experiment. A standard chair was placed 1m in front of a 2m x 1.5m projection screen. Participants sat comfortably with feet flat on the floor and hips and knees at 90°. The starting position of the reaching arm was hanging on the side of the body with the elbow flexed to 30°, wrist in neutral position, and third finger resting on a support placed next to the chair.

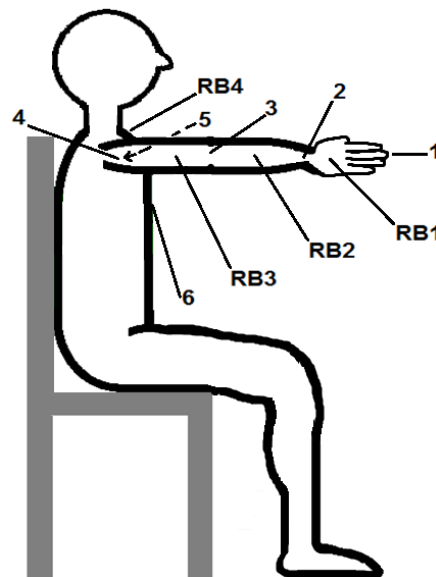


3.5.2.2 RECORDING AND ONLINE CONTROL OF MOVEMENT IN THE VE

Planar and rotation movements of the trunk, arm and hand were recorded with two camera bars of the Optotrak Certus motion analysis system. One bar was suspended horizontally from the ceiling directly above the screen. The other bar was placed vertically on the floor two metres away from the subject on the side being tested. The Optotrak system tracked the positions of 24 infrared-emitting markers placed on various body landmarks – four rigid bodies and seven

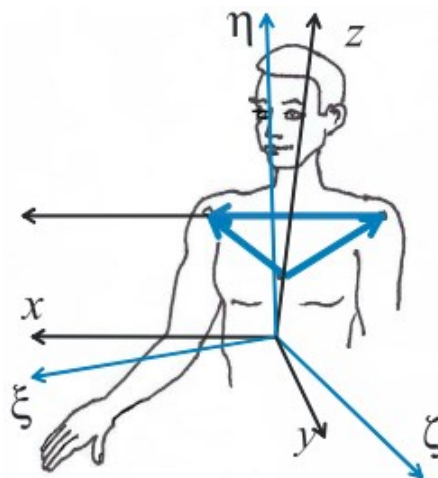
individual markers ([Figure 2](#)). Four rigid bodies consisting of groups of non-coaxial markers were placed on the hand (six markers), mid-forearm (six markers), mid-upper arm (three markers), and sternal notch (three markers). They were used to track hand, forearm, upper arm and trunk rotations. Six individual markers were placed on the mid-sternum, ipsilateral and contralateral acromio-humeral joints, lateral epicondyle of the elbow, mid-point between the dorsal aspect of the styloid processes of the wrist, and the dorsal surface of the distal phalange of the middle finger. The configuration of the five arm markers defined the arm segments. The trunk was defined by the shoulder markers and the mid-sternum marker. Data from individual markers were used to calculate endpoint, trunk and arm joint planar kinematics (shoulder flexion, shoulder horizontal adduction, elbow extension, wrist flexion, wrist abduction, and trunk flexion angles).

[Figure 2](#): Placement of the infrared markers on the reaching arm and trunk. Six individual markers were placed on the dorsal surface of the distal phalange of the middle finger (1), mid-point between the dorsal aspect of the styloid processes of the wrist (2), lateral epicondyle of the elbow (3), ipsilateral (4) and contralateral (5) acromio-humeral joints and the mid-sternum (6). Four rigid bodies (RB) consisting of groups of non-coaxial markers were placed on the hand (six markers, RB1), mid-forearm (six markers, RB2), mid-upper arm (three markers, RB3), and sternal notch (three markers, RB4).



The arms of the participants were represented in real time by an avatar in the VE. Participants saw an avatar of their forearm and hand on the screen. Vision of their real arm and hand was reduced by blocking peripheral vision with black felt mounted on the frame of the 3D glasses and by turning off all lights in the physical environment. They controlled the position of the forearm and hand by moving their shoulder, elbow and wrist joints. Online monitoring of the spatial positions of the individual upper limb joint markers as well as the rotation of the rigid bodies attached to the forearm and upper arm allowed the subject to have online control of the avatar's position within the VE. Trunk flexion and rotation were measured within an absolute frame of reference with respect to the room – ξ , ζ , η . The vertical axis, η , was perpendicular with the floor. All other angles were defined in a body-centred coordinate system. The X axis was defined by line connecting the right shoulder marker with the left shoulder marker. The Z axis was along the line connecting the sternum marker with the middle point on the inter-shoulder line. The Y axis projected forward from the trunk ([Figure 3](#)). The frontal plane was defined as the XZ plane, the sagittal plane as the YZ plane, and the coronal plane as the XY plane. Positive X values indicated rightward lateral movement. Positive Y values indicated anterior displacement. Positive Z values indicated movement in the upward direction.

[Figure 3](#): The frames of reference used to measure movement kinematics. The absolute vertical axis, η , was perpendicular to the floor and was used as the reference zero position to measure trunk flexion and rotation. All other joint angles were defined in the illustrated X, Y, Z coordinate system. The X axis was defined by a line connecting the right shoulder marker with the left shoulder marker. The Z axis was along the line connecting the sternum marker with the middle point on the inter-shoulder line. The Y axis projected forward from the trunk.



There were two calibration positions for the arm. The first was used to set the origin ($X=0$, $Y=0$, $Z=0$) for the VE and the second was used to set the size and position of the avatar arm to visualize in the VE. In the first calibration position, the straight arm was pointed forward, parallel with the floor and slightly horizontally adducted so that the middle metacarpophalangeal (MCP) joint was in line with the middle of the trunk. The position of the MCP joint was set as the origin of the VE.

In order to ensure that the avatar arm was representative of both location and size of the participant's actual arm, a second calibration procedure was performed. In the second calibration position, the straight arm was pointed forward and parallel with the floor, and the hand was positioned in line with the shoulder. The positions of the markers on the middle finger, wrist, elbow, shoulders and trunk were used to determine: (1) the dimensions of the avatar forearm and hand displayed on the screen; and (2) the position of the avatar in the VE so that it corresponded to subject's own forearm and hand position. The distance between the person and the target object was calibrated so that the person perceived the avatar forearm and hand as being an extension of his or her own body. To ensure that the subjects perceived that the target was at a reachable distance, the target was placed at an arm distance equal to length between the axilla and wrist crease (Mark et al., 1997). This also ensured that the movements were ecologically valid (Mon-Williams et al., 2001).

3.5.2.3 THE VIRTUAL ENVIRONMENT

The nature of upper limb movements in physical environments (PE) and VE has been shown to be similar (Viau et al., 2004). In a PE, it is difficult to set up a task requiring critical timing and obstacle avoidance. It is also hard to have full control of the spatial and temporal characteristics of the task in order to tailor the task for each individual. Therefore, the obstacle avoidance task was created in an ecologically valid VE. The ecological validity of an experiment can be judged by how closely the nature of the experimental setting, stimuli and resulting behaviour represent real-world phenomena (Schmuckler 2001). Well-designed VEs can be ecologically valid for stroke subjects. For example, stroke subjects participating in a movement training protocol using a VE had improvements in clinical functional scores (Rand et al., 2005).

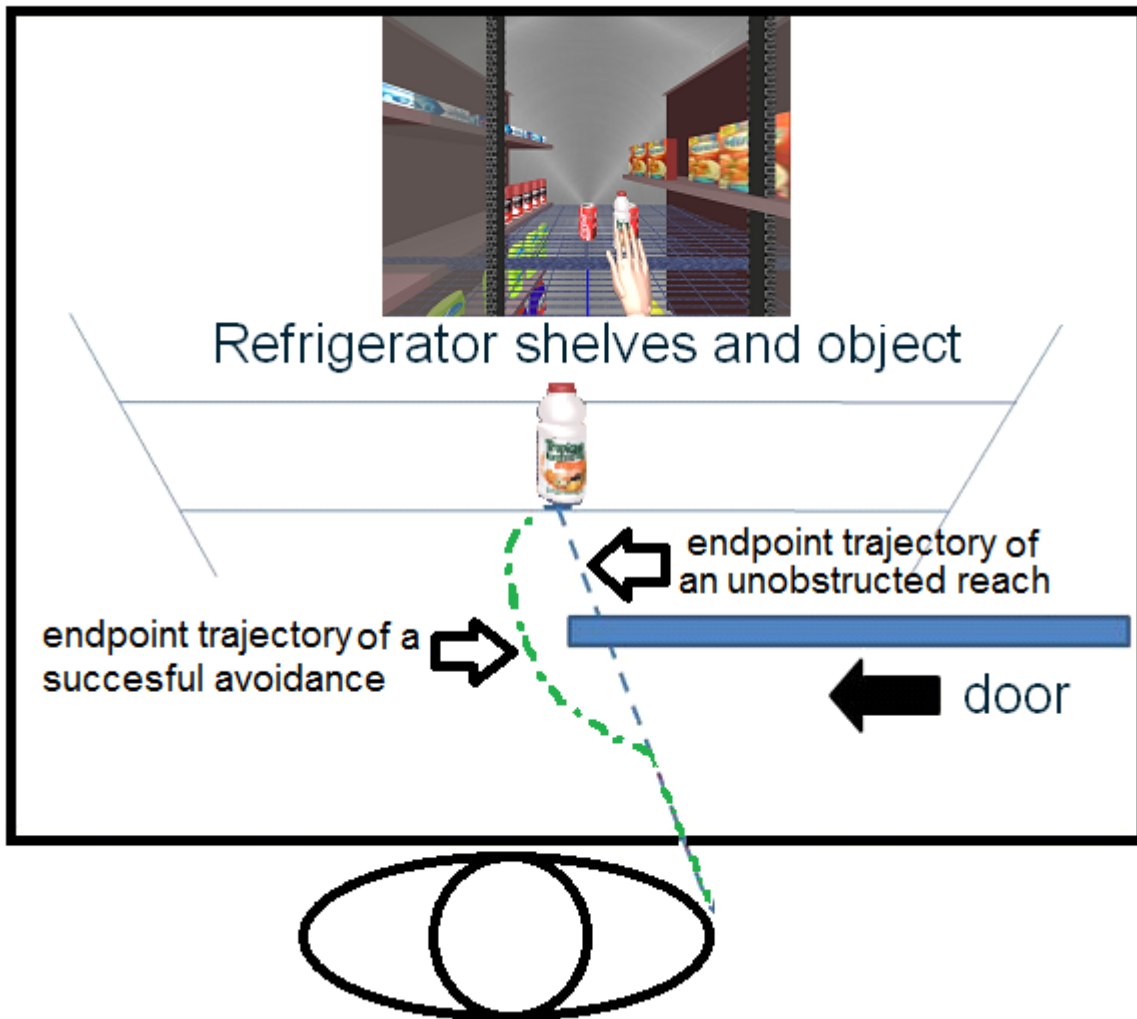
The VE setting was designed as a grocery store commercial refrigerator with double sliding doors. The target object for the experimental task was a juice bottle placed on a shelf in the refrigerator. A subject was asked to reach for and touch the juice bottle. After touching the bottle, the bottle attached to the virtual hand and the subject was able to bring it back towards their side.

In order to create a sense of immersion in the VE, a stereoscopic technique of presenting the VE image was used. The environment image was projected from two projectors onto the screen and the two images were offset. The resulting overlapping images were viewed through stereoscopic glasses with black felt restricting peripheral vision. Also, the room was darkened to optimize viewing.

When testing involved the right arm, the centre of the juice bottle was 10cm to the right (X axis) and 15cm in front (Y axis) of the origin. The object was moved to -10cm to the left along the X axis from the origin when the left arm was being tested.

To obstruct the juice bottle, glass refrigerator doors were programmed to close by sliding either from the right or left side of the screen ([Figure 4](#)). The doors moved along the X axis and were positioned 7cm posterior to the origin. When the right or left door was in the closed position, the medial edge of the door was positioned at 9cm to the right of the origin along the X axis, thus only partially obstructing the juice bottle. The door was timed to begin closing when the tangential velocity of the wrist marker reached 10% of the mean peak wrist tangential velocity, which was calculated by averaging the peak velocities of a block of unobstructed fast reach trials (Block 1; refer to next section for block descriptions). The door then completely closed by the time the wrist had attained peak velocity. This value was chosen since Ghafoury and Feldman (2002) showed that because of the high speed of the motor command, by the time the peak velocity of the arm movement was attained, the command shift for the initial movement was already completed. This also implied that secondary or corrective movements could only occur after the time of peak velocity of the first hand movement.

[Figure 4](#): Schematic of the obstacle avoidance task. In this illustration, participants are seated in front of the refrigerator. When they begin to reach forward, the right door begins to close and obstructs a straight line path to the target in random trials. Participants reach for the target as quickly as possible without touching the door.



3.5.2.4 EXPERIMENTAL PROCEDURE

Before testing, all subjects went through a familiarization procedure to habituate them to the VE and the reaching task. Subjects repetitively reached for the juice bottle for a minimum of 30 trials. At the beginning of each trial, a single tone indicated that the subject was to start reaching as fast as possible toward the juice bottle on the refrigerator shelf. When they touched the juice bottle, they received a “ping” sound. After familiarization, subjects completed four blocks. Subjects were instructed that there would be 3 conditions in the experiment that could occur during a reach: unobstructed; left door obstruction; and right door obstruction. For this thesis, only the trials in which the door closed from the more-affected side were used for determining the obstacle avoidance strategy. Trials in which the door closed from the opposite side were included so that subjects would not anticipate the order of the task conditions in the randomized block (see Block 4 below).

In Block 1 (Template block; $n=15$ trials), the reaching path was unobstructed when the subjects reached for the juice bottle as fast as possible. Performance in all trials of this block were averaged and used as the template against which all other conditions were compared.

In Block 2 the left door closed and in Block 3 the right door closed during the reach ($n=30$ obstructed trials for each block). These blocks were practice blocks so that subjects could learn how to avoid each door. Subjects reached around the door without touching it with any surface of the hand and forearm. Subjects were given visual and auditory feedback about their task performance. If they avoided the door and touched the juice bottle, subjects heard a ping sound indicating success. If the arm collided with the door, there was a sound of cracking glass and they were no longer able to touch the juice bottle. Subjects were considered to have learned the task when they successfully avoided the door for at least 10 trials in a row. A block was repeated if subjects needed more practice.

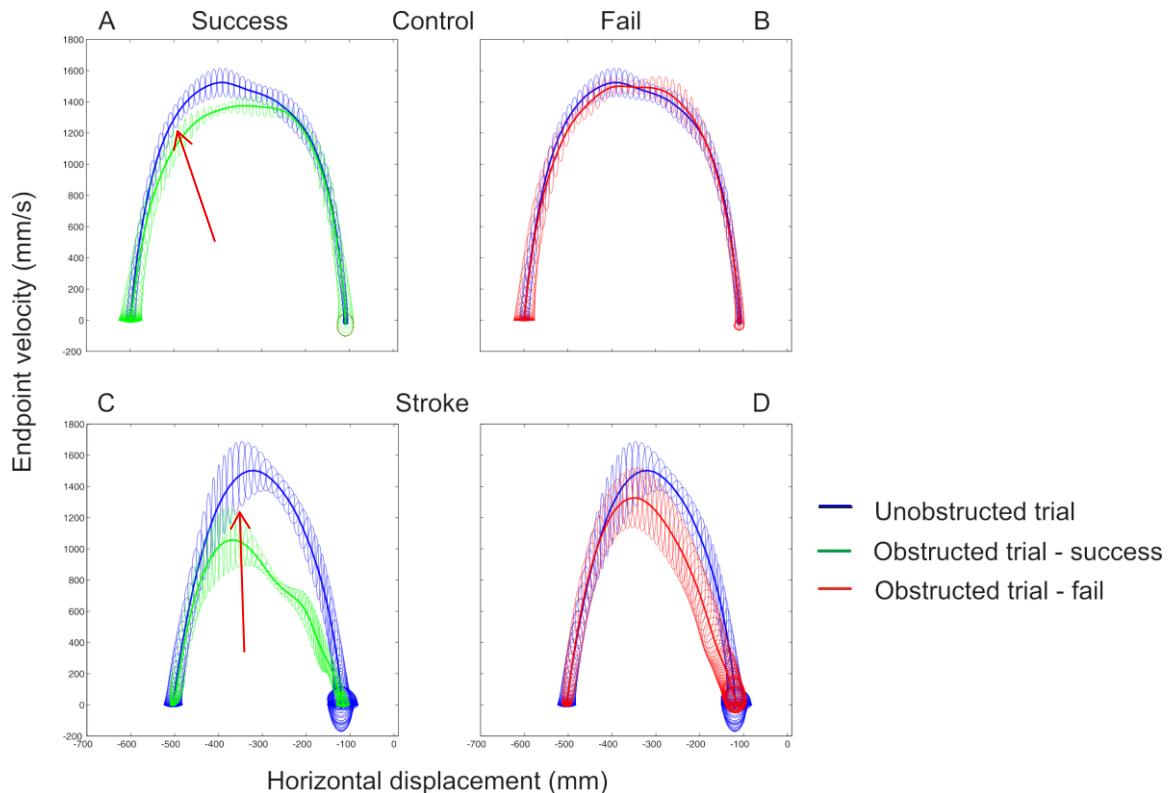
Block 4 was the “Randomized block” consisting of 60 trials. In $1/3$ of the trials the right door closed and in another $1/3$ of trials the left door closed. The remaining $1/3$ of trials in Block 4 were unobstructed. All conditions were pseudo-randomized (pre-set randomized condition order) within Block 4. To avoid fatigue, subjects were allowed to rest whenever they needed.

3.6 DATA ANALYSIS

3.6.1 OUTCOME MEASURES

Divergence point, DP: The tangential velocity/position phase profiles of each trial in a task condition were averaged and represented as a mean phase plot. The mean phase plot represented the tangential velocity of the endpoint at any given time along the reach trajectory from the start position to the final position in the sagittal (YZ) plane. In [Figure 5](#), the phase plot of the template block (blue) was overlaid with the phase plots of the successful or failed obstructed blocks (green or red, respectively). The ellipses overlaid on each of the phase plots represented the standard deviation. The DP was defined as the point on the overlaid phase plots where the template phase plot and obstructed phase plots deviated from each other (running t-test between adjacent coordinates on the blue and green/red traces, $p < 0.05$) and expressed as the percent of total reach distance from the starting position.

[Figure 5](#): Divergence points of average phase (position/velocity) plots of successful (left column) and failed (right column) trials from template phase plots (blue traces in all graphs). The first row contains examples of successful (A) and failed (B) phase trials for one control subject. The second row contains examples of successful (C) and failed (D) for one stroke subject. S5 had an FMA=57.



Success rate was calculated as the number of successful trials in the randomized block divided by the total number of trials in the block. Success was defined as not hitting the door with the hand or arm. Success rates were compared between groups. A comparison of the proportion of individuals in each group who achieved different levels of success rates was then performed.

ENDPOINT TASK PERFORMANCE VARIABLES:

Endpoint peak velocity (EPV; m/s) was defined as the highest value in the wrist tangential velocity profile during the reach. Tangential velocity was computed from the X, Y, Z position data of the wrist marker. Movement initiation was defined as the time at which the arm tangential velocity went above and remained above 10% of the peak arm tangential velocity for at least 50ms. Movement end was defined as the time at which the arm tangential velocity went below and remained below 10% of the peak arm tangential velocity for at least 50ms.

Endpoint time to peak velocity (ETPV; s) was defined as the length of time that the wrist marker moved from movement initiation, 10% of peak velocity on the tangential velocity profile, to peak velocity, the highest value in the tangential velocity profile.

Endpoint trajectory length (ETL; mm) was defined as the length of the path travelled by the endpoint from the time of movement initiation to movement end, determined from the tangential velocity profile.

Index of curvature (IC) measured the shape of the endpoint trajectory, thus representing movement smoothness. IC was defined as the ratio of endpoint trajectory length to length of a straight line joining the initial and final endpoint positions. A straight line would have an index of 1, whereas a semi-circle would have an index of $\pi/2=1.57$.

MOVEMENT QUALITY VARIABLES (3D ANGLES):

Shoulder flexion angle (degrees, °): The shoulder flexion angle was measured in the local frame of reference of the subject at the shoulder. The 0° position was defined as the vertical position (Z) of the upper arm when the arm was in line with the trunk. A positive increase in the shoulder flexion angle indicated forward movement of the upper arm. For example, the straight

arm pointed forward and parallel to the floor was at 90° . Shoulder flexion range of motion was defined as the difference between the initial and final shoulder flexion angles during the forward reaching movement.

Shoulder horizontal adduction angle (degrees, $^{\circ}$): The shoulder horizontal adduction angle was measured in the relative frame of reference of the subject. The 0° position was defined as the horizontal projection of the upper arm vector on the frontal (XZ) plane, lying along the X axis and directed away from the trunk. The positive values of the shoulder horizontal adduction angle occurred when the horizontal projection of the upper arm vector moved from outward to inward across the trunk. Shoulder horizontal adduction range of motion was defined as the difference between the initial and final shoulder horizontal adduction angles during the forward reaching movement.

Elbow extension (degrees, $^{\circ}$): The elbow extension angle was measured in the relative frame of reference of the subject. The elbow angle when the arm was straight in full elbow extension was defined as 180° . The angular value decreased as the forearm moved towards the upper arm into a flexed position. Elbow extension range of motion was defined as the difference between the initial and final elbow extension angles during the forward reaching movement.

Wrist flexion (degrees, $^{\circ}$): The wrist extension angle was measured in the relative frame of reference of the subject. The 0° position was defined as the hand in neutral position, midway between wrist flexion and extension, and in line with the forearm. Positive values of the wrist flexion angle indicated when the hand moved anteriorly towards the forearm into a flexed position. Wrist flexion range of motion was defined as the difference between the initial and final wrist flexion angles during the forward reaching movement.

Wrist abduction (degrees, $^{\circ}$): The wrist abduction angle was measured in the relative frame of reference of the subject. The 0° position was defined as the hand in neutral position, midway between wrist adduction (towards the ulnar side) and abduction (towards the radial side), and in line with the forearm. Positive values of the wrist abduction angle indicated when the hand moved towards the thumb side into an abducted position. Wrist abduction range of motion was defined as the difference between the initial and final wrist abduction angles during the forward reaching movement.

Trunk pitch angle (degrees, °): The initial resting vertical position of the trunk in the absolute frame of reference was set as the initial position. Moving the trunk forward was associated with positive pitch angles. The trunk pitch range of motion was the difference between the initial and end trunk pitch angles during the forward reaching movement.

Arm joint axial rotation (degrees, °): For all axial rotations of the upper arm, forearm and hand the rotation angles were set to 0° at the initial starting position. During the forward reaching movement, positive values indicated movement into supination, while negative values indicated movement into pronation. (Hereafter, the upper arm rotation is referred to as “shoulder rotation”, forearm rotation as “elbow rotation”, and hand rotation as “wrist rotation”. This nomenclature is consistent with the analysis program used for the project. For simplicity of interpretation from the analysis program, nomenclature was retained in this thesis.)

Trunk rotation (degrees, °): The initial resting position of the trunk around the vertical axis in the absolute frame of reference was set as the initial position. Positive values indicated that the trunk was rotating away from the reaching arm. In the case of right arm reaching, the rotation was in a counter clockwise direction.

3.6.2 STATISTICAL ANALYSIS

Homogeneity of variance of the data for each group was determined using Levine’s test. If data did not meet requirements for parametric statistical tests, non-parametric tests were used. A significance level of $p < 0.05$ was used for all tests.

For Hypothesis 1A, the task success rates for each group were compared using Student’s t-tests. Also, a comparison of the proportion of individuals in each group who achieved different levels of success rates was performed.

For Hypothesis 1B, the divergence points of the endpoint phase plots were compared between the control and stroke groups using Student’s t-tests. The performance and movement quality variables of the control and stroke groups were compared using repeated-measures ANOVA.

For Hypothesis 2, the strength of the association between task success rates and clinical measures (shoulder strength and range of motion, CSS, CMSA arm and hand subscales, modified

WMFT, FMA, RPS, MAL, MAL-SES) was assessed using appropriate correlational analysis (Pearson's correlations).

For Hypothesis 3A, 3B and 3C, the task success rates and divergence points of the endpoint phase plots were compared between the first and second visits of the stroke subjects using Student's t-tests and the proportion analysis described for Hypothesis 1. The performance and movement quality variables from the first and second visits were compared using repeated-measures ANOVA. In addition, the strength of the association between task success rates and clinical measures for the second visit (shoulder strength and range of motion, CSS, CMSA arm and hand subscales, modified WMFT, FMA, RPS, MAL, MAL-SES) was assessed using appropriate correlational analysis (Pearson's correlations).

3.6.3 MISSING DATA

In cases where markers were missing for a small number of frames during a trial, data were interpolated using a cubic-spline method.

For three subjects, the MoCA was not completed. A median imputation procedure was performed to fill in the missing data. The median imputation procedure involved replacing the missing values with the median of the non-missing values of the variable of interest.

3.7 ETHICAL CONSIDERATIONS

Ethics approval was obtained from the Research Ethics Boards of CRIR and the HRVM. Subjects were made aware of the risks and advantages of study participation, and understood that they could withdraw without reason without effect on their current or future medical care. There was reimbursement for travel expenses and parking up to a sum of \$30, but participation itself was voluntary. All personal data were kept confidential. Paper records were kept in a locked cabinet at the JRH Sensorimotor Control and Virtual Reality Laboratory. Digital data were stored on a password protected server. Access to medical charts was limited to the current project, and only to the relevant details. Only members of the research team had access to the information collected during the project.

It was the intention of the author to reveal the results of the project only in the form of scientific presentations or publication, and with no exposure of subject identity.

CHAPTER 4: RESULTS

4.1 DEMOGRAPHIC DATA AND CLINICAL ASSESSMENT

The study sample population was comprised of two groups: 17 participants with stroke (4 female) and 12 control (9 female) participants. There were no differences between demographic data of the two groups in age (stroke = 62.7 ± 9.3 , control = 59.8 ± 8.5) or dominance (stroke = 3 left, control = 4 left; [Table 4](#)).

Although the individuals in the stroke group were well-recovered (FMA score range = 49-66; [Table 5](#)), they were 13-29% weaker and had 6-40% less range of motion in the affected shoulder compared to their less affected side ([Table 6](#), [Table 7](#)). The stroke group compared to the control group also had lower shoulder strength and decreased ranges of shoulder extension, horizontal adduction, and internal rotation (anterior deltoid mean difference=18.3kg, $t_{27}=2.5$, $p<0.05$; medial deltoid mean difference=13.2kg, $t_{27}=2.1$, $p<0.05$; posterior deltoid mean difference=28.3kg, $t_{27}=4.5$, $p<0.01$; extension mean difference=26.6°, $t_{27}=3.7$, $p<0.01$; horizontal adduction mean difference=20.4°, $t_{27}=2.1$, $p<0.05$; internal rotation mean difference=41.0°, $t_{27}=3.2$, $p<0.01$; [Table 8](#)). The Box and Blocks Test was completed by both groups and the stroke group was less dextrous in the task (mean difference=12.9 blocks, $t_{27}=3.0$, $p<0.01$; [Table 8](#)).

Table 4: Individual demographic data of stroke subjects and control subjects

| Subject | Age (years; 1 st visit/2 nd visit) | Sex | Time since stroke (months; 1 st visit/2 nd visit) | Dominant side/Lesion location |
|---------|--|-----|---|--|
| S1 | 72.0/73.0 | M | 27/39 | R / L putamen and internal capsule and subarachnoid (hemorrhagic) |
| S2 | 50.1/51.2 | M | 16/29 | R / L putamen (hemorrhagic) |
| S3 | 55.4/56.4 | M | 12/24 | L / R internal capsule (ischaemic) |
| S4 | 49.3/50.1 | F | 7/17 | R / L Sylvian |
| S5 | 70.4 | F | 80 | R / L internal capsule |
| S6 | 75.7 | M | 11 | R / L lentiform nucleus and internal capsule (hemorrhagic) |
| S7 | 51.3 | M | 10 | R / L fronto-parietal sylvian, L paracentral gyrus, L Rolandic fissure (ischaemic) |
| S8 | 78.1/79.0 | M | 33/44 | R / L lacunar, corona radiata (ischaemic) |
| S9 | 63.4/64.4 | M | 20/32 | R / L paramedian, pontine |
| S10 | 56.2 | M | 20 | R / L corona radiata and corpus collosum |
| S11 | 69.3 | M | 8 | L / R bulbar |
| S12 | 69.8 | M | 25 | R |
| S13 | 67.1 | F | 23 | R / L ACA with extension to L paracentral gyrus and basal ganglia |
| S14 | 66.6/67.8 | M | 20/35 | L / R head of caudate nucleus, lenticular nucleus, internal capsule |
| S15 | 52.0/53.0 | F | 8/20 | R / L sylvian (MCA); posterior aspect L frontal lobe |
| S16 | 58.8/60.9 | M | 6/31 | R / L occipital, internal capsule, cerebellum |
| S17 | 61.3/62.3 | M | 36/48 | R / L corona radiata, bilateral subcortical (ischemic) |
| C1 | 56.7 | F | | R |
| C2 | 67.0 | F | | R |
| C3 | 65.3 | F | | R |
| C4 | 69.2 | M | | L |
| C5 | 62.0 | M | | R |
| C6 | 47.2 | F | | L |
| C7 | 48.4 | F | | L |
| C8 | 70.3 | F | | R |
| C9 | 57.5 | F | | R |
| C10 | 68.5 | M | | R |
| C11 | 56.6 | F | | L |
| C12 | 48.4 | F | | R |

S – stroke subject; C – control subject; M – male; F – female; R – right; L – left; ACA – anterior cerebral artery; MCA – middle cerebral artery

Table 5: Individual clinical data of stroke subjects: Clinical scales for each stroke subject at Visit 1 (and Visit 2 if applicable)

| Subject | FMA (x/66) | | WMFT (x/30) | | CSS (x/16) | | RPSS (x/18): close/far | | BBT (% of less affected side) | | MAL Amount of Use (x/5) | | MAL Quality of Movement (x/5) | | MAL Self- Efficacy Scale (x/10) | |
|------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|------------------------------|-----------------|--|-----------------|----------------------------------|-----------------|--|-----------------|---|-----------------|
| | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd |
| S1 | 49 | 52 | 22 | 24 | 6 | 5 | 16;17 | 17;14 | 67.0 | nc | 4.7 | 3.2 | 4.3 | 3.0 | 8.8 | 8.4 |
| S2 | 49 | 53 | 21 | 27 | 7 | 5 | 17;15 | 18;17 | 83.2 | 79.8 | 3.8 | 4.1 | 3.1 | 4.1 | 6.5 | 6.5 |
| S3 | 53 | 62 | 20 | 24 | 7 | 9 | 18;16 | 17;16 | 62.1 | 90.0 | 4.9 | 5.0 | 4.4 | 4.8 | 9.7 | 10.0 |
| S4 | 54 | 46 | 28 | 16 | 4 | 4 | 17;17 | 18;17 | 83.3 | 69.8 | 4.9 | 4.8 | 4.0 | 4.1 | 8.2 | 9.9 |
| S5 | 55 | | 25 | | 4 | | 18;18 | | 75.8 | | 4.4 | | 4.3 | | nc | |
| S6 | 56 | | 26 | | 5 | | 16;17 | | 94.4 | | 4.6 | | 4.3 | | 9.3 | |
| S7 | 59 | | 28 | | 5 | | 18;17 | | 86.9 | | 4.9 | | 4.7 | | 8.5 | |
| S8 | 60 | 60 | 27 | 28 | 0 | 4 | 18;17 | 18;18 | 96.3 | 98.9 | 5.0 | 5.0 | 4.9 | 4.9 | 9.4 | 10.0 |
| S9 | 60 | 60 | 29 | 29 | 4 | 4 | 18;18 | 18;18 | 94.7 | 103.3 | 4.8 | 5.0 | 4.8 | 5.0 | 9.4 | 10.0 |
| S10 | 61 | | 30 | | 4 | | 18;18 | | 109.6 | | 5.0 | | 5.0 | | 9.9 | |
| S11 | 62 | | 30 | | 5 | | 18;18 | | 89.2 | | 4.8 | | 5.0 | | 7.6 | |
| S12 | 64 | | 28 | | 4 | | 17;17 | | 82.6 | | 4.8 | | 3.8 | | 6.3 | |
| S13 | 64 | | 28 | | 5 | | 18;18 | | 103.1 | | 4.9 | | 4.2 | | 8.0 | |
| S14 | 65 | 60 | 30 | 30 | 6 | 5 | 18;18 | 18;18 | 85.2 | 93.7 | 5.0 | 4.5 | 4.8 | 4.6 | 8.2 | 9.7 |
| S15 | 65 | 66 | 30 | 30 | 2 | 5 | 18;18 | 18;18 | 103.8 | 102.0 | 4.8 | 4.8 | 4.5 | 4.7 | 8.6 | 9.3 |
| S16 | 66 | 64 | 30 | 24 | 4 | 6 | 18;18 | 17;17 | 69.4 | 66.3 | 3.3 | 3.3 | 3.2 | 3.9 | 9.7 | 6.6 |
| S17 | 66 | 62 | 30 | 29 | 2 | 7 | 18;18 | 18;18 | 94.3 | 101.1 | 5.0 | 5.0 | 4.8 | 4.8 | 9.8 | 9.2 |

FMA – Fugl-Meyer Assessment; WMFT – Wolf Motor Function Test; CSS – Composite Spasticity Scale; RPSS – Reaching Performance Scale for Stroke; BBT – Box and Blocks Test; MAL – Motor Activity Log

Table 6: Individual clinical data of stroke subjects: Grip and shoulder strength for each stroke subject at Visit 1 (and Visit 2 if applicable)

| Subject | Grip strength (% of less affected side) | | DA mean strength (kg) | | DM mean strength (kg) | | DP mean strength (kg) | | DA (% of less affected side) | | DM (% of less affected side) | | DP (% of less affected side) | |
|---------|---|-----------------|-----------------------|-----------------|-----------------------|-----------------|-----------------------|-----------------|------------------------------|-----------------|------------------------------|-----------------|------------------------------|-----------------|
| | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd |
| S1 | 79.8 | 79.7 | 15.5 | 16.8 | 11.0 | 19.7 | 13.5 | 17.7 | 76.1 | 92.0 | 73.9 | 100.9 | 82.6 | 94.6 |
| S2 | 68.3 | 50.5 | 5.7 | 12.0 | 5.7 | 9.6 | 10.3 | 11.3 | 27.6 | 78.4 | 36.2 | 55.7 | 68.6 | 84.3 |
| S3 | 97.0 | 100.0 | 20.0 | 21.8 | 17.0 | 20.0 | 11.1 | 21.1 | 95.2 | 94.1 | 85.8 | 96.1 | 66.6 | 99.1 |
| S4 | 90.4 | 63 | 4.6 | 3.7 | 4.2 | 3.5 | 3.7 | 4.2 | 33.7 | 33.0 | 38.6 | 31.0 | 34.1 | 53.9 |
| S5 | 100.0 | | 12.8 | | 11.8 | | 12.3 | | 93.4 | | 75.2 | | 84.2 | |
| S6 | nc | | 11.1 | | 10.4 | | 10.9 | | 64.1 | | 85.5 | | 79.4 | |
| S7 | 84.8 | | 16.3 | | 15.1 | | 14.3 | | 83.0 | | 91.9 | | 80.0 | |
| S8 | 88.0 | 91.7 | 17.3 | 14.3 | 16.8 | 13.1 | 15.2 | 13.7 | 98.9 | 100.0 | 98.6 | 99.2 | 89.4 | 106.2 |
| S9 | 93.0 | 91.3 | 16.0 | 17.0 | 16.6 | 11.9 | 12.4 | 10.2 | 93.6 | 166.3 | 113.0 | 96.7 | 86.7 | 101.0 |
| S10 | 92.3 | | 14.9 | | 12.3 | | 12.7 | | 63.5 | | 91.3 | | 92.7 | |
| S11 | 85.8 | | 16.5 | | 17.5 | | 16.2 | | 90.8 | | 97.5 | | 83.9 | |
| S12 | 71.4 | | 9.2 | | 8.1 | | 9.1 | | 82.4 | | 86.5 | | 82.4 | |
| S13 | 100.0 | | 10.2 | | 9.0 | | 9.0 | | 100.7 | | 91.0 | | 89.4 | |
| S14 | 83.7 | 98.2 | 14.6 | 17.0 | 15.1 | 13.8 | 13.8 | 12.9 | 90.7 | 101.0 | 100.7 | 94.3 | 81.8 | 77.4 |
| S15 | 52.2 | 92.6 | 10.4 | 13.2 | 9.4 | 12.0 | 7.5 | 10.2 | 67.9 | 96.4 | 78.8 | 89.5 | 61.6 | 84.8 |
| S16 | 93.9 | 86.6 | 23.3 | 11.1 | 27.4 | 13.1 | 24.1 | 10.6 | 59.6 | 72.9 | 69.9 | 87.3 | 58.3 | 81.3 |
| S17 | 82.4 | 100.0 | 17.6 | 17.0 | 17.3 | 19.6 | 11.9 | 18.1 | 85.2 | 98.1 | 100.6 | 106.7 | 78.7 | 102.4 |

DA – anterior deltoid; DM – medial deltoid; DP – posterior deltoid

Table 7: Individual clinical data of stroke subjects: Shoulder range of motion (% of affected/less affected side) for each stroke subject at Visit 1 (and Visit 2 if applicable)

| Subject | Flexion | | Extension | | Abduction | | Adduction | | Internal Rotation | | External Rotation | |
|------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-------------------|-----------------|-------------------|-----------------|
| | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd | 1 st | 2 nd |
| S1 | 94.1 | 103.2 | 80.0 | 122.2 | 93.8 | 101.4 | 88.9 | 105.0 | 71.4 | 125.0 | 81.8 | 105.3 |
| S2 | 88.9 | 83.3 | 100.0 | 71.4 | 8.3 | 97.0 | 100.0 | 100.0 | 80.0 | 100.0 | 90.0 | 80.0 |
| S3 | 100.0 | 107.7 | 100.0 | 152.0 | 100.0 | 104.8 | 100.0 | 137.9 | 100.0 | 128.9 | 100.0 | 84.2 |
| S4 | 77.8 | 79.4 | 100.0 | 91.7 | 88.9 | 41.2 | 100.0 | 66.7 | 14.3 | | 87.0 | 82.6 |
| S5 | 85.2 | | 100.0 | | 97.1 | | 100.0 | | 50.0 | | 88.9 | |
| S6 | 83.9 | | 83.3 | | 93.9 | | 60.0 | | 83.3 | | 90.0 | |
| S7 | 100.0 | | 100.0 | | 100.0 | | 100.0 | | 100.0 | | 100.0 | |
| S8 | 95.1 | 100.0 | 79.2 | 100.0 | 96.6 | 102.5 | 86.5 | 115.6 | 60.5 | 95.5 | 100.0 | 92.9 |
| S9 | 90.3 | 91.9 | 66.7 | 97.6 | 97.1 | 95.3 | 100.0 | 117.4 | 66.7 | 42.9 | 87.0 | 97.4 |
| S10 | 115.9 | | 106.9 | | 118.6 | | 120.0 | | 80.0 | | 139.7 | |
| S11 | 69.9 | | 62.5 | | 56.3 | | 66.7 | | 100.0 | | 60.0 | |
| S12 | 81.1 | | 80.0 | | 111.9 | | 106.7 | | 27.3 | | 67.1 | |
| S13 | 94.1 | | 75.0 | | 88.2 | | 100.0 | | 100.0 | | 115.0 | |
| S14 | 90.3 | 104.5 | 70.0 | 126.7 | 88.2 | 105.0 | 120.0 | 136.4 | 33.3 | 100.0 | 72.0 | 128.9 |
| S15 | 92.0 | 106.3 | 74.1 | 91.8 | 78.8 | 94.3 | 46.5 | 94.7 | 44.4 | 56.9 | 73.1 | 83.7 |
| S16 | 82.9 | 100.0 | 81.3 | 60.0 | 62.9 | 98.8 | 68.8 | 80.0 | nc | 66.7 | nc | 90.2 |
| S17 | 100.0 | 94.3 | 63.3 | 90.5 | 97.1 | 100.0 | 75.0 | 133.3 | 69.2 | 76.9 | 100.0 | 102.2 |

Table 8: Group means of shoulder strength, shoulder range of motion, and Box and Blocks Test score at Visit 1 and Visit 2

| | Control (% of non-dominant / dominant side) | Stroke (Visit 1) (% of affected / less affected side) |
|---|--|--|
| Grip strength, kg ($\bar{x} \pm SD$) | 91.1±10.2 | 84.5±21.6 |
| Deltoid strength, kg ($\bar{x} \pm SD$): | | |
| Anterior | 96.9±16.0 | 78.6±21.7 ^b |
| Medial | 96.5±8.3 | 83.4±19.9 ^b |
| Posterior | 104.8±19.6 | 76.1±14.3 ^a |
| Shoulder range of motion, degrees ($\bar{x} \pm SD$): | | |
| Flexion | 97.5±7.4 | 91.2±10.4 |
| Extension | 110.3±23.2 | 84.6±14.7 ^a |
| Horizontal abduction | 96.9±6.4 | 87.6±24.7 |
| Horizontal adduction | 110.9±29.8 | 89.0±21.1 ^b |
| Internal rotation | 108.5±40.1 | 66.2±27.2 ^a |
| External rotation | 102.5±37.2 | 91.3±18.9 |
| Box and Blocks Test ($\bar{x} \pm SD$) | 100.0±8.4 | 87.1±13.3 ^a |

compared to controls ^a p < 0.01, ^b p < 0.05

4.2 PERFORMANCE OF CONTROL AND STROKE GROUPS IN OBSTACLE AVOIDANCE TASK

4.2.1 TEMPLATE REACHING

In the template reach block, endpoint trajectories of both groups were slightly curved (index of curvature > 1). Endpoint peak velocity, time to peak velocity, trajectory length, as well as shoulder, elbow, and trunk displacement were similar between groups ([Table 9](#)). However, stroke subjects used $\sim 10^\circ$ less shoulder external rotation ($t_{27}=2.1$, $p<0.05$), $\sim 10^\circ$ less wrist flexion ($t_{27}=2.7$, $p<0.05$), and abducted instead of adducted their wrists ($t_{27}=-3.2$, $p<0.01$) compared to control subjects near the time of object contact ([Table 10](#)).

[Table 9](#): Endpoint variables (mean \pm SD) of reaching performance by control and stroke subjects (Visit 1) for template, success and fail blocks

| | Control | | | Stroke (Visit 1) | | |
|------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| | Template | Success | Fail | Template | Success | Fail |
| Endpoint peak velocity (mm/s) | 2241 \pm 766 | 1162 \pm 317 | 1466 \pm 431 | 1979 \pm 595 | 1299 \pm 517 | 1547 \pm 479 |
| Endpoint time to peak velocity (s) | 0.257 \pm 0.068 | 0.422 \pm 0.144 | 0.322 \pm 0.078 | 0.308 \pm 0.102 | 0.534 \pm 0.236 | 0.377 \pm 0.171 |
| Index of curvature | 1.09 \pm 0.06 | 1.18 \pm 0.08 | 1.14 \pm 0.07 | 1.07 \pm 0.03 | 1.17 \pm 0.18 | 1.18 \pm 0.23 |
| Endpoint trajectory length (mm) | 687 \pm 100 | 813 \pm 119 | 727 \pm 108 | 705 \pm 84 | 821 \pm 180 | 802 \pm 243 |

[Table 10](#): Joint angles (degrees; mean \pm SD) during reaching by control and stroke subjects (Visit 1) for template, success and fail blocks

| | Control | | | Stroke (Visit 1) | | |
|---------------------------------|------------------|-------------------------------|---------------------------------|------------------|-------------------------------|-----------------------------|
| | Template | Success | Fail | Template | Success | Fail |
| Wrist flexion | 14.1 \pm 10.1 | 8.4 \pm 12.5 | 9.2 \pm 9.5 | 4.2 \pm 9.4* | 1.6 \pm 10.5 | 6.5 \pm 7.8 |
| Wrist abduction | -7.5 \pm 11.7 | -1.0 \pm 18.2 | -1.1 \pm 13.5 | 6.9 \pm 12.0** | 10.2 \pm 15.1 | 5.9 \pm 9.9 |
| Wrist rotation | 21.3 \pm 25.0 | 13.1 \pm 18.5 | 14.4 \pm 21.5 | 11.5 \pm 15.4 | 18.8 \pm 16.0 | 17.0 \pm 14.8 |
| Elbow extension (full ext=180°) | 129.4 \pm 16.4 | 108.6 \pm 20.0 ^a | 114.2 \pm 18.5 ^{a,c} | 124.5 \pm 13.0 | 122.0 \pm 12.4* | 121.7 \pm 13.4 |
| Elbow rotation | 18.4 \pm 9.2 | 14.8 \pm 10.6 | 16.6 \pm 12.1 | 10.9 \pm 12.5 | 13.5 \pm 13.7 | 11.6 \pm 15.9 |
| Shoulder horizontal adduction | 35.0 \pm 20.6 | 41.1 \pm 31.2 | 30.5 \pm 27.2 | 27.0 \pm 23.7 | 52.2 \pm 36.7 | 31.0 \pm 31.7 |
| Shoulder flexion | 28.6 \pm 15.6 | 14.1 \pm 15.0 | 17.1 \pm 18.1 | 21.0 \pm 16.8 | 15.9 \pm 14.0 | 16.1 \pm 15.8 |
| Shoulder rotation (+ ER) | 14.1 \pm 15.0 | 3.5 \pm 11.7 | 8.4 \pm 13.7 | 4.4 \pm 10.1* | -6.7 \pm 11.7 | 0.08 \pm 10.8 |
| Trunk pitch | 8.5 \pm 3.9 | 5.0 \pm 3.7 ^a | 5.2 \pm 3.0 ^a | 7.6 \pm 3.3 | 8.8 \pm 5.2* | 7.4 \pm 4.0 |
| Trunk rotation | 6.0 \pm 6.9 | 3.9 \pm 3.5 | 2.7 \pm 2.5 | 6.2 \pm 4.4 | 10.2 \pm 5.3 ^{b**} | 6.5 \pm 4.6 ^{c*} |

compared to template^a $p<0.01$, ^b $p<0.05$; compared to success^c $p<0.01$

compared to controls^{**} $p<0.01$, ^{*} $p<0.05$; red stars * indicate t-test for template condition planned comparison only

4.2.2 OBSTACLE AVOIDANCE TASK VS. TEMPLATE

The mean success rate did not differ between groups likely due to the large variance (control = $55.9\% \pm 24.0\%$, stroke = $43.5\% \pm 23.2\%$). Therefore, we compared the number of individuals between groups who achieved successively increasing levels of success (50%, 60%, 70%, etc.). At the success level of 65%, the proportion of subjects who achieved success was significantly lower for the stroke group (12%) compared to controls (42%; $z=1.85$, $p<0.05$; [Table 11](#)).

[Table 11](#): Success rates for each control and stroke subject in the randomized block of the obstacle avoidance task (Visit 2 reported if applicable)

| Control group | Success rate (%) | Stroke group | Success rate (%): Visit 1 | Success rate (%): Visit 2 |
|---------------|------------------|--------------|---------------------------|---------------------------|
| C1 | 58.8 | S1 | 88.9 | 94.7 |
| C2 | 88.9 | S2 | 35.0 | 100.0 |
| C3 | 80.0 | S3 | 50.0 | 50.0 |
| C4 | 36.8 | S4 | 20.0 | 85.0 |
| C5 | 21.4 | S5 | 10.5 | |
| C6 | 73.7 | S6 | 47.1 | |
| C7 | 90.0 | S7 | 50.0 | |
| C8 | 43.3 | S8 | 48.4 | 45.0 |
| C9 | 70.0 | S9 | 60.0 | 70.0 |
| C10 | 36.8 | S10 | 57.9 | |
| C11 | 30.0 | S11 | 52.6 | |
| C12 | 40.7 | S12 | 10.5 | |
| | | S13 | 15.0 | |
| | | S14 | 55.0 | 60.9 |
| | | S15 | 7.1 | 35.0 |
| | | S16 | 90.0 | 60.0 |
| | | S17 | 8.7 | 20.0 |

The hand trajectory initially followed the template path in trials where the door on the affected side closed. As the door closed, the trajectory deviated more medially than the template reach ([Figure 6](#)). For both groups, the trajectory lengthened by $\sim 125\text{mm}$ ($F_{2,26}=10.9$, $p<0.01$) and the index of curvature was greater ($F_{2,26}=9.6$, $p<0.05$) for successful compared to template reaches but not for failed reaches ([Table 12](#)). Subjects in both groups decreased their endpoint peak velocity by $\sim 40\%$ for successful and by $\sim 28\%$ for unsuccessful compared to template reaches with no difference between groups ($F_{2,26}=35.8$, $p<0.01$).

Figure 6: Examples of endpoint and trunk trajectories. The black traces represent template reach trials, the left column green traces represent successful reach trials, and the right column red traces represent failed reach trials. The first row contains examples of successful (A) and failed (B) trials for one control subject. The second row contains examples of successful (C) and failed (D) for one stroke subject. S5 had an FMA=57.

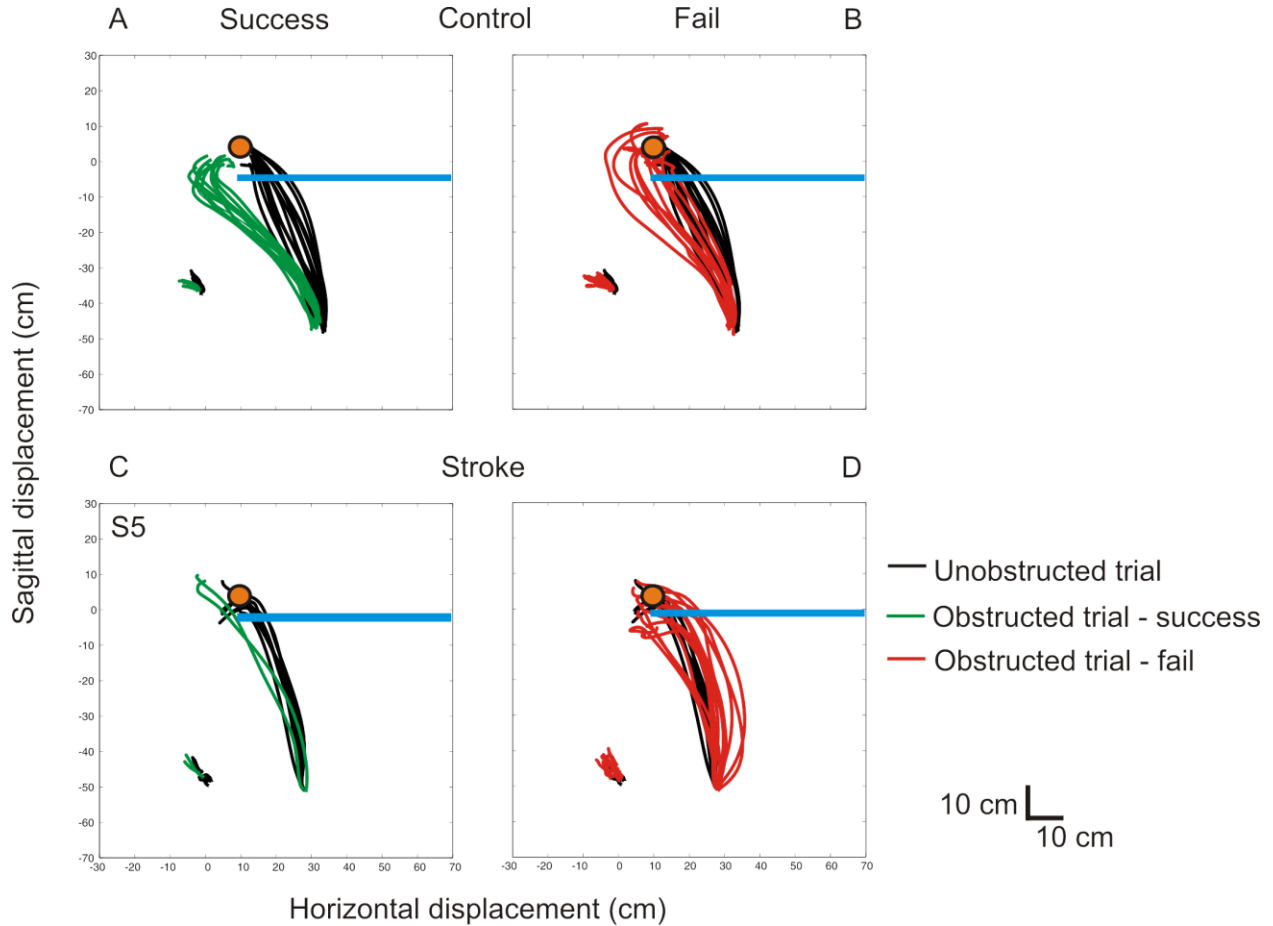


Table 12: Endpoint variables (mean \pm SD) of reaching performance by control and stroke subjects (Visit 1; mean across groups) for template, success and fail blocks

| | Template | Success | Fail |
|------------------------------------|-------------------|--------------------------------|----------------------------------|
| Endpoint peak velocity (mm/s) | 2087 \pm 671 | 1242 \pm 444 ^a | 1513 \pm 454 ^{a,c} |
| Endpoint time to peak velocity (s) | 0.287 \pm 0.092 | 0.488 \pm 0.208 ^a | 0.354 \pm 0.141 ^{b,c} |
| Index of curvature | 1.08 \pm 0.04 | 1.17 \pm 0.15 ^a | 1.16 \pm 0.18 |
| Endpoint trajectory length (mm) | 698 \pm 90 | 818 \pm 155 ^a | 771 \pm 199 ^d |

compared to template^a $p < 0.01$, ^b $p < 0.05$; compared to success^c $p < 0.01$, ^d $p < 0.05$

For the shoulder, both groups increased horizontal adduction by 6-25° ($F_{2,26}=10.2$, $p<0.01$), decreased flexion by 5-15° ($F_{2,26}=8.1$, $p<0.01$), and increased internal rotation by ~11° ($F_{2,26}=25.7$, $p<0.01$) in the successful reaches compared to template reaches ([Table 13](#)). In the failed condition, the shoulder angles changed in the same direction (increased horizontal adduction, decreased flexion, increased internal rotation). However, there was no difference between the successful and failed reaches for shoulder flexion, so the decrease in shoulder flexion was not a condition of success. In contrast, greater shoulder internal rotation (Success: $-2.5^\circ \pm 12.6^\circ$, Fail: $3.5^\circ \pm 12.5^\circ$, $F_{2,26}=25.7$, $p<0.01$) and horizontal adduction (Success: $47.6^\circ \pm 34.4^\circ$, Fail: $30.8^\circ \pm 29.4^\circ$, $F_{2,26}=10.2$, $p<0.01$) were used in successful compared to non-successful trials. Furthermore, the stroke group actually used more internal rotation than the control group across all conditions (Control= $8.7^\circ \pm 13.9^\circ$, Stroke= $-0.7^\circ \pm 11.6^\circ$, $F_{1,27}=5.188$, $p<0.05$; [Table 14](#)).

[Table 13](#): Joint angles (degrees; mean \pm SD) during reaching by control and stroke subjects (Visit 1; mean across groups) for template, success and fail blocks

| | Template | Success | Fail |
|--------------------------------------|-----------------|------------------------------|-------------------------------|
| Shoulder horizontal adduction | 30.3 \pm 22.4 | 47.6 \pm 34.4 ^a | 30.8 \pm 29.4 ^c |
| Shoulder flexion | 24.1 \pm 16.4 | 15.2 \pm 14.2 ^a | 16.5 \pm 16.5 ^a |
| Shoulder rotation | 8.4 \pm 13.1 | -2.5 \pm 12.6 ^a | 3.5 \pm 12.5 ^{b,c} |

compared to template^a $p<0.01$, ^b $p<0.05$; compared to success^c $p<0.01$

[Table 14](#): Joint angles (degrees; mean \pm SD) during reaching by control and stroke subjects (Visit 1; mean across conditions)

| | Control | Stroke (Visit 1) |
|--------------------------|-----------------|------------------------------|
| Wrist flexion | 10.5 \pm 10.8 | 4.1 \pm 9.3 [*] |
| Wrist abduction | -3.2 \pm 14.6 | 7.6 \pm 12.4 [*] |
| Shoulder rotation | 8.7 \pm 13.9 | -0.7 \pm 11.6 [*] |

compared to controls^{*} $p<0.05$; however, t-test suggests this significance is driven mainly by the change in template condition

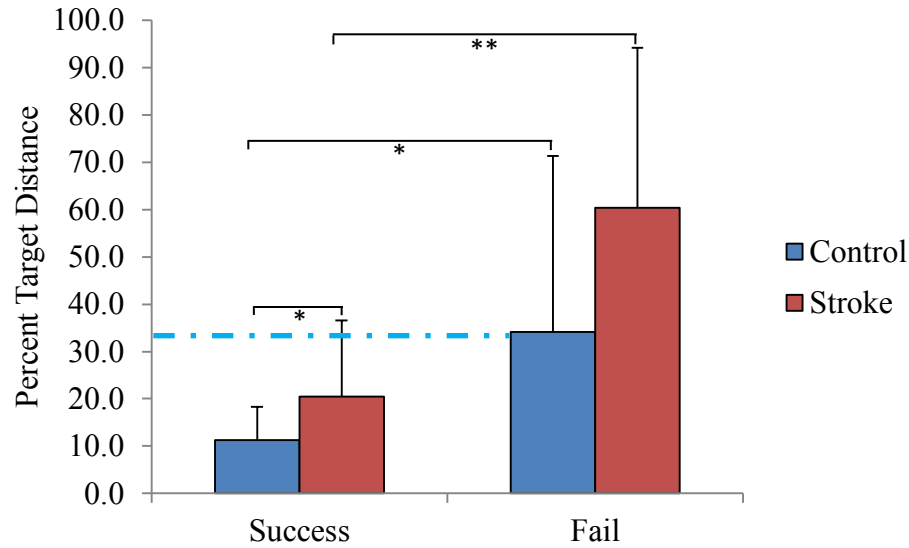
Differences between the control and stroke groups were also found in the wrist, elbow, and trunk angles near the time of object contact. There was a group difference where the stroke group used less wrist flexion (Control= $10.5^\circ \pm 10.8^\circ$, Stroke= $4.1^\circ \pm 9.3^\circ$, $F_{1,27}=4.8$, $p<0.05$) and more wrist abduction (Control= $-3.2^\circ \pm 14.6^\circ$, Stroke= $7.6^\circ \pm 12.4^\circ$, $F_{1,27}=7.0$, $p<0.05$) across all conditions ([Table 14](#)). There were significant interaction effects for elbow extension ($F_{2,26}=9.2$, $p<0.01$), trunk pitch ($F_{2,26}=8.0$, $p<0.01$), and trunk rotation ($F_{2,26}=4.9$, $p<0.01$;

[Table 10](#)). Controls used more elbow flexion to avoid hitting the door, while stroke subjects did not vary elbow angle across all conditions. Control subjects did not use as much trunk flexion in all obstructed trials compared to their template trials, while stroke subjects were more flexed than the controls in the successful trials. Stroke subjects used more trunk rotation to the side contralateral to the affected arm than controls in all obstructed trials. Additionally in the stroke group, trunk rotation was most pronounced in the successful trials.

4.2.3 DIVERGENCE POINT

[Figure 5](#) illustrates divergence points for one control and one stroke subject. The blue curves in all diagrams are phase plots representing all template reaches, whereas the red and green curves are phase plots representing successful (5A, 5C) and failed (5B, 5D) obstructed reaches. The successful divergence point of the control subject (top row) occurred closer to the start position of the hand, ~100mm, than the failed divergence point, ~300mm. The successful divergence point of the first stroke subject example occurred at ~150mm. The stroke subject did not have a significant failed divergence point when they failed to avoid the door, indicating that their endpoint phase plot was not different from their template phase plot. Overall, for all examples of stroke subjects, successful door avoidance was characterized by the divergence point occurring closer to the starting position of the hand compared to failed trials. The distance measured was equivalent to a latency measure. For both groups, the successful divergence point occurred at $18.2\% \pm 17.7\%$ SD from the starting position while the failed divergence point occurred at $45.7\% \pm 34.6\%$ from the starting position. ($F_{2,26}=24.799$, $p<0.01$). When considering the groups separately, the mean successful divergence point for the control group occurred at $11\% \pm 6\%$ SD from the starting position and the divergence point for failed trials occurred at $33\% \pm 38\%$ from the starting position. Thus the difference between the divergence points of the success and failed trials of the controls was ~22% of the total hand path ([Figure 7](#)). The mean failed divergence point of the control subjects was considered to be the mean critical failure point for both groups (the blue horizontal dashed line in [Figure 7](#)). In contrast, the success divergence point of the stroke group occurred further from the starting position, $23\% \pm 21\%$. The difference between the success divergence point of the stroke subjects and the mean critical failure point was only ~10%.

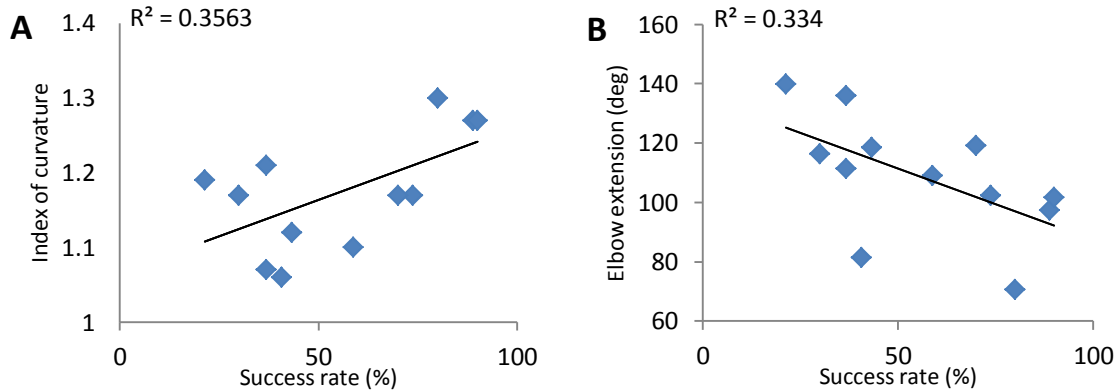
Figure 7: Mean divergence point of the phase plots for each group. The 2 left bars represent the mean divergence point of the successful trials for control (blue bar) and stroke (red bar) groups. The 2 right bars represent the divergence point of the failed trials for each group. Asterisks indicate significance (*: $p < 0.05$, **: $p < 0.01$). The blue dashed horizontal line indicates the critical threshold at which the control subjects failed the task.



4.3 RELATIONSHIPS OF SUCCESS RATE AND SUCCESSFUL DIVERGENCE POINT WITH CLINICAL SCORES AND KINEMATIC MEASURES

Success rates were correlated with the kinematic variables of successful trials as well as the clinical scores for all subjects. Success rate for the control group was related to the index of curvature and the amount of elbow extension used in the task. Increases in the index of curvature ($r=0.60$, $p < 0.05$) and decreases in elbow extension near the position of door contact ($r=-0.58$, $p < 0.05$) were related to higher success rates (Figure 8).

Figure 8: Correlations of success rate with index of curvature (A) and elbow extension (B) were performed for the control group. Higher task success rates were correlated with increased trajectory curvature ($r=0.60$, $p<0.05$) and decreased elbow extension ($r=-0.58$, $p<0.05$) near the position of door contact. Full elbow extension was defined as 180° .



Success rate for the stroke group was related to the endpoint peak velocity, endpoint time to peak velocity, and shoulder rotation position near the point of door contact ([Figure 9](#)). For the endpoint performance variables, lower endpoint peak velocities and slower time to reach endpoint peak velocity were related to higher success rates ($r=-0.50$, $p<0.05$; $r=0.48$, $p<0.05$ respectively). For the movement quality variables, greater use of shoulder external rotation ($r=0.65$, $p<0.01$) near the point of door contact was related to higher success rates. No relationships were found between success rate and scores on clinical assessments.

When successful divergence points were correlated with scores on clinical assessments, earlier successful divergence points were related to higher Box and Blocks Test scores ($r=-0.53$, $p<0.05$; [Figure 10](#)). No other relationships were found between successful divergence points and endpoint performance/ movement quality variables or scores on clinical assessments.

A 10-point confidence rating scale for each of the 30 questions from the MAL was administered at the clinical evaluation. The total score of the MAL self-efficacy scale (MAL-SES_T) was highly correlated with the total scores of the MAL-AOU_T and MAL-QOM_T ($r=0.85$, $p<0.01$, $r=0.89$, $p<0.01$ respectively, [Figure 11](#)). Higher MAL-SES_T were related to higher scores on the CMSA arm subscale ($r=0.48$, $p<0.05$), higher anterior deltoid strength ($r=0.44$, $p<0.05$), and higher medial deltoid strength ($r=0.50$, $p<0.05$; [Figure 12](#)).

Figure 9: Correlations of success rate with endpoint peak velocity (EPV; A), time to peak velocity (ETPV; B), and shoulder rotation (C) for the stroke group at first visit were performed. Higher task success rates were correlated with decreased endpoint peak velocity ($r=-0.50$, $p<0.05$) and increased time to peak velocity ($r=0.48$, $p<0.05$). Greater use of shoulder external rotation ($r=0.65$, $p<0.01$) near the point of door contact was related to higher success rates.

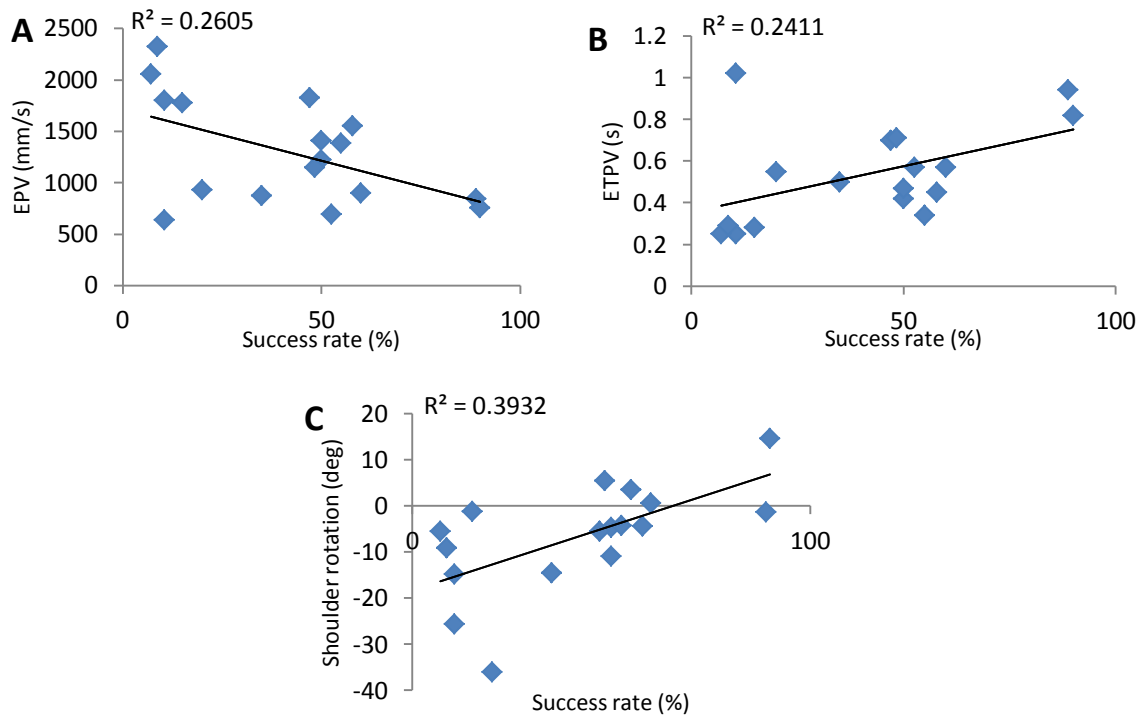


Figure 10: A correlation of successful divergence point with Box and Blocks Test for the stroke group at first visit was performed. Earlier successful divergence points (i.e. larger margin of error) were related to higher Box and Blocks Test scores ($r=-0.53$, $p<0.05$).

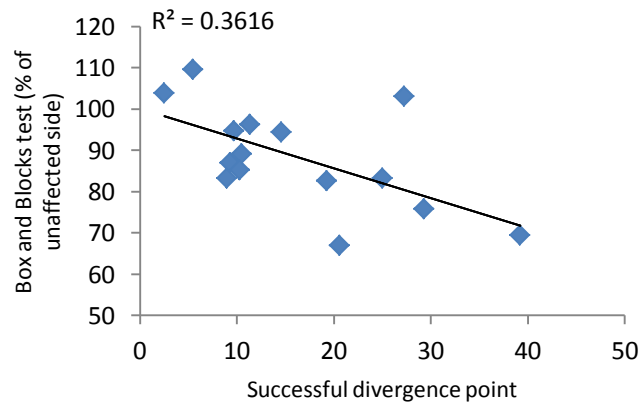


Figure 11: Correlations of MAL-SES_T with MAL-AOU_T (A) and MAL-QOL_T (B) for the stroke group at first visit were performed. Higher AOU and QOM scores were related to higher confidence levels in completing the MAL tasks ($r=0.85$, $p<0.01$, $r=0.89$, $p<0.01$ respectively).

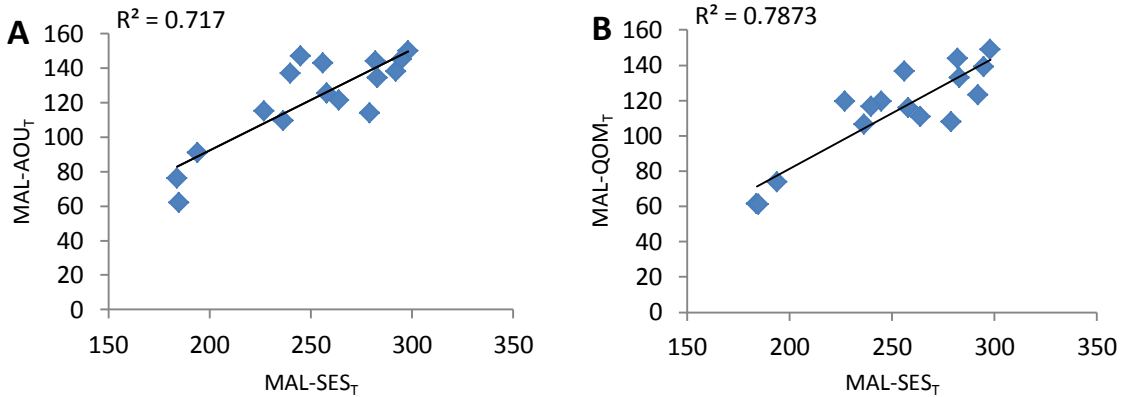
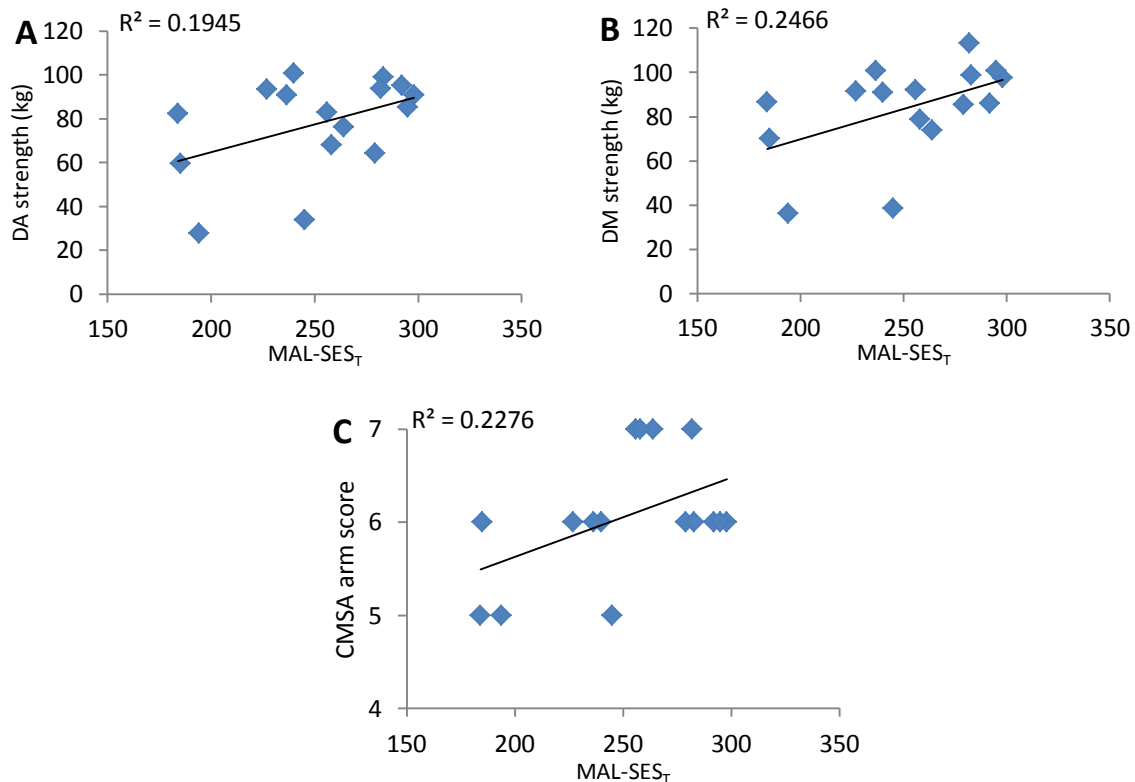


Figure 12: Correlations of MAL-SES_T with anterior deltoid strength (DA; A), medial deltoid strength (DM; B), and CMSA arm score (C) for the stroke group at first visit were performed. Higher MAL-SES_T were related to higher scores on the CMSA arm subscale ($r=0.48$, $p<0.05$), higher anterior deltoid strength ($r=0.44$, $p<0.05$), and higher medial deltoid strength ($r=0.50$, $p<0.05$).



4.4 PERFORMANCE OF THE STROKE GROUP COMPARING 1ST AND 2ND VISITS

Previous literature demonstrated that there was decreased daily arm use in well-recovered stroke patients (Rand & Eng, 2012). It was hypothesized that after one year (Visit 2), task success rates would decrease compared to the first visit (Visit 1), the stroke group would have a decreased adaptability of arm and trunk movements, and lower task success rates would be related to lower clinical measures of arm impairment, activity and participation.

4.4.1 GROUP DESCRIPTION

Subjects from the stroke group were contacted 1 year after their initial visit and, if agreeable, repeated the clinical and experimental sessions. Of the 17 original participants (Visit 1), 10 returned for a second visit (Visit 2). Clinical testing revealed that CMSA, FMA, MAL, WMFT, RPS, CSI and grip strength were similar to the initial visit ([Table 5](#); [Table 6](#)). However, at the second visit there were increases in anterior deltoid strength, posterior deltoid strength, shoulder flexion, shoulder adduction, shoulder internal rotation (individual means: [Table 6](#) and [Table 7](#); Visit means: [Table 15](#)).

[Table 15](#): Stroke group (n=10): Means of shoulder strength and shoulder range of motion at Visit 1 and Visit 2

| | Visit 1 | Visit 2 |
|---|-----------|------------|
| Anterior deltoid strength (kg) | 72.9±25.5 | 93.2±32.9 |
| Posterior deltoid strength (kg) | 70.8±16.8 | 88.5±15.7 |
| Shoulder flexion (degrees) | 91.1±6.9 | 97.1±9.7 |
| Shoulder adduction (degrees) | 88.6±20.7 | 108.7±24.1 |
| Shoulder internal rotation (degrees) | 65.7±20.6 | 90.8±30.4 |

4.4.2 TEMPLATE REACHING

When comparing the template reach block of each visit, endpoint trajectories were slightly curved (index of curvature > 1). Endpoint peak velocity, time to peak velocity, trajectory length, as well as wrist rotation, elbow rotation, shoulder rotation, trunk rotation, wrist flexion, wrist abduction, elbow extension, shoulder horizontal adduction, shoulder flexion, and trunk pitch

were similar between Visits 1 and 2 (paired t-tests not significant; [Table 16](#), [Table 17](#), [Table 18](#), [Table 19](#)).

Table 16: Endpoint variables (mean \pm SD) of reaching performance by stroke subjects in Visit 1 and Visit 2 for template, success and fail blocks

| | Stroke (Visit 1) | | | Stroke (Visit 2) | | |
|------------------------------------|-------------------|-------------------|-------------------|-------------------|-------------------|-------------------|
| | Template | Success | Fail | Template | Success | Fail |
| Endpoint peak velocity (mm/s) | 1924.7 \pm 632 | 1302.5 \pm 555 | 1563.0 \pm 473 | 2232.7 \pm 695 | 1555.9 \pm 762 | 1776.5 \pm 730 |
| Endpoint time to peak velocity (s) | 0.323 \pm 0.117 | 0.542 \pm 0.243 | 0.346 \pm 0.091 | 0.274 \pm 0.063 | 0.457 \pm 0.201 | 0.357 \pm 0.112 |
| Index of curvature | 1.07 \pm 0.03 | 1.15 \pm 0.07 | 1.12 \pm 0.040 | 1.07 \pm 0.03 | 1.14 \pm 0.06 | 1.12 \pm 0.036 |
| Endpoint trajectory length (mm) | 718 \pm 98 | 814 \pm 123 | 774 \pm 81 | 674 \pm 57 | 767 \pm 90 | 740 \pm 60 |

Table 17: Endpoint variables (mean \pm SD) of reaching performance by stroke subjects in Visit 1 and Visit 2 (mean across groups) for template, success and fail blocks

| | Template | Success | Fail |
|------------------------------------|-------------------|--------------------------------|---------------------------------|
| Endpoint peak velocity (mm/s) | 2078.7 \pm 663 | 1429.2 \pm 659 ^a | 1669.7 \pm 607 ^{a,c} |
| Endpoint time to peak velocity (s) | 0.298 \pm 0.095 | 0.500 \pm 0.220 ^a | 0.352 \pm 0.099 ^c |
| Index of curvature | 1.07 \pm 0.03 | 1.14 \pm 0.06 ^a | 1.12 \pm 0.04 ^a |
| Endpoint trajectory length (mm) | 696 \pm 81 | 791 \pm 107 ^a | 757 \pm 71 ^a |

compared to template^a p<0.01; compared to success^c p<0.01

Table 18: Joint angles (degrees; mean \pm SD) during reaching by stroke subjects in Visit 1 and Visit 2 for template, success and fail blocks

| | Stroke (Visit 1) | | | Stroke (Visit 2) | | |
|-------------------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| | Template | Success | Fail | Template | Success | Fail |
| Wrist flexion | 4.0 \pm 11.3 | 5.0 \pm 10.2 | 9.8 \pm 6.0 | 8.2 \pm 6.4 | 5.0 \pm 14.6 | 7.6 \pm 8.3 |
| Wrist abduction | 0.8 \pm 8.6 | 6.8 \pm 14.0 | 2.9 \pm 9.2 | -1.4 \pm 9.4 | 5.0 \pm 10.4 | -2.9 \pm 10.5 |
| Wrist rotation | 4.1 \pm 15.0 | 16.4 \pm 19.3 | 11.0 \pm 12.3 | 13.0 \pm 16.7 | 15.6 \pm 17.0 | 18.1 \pm 22.5 |
| Elbow extension | 124.7 \pm 16.3 | 125.2 \pm 15.3 | 124.5 \pm 16.6 | 129.6 \pm 15.4 | 123.7 \pm 15.5 | 126.3 \pm 12.8 |
| Elbow rotation | 6.3 \pm 15.1 | 13.1 \pm 18.3 | 7.7 \pm 20.2 | 14.9 \pm 12.0 | 15.7 \pm 14.6 | 13.9 \pm 12.0 |
| Shoulder horizontal adduction | 28.1 \pm 23.5 | 62.1 \pm 43.4 | 38.4 \pm 36.3 | 46.8 \pm 40.4 | 69.7 \pm 38.1 | 50.5 \pm 40.8 |
| Shoulder flexion | 22.8 \pm 19.5 | 15.8 \pm 16.8 | 16.4 \pm 18.8 | 25.3 \pm 17.5 | 17.6 \pm 10.8 | 22.6 \pm 19.3 |
| Shoulder rotation | 7.4 \pm 11.1 | -3.6 \pm 14.0 | 3.7 \pm 12.9 | 0.78 \pm 15.3 | -7.8 \pm 18.2 | -1.5 \pm 16.5 |
| Trunk pitch | 7.7 \pm 3.2 | 8.8 \pm 4.7 | 7.6 \pm 4.1 | 4.4 \pm 5.5 | 5.3 \pm 7.0 | 4.6 \pm 5.3 |
| Trunk rotation | 5.5 \pm 5.0 | 9.3 \pm 5.7 | 5.5 \pm 5.5 | 3.3 \pm 3.0 | 5.8 \pm 4.5 | 4.1 \pm 3.4 |

Table 19: Joint angles (degrees; mean \pm SD) during reaching by stroke subjects in Visit 1 and Visit 2 (mean across groups) for template, success and fail blocks

| | Template | Success | Fail |
|--------------------------------------|-----------------|------------------------------|------------------------------|
| Shoulder flexion | 24.0 \pm 18.0 | 16.7 \pm 13.8 ^b | 19.5 \pm 18.7 |
| Shoulder horizontal adduction | 37.5 \pm 33.5 | 65.9 \pm 39.8 ^a | 44.5 \pm 38.0 ^c |
| Shoulder rotation | 4.1 \pm 13.4 | -5.7 \pm 15.9 ^a | 1.1 \pm 14.6 ^c |
| Trunk rotation | 4.4 \pm 4.2 | 7.6 \pm 5.3 ^b | 4.8 \pm 4.5 ^c |

compared to template^a $p < 0.01$, ^b $p < 0.05$; compared to success^c $p < 0.01$, ^d $p < 0.05$

4.4.3 OBSTACLE AVOIDANCE TASK VS. TEMPLATE

The success rate at Visit 1 was 46.3 \pm 29.4%, where 2/10 individuals attained an individual success rate greater than 65%. The success rate at Visit 2 was 62.1 \pm 25.9%, where and the number individuals with success rates greater than 65% increased to 4/10 ([Table 11](#)). Success rate was not significantly different between the visits. However, there were three subjects that had a large improvement in their success rates and one subject with a worse performance in Visit 2.

The strategies in the obstacle avoidance task for Visit 2 were similar to Visit 1. The hand trajectory of both groups initially followed the template path, but as the door closed, the trajectories deviated medially away from the door. Endpoint trajectory lengthened by \sim 95mm ($F_{2,15}=12.5$, $p < 0.01$) and the index of curvature was greater ($F_{2,15}=15.6$, $p < 0.01$) for successful compared to template reaches. The trajectory lengths and curvatures of the failed reaches were also increased, but not as high as in the success trials. Subjects in both visits decreased endpoint peak velocity by \sim 31% for successful and by 20% for unsuccessful compared to template reaches ($F_{2,15}=18.4$, $p < 0.01$) with no difference between the visits. Similarly, subjects in both visits increased time to endpoint peak velocity by an average of 202ms in the successful trials versus 54ms in the unsuccessful trials compared to the template endpoint time to peak velocity ($F_{2,15}=10.5$, $p < 0.01$; [Table 16](#), [Table 17](#)) with no difference between the visits.

For the shoulder, subjects in both visits increased horizontal adduction by 28° ($F_{2,15}=8.7$, $p < 0.01$), increased internal rotation by 10° ($F_{2,15}=21.5$, $p < 0.01$), and decreased shoulder flexion by 7° ($F_{2,15}=4.7$, $p < 0.05$) in the successful reaches compared to template reaches. Subjects in both visits increased trunk rotation to the side contralateral to the affected arm (successful=7.6° versus template=4.4°, $F_{2,15}=9.8$, $p < 0.01$; [Table 18](#), [Table 19](#)).

4.4.4 DIVERGENCE POINT

Overall, successful door avoidance as characterized by the divergence point was similar between Visits 1 and 2. Successful divergence point occurred closer to the starting position compared to failed trials. In both visits, the successful divergence point occurred at $27.4 \pm 22.1\%$ from the starting position while the failed divergence point occurred at $55.9 \pm 35.0\%$ from the starting position ($F_{1,16}=17.3$, $p<0.01$). Although the means of the successful and failed DPs at the second visit tended to be closer to the starting position than the means of the first visit, there was no statistical difference.

4.5 RELATIONSHIPS OF SUCCESS RATE AND SUCCESSFUL DIVERGENCE POINT WITH CLINICAL SCORES AND KINEMATIC MEASURES FOR THE STROKE GROUP AT 2ND VISIT

Success rate for the stroke group at the second visit was related to the endpoint peak velocity, endpoint time to peak velocity, index of curvature and trunk forward flexion when the hand arrived at the point of door contact ([Figure 13](#)). Lower endpoint peak velocities and slower time to reach endpoint peak velocity were related to higher success rates ($r=-0.73$, $p<0.01$; $r=0.60$, $p<0.05$ respectively). A higher index of curvature was related to better success rates ($r=0.67$, $p<0.05$). Less trunk forward flexion near the point of door contact was related to higher success rates ($r=-0.59$, $p<0.05$). No other correlations were present between success rates and clinical tests.

Earlier successful divergence points were related to higher posterior deltoid strength ($r=-0.6$, $p<0.05$; [Figure 14](#)). No other correlations were present between successful divergence points and kinematic variables or other clinical tests.

As with the first visit, the MAL-SES_T at visit 2 was correlated with the total scores of the MAL-AOU_T and MAL-QOM_T ($r=0.73$, $p<0.01$, $r=0.78$, $p<0.01$ respectively, [Figure 15](#)). Higher MAL-SES_T were related to higher anterior deltoid strength ($r=0.57$, $p<0.05$) and larger shoulder extension ROM ($r=0.58$, $p<0.05$; [Figure 16](#)).

Figure 13: Correlations of success rate with endpoint peak velocity (EPV; A), time to peak velocity (ETPV; B), index of curvature (C), and trunk flexion (D) for stroke group at second visit were performed. Lower EPVs and slower ETPVs were related to higher success rates ($r=-0.73$, $p<0.01$; $r=0.60$, $p<0.05$ respectively). A higher index of curvature was related to better success rates ($r=0.67$, $p<0.05$). Decreased trunk forward flexion near the point of door contact was related to higher success rates ($r=-0.59$, $p<0.05$).

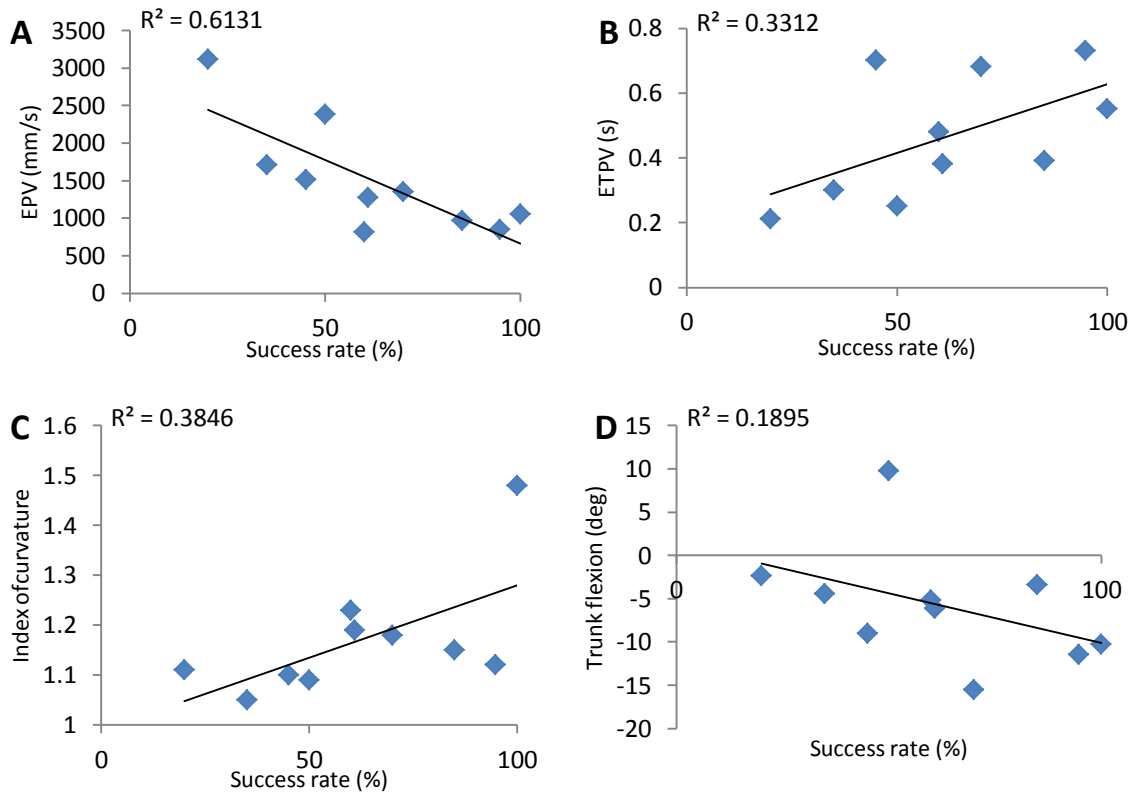


Figure 14: A correlation of successful divergence point with posterior deltoid strength (DP) for stroke group at second visit was performed. Earlier successful divergence points (i.e. larger margin of error) were related to higher posterior deltoid strength ($r=-0.6$, $p<0.05$).

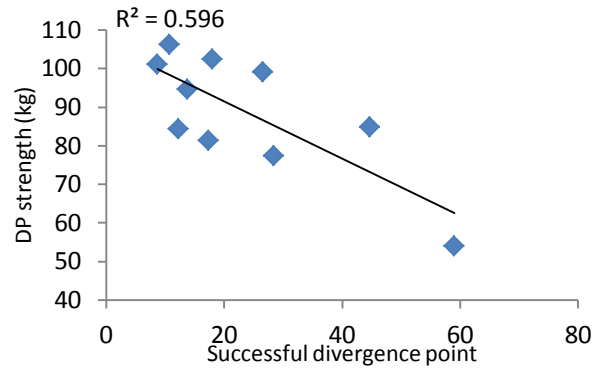


Figure 15: Correlations of MAL-SES_T with MAL-AOU_T (A) and MAL-QOL_T (B) for the stroke group at second visit were performed. As with the first visit, the MAL-SES_T at Visit 2 was correlated with the total scores of the MAL-AOU_T and MAL-QOM_T ($r=0.73$, $p<0.01$; $r=0.78$, $p<0.01$ respectively).

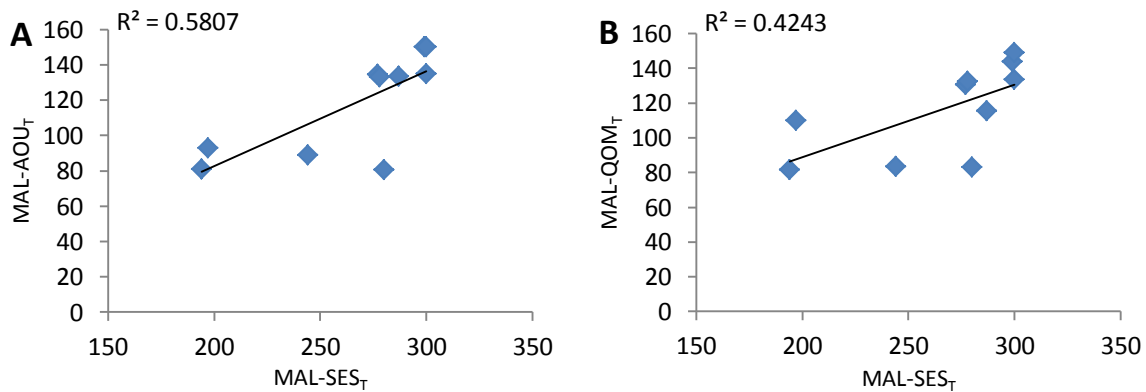
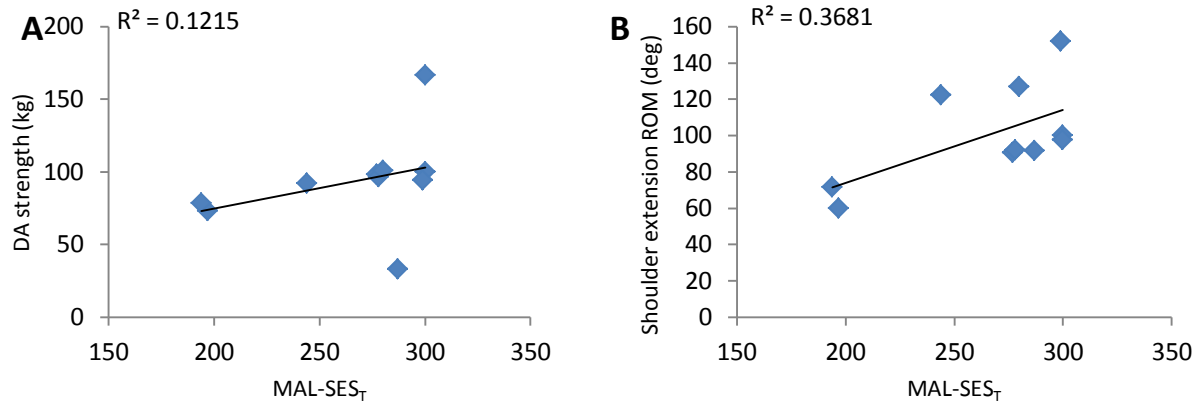


Figure 16: Correlations of MAL-SES_T with the anterior deltoid strength (DA; A) and shoulder extension range of motion (B) for stroke group at second visit were performed. Higher MAL-SES_T were related to higher anterior deltoid strength ($r=0.57$, $p<0.05$) and larger shoulder extension ROM ($r=0.58$, $p<0.05$).



CHAPTER 5: DISCUSSION

Reaching and obstacle avoidance behaviours in control and stroke subjects were described in terms of endpoint performance variables (endpoint peak tangential velocity, EPV; endpoint time to peak tangential velocity, ETPV; index of curvature, IC; endpoint trajectory length, ETL) and movement quality variables (wrist, elbow, shoulder, and trunk displacement). The dependent variables were compared between groups and then to clinical data to identify the relationships between functional ability and success in the experimental task. One year after the initial testing session, stroke subjects repeated the experiment. Data were compared between Visits 1 and 2 to identify if these relationships change over time.

The stroke subjects participating in our study were considered well-recovered as indicated by high functional scores in the clinical assessment; but they still exhibited mild motor deficits in the arm. For example, compared to their unaffected side, there was still residual weakness (~20% decrease) and decreased range of motion (~18% decrease) in their affected shoulders.

5.1 UNOBSTRUCTED REACHING

In Visit 1, performance and movement quality variables in the template reaching block were compared between groups. Endpoint performance variables (EPV, ETPV, IC, ETL), trunk and elbow joint angle excursions were similar between the two groups. The use of similar joint ranges between the groups partly agrees with results from Prange and colleagues (2010), in which shoulder elevation and elbow angles of mildly affected stroke patients were similar to controls when performing ADL-like upper limb movements. However, our study measured additional joint excursions. The stroke group compared to controls had decreased shoulder external rotation, decreased wrist flexion, and used wrist abduction instead of adduction near the time of object contact. The differences were small, but suggest that the stroke group demonstrated an altered pattern of movement for an unobstructed, rapid reaching task. Keeping in mind that the average Chedoke-McMaster Stroke Assessment (CMSA) arm and hand scores were 6, the results support the definition of Stage 6 in the CMSA scoring: abnormal movement patterns emerge when rapid or complex actions are performed. The appearance of alternate strategies may reflect how our participants with hemiparesis combined different degrees of

freedom still available to them in order to succeed in the task (Bernstein 1967; Latash & Anson, 1996).

Another argument for the presence of group differences is that, in fact, unobstructed reaching requires the coordination of multiple joints and the fine control of the speed and accuracy of the endpoint. Problems with single and multi-joint coordination and endpoint control have been found in other studies investigating the upper limb in mildly affected stroke subjects. In a study by Reisman and Scholz (2003), mildly and moderately affected hemiparetic post-stroke subjects and control subjects performed multiple reaches to two targets within their functional arm length (distance of acromion to metacarpophalangeal joint of the index finger). Individual joint ROM, interjoint coordination, and variability in performance variables were measured. Both the mildly and moderately affected stroke group had limited shoulder and elbow ROM and altered patterns of coordination between the shoulder and elbow. The variance of joint combinations possible for the task were divided into 1) variation of joint angle combinations leading to a consistent hand position (goal-equivalent variance) vs 2) combinations leading to variable hand positions other than the goal position (non-goal-equivalent variance). The mild-to-moderately affected stroke subjects used joint configurations that limited hand path variance while still having goal-equivalent variance similar to the control subjects. A principle component analysis was performed to evaluate what components (combination of joint angles) accounted for most of the variance in the observed variables. The hemiparetic individuals did have different joint coupling patterns. Similarly, the well-recovered post-stroke individuals from our study also had differences in the patterns of joint positions compared to controls.

The group differences in the shoulder and wrist kinematic variables during the template reach block are important because they objectively identify task-related residual deficits of joint movement in well-recovered stroke subjects. Many clinical tests of functional tasks objectively measure task completion, but only allow subjective observation of movement quality. One exception is the Reaching Performance Scale for Stroke (RPSS) which was specifically developed to identify deficits in movement kinematics during performance of a reach to grasp task (Levin et al., 2004). However, even this clinical movement quality scale did not identify the residual deficits in shoulder rotation and wrist movement because of the difference between the experimental and RPSS grasping task. In addition the RPSS task does not measure wrist

movements nor take into account the speed of the movement. Indeed, on this clinical scale, only 5/17 subjects had slight movement segmentation and the remaining 12 subjects achieved the maximum RPS score.

Another reason why deficits were revealed was that subjects had to reach as fast as possible. This requirement added difficulty to the task (Fitts 1954) and may have unmasked compensatory behaviours. In a previous study, DeJong and Lang (2012) found that when asked to make a reaching movement as fast as possible, the subjects with hemiparesis were able to achieve reach durations equal to the preferred speed of reaches in the control group. Both groups also achieved straighter reach paths when reaching faster compared to their respective preferred speeds, leading the authors to conclude that faster movements improved movement quality. However, the straightness of the reach trajectory is actually a descriptor of task performance and not movement quality as defined by Levin and colleagues (2009), as it describes the behaviour of the end effector, in this case the hand. Our results agree with Dejong and colleagues (2012) that stroke subjects can succeed in performing a task in a similar manner to controls. On the contrary, movement quality variables should describe how the movement is achieved both at the level of single joints and segments as well as at the level of the coordination between joints and segments (Cirstea & Levin, 2000; Michaelson et al., 2006; Levin et al., 2009). Our measures of joint excursions highlight that while the control and well-recovered stroke groups performed at similar speeds and had similar trajectory profiles, the stroke group used alternate joint patterns to complete the task, similar to previous work (Cirstea & Levin, 2000; Levin et al., 2002; Knaut et al., 2009; Subramanian et al., 2010).

5.2 OBSTRUCTED REACHING AND OBSTACLE AVOIDANCE STRATEGIES

The overall success rate was calculated as the number of successful trials over the total number of trials in the random block. Controls achieved a success rate of $55.9 \pm 24.0\%$, while the stroke group achieved $43.5 \pm 23.2\%$ suggesting that the task was similarly difficult for both groups. However, the comparison of the proportion of individuals who achieved different levels of success rates indicated that at the success level of 65%, the proportion of subjects who achieved success was significantly lower for the stroke group (12%) compared to controls. The poorer

performance of the stroke group at this level was possibly due to the differences in the spatial movement kinematic patterns between groups (see below).

5.2.1 TASK PERFORMANCE VARIABLES

The temporal variables pertaining to endpoint performance did not differ between the groups. To successfully avoid the door, all subjects reached more slowly, evidenced by decreased EPV (58% decrease) and increased ETPV (170% increase) with no differences between the groups. The strategy of decreasing velocity in order to improve accuracy follows an extension of Fitts' law (Fitts 1954; Jax et al., 2007; Vaughan et al., 2010), which predicts that the movement time of the end effector is affected by the final target distance, the amount by which an obstacle extends beyond the straight line between the targets, and the movement's index of difficulty. Healthy individuals reached slower when obstacles were within the reach distance and were close to the reaching hand (Chapman & Goodale, 2008). Individuals with mild stroke impairments decreased velocity more than controls in order to maintain endpoint precision in unobstructed pointing tasks (Subramanian et al., 2011; Mani et al., 2013). In movements with online correction, mildly and moderately affected post-stroke individuals increased their movement time compared to control subjects when they reached to targets and then corrected their path when the same target unexpectedly changed position (jumped; Schaefer et al., 2012). The movement times for the target jump trials almost doubled compared to baseline trials (controls: 913ms vs. 554ms; stroke: 1349ms vs. 815ms) and the mean movement time of the stroke group was significantly longer than controls. Our subjects also increased movement times to avoid the obstacle, but with no differences between groups (mean of all subjects: successful trials = 1420ms vs. template trials = 805ms).

As well, some spatial variables were similar between groups. For example, the length and curvature of the trajectory path (ETL and IC) of successful trials was increased compared to the template and failed trials for all subjects. In other words, increasing trajectory length led to successful obstacle avoidance in both groups. However, it should be noted that although not statistically significant, the trajectory length of successful trials in the stroke group was only 19mm greater than the failed trials (821mm vs. 802mm), while the controls had a difference of

86mm (813mm vs. 727mm). On the contrary, the stroke group had similar trajectory lengths in the successful and failed trials, indicating that while the stroke group maintained a longer trajectory throughout the random block, lengthening the trajectory did not ensure task success. In a similar trend, the curvature of the trajectory path of the failed trials in the stroke group was similar to that of the successful trials in the control group. This indicates that even though stroke subjects used a strategy of increasing both trajectory length and curvature throughout the random block, they still did not always succeed in avoiding the obstacle. The stroke subjects may have had difficulty making rapid corrections to their endpoint trajectories in order to change their reach strategy, particularly because of the increased difficulty of the obstructed condition compared to the template condition. The root of the difficulty, however, could not be found in the performance outcome variables when considered separately because there were no group differences for EPV, ETPV, ETL and IC values. The differences between our groups were better explained by the movement quality variables.

5.2.2 MOVEMENT QUALITY VARIABLES

Controls used more wrist flexion and elbow flexion than stroke subjects to avoid the door. The relative increased use of the distal joints (wrist and elbow) in the avoidance reach strategy has also been observed by Jaric and Latash (1998). In their study, healthy subjects practiced moving their hand as fast as possible between two targets while also avoiding stationary obstacles placed in the arm path. Before practice, subjects performed the task by predominantly using shoulder and elbow movements. As they learned the task, subjects changed their reaching strategy and used more wrist excursions to avoid the obstacles. The degrees of freedom (DF) utilized to avoid obstacles were specific to the body segment that was more involved with avoiding the obstacle (the wrist) rather than the more proximal DFs (the shoulder and trunk). Our subjects were well-practiced in avoiding the obstacle before attempting the randomized block, so their strategy of increased wrist and elbow use to avoid the obstacle was consistent with the data from Jaric and Latash (1998).

Similar to the control group, the stroke group used more shoulder horizontal adduction and shoulder internal rotation to avoid the door. The similarities in the shoulder strategy of the two

groups suggest that the stroke group had largely recovered shoulder functional use for reaching, but there was a difference in how shoulder rotation was used by the stroke group. Although the range of shoulder rotation was similar between groups, the stroke subjects had a more internally rotated initial shoulder position. To successfully avoid the door, the shoulders of stroke subjects reached a more internally rotated position (-6.7°) compared to control subjects (3.5°). Indeed, in the clinical test of active shoulder ROM in the stroke group, external shoulder rotation of the affected arm was less than the unaffected arm, supporting the suggestion that the initial position of the stroke subjects was more internally rotated. Thus, although stroke subjects used the same strategy as the control group, movements were restricted by their limited range of shoulder external rotation. The decreased use of shoulder external rotation in our task indicated impairments of the proximal as well as the distal arm in our clinically well-recovered chronic stroke subjects. Previous work investigating the progression of early and late motor recovery in moderately-affected patients suggests that recovery does not necessarily occur in a proximal to distal direction in the upper limb post-stroke (Lang & Beebe, 2007; Beebe & Lang, 2008). In the Beebe and Lang study (2008), regression analysis showed that the level of upper limb active range of motion (AROM) could explain 82% of the variance in hand function, with the variance shared equally among proximal, middle and distal joints of the UL. Furthermore, hierarchical regression analysis showed that 88% of the variance in hand function could be explained by shoulder AROM alone. Our findings concur with these previous results, suggesting that mild shoulder impairments in well-recovered post-stroke individuals can be present even if they have good hand function and continue to affect how the arm is used.

The stroke subjects also used different strategies involving elbow and trunk movement. Their elbow ROM did not vary throughout the experiment, such that the elbow was in a slightly flexed position ($\sim 123^{\circ}$) in the template, successful and failed trials. In comparison, controls varied the amount of elbow ROM according to the condition (successful: 109° , failed: 115° , template: 129°). In another study of reaching ability in individuals post-stroke, the amount of active elbow extension while reaching for a drinking glass was decreased in the stroke subjects with mild impairments compared to healthy participants (Alt Murphy et al., 2011). However, our stroke subjects had full elbow PROM during the clinical evaluation. Rather than having decreased ROM, our stroke subjects may have had difficulty incorporating the appropriate amount of elbow movement into their reach because they could not quickly adjust their reaching strategy when

faced with an obstacle in the reaching path. Stroke subjects have exhibited greater difficulty in adjusting movements in unpredictable environments such as producing elbow flexion and extension movements while reaching for targets against position-dependent (Dancause et al., 2002; Subramanian et al., 2014) and velocity-dependent forces (Scheidt & Stoeckmann, 2007). In these studies, stroke subjects had difficulty using visual and proprioceptive information about the perturbations and movement errors from previous trials to modify hand trajectories in subsequent trials. In our study, stroke subjects did not receive force perturbations, but still had deficits in adapting their movements based on perceived information in the randomized block. Their difficulties may have been due to the inability to rapidly integrate visual information and modify the motor plan. It could be questioned if the deficits were a result of poor motor learning from the previous blocks, but the practice trials were repeated until we were ensured that the subjects had established their obstacle avoidance strategy for both doors. It could also be questioned if the visual information that the subjects received from the VE was sufficient for the stroke subjects to control their movements. The position of the elbow was not visible, so subjects had to depend on proprioceptive information about the location of their elbow in space. If there was any proprioceptive impairment, stroke subjects would not have any visual information to compensate for proprioceptive loss. However, the presence of proprioceptive impairment was assessed and cause for exclusion from the study.

Another reason for the stroke group using a limited elbow ROM in their reaching movements was that the task required fast movements. When fast arm movements are performed, the threshold of the stretch reflex in the biceps is decreased and flexor spasticity can limit rapid arm extension (Levin & Feldman, 1994). Mild spasticity was present in eight out of 17 subjects. Since spasticity is velocity-dependent and the obstacle avoidance movement is rapid, the fast arm extension could have caused a pathological stretch response to occur in the arm flexor muscles. The stretch response is part of the flexor synergy. The flexor synergy is an abnormal stereotypical movement pattern that does not adapt to changes in task or environmental demands (Shumway-Cook & Woollacott, 2012). In the UL, the flexor synergy is characterized by a fixed combination of muscular activity including scapular retraction and elevation, shoulder abduction and external rotation, elbow flexion, forearm supination, and wrist and finger flexion (Bobath 1990; Brunnstrom 1970). All of the listed components are not equal in strength, with elbow flexion being the strongest component of the synergy. The flexor synergy movement pattern

emerges when patients with moderate to severe upper limb hemiparesis attempt movements of their paretic limb. As recovery progresses, patients gain control of individual joint rotations within the synergy, but are unable to initiate movement outside of the synergy. With further recovery, patients are able to move out of synergy, but abnormal elbow flexion is one of the last components to disappear (Brunnstrom 1970). Post-stroke individuals with milder deficits do not generally present with the flexion synergy pattern. When our well-recovered stroke group performed the obstacle avoidance task, elbow flexion was not modulated with the task conditions and they tended to have a more supinated forearm posture compared to controls. This suggests that some components of the synergy, namely abnormal elbow flexion and forearm supination, only become apparent when performing challenging arm movements.

Trunk forward flexion and trunk rotation toward the less affected side were more pronounced in the obstructed trials for the stroke group compared to the control group. Therefore, even in mildly affected stroke subjects, trunk compensatory patterns were used in reaching, which agrees with previous work from our lab (Cirstea & Levin, 2000; Levin et al., 2002; Michaelson et al., 2006; Subramanian et al., 2010). For post-stroke individuals, increasing trunk use during reaching may be a common compensatory strategy. In Cirstea & Levin (2000), seated stroke and control subjects made multiple pointing movements to targets at different distances and locations in their arm workspace. The different target positions included targets at arm's length, beyond arm's length, and locations in the ipsi- and contralateral workspace. Regardless of the target location or distance, all stroke subjects incorporated more trunk flexion and rotation to move the hand to the target (Cirstea & Levin, 2000; Levin et al., 2002). In contrast to our study, the stroke subjects of the previous studies had a wide range of arm impairment, with Fugl-Meyer arm scores of 15 to 65 out of a possible total score of 66. The authors noted that the mildly affected stroke subjects tended to use less trunk movement than the moderately to severely affected subjects, but also tended to use slightly more trunk movement than the control subjects. The unobstructed reaching task of the previous studies may not have targeted the motor elements that the mildly affected stroke subjects were having difficulty performing. Our obstacle avoidance task was more complicated than the tasks of the previous studies, which would bring out the residual deficit of mildly affected stroke subjects. Overall, the stroke subjects used different joint configurations from the controls in their reaching avoidance strategy even though they were considered clinically well-recovered.

5.3 MARGIN OF ERROR

Recall that the difference between the successful and failed DP was defined as the spatiotemporal margin of error for task success. The failed DP of the control group was considered the critical temporal-spatial threshold where the obstacle could no longer be avoided. Since the successful DP of the stroke group occurred closer to the critical threshold than the control successful DP (see Section 5.2 above), the stroke group had reduced time and space to avoid the obstacle. Previous studies of obstacle avoidance while reaching exhibited a similar strategy of maintaining distance from obstacles (Aivar et al., 2008; Chapman & Goodale, 2010). Maintenance of distance from the obstacle may be impaired in post-stroke individuals because of existing motor control deficits.

Most upper limb obstacle avoidance literature characterizes behaviour in healthy individuals, and only a few groups have studied individuals with post-stroke hemiparesis. In earlier studies, 2D reaching of healthy individuals described a three-segment curved hand path when avoiding an obstacle (Abend et al., 1982; Flash & Hogan, 1985). First, a person moved their hand in a straight or slightly curved path to an intermediate position close to the obstacle, termed a via point. Around the via point there was a greater hand path curvature. When the via point was passed, the subjects then again displayed a straight or slightly curved hand path towards the goal. Flash and Hogan (1985) viewed the via point as the control variable that acted as the axis of movement. Other studies of obstacle avoidance in 3D reaching characterized a hand path with a more constant curvature and timing (Dean & Brüwer, 1994, 1997; Grimme et al., 2012). A single via point would not explain the more uniformly curved hand path. Some researchers viewed the via point as an emergent property rather than the control variable that modified the characteristics of the hand path (Torres et al., 2006; Vaughan et al., 2010). In this case, the focus of movement could be on the proximity of the hand to the obstacle and the margin of error. Margins of error were observed to be larger when the obstacle was closer to the person and their reaching arm (Chapman & Goodale, 2008). Margins of error were also larger when there was a higher level of visual uncertainty (Chapman & Goodale, 2010). In agreement with previous literature, our control subjects maintained a large margin of error in their obstructed reach profile in order to avoid the obstacle. However, the participants with stroke did not have as large a margin as the controls. It is possible that existing motor control deficits did not allow them to

initiate a quick change in their hand path as early as the controls, thus reducing the time and space where they could modulate movement patterns and successfully avoid the obstacle.

5.4 RELATIONSHIP BETWEEN SUCCESS RATE AND KINEMATIC MEASURES

For the control group, higher success rates were related to increased endpoint trajectory curvature and elbow flexion. These relationships further support the idea that the strategy of the control subjects was to avoid the obstacle by adjusting the position of the distal rather than the proximal portion of the limb, namely the forearm and hand (Jaric & Latash, 1998). Higher task success rates of the stroke group were related to lower EPVs, slower ETPVs and more shoulder internal rotation near the point of obstacle contact. Stroke subjects used a strategy of slowing down their movements and used the proximal segments of their body (shoulder and trunk) to help manoeuvre the position of the endpoint. This is consistent with previous studies in stroke subjects showing that the trunk is used to assist arm movement for various tasks. For example, mildly and moderately affected stroke subjects used greater trunk anterior displacement and rotation as well as compensatory shoulder involvement compared to controls for transporting the hand to a target and to orient the hand for grasping (Michaelson et al., 2004; Robi-Brami et al., 2003). Also, when individuals with post-stroke hemiparesis performed in-phase and anti-phase bilateral arm swinging, they rotated the trunk to assist the forward movement of the hemiparetic arm unlike controls (Ustinova et al., 2004). However, excessive trunk use has been mainly seen in patients with more severe hemiparesis than the stroke subjects in our obstacle avoidance task. The deficits in trunk use in the less-severely affected subjects were likely revealed by our study because of the higher task demands.

5.5 RELATIONSHIP BETWEEN SUCCESSFUL DIVERGENCE POINT AND CLINICAL MEASURES

Correlations between successful divergence points and the clinical scores highlighted the contributions of the existing level of upper limb impairment (ICF Body Structure and Function) to the task performance in the stroke group. Only one clinical assessment, Box and Blocks Test, was related to successful divergence point, contrary to our initial hypothesis. This may suggest

that current clinical assessments do not measure the higher-order motor control deficits that exist in well-recovered individuals.

Results from the Box and Blocks Test indicated an individual's level of manual dexterity in a common upper limb activity. The experimental reaching task was designed to mimic a common upper limb activity and measure arm dexterity. Higher Box and Blocks Test scores were positively correlated with earlier successful divergence points, indicating that the BBT could be used to predict the level of arm dexterity in stroke subjects. High levels of upper limb capacity (potential level of performance) have been positively correlated with actual arm performance (Michielsen et al., 2009). The positive relationship between the task performance and BBT in our study is consistent with previously results.

5.5 SELF-EFFICACY

A measure of self-efficacy of arm use after stroke (MAL-SES) based on the questions from the MAL was developed for this study since few studies to date have explored specifically self-efficacy of arm use. For the stroke population, more self-efficacy scales have been developed for functional activity as a whole, mobility, or self-care (Jones et al., 2008; Hellström et al., 2003; Haworth et al., 2009; Robinson-Smith et al., 2000). The MAL-SES was positively correlated with the MAL-AOU and MAL-QOM subscales. In other words, subjects with higher AOU and QOM scores reported higher confidence levels in completing the various activities of the MAL. The MAL-SES was positively related to the CMSA arm subscale, anterior deltoid strength, and medial deltoid strength. Therefore, the subjects seemed to be aware of the limitations in their arm, which in turn may have been reflected in their MAL-SES scores. On the other hand, MAL-SES was not related to any of the kinematic measures (movement quality or endpoint performance variables) in the experimental task. Recall that kinematic variables were measured because they were shown to be more precise in detecting residual motor impairment particularly in mildly- to moderately-affected hemiparetic post-stroke individuals (Subramanian et al., 2010). The lack of correlation with kinematic measures suggests that the MAL-SES had low sensitivity to changes in higher functioning post-stroke individuals because they had high scores with little variability between subjects ($\text{MAL-SES}_{\text{mean}}=8.7\pm1.2$ out of a possible score of 10). Closer

analysis of the subscale MAL scores revealed that there was no clear trend in the direction of the relationships between MAL subscales and the FMA.

The only factors that had a relationship with success rate were the various kinematic variables mentioned in Section 5.4. The self-efficacy results indicated that confidence alone could not influence and predict successful outcome. Therefore, another possible factor affecting success rate could have been the cognitive load of the task. Because of the task difficulty, there may have been increased cognitive load and thus more cognitive processing needed to coordinate the requirements of accuracy and speed necessary for the experimental task. There may have been an effect of cognitive levels or level of executive function on performance of our task, but the effects were not assessed in the present study.

5.6 STROKE GROUP AT 2ND VISIT

Stroke subjects were approached one year after Visit 1 to participate in a follow-up session where all clinical and experimental procedures were repeated. The purpose was to identify if reaching performance and quality changed over time.

5.6.1 CLINICAL AND TASK DIFFERENCES BETWEEN VISITS

At Visit 2, individuals improved deltoid strength as well as shoulder flexion, extension, internal rotation and horizontal adduction ROM compared to Visit 1. This represents an improvement towards movement patterns observed in the healthy subjects. Therefore, it was reasonable to expect differences in the way that they performed the experimental task. For the obstacle avoidance task, the overall success rate tended to be higher at Visit 1 but the difference was not significant (62% vs 46%). Comparison of individual success rates revealed that the number of individuals achieving a success rate over 65% increased to 4/10 individuals from 2/10 at Visit 1. However, the lack of significant differences may be due to the small sample size (n=10) which may not have had enough power to reveal differences in these variables. Nonetheless, comparison of some of the task movement quality variables portrayed trends towards improvement after 1 year. At Visit 2, there was a tendency of individuals to decrease trunk

rotation to the contralateral side. Improvement of functional reaching has been associated with decreased trunk involvement (Michaelson et al., 2006). On the other hand, an interesting observation was made concerning elbow and shoulder involvement in successful obstacle avoidance. First, subjects kept a fairly constant elbow flexed position in all obstructed reaching trials at both visits. This suggests that the flexion synergy or a certain level of elbow flexor spasticity may still have been present and was unmasked when the reaching task was more difficult. Thus, the stroke subjects would show evidence of using compensatory movements in the other upper limb and trunk segments.

The next observation was that controls and the stroke subjects at both visits used a similar shoulder strategy to successfully avoid the obstacle compared to the template condition. All subjects increased shoulder adduction and internal rotation as well as decreased shoulder flexion. To further support this idea of alternate compensatory patterns mentioned above, stroke subjects at Visit 2 had a tendency to increase shoulder adduction, internal rotation and decrease shoulder flexion more than in their initial visit as well as compared to the control group. Though these differences were not statistically significant, it could suggest that the reaching strategy of the stroke group at the second visit was becoming similar to the control group strategy, particularly when considered with other clinical and kinematic outcomes. Remember that shoulder strength and ROM significantly improved in the second clinical evaluation and that trunk involvement in the reaching task decreased. With the improvement of the shoulder function, the shoulder could actually be used to a much higher extent, and trunk compensation would not be necessary or as functionally efficient.

5.6.2 RELATIONSHIP BETWEEN SUCCESS RATE AND KINEMATIC MEASURES

In the first visit, the improvement of success rates were correlated with lower velocities, more curved trajectories, more shoulder external rotation and less shoulder flexion. Similarly, in the second visit, higher success rates were also related to lower velocities and more curved trajectories. Also, less trunk forward flexion was related to higher success rates, further supporting the observation in the previous section that trunk compensation was not as prevalent in Visit 2.

CHAPTER 6: SUMMARY AND CONCLUSIONS

6.1 SUMMARY OF RESULTS

The objective of this thesis was to identify and quantify impairments in higher-order motor function among individuals with well-recovered upper limbs post-stroke and to evaluate the effect of the impairments on daily use of the arm. It was hypothesized that well-recovered post-stroke individuals would be less successful at performing a higher-order motor task compared to healthy individuals. The well-recovered stroke group did have a difficult time with the obstacle avoidance task and only two subjects were able to achieve higher than a 65% success rate compared to the control group. The second hypothesis was that the stroke group would have subtle impairments in higher-order motor function. This hypothesis was confirmed when significant differences in reaching movement quality were observed in the stroke group. The stroke group, though well-recovered, did not leave themselves a large margin of error when they were trying to avoid the obstacle in their reaching path. Also, the stroke group showed evidence of compensatory movements such as increased involvement of the trunk forward flexion in the reaching task.

To evaluate the effect of the impaired obstacle avoidance ability on daily use of the arm, it was hypothesized that higher task success rates would positively correlate with better clinical measures of arm function. The task success rates did not correlate with any arm function clinical measures, but did correlate with movement quality variables from the obstacle avoidance task. The stroke subjects had the ability to successfully perform the clinical tasks, so it was suggested that the clinical measures were not sensitive enough or did not measure the ability to perform higher-order motor functions such as obstacle avoidance with the upper limb. In addition, earlier successful divergence points were positively correlated with Box and Blocks Test scores. This relationship would suggest that the primary outcome measure of spatiotemporal coordination in the obstacle avoidance task was affected by level of arm dexterity. It was also hypothesized that having mild upper limb deficits would affect the confidence of the post-stroke individuals in using their arm to its full capacity. The MAL-SES_T was related to the CMSA arm subscale, anterior deltoid strength and medial deltoid strength, so subjects were aware of their subtle impairments and had lower confidence scores in the MAL-SES_T questionnaire.

Finally, the last hypothesis of the study concerned the longitudinal changes in obstacle avoidance ability and arm function clinical measures one year after the initial visit. Although there were no statistically significant differences between the two visits, the returning group did show trends of increasing shoulder strength and range of motion in the clinical assessment, increasing shoulder external rotation and decreasing trunk involvement in the obstacle avoidance task, and improving task success rates.

6.2 CLINICAL RELEVANCE

The results presented in this thesis have the following implications for clinicians and rehabilitation experts:

Individuals who are considered well-recovered after a stroke may have impairments in higher-order motor control that is not detected with current clinical assessments. Participants in the stroke group were considered high-functioning, independent, well-recovered individuals after a stroke. However, they had difficulty succeeding in a task requiring quick movements and online corrections of hand and arm movement. This implies that current clinical measures of upper limb function are not detecting the higher-order motor deficits. This places well-recovered post-stroke individuals at a disadvantage, where they are deemed “too good” for enrollment into rehabilitation programs. Kinematic measures of upper limb daily activities may be more appropriate for detecting changes in movement quality and compensatory strategies. Developing a test for higher-order motor impairments using virtual environments and motion analysis allows assessment of task performance and movement quality in ecologically valid tasks while still having control of task parameters.

Lower self-confidence in upper limb ability may be reflective of the subtle deficits found in well-recovered post-stroke individuals. Although many well-recovered post-stroke individuals may achieve good scores on clinical sensorimotor function assessments, clinicians should be aware of other factors that affect motor performance. One question of this thesis was concerned with the effect of self-efficacy of arm use on actual arm use and found that it was related to shoulder strength and range of motion. Rating self-efficacy of arm use could be useful for clinicians and rehabilitation experts to detect the presence of subtle sensorimotor deficits that are often missed

in clinical evaluations in order to improve recovery of arm function and increase arm use in the well-recovered post-stroke population. It is important to note that many more factors, such as cognition and depression affect motor performance, so they should be measured and factored into the rehabilitation programs aimed at improving sensorimotor function of the upper limb.

6.3 LIMITATIONS

Kinematics were used to quantify task performance (e.g. EPV, IC), and movement quality (e.g. shoulder rotation, trunk forward flexion) and were effective in detecting the alterations in movement patterns hypothesized to exist in the well-recovered stroke group. However, one limitation of the study was that electromyography was not used to evaluate muscle activations, so analysis and interpretation of muscle coordination was not possible (e.g. coordination between different segments of the arm, or the arm and trunk).

A second limitation of the study was that post-stroke individuals were only assessed two times, one year between each visit. There was a possibility that the well-recovered stroke subjects were improving in their reaching obstacle avoidance ability one year after the first evaluation. Therefore, more frequent visits before and after the one year mark would have created a more complete picture of long-term recovery in an individual who is mildly affected post-stroke.

Another limitation of this study is that the use of a VE may decrease the external validity of our study. To decrease this possibility, we designed the task to mimic a real-world situation requiring a typical behavioural reaction. Using an ecologically valid VE task allows the possibility that the learned behaviour could be transferred to a physical environment.

More stroke subjects would need to be recruited in order to generalize the results to the greater population of well-recovered post-stroke individuals. It is important to note that the results from the current study also cannot be generalized to subjects with more severe arm impairments, more than one incidence of stroke, cognitive deficits, mood disorders, and lesions in the non-dominant hemisphere.

This study illustrated that deficits in higher-order motor control could be subtle but still detrimental to the quality of upper limb movement in mildly affected post-stroke individuals. To

isolate the effect of impaired obstacle avoidance ability, exclusion criteria were applied during the recruitment of participants to remove possible confounding variables. However, factors that influence motor performance, such as cognition should also be measured for their effects on higher-order motor function.

6.4 FUTURE DIRECTIONS

The results presented in this thesis raise new questions for future research. Deficits can be identified in individuals who are considered well-recovered, but current clinical tools do not have the same sensitivity as research laboratory measures. Tools should be developed to help detect subtle impairments, but these tools must be accessible to the clinician. This thesis presents novel work in which higher-order motor control deficits can be detected by measuring not only task performance outcomes, but also movement quality while performing challenging real-world tasks. Through the use of virtual reality platforms, individuals can be assessed performing daily tasks in a safe and controlled environment. Furthermore, well-recovered post-stroke individuals perform well on the current clinical tests, so it follows that stroke rehabilitation programs designed for moderately to severely affected patients will not be appropriate for their level of impairment. A better understanding of what is being controlled when a person performs higher-order motor activities can help to direct rehabilitation towards improving impairments specific to well-recovered post-stroke individuals.

REFERENCES

- Abend W, Bizzi E, & Morasso P. (1982). Human arm trajectory formation. *Brain*, 105(2), 331-348.
- Aglioti S, DeSouza JF, & Goodale MA. (1995). Size-contrast illusions deceive the eye but not the hand. *Current Biology*, 5(6), 679-685.
- Agrell B & Dehlin O. (1989). Comparison of six depression rating scales in geriatric stroke patients. *Stroke*, 20, 1190-1194.
- Agrell BM, Dehlin OI, & Dahlgren CJ. (1997). Neglect in elderly stroke patients: A comparison of five tests. *Psychiatry and Clinical Neurosciences*, 51(5), 295-300.
- Aivar MP, Brenner E, & Smeets JBJ. (2008). Avoiding moving obstacles. *Experimental Brain Research*, 190(3), 251-264.
- Alexander GE. (1994). Basal ganglia-thalamocortical circuits: Their role in control of movements. *Journal of Clinical Neurophysiology*, 11(4), 420-431.
- Alt Murphy M, Willén C, & Sunnerhagen KS. (2011). Kinematic variables quantifying upper-extremity performance after stroke during reaching and drinking from a glass. *Neurorehabilitation and Neural Repair*, 25(1), 71-80.
- American Guidance Service. (1969). *The Minnesota Rate Manipulative Tests. Examiner's manual*. Circle Pines, MN: Author.
- Amiez C & Petrides M. (2014). Neuroimaging evidence of the anatomo-functional organization of the human cingulate motor areas. *Cerebral Cortex*, 24(3), 563-578.
- Andersen RA & Buneo CA. (2002). Intentional maps in posterior parietal cortex. *Annual Review of Neuroscience*, 25(1), 189-220.
- Appelros P. (2006). Prevalence and predictors of pain and fatigue after stroke: a population-based study. *International Journal of Rehabilitation Research*, 29(4), 329-333.

- Archambault P, Pigeon P, Feldman AG, & Levin MF. (1999). Recruitment and sequencing of different degrees of freedom during pointing movements involving the trunk in healthy and hemiparetic subjects. *Experimental Brain Research*, 126(1), 55-67.
- Arienzo D, Babiloni C, Ferretti A, Caulo M, Del Gratta C, Tartaro A, Rossini PM, & Romani GL. (2006). Somatotopy of anterior cingulate cortex (ACC) and supplementary motor area (SMA) for electric stimulation of the median and tibial nerves: an fMRI study. *Neuroimage*, 33(2), 700–705.
- Astrup J, Siesjo BK, & Symon L. (1981). Thresholds in cerebral ischemia-the ischemic penumbra. *Stroke*, 12(6), 723-725.
- Atkeson CG & Hollerbach JM. (1985). Kinematic features of unrestrained vertical arm movements. *The Journal of Neuroscience*, 5(9), 2318-2330.
- Augustine JR. (2008). *Human neuroanatomy: An introduction*. London: Elsevier.
- Bailey MJ, Riddoch MJ, & Crome P. (2000). Evaluation of a test battery for hemineglect in elderly stroke patients for use by therapists in clinical practice. *NeuroRehabilitation*, 14(3), 139-150.
- Bandura A. (1977). Self-efficacy: Toward a unifying theory of behavioral change. *Psychological Review*, 84(2), 191-215.
- Bandura A. (1986). The explanatory and predictive scope of self-efficacy theory. *Journal of Social and Clinical Psychology*, 4(3), 359-373.
- Bandura A. (2001). *Guide for constructing self-efficacy scales*. Retrieved from <http://www.uky.edu/~eushe2/BanduraPubs/BanduraGuide2006.pdf>
- Beebe JA & Lang CE. (2008). Absence of a proximal to distal gradient of motor deficits in the upper extremity early after stroke. *Clinical Neurophysiology*, 119(9), 2074-2085.

- Beer RF, Dewald JP, & Rymer WZ. (2000). Deficits in the coordination of multijoint arm movements in patients with hemiparesis: Evidence for disturbed control of limb dynamics. *Experimental Brain Research*, 131(3), 305-319.
- Beggs WDA & Howarth CI. (1972). The movement of the hand towards a target. *The Quarterly Journal of Experimental Psychology*, 24(4), 448-453.
- Bernstein NA. (1967). *The coordination and regulation of movements*. Oxford: Pergamon.
- Berkinblit MB, Gel'fand IM, & Fel'dman AG. (1986). [A model of control of the movement of the multiarticular extremity]. *Biofizika*, 31(1), 128-138.
- Binkofski F & Buccino G. (2006). The role of ventral premotor cortex in action execution and action understanding. *Journal of Physiology-Paris*, 99(4), 396-405.
- Blumenfeld H. (2010). *Neuroanatomy through clinical cases* (2nd ed.). Sunderland, MA: Sinauer.
- Bobath B. (1990). *Adult hemiplegia. Evaluation and treatment* (3rd ed.). Oxford: Butterworth-Heinemann.
- Bogard K, Wolf S, Zhang Q, Thompson P, Morris D & Nichols-Larsen D. (2009). Can the Wolf Motor Function Test be streamlined? *Neurorehabilitation and Neural Repair*, 23(5), 422-428.
- Bohannon RW. (1988). Make tests and break tests of elbow flexor muscle strength. *Physical Therapy*, 68(2), 193-194.
- Bourbonnais D, Noven SV, Carey KM, & Rymer WZ. (1989). Abnormal spatial patterns of elbow muscle activation in hemiparetic human subjects. *Brain*, 112(1), 85-102.
- Bourbonnais D & Noven SV. (1989). Weakness in patients with hemiparesis. *American Journal of Occupational Therapy*, 43(5), 313-319.
- Brass M, Bekkering H, Wohlschlaeger A, & Prinz W. (2000). Compatibility between observed and executed finger movements: Comparing symbolic, spatial and imitative cues. *Brain and Cognition*, 44(2), 124-143.

- Brenner E & Smeets JBJ. (1997). Fast responses of the human hand to changes in target position. *Journal of Motor Behavior*, 29(4), 297-310.
- Brodal A. (1973). Self-observations and neuro-anatomical considerations after a stroke. *Brain*, 96(4), 675-694.
- Broeks JG, Lankhorst GJ, Rumping K, & Prevo AJ. (1999). The long-term outcome of arm function after stroke: Results of a follow-up study. *Disability and Rehabilitation*, 21(8), 357-364.
- Brown CE & Murphy TH. (2007). Livin' on the edge: Imaging dendritic spine turnover in the peri-infarct zone during ischemic stroke and recovery. *The Neuroscientist*, 14(2), 139-146.
- Brown CE, Aminoltejari K, Erb H, Winship IR, & Murphy TH. (2009). In vivo voltage-sensitive dye imaging in adult mice reveals that somatosensory maps lost to stroke are replaced over weeks by new structural and functional circuits with prolonged modes of activation within both the peri-infarct zone and distant sites. *The Journal of Neuroscience*, 29(6), 1719-1734.
- Brunnstrom S. (1970). *Movement therapy in hemiplegia: a neurophysiological approach*. New York: Harper & Row.
- Buccino G, Binkofski F, & Riggio L. (2004). The mirror neuron system and action recognition. *Brain and Language*, 89(2), 370-376.
- Burke D. (1988). Spasticity as an adaptation to pyramidal tract injury. [Review]. *Advances in Neurology*, 47, 401-423.
- Cailliet R. (1980). *The shoulder in hemiplegia*. Philadelphia, PA: FA Davis.
- Calota A, Feldman AG, & Levin MF. (2008). Spasticity measurement based on tonic stretch reflex threshold in stroke using a portable device. *Clinical Neurophysiology*, 119(1), 2329-2337.

- Capaday C, Ethier C, Van Vreeswijk C, & Darling WG. (2013). On the functional organization and operational principles of the motor cortex. *Frontiers in Neural Circuits*, 7(66).
- Carlsson GE, Möller A, & Blomstrand C. (2003). Consequences of mild stroke in persons <75 years – A 1-year follow-up. *Cerebrovascular Diseases*, 16(4), 383-388.
- Carlsson GE, Möller A, & Blomstrand C. (2004). A qualitative study of the consequences of ‘hidden dysfunctions’ one year after a mild stroke in persons < 75 years. *Disability and Rehabilitation*, 26(23), 1373-1380.
- Carlsson GE, Möller A, & Blomstrand C. (2009). Managing an everyday life of uncertainty – A qualitative study of coping in persons with mild stroke. *Disability and Rehabilitation*, 31(10), 773-782.
- Carlton LG. (1980). Movement control characteristics of aiming responses. *Ergonomics*, 23(11), 1019-1032.
- Carmichael ST. (2006). Cellular and molecular mechanisms of neural repair after stroke: Making waves. *Annals of Neurology*, 59(5), 735-742.
- Carr JH & Shepherd RB. (1987). A motor learning model for rehabilitation. In JH Carr, RB Shepherd, J Gordon, AM Gentile, JN Held (Eds.), *Movement science: Foundations for physical therapy in rehabilitation* (pp. 31-91). Rockville, MD: Aspen.
- Castiello U. (1996). Grasping a fruit: Selection for action. *Journal of Experimental Psychology: Human Perception and Performance*, 22(3), 582-603.
- Chapman CS & Goodale MA. (2008). Missing in action: The effect of obstacle position and size on avoidance while reaching. *Experimental Brain Research*, 191(1), 83-97.
- Chapman CS & Goodale MA. (2010). Obstacle avoidance during online corrections. *Journal of Vision*, 10(11), 17.

- Chen HM, Chen CC, Hsueh IP, Huang SL, & Hsieh CL. (2009). Test-retest reproducibility and smallest real difference of 5 hand function tests in patients with stroke. *Neurorehabilitation and Neural Repair*, 23(5), 435-440.
- Chen TL, Babiloni C, Ferretti A, Perrucci MG, Romani GL, Rossini PM, Tartaro A, & Del Gratta C. (2008). Human secondary somatosensory cortex is involved in the processing of somatosensory rare stimuli: An fMRI study. *Neuroimage*, 40(4), 1765-1771.
- Chollet F, DiPiero V, Wise RJS, Brooks DJ, Dolan RJ, & Frackowiak RSJ. (1991). The functional anatomy of motor recovery after stroke in humans: A study with positron emission tomography. *Annals of Neurology*, 29(1), 63-71.
- Cirstea MC & Levin MF. (2000). Compensatory strategies for reaching in stroke. *Brain*, 123(5), 940-953.
- Cirstea MC, Mitnitski AB, Feldman AG, & Levin MF. (2003). Interjoint coordination dynamics during reaching in stroke. *Experimental Brain Research*, 151(3), 289-300.
- Claassen J, Carhuapoma JR., Kreiter KT, Du EY, Connolly ES, & Mayer SA. (2002). Global cerebral edema after subarachnoid hemorrhage frequency, predictors, and impact on outcome. *Stroke*, 33(5), 1225-1232.
- Clavagnier S, Prado J, Kennedy H, & Perenin MT. (2007). How humans reach: Distinct cortical systems for central and peripheral vision. *The Neuroscientist*, 13(1), 22-27.
- Coderre AM, Zeid AA, Dukelow SP, Demmer MJ, Moore KD, Demers MJ, Bretzke H, Herter TM, Glasgow JI, Norman KE, Bagg SD, & Scott, S. H. (2010). Assessment of upper-limb sensorimotor function of subacute stroke patients using visually guided reaching. *Neurorehabilitation and Neural Repair*, 24(6), 528-541.
- Cohen DAD, Prud'Homme MJL, & Kalaska JF. (1994). Tactile activity in primate primary somatosensory cortex during active arm movements: Correlation with receptive field properties. *Journal of Neurophysiology*, 71(1), 161-172.

- Cohen RG, Biddle JC, & Rosenbaum DA. (2010). Manual obstacle avoidance takes into account visual uncertainty, motor noise, and biomechanical costs. *Experimental Brain Research*, 201(3), 587-592.
- Connell LA, Lincoln NB, & Radford KA. (2008). Somatosensory impairment after stroke: Frequency of different deficits and their recovery. *Clinical Rehabilitation*, 22(8), 758-767.
- Corradi-Dell'Acqua C, Tomasino B, & Fink GR. (2009). What is the position of an arm relative to the body? Neural correlates of body schema and body structural description. *Journal of Neuroscience*, 29(13), 4162-4171.
- Corser T. (1974). Temporal discrepancies in the electromyographic study of rapid movement. *Ergonomics*, 17(3), 389-400.
- Crago PE, Houk JC, & Hasan Z. (1976). Regulatory actions of human stretch reflex. *Journal of Neurophysiology*, 39(5), 925-935.
- Craighero L, Metta G, Sandini G, & Fadiga L. (2007). The mirror-neurons system: Data and models. *Progress in Brain Research*, 164, 39-59.
- Crammond DJ & Kalaska JF. (1996). Differential relation of discharge in primary motor cortex and premotor cortex to movements versus actively maintained postures during a reaching task. *Experimental Brain Research*, 108(1), 45-61.
- Crammond DJ & Kalaska JF. (2000). Prior information in motor and premotor cortex: Activity during the delay period and effect on pre-movement activity. *Journal of Neurophysiology*, 84(2), 986-1005.
- Cromwell FS. (1976). *Occupational therapist's manual for basic skill assessment: Primary prevocational evaluation*. Pasedena, CA: Fair Oaks Printing.
- Crow JL & Harmeling-van der Wel BC. (2008). Hierarchical properties of the motor function sections of the Fugl-Meyer Assessment Scale for people after stroke: A retrospective study. *Physical Therapy*, 88(12), 1554-1567.

- Culham JC & Valyear KF. (2006). Human parietal cortex in action. *Current Opinion in Neurobiology*, 16(2), 205-212.
- Dancause N, Barbay S, Frost SB, Plautz EJ, Chen D, Zoubina EV, Stowe AM, & Nudo RJ. (2005). Extensive cortical rewiring after brain injury. *The Journal of Neuroscience*, 25(44), 10167-10179.
- Dancause N, Ptito A, & Levin MF. (2002). Error correction strategies for motor behavior after unilateral brain damage: Short-term motor learning processes. *Neuropsychologia*, 40(8), 1313-1323.
- Day BL & Lyon IN. (2000). Voluntary modification of automatic arm movements evoked by motion of a visual target. *Experimental Brain Research*, 130(2), 159-68.
- de NAP Shelton F & Reding MJ. (2001). Effect of lesion location on upper limb motor recovery after stroke. *Stroke*, 32(1), 107-112.
- Dean J & Brüwer M. (1994). Control of human arm movements in two dimensions: Paths and joint control in avoiding simple linear obstacles. *Experimental Brain Research*, 97(3), 497-514.
- Dean J & Brüwer M. (1997). Control of human arm movements in two dimensions: Influence of pointer length on obstacle avoidance. *Journal of Motor Behavior*, 29(1), 47-63.
- DeJong SL & Lang CE. (2012). The bilateral movement condition facilitates maximal but not submaximal paretic-limb grip force in people with post-stroke hemiparesis. *Clinical Neurophysiology*, 123(8), 1616-1623.
- Desrosiers J, Bravo G, Hébert R, Dutil E, & Mercier L. (1994). Validation of the Box and Block Test as a measure of dexterity of elderly people: Reliability, validity, and norms studies. *Archives of Physical Medicine and Rehabilitation*, 75(7), 751-755.
- Desrosiers J, Héber R, Bravo G, & Dutil É. (1995). Upper-extremity motor co-ordination of healthy elderly people. *Age and Ageing*, 24(2), 108-112.

- Desmurget M & Grafton S. (2000). Forward modeling allows feedback control for fast reaching movements. *Trends in Cognitive Sciences*, 4(11), 423-431.
- Desmurget M, Epstein CM, Turner RS, Prablanc C, Alexander GE, & Grafton ST. (1999). Role of the posterior parietal cortex in updating reaching movements to a visual target. *Nature Neuroscience*, 2(6), 563-567.
- Dewald JP, Pope PS, Given JD, Buchanan TS, & Rymer WZ. (1995). Abnormal muscle coactivation patterns during isometric torque generation at the elbow and shoulder in hemiparetic subjects. *Brain: A Journal of Neurology*, 118(2), 495-510.
- Disbrow E, Roberts T, & Krubitzer L. (2000). Somatotopic organization of cortical fields in the lateral sulcus of Homo sapiens: Evidence for SII and PV. *Journal of Comparative Neurology*, 418(1), 1-21.
- Duffy FH & Burchfiel JL. (1971). Somatosensory system: Organizational hierarchy from single units in monkey area 5. *Science*, 172(3980), 273-275.
- Dum RP & Strick PL. (1993). Cingulate motor areas. In B Vogt, M Gabriel (Eds.), *Neurobiology of cingulate cortex and limbic thalamus: A comprehensive handbook* (pp.415-441). Boston: Birkhäuser.
- Duncan PW, Goldstein LB, Horner RD, Landsman PB, Samsa GP, & Matchar DB. (1994). Similar motor recovery of upper and lower extremities after stroke. *Stroke*, 25(6), 1181-1188.
- Edlow JA, Newman-Toker DE, & Savitz SI. (2008). Diagnosis and initial management of cerebellar infarction. *The Lancet Neurology*, 7(10), 951-964.
- Edwards DF, Hahn M, Baum C, & Dromerick AW. (2006). The impact of mild stroke on meaningful activity and life satisfaction. *Journal of Stroke and Cerebrovascular Diseases*, 15(4), 151-157.
- Egelko S, Gordon WA, Hibbard MR, Diller L, Lieberman A, Holliday R, Ragnarsson K, Shaver MS, & Orazem J. (1988). Relationship among CT scans, neurological exam, and

- neuropsychological test performance in right-brain-damaged stroke patients. *Journal of Clinical and Experimental Neuropsychology*, 10(5), 539-564.
- Ellis MD, Drogos J, Carmona C, Keller T, & Dewald JP. (2012). Neck rotation modulates flexion synergy torques, indicating an ipsilateral reticulospinal source for impairment in stroke. *Journal of Neurophysiology*, 108(11), 3096-3104.
- Ellis-Hill CS, Payne S, & Ward C. (2000). Self-body split: issues of identity in physical recovery following a stroke. *Disability & Rehabilitation*, 22(16), 725-733.
- Fang F & He S. (2005). Cortical responses to invisible objects in the human dorsal and ventral pathways. *Nature Neuroscience*, 8(10), 1380-1385.
- Fadiga L, Fogassi L, Pavesi G, & Rizzolatti G. (1995). Motor facilitation during action observation: A magnetic stimulation study. *Journal of Neurophysiology*, 73(6), 2608-2611.
- Faul F, Erdfelder E, Lang AG, & Buchner A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39(2), 175-191.
- Feeney DM & Baron JC. (1986). Diaschisis. *Stroke*, 17(5), 817-830.
- Feldman AG & Levin MF. (1995). The origin and use of positional frames of reference in motor control. *Behavioral and Brain Sciences*, 18(4), 723-744.
- Feys H, Hetebrij J, Wilms G, Dom R, & De Weerd W. (2000). Predicting arm recovery following stroke: Value of site of lesion. *Acta Neurologica Scandinavica*, 102(6), 371-377.
- Fisher CM. (1982). Lacunar strokes and infarcts: A review. *Neurology*, 32(8), 871-876.
- Fisk JD & Goodale MA. (1988). The effects of unilateral brain damage on visually guided reaching: Hemispheric differences in the nature of the deficit. *Experimental Brain Research*, 72(2), 425-435.

- Fitts PM. (1954). The information capacity of the human motor system in controlling the amplitude of movement. *Journal of Experimental Psychology*, 47(6), 381-391.
- Flanagan JR, Ostry DJ, & Feldman AG. (1993). Control of trajectory modifications in target-directed reaching. *Journal of Motor Behavior*, 25(3), 140-152.
- Flash T & Henis E. (1991). Arm trajectory modifications during reaching towards visual targets. *Journal of Cognitive Neuroscience*, 3(3), 220-230.
- Flash T & Hogan N. (1985). The coordination of arm movements: An experimentally confirmed mathematical model. *The Journal of Neuroscience*, 5(7), 1688-1703.
- Fogassi L, Gallese V, Buccino G, Craighero L, Fadiga L, & Rizzolatti G. (2001). Cortical mechanism for the visual guidance of hand grasping movements in the monkey: A reversible inactivation study. *Brain*, 124(3), 571-586.
- Fogassi L & Luppino G. (2005). Motor functions of the parietal lobe. *Current Opinion in Neurobiology*, 15(6), 626-631.
- Fredericks CM & Saladin LK. (Eds.). (1996). *Pathophysiology of the motor systems: Principles and clinical presentations*. Philadelphia, PA: FA Davis.
- Fries W, Danek A, Scheidtmann K, & Hamburger C. (1993). Motor recovery following capsular stroke: Role of descending pathways from multiple motor areas. *Brain*, 116(2), 369-382.
- Fugl-Meyer AR, Jääskö L, Layman I, Olsson S, & Steglind S. (1975). The post-stroke hemiplegic patient 1. A method for evaluation of physical performance. *Scandinavian Journal of Rehabilitation Medicine*, 7(1), 13-31.
- Furlan M, Marchal G, Derlon JM, Baron JC, & Viader F. (1996). Spontaneous neurological recovery after stroke and the fate of the ischemic penumbra. *Annals of Neurology*, 40(2), 216-226.

- Galea JM, Vazquez A, Pasricha N, de Xivry JJO, & Celnik P. (2011). Dissociating the roles of the cerebellum and motor cortex during adaptive learning: The motor cortex retains what the cerebellum learns. *Cerebral Cortex*, 21(8), 1761-1770.
- Gallese V, Fadiga L, Fogassi L, & Rizzolatti G. (1996). Action recognition in the premotor cortex. *Brain*, 119(2), 593-609.
- Gandevia SC. (1982). The perception of motor commands or effort during muscular paralysis. *Brain*, 105(1), 151-159.
- Ghafoury M, Archambault PS, Adamovich SV, & Feldman AG. (2002). Pointing movements may be produced in different frames of reference depending on the task demand. *Brain Research*, 929(1), 117-128.
- Gladstone DJ, Danells CJ, & Black SE. (2002). The Fugl-Meyer assessment of motor recovery after stroke: A critical review of its measurement properties. *Neurorehabilitation and Neural Repair*, 16(3), 232-240.
- Georgopoulos AP, Kalaska JF, & Massey JT. (1981). Spatial trajectories and reaction times of aimed movements: Effects of practice, uncertainty, and change in target location. *Journal of Neurophysiology*, 46(4), 725-743.
- Georgopoulos AP, Kalaska JF, Caminiti R, & Massey JT. (1982). On the relations between the direction of two-dimensional arm movements and cell discharge in primate motor cortex. *The Journal of Neuroscience*, 2(11), 1527-1537.
- Geyer S, Matelli M, Luppino G, & Zilles K. (2000). Functional neuroanatomy of the primate isocortical motor system. *Anatomy and Embryology*, 202(6), 443-474.
- Go AS, Mozaffarian D, Roger VL, Benjamin EJ, Berry JD, Blaha MJ, Dai S, Ford ES, Fox CS, Franco S, Fullerton HJ, Gillespie C, Hailpern SM, Heit JA, Howard VJ, Huffman MD, Judd SE, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Mackey RH, Magid DJ, Marcus GM, Marelli A, Matchar DB, McGuire DK, Mohler 3rd ER, Moy CS, Mussolino ME, Neumar RW, Nichol G, Pandey DK, Paynter NP, Reeves MJ, Sorlie PD, Stein J, Towfighi A, Turan TN, Virani SS, Wong ND, Woo D, Turner MB, & American

- Heart Association Statistics Committee and Stroke Statistics Subcommittee. (2014). Executive summary: Heart disease and stroke statistics--2014 update: A report from the American Heart Association. *Circulation*, 129(3), 399-410.
- Godschalk M, Lemon RN, Kuypers HG, & van der Steen J. (1985). The involvement of monkey premotor cortex neurones in preparation of visually cued arm movements. *Behavioral Brain Research*, 18(2), 143-157.
- Goodale MA. (2011). Transforming vision into action. *Vision Research*, 5(13), 1567-1587.
- Goodale MA, Pélisson D, & Prablanc C. (1986). Large adjustments in visually guided reaching do not depend on vision of the hand or perception of target displacement. *Nature*, 320(6064), 748-750.
- Goodale MA & Milner AD. (1992). Separate visual pathways for perception and action. *Trends in Neurosciences*, 15(1), 20-25.
- Gordon J, Ghilardi MF, & Ghez C. (1994). Accuracy of planar reaching movements. I. Independence of direction and extent variability. *Experimental Brain Research*, 99(1), 97-111.
- Goulet C, Arsenault AB, Bourbonnais D, Laramée MT, & Lepage Y. (1996). Effects of transcutaneous electrical nerve stimulation on H-reflex and spinal spasticity. *Scandinavian Journal of Rehabilitation Medicine*, 28(3), 169-176.
- Gowland C, Stratford P, Ward M, Moreland J, Torresin W, Van Hullenar S, Sanford J, Barreca S, Vanspall B, & Plews N. (1993). Measuring physical impairment and disability with the Chedoke-McMaster Stroke Assessment. *Stroke*, 24(1), 58-63.
- Grafton ST, Fagg AH, Woods RP, & Arbib MA. (1996). Functional anatomy of pointing and grasping in humans. *Cerebral Cortex*, 6(2), 226-237.
- Grafton ST, Woods RP, & Mazziotta JC. (1993). Within-arm somatotopy in human motor areas determined by positron emission tomography imaging of cerebral blood flow. *Experimental Brain Research*, 95(1), 172-176.

- Gréa H, Pisella L, Rossetti Y, Desmurget M, Tilikete C, Grafton S, Prablanc C, & Vighetto A. (2002). A lesion of the posterior parietal cortex disrupts on-line adjustments during aiming movements. *Neuropsychologia*, 40(13), 2471-2480.
- Grefkes C & Ward NS. (2014). Cortical reorganization after stroke: How much and how functional? *The Neuroscientist*, 20(1), 56-70.
- Grimme B, Lipinski J, & Schöner G. (2012). Naturalistic arm movements during obstacle avoidance in 3D and the identification of movement primitives. *Experimental Brain Research*, 222(3), 185-200.
- Halsband U, Ito N, Tanji J, & Freund HJ. (1993). The role of premotor cortex and the supplementary motor area in the temporal control of movement in man. *Brain*, 116(1), 243-266.
- Hamzei F, Rijntjes M, Dettmers C, Glauche V, Weiller C, & Buchel C. (2003). The human action recognition system and its relationship to Broca's area: An fMRI study. *Neuroimage*, 19(3), 637-644.
- Hare R, Rogers H, Lester H, McManus RJ, & Mant J. (2006). What do stroke patients and their carers want from community services? *Family Practice*, 23(1), 131-136.
- Haworth J, Young C, & Thornton E. (2009). The effects of an 'exercise and education' programme on exercise self-efficacy and levels of independent activity in adults with acquired neurological pathologies: An exploratory, randomized study. *Clinical Rehabilitation*, 23(4), 371-383.
- Hellström K, Lindmark B, Wahlberg B, & Fugl-Meyer AR. (2003). Self-efficacy in relation to impairments and activities of daily living disability in elderly patients with stroke: A prospective investigation. *Journal of Rehabilitation Medicine*, 35(5), 202-207.
- Herrmann N, Black SE, Lawrence J, Szekely C, & Szalai JP. (1998). The Sunnybrook Stroke Study: A prospective study of depressive symptoms and functional outcome. *Stroke*, 29(3), 618-624.

- Hoppenfeld S. (1976). *Physical examination of the spine and extremities*. New York: Appleton-Century-Crofts.
- Hoshi E & Tanji J. (2007). Distinctions between dorsal and ventral premotor areas: Anatomical connectivity and functional properties. *Current Opinion in Neurobiology*, 17(2), 234-242.
- Houk JC & Wise SP. (1995). Distributed modular architectures linking basal ganglia, cerebellum, and cerebral cortex: Their role in planning and controlling action. *Cerebral Cortex*, 5(2), 95-110.
- Howard LA & Tipper SP. (1997). Hand deviations away from visual cues: Indirect evidence for inhibition. *Experimental Brain Research*, 113(1), 144-152.
- Hsueh IP, Hsu MJ, Sheu CF, Lee S, Hsieh CL, & Lin JH. (2008). Psychometric comparisons of 2 versions of the Fugl-Meyer Motor Scale and 2 versions of the Stroke Rehabilitation Assessment of Movement. *Neurorehabilitation and Neural Repair*, 22(6), 737-744.
- Inman VT, Ralston HJ, De CM Saunders JB, Bertram Feinstein MB, & Wright Jr EW. (1952). Relation of human electromyogram to muscular tension. *Electroencephalography and Clinical Neurophysiology*, 4(2), 187-194.
- Iwata J, Shima K, Tanji J, & Mushiake H. (2013). Neurons in the cingulate motor area signal context-based and outcome-based volitional selection of action. *Experimental Brain Research*, 229(3), 407-417.
- Jackson SR, Jackson GM, & Rosicky J. (1995). Are non-relevant objects represented in working memory? The effect of non-target objects on reach and grasp kinematics. *Experimental Brain Research*, 102(3), 519-530.
- Jakobson LS, Archibald YM, Carey DP, & Goodale MA. (1991). A kinematic analysis of reaching and grasping movements in a patient recovering from optic ataxia. *Neuropsychologia*, 29(8), 803-809.

- James TW, Culham J, Humphrey GK, Milner AD, & Goodale MA. (2003). Ventral occipital lesions impair object recognition but not object-directed grasping: An fMRI study. *Brain*, 126(11), 2463-2475.
- Jankowska E & Edgley SA. (2006). How can corticospinal tract neurons contribute to ipsilateral movements? A question with implications for recovery of motor functions. *The Neuroscientist*, 12(1), 67-79.
- Jaric S & Latash ML. (1998). Learning a motor task involving obstacles by a multi-joint, redundant limb: Two synergies within one movement. *Journal of Electromyography and Kinesiology*, 8(3), 169-176.
- Jax SA, Rosenbaum DA, & Vaughan J. (2007). Extending Fitts' Law to manual obstacle avoidance. *Experimental Brain Research*, 180(4), 775-779.
- Jeannerod M, Decety J, & Michel F. (1994). Impairment of grasping movements following a bilateral posterior parietal lesion. *Neuropsychologia*, 32(4), 369-380.
- Jones EG & Powell TPS. (1970). An anatomical study of converging sensory pathways within the cerebral cortex of the monkey. *Brain*, 93(4), 793-820.
- Jones EG, Coulter JD, & Hendry SHC. (1978). Intracortical connectivity of architectonic fields in the somatic sensory, motor and parietal cortex of monkeys. *Journal of Comparative Neurology*, 181(2), 291-347.
- Jones F, Partridge C, & Reid F. (2008). The Stroke Self-Efficacy Questionnaire: Measuring individual confidence in functional performance after stroke. *Journal of Clinical Nursing*, 17(7b), 244-252.
- Jones F & Riazi A. (2011). Self-efficacy and self-management after stroke: A systematic review. *Disability and Rehabilitation*, 33(10), 797-810.
- Jørgensen HS, Nakayama H, Raaschou HO, Vive-Larsen J, Støier M, & Olsen TS. (1995). Outcome and time course of recovery in stroke. Part I: Outcome. The Copenhagen Stroke Study. *Archives of Physical Medicine and Rehabilitation*, 76(5), 399-405.

- Kaas JH, Nelson RJ, Sur M, Lin CS, & Merzenich MM. (1979). Multiple representations of the body within the primary somatosensory cortex of primates. *Science*, 204(4392), 521-523.
- Kakei S, Hoffman DS, & Strick PL. (2001). Direction of action is represented in the ventral premotor cortex. *Nature Neuroscience*, 4(10), 1020-1025.
- Kalaska JF. (2009). From intention to action: Motor cortex and the control of reaching movements. In *Progress in Motor Control* (pp. 139-178). Springer US.
- Kalaska JF, Cohen DA, Prud'Homme M, & Hyde ML. (1990). Parietal area 5 neuronal activity encodes movement kinematics, not movement dynamics. *Experimental Brain Research*, 80(2), 351-364.
- Kandel ER, Schwartz JH, Jessell TM, Siegelbaum SA, & Hudspeth AJ. (Eds.). (2012). *Principles of Neural Science*, 5th ed. New York: McGraw-Hill.
- Karnath HO & Perenin MT. (2005). Cortical control of visually guided reaching: Evidence from patients with optic ataxia. *Cerebral Cortex*, 15(10), 1561-1569.
- Kinsella G, Olver J, Ng K, Packer S, & Stark R. (1993). Analysis of the syndrome of unilateral neglect. *Cortex*, 29(1), 135-140.
- Knaut LA, Subramanian SK, McFadyen BJ, Bourbonnais D, & Levin MF. (2009). Kinematics of pointing movements made in a virtual versus a physical 3-dimensional environment in healthy and stroke subjects. *Archives of Physical Medicine and Rehabilitation*, 90(5), 793-802.
- Kohler E, Keysers C, Umiltà MA, Fogassi L, Gallese V, & Rizzolatti G. (2002). Hearing sounds, understanding actions: Action representation in mirror neurons. *Science*, 297(5582), 846-848.
- Korpershoek C, van der Bijl J, & Hafsteinsdóttir TB. (2011). Self-efficacy and its influence on recovery of patients with stroke: A systematic review. *Journal of Advanced Nursing*, 67(9), 1876-1894.

- Krishnamoorthy V, Scholz JP, & Latash ML. (2007). The use of flexible arm muscle synergies to perform an isometric stabilization task. *Clinical Neurophysiology*, 118(3), 525-537.
- Kwakkel G, Kollen BJ, van der Grond J, & Prevo AJ. (2003). Probability of regaining dexterity in the flaccid upper limb: Impact of severity of paresis and time since onset in acute stroke. *Stroke*, 34(9), 2181-2186.
- Kumral E, Bayukem G, Evyapan D, & Yuntun N. (2002). Spectrum of anterior cerebral artery territory infarction: Clinical and MRI findings. *European Journal of Neurology*, 9(6), 615–624.
- Kunesch E, Binkofski F, Steinmetz H, & Freund HJ. (1995). The pattern of motor deficits in relation to the site of stroke lesions. *European Neurology*, 35(1), 20-26.
- Lacquaniti F & Soechting JF. (1982). Coordination of arm and wrist motion during a reaching task. *The Journal of Neuroscience*, 2(4), 399-408.
- Lamme VA & Roelfsema PR. (2000). The distinct modes of vision offered by feedforward and recurrent processing. *Trends in Neurosciences*, 23(11), 571-579.
- Lance JW. (1980). Pathophysiology of spasticity and clinical experience with baclofen. In RG Feldman, RR Young , & WP Koella (Eds.), *Spasticity: Disordered Motor Control* (pp. 185–220). Chicago, IL: Year Book Medical Publisher.
- Lang CE & Beebe JA. (2007). Relating movement control at 9 upper extremity segments to loss of hand function in people with chronic hemiparesis. *Neurorehabilitation and Neural Repair* 21(3), 279-291.
- Lang CE, Wagner JM, Edwards DF, & Dromerick AW. (2007). Upper extremity use in people with hemiparesis in the first few weeks after stroke. *Journal of Neurologic Physical Therapy*, 31(2), 56-63.
- Latash ML & Anson JG. (1996). What are “normal movements” in atypical populations? *Behavioral and Brain Sciences*, 19(1), 55-68.

- Lawrence DG & Kuypers HG. (1968a). The functional organization of the motor system in the monkey. I. The effects of bilateral pyramidal lesions. *Brain*, 91(1), 1-14.
- Lawrence DG & Kuypers HG. (1968b). The functional organization of the motor system in the monkey. II. The effects of lesions of the descending brain-stem pathways. *Brain*, 91(1), 15-36.
- Lemon RN. (2008). Descending pathways in motor control. *Annual Review of Neuroscience*, 31, 195-218.
- Lev EL & Owen SV. (1996). A measure of self-care self-efficacy. *Research in Nursing & Health*, 19(5), 421-429.
- Levin MF. (1996). Interjoint coordination during pointing movements is disrupted in spastic hemiparesis. *Brain*, 119(1), 281-293.
- Levin MF, Desrosier, J, Beauchemin D, Bergeron N, & Rochette A. (2004). Development and validation of a scale for rating motor compensations used for reaching in patients with hemiparesis: The reaching performance scale. *Physical Therapy*, 84(1), 8-22.
- Levin MF & Hui-Chan CW. (1992). Relief of hemiparetic spasticity by TENS is associated with improvement in reflex and voluntary motor functions. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 85(2), 131-142.
- Levin MF, Kleim JA, & Wolf SL. (2009). What do motor “recovery” and “compensation” mean in patients following stroke? *Neurorehabilitation and Neural Repair*, 23(4), 313-319.
- Levin MF, Michaelsen SM, Cirstea CM, & Roby-Brami A. (2002). Use of the trunk for reaching targets placed within and beyond the reach in adult hemiparesis. *Experimental Brain Research*, 143(2), 171-180.
- Levin MF, Selles RW, Verheul MHG, & Meijer OG. (2000). Deficits in the coordination of agonist and antagonist muscles in stroke patients: Implications for normal motor control. *Brain Research*, 853(2), 352-369.

- Lin JH, Hsueh IP, Sheu CF, & Hsieh CL. (2004). Psychometric properties of the sensory scale of the Fugl-Meyer Assessment in stroke patients. *Clinical Rehabilitation*, 18(4), 391-397.
- Lo EH, Dalkara T, & Moskowitz MA. (2003). Mechanisms, challenges and opportunities in stroke. *Nature Reviews Neuroscience*, 4(5), 399-414.
- Lo EH, Moskowitz MA, & Jacobs TP. (2005). Exciting, radical, suicidal how brain cells die after stroke. *Stroke*, 36(2), 189-192.
- Lo EH. (2008). A new penumbra: transitioning from injury into repair after stroke. *Nature medicine*, 14(5), 497-500.
- Luppino G, Matelli M, Camarda RM, Gallese V, & Rizzolatti G. (1991). Multiple representations of body movements in mesial area 6 and the adjacent cingulate cortex: An intracortical microstimulation study in the macaque monkey. *Journal of Computational Neurology*, 311(4), 463–482.
- Mackay J & Mensah GA. (2004). *The atlas of heart disease and stroke*. Geneva: World Health Organization.
- Mani S, Mutha PK, Przybyla A, Haaland KY, Good DC, & Sainburg RL. (2013). Contralesional motor deficits after unilateral stroke reflect hemisphere-specific control mechanisms. *Brain*, 136, 1288-1303.
- Manto M, Bower JM, Conforto AB, Delgado-García JM, da Guarda SNF, Gerwig M, Habas C, Hagura N, Ivry RB, Mariën P, Molinari M, Naito E, Nowak DA, Taib NOB, Pelisson D, Tesche CD, Tilikete C, & Timmann D. (2012). Consensus paper: Roles of the cerebellum in motor control – The diversity of ideas on cerebellar involvement in movement. *The Cerebellum*, 11(2), 457-487.
- Mark LS, Nemeth K, Gardner D, Dainoff MJ, Paasche J, Duffy M, & Grandt K. (1997). Postural dynamics and the preferred critical boundary for visually guided reaching. *Journal of Experimental Psychology: Human Perception and Performance*, 23(5), 1365–1379.

- Marsh NV & Kersel DA. (1993). Screening tests for visual neglect following stroke. *Neuropsychological Rehabilitation*, 3(3), 245-257.
- Matelli M & Luppino G. (1996). Thalamic input to mesial and superior area 6 in the macaque monkey. *Journal of Comparative Neurology*, 372(1), 59-87.
- Mayo NE, Wood-Dauphinee S, Ahmed S, Gordon C, Higgins J, McEwen S, & Salbach N. (1999). Disablement following stroke. *Disability and Rehabilitation*, 21(5-6), 258-268.
- Mayo NE, Wood-Dauphinee S, Côté R, Durcan L, & Carlton J. (2002). Activity, participation, and quality of life 6 months poststroke. *Archives of Physical Medicine and Rehabilitation*, 83(8), 1035-1042.
- Michaelson SM, Dannenbaum R, & Levin MF. (2006). Task-specific training with trunk restraint on arm recovery in stroke randomized control trial. *Stroke*, 37(1), 186-192.
- Michaelson SM, Jacobs S, Roby-Brami A, & Levin MF. (2004). Compensation for distal impairments of grasping in adults with hemiparesis. *Experimental Brain Research*, 157(2), 162-173.
- Michielsen ME, de Niet M, Ribbers GM, Stam, HJ, & Bussmann JB. (2009). Evidence of a logarithmic relationship between motor capacity and actual performance in daily life of the paretic arm following stroke. *Journal of Rehabilitation Medicine*, 41(5), 327-331.
- Michielsen ME, Selles RW, Stam HJ, Ribbers GM, & Bussmann JB. (2012). Quantifying nonuse in chronic stroke patients: A study into paretic, nonparetic, and bimanual upper-limb use in daily life. *Archives of Physical Medicine and Rehabilitation*, 93(11), 1975-1981.
- Mihaltchev P, Archambault PS, Feldman AG, & Levin MF. (2005). Control of double-joint arm posture in adults with unilateral brain damage. *Experimental Brain Research*, 163(4), 468-486.
- Milner AD, Dijkerman HC, McIntosh RD, Rossetti Y, & Pisella L. (2003). Delayed reaching and grasping in patients with optic ataxia. *Progress in Brain Research*, 142, 225-242.

- Milner AD & Goodale MA. (2008). Two visual systems re-viewed. *Neuropsychologia*, 46(3), 774-785.
- Milner AD, Paulignan Y, Dijkerman HC, Michel F, & Jeannerod M. (1999). A paradoxical improvement of misreaching in optic ataxia: New evidence for two separate neural systems for visual localization. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 266(1434), 2225-2229.
- Milner AD, Perrett DI, Johnston RS, Benson PJ, Jordan TR, Heeley DW, Bettucci D, Mortara F, Mutani R, Terazzi E, & Davidson DLW. (1991). Perception and action in 'visual form agnosia'. *Brain*, 114(1), 405-428.
- Milner-Brown HS & Stein RB. (1975). The relation between the surface electromyogram and muscular force. *The Journal of Physiology*, 246(3), 549-569.
- Mon-Williams M, Tresilian JR, Coppard VL, & Carson RG. (2001). The effect of obstacle position on reach-to-grasp movements. *Experimental Brain Research*, 137(3-4), 497-501.
- Morasso P. (1981). Spatial control of arm movements. *Experimental Brain Research*, 42(2), 223-227.
- Moreland J, Gowland C, Van Hullenar S, & Huijbregts M. (1993). Theoretical basis of the Chedoke-McMaster Stroke Assessment. *Physiotherapy Canada*, 45, 231-238.
- Mountcastle VB, Lynch JC, Georgopoulos A, Sakata H, & Acuna C. (1975). Posterior parietal association cortex of the monkey: Command functions for operations within extrapersonal space. *Journal of Neurophysiology*, 38(4), 871-908.
- Mulliken GH, Musallam S, & Andersen RA. (2008). Forward estimation of movement state in posterior parietal cortex. *Proceedings of the National Academy of Sciences*, 105(24), 8170-8177.
- Murayama T, Numata K, Kawakami T, Tosaka T, Oga M, Oka N, Katano M, Takasugi J, & Shimizu E. (2011). Changes in the brain activation balance in motor-related areas after

- constraint-induced movement therapy: A longitudinal fMRI study. *Brain Injury*, 25(11), 1047-1057.
- Murphy TH & Corbett D. (2009). Plasticity during stroke recovery: From synapse to behaviour. *Nature Reviews Neuroscience*, 10(12), 861-872.
- Musampa NK, Mathieu PA, & Levin MF. (2007). Relationship between stretch reflex thresholds and voluntary arm muscle activation in patients with spasticity. *Experimental Brain Research*, 181(4), 579-593.
- Nasreddine ZS, Phillips NA, Bédirian V, Charbonneau S, Whitehead V, Collin I, Cummings JL, & Chertkow H. (2005). The Montreal Cognitive Assessment, MoCA: A brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, 53(4), 695-699.
- Newell KM & Houk JC. (1983). Speed and accuracy of compensatory responses to limb disturbances. *Journal of Experimental Psychology: Human Perception and Performance*, 9(1), 58-74.
- Nijland R, van Wegen E, Verbunt J, van Wijk R, van Kordelaar J, & KwakkeL G. (2010). A comparison of two validated tests for upper limb function after stroke: The Wolf Motor Function Test and the Action Research Arm Test. *Journal of Rehabilitation Medicine*, 42(7), 694-696.
- Nudo RJ. (2013). Recovery after brain injury: Mechanisms and principles. *Frontiers in Human Neuroscience*, 7, 1-14.
- Nudo RJ, Wise BM, SiFuentes F, & Milliken GW. (1996). Neural substrates for the effects of rehabilitative training on motor recovery after ischemic infarct. *Science*, 272(5269), 1791-1794.
- O'Brien MD. (1979). Ischemic cerebral edema. A review. *Stroke*, 10(6), 623-628.
- Oldfield RC. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia*, 9(1), 97-113.

- Patten C, Lexell J, & Brown HE. (2004). Weakness and strength training in persons with poststroke hemiplegia: rationale, method, and efficacy. *Journal of Rehabilitation Research and Development*, 41(3), 293-312.
- Patten J. (1996). Neurological differential diagnosis (2nd ed.). London: Springer-Verlag.
- Paus T. (2001). Primate anterior cingulate cortex: Where motor control, drive and cognition interface. *Nature Reviews Neuroscience*, 2(6), 417-424.
- Pearson RCA & Powell TPS. (1985). The projection of the primary somatic sensory cortex upon area 5 in the monkey. *Brain Research Reviews*, 9(1), 89-107.
- Penfield W & Boldrey E. (1937). Somatic motor and sensory representation in the cerebral cortex of man as studied by electrical stimulation. *Brain: A Journal of Neurology*, 60, 389-443.
- Pisella L, Gréa H, Tilikete C, Vighetto A, Desmurget M, Rode G, Boisson D, & Rossetti Y. (2000). An 'automatic pilot' for the hand in human posterior parietal cortex: Toward reinterpreting optic ataxia. *Nature Neuroscience*, 3(7), 729-736.
- Platz T, Bock S, & Prass K. (2001). Reduced skilfulness of arm motor behaviour among motor stroke patients with good clinical recovery: Does it indicate reduced automaticity? Can it be improved by unilateral or bilateral training? A kinematic motion analysis study. *Neuropsychologia*, 39(7), 687-698.
- Platz T & Denzler P. (2002). Do psychological variables modify motor recovery among patients with mild arm paresis after stroke or traumatic brain injury who receive the Arm Ability Training? *Restorative Neurology and Neuroscience*, 20(1), 37-49.
- Platz T, Pinkowski C, van Wijck F, Kim IH, di Bella P, & Johnson G. (2005). Reliability and validity of arm function assessment with standardized guidelines for the Fugl-Meyer Test, Action Research Arm Test and Box and Block Test: A multicentre study. *Clinical Rehabilitation*, 19(4), 404-411.

- Plummer P, Morris ME, & Dunai J. (2003). Assessment of unilateral neglect. *Physical Therapy*, 83(8), 732-740.
- Potvin AR & Tourtelotte WW. (1985). *Quantitative examination of neurologic functions*. Boca Raton, FL: CRC Press.
- Prablanc C & Martin O. (1992). Automatic control during hand reaching at undetected two-dimensional target displacements. *Journal of Neurophysiology*, 67(2), 455-469.
- Prado J, Clavagnier S, Otzenberger H, Scheiber C, Kennedy H, & Perenin MT. (2005). Two cortical systems for reaching in central and peripheral vision. *Neuron*, 48(5), 849-858.
- Prablanc C, Desmurget M, & Gréa H. (2003). Neural control of on-line guidance of hand reaching movements. *Progress in Brain Research*, 142, 155-170.
- Prange GB, Jannink MJ, Stienen AH, van der Kooij H, IJzerman MJ, & Hermens HJ. (2010). An explorative, cross-sectional study into abnormal muscular coupling during reach in chronic stroke patients. *Journal of Neuroengineering and Rehabilitation*, 7(14).
- Prud'Homme MJ, Cohen DA, & Kalaska JF. (1994). Tactile activity in primate primary somatosensory cortex during active arm movements: Cytoarchitectonic distribution. *Journal of Neurophysiology*, 71(1), 173-181.
- Prud'Homme MJ & Kalaska JF. (1994). Proprioceptive activity in primate primary somatosensory cortex during active arm reaching movements. *Journal of Neurophysiology*, 72(5), 2280-2301.
- Purves D, Augustine GJ, Fitzpatrick D, Katz LC, Lamantia A-S, McNamara JO, & Williams SM. (2001). *Neuroscience (2nd ed.)*. Sunderland, MA: Sinauer.
- Raghavan P, Santello M, Gordon AM, & Krakauer JW. (2010). Compensatory motor control after stroke: An alternative joint strategy for object-dependent shaping of hand posture. *Journal of Neurophysiology*, 103(6), 3034-3043.

- Ralston HJ, Todd FN, & Inman VT. (1976). Comparison of electrical activity and duration of tension in the human rectus femoris muscle. *Electromyography and Clinical Neurophysiology*, 16(2-3), 271-280.
- Rand D & Eng JJ. (2012). Disparity between functional recovery and daily use of the upper and lower extremities during subacute stroke rehabilitation. *Neurorehabilitation and Neural Repair*, 26(1), 76-84.
- Rand D, Kizony R, Feintuch U, Katz N, Josman N, & Weiss PLT. (2005). Comparison of two VR platforms for rehabilitation: Video capture versus HMD. *Presence: Teleoperators and Virtual Environments*, 14(2), 147-160.
- Rathore SS, Hinn AR, Cooper LS, Tyroler HA, & Rosamond WD. (2002). Characterization of incident stroke signs and symptoms: Findings from the Atherosclerosis Risk In Communities (ARIC) study. *Stroke*, 33(11), 2718-2721.
- Rawley JB & Constantinidis C. (2009). Neural correlates of learning and working memory in the primate posterior parietal cortex. *Neurobiology of Learning and Memory*, 91(2), 129-138.
- Rehme AK, Fink GR, Von Cramon DY, & Grefkes C. (2010). The role of the contralesional motor cortex for motor recovery in the early days after stroke assessed with longitudinal FMRI. *Cerebral Cortex*, 21(4), 756-768.
- Reisman DS & Scholz JP. (2003). Aspects of joint coordination are preserved during pointing in persons with post-stroke hemiparesis. *Brain*, 126(11), 2510-2527.
- Riehle A & Requin J. (1989). Monkey primary motor and premotor cortex: Single-cell activity related to prior information about direction and extent of an intended movement. *Journal of Neurophysiology*, 61(3), 534-549.
- Rizzolatti G & Craighero L. (2004). The mirror-neuron system. *Annual Review of Neuroscience*, 27, 169-192.
- Rizzolatti G, Fadiga L, Gallese V, & Fogassi L. (1996). Premotor cortex and the recognition of motor actions. *Cognitive Brain Research*, 3(2), 131-141.

- Rizzolatti G, Ferrari PF, Rozzi S, & Fogassi L. (2006). The inferior parietal lobule: Where action becomes perception. In *Novartis Foundation Symposium* (Vol. 270, p. 129). Chichester, NY: John Wiley.
- Rizzolatti G, Fogassi L, & Gallese V. (2002). Motor and cognitive functions of the ventral premotor cortex. *Current Opinion in Neurobiology*, 12(2), 149-154.
- Robinson-Smith G, Johnston MV, & Allen J. (2000). Self-care self-efficacy, quality of life, and depression after stroke. *Archives of Physical Medicine and Rehabilitation*, 81(4), 460-464.
- Roby-Brami A, Feydy A, Combeaud M, Biryukova EV, Bussel B, & Levin MF. (2003). Motor compensation and recovery for reaching in stroke patients. *Acta neurologica Scandinavica*, 107(5), 369-381.
- Rode G, Rossetti Y, & Boisson D. (1996). Inverse relationship between sensation of effort and muscular force during recovery from pure motor hemiplegia: A single-case study. *Neuropsychologia*, 34(2), 87-95.
- Roh J, Rymer WZ, Perreault EJ, Yoo SB, & Beer RF. (2013). Alterations in upper limb muscle synergy structure in chronic stroke survivors. *Journal of Neurophysiology*, 109(3), 768-781.
- Ropper AH. (1984). Brain edema after stroke: clinical syndrome and intracranial pressure. *Archives of Neurology*, 41(1), 26-29.
- Roy CW, Sands MR, & Hill LD. (1994). Shoulder pain in acutely admitted hemiplegics. *Clinical Rehabilitation*, 8(4), 334-340.
- Ruber T, Schlaug G & Lindenberg R. (2012). Compensatory role of the cortico-rubro-spinal tract in motor recovery after stroke. *Neurology*, 79, 515-522.
- Rubio KB & Van Deusen J. (1995). Relation of perceptual and body image dysfunction to activities of daily living of persons after stroke. *American Journal of Occupational Therapy*, 49(6), 551-559.

- Ryerson S & Levit K. (1987). The shoulder in hemiplegia. In *Physical therapy of the shoulder* (pp. 105-31). New York: Churchill Livingstone.
- Sabes PN. (2000). The planning and control of reaching movements. *Current Opinion in Neurobiology*, 10(6), 740-746.
- Sabes PN, Jordan MI, & Wolpert DM. (1998). The role of inertial sensitivity in motor planning. *The Journal of Neuroscience*, 18(15), 5948-5957.
- Sackett D, Richardson W, Rosenberg W, & Haynes R. (1997). *Evidence-based medicine: How to practice and teach EBM*. New York: Churchill Livingstone.
- Sadato N, Yonekura Y, Waki A, Yamada H, & Ishii Y. (1997). Role of the supplementary motor area and the right premotor cortex in the coordination of bimanual finger movements. *The Journal of Neuroscience*, 17(24), 9667-9674.
- Saeki S & Toyonaga T. (2010). Determinants of early return to work after first stroke in Japan. *Journal of Rehabilitation Medicine*, 42(3), 254-258.
- Sakata H, Takaoka Y, Kawarasaki A, & Shibutani H. (1973). Somatosensory properties of neurons in the superior parietal cortex (area 5) of the rhesus monkey. *Brain Research*, 64, 85-102.
- Sakata H, Taira M, Murata A, & Mine S. (1995). Neural mechanisms of visual guidance of hand action in the parietal cortex of the monkey. *Cerebral Cortex*, 5(5), 429-438.
- Salenius S, Portin K, Kajola M, Salmelin R, & Hari R. (1997). Cortical control of human motoneuron firing during isometric contraction. *Journal of Neurophysiology*, 77(6), 3401-3405.
- Saling M, Alberts J, Stelmach GE, & Bloedel JR. (1998). Reach-to-grasp movements during obstacle avoidance. *Experimental Brain Research*, 118(2), 251-258.
- Scott SH. (1999). Apparatus for measuring and perturbing shoulder and elbow joint positions and torques during reaching. *Journal of Neuroscience Methods*, 89, 119-127.

- Schaefer SY, Mutha PK, Haaland KY, & Sainburg RL. (2012). Hemispheric specialization for movement control produces dissociable differences in online corrections after stroke. *Cerebral Cortex*, 22(6), 1407-1419.
- Scheidt RA & Stoeckmann T. (2007). Reach adaptation and final position control amid environmental uncertainty after stroke. *Journal of Neurophysiology*, 97(4), 2824-2836.
- Schenkenberg T, Bradford DC, & Ajax ET. (1980). Line bisection and unilateral visual neglect in patients with neurologic impairment. *Neurology*, 30, 509-517.
- Schmitz TJ. (2001). Coordination assessment. In SB O'Sullivan & TJ Schmitz (Eds.), *Physical Rehabilitation: Assessment and Treatment* (4th ed.). Philadelphia, PA: FA Davis.
- Schmuckler MA. (2001). What is ecological validity? A dimensional analysis. *Infancy*, 2(4), 419-436.
- Scholz JP & Schöner G. (1999). The uncontrolled manifold concept: Identifying control variables for a functional task. *Experimental Brain Research*, 126(3), 289-306.
- Seitz RJ, Azari NP, Knorr U, Binkofski F, Herzog H, & Freund HJ. (1999). The role of diaschisis in stroke recovery. *Stroke*, 30(9), 1844-1850.
- Seitz RJ, Nickel J, & Azari NP. (2006). Functional modularity of the medial prefrontal cortex: Involvement in human empathy. *Neuropsychology*, 20(6), 743-751.
- Sethi A, Patterson T, McGuirk T, Patten C, Richards LG, & Stergiou N. (2013). Temporal structure of variability decreases in upper extremity movements post stroke. *Clinical Biomechanics (Bristol, Avon)*, 28(2), 134-139.
- Sheikh JI & Yesavage JA. (1986). Geriatric Depression Scale (GDS): Recent evidence and development of a shorter version. In *Clinical Gerontology: A Guide to Assessment and Intervention* (pp. 165-173). New York: The Haworth Press.
- Shima K & Tanji J. (1998). Role for cingulate motor area cells in voluntary movement selection based on reward. *Science*, 282(5392), 1335-1338.

- Shmuelof L & Krakauer JW. (2011). Are we ready for a natural history of motor learning? *Neuron*, 72(3), 469-476.
- Shumway-Cook A & Woollacott MH. (2012). *Motor control: Translating research into clinical practice* (4th ed.). Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins.
- Schwartz AB. (1994). Distributed motor processing in cerebral cortex. *Current Opinion in Neurobiology*, 4(6), 840-846.
- Soechting JF. (1984). Effect of target size on spatial and temporal characteristics of a pointing movement in man. *Experimental Brain Research*, 54(1), 121-132.
- Soechting JF & Lacquaniti F. (1981). Invariant characteristics of a pointing movement in man. *The Journal of Neuroscience*, 1(7), 710-720.
- Soechting JF & Lacquaniti F. (1983). Modification of trajectory of a pointing movement in response to a change in target location. *Journal of Neurophysiology*, 49(2), 548-564.
- Stephan KM, Binkofski F, Halsband U, Dohle C, Wunderlich G, Schnitzler A, Tass P, Posse S, Herzog H, Sturm V, Zilles K, Seitz RJ, & Freund H-J. (1999). The role of ventral medial wall motor areas for bimanual coordination: A combined lesion and activation study. *Brain*, 122(2), 351–368.
- Stroke Engine Team. (2013). *Stroke Engine Assess*, Retrieved from <http://strokengine.ca/assess/>
- Subramanian SK & Levin MF. (2011). Viewing medium affects arm motor performance in 3D virtual environments. *Journal of NeuroEngineering and Rehabilitation*, 8(36).
- Subramanian SK, Yamanaka J, Chilingaryan G, & Levin MF. (2010). Validity of movement pattern kinematics as measures of arm motor impairment poststroke. *Stroke*, 41(10), 2303-2308.

- Subramanian SK, Chilingaryan G, Sveistrup H, & Levin MF. (2014). Depression and cognitive deficits influence use of feedback for motor learning and recovery in chronic stroke. *Neurorehabilitation and Neural Repair*, 28(4), NP18.
- Summers JJ, Kagerer FA, Garry MI, Hiraga CY, Loftus A, & Cauraugh JH. (2007). Bilateral and unilateral movement training on upper limb function in chronic stroke patients: A TMS study. *Journal of the Neurological Sciences*, 252(1), 76-82.
- Swaine BR & Sullivan SJ. (1992). Relation between clinical and instrumented measures of motor coordination in traumatically brain injured persons. *Archives of Physical Medicine and Rehabilitation*, 73(1), 55-59.
- Swaine BR & Sullivan SJ. (1993). Reliability of the scores for the finger-to-nose test in adults with traumatic brain injury. *Physical Therapy*, 73(2), 71-78.
- Takada M, Tokuno H, Hamada I, Inase M, Ito Y, Imanishi M, Hasegawa N, Akazawa T, Hatanaka N, & Nambu A. (2001). Organization of inputs from cingulate motor areas to basal ganglia in macaque monkey. *European Journal of Neuroscience*, 14(10), 1633-1650.
- Takeuchi N, Chuma T, Matsuo Y, Watanabe I, & Ikoma K. (2005). Repetitive transcranial magnetic stimulation of contralesional primary motor cortex improves hand function after stroke. *Stroke*, 36(12), 2681-2686.
- Tanji J. (1996). New concepts of the supplementary motor area. *Current Opinion in Neurobiology*, 6(6), 782-787.
- Taub E, Crago JE, Burgio LD, Groomes TE, Cook EW, DeLuca SC, & Miller NE. (1994). An operant approach to rehabilitation medicine: Overcoming learned nonuse by shaping. *Journal of the Experimental Analysis of Behavior*, 61(2), 281-293.
- Taub E, Miller NE, Novack TA, Cook 3rd EW, Fleming WC, Nepomuceno CS, Connell JS, & Crago JE. (1993). Technique to improve chronic motor deficit after stroke. *Archives of Physical Medicine and Rehabilitation*, 74(4), 347-354.

- Thach WT. (1998). A role for the cerebellum in learning movement coordination. *Neurobiology of learning and memory*, 70(1), 177-188.
- Teasell R, Richardson M, Allen L, & Hussein N. (2013). Upper extremity interventions. In *Evidence-Based Review of Stroke Rehabilitation (Evidence review 10)*. Retrieved from <http://www.ebrsr.com/evidence-review/10-upper-extremity-interventions>
- Tellier M & Rochette A. (2009). Falling through the cracks: a literature review to understand the reality of mild stroke survivors. *Topics in Stroke Rehabilitation*, 16(6), 454-462.
- Tettamanti M, Buccino G, Saccuman MC, Gallese V, Danna M, Scifo P, Fazio F, Rizzolatti G, Cappa SF, & Perani D. (2005). Listening to action-related sentences activates fronto-parietal motor circuits. *Journal of Cognitive Neuroscience*, 17(2), 273-281.
- Tresilian J R. (1998). Attention in action or obstruction of movement? A kinematic analysis of avoidance behavior in prehension. *Experimental Brain Research*, 120(3), 352-368.
- Torres E & Andersen R. (2006). Space–time separation during obstacle-avoidance learning in monkeys. *Journal of Neurophysiology*, 96(5), 2613-2632.
- Umphred DA, Lazaro RT, Roller M, & Burton G. (Eds.). (2013). *Neurological rehabilitation* (6th ed.). St. Louis, MI: Elsevier Health Sciences.
- Ustinova KI, Goussev VM, Balasubramaniam R, & Leven MF. (2004). Disruption of coordination between arm, trunk, and center of pressure displacement in patients with hemiparesis. *Motor Control-Champaign*-, 8(2), 139-159.
- Uswatte G, Taub E, Morris D, Light K, & Thompson PA. (2006). The Motor Activity Log-28: Assessing daily use of the hemiparetic arm after stroke. *Neurology*, 67(7), 1189-1194.
- Uswatte G, Taub E, Morris D, Vignolo M, & McCulloch K. (2005). Reliability and validity of the upper-extremity Motor Activity Log-14 for measuring real-world arm use. *Stroke*, 36(11), 2493-2496.

- Van der Lee JH, Beckerman H, Knol DL, De Vet HCW, & Bouter LM. (2004). Clinimetric properties of the Motor Activity Log for the assessment of arm use in hemiparetic patients. *Stroke*, 35(6), 1410-1414.
- Vaughan J, Barany DA, Sali AW, Jax SA, & Rosenbaum DA. (2010). Extending Fitts' Law to three-dimensional obstacle-avoidance movements: Support for the posture-based motion planning model. *Experimental Brain Research*, 207(1-2), 133-138.
- Viau A, Feldman AG, McFadyen BJ, & Levin MF. (2004). Reaching in reality and virtual reality: A comparison of movement kinematics in healthy subjects and in adults with hemiparesis. *Journal of NeuroEngineering and Rehabilitation*, 1(11).
- Vidoni ED, Acerra NE, Dao E, Meehan SK, & Boyd LA. (2010). Role of the primary somatosensory cortex in motor learning: An rTMS study. *Neurobiology of Learning and Memory*, 93(4), 532-539.
- Weiller C, Chollet F, Friston KJ, Wise RJ, & Frackowiak RS. (1992). Functional reorganization of the brain in recovery from striatocapsular infarction in man. *Annals of Neurology*, 31(5), 463-472.
- Werhahn KJ, Conforto AB, Kadom N, Hallett M, & Cohen LG. (2003). Contribution of the ipsilateral motor cortex to recovery after chronic stroke. *Annals of Neurology*, 54(4), 464-472.
- Whitall J, Savin DN Jr, Harris-Love M, & Waller SM. (2006). Psychometric properties of a modified Wolf Motor Function Test for people with mild and moderate upper-extremity hemiparesis. *Archives of Physical Medicine and Rehabilitation*, 87(5):656-660.
- Wiesendanger M. (1990). Weakness and the upper motoneurone syndrome: A critical pathophysiological appraisal. In A Berardelli & R Benecke (Eds.), *Motor disturbances II* (pp. 319-331). London: Academic Press.
- Williams PT, Kim S, & Martin JH. (2014). Postnatal maturation of the red nucleus motor map depends on rubrospinal connections with forelimb motor pools. *Journal of Neuroscience*, 34(12), 4432-4441.

- Winship IR & Murphy TH. (2009). Remapping the somatosensory cortex after stroke: Insight from imaging the synapse to network. *The Neuroscientist*, 15(5), 507-524.
- Wolf SL, Catlin PA, Ellis M, Archer AL, Morgan B, & Piacentino A. (2001). Assessing Wolf Motor Function Test as outcome measure for research in patients after stroke. *Stroke*, 32(7), 1635-1639.
- Woodbury ML, Velozo CA, Richards LG, Duncan PW, Studenski S, & Lai S. (2008). Longitudinal stability of the Fugl-Meyer Assessment of the Upper Extremity. *Archives of Physical Medicine and Rehabilitation*, 89(8), 1563-1569.
- Wood-Dauphinee SL, Williams JI, & Shapiro SH. (1990). Examining outcome measures in a clinical study of stroke. *Stroke*, 21(5), 731-739.
- Wu CY, Chuang LL, Lin KC, Chen HC, & Tsay PK. (2011). Randomized trial of distributed constraint-induced therapy versus bilateral arm training for the rehabilitation of upper-limb motor control and function after stroke. *Neurorehabilitation and Neural Repair*, 25(2), 130-139.
- Wu CY, Fu T, Lin KC, Feng CT, Hsieh KP, Yu HW, Lin CH, Hsieh CJ, & Ota H. (2011). Assessing the streamlined Wolf Motor Function Test as an outcome measure for stroke rehabilitation. *Neurorehabilitation and Neural Repair*, 25(2), 194-199.
- Yeo SS & Jang SH. (2010). Changes in red nucleus after pyramidal tract injury in patients with cerebral infarct. *Neurorehabilitation*, 27, 373-377.
- Yesavage JA, Brink TL, Rose TL, Lum O, Huang V, Adey M, & Leirer VO. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research*, 17(1), 37-49.
- Zaaimi B, Edgley SA, Soteropoulos DS, & Baker SN. (2012). Changes in descending motor pathway connectivity after corticospinal tract lesion in macaque monkey. *Brain*, 135(7), 2277-2289.

APPENDIX 1: CLINICAL ASSESSMENT

Identification

Date: _____
(dd / mm / yr)

Subject name: _____

Address: _____

Telephone number: _____

Date of birth: _____

Gender: _____

Dominance: _____

Time since stroke (mo): _____

Site of stroke (specific lesion site): _____

Current Therapy received: _____

Results of Evaluations

Subject Name: _____

Test date: _____

1. Deltoid Strength:

Right

Left

Anterior

Middle

Posterior

2. Shoulder ROM:

Right

Left

Flex

Ext

Abd

Add

Int Rot

Ext Rot

3. Edinburgh Handedness Inventory

4. Line Bisection Test

5. Montreal Cognitive Assessment

_____ / 30

6. Geriatric Depression Scale

7. Chedoke McMaster Stroke Assessment

Arm

Hand

Subject Name: _____

Test date: _____

8. Motor Activity Log and MAL self-efficacy scale

9. Box and Block Test: Right _____ Left _____

10. Fugl Meyer Assessment (UE): _____ / 66

Light Touch: _____ / 4

Proprioception: _____ / 8

Passive range of motion: _____ / 24

Pain: _____ / 24

11. Modified Wolf Motor Function Test: _____ / 30

Grip strength: Right _____ Left _____ Time Median
NA _____ / A _____

12. The Reaching Performance Scale for stroke (RPS):

Close target: _____ / 18

Far target: _____ / 18

13. Composite Spasticity Index: _____ / 16

APPENDIX II

*Medical Research Council Speech & Communication Unit**EDINBURGH HANDEDNESS INVENTORY*

Surname..... Given Names.....

Date of Birth..... Sex.....

Please indicate your preferences in the use of hands in the following activities *by putting + in the appropriate column*. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, *put ++*. If in any case you are really indifferent *put + in both columns*.

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

Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

| | | LEFT | RIGHT |
|----|---|------|-------|
| 1 | Writing | | |
| 2 | Drawing | | |
| 3 | Throwing | | |
| 4 | Scissors | | |
| 5 | Toothbrush | | |
| 6 | Knife (without fork) | | |
| 7 | Spoon | | |
| 8 | Broom (upper hand) | | |
| 9 | Striking Match (match) | | |
| 10 | Opening box (lid) | | |
| | | | |
| i | Which foot do you prefer to kick with? | | |
| ii | Which eye do you use when using only one? | | |

| | |
|------|--|
| L.Q. | |
|------|--|

Leave these spaces blank

| | |
|--------|--|
| DECILE | |
|--------|--|

MARCH 1970

A series of horizontal black bars of varying lengths and positions, resembling a barcode or a stylized text representation. The bars are arranged in a non-uniform, staggered pattern across the image.

Name: _____

Date: _____

Geriatric Depression Scale (Short form)

Choose the best answer for how you have felt over the past week:

1. Are you basically satisfied with your life? YES / NO
2. Have you dropped many of your activities and interests? YES / NO
3. Do you feel that your life is empty? YES / NO
4. Do you often get bored? YES / NO
5. Are you in good spirits most of the time? YES / NO
6. Are you afraid that something bad is going to happen to you? YES / NO
7. Do you feel happy most of the time? YES / NO
8. Do you often feel helpless? YES / NO
9. Do you prefer to stay at home, rather than going out and doing new things? YES / NO
10. Do you feel you have more problems with memory than most? YES / NO
11. Do you think it is wonderful to be alive now? YES / NO
12. Do you feel pretty worthless the way you are now? YES / NO
13. Do you feel full of energy? YES / NO
14. Do you feel that your situation is hopeless? YES / NO
15. Do you think that most people are better off than you are? YES / NO

Name: _____

Date: _____

Chedoke-McMaster Stroke Assessment**SCORE FORM Page 2 of 4****IMPAIRMENT INVENTORY: STAGE OF RECOVERY OF ARM AND HAND**

ARM and HAND: Start at Stage 3. Starting position: sitting with forearm in lap in a neutral position, wrist at 0° and fingers slightly flexed. Changes from this position are indicated by underlining. Place an X in the box of each task accomplished. Score the highest Stage in which the client achieves at least two Xs.

ARM

- 1 ☐ not yet Stage 2
- 2 ☐ resistance to passive shoulder abduction or elbow extension
☐ facilitated elbow extension
☐ facilitated elbow flexion
- 3 ☐ touch opposite knee
☐ touch chin
☐ shoulder shrugging > ½ range
- 4 ☐ extension synergy, then flexion synergy
☐ shoulder flexion to 90°
☐ elbow at side, 90° flexion: supination, then pronation
- 5 ☐ flexion synergy, then extension synergy
☐ shoulder abduction to 90° with pronation
☐ shoulder flexion to 90°: pronation then supination
- 6 ☐ hand from knee to forehead 5 x in 5 sec.
☐ shoulder flexion to 90°: trace a figure 8
☐ arm resting at side of body: raise arm overhead with full supination
- 7 ☐ clap hands overhead, then behind back 3 x in 5 sec
☐ shoulder flexion to 90°: scissor in front 3 x in 5 sec
☐ elbow at side, 90° flexion: resisted shoulder external rotation

STAGE OF ARM

☐**HAND**

- 1 ☐ not yet Stage 2
- 2 ☐ positive Hoffman
☐ resistance to passive wrist or finger extension
☐ facilitated finger flexion
- 3 ☐ wrist extension > ½ range
☐ finger/wrist flexion > ½ range
☐ supination, thumb in extension: thumb to index finger
- 4 ☐ finger extension, then flexion
☐ thumb extension > ½ range, then lateral prehension
☐ finger flexion with lateral prehension
- 5 ☐ finger flexion, then extension
☐ pronation: finger abduction
☐ hand unsupported: opposition of thumb to little finger
- 6 ☐ pronation: tap index finger 10 x in 5 sec
☐ pistol grip: pull trigger, then return
☐ pronation: wrist and finger extension with finger abduction
- 7 ☐ thumb to finger tips, then reverse 3 x in 12 sec
☐ bounce a ball 4 times in succession, then catch
☐ pour 250 ml. from 1 litre pitcher, then reverse

STAGE OF HAND

☐

COPY FREELY - DO NOT CHANGE

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Name: _____

Date: _____

MAL Score Sheet

| | Yes/No | AS | HW |
|--|--------|----|-------------------------------------|
| 1. Turn on a light with a light switch | | | If no, why? _____ Comments _____ |
| 2. Open drawer | | | If no, why? _____ Comments _____ |
| 3. Remove an item of clothing from a drawer | | | If no, why? _____ Comments _____ |
| 4. Pick up a phone | | | If no, why? _____ Comments _____ |
| 5. Wipe off a kitchen counter or other surface | | | If no, why? _____ Comments _____ |
| 6. Get out of a car (includes only the movement needed to get the body from sitting to standing outside of the car, once the door is open) | | | If no, why? _____ Comments _____ |
| 7. Open refrigerator | | | If no, why? _____ Comments _____ |
| 8. Open a door by turning a door knob | | | If no, why? _____ Comments _____ |
| 9. Use a TV remote control | | | If no, why? _____ Comments _____ |
| 10. Wash your hands (includes lathering and rinsing hands; does not include turning water on and off with a faucet handle) | | | If no, why? _____ Comments _____ |
| 11. Turning water on/off with knob/lever on faucet | | | If no, why? _____ Comments _____ |
| 12. Dry your hands | | | If no, why? _____ Comments _____ |
| 13. Put on your socks | | | If no, why? _____ Comments _____ |

Name: _____

Date: _____

| | Yes/No | AS | HW |
|---|--------|----|-------------------------------------|
| 14. Take off your socks | | | If no, why? _____ Comments _____ |
| 15. Put on your shoes (includes tying shoestrings and fastening straps) | | | If no, why? _____ Comments _____ |
| 16. Take off your shoes (includes untying shoestrings and unfastening straps) | | | If no, why? _____ Comments _____ |
| 17. Get up from a chair with armrests | | | If no, why? _____ Comments _____ |
| 18. Pull chair away from table before sitting down | | | If no, why? _____ Comments _____ |
| 19. Pull chair toward table after sitting down | | | If no, why? _____ Comments _____ |
| 20. Pick up a glass, bottle, drinking cup, or can (does not need to include drinking) | | | If no, why? _____ Comments _____ |
| 21. Brush your teeth (does not include preparation of toothbrush or brushing dentures) | | | If no, why? _____ Comments _____ |
| 22. Put on makeup base, lotion, or shaving cream on face | | | If no, why? _____ Comments _____ |
| 23. Use a key to unlock a door | | | If no, why? _____ Comments _____ |
| 24. Write on paper (if dominant arm was most affected, "do you use it to write?": if non-dominant arm was most affected, drop the item and assign "NA") | | | If no, why? _____ Comments _____ |

Name: _____

Date: _____

| | Yes/No | AS | HW |
|--|--------|----|-------------------------------------|
| 25. Carry an object in your hand (draping an item over the arm is not acceptable) | | | If no, why? _____ Comments _____ |
| 26. Use a fork or spoon for eating (refers to the action of bringing food to the mouth with fork or spoon) | | | If no, why? _____ Comments _____ |
| 27. Comb your hair | | | If no, why? _____ Comments _____ |
| 28. Pick up a cup by a handle | | | If no, why? _____ Comments _____ |
| 29. Button a shirt | | | If no, why? _____ Comments _____ |
| 30. Eat half a sandwich or finger foods | | | If no, why? _____ Comments _____ |

Amount Scale

0 – My weaker arm was not used at all for the activity (not used).

0.5 –

1 – My weaker arm was moved during that activity, but was not helpful (very rarely).

1.5 –

2 – My weaker arm was of some use during that activity but needed some help from the stronger arm or moved very slowly or with difficulty (rarely).

2.5 –

3 – My weaker arm was used for the activity but the movements were slow or were made only with some effort (half pre stroke).

3.5 –

4- The movements made by my weaker arm for that activity were almost normal but not quite as fast or accurate as normal (3\4th or 75% pre stroke).

4.5 –

5 – My ability to use the weaker arm for that activity was as good as before the injury (same as pre-stroke).

How Well Scale

0 – My weaker arm was not used at all for the activity (of no use).

0.5 –

1 – My weaker arm was moved during that activity, but was not helpful (very poor).

1.5 –

2 – My weaker arm was of some use during that activity but needed some help from the stronger arm or moved very slowly or with difficulty (poor).

2.5 –

3 – My weaker arm was used for the activity but the movements were slow or were made only with some effort (fair).

3.5 –

4 – The movements made by my weaker arm for that activity were almost normal but not quite as fast or accurate as normal (almost normal).

4.5 –

5 – My ability to use the weaker arm for that activity was as good as before the injury.

Name: _____

Date: _____

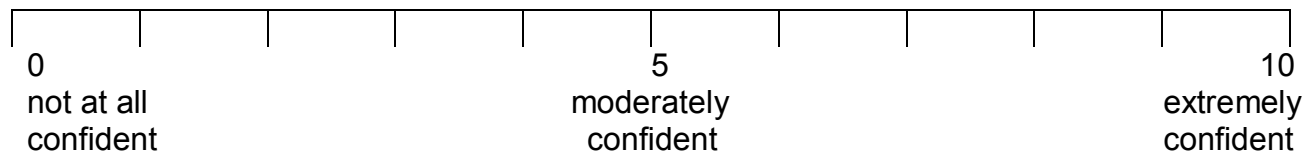
*Coordination and motor recovery of the upper limb in stroke
LEVIN, Mindy F., KORNER-BITENSKY, Nicol, FELDMAN, Anatol G.*

Self-Efficacy Scale for Items on Motor Activity Log

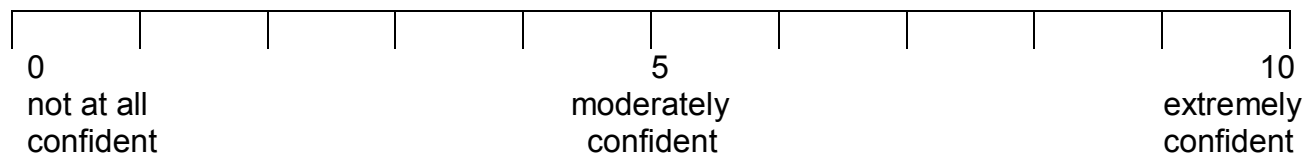
We would like to know how confident you are in performing certain daily activities. For each of the following questions, please mark the line at the point that corresponds to your certainty that you can perform the tasks with your **more affected hand** as of now, without assistive devices or help from another person. Please consider what you routinely can do, not what would require a single extraordinary effort.

AS OF NOW, HOW CONFIDENT ARE YOU THAT YOU CAN:

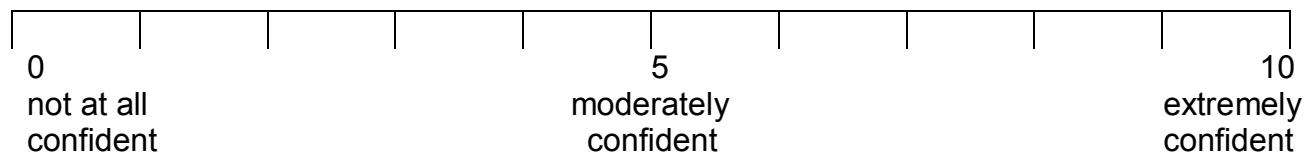
1. Turn on a light switch?



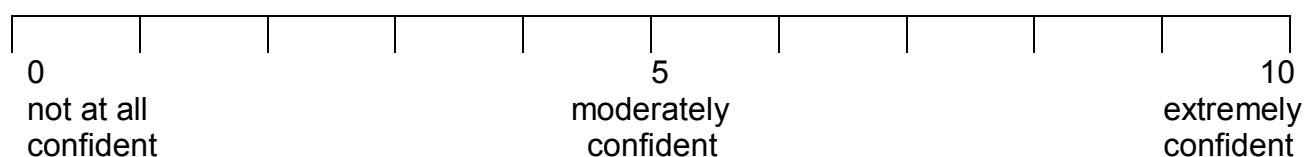
2. Open a drawer?



3. Remove an item of clothing from a drawer?



4. Pick up the phone?

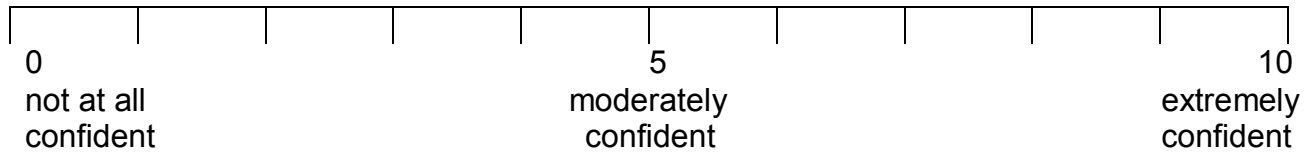


Name: _____

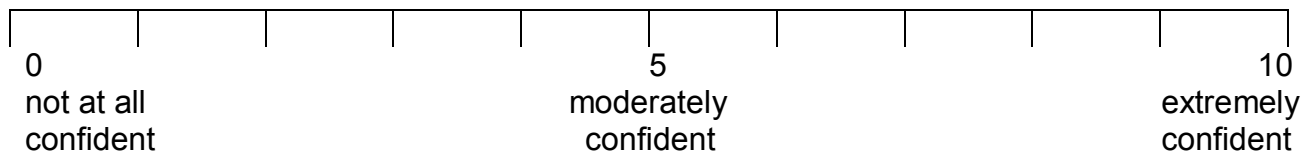
Date: _____

Coordination and motor recovery of the upper limb in stroke
LEVIN, Mindy F., KORNER-BITENSKY, Nicol, FELDMAN, Anatol G.

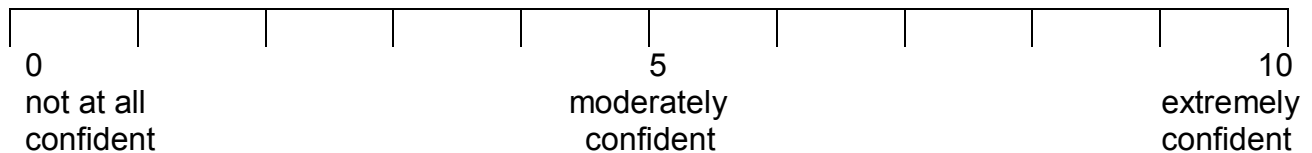
5. Wipe off a kitchen counter or other surface?



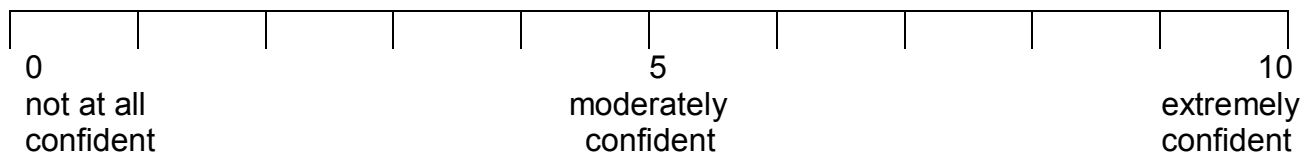
6. Get out of a car?



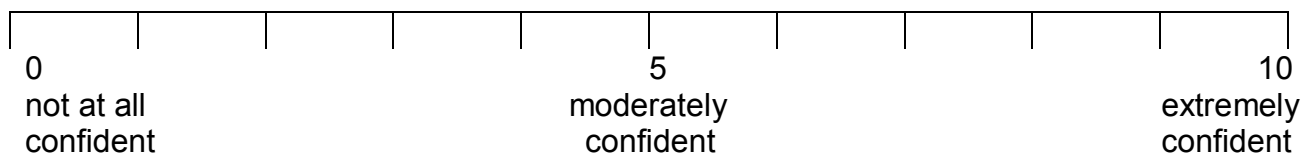
7. Open the refrigerator?



8. Open a door by turning a door knob?



9. Use a TV remote control?

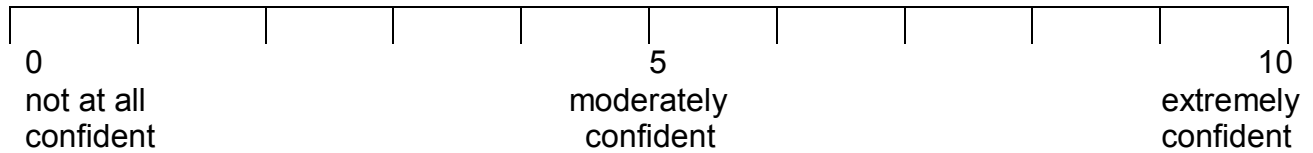


Name: _____

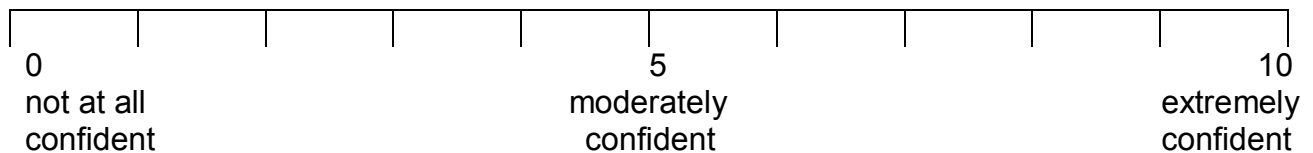
Date: _____

Coordination and motor recovery of the upper limb in stroke
LEVIN, Mindy F., KORNER-BITENSKY, Nicol, FELDMAN, Anatol G.

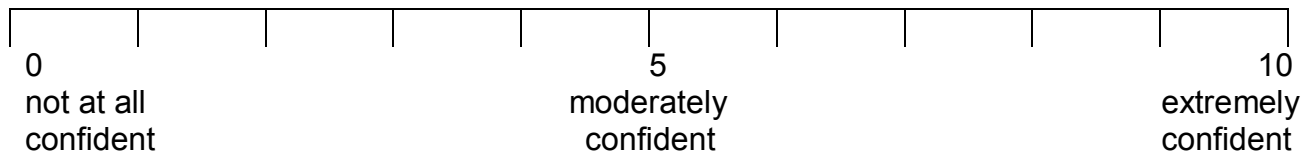
10. Wash your hands?



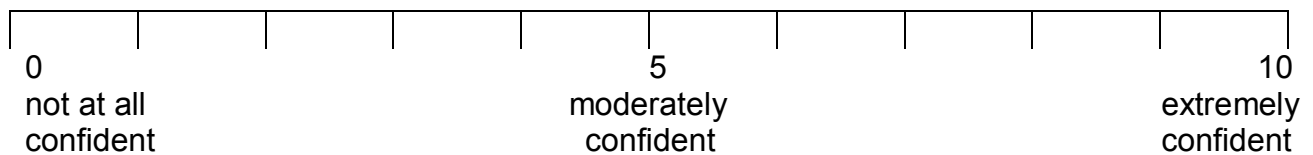
11. Turn water on/off with knob/lever on faucet?



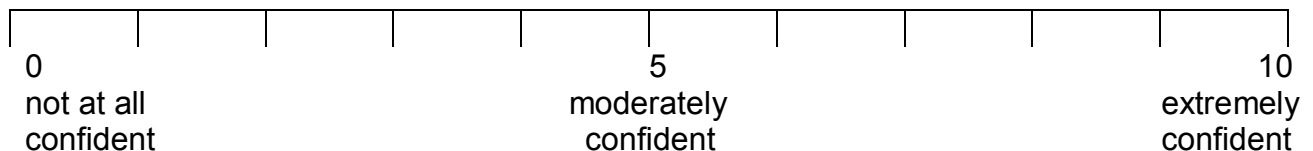
12. Dry your hands?



13. Put on your socks?



14. Take off your socks?

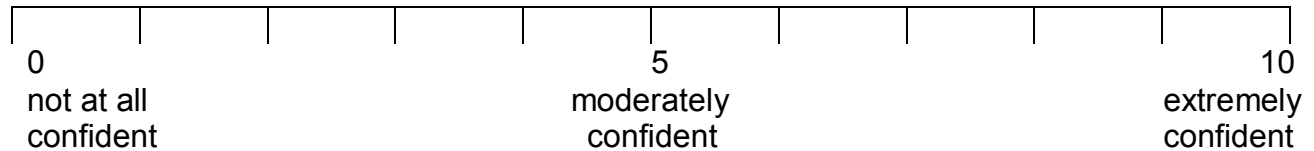


Name: _____

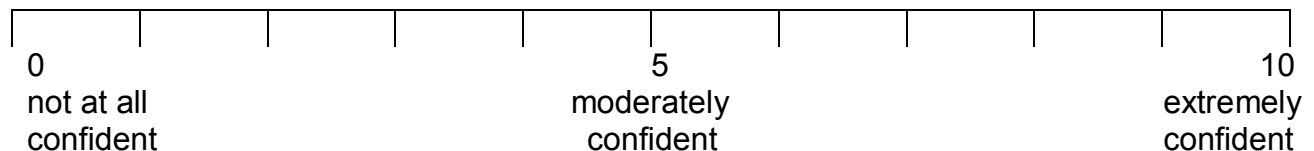
Date: _____

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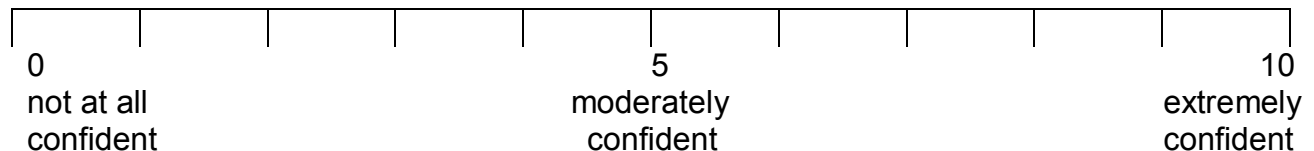
15. Put on your shoes?



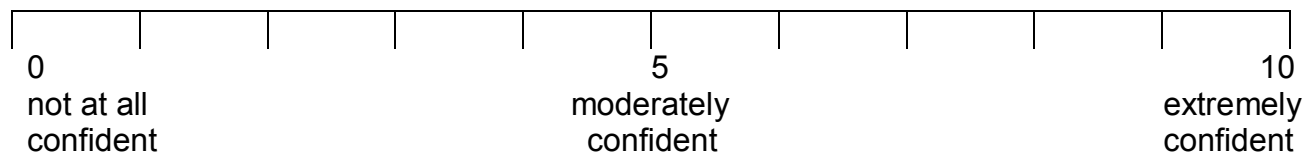
16. Take off your shoes?



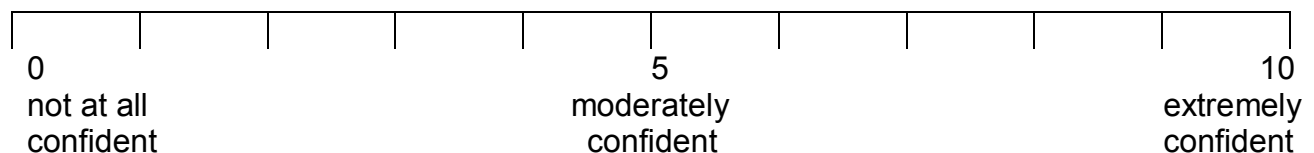
17. Get up from a chair with armrests?



18. Pull chair away from a table before sitting down?



19. Pull chair toward a table after sitting down?

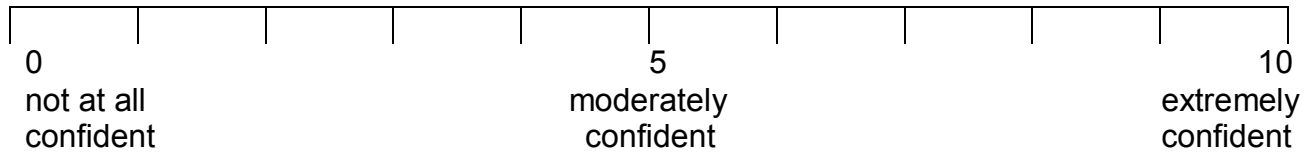


Name: _____

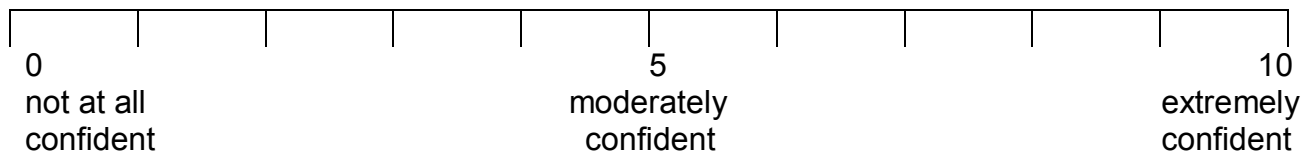
Date: _____

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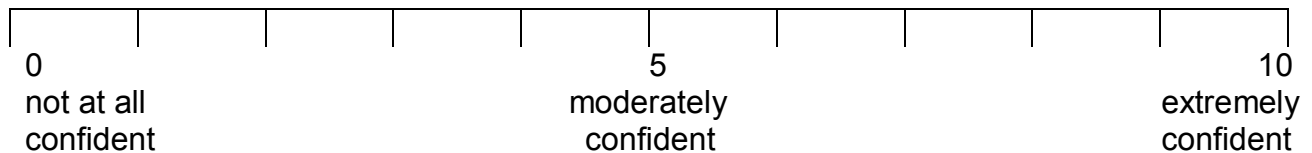
20. Pick up a glass, bottle, drinking cup or can?



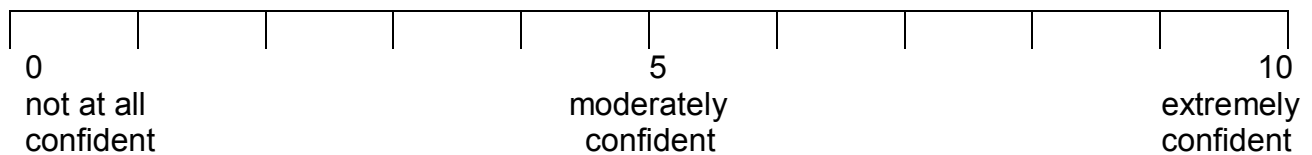
21. Brush your teeth?



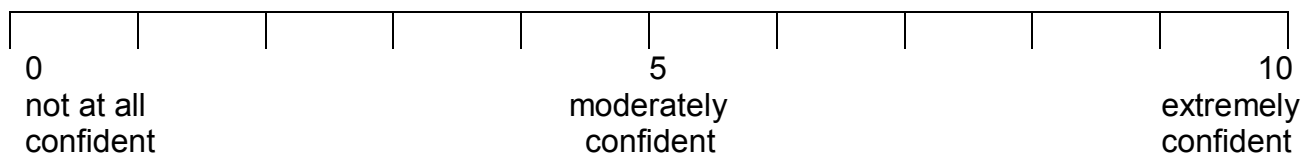
22. Put on makeup base, lotion or shaving cream on face?



23. Use a key to unlock a door?



24. Write on paper?

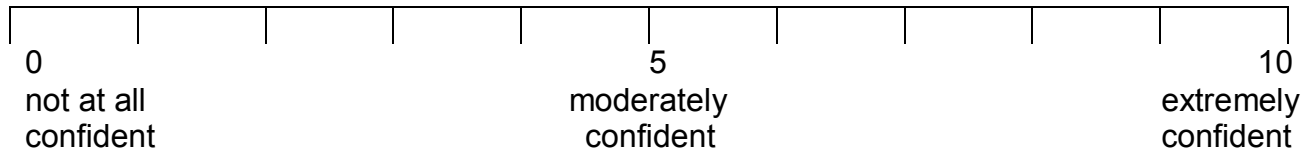


Name: _____

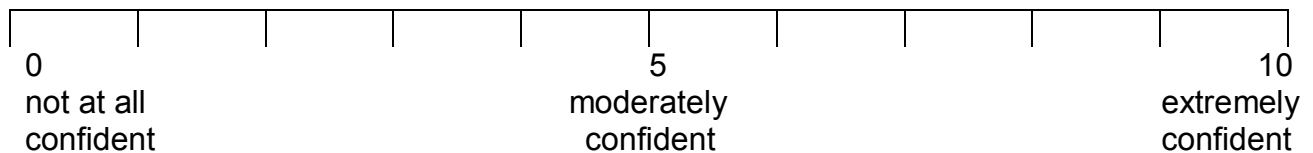
Date: _____

Coordination and motor recovery of the upper limb in stroke
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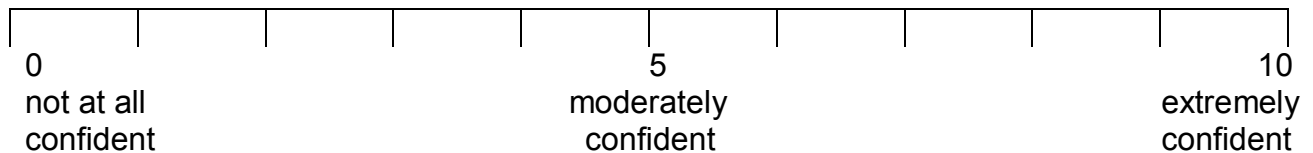
25. Carry an object in your hand?



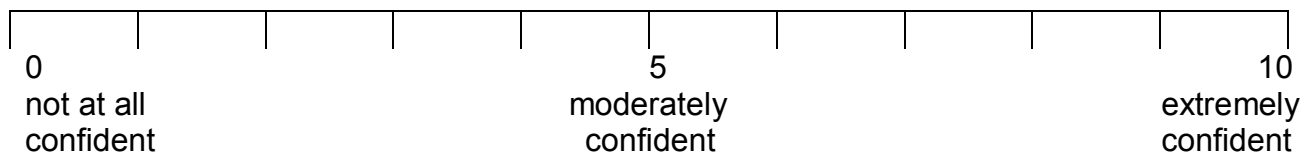
26. Use a fork or spoon for eating?



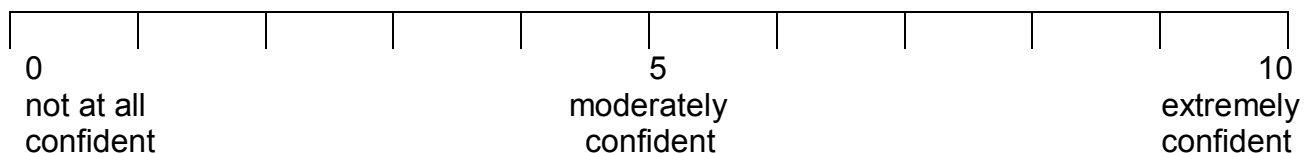
27. Comb your hair?



28. Pick up a cup by a handle?



29. Button a shirt?

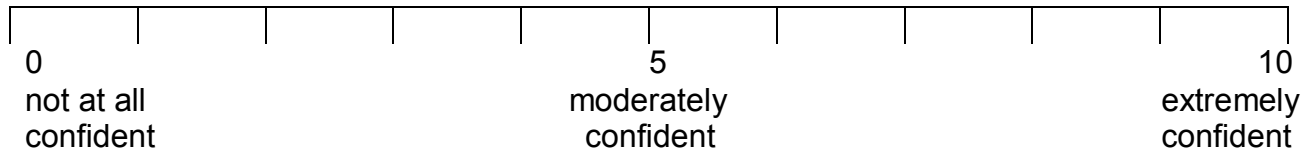


Name: _____

Date: _____

Coordination and motor recovery of the upper limb in stroke
LEVIN, Mindy F., KORNER-BITENSKY, Nicol, FELDMAN, Anatol G.

30. Eat half a sandwich or finger food?



EVALUATION OF DEXTERITY BOX AND BLOCKS TEST

IDENTITY SHEET

NAME: _____ AGE: _____ DATE: _____

DIAGNOSIS: _____ DOMINANCE: _____

NUMBER OF BLOCKS TRANSFERRED FROM ONE COMPARTMENT TO ANOTHER IN 60 SECONDS

| | Right | Left |
|-----------------|-------------|-------------|
| 1 st | <div></div> | <div></div> |
| 2 nd | <div></div> | <div></div> |

Comments:

Therapist: _____

MATHIOVETZ V, VOLLAND G, KASHMAN N, WEBER K (1985). ADULT NORMS FOR BOX AND BLOCK TEST FOR MANUAL DEXTERITY. AMERICAN JOURNAL OF OCCUPATIONAL THERAPY. 39:386-391.

© 1985 BY THE AMERICAN OCCUPATIONAL THERAPY ASSOCIATION, INC.

Name: _____

Date: _____

FUGL-MEYER ASSESSMENT OF PHYSICAL PERFORMANCE

| Motor Function Upper Extremity | | | | |
|---------------------------------------|--|-------|------|---|
| TEST | ITEM | SCORE | | SCORING CRITERIA |
| | | Pre | Post | |
| I. Reflexes | Biceps | | | 0 – No reflex activity can be elicited |
| | Triceps | | | 2 – Reflex activity can be elicited |
| II. Flexor Synergy | Elevation | | | 0 – Cannot be performed at all |
| | Shoulder retraction | | | 1 – Performed partly |
| | Abduction (at least 90°) | | | 2 – Performed faultlessly |
| | External rotation | | | |
| | Elbow flexion | | | |
| | Forearm supination | | | |
| III. Extensor Synergy | Shoulder add / int rot | | | 0 – Cannot be performed at all |
| | Elbow extension | | | 1 – Performed partly |
| | Forearm pronation | | | 2 – Performed faultlessly |
| IV. Movement combining synergies | Hand to lumbar spine | | | 0 – No specific action performed 1 – Hand must pass anterior superior iliac spine 2 – Performed faultlessly |
| | Shoulder flexion to 90°, elbow at 0° | | | 0 – Arm is immediately abducted, or elbow flexes at start of motion 1 – Abduction or elbow flexion occurs in later phase of motion 2 – Performed faultlessly |
| | Pronation/supination of forearm with elbow at 90° & shoulder at 0° | | | 0 – Correct position of shoulder and elbow cannot be attained, and/or pronation or supination cannot be performed at all 1 – Active pronation or supination can be performed even within a limited range of motion, and at the same time the shoulder and elbow are correctly positioned 2 – Complete pronation and supination with correct positions at elbow and shoulder |
| V. Movement out of synergy | Shoulder abduction to 90°, elbow at 0°, and forearm pronated | | | 0 – Initial elbow flexion occurs, or any deviation from pronated forearm occurs 1 – Motion can be performed partly, or if during motion, elbow is flexed, or forearm cannot be kept in pronation 2 – Performed faultlessly |
| | Shoulder flexion 90-180°, elbow at 0°, and forearm at mid-position | | | 0 – Initial flexion of elbow or shoulder abduction occurs 1 – Elbow flexion or shoulder abduction occurs during shoulder flexion 2 – Performed faultlessly |
| | Pronation/supination of forearm, elbow at 0° and shoulder between 30-90° of flexion | | | 0 – Supination and pronation cannot be performed at all, or elbow and shoulder positions cannot be attained 1 – Elbow and shoulder properly positioned and pronation and supination performed in a limited range 2 – Performed faultlessly |
| VI. Normal reflex activity | Biceps and/or finger flexors and triceps (This item is only included if the patient achieves a maximum score on all previous items, otherwise score 0) | | | 0 – At least 2 of the 3 phasic reflexes are markedly hyperactive 1 – One reflex is markedly hyperactive, or at least 2 reflexes are lively 2 – No more than one reflex is lively and none are hyperactive |

Name: _____

Date: _____

| TEST | ITEM | SCORE | | SCORING CRITERIA |
|---|--|-------|--|---|
| VII. Wrist | Stability, elbow at 90°, shoulder at 0° | | | 0 – Patient cannot dorsiflex wrist to required 15° 1 – Dorsiflexion is accomplished, but no resistance is taken 2 – Position can be maintained with some (slight) resistance |
| | Flexion/extension, elbow at 90°, shoulder at 0° | | | 0 – Volitional movement does not occur 1 – Patient cannot actively move the wrist joint throughout the total ROM 2 – Faultless, smooth movement |
| | Stability, elbow at 0°, shoulder at 0° | | | 0 – Patient cannot dorsiflex wrist to required 15° 1 – Dorsiflexion is accomplished, but no resistance is taken 2 – Position can be maintained with some (slight) resistance |
| | Flexion/extension, elbow at 0°, shoulder at 30° | | | 0 – Volitional movement does not occur 1 – Patient cannot actively move the wrist joint throughout the total ROM 2 – Faultless, smooth movement |
| | Circumduction | | | 0 – Cannot be performed 1 – Jerky motion or incomplete circumduction 2 – Complete motion with smoothness |
| VIII. Hand | Finger mass flexion | | | 0 – No flexion occurs 1 – Some flexion, but not full motion 2 – Complete active flexion (compared with unaffected hand) |
| | Finger mass extension | | | 0 – No extension occurs 1 – Patient can release an active mass flexion grasp 2 – Full active extension |
| | Grasp I – MCP joints extended and proximal & distal IP joints are flexed; grasp is tested against resistance | | | 0 – Required position cannot be acquired 1 – Grasp is weak 2 – Grasp can be maintained against relatively great resistance |
| | Grasp II – Patient is instructed to adduct thumb, with a scrap of paper interposed | | | 0 – Function cannot be performed 1 – Scrap of paper interposed between the thumb and index finger can be kept in place, but not against a slight tug 2 – Paper is held firmly against a tug |
| | Grasp III – Patient opposes thumb pad against pad of index finger, with a pencil interposed | | | 0 – Function cannot be performed 1 – Pencil interposed between the thumb and index finger can be kept in place, but not against a slight tug 2 – Pencil is held firmly against a tug |
| | Grasp IV – The patient should grasp a can by opposing the volar surfaces of the 1 st and 2 nd digits | | | 0 – Function cannot be performed 1 – A can interposed between the thumb and index finger can be kept in place, but not against a slight tug 2 – Can is held firmly against a tug |
| | Grasp V – The patient grasps a tennis ball with a spherical grip or is instructed to place his/her fingers in a position with abduction position of the thumb and abduction flexion of the 2 nd , 3 rd , 4 th & 5 th fingers | | | 0 – Function cannot be performed 1 – A tennis ball can be kept in place with a spherical grasp, but not against a slight tug 2 – Tennis ball is held firmly against a tug |
| IX. Coordination/ Speed – Finger from knee to nose (5 repetitions in rapid succession) | Tremor | | | 0 – Marked tremor 1 – Slight tremor 2 – No tremor |
| | Dysmetria | | | 0 – Pronounced or unsystematic dysmetria 1 – Slight or systematic dysmetria 2 – No dysmetria |
| | Speed | | | 0 – Activity is more than 6 seconds longer than unaffected hand 1 – (2-5.9) seconds longer than unaffected hand 2 – Less than 2 seconds difference |
| Upper Extremity Total | | | | Maximum = 66 |

Name: _____

Date: _____

| Sensation | | | | |
|--------------------------------------|--------------|-------|------|--|
| TYPE OF SENSATION | AREA | SCORE | | SCORING CRITERIA |
| | | Pre | Post | |
| I. Light Touch | Upper Arm | | | 0 – Anesthesia 1 – Hyperesthesia / dysesthesia 2 - Normal |
| | Palm of Hand | | | |
| | Thigh | | | |
| | Sole of Foot | | | |
| II. Proprioception | Shoulder | | | 0 – No sensation 1 – 75% of answers are correct, but considerable difference in sensation relative to unaffected side 2 – All answers are correct, little or no difference |
| | Elbow | | | |
| | Wrist | | | |
| | Thumb | | | |
| | Hip | | | |
| | Knee | | | |
| | Ankle | | | |
| | Toe | | | |
| Total Sensation Score | | | | Maximum = 24 |
| Total Motor and Sensory Score | | | | Maximum = 124 |
| Comments | Pre: | | | |
| | Post: | | | |

Évaluation de l'amplitude articulaire passive et de la douleur

Formule d'évaluation

**TEST DE
FUGL-MEYER**

IDENTIFICATION

Nom: _____
No: _____
Côté atteint: Gauche ☐ Droit ☐

Prénom: _____
Date: _____
jour mois an

Session
① ② ③ ④

I- MEMBRE SUPÉRIEUR

Épaule

Flexion (0-180°)

Abduction (0-90°)

Rotation externe

Rotation interne

Coude

Flexion

Extension

Avant-bras

Pronation

Supination

Poignet

Flexion

Extension

Doigts

Flexion

Extension

AMPLITUDE

0 1 2

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DOULEUR

0 1 2

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Total D G

24

Amplitude articulaire passive et douleur

35

MODIFIED WOLF MOTOR FUNCTION TEST – DATA COLLECTION FORM

Subject's name: _____

Date: _____

Side of hemiparesis

Right

Left

| Task | Time | Functional Ability | Time |
|--------------------------|----------------------|--------------------|----------------------|
| | Less Affected | | More Affected |
| 5. Hand to table (front) | | 0 1 2 3 4 5 | |
| 6. Hand to box (front) | | 0 1 2 3 4 5 | |
| 8. Reach and retrieve | | 0 1 2 3 4 5 | |
| 9. Lift can | | 0 1 2 3 4 5 | |
| 10. Lift pencil | | 0 1 2 3 4 5 | |
| 16. Fold towel | | 0 1 2 3 4 5 | |

Grip strength: Right _____

Left _____

Functional Ability Scale

- 0 – Does not attempt with upper extremity (UE) being tested.**
- 1 –UE being tested does not participate functionally; however, attempt is made to use the UE. In unilateral tasks the UE not being tested may be used to move the UE being tested.**
- 2 – Does, but requires assistance of the UE not being tested for minor readjustments or change of position, or requires more than two attempts to complete, or accomplishes very slowly. In bilateral tasks the UE being tested may serve only as a helper.**
- 3 – Does, but movement is influenced to some degree by synergy or is performed slowly or with effort.**
- 4 – Does; movement is close to normal *, but slightly slower; may lack precision, fine coordination or fluidity.**
- 5 – Does; movement appears to be normal *.**

(*) For the determination of normal, the less-involved UE can be utilized as an available index for comparison, with pre-morbid UE dominance taken into consideration.

The Reaching Performance Scale for Stroke (RPS)

Name of patient: _____
 Today's Date: _____
 Name of Evaluator: _____
 Evaluation Number: _____

1. TRUNK DISPLACEMENT

| | Close Target | | Far Target |
|----|--|----|---|
| 3. | No or almost no forward trunk Displacement | 3. | Appropriate forward trunk displacement related to the amount of elbow extension |
| 2. | Small displacement of the trunk (flexion, rotation or flexion accompanied by rotation) | 2. | Excessive trunk displacement related to a limitation of the active movement of the elbow or shoulder |
| 1. | More than half the movement is made by the trunk | 1. | Excessive trunk displacement: About half of the displacement of the hand towards the target is accomplished by the trunk but the hand arrives at the target |
| 0. | Task is accomplished only by forward trunk displacement | 0. | Excessive trunk displacement: More than $\frac{3}{4}$ of the displacement of the hand to the target is accomplished by the trunk and the hand does not arrive at the target |

Rating: _____

Rating: _____

2. MOVEMENT SMOOTHNESS*

| | Close Target | | Far Target |
|----|---|----|---|
| 3. | The combination of movement of the arm and trunk is fluid and smooth | 3. | The combination of movement of the arm and trunk is fluid and smooth |
| 2. | More than one movement of the arm is made to perform the task or the movement is segmented (not smooth) | 2. | More than one movement of the arm is made to perform the task or the movement is segmented (not smooth) |
| 1. | Several small movements of the arm <i>and trunk</i> are made in a sequential manner | 1. | Several small movements of the arm <i>and trunk</i> are made in a sequential manner |
| 0. | Complete segmentation of arm and trunk movement | 0. | Complete segmentation of arm and trunk movement |

- exclude assessment of tremor or dysmetria

Rating: _____

Rating: _____

3. SHOULDER MOVEMENTS

| | Close Target | | Far Target |
|----|---|----|--|
| 3. | Adequate shoulder flexion and horizontal adduction with scapular elevation to perform the task | 3. | Adequate shoulder flexion and horizontal adduction with scapular protraction and elevation to perform the task |
| 2. | Shoulder flexion and horizontal adduction occurs with excessive scapular elevation | 2. | Shoulder flexion and horizontal adduction occurs with excessive scapular protraction or elevation |
| 1. | Shoulder flexion only occurs in combination with excessive scapular elevation. Shoulder horizontal adduction is decreased | 1. | Shoulder flexion is combined with scapular elevation. Shoulder horizontal adduction is decreased |
| 0. | No or almost no shoulder flexion or horizontal adduction is possible (all the movement is made by the scapula) | 0. | No or almost no shoulder flexion or horizontal adduction is possible (all the movement is made by the scapula) |

Rating: _____

Rating: _____

4. ELBOW MOVEMENTS

| | Close Target | | Far Target |
|----|---|----|--|
| 3. | Extending the hand to the target is principally attributed to elbow extension | 3. | Elbow extension is almost full |
| 2. | More than half of the reaching movement is attributed to elbow extension | 2. | More than half of the reaching movement is attributed to elbow extension |
| 1. | Less than half of the reaching movement is attributed to elbow extension | 1. | Less than half of the reaching movement is attributed to elbow extension |
| 0. | No elbow extension occurs | 0. | No elbow extension occurs |

Rating: _____

Rating: _____

5. PREHENSION

| | Close Target | | Far Target |
|----|--|----|--|
| 3. | Adequate hand opening and closure to perform the task | 3. | Adequate hand opening and closure to perform the task |
| 2. | Opening or relaxing the hand is difficult | 2. | Opening or relaxing the hand is difficult |
| 1. | Use of compensatory grasping strategies: for example: winding, downward grasping | 1. | Use of compensatory grasping strategies: for example: winding, downward grasping |
| 0. | Prehension is not possible | 0. | Prehension is not possible |

Rating: _____

Rating: _____

6. GLOBAL SCORE

| | Close Target | | Far Target |
|----|--|----|--|
| 3. | The task can be done easily, with or without mild tremor or dysmetria, following a smooth and direct trajectory | 3. | The task can be done easily, with or without mild tremor or dysmetria, following a smooth and direct trajectory |
| 2. | The task is done in the presence of tremor, dysmetria, small, jerky movements, arc shaped trajectory or segmentation. Prehension is possible but may be modified or difficult | 2. | The task is done in the presence of tremor, dysmetria, small, jerky movements, arc shaped trajectory or segmentation. Prehension is possible but may be modified or difficult |
| 1. | The task is done partially (more than 50%) or with modification (such as stabilization of the cone, sliding the cone on the table, modification of table height, shorter distance to the cone). Prehension may be absent | 1. | The task is done partially (more than 50%) or with modification (such as stabilization of the cone, sliding the cone on the table, modification of table height, shorter distance to the cone). Prehension may be absent |
| 0. | Less than half the task is accomplished despite modifications | 0. | Less than half the task is accomplished despite modifications |

Rating: _____

Rating: _____

COMPOSITE SPASTICITY INDEX
MOTOR CONTROL IN REHABILITATION LABORATORY
MINDY LEVIN, PT, PH.D.

NAME: _____ TODAY`S DATE (d/m/y)_____

DATE OF BIRTH (d/m/y)_____ AGE:____ DATE OF INJURY (d/m/y) _____

DESCRIPTION OF INJURY AND TREATMENT HISTORY:

MEDICATIONS: _____

RANGE OF MOTION: WRIST: _____ ELBOW _____

=====

EVALUATION

TENDON JERK (BICEPS)

- 0 No response
- 1 Normal response
- 2 Mildly hyperactive response
- 3 Moderately hyperactive response
- 4 Maximally hyperactive response

RESISTANCE TO FULL RANGE PASSIVE JOINT DISPLACEMENT (e.g., elbow extension)

* performed at moderate speed (> 100 deg/s)

- 0 No resistance (hypotonic)
- 2 Normal resistance
- 4 Mildly increased resistance
- 6 Moderately increased resistance
- 8 Maximally increased resistance

CLONUS (wrist or ankle)

- 1 Clonus not elicited
- 2 1 – 3 beats of clonus elicited
- 3 3 – 10 beats of clonus elicited
- 4 Sustained clonus

=====

COMPOSITE SPASTICITY SCORE _____ / 16

APPENDIX 2: CONSENT FORM

INFORMED CONSENT FORM

Project Investigators

Mindy F. Levin, PhD, PT

School of Physical and Occupational Therapy, McGill University and CRIR - JRH

Melanie C. Baniña, MSc

PhD candidate, School of Physical and Occupational Therapy, McGill University & CRIR-JRH

Background

We are asking you to participate in a research project looking at upper limb movement and chronic musculoskeletal problems after stroke. This consent form explains the aim of this study, the procedures, advantages, risks and inconvenience as well as the persons to contact, if necessary. Before agreeing to participate in this project, please take the time to read and carefully consider the following information.

This consent form may contain words that you do not understand. We invite you to ask any question that you deem useful to the researcher and the other members of the staff assigned to the research project and ask them to explain any word or information which is not clear to you.

Individuals who have had a stroke often have difficulty moving their arm, even after having completed their rehabilitation program. In addition, chronic musculoskeletal problems including, but not limited to pain, joint damage, and muscle spasms may have a negative effect on the recovery of arm movement. This project will assess the coordination and dexterity of the arm while reaching for objects and avoiding obstacles.

Objectives

The main goal of this study is to evaluate the coordination of arm movements in persons who have chronic musculoskeletal problems and have had a stroke. Their characteristics will be compared to three additional groups: a healthy group, subdivided into those with and without musculoskeletal problems, and a group of stroke survivors who do not have chronic musculoskeletal problems.

Nature of my participation

This study will take place at the Sensorimotor Control and Rehabilitation Laboratory of the Jewish Rehabilitation Hospital. I shall be attending 2 evaluation sessions that will take a total of approximately 2 hours of my time. The first session (1 hour) will consist of a clinical evaluation of my ability to move my arm. The second session (1 hour: 15 minutes for set-up, 45 minutes for evaluation) will be an experimental session in which the movement characteristics (kinematic evaluation) of my arm will be measured.

Preparation

Coordination and motor recovery of the upper limb in stroke

Markers will be placed onto my trunk, arms, forearms, and hands in order to measure my movement patterns using a special camera. The application of the markers means I will need to bring a sleeveless top to wear during the study. I will also wear glasses that will enable me to look at the virtual environment in 3D.

Evaluation

The kinematic evaluation consists of the recording of movements made with my arms. I will be sitting in front of a screen and viewing an image of a grocery store refrigerator. The task will be to reach for a can on the refrigerator shelf. At the start of the experiment I will be given time to get used to moving my arm in the virtual environment. (Please see the photo of the experimental setup, next page). There will be 6 blocks of trials (3 minutes each), and after each block I will be able to rest if needed.

During the trials, one of the refrigerator doors may close while I am reaching for the object. I will be asked to continue to reach despite these disturbances. I will not be asked to do these trials unless I am confident that I can do so safely.



Experimental setup

Risks and disadvantages

There is no medical risk associated with my participation in this research study. My participation in the study will not affect the care and services that I receive at the Jewish Rehabilitation Hospital. During the first evaluation a therapist will always be present to provide any assistance. I may, however, feel tired following the evaluation. Although it is hypo-allergenic, the adhesive tape used to fix the markers on my skin may occasionally produce some slight skin irritation. If this happens, a calming lotion is available and will be applied to the skin. New adhesive tape is used for each session.

Benefits

I will not personally benefit from participating in this study. However, the results from this study will provide information that may help in developing better techniques for the rehabilitation of persons who have had a stroke.

Financial compensation

Coordination and motor recovery of the upper limb in stroke

Transportation and parking costs incurred through my participation in this project will be reimbursed up to a maximum of \$30 per session, upon presentation of receipts.

Access to my medical chart

I authorize access to my medical file to the persons responsible for this project. I understand that only the relevant information concerning my medical history will be used by members of this research team.

Confidentiality

Any personal information making it possible to identify me will be kept confidential and will be filed by the research coordinator in a locked cabinet at the Jewish Rehabilitation Hospital Sensorimotor Control and Rehabilitation Laboratory. The data relating to my evaluations will be transferred onto a computer file server where access is protected by passwords. Only members of the research team will have access to the information collected during the project. Otherwise, the information will be preserved for a maximum duration of 5 years following the end of the study, after which time it will be destroyed. The results of this research study will only be revealed in the form of scientific presentations or publications, without my name or identity exposed.

Questions concerning the study

The researchers present during the evaluation session should answer any questions I may have concerning the project in a satisfactory manner.

Withdrawal of subject from study

My participation in the research project described above is completely free and voluntary. I understand that I have the right to withdraw from the study at any moment without giving reason. This will not affect the health care and services I receive. Should I withdraw from the study, all documents and research data concerning myself will be destroyed.

Responsibility

By accepting to participate in this study, I do not surrender any of my rights and I do not liberate the researchers, their sponsors or the institutions involved from their legal and professional obligations.

Contact persons

If I need to ask questions about the project, signal an adverse effect and/or an incident, I can contact Melanie Baniña at (450) 688-9550 ext. 4824 or by email: melanie.banina@mail.mcgill.ca, or Mindy Levin, PhD, PT, at (514) 398-3894 or by email: mindy.levin@mcgill.ca.

If I have any questions regarding my rights and recourse concerning my participation in this study, I can contact Ms. Anik Nolet, Research Ethics Co-ordinator of the CRIR establishments at (514) 527-4527 ext. 2643, or by e-mail at: anolet.crir@ssss.gouv.qc.ca.

CONSENT

I declare to have read and understood the project, the nature and the extent of the project, as well as the risks and inconveniences I am exposed to as described in the present document. I have had the opportunity to ask all my questions concerning the different aspects of the study and to receive explanations to my satisfaction.

I, undersigned, voluntarily accept to participate in this study. I can withdraw at any time without any prejudice. I certify that I have received enough time to take my decision and I know that a copy of this consent form will be added to my medical file.

A signed copy of this information and consent form will be provided to me for my record.

Participant: _____ Date: _____

(Signature)

_____ Tel: _____

(Print name)

COMMITMENT OF RESEARCHER

I, undersigned, _____ certify

- (a) having explained to the signatory the terms of the present form;
- (b) having answered all questions he/she asked concerning the study;
- (c) having clearly told him/her that he/she is at any moment free to withdraw from the research project described above; and
- (d) that I will give him/her a signed and dated copy of the present document.

Signature: _____ Date: _____