CHANGES IN PATTERNS OF EMG ACTIVITY IN POST-STROKE SUBJECTS FOLLOWING ROBOT-ASSISTED HAND REHABILITATION

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ABSTRACT

A feasibility study of robot-assisted hand rehabilitation post-stroke was conducted to investigate changes in activation patterns of forearm and hand muscles. Four stroke survivors received robot-assisted hand rehabilitation twice a week for two months. Eight healthy age-matched control subjects performed the same exercises as stroke subjects in a single training session. The pattern of activation of forearm and hand muscles was analyzed by performing Principal Component Analysis on the root-mean-square electromyogram. No differences were found in the number of principal components required to account for 95% of the variance between stroke and healthy subjects or pre- and post-rehab even though the majority of subjects used different muscle activation patterns for different exercises and changed their activation patterns from pre- to post-rehab. Stroke subjects were found to have more co-contraction of antagonistic muscles than healthy subjects pre-rehab. In most cases, the amount of co-contraction did not decrease from pre- to post-rehab.
DEDICATION

This work is dedicated to my family and especially to my grandmother.
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CHAPTER 1: LITERATURE REVIEW

1.1 Stroke

A stroke, or cerebrovascular accident (CVA), is the rapidly developing loss of brain function(s) due to disturbance in the blood supply to the brain. Strokes are categorized as either occlusive (due to closure of a blood vessel) or hemorrhagic (due to bleeding from a vessel). They both interrupt the essential supply of oxygen and glucose to the nervous tissues and may cause temporary or severe ischemia (insufficiency of blood supply), therefore an infarction (death of neurons and other cellular elements). Hemorrhage either can be extraparenchymal (occur at the brain surface) or intraparenchymal which may involve cerebral hemispheres, brain stem, or cerebellum. On average, 70-75% of strokes are found to be ischemic in origin, 10-15% are the result of primary intracerebral hemorrhage, around 5% are due to subarachnoid hemorrhage and the rest are of unknown origin (Losseff, 2004). Most of the ischemic strokes are the result of anterior circulation infarcts which involve the anterior and middle cerebral arteries (Winstein et al., 1999).

The most common reasons for occlusive strokes are atherosclerosis and thrombosis while the reasons for hemorrhagic strokes are hypertension or aneurysms. However, both types of stroke may occur at any age from many causes, such as cardiac disease, trauma, infection, neoplasm, blood dyscrasia,
vascular malformation, immunological disorder, and exogenous toxins (Kandel et al., 1991).

Stroke is the third most common cause of death in the developed countries, after ischemic heart disease and all types of cancers combined, second cause of death worldwide and the most important cause of disability among adults (Hankey, 2005).

Epidemiological studies have reported that 15 million people suffer from stroke worldwide each year. From those, 5 million die and another 5 million are permanently disabled (WHO Report, 2007). According to Heart and Stroke Foundation Canada, stroke is the fourth leading cause of death in Canada, and approximately 40,000 to 50,000 strokes occur each year in Canada. About 16,000 of these strokes result in death. About 300,000 people currently live with the effects of stroke. The rate of strokes is likely to increase due to the aging of the population (WHO Report, 2007). The incidence of new cases of first-ever stroke is about 0.2% of the population (Hankey, 2005).

The severity of the impairment and the 30-day fatality rate was found to be much higher (30%) in patients with hemorrhages than in the cerebral infarction category. Lacunar stroke had the lowest fatality rate (1%) within the cerebral infarction category compared to the other subtypes (10-13%) (Foulkes et al., 1988). In another source, the 1-month overall fatality rate was reported as 20-25% after the first stroke, 10-15% after cerebral infarction and 40-50% after primary intracerebral or subarachnoid hemorrhage (Losseff, 2004). There is a
20% chance for a stroke survivor to have another stroke within 2 years (Heart and Stroke Foundation of Canada).

Some of the impairments that can result from stroke are impaired pain and temperature sensation, limb ataxia (gross lack of coordination of muscle movements), Homonymous hemianopsia (loss of vision in the same visual field of both eyes), aphasia (difficulty in speech or speech comprehension and reading), motor apraxia (disturbance of learned motor acts), alexia (inability to read) and behavioural disturbances.

Stroke has a big economic burden on both the health care system and the patients. It has been reported that 4-6% of health care budgets, depending on the country, goes to stroke treatment and prevention each year (Hankey, 2005), and the mean lifetime cost of ischemic stroke for a patient is approximately $150,000 (Barnes, 2005). Yet, it is difficult to estimate the real cost of stroke because of the loss of earnings and production and unpaid caregiver time of the family.

Finally, stroke has a negative physiological, social and economic effect on both patients and their relatives. Therefore understanding the underlying mechanisms of stroke in order to improve the treatment techniques and outcomes has substantial financial and social implications.

1.2 Motor and sensory impairments after stroke

Stroke, which causes damage to the brain tissue by cutting off the blood flow, impairs motor and sensory functions of the contralateral limbs. A large
proportion of ischemic strokes occur in the distribution of the middle cerebral artery which includes the blood supply to the primary sensorimotor cortices (Winstein et al., 1999). The severity and the nature of impairment are highly dependent on the etiology, location and the intensity of the stroke. For example, Lacunar strokes require much shorter hospitalization and produce less severity of impairment than any other type of stroke (Foulkes et al., 1988). Location of a stroke is associated with the type of damage to brain functions. For example, a stroke in the brainstem, thalamus and sensory cortex would cause damage to the sensory system, whereas a stroke in the parietal lobe and motor cortex would result in damage to the motor system (Pause et al., 1989). Some of the sensory impairments include discriminative sensory impairment i.e. stereognosis, loss of proprioception or somatosensory impairments i.e. loss of tactile stimuli recognition. However, motor impairments appear more frequently than sensory impairments following stroke.

Upper motor neuron (UMN) syndrome is used to describe patients with motor dysfunction secondary to lesion of cortical, subcortical or spinal cord structures. Burke (Burke, 1988) has categorized motor impairments of patients with UMN syndrome as having positive and negative symptoms. Positive symptoms, or abnormal behaviours, are abnormal posture, exaggerated exteroceptive (cutaneous) reflexes, flexor withdrawal spasm, extensor spasm and Babinski reflex. Negative symptoms, or performance deficits, are weakness and loss of dexterity, particularly for fine hand manipulation. Negative symptoms are more frequently observed in hemiparetic patients such as those suffering
from cerebrovascular accidents (Katz and Rymer, 1989), and major functional deficits following brain damage are largely due to negative features (Burke, 1988).

Spasticity, one component of UMN syndrome, is described as a motor neuron disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks resulting from hyperexcitability of the stretch reflex (Lance and Burke, 1974). Muscle tone is normally regulated by the corticoreticulospinal tract which exerts a tonic inhibition of the stretch reflex arc. This inhibition balances the constant background facilitation from the pontine reticulospinal and lateral vestibulospinal pathways to adjust the muscle tone appropriately for voluntary movement. Consequently, a lesion of the descending tracts contributes to spastic hypertonia either via decreased presynaptic inhibition to lower motoneurons (e.g. from the lateral vestibulospinal tract), or indirectly, by reduced inhibition from inhibitory interneurons within the spinal reflex pathway (Katz and Rymer, 1989).

Powers and colleagues suggested that a decrease in the stretch reflex threshold, the threshold for activation of a relaxed muscle by the tonic stretch reflex, of a muscle following stroke is the main cause of spastic hypertonia. Their study compared the static and dynamic torque and electromyographic (EMG) activity of initially active spastic elbow muscles to pre-activated normal muscles at different velocities (Powers et al, 1989). Results of the study suggested that the velocity dependence of the stretch reflex in spastic muscles results from a lower stretch reflex threshold (amount of stretch required to activate a muscle).
The most likely explanation for the reduced threshold of the stretch reflex, which is equivalent to the threshold for motoneuron recruitment, is that motoneurons are more depolarized as a result of a net increase in tonic excitatory synaptic input (resulting from the loss of descending inhibitory pathways) from descending pathways or regional interneurons within the spinal reflex arc (Katz and Rymer, 1989). Moreover, it has been postulated that spastic hypertonia is the result of a decrease in stretch reflex threshold without a significant increase in the amount of force required to extend the limb in proportion to an increase in joint angle which is determined as reflex “gain” and measured by the angular stiffness (Katz and Rymer, 1989). Because the stretch reflex threshold of a spastic muscle decreases following stroke or, in other words, the excitability of the motoneurons increase, a smaller amount of stretch and/or slower velocity of motion would be sufficient to elicit a stretch reflex (Powers et al., 1989). Thus, stretch-induced activation of spastic muscles would limit the range of voluntary movement of the paretic limb that could be achieved by the opposing muscles.

Although the hyperexcitability of the motoneurons is the most widely accepted explanation for the increased spastic hypertonia in post-stroke, another school of thought suggests that pathological changes in passive muscle stiffness and contracture in muscles\(^1\) are responsible for the spasticity following stroke. According to O’Dwyer and colleagues (O’Dwyer et al., 1996), muscle contracture

\(^1\) In this study, the muscle contracture is defined as “shortening of muscle length due to a decrease in the number of sarcomeres in series along the myofibrils, accompanied by an increase in the resistance to passive stretch”. Consequently, the range of joint motion is reduced both by the shortening of the muscle fibres and by the loss of muscle compliance (O’Dwyer et al., 1996).
causes the muscle and its spindles to be shorter than normal, causing force enhancement during stretch by increasing the muscle stiffness and the size of the stretch reflex.

Spastic hypertonia has clinical importance for those taking care of patients in clinics. However, the negative symptoms of UMN syndrome such as weakness and/or lack of dexterity in the hand, as well as co-contraction of agonist and antagonist muscles, are probably more disabling than spasticity for the patients who suffer from stroke. Muscle weakness, one of the most common stroke related impairments, might occur both contralateral and ipsilateral to the lesion (Ada et al., 2003; Colebatch and Gandevia, 1989; Tyson et al, 2006; Lawrence et al., 2001; Chae et al., 2002; Gowland et al., 1992; Tang and Rymer, 1981), and is associated with increased muscle tone as well as hypersensitive stretch reflexes (Delisa, 1982; Powers et al, 1989; Colebatch and Gandevia, 1989; Levin et al, 2000).

Kamper and Rymer (Kamper and Rymer, 2001) found that patients with flexor spasticity have great difficulty in contracting extensor muscles because of reduced voluntary activation (i.e. weakness) of these muscles and increased co-activation of flexor and extensor muscles. They have attributed inappropriate co-activation to alteration in reciprocal inhibition between flexor and extensor muscles, selective activation of cortical areas responsible for co-activation, and central reorganization following stroke. Abnormal muscle co-contraction or spastic restraint was observed particularly during voluntary motion. However, there has been a controversy whether abnormal co-contraction of muscles is a
contributor to the voluntary movement deficit in post-stroke (Knuttson and Martensson, 1980; Hammond et al., 1988) or not (Gowland et al., 1992; Sahrmann and Norton, 1977; Tang and Rymer, 1981). Some concluded that the abnormal co-activation of agonist and antagonist muscles resulting from impaired antagonist inhibition is a crucial component of the motor impairment post-stroke (Knuttson and Martensson, 1980; Hammond et al., 1988), while others suggested that the impairment of voluntary movement is attributable to weakness resulting from “inadequate recruitment of motor units in the agonist muscles which creates an inability to generate sufficient force to complete a motor task”, rather than impaired antagonist inhibition (Gowland et al., 1992; Sahrmann and Norton, 1977; Tang and Rymer, 1981). Because of the decreased force output of individual motor units caused by the reduction in the mean motor unit discharge rate, many more motor units would have to be recruited in order to produce a given level of force. This situation could give rise to weakness when all readily accessible motor units are recruited, and may lead to early fatigue because of increased sense of effort at lower levels of activation (Tang and Rymer, 1981).

In a more recent study, Levin and colleagues (Levin et al., 2000) measured the ability of hemiparetic subjects to produce coordinated changes in agonist and antagonist stretch reflex thresholds during voluntary elbow flexion and extension. In this study, agonist/antagonist muscle co-activation was investigated based on the equilibrium point model which associates the range of reciprocal innervations with stretch reflex threshold angles. The model allows
these threshold angles to move beyond the limits of the physiological range of joint motion. According to the model, co-activation of flexors and extensor muscles is triggered when the threshold angle is outside of this physiological range. In healthy subjects, the range of regulation of stretch reflex is larger than the physiological range of motion. This enables the full activation to full relaxation of the muscle, which is necessary for the full repertoire of possible movements. However, in the case of CNS lesion, the limits of regulation of stretch reflex narrows. Therefore, the ability to regulate muscle force in all parts of the physiological range is lost (Levin et al., 2000). Levin and colleagues have summarized the possible mechanisms behind the disturbed coordination of agonist/antagonist muscle activity as a reduced stretch reflex threshold of flexors which interrupts the relaxation of flexor muscles at any angle beyond the stretch reflex threshold of the flexors. This will cause an increase in the extensor's threshold, thus causing a co-activation of flexor and extensor muscles. They have also concluded that the voluntary movement deficit in the arm flexion is related to not only spasticity of flexors, but also weakness in the extensors to the extent that stroke survivors were found to be unable to generate torque in the weak extensors to overcome the resistance of reflexively co-activated flexors.

In addition, there have been conflicting reports about the degree of co-activation of agonist and antagonist muscles in the upper limb following cerebral damage as measured by the EMG activity during isometric contraction (Tang and Rymer, 1981; Hammond et al., 1988; Dewald et al., 1995). Tang and Rymer found no co-contraction in antagonistic muscles of hemiparetic arm after
measuring the EMG activity of elbow flexors, while Hammond and colleagues reported a greater co-contraction ratio in wrist flexor and extensor muscles for stroke patients than controls and finally, Dewald and colleagues showed strong co-activation selectively between elbow flexors and extensors in some of their hemiparetic subjects while others did not exhibit the same behaviour. They have also reported additional novel co-activation patterns of different muscle pairs of spastic-paretic limb, in addition to consistent EMG co-activation patterns seen in both normal and hemiparetic limbs. They stated that the abnormal co-activation patterns in the hemiparetic limb may be the consequence of a decrease in the number of possible muscle synergies that the CNS is able to produce\(^2\) and could be the primary source of dysfunction in many hemiparetic patients after stroke. The reason for the depletion in synergies might be due to the damage to CNS descending pathways, increased reliance on residual descending brainstem pathways or to changes in spinal interneuronal excitability.

There are several studies that summarize the contributing factors of the pattern of muscle weakness, one of the negative symptoms of UMN. The factors listed in those studies are: decreased supraspinal efferent input by the interruption of the corticospinal pathways and likely involvement of several descending fibre systems, reduced recruitment of motoneurons (Tang and Rymer, 1981) associated with both alteration in frequency of firing of motor units and reduction in the number of active motor units (Gowland et al., 1992), and; loss of inhibitory mechanisms which can cause hyperexcitability of the spinal

\(^2\) “Muscle synergies” refers to coordinated activation of different muscles used by the CNS to reduce the complexity of the control of the neuromuscular system for a given task (as explained on page 65).
motor pool and lead to loss of the orderly recruitment of motor units (Chae et al., 2002; Levin et al., 2000). In one study, Tyson and colleagues (Tyson et al., 2006) assessed the distribution of weakness in the acute post-stroke stage and found more severe weakness in the arm than in the leg. The difference between the upper and lower limb weakness ranged from 6% to 33% (Lawrence et al., 2001). Furthermore, selective weakness of particular muscle groups was observed with hemiplegia, such as more noticeable weakness of distal rather than proximal muscles for both the arm and leg (Colebatch and Gandevia, 1989; Chae et al., 2002), and more severe weakness in flexor muscles of the wrist and fingers (Colebatch and Gandevia, 1989).

Reach-to-grasp (Lang et al., 2005), or producing coordinated grip forces to grasp and lift an object (Nowak et al., 2003), and the individuation of a single finger (Raghavan et al., 2006; Cruz et al., 2005) with the contralesional hand, are other impaired upper extremity functions following stroke.

Motor impairments of the upper extremity have a higher impact on stroke survivors’ independency because of their effect in the ability to perform activities of daily living (ADL), which are significantly dependent on a functional hand grasp and release (Chae et al., 2002). Only about 5% of individuals paralyzed by stroke regain full arm and hand function, and about 20% regain no functional use (Gowland et al., 1992). Considering the fact that 73% to 88% of the first-time-ever strokes result in an acute hemiparesis of the upper and/or lower extremity (Foulkes et al., 1988), and the greatest motor and sensory loss following stroke is found in the hand (Colebatch and Gandevia, 1989; Kandel et al., 1991) and
fingers (Colebatch and Gandevia, 1989), it is crucial to understand the effects of stroke on hand functions, as well as to investigate techniques to help for recovery in order to improve the life quality and independency of stroke survivors.

Previous studies measuring the deficits in dexterity of the stroke survivor’s hand while performing functional tasks, used quantitative measures such as speed, accuracy, efficiency of the movement, performance (Lang et al., 2005) or grip forces (Nowak et al., 2007, Raghavan et al., 2006). However, wrist and finger muscle activation patterns during functional activities, or the abnormalities in these patterns, as well as the changes in these patterns from pre- to post-rehab have not been investigated yet. Furthermore, the degree of co-activation in forearm and hand muscles while performing a hand related motor task (i.e. flexion and extension of fingers), has not been quantified in any study. Understanding the muscle activation patterns occurring post stroke and the abnormalities in these patterns would consequently help clinicians to design the most effective therapies and to choose the most suitable hand therapy techniques for their patients. It would also give valuable information to follow-up patients’ improvement by the changes in these patterns.

1.3 Recovery after stroke

1.3.1 Epidemiology of Recovery

According to the Heart and Stroke Foundation of Canada, only 10% of the strokes that occur in Canada are followed by complete recovery, 25% of stroke survivors are left with a minor impairment or disability, and 40% with a moderate
to severe impairment. Another 10% stay so severely disabled that they require long-term care (Heart and Stroke Foundation of Canada Report, 2000).

Recent longitudinal studies have found that at 6-12 months post stroke, 25-30% of survivors have difficulty with bathing or using stairs, 30-40% are depressed (10-15% are severely so), 50% need help for performing their activities of daily living, such as housework, meal preparation or shopping, and a similar number lack a meaningful social, recreational or occupational activity during the day (Losseff, 2004). Between 3 and 12 months and 5 years post stroke, motor performance and functional independence deteriorate in up to 40% of survivors, largely due to ageing and co-morbidity rather than recurrent stroke (Losseff, 2004). For the upper extremity, only about 5% of individuals paralyzed by stroke regain full arm and hand function, and about 20% regain no functional use (Gowland et al., 1992).

1.3.2 Factors Affecting Recovery

Like post-stroke impairments, the recovery from stroke also depends on several anatomical, physiological and treatment-related factors, such as etiology, location, and severity of stroke or the onset, the amount or the duration of therapy, and the type and context of the tasks practiced by the patient (Dobkin, 2008). For instance, hemorrhagic strokes usually have poorer prognosis than ischemic stroke (Foulkes et al., 1988) and the earlier the treatment starts, the more improvement can be achieved. There is individual variability in patients recovering from stroke which makes the prediction of timing and the extent of the recovery relatively difficult. Different sources provide different evidence for the
timeline of recovery. Some state that the most of the recovery occurs by six months post-stroke or the functions remain permanently lost, while the others state that the patients who are severely impaired initially have longer recovery times after stroke onset (Connell, 2007). It has been reported that motor function improves for more than half a year after stroke (Fugl-Meyer, 1975) or up to one year (Kotila et al., 1984). There is evidence to suggest that the improvement of the hand functions can continue four to fifteen years after stroke onset (Johannson, 2000).

Animal studies of stroke showed that specific types of motor training (e.g. skill, strength, endurance) can change the structure and function of the distributed sensorimotor systems. For instance, skills training induces plasticity in M1 and related motor cortices which are involved in performing skilled movements, while strength training can alter the excitability of spinal motor neurons, but does not result in reorganization of the motor map. By strength training a patient can increase the rate of torque production and gain the ability to vary the discharge rate of motor units which would influence the ability to control the submaximal forces that are required to perform everyday tasks. Consequently, the experience-specific plasticity provides opportunities to enhance post-stroke recovery in different ways (Dobkin, 2008).

On the other hand, task-specific training seems to benefit stroke patients more than general exercise patterns in terms of improving daily functions (Dobkin, 2008; Winstein et al., 2004). Winstein and colleagues showed that the long-term benefits of rehabilitation in humans using a “functional task approach”
which involved repeatable tasks and tasks with functional goals (i.e. grasping) were significantly greater 9 months after stroke compared to rehabilitation with a “resistance-strength approach”. Furthermore, repeated attempts to use the affected hand in daily activities can improve performance further in patients with less severe stroke even after retraining is terminated (Han et al., 2008).

Even though recovery in the upper extremity takes longer than recovery in the lower extremity, it has been reported that it usually recovers to nearly the same degree as the leg (Fugl-Meyer, 1975; Duncan et al., 1994). However, the longer time period of recovery of the upper extremity, specifically the hand, is much more disabling because of the difficulty of performing activities of daily living (ADL), and the difficulty in compensating for upper extremity functions which require finer motor control (Duncan et al., 1994).

In terms of the other factors affecting the recovery from stroke, Kotila et al. (1984) showed that patients with a lesion in the left hemisphere did not have differences in recovery in terms of independency in ADL compared to those who had a lesion in the right hemisphere. Even though the improvement of neurological and neuropsychological deficits can be seen from an early stage up to one year after stroke, younger patients (<65 years) are found to have better recovery and independency in activities of daily living than the older (>65 years) age group (Kotila et al., 1984).
1.3.3 Neuroplasticity

It is widely accepted that spontaneous recovery of motor functions following stroke occurs in acute\(^3\) or subacute\(^4\) stages of stroke probably due to the recovery of injured neurons. Further improvement in function is accompanied by relearning which involves neuroplasticity\(^5\) (Nudo and Duncan, 2004; Brown, 2006; Dobkin, 2008; Rossini et al., 2007).

Functional neuroimaging studies have revealed that cortical plasticity in both hemispheres is associated with sensorimotor recovery, such that the activity of contralesional non-primary motor areas decreases, while the activity of ipsilesional primary sensorimotor areas increases with the improvement of motor performance. (Zemke et al., 2003; Loubinoux et al.,2003; Dobkin, 2008; Rossini et al., 2007). Although poor recovery is reported to be associated with the activation of the contralesional sensory motor area (Rossini et al., 2007), the neural mechanism of plasticity are poorly understood. Dobkin (2008) has proposed several possible mechanisms that might account for the neuroplasticity, e.g. the spared pathways of supraspinal descending projections can be used to relearn a movement pattern when some of M1 or the corticospinal pathway is infarcted or uncrossed fibres of the corticospinal tract and axons that recross might provide input that can partially compensate for the loss of motor

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\(^3\) Acute stage of stroke refers to the period of a few hours, just after the stroke has occurred. This phase is characterized by a need of urgent care and rapid progress. It ends when the vital functions (e.g. respiration or circulation of the blood) are stable.

\(^4\) Subacute stage of stroke usually refers to the early phase of stroke and lasts between four weeks and six months. During this period, progress occurs at a slower rate than for the acute phase, but stroke patients can expect some degree of spontaneous recovery.

\(^5\) Cerebral plasticity is the neural ability to change the properties of the activation induced by inputs from peripheral receptors and/or other brain areas (Rossini et al, 2007).
function. In addition, interhemispheric connections via the corpus colloseum between cortical motor areas might also contribute to the gain of motor function. However, it is still not well known whether reorganization is due to modulation of existing synapses or to the physical growth of new axonal processes (Nudo et al., 1996).

1.3.4 Progress of Recovery Post-Stroke

The restoration of motor function in both upper and lower extremities follows a general pattern in which patients exhibit certain phenomena during distinct phases or stages of the recovery process. However, it should be noted that the recovery process is continuous and new phenomena might appear gradually and/or these phenomena might overlap in different stages or phases of recovery (Twitchell, 1951).

Immediately following the onset of stroke, the patient loses the ability to move the affected limbs voluntarily, the so-called total paralyses. First, tendon reflexes in the involved extremities and later resistance to passive movement decrease or disappear. This is called flaccidity, if no tendon reflexes can be elicited in the involved extremity's muscles. As the acute stage of stroke ends, the tendon reflexes become more active on the side contralateral than the side ipsilateral to the lesion, followed by minimal resistance to passive movement. This is reported as first seen in flexors of wrist and hand muscles (Twitchell, 1951). Spasticity develops and involves other muscle groups as the resistance gradually increases in intensity.
As the recovery continues, patients regain their first voluntary movements. Usually, these voluntary movements occur in *movement synergies* ("associated reactions"\(^6\)) of the hemiplegic limb, if the voluntary effort which elicits the reactions is strong and of some duration (Brunnstrom, 1956). Patients often have difficulty in performing a single, isolated movement of a joint compared to strong synergistic movements of multiple joints. The flexor and extensor synergies were defined by Brunnstrom, (1956) (discussed in more detail in Chapter 1.4.1). Flexor synergies often develop earlier than extensor synergies in the upper extremity. Severe spasticity is present at this level of recovery and starts to decrease in the next stage.

At the next stage of recovery, the patient is able perform a few simple movements which do not involve the synergies. However, the movements are usually slow or not well coordinated. Reciprocal movements in the synergies can also be observed at this stage. As the recovery progress, some individual joint movements are possible, spasticity continues to decrease, and the patient is able to perform several functional activities without synergistic movements. At the last stage of recovery the spasticity almost disappears. Individual finger movements can be performed with controlled speed and direction. Although some incoordination might be present, it is often only apparent during rapid reciprocal movements (Twitchell, 1951). Progress during recovery always follows the same pattern and never skips a stage. However, it might reach a plateau depending on the extent of the damage (Perry, 1967). Usually, the recovery of distal functions

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\(^6\) The associated reactions are automatic responses that reinforce or change the posture of the affected extremity or extremities when some other part of the body is brought into action by voluntary effort and/or artificial reflex stimulation (Brunnstrom, 1956).
takes much longer than proximal functions, as does the upper extremity compared to the lower extremity (Fugl-Meyer, 1975; Duncan et al., 1994; Otman et al., 2004).

1.3.5 Recovery Pattern of Hand Functions

Because of the high degree of cortical control, recovery of hand functions does not follow a distinct pattern as in the upper and lower extremity. Reflex activity and mass reactions become active in the early stage of wrist and hand recovery, and these reflex movements are used in rehabilitation in order to enable gross functions (Perry, 1967).

Even though the recovery of wrist and fingers does not follow a well defined pattern, Twitchell (Twitchell, 1951) described the process in a similar order. In early stroke, tendon reflexes become hyperactive and spasticity develops. Increased spasticity first appears in palmar flexors of the wrist and fingers. At this stage, voluntary finger flexion can be obtained by proprioceptive stimuli such as finger-jerks or passive stretches while the patient tries to flex his/her fingers simultaneously. In addition, a proximal traction response can be elicited. Later, patients can reach a level in which movements can be initiated without proprioceptive stimuli. With the increased control of voluntary flexion in the upper extremity and fingers, spasticity significantly decreases in these muscle groups. At this stage of recovery, flexion of all fingers, and possibly individual finger flexion, can be facilitated by a tactile stimulus of the palm by

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7 Proximal traction response: Passive traction upon the flexor muscles of an upper extremity can by itself causes an active contraction of all the flexor muscles of the limb.
moving distally across the palm of the hand. Spasticity continues to decrease in wrist and fingers while finger movements become more powerful and dexterous, and a “well-defined grasp reflex”\textsuperscript{8} can be obtained. Progress of hand function recovery is discussed further under section 1.4 Post-stroke rehabilitation.

1.3.6 Why is rehabilitation important for the recovery of hand functions?

Nudo and colleagues (Nudo et al., 1996) demonstrated that rehabilitative training plays a major role in the functional reorganization in the hand area of motor cortex of adult primates after a focal ischemic infarct, as might occur in stroke. They have examined lesion-induced plasticity in primary motor cortex of primates by using the intracortical microstimulation (ICMS) technique and derived maps of the hand representation in monkeys before and after focal ischemic infarcts. An intensive rehabilitation procedure was applied to one group of the monkeys (rehabilitation group) but not to others (control group) after the infarction and between two mapping sessions. The results of the study revealed that rehabilitative training prevents further losses of hand area in the adjacent, intact tissue and may direct the intact tissue to “take over” the function of the damaged region. It is possible that rehabilitative training after the injury could result in enhancement of representational plasticity and of functional motor recovery in humans.

Dobkin (2008) reported that if an adequate percentage of ascending and descending pathways are present in poststroke, a rehabilitation program adapted

\textsuperscript{8}Grasp reflex: A reflex which can be facilitated by a tactile stimulus applied on certain parts of the palmar surface of hand and fingers. These parts cover most of the palm areas, with the exception of ulnar side, and become weaker on thumb. In order to elicit the reflex, the stimulus must be in a direction from distal to proximal.
to the needs of a patient can be used to retrain adequate reaching and grasping with the hand. He stated that well-designed post-stroke training is an essential factor which can shape the final functional status of the patient. Physiotherapy, therefore, is a vital part of the recovery process after stroke in which physiotherapists can retrain individuals to improve functional activities rather than only isolated neuromuscular components of movements or movement patterns. A person with a stroke may improve grip force as measured with a dynamometer but may not be able to use that force to pick up a jar (Richards and Pohl, 1999). In such a case, a physiotherapist can evaluate the current abilities and/or disabilities of the patient and can retrain these particular functions that the patient has difficulty performing and/or enhance the abilities of the patient by using several rehabilitative techniques.

1.4 Post-stroke Rehabilitation

Immediate acute care starts in hospital right after patients have been diagnosed with stroke. A treatment program can be designed in 48 hours, if the patient’s clinical condition is stable. The program design depends on severity of impairment, level of activity and participation, and needs or priorities of the patient. In order to promote the greatest gain, patients receive specific therapies in organized stroke units as well as supportive medical care. These units may include physiotherapy, occupational therapy, speech and language therapy, clinical neuropsychology and nutrition and dietetic service (Losseff, 2004).
1.4.1 Physical Therapy and Rehabilitation

Physical therapy is the most commonly used intervention for recovery after stroke and primarily aims to improve motor functions and independency in ADL by reducing spasticity and strengthening the muscles (Peppen and Kwakkel, 2004). Conventional therapy techniques involve range of motion (ROM), strengthening exercises, mobilization activities, and compensation techniques.

1.4.1.1 Neurophysiologic approaches

Neurophysiologic approaches, which are designed as neuromuscular re-education techniques in order to excite neural and physiologic structures, are often used in post-stroke rehabilitation. There are several techniques that are structured based on the stages of recovery after hemiplegia such as Brunnstrom’s movement therapy (Brunnstrom, 1956), Bobath’s treatment (Bobath, 1977), proprioceptive neuromuscular facilitation (PNF) principles, Rood’s sensorimotor approach, and Margaret Johnstone’s method.

These facilitation models are based on the most accepted top-down model of the CNS. In this model, newer brain structures control more primitive brain structures. After stroke, primitive brain centers are released from the control of higher brain centres due to the damage of CNS. The abnormal movements resulting from stroke are, therefore, viewed as being under the control of primitive brain centres. Motor rehabilitation assists the higher-level centers to re-learn the control of the primitive centres by inhibiting abnormal movement and facilitating normal movement through the delivery of controlled sensory input. Even though Bobath’s technique is the most widely practiced approach in many countries
(Barnes et al., 2005), Brunnstrom’s movement therapy, PNF and Margaret Johnstone’s method are also commonly used therapy techniques.

1.4.1.1 Brunnstrom’s movement therapy

The Brunnstrom technique was developed by a physiotherapist, Signe Brunnstrom, as the first systematic technique for the treatment of motor function deficits after stroke. The theoretical basis of this technique is based on normal development of CNS and sensory-motor system. According to Brunnstrom, after a lesion in CNS, the patient returns to a subcortical level of control of movement (Brunnstrom, 1956) because of the lesion in higher motor centers and the loss of inhibitory control mechanism of the CNS. Primitive reflexes such as tonic neck reflex\textsuperscript{9}, tonic lumbar reflex\textsuperscript{10} or tonic labyrinthine reflex\textsuperscript{11} and increased tendon reflexes appear as the first movement ability of the patient.

The objective of this technique is to progress from subcortical to cortical control of muscle function by using movement synergies and reflex training after a critical evaluation of the patient to determine his/her stage of recovery. The Brunnstrom approach follows four stages: 1- elicitation of movement synergies on a reflex level; 2- capturing voluntary control of synergies; 3- inhibition of synergies by using antagonistic synergies, and; 4- eliciting voluntary hand and finger functions (Perry, 1967; Otman et al., 2004).

\begin{itemize}
  \item \textsuperscript{9} Tonic Neck Reflex: Flexion or extension of the neck elicits the same movement in arms and the antagonistic movement in legs (Symmetrical Tonic Neck Reflex). Rotation of the head elicits limb extension on the side towards the rotation and limb flexion on the side opposite the rotation (Asymmetrical Tonic Neck Reflex).
  \item \textsuperscript{10} Tonic Lumbar Reflex: Rotation of trunk toward the affected side facilitates flexion of upper and extension of lower extremities, rotation away from affected side facilitates extension of upper and flexion of lower extremities.
  \item \textsuperscript{11} Tonic Labyrinthine Reflex: Supine position of the patient increases extensor tonus and prone position increases flexor tonus.
\end{itemize}
Synergistic movement patterns in hemiplegia involve certain movements which are difficult for patients to perform individually. Flexor synergy, which is found to be more dominant in the upper extremity, is a shortening synkinesis. It involves muscle groups, which shorten the extremity when they contract. Extensor synergy is a lengthening synkinesis and the muscle groups involved in the extensor synergy lengthen the extremity when they are activated. Wrist and fingers do not follow either synergy pattern of the upper extremity. Wrist extension might be a component of extensor synergy, while wrist flexion might belong to the flexor synergy. However, this is not certain and could be different for each patient (Otman et al., 2004). Finger flexion might accompany both flexion and extension synergies, but finger extension never appears as a component of either of the two synergies (Brunnstrom, 1956). Brunnstrom reported that post-stroke recovery occurs in specific phases (Table 1).
Table 1: Recovery phases of hand functions.

<table>
<thead>
<tr>
<th>PHASE 1</th>
<th>PHASE 2</th>
<th>PHASE 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>No hand function</td>
<td>Gross grasp and minimal finger flexion</td>
<td>Gross grasp and hook grip</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Patient cannot release the object</td>
</tr>
<tr>
<td>PHASE 4</td>
<td>PHASE 5</td>
<td>PHASE 6</td>
</tr>
<tr>
<td>Gross grasp, lateral grip, limited finger extension, some thumb movements</td>
<td>Palmar, spherical and cylinder grip</td>
<td>All grips, individual finger movements and voluntary extension</td>
</tr>
<tr>
<td></td>
<td>Patient can release the object</td>
<td></td>
</tr>
</tbody>
</table>

Six phases of motor improvement of hand reported by Brunnstrom S. Adapted from Otman et al., 2004.

Because hand and finger functions have a high degree of cortical control, the training of these functions requires a different process than the upper extremity. Hand rehabilitation in the Brunnstrom technique follows the stages of recovery reported by Twitchell (Twitchell, 1951) (see section 1.3.5). Positioning and methods of sensory stimulation are often used for hand rehabilitation in the Brunnstrom technique (Perry, 1967). The first objective of the rehabilitation is to elicit gross grasp by using reflex movements and mass reactions that appear in early stages of recovery, such as the proximal traction response and the grasp reflex. The second objective is to obtain wrist stabilization, and finally to achieve hand release by finger extension (Otman et al., 2004).
1.4.1.2 Bobath’s treatment technique

Berta Bobath developed and practiced neurodevelopment techniques on children with cerebral palsy in 1950’s and 60’s and began applying her approach (i.e. neurodevelopmental treatment) to patients with CNS lesions in the 1970’s.

According to Bobath, a lesion resulting from UMN syndrome causes a disruption of the “normal postural reflex mechanism” which is controlled by the CNS. This mechanism is responsible for normal postural tone, reciprocal innervations, and automatic movement patterns. After a lesion in higher control centers, such as in stroke, patterns of activity appear to be controlled at a lower level which results in abnormalities in postural tone (e.g. hypertonia), reciprocal innervations (e.g. excessive co-contraction) and movement patterns (Barnes et al., 2005). Disinhibition of tonic reflex patterns associated with spasticity, abnormal reciprocal innervations, loss of memory of former movement patterns and sensory deficits are the common problems which interferes with normal function or makes normal functions impossible.

Bobath (1977) has stated that, “compensation therapies are largely responsible for an increase in the spasticity and a decrease in the activity of the involved side. Spasticity is also reinforced by associated reactions produced by the effort involved in the unilateral use of the unaffected side, and through lack of balance and fear of falling. Synergy patterns are useless for performing selective functional skills. It is because a patient needs to be able to perform in any required pattern in order to achieve a functional skill, not only in a couple of movement synergies”. Consequently, Bobath’s method avoids these synergy
patterns and introduces selective movements as early in treatment as possible by focusing on re-experiencing the sensation of normal movements without spasticity. The objective of the Bobath approach is to prepare the patient for functional activity by inhibiting “abnormal” movement patterns and facilitating more “normal” movement (Barnes et al., 2005). Since the spasticity of neck and trunk influences that of the limbs and spasticity of the upper extremity influences the lower extremity or vice versa, arm and leg are not treated separately in this method.

The basic principles of the Bobath’s treatment technique rely on: 1- reducing the spasticity in order to have effortless movement; 2- increasing the postural tonus, if the tonus is too low or certain muscles appear to be weak, with the help of proprioceptive and tactile stimulation; 3- introducing selective movements as early as possible; 4- avoiding or inhibiting the associated reactions by counteracting the patterns of spasticity, and; 5- facilitating and stimulating balance reactions and the use of arm/hand for support together with weight bearing of the affected side because patients need well developed normal sensation on the affected side (Bobath, 1977).

According to Bobath, ideal therapy must be designed in three steps. First, the therapist must prepare specific movement patterns necessary for the function that the patient wants to improve; second, to be able to do this, the therapist needs to observe and analyze the patient’s abnormal movement and posture patterns in order to know how these movements disturb normal functions, and; third, the therapist needs to find out which normal patterns that are necessary for
the skilled movement are missing. Every movement practiced in treatment should serve as a preparation for functional use and should be as similar as possible to movements needed in daily life. It should be noted that even though the muscles may function well during the developmental exercise, it does not mean that the patient can adapt these movement patterns into the activities of daily living.

1.4.1.1.3 Margaret Johnstone’s method

Margaret Johnstone established her technique in early 1980’s. The principle of Johnstone’s method relies on establishing a balance between facilitation-inhibition mechanisms of the sensorimotor system. It was originally developed for hemiplegic patients. The method follows the normal development of the CNS where recovery occurs from proximal to distal. The objective is first to improve gross motor functions and then to move on to fine motor functions. It supports the idea of inhibition of abnormal movement patterns as early in the treatment as possible. One of the differences compared to other facilitation techniques is that Johnstone’s method emphasizes the importance of positioning in the stages of recovery, and uses pressure splints in order to position the extremity (Otman et al., 2004).

The main difference between these techniques is that Bobath’s and Johnstone’s treatment techniques emphasize the inhibition of synergistic patterns and spasticity from the early stages of the therapy while Brunnstrom’s technique supports the facilitation and usage of synergistic patterns in order to have normal movement patterns; Brunnstrom’s technique is more effectively used in chronic patients, while Johnstone and Bobath approaches are preferred in the acute
stages of stroke for both upper and lower extremities. During the late recovery of stroke, all these methods follow similar principles; therefore, exercises developed by these methods can be used together in these late recovery stages.

Several studies have compared the outcomes of different facilitation models. For example, Dickstein (1986) compared outcomes of PNF, Bobath and traditional exercises by measuring spasticity, isolated wrist extension, ability to perform ADL, and Wagenaar et al. (1990) compared Bobath and Brunnstrom methods by measuring upper limb functions. Neither study found a relationship between the amount or the type of upper extremity improvement and the type of intervention. Both showed improved ability to perform ADL; however, they failed to measure the ability to perform ADL specifically with the affected extremity, which made it unclear whether the better performance in ADL was the result of improved motor skills, increased compensatory behaviour or both.

Rehabilitation can also be enhanced by several newer techniques, such as functional electric stimulation (FES, which is the delivery of electric pulses to affected muscles to elicit muscle contraction for movement), electromyographic biofeedback (auditory or visual signal that provides the patient with information about muscle contraction), constraint induced therapy (CIT, a protocol which forces the use of affected limb by restraining the use of the less affected limb with a mitt) and transcranial electric stimulation (TMS, a non-invasive method to excite neurons in the brain).
1.4.1.2 Effectiveness of facilitation techniques in performing motor skills with upper extremity and hand

In a study, Bütefisch et al. (1995) compared standardized training of hand and finger movements with a therapeutic rehabilitation technique following the Bobath concept. The training consisted of repetitive hand and finger flexion and extensions against various loads, and the therapeutic approach aimed at reducing spasticity without reinforcing the activity of the affected hand. Patients who received therapeutic rehabilitation alone did not show a significant improvement in the motor capacity of the hand. On the other hand, the training program which emphasized early initiation of active movement rather than focusing on spasticity reduction resulted in significant improvement in hand motor performance. Kraft et al. (1992) showed that even though specific types of therapy (i.e. electric stimulation, biofeedback and PNF technique) can improve the gross function and strength of the paretic upper limb, they are not effective in restoring the fine hand and finger movements in chronic stroke patients (i.e. finger tapping and card turning). Later, Richards and Pohl (1999) stated that the most common methods used in the interventions for upper extremity rehabilitation focus on motor rehabilitation facilitation techniques at the movement level. Practice typically involves repetitions of particular movements in a limited number of contexts. Since individuals must be able to vary their upper extremity movements in order to perform real-life actions, improvements in motor skills observed during the practice of training movements may not be indicative of the performance of ADL.
In conclusion, there are many difficulties in rehabilitation of the upper extremity, particularly rehabilitation of hand functions after stroke. Rehabilitation techniques have been more successful for the lower extremity than the upper extremity (Kraft et al., 1992) mostly due to the complex neuromuscular structure and the higher cortical level of control of hand and finger functions. Recovery of upper extremity function requires improved sensation, manipulation and fine control of hand and wrist muscles. Apart from consideration of the anatomical and neurological complexity of the upper limb, other factors can play important role in the success of rehabilitation reported in these studies. For example, the skill of the physiotherapist who applies the therapy, physiological limitations of the patient (e.g. medical problems such as cardiovascular disease, side effects of medication) or psychological conditions (e.g. depression) are some of the important factors affecting the outcomes of therapies. Above all, heterogeneity of the subject groups (i.e. location, severity and age at occurrence of the lesion, previous or subsequent medical problems of patients, psychosocial influences) is a problem intrinsic to stroke rehabilitation research.

In summary, neurophysiologic facilitation techniques are commonly used in rehabilitation therapies of upper and lower extremity together with other treatment approaches. The earliest therapeutic technique was developed by Brunnstrom based on the recovery process of stroke reported by Twitchell and neurological findings of other scientists of the time. Brunnstrom has proposed that the neurological reactions associated with upper motor neuron syndrome can be used to facilitate synergistic movements, and these movements can be
used to entrain normal movement patterns in late recovery. Bobath opposed this idea by stating that recruitment of spastic synergies is not useful for performing functional movements as they might become a habit later in recovery. She claimed that patients need to perform “normal” movement patterns as early in treatment as possible; thus, the spasticity should be reduced starting from the early phases of the therapy and the patient should attempt to perform functional movements. Another therapeutic intervention, Johnstone’s treatment method, followed the basic principles of Bobath and emphasized the importance of positioning of the upper extremity during rehabilitation. All of these facilitation techniques, as well as many others (i.e. PNF), adopt a top-down hierarchical model of CNS. Most therapies used in upper extremity rehabilitation are influenced by these facilitation techniques; however, the effectiveness of these neurofacilitation approaches for improving fine motor function of hand and fingers, and their applicability to activities of daily living is debatable.

1.4.1.3 Motor learning approach

Recently, new therapy approaches have been adopted in which motor learning and active involvement of the patient are being emphasized. The key assumption behind these approaches is that regaining the ability to perform activities of daily living after stroke requires a relearning process that is similar to the learning process for non-impaired individuals (Barnes et al., 2005). Weinstein et al. (1999) showed that damage to sensorimotor areas after stroke impairs “the control and execution of motor skills but not the learning process” of those skills. The aim of a motor learning program is for the patient to relearn everyday tasks
by practicing task- and context-specific activities. In this treatment, the therapist teaches patients to perform the “missing components” of their movements by using goal setting, instruction, feedback and manual guidance, and having them carry out these tasks in a more functional context (Barnes et al., 2005). According to Richards and Pohl (1999), practice is a cornerstone of interventions designed to train skilled functions. There are many dimensions of practice that must be considered in structuring an effective therapy program for the upper extremity of stroke patients to facilitate motor skill learning. These include: variability (practice should be variable to facilitate long-term retention of skilled action performance); part- versus whole-task practice (depending on whether the action to be learned can be broken into parts); context of practice (practice in performing a skilled action must be in different contexts if that action is to be performed in different contexts); feedback (infrequent and somewhat delayed feedback facilitates learning, particularly if the feedback is not an actual part of the skill); and attentional demands (learners must actively participate in practice and attend to their performance). Bütefisch et al. (1995) stated that “repetition of identical movements forms the physiological basis of motor learning and that it is essential to overcome deficits in force production and movement execution”. However, these repetitive elements do not appear to be emphasized sufficiently in conventional physiotherapeutic approaches.

There is potential for implementing motor learning principles in neurological rehabilitation; however, very few studies using this paradigm have been conducted with patients with neurological deficits (Barnes et al., 2005). New
approaches such as robotics and robot-assisted rehabilitation techniques can incorporate motor learning principles into post-stroke rehabilitation together with the training of functional movements. For example, it has been shown that robot-assisted rehabilitation involving repeated practice against a resistive force improves upper extremity strength and functions of the wrist and hand in chronic stroke patients (Hu et al., 2007).

1.5 Robot-assisted rehabilitation

For the purpose of achieving better functional improvement and enhancing quality of life, new rehabilitative approaches are being adopted and/or previous techniques are being modified. Technological interventions such as virtual reality exercises and robotics are under development and becoming more common. Robot-assisted rehabilitation, in general, can be divided into two categories: rehabilitation of active movement and rehabilitation of passive movement (i.e. to provide continuous passive motion of the hand). There are four different approaches that have been developed for robotic hand rehabilitation (Dovat et al., 2008).

The first rehabilitative approach is to provide continues passive motion (CPM) of the hand. The Amadeo System for Tyromotion is one of the examples for this type of rehabilitation. It is designed to help subjects reduce joint stiffness of the fingers by assisting flexion and extension. It can work in passive or active modes with individual fingers, i.e. it has five degrees of freedom (DOF). The second method for the robotic hand rehabilitation involves training with an additional hand module attached to robotic structures. For example, the Hand
Robot Alpha-Prototype II (Masia et al., 2007) trains hand grasp and release by providing high force to the palm. It works in active and assistive modes in one DOF (i.e. flexion/extension of five fingers together). The third and fourth type of robotic designs are dedicated to training wrist and hand functions, for example, the Hand-Wrist Assisting Robotic Device-HWARD (Takahashi et al., 2005) and finger functions, for example, Rutgers Master II (Bouzit et al., 2002). HWARD is a pneumatically-actuated robotic device designed to train grasping and releasing movements. It has three DOF (flexion/extension of four fingers, thumb or wrist) and functions in active and assistive modes with a large ROM. Rutgers Master II is a haptic glove which samples hand position so as to provide suitable resistive forces to individual fingers (4 DOF, no little finger).

Following stroke, patients are discharged from hospitals as soon as they have mobilization to limit the cost of the treatment because post-stroke rehabilitation programs are time-consuming and labor-intensive (Hu et al., 2007). Upper extremity and hand rehabilitation has a long recovery process. After being discharged from a hospital or a rehabilitation center, patients can perform hand exercises at home with the help of several rehabilitation products (Figure 1) in order to improve the strength and functions of the hand (e.g. rubber ball, grip spring, Thera-Band, power web, Digi-Flex). However, the exercises performed with these products are not force-controlled, measurable and often lack of motivating factors.
It has been shown that passive movement provided by CPM therapy is not beneficial for improvement of motor functions, even though it can temporarily reduce spasticity and maintain joint flexibility and stability in the early post-stroke stage. This is because passive movement therapies do not involve the active movement necessary for motor learning and/or building strength. On the other hand, active and active-assistive robotic therapies have been shown to improve strength and motor function in post-stroke rehabilitation (Hu et al., 2007). Robot-assisted rehabilitation can complement traditional therapy in many ways: Robotic devices can motivate patients to continue their training without clinical observation. This is because these devices can be used for the purpose of treatment, diagnosis and assessment of motor impairments (i.e. spasticity, strength) with great accuracy (Dovat et al., 2008), as they can measure variables, such as position and force. They can automate repetitive movements to enhance motor learning and provide passive\textsuperscript{12} or active\textsuperscript{13} movements as well.

\textsuperscript{12} Passive movement: Movement performed without voluntary contraction by the individual.

\textsuperscript{13} Active movement: Movement performed by the individual.
as motivate patients with virtual reality game-like exercise feedback. The training can be modified based on each subject's needs and degree of recovery. There is evidence showing that active and active-assistive robotic therapies can decrease the amount of co-contraction in the shoulder and elbow, increase the force production from the shoulder and the elbow as well as enhance the functional improvement of the wrist and hand in chronic stroke (Hu et al., 2007).

However, current active robotic devices for hand rehabilitation are often too large to be used at home, have too limited range of force, ROM or limited DOF (i.e. no individual finger movements) and they may not be adaptable for training a variety of tasks and movements (Dovat et al., 2008; Lambercy et al., 2007). Therefore, two novel robotic devices, HandCARE and Haptic Knob, were designed. Both of these interfaces can produce adequate forces within a large workspace and their portability would allow them to be used at home or in rehabilitation centers and clinics. Furthermore, they can be adapted to allow patients to perform different tasks as they need (Dovat et al., 2008; Lambercy et al., 2007). With these systems, subjects can train active, active-assistive and passive movements in order to improve both motor functions and ROM.

We have developed exercises with the HandCARE and the Haptic Knob robotic devices to train specific hand and finger functions by focusing on the difficulties in performing ADL (i.e. manipulating small objects, grasping, key turning and individual finger movements) after stroke. There are three exercises designed with each robotic system with the aim of improving hand and finger

\[13\text{ Active movement: Voluntary movement of a joint.}\]
functions commonly used in ADL. The exercises focus on practicing similar movements to the tasks of daily life (e.g. opening/closing of the hand, using a key to open a door) with the help of virtual reality game-like feedback.

1.5.1 HandCARE: Cable Actuated REhabilitation system

The HandCARE (Dovat et al., 2008) is a cable-driven robot that consists of five loops— one for each finger— moving in linear displacement independently or all together (Figure 2). It is designed to train hand functions such as flexion/extension of fingers, finger independence, coordination and force control of fingers. The interface can provide assistive or resistive forces to the fingers by a clutch system actuated by one motor. There are five clutches, so it can train all fingers at the same time (e.g. grasping) as well as independent movements of each finger (e.g. flexion/extension of index finger). Each clutch, used for one finger, can be operated in three different modes: fixed mode to train isometric force tasks; free mode to move the fingers(s) without restriction along the path defined by the cable; and active mode to train the finger(s) with a resistive force. The position and the velocity of the clutches and the currents of the motor can be read by the system (at a frequency of 100 Hz) and the forces applied by each finger can be measured by five 2 N force sensors. The system can provide visual, tactile and audio feedback to keep the subject informed during the training. The device includes a support system for the forearm and elbow in order to provide comfort and to isolate the hand movements from other body parts while supporting the weight of the forearm and the elbow. Five mechanical stops and one emergency switch are incorporated for safety. The patient holds a
pneumatic switch during the experiment which also can stop the system if squeezed. The complete features of the HandCARE system are listed in Table 2.

Figure 2: Parts of the HandCARE system

Control box includes the motor, the control card, the clutch systems and the sensing system. Five adjustable pulley fixtures allow changing the direction of the hand movement.
Table 2: Characteristics of the HandCARE system

<table>
<thead>
<tr>
<th>Description</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>linear motion of each finger</td>
<td>8.5 cm</td>
</tr>
<tr>
<td>flexion of each finger</td>
<td>0 – 70 deg</td>
</tr>
<tr>
<td>maximal opening (between thumb and opposing fingers)</td>
<td>18.5 cm</td>
</tr>
<tr>
<td>minimal opening (between thumb and opposing fingers)</td>
<td>1.5 cm</td>
</tr>
<tr>
<td>maximal/minimal force generated at the output</td>
<td>±75 N</td>
</tr>
<tr>
<td>force measuring range</td>
<td>±15 N</td>
</tr>
<tr>
<td>force sensitivity</td>
<td>0.2 N</td>
</tr>
<tr>
<td>control frequency</td>
<td>100 Hz</td>
</tr>
<tr>
<td>sensor sampling frequency</td>
<td>1000 Hz</td>
</tr>
<tr>
<td>weight (with motor and control system)</td>
<td>5 kg</td>
</tr>
<tr>
<td>external dimensions</td>
<td>60 × 30 × 30 cm³</td>
</tr>
</tbody>
</table>

Adapted from Dovat et al., 2008

Three exercises have been designed for the HandCARE robotic device, namely the isometric Hangman exercise, the elastic exercise and the somatosensory exercise.

1.5.1.1 Isometric Hangman exercise

The isometric Hangman exercise has been designed by considering the post-stroke impairment of diminished control of individual finger movements. The objective of this exercise is to strengthen muscles of individual fingers in order to improve finger independence, and to improve the ability to control the force
applied by each finger. The exercise consists of isometric forces generated by individual fingers. Each column on the feedback screen represents a finger (Figure 3A). The instruction is to select the target letters to compose a word such as in the game of Hangman, a game in which the player tries to find a word by guessing its letters one-by-one. In the game, every wrong letter chosen is registered as a line in a drawing of a stick man hanging on a gallows. To select a letter, patients have to apply certain amount of isometric force (between 5% to 25% of maximal voluntary contraction (MVC)) with the chosen finger on the screen and hold the force level for several seconds before the system will confirm the selected letter. Any force applied by the finger other than the selected finger has a negative effect on the applied force of the selected finger. Through this application, the patient is encouraged to use the selected finger individually and improve coordination between fingers. Rather than a hanging stick man, an aesthetically appealing picture appears in fragments on the computer screen as letters are correctly selected. The success of the exercise is associated with completion of the picture which depends on the number of successfully selected letters during the set, i.e. the more correct letters that are chosen, the more complete the displayed picture (Figure 3B).

There are number of parameters that can be changed in order to modify the exercise to the patient’s needs and to change the difficulty level of the exercise. One parameter that can be changed is whether to use the negative force effect of unselected fingers or not. By eliminating the negative force effect, the exercise becomes easier for patients to perform because they can practice
the exercise without having to control more than one finger. Another parameter that can be modified is the distribution of the letters in the columns. For example, the letters that are most often used (e.g. vowels) can be placed higher in the columns so that the patient must apply larger forces to reach them. A third parameter is the method of letter confirmation either by switch or timing. Choosing the letters by pneumatic switch gives the patient a chance to practice coordination of the two hands.
Figure 3:  
B: Corresponding reward view of the HandCARE- Isometric Hangman exercise

A: The force applied by each finger is displayed in different columns where the cursors alternate according to the force amplitude. A selection of letters is displayed besides each column. The target letter flashes green in order to help the patient to recognize it. The finger indicator also shows the selected finger to apply force. On the feedback screen, the red cursor represents the force applied by the selected finger while the blue cursor shows the force applied by other fingers. The blue cursor in the column of the selected finger (middle finger) represents the total amount of force applied by other fingers (index and ring fingers). B: At the end of each set, a complete or a fragmented picture is displayed.

1.5.1.2 Elastic exercise

One of the motor disabilities found in post-stroke is impaired coordination between multiple joints. The elastic exercise, therefore, has been designed to train coordination between individual fingers (Figure 4). The exercise involves
active finger movements against an elastic load (between 5% to 25% MVC) generated by the robot. The visual effect for this exercise consists of a picture, fragmented into five parts (Figure 5). Each fragment of the picture represents an individual finger and moves when force is applied by the corresponding finger. Subjects are instructed to apply the same amount of force with each finger while closing the hand against the elastic load in order to keep the picture complete during the movement. The exercise starts when the phase indicator flashes green. Once the hand is completely closed as displayed by the “position indicator” (Figure 5), the patient can extend the fingers (active extension) or the robotic interface can assist the patient for finger extension (active-assistive movement).

During training, several parameters can be modified. The amount of resistive force can be increased in order to strengthen fingers, the range of the opening movement can be adjusted based on the patient’s ROM, the difficulty level of the exercise can be adjusted by changing the sensitivity of the visual feedback. For example, a force target window is set for each patient in order to adjust the sensitivity level of the exercise. If the force applied by any finger moves outside the force target window set for the exercise while closing the hand, the corresponding slice of the picture moves proportional to the amount of force applied by the finger and an uneven picture is displayed on the computer screen (Figure 5). If the forces applied by each finger stay in the force target window during the movement, the picture remains unaltered. Lastly, the level of assistive force can be adjusted in order to help or train finger extension by
changing the elastic constant of the load. The success of the exercise is measured by the amount of fluctuation of the columns as well as the amount of active extension that can be completed. Therefore, patients are informed about their performance in terms of whether they apply too much or too little force with any of the fingers and are encouraged to reach fully open the hand as indicated by the position indicator.

Figure 4: A view from a patient performing the elastic exercise.
1.5.1.3 Somatosensory exercise

The somatosensory exercise has been designed to improve the processing of somatosensory information by applying somatosensory feedback, and to train subjects to regulate force by maintaining the force applied by individual fingers.

During training, no visual feedback is provided for the subject in order to focus on tactile feedback. The tactile feedback is given in the form of vibration generated by a small motor, similar the ones used in cell phones, attached to a finger loop. A feedback screen is available for the instructor so as to follow the amount of force applied by the subject (Figure 6). Each column on the screen represents an individual finger. The red cursors show the amount of force applied by the finger being trained. The “success LED” flashes green when the subject applies an adequate amount of force to reach at the target force level.
Subjects are instructed to reach the target by applying force with the selected finger and maintain the force for a prescribed period of time. The number of fingers involved in the movement can be changed. For example, moving the index and the thumb fingers together can simulate a pinching motion. Once the target force level is reached, the subject feels the vibration feedback on a selected finger (thumb or index). The target force level can be adjusted based on the subject’s ability to exert force. In order to change the difficulty level, the exercise can be modified by changing the force target window. The success of the exercise is defined by the amount of fluctuation around the target force level and the time taken to find the right target level.

Figure 6: Feedback screen of the HandCARE-somatosensory exercise
1.5.2 Haptic Knob

The Haptic Knob (Lambercy et al., 2007) is a robotic interface with two DOF designed to train specific functions: opening/closing of the hand, pronation/supination of the forearm and the force control during grasping based on the functions that patients have most difficulty to perform in a daily life. For example, turning a door knob, using a key and grasping objects of different sizes are some of the functions that stroke survivors have the most desire to recover.

The first DOF of the device is linear opening of the hand, while the second one is rotation of the forearm by the rotation of the robot along the axis of the forearm (figure1). The system is controlled by two motors, one controlling the linear movement while the second motor actuates the rotation of the system. The Haptic Knob can train patients in both assistive and resistive modes by generating forces up to 50 N in both opening and closing directions, and torques up to 1.5 Nm on the rotational DOF. The grasping force and the perpendicular forces (Figure 7A, r and z-directions, respectively) can be measured with four miniature force sensors. The position of the linear movement, i.e. hand opening, and the pronation/supination angle can be read by the motor encoders. The complete features of the Haptic Knob system are listed in Table 3.
Table 3: Characteristics of the Haptic Knob system

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>maximum opening of the interface</td>
<td>150 mm</td>
</tr>
<tr>
<td>minimal opening of the interface</td>
<td>30 mm</td>
</tr>
<tr>
<td>maximum rotation of the interface</td>
<td>±180 deg</td>
</tr>
<tr>
<td>maximum rotation of the moving parallelogram</td>
<td>±45 deg</td>
</tr>
<tr>
<td>maximum generated opening/closing force</td>
<td>50 N</td>
</tr>
<tr>
<td>maximum generated torque</td>
<td>1.5 Nm</td>
</tr>
<tr>
<td>friction force for the linear DOF</td>
<td>9 N</td>
</tr>
<tr>
<td>friction torque for the rotation DOF</td>
<td>0.02 Nm</td>
</tr>
<tr>
<td>inertia (closed position)</td>
<td>4.83.10^{-4} Kgm²</td>
</tr>
<tr>
<td>inertia (open position)</td>
<td>19.3.10^{-4} Kgm²</td>
</tr>
<tr>
<td>workspace of one finger (MCP flex./ext)</td>
<td>60 deg</td>
</tr>
</tbody>
</table>

Adapted from Lambercy et al., 2007.

Fixtures of different sizes and shapes can be attached to the Haptic Knob in order to train different functional tasks (Figure 7B). Furthermore, visual, tactile and audio feedback have been implemented with the Haptic Knob in order to motivate and inform patients about their performances. The device incorporates an adjustable support system for the forearm and the elbow in order to provide comfort and prevents patients from rotating their body while performing the exercises. Both software and hardware emergency systems have been implemented to prevent any harm to the subjects. There is a safety switch for the instructor and a pneumatic switch for the patient to turn off the system. Three
exercises have been designed for this robotic device, which focus on improving ROM, movement coordination, precision and fine force tuning.

Figure 7:  
A: Parts of the Haptic Knob system  
B: Fixtures of the Haptic Knob

A: The Haptic Knob consists of two moving parallelograms which enable the device to move in linear direction. B: Fixtures of different sizes and shapes can be attached to the Haptic Knob.
1.5.2.1 Opening/closing exercise

Flexor spasticity in the upper extremity is a common result of stroke accompanied by the weakness in the extensors. Because of the spasticity in wrist and finger flexor muscles and weakness in extensor muscles patients have a very limited ROM of finger extensors.

The opening/closing exercise, therefore, has been designed to decrease flexor spasticity, increase the ROM of finger extensors, and to increase control and muscle strength in finger flexors. The exercise is performed using the linear opening DOF of the Haptic Knob, while the rotational DOF is held in static position. The patient is instructed to place the thumb and fingers on two separate parts of the knob (Figure 8A). The exercise starts with passive extension of the fingers to an initially selected position. The displayed picture disappears progressively while the robot is opening the hand (Figure 8B). At this time, the subject is instructed to make the picture completely disappear by relaxing the hand. Passive extension is followed by active flexion of the hand against a resistive force generated by the robot. The picture progressively appears as a function of the closing movement. The resistive force can be a constant, elastic or velocity dependent load. The amount of resistive force can be changed in order to adjust the difficulty and thus the intensity of the exercise. A damping effect, which might make the movement smoother, can be added. The opening range of the knob is modified for each patient based on their maximum ROM.
Figure 8:  
A: A view from a patient training with the Haptic Knob, opening/closing exercise  
B: Feedback view of the Haptic Knob-opening/closing exercise

A: Patient's hand is being opened by the robot to a position set for the patient. B: The displayed picture disappears as a function of the opening movement while the device is opening the hand passively and appears as a function of the closing movement.

Performance is measured by the velocity at the end of the closing movement and the amount of force applied during the opening movement. Therefore, subjects are instructed to try to keep a constant speed while closing the hand, so as to
avoid closing too rapidly, and to relax the hand while opening to have the robot open the hand completely.

1.5.2.2 Pronation/supination exercise

This exercise trains pronation and supination of the forearm, as well as fine control of the movement, while generating and maintaining grasping force. This is because stroke survivors have great difficulty in performing certain activities (e.g. using a key, rotating a door knob) which involve coordination between forearm and hand movements. Patients often report a difficulty in maintaining the grasping force applied by the hand while rotating the forearm, which also requires torque production. In this exercise, the patient is asked to grasp the two sections of the knob and rotate it to match the picture with the frame shown on the computer screen by rotating the forearm (Figure 9 A and B). A scoring system has been developed to encourage the subject to precisely control the end of the movement while trying to match the picture with the frame. The score is calculated based on the time spent outside the target window to adjust the position. A higher number of oscillations around the target results in a lower score.

The size of the target window can be adjusted in order to set the accuracy level of the exercise. The difficulty level of the exercise can be modified by adding a force field, changing the degree of rotation or the accuracy level of the exercise.
1.5.2.3 Proprioception exercise

Following stroke, patients often report difficulty in carrying objects without dropping them and/or in gently grasping objects. This problem emanates from the lack of ability to regulate force while using the hand in functional tasks. The proprioception exercise, therefore, has been developed to train subjects to
regulate force by giving proprioceptive feedback. In this exercise, the patient is asked to maintain a target force by grasping the two sections of the knob. During the exercise, the patient has two seconds to reach the target force with the help of visual, audio or audio-visual feedback (Figure 10) in order to learn the amount of force that needs to be applied. As soon as the patient maintains the target force, the visual feedback mechanism is eliminated and the exercise begins. The Haptic Knob is programmed to remain stationary if the patient maintains the correct amount of force, and to rotate in one direction if the grip force is greater than the target force or in the other direction if the grip force is smaller than the target force. A score is calculated based on the force applied to both sections of the knob. The score drops with a variation of the force in either section of the knob.

Figure 10: Feedback screen of the Haptic Knob- proprioception exercise

The patient places his hand on the knob so that fingers are in contact with the section of the knob in the upper display, while the thumb is in contact with the section on the lower display. The target force is displayed beside the force indicator. The yellow phase indicator shows that the applied force is not in the target window.
Several parameters can be adapted to the needs of the patient such as the target force level, the opening range of the knob, the accuracy level of the exercise, i.e. the size of the force target window, and the duration for which the subject must maintain the grasping force.

A pilot study was conducted in order to test the usability and the effectiveness of the two robotic devices for post-stroke hand rehabilitation. The results of this pilot study can be found in Dovat (2009), Lambercy (2008), Lambercy et al. (2007), Dovat et al. (2007), Lambercy et al. (2008), Dovat et al. (2008).

1.6 Objective

After stroke, the number of possible muscle synergies that the CNS is able to produce decreases. This might be due to a decrease in the variety of muscle activation patterns. Fewer muscle synergies and impairment in the ability to control available muscle activation patterns could be the reason for the abnormal co-activation patterns observed post-stroke.

The objective of the current study is to quantify the changes in muscle activity of stroke survivors before and after robot-assisted hand rehabilitation with the HandCARE and Haptic Knob robotic devices, and specifically, to compare the patterns of muscle activity with healthy age-matched subjects.

The results should advance our understanding of the muscle activation patterns occurring post-stroke and the abnormalities in these patterns which would consequently help clinicians to design the most effective therapies and to
choose the most suitable hand therapy techniques for their patients. It would also give valuable information to follow up patients' improvement by the changes in these patterns.

Three hypotheses have been formulated to achieve the objective of the study:

1. Post-stroke subjects co-contract antagonistic muscles more than healthy age-matched subjects performing the same exercises

2. Post-stroke subjects have less variety in their activation patterns than healthy age-matched subjects performing the same exercises

3. The muscle activation patterns of post-stroke subjects will show (1) less co-contraction and (2) more variety after robot-assisted hand rehabilitation

The methods and the protocol used in this study will be explained in Chapters 3.
CHAPTER 2: ACTIVITY PATTERNS OF HAND MUSCLES

2.1 Introduction to Hand Anatomy

The hand is one of the most complex structures of the human anatomy with its 19 bones, 34 muscles in finger, hand and forearm, 14 joints and 25 degrees of freedom.

The hand skeleton is composed of five metacarpals and fourteen phalanges (Figure 11). The metacarpals connect each individual finger to the wrist and the phalanges form the individual fingers. Each finger has three phalanges (proximal, middle and distal) with the exception of the thumb which has only two phalanges. The separated position of the thumb from other digits enables it to have more DOF. There are eight bones at the wrist called “carpals” connecting metacarpals to the radius and ulna by carpometacarpal (CMC) joints. The joints connecting the phalanges to the metacarpal bones are called metacarpophalangeal (MCP) joints. There are two interphalangeal joints (IP, between phalanges) located in each of the four fingers and only one in the thumb. The ones located proximally are called proximal interphalangeal (PIP) joints, and the distal ones are called distal interphalangeal (DIP) joints in the four fingers.
There are two muscle groups producing finger, hand and forearm movements: intrinsic muscles, located within the hand itself, and extrinsic muscles, located proximally in the forearm which insert on the hand skeleton by long tendons. The intrinsic muscles are composed of interosseus muscles (4 dorsal and 3 palmar), thenar muscles (located on the thumb side of the hand), hypothenar muscles (located on the little finger side of the hand), and lumbricals (4 muscles). The intrinsic muscles can also be divided as dorsal and palmar intrinsic muscles based their location (Table 4).
Table 4: Intrinsic muscles of the hand

<table>
<thead>
<tr>
<th>Dorsal Intrinsic Muscles</th>
<th>Palmar (Volar) Intrinsic Muscles</th>
</tr>
</thead>
<tbody>
<tr>
<td>(DI) Dorsal Interossei</td>
<td>Superficial</td>
</tr>
<tr>
<td>Abductor digit minimi (ADM)</td>
<td>Profundus</td>
</tr>
<tr>
<td>Lumbricals</td>
<td>Palmar interossei</td>
</tr>
<tr>
<td>Flexor digit minimi</td>
<td></td>
</tr>
<tr>
<td>Abductor pollicis brevis (APB)</td>
<td>Thenar muscles</td>
</tr>
<tr>
<td>Opponens pollicis</td>
<td>Opponens digit minimi</td>
</tr>
<tr>
<td>Adductor pollicis</td>
<td>Hypothenar muscles</td>
</tr>
<tr>
<td>Flexor pollicis brevis</td>
<td></td>
</tr>
</tbody>
</table>

Dorsal and palmar distribution of intrinsic muscles. Palmar muscles can also be divided into superficial (located on the surface) and profound (located deep) muscles. Abductor digit minimi and flexor digit minimi are also hypothenar muscles.

The extrinsic muscle groups are composed of long flexors and extensors (Table 5). The lumbricals and all of the interosseus, except the first dorsal interosseus, are also involved in interphalangeal joint extension through their insertion onto to extensor mechanism. The extrinsic extensors consist of three wrist extensors and a larger group of thumb and digit extensors.
### Table 5: Extrinsic muscles of the hand

<table>
<thead>
<tr>
<th>Extensors (Ext.)</th>
<th>Flexors (Flex.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ext. carpi radialis longus (ECR)</td>
<td>Flex. carpi radialis</td>
</tr>
<tr>
<td>Ext. carpi radialis brevis</td>
<td>Flex. carpi ulnaris (FCU)</td>
</tr>
<tr>
<td>Ext. carpi ulnaris</td>
<td>Palmaris longus</td>
</tr>
<tr>
<td>Abductor pollicis longus</td>
<td></td>
</tr>
<tr>
<td>Ext. pollicis brevis</td>
<td>Flex. digitorum superficialis (FDS)</td>
</tr>
<tr>
<td>Ext. pollicis longus</td>
<td>Intermediate muscles</td>
</tr>
<tr>
<td>Ext. indicis</td>
<td>Flex. digitorum profundus</td>
</tr>
<tr>
<td>Ext. digitii minimi</td>
<td>Flex. pollicis longus</td>
</tr>
<tr>
<td>Ext. digitorum communis (EDC)</td>
<td>Primary activator for digits</td>
</tr>
</tbody>
</table>

The extrinsic muscles can be separated based on their functions (i.e. extensors or flexors of wrist or fingers) or their location (i.e. superficialis or profundus muscles).

Given the number of muscles and degrees of freedom, the hand is capable of performing a considerable number of combinations of movement. Nine primary movements are shown in Figure 12.
2.2 Literature Review of Hand Muscle Activation Patterns

Early studies investigated the muscle activations in anatomically defined muscle groups such as palmar and dorsal interossei (Backhouse et al., 1968), thenar and hypothenar participation (Forrest and Basmajian, 1965) or intrinsic and extrinsic cooperation (Close and Kidd, 1969). Several experiments have been performed in order to study the functions of muscles either in power grip, performing ADL or during pinch.

However, the study of Long and Conrad (1970) was the first one to determine the functions of intrinsic and extrinsic hand muscles in well defined motions such as power grip, i.e. squeeze, disc, hook and spherical grips, precision handling, i.e. rotation and translation of the object held by thumb and first one or two fingers, and pinch, i.e. between thumb and index or thumb, index
and middle fingers. Their results demonstrated that in power grip and pinch, intrinsic and extrinsic muscles work together, with the extrinsics providing the major gripping force which increases in proportion to the applied force. More recently, Johansson and Westling (1988) showed evidence of co-contraction patterns in which the agonist and antagonist\textsuperscript{14} muscles of the hand and arm were active at the same time during the lift and hold phase of a lifting task and during load perturbations. However, during the loading as well as unloading phases, the activity changes in reciprocal manner for both extrinsic and intrinsic hand muscles. Co-contraction is necessary during the hold phase to provide postural stabilization of the multiarticulate hand and arm. Adequate stiffness during the loading and unloading phases is provided by activity in one muscle group while relaxing the antagonists.

During a precision grip task in which a person applies fine-graded isometric forces rather than a dynamic task (e.g. grip-and-lift), co-activation patterns can be seen in all extrinsic and intrinsic hand muscles and the activity of muscles increases in parallel with force (Maier and Hepp-Reymond, 1995a). Moreover, it has been demonstrated that the intrinsic muscles are as important as, or possibly more so than the extrinsic muscles in the generation and regulation of low forces in precision grip in contrast to the findings of Long et al. (1970) who applied considerable loads during pinch. Extrinsic hand muscles

\textsuperscript{14} An antagonistic muscle acts in opposition to the specific movement generated by an agonist muscle. Most muscles of the musculoskeletal system have antagonistic muscles. Antagonistic pairs consisting of flexor and extensor muscles work together during a movement such that the extensor muscle stretches while the flexor muscle contracts. Antagonist muscles are responsible for returning a limb to its initial position as well as counteracting acceleration of the limb produced by the agonist muscle.
were found to be strongly active during reaching, while the intrinsic hand muscles were mainly active when shaping the hand around the object and at the onset of manipulation (Lemon et al., 1995). FDS and EDC, which are involved both in the orientation of the palm and positioning of the finger tips, showed high EMG activity during reaching, whereas the largest relative responses in 1DI and APB were found after the subject first touched the object (Lemon et al., 1995) with APB showing high activity prior to contact because of the final adjustment of the grip aperture. In accordance with the previous results, Milner and Dhaliwal (2002) showed that a primary role of intrinsic muscles (1DI and LUM) was to control the direction of fingertip force, while extrinsic muscles (FDS and EDC) produced most of the force in an isometric force control task. Co-contraction of extrinsic extensor muscles was necessary to stabilize the joints while applying large forces at the fingertip.

The majority of the previously described studies demonstrated agonist/antagonist co-activation patterns of finger muscles in variety of functional tasks involving several fingers. For example, during grasping, all four fingers move together or in a pinching task, the first two or three fingers move together. However, fingers can also move independently of one another. Independency of finger movements can be demonstrated in two ways: individuation of finger movements, in which a finger would move without accompanying motion of other fingers, and stationarity of a finger, in which a finger would stay stationary during the movement of other fingers. Indeed, there are tasks which require multiple fingers to move independently and synchronously within the same performance.
For example, playing a piano would involve both characteristics, such as the fingers of the left hand moving synchronously for playing chords, while the fingers of the right hand play a melody which requires mostly individual finger movements. Schieber (1995) studied the mechanism underlying individuated finger movements by recording EMG activity of forearm muscles in trained rhesus monkeys which have multi-tendoned extrinsic muscles that flex and extend the fingers similar to human muscles. The study revealed that the individuated movements of an instructed or a noninstructed digit, i.e. individual finger movements, are produced by the “net effect of several muscles including agonists, antagonists and stabilizers of a digit acting simultaneously on many or all digits”. Movements are produced by complex combinations of various degrees of activity in participating muscles, i.e. coordination and in this way “more distinct movements can be produced than there are muscles to produce them” (Schieber, 1995; Valero-Cuevas, 2000). This might be the solution of Bernstein’s degrees-of-freedom problem where the available number of muscles is higher than necessary to control the mechanical DOF (Latash, 2001), or it may be related to mechanical constraints which may limit individual finger movements, i.e. the 3rd and 4th digits are controlled by extrinsic muscles that attach to more than one finger by means of multiple tendons (Schieber, 1995). The CNS overcomes this potential problem by co-activating groups of “muscle synergies” in each movement to reduce the number of individual muscles that need to be controlled, and achieves it by selecting a suitable combination of joints, muscles and motor command to use for a given task.
Other evidence for the synergistic muscle activation comes from Maier and Hepp-Reymond (1995b) who stated that muscles are activated in synergistic patterns i.e. components of muscle activation patterns more frequently than independently during a precision grip, and among the synergistic muscles combinations, co-activation is the most commonly observed pattern. The synergies are not rigid, instead they are task-dependent such that they are dependent on joint angle, direction, and finally, magnitude of the forces applied (Maier and Hepp-Reymond, 1995b; Valero-Cuevas, 2000; Milner and Dhaliwal, 2002). The CNS uses a simplifying strategy to control the redundant musculature of the digits rather than generating new muscle coordination patterns for different force levels (Valero-Cuevas, 2000). According to this strategy, muscle coordination patterns to produce fingertip forces of lower magnitudes are effortlessly selected by scaling down the pattern used for producing the largest expected fingertip forces.

There is evidence that many behaviors can be described by combinations of small number of muscle synergies, i.e. components of muscle activation patterns (Tresch et al., 2006; d’Avella et al., 2003; Weiss and Flanders, 2004). Principal components analysis (PCA) is a factorization algorithm which can be beneficially used to examine these muscle synergies underlying behavior (Tresch et al., 2006). In this study, we would like to investigate the components of muscle activation patterns in chronic stroke survivors’ hand and finger muscles and PCA is a good method for this purpose.
CHAPTER 3: METHODS

3.1 Subjects

Four right-handed chronic stroke subjects (S; 1 female, 3 male; age between 54-83 y) with right hemiplegia and 8 healthy right-handed subjects (H; 4 female, 4 male; age between 50-80 y; one ambidextrous) participated in this study. The healthy subject group was composed of one female and one male subject from each 10-year-age-group from 50 to 80 years old (Table 6). They are referred to as healthy age-matched subjects.

<table>
<thead>
<tr>
<th>Subject Group #</th>
<th>Age Group</th>
<th>Gender</th>
<th>Healthy subjects</th>
<th>Post-stroke subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50-60</td>
<td>M F</td>
<td>H1 H2</td>
<td>S1</td>
</tr>
<tr>
<td>2</td>
<td>60-70</td>
<td>M F</td>
<td>H3 H4</td>
<td>S2</td>
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<tr>
<td>3</td>
<td>80-up</td>
<td>M F</td>
<td>H5 H6</td>
<td>S3</td>
</tr>
<tr>
<td>4</td>
<td>70-80</td>
<td>M F</td>
<td>H7 H8</td>
<td>S4</td>
</tr>
</tbody>
</table>

All stroke subjects were recruited through advertisements in newspapers and during the visits to the community centres and stroke clubs in the Greater
Mainland area in Vancouver, BC. A first interview was done by telephone and/or face-to-face meeting in order to ensure that the stroke survivors met the inclusion criteria of the study. Only right hemiplegic patients were included in this study. Stroke subjects with severe hand impairments, which prevented them from positioning the hand on the robotic devices, i.e. no functional use of the right hand in any ADL due to severe spasticity, as well as subjects with cognitive disabilities, e.g. agnosia, alexia, neurological diseases, e.g. Parkinson, MS, medical problems which can affect the performance of the patient, e.g. chronic heart disease, or patients with visual or perceptual deficits, e.g. homonimus hemianopsia, and neurological disorders, e.g. apraxia, receptive aphasia, were excluded from this study. Healthy subjects were recruited through community centres located in the Greater Mainland area in Vancouver, BC. Inclusion criteria for the healthy subjects involved good general health with no severe medical problems and no previous hand injuries, as well as age-matching in 10-year brackets between 50 and 90 years old.

The stroke subjects who participated in this study had only one ischemic stroke more than two years prior to the study (S1: two-year post-stroke, S2: four-year post-stroke, S3: six-year post-stroke, S4: eighteen-year post-stroke) with the exception of one subject (S2) who had two consecutive strokes within the same year. Two of the subjects had the ischemic stroke in the internal capsule (S1: posterior limb of the internal capsule, S2: anterior limb of the internal capsule and caudate nucleus). One of the subjects did not provide a medical report (S3), and the other subject (S4) had the stroke in the white matter along the distribution of
the left middle cerebral artery. The initial impairment levels of individual subjects were second and third stages for two subjects (S1 and S3, respectively) and fifth stage for the other subjects (S2 and S4) based on Chedoke McMaster Impairment Inventory/ Stages of Hand (Chedoke Assessment).

All subjects provided informed written consent (Appendix A and B), and the experimental protocol was approved by Simon Fraser University, Office of Research Ethics (Appendix C).

3.1.1 Experimental Protocol

Subjects underwent robot-assisted rehabilitation for 8 weeks, twice a week in a rehabilitation program based on each subject’s level of functional impairment and types of functions which the subject desired to recover. The rehabilitation program involved two to three exercises on Haptic Knob and HandCARE robotic systems during a one-hour training session. Each exercise was performed for at least two sets of ten repetitions, depending on the performance and the physical condition of the subject. Figure 13 shows the protocol for the experiment. A preliminary session was conducted prior to the experiment to determine whether the subjects were capable of using the robotic systems and which exercises were most appropriate.

During the 8-week training sessions, either the difficulty level of the exercise or the type of exercise was changed once the subject’s performance reached a plateau. The healthy age-matched subjects had only one training session in which they performed the same exercise program as their age-
matched stroke subject performed in the first training session. Table 7 summarizes the exercises that each stroke subject performed with the number of sessions.

Figure 13: Flow chart of the robot-assisted hand rehabilitation protocol
Table 7: Number of performed training sessions for each exercise.

<table>
<thead>
<tr>
<th>Post-stroke subject</th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
<th>S4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of sessions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**HandCARE exercises**

<table>
<thead>
<tr>
<th>Exercise</th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
<th>S4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isometric Hangman</td>
<td>0</td>
<td>0</td>
<td>16</td>
<td>0</td>
</tr>
<tr>
<td>Elastic</td>
<td>16</td>
<td>4</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Sensorymotor</td>
<td>0</td>
<td>12</td>
<td>16</td>
<td>11</td>
</tr>
</tbody>
</table>

**Haptic Knob exercises**

<table>
<thead>
<tr>
<th>Exercise</th>
<th>S1</th>
<th>S2</th>
<th>S3</th>
<th>S4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening/closing</td>
<td>16</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Pronation/supination</td>
<td>16</td>
<td>7</td>
<td>16</td>
<td>4</td>
</tr>
<tr>
<td>Proprioception</td>
<td>0</td>
<td>9</td>
<td>0</td>
<td>12</td>
</tr>
</tbody>
</table>

I assessed each stroke subject prior to the start of the rehabilitation program in what is referred to as a pre-rehab assessment session. This assessment session involved the application of five assessments related to the upper extremity functions. Following the pre-rehab assessment session, I designed an exercise plan for each subject in the rehabilitation program based on the subject’s performance and assessment outcome. The assessments used in this study were:

1. Modified\(^{15}\) Hand Function Interview: a self-reported interview to record the types of activities that the patient has the most difficulty in performing and the desire to recover (modified from Petersen, 2004) (Appendix D).

\(^{15}\) The original Hand Function interview was modified by adding questions for stroke subjects to describe changes in their hand functions after rehabilitation. The number of questions was reduced by removing questions to rank difficulty of activities that are not related to the current rehabilitation program.
2. The Action Research Arm Test: tests arm and hand motor function on 20 tasks. Patients are asked to grasp, grip, and pinch several objects and perform gross movements (Figure 14) (Lyle, 1981).

3. Chedoke McMaster Impairment inventory/ Stages of Hand: evaluates the wrist and finger functions by asking patients to perform motor tasks related to seven different stages of recovery. Each stage involves three tasks (Chedoke Assessment).

4. Jebsen Test of Hand Function: measures the time spent in performing seven upper extremity tasks related to ADL (i.e. writing) (Jebsen et al., 1969).

5. Purdue Pegboard Test: tests the dexterity of the hand(s) in placing pins on a pegboard (Lafayette Instrument Company).

If the patient had fatigue or lapse in concentration, the assessment sessions were divided into two days.
A stroke survivor is performing the Action Research Arm test. Severe flexor spasticity together with weakness in extensors makes the grasping difficult.

In the same assessment session, I measured parameters related to the subject’s hand function such as muscle strength measured by Manual muscle testing (MMT)\textsuperscript{16}, level of spasticity measured by the Modified Ashworth Scale (Blackbum et al., 2002), ROM measured by the passive movement of the extremity and maximal grasping force measured using a Jamar grip dynamometer (Jamar\textsuperscript{®}). These measures were used in setting the level of difficulty of each exercise. Even though this was not the main focus of the experiment, I applied the same assessments and measures of hand parameters at the end (post-rehab assessment session) and one month after completion of

\textsuperscript{16} MMT is a subjective method used to define and measure muscle strength. It is used to determine the ability to contract a muscle or muscle group voluntarily. The grading system for muscle strength is based on specific factors such as 1) the amount of manual resistance used to oppose the contracting muscle or muscle group, 2) the ability of a muscle or muscle group to move a body segment through its complete ROM, 3) evidence of the presence or absence of muscle contraction, and 4) ability to counteract gravity or manual resistance, i.e. light segments with short lever arms (finger and toes) are not significantly influenced by gravity; thus their grades are determined using manual resistance only (Palmer 1998).
the rehabilitation program (re-call assessment session) for the purpose of looking at the changes in the outcomes after the training.

### 3.2 Data Recording

The surface electromyogram (EMG) comprises the sum of the electrical contributions made by the active motor units as detected by electrodes placed on the skin overlying the muscles (Farina et al. 2004). I recorded the EMG activity of nine hand and forearm muscles of stroke subjects on the first and last day of the robot-assisted rehabilitation, which are called “recording sessions”, and of healthy age-matched subjects while they performed the same exercises with the robots. EMG of the following muscles was recorded: extensor carpi radialis brevis (ECR), extensor digitorum communis (EDC), flexor carpi ulnaris (FCU), flexor digitorum superficialis (FDS), pronator teres (PT), biceps brachii (BI), first dorsal interosseous (1DI), abductor pollicis brevis (APB), abductor digiti minimi (ADM).

I chose to record from these particular muscles because they are superficial muscles of hand and forearm (except for pronator teres), they have functions related to the exercises and they are sufficiently separated anatomically to avoid cross-talk between EMG channels. The relevant functions of the selected muscles for the exercises performed by the subjects are listed below.

- ECR produces extension and radial deviation of wrist. EDC extends the four fingers at the MCP, PIP and DIP joints. FCU produces flexion and ulnar deviation of the wrist. FDS flexes the four digits at the MCP and PIP joints. PT
pronates the forearm (wrist). BI supinates the forearm (wrist). 1DI flexes and abducts the index finger at the MCP joint., APB produces abduction and opposition of the thumb at the CMC joint and flexion at the MCP joint. ADM produces opposition of the little finger at the CMC joint and abduction and extension at the MCP joint of the little finger.

I used custom built active bipolar electrodes with a 20 Hz and 500 Hz bandwidth and variable gain. The EMG was sampled at 2 kHz and stored on disk for subsequent analysis. During the positioning of the electrodes, I asked subjects to contract various muscles with the intention of activating only one muscle at a time. Before the start of recording, we monitored the real time EMG responses while subjects performed various voluntary contractions to evaluate the functional selectivity of the muscles.

For some stroke subjects it can be very challenging to activate a selected muscle due to high spasticity, weakness or both. Both limitations often made the process of electrode placement difficult. Therefore, I used an “electrode placement protocol” based on anatomical landmarks (Perotto, 2005; Arinci, 1994) (Appendix E). Prior to the electrode placement, the surface of the skin was cleaned with alcohol and abrasive gel to increase the conductivity.

Prior to the recording session, we measured the EMG during maximal voluntary contraction (MVC) in sixteen different functional movements and recorded the force using a 6-axis force/torque transducer with a sensitivity of 0.1 N (100Hz sampling rate). The selected functional movements and the placement of the transducer are explained in detail in Appendix F. We provided subjects
with real-time force feedback on a computer screen to motivate them to reach their maximum and to allow the instructor to observe the force applied by the subject. Subjects were allowed to rest between contractions.

3.3 Data Analysis

3.3.1 Introduction to Principal Component Analysis

In this study, EMG activity was recorded from nine muscles of the arm and hand. However, the activity of only eight of the muscles was analyzed because of the poor quality of the recorded signals from one muscle. Thus, the recorded muscle activity is made of eight dimensions. PCA of the muscle activity can be used to determine whether the eight dimensional space representing arm and hand muscle activity can effectively be represented by a smaller lower dimensional space. The principal components (PCs) obtained by this analysis represent predominant combinations of muscle activity that are common across the data set. Because the PCs represent a set of basis vectors that span the space of the data set a linear combination of PCs can be used to reconstruct the elements of the data set. In this case, the data set consists of repetitions of an exercise. The coefficient of each PC in the linear combination is known as the loading score (LS). Although the muscle activity for a given repetition of the exercise could be exactly reproduced from eight PCs and the corresponding set of eight loading scores, i.e. a specific linear combination of the eight PCs, it may be possible to represent the predominant features of the muscle activity with fewer than eight PCs. This can be determined from the variance accounted for across the data set by each PC. For example, if one predominant pattern of
muscle activity is employed in all repetitions of the exercise, i.e. the variability is small, it may turn out that most of the variance is accounted for by a single PC.

### 3.3.2 Calculation of the amount of variation in muscle activation

Root mean square (RMS) values of the EMG signals recorded during MVC measurements were calculated, and the maximum RMS-EMG values for each of the nine muscles were found and saved as “normalization values”. Next, the RMS-EMG for each muscle in each repetition an exercise was calculated and normalized by the subject’s corresponding normalization value. The EMG activity recorded from the APB muscle was excluded from the analysis because the signal was found to have an excessive amount of noise. To determine the amount of variation in the pattern of muscle activation in stroke subjects compared to age-matched healthy subjects, sixteen Principal Components Analyses were performed using normalized RMS-EMG data set recorded from stroke subjects ($2 \times 4 \times 2$, robot $\times$ patient $\times$ pre-and post-rehab) and from healthy subjects ($2 \times 8$, robot $\times$ subject) including all the performed exercises. Wolfram Mathematica Version 7.0 was used for the analysis.

Most of the exercises had two distinct phases in each repetition. These phases were: opening and closing the hand for the Elastic Exercise performed with the HandCARE, “setting” and “tuning” phases for the Proprioception exercise performed with the Haptic Knob, opening and closing the hand for the Open/Close exercise performed with the Haptic Knob and pronation and supination movements for the Pronation/Supination exercise performed with the
Haptic Knob. The RMS-EMG was calculated for each phase and entered as a separate element in the PCA.

Prior to the PCA, normalized RMS-EMG values were organized as p X N matrix A, where p=8 muscles and N=total number of repetitions x number of phases for the exercises performed with each robot. A custom written program was used to perform PCA. The covariance matrix B was calculated from the data A. The principal component weightings for each muscle were determined from the eigenvectors $\alpha$ of covariance matrix B. The PCs were ordered (from 1 to 8) according to the absolute eigenvalues corresponding to each eigenvector, i.e. the greatest eigenvalue corresponded to PC1 and the smallest eigenvalue corresponded to PC8. The relative proportion of the EMG patterns explained by each PC was given by $\alpha'B\alpha$ and the loading scores for each PC for the N repetitions were given by $\alpha'A$. The pattern of activity for each repetition can be reconstructed from the vector product of the PC weightings and the PC loading scores (Wakeling and Horn, 2009). It should be noted that PCA studies commonly subtract the mean EMG before the PCs are calculated and this results in the PCs describe the variance from the original data. In this study, the mean is not subtracted and so the PC defines the components of the entire signal. In order to be consistent with other studies the $\alpha'B\alpha$ is termed the variance.

PCA calculates 8 principal components (PCs) with weighting coefficients for each muscle; loading scores (LS) for each repetition (and phase) and the percentage of the variance across the data set accounted for by each PC. The results for the stroke subjects and their age-matched healthy subjects were
compared in four subject groups. A subject group is defined as one stroke and two age-matched healthy subjects who practiced the same rehabilitation program. The exercises performed by the subject groups are listed in Table A for each robot. The numbers of repetitions analyzed for each exercise were kept constant for each subject in a subject group.

The PCs are generally ranked in terms of the amount of the total variance for which each accounts, i.e. PC1 explains the greatest part of the variance since it would be comparable to the mean muscle activity vector. The amount of variance explained is progressively less in going down the rankings.

If different patterns of muscle activation are expected to predominate for different exercises then one would expect that PCA conducted on a data set that includes different exercises would show that several PCs would be needed to account for most of the variance. However, if individuals are impaired in their ability to generate different patterns of muscle activation, as might be expected after stroke, then most of the variance might be accounted for by one or two PCs. Thus, it is hypothesized, in this study, that fewer PCs will be needed to account for 95% of the muscle activity in stroke patients than healthy subjects, and that the number of PCs needed to account for 95% of the variance in the muscle activity of stroke patients will increase after rehabilitation.
Table 8: Subject groups and the name of the exercises performed by each subject group on each robot.

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>Performed Exercise</th>
<th>Robot</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Elastic</td>
<td>HandCARE</td>
</tr>
<tr>
<td></td>
<td>Open/close</td>
<td>Haptic Knob</td>
</tr>
<tr>
<td></td>
<td>Pronation/supination</td>
<td>Haptic Knob</td>
</tr>
<tr>
<td>2</td>
<td>Somatosensory</td>
<td>HandCARE</td>
</tr>
<tr>
<td></td>
<td>Proprioception</td>
<td>Haptic Knob</td>
</tr>
<tr>
<td>3</td>
<td>Hangman</td>
<td>HandCARE</td>
</tr>
<tr>
<td></td>
<td>Somatosensory</td>
<td>HandCARE</td>
</tr>
<tr>
<td></td>
<td>Pronation/supination</td>
<td>Haptic Knob</td>
</tr>
<tr>
<td>4</td>
<td>Somatosensory</td>
<td>HandCARE</td>
</tr>
<tr>
<td></td>
<td>Proprioception</td>
<td>Haptic Knob</td>
</tr>
</tbody>
</table>

To test the hypothesis that the pattern of stroke subjects’ muscle activity is less variable than healthy subjects, the number of PCs required to account for 95% of the variance was determined and compared between stroke subjects (pre-rehab) and healthy subjects. To test the hypothesis that the variability in the pattern of muscle activation increases with training; the number of PCs needed to account for 95% of the variance was compared for stroke subjects before and after rehab.

3.3.3 Co-contraction measurement

Four muscle pairs were created to test the co-activation levels of antagonistic hand and forearm muscles. The selected muscle pairs are; EDC (extension at MCP, PIP and DIP joints) vs. FDS (flexion at MCP and PIP joints), ECR (extension with radial deviation at wrist) vs. FCU (flexion with ulnar
deviation at wrist), PT (pronation at elbow joint) vs. BI (flexion and assistance to supination at elbow joint); and, 1DI (flexion and abduction of the index finger at MCP joint) vs. EDC (extension of the index finger at MCP joint).

Normalized mean RMS-EMG values were used to create a co-contraction index for each muscle pair for the different exercises. The co-contraction index (CI) was defined as the lower of the mean RMS-EMG values of each muscle pair, i.e. amount of activity that could be considered as common to the two antagonistic muscles. Co-contraction indices for each exercise were found for each subject. The co-contraction indices of stroke subjects in the pre-rehab condition were compared to those of healthy subjects for each muscle pair. Different factors included in comparison of CI between subjects, such as muscle pair and exercise, are referred as a case. The total number of cases in which the CI of stroke subjects in the pre-rehab condition was higher than the CI of healthy subjects was determined and tested for 50% chance occurrence based on a binomial distribution. Chance occurrence was rejected if its probability was less than 5%, i.e. 95% confidence level.

To test the hypothesis that amount of co-contraction decreased after rehabilitation, the co-contraction indices were compared for stroke subjects between pre-and post-rehab conditions and the number of cases where it decreased was tested for chance occurrence in the same way.
CHAPTER 4: RESULTS

4.1.1 Comparison of muscle activation patterns

Prior to Principal Component Analysis (PCA), RMS-EMG values of eight muscles were calculated for each repetition and phase of the different exercises and normalized by the maximum RMS-EMG values recorded during MVC measurements for each subject. Examples of raw EMG signals recorded from a stroke subject (pre-rehab) and a healthy age-matched subject are shown in Figure 15 for one set of the Open/Close exercise with the Haptic Knob. The exercise consisted of ten passive opening (robot-driven extension) and active closing (flexion) movements of all fingers. The bursts of activity in the healthy subject’s EMG traces (Figure 15B) correspond to the onset of the ten flexion movements. The activity of most muscles is considerably reduced during passive extension of fingers. The EMG activity of the stroke subject (Figure 15A) shows much less modulation of muscle activity in all muscles (with the exception of 1DI during the closing movement) with little difference in activity between the two movement phases of the exercise. The mean RMS-EMG values (non-normalized) of each muscle, corresponding to the ten trials shown in Figure 15, are listed in Table 9 for both subjects.
Figure 15: Examples of raw EMG activity

A
Raw EMG activity recorded from muscles of one A) stroke subject (S1) pre-rehab and B) healthy age-matched subject (H5).
Table 9: Mean RMS-EMG values (SEM)

<table>
<thead>
<tr>
<th></th>
<th>Healthy</th>
<th>Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Open</td>
<td>Close</td>
</tr>
<tr>
<td>ECR</td>
<td>0.0348 (0.0030)</td>
<td>0.0546 (0.0030)</td>
</tr>
<tr>
<td>EDC</td>
<td>0.0651 (0.0043)</td>
<td>0.0923 (0.0046)</td>
</tr>
<tr>
<td>FCU</td>
<td>0.0150 (0.0015)</td>
<td>0.0237 (0.0020)</td>
</tr>
<tr>
<td>FDS</td>
<td>0.0126 (0.0009)</td>
<td>0.0240 (0.0015)</td>
</tr>
<tr>
<td>PT</td>
<td>0.0101 (0.0010)</td>
<td>0.0176 (0.0030)</td>
</tr>
<tr>
<td>BI</td>
<td>0.0151 (0.0010)</td>
<td>0.0145 (0.0007)</td>
</tr>
<tr>
<td>1DI</td>
<td>0.0427 (0.0165)</td>
<td>0.1088 (0.0067)</td>
</tr>
<tr>
<td>ADM</td>
<td>0.0107 (0.0012)</td>
<td>0.0163 (0.0008)</td>
</tr>
</tbody>
</table>

Individual PCAs for each stroke and healthy subject generated 8 principal components (PCs), consisting of weighting coefficients for each muscle, loading scores for each PC for each phase of a repetition and the percentage of the total variance in the data set accounted for by each PC. Data recorded from all exercises on one robot were included in each PCA. The weighting coefficients for each muscle for PC1 from two representative PCAs (one for a stroke and one for a healthy subject) are shown in Figure 16. Subjects performed the Proprioception exercise with the Haptic Knob.
Figure 16: Weighting coefficients calculated by two PCAs

A

PC1 = 96.7%

Weighting coefficient

ECR  EDC  FCU  FDS  PT  BI  1DI  ADM

B

PC1 = 98%

Weighting coefficient

ECR  EDC  FCU  FDS  PT  BI  1DI  ADM

Weighting coefficients of the first principal component of a PCA calculated for A) a stroke subject’s (S4) pre-rehab exercises and for the same exercise for B) an age-matched healthy subject (H7).
PC1 of the stroke subject had approximately twice as much muscle activation (relative to the subject’s MVC) in FCU and more than twice as much in the remaining muscles (FDS, PT, BI, 1DI, ADM) compared to the healthy age-matched subject. The EDC muscle is almost silent for the stroke subject while it is the predominant active muscle in the case of the healthy subject. PC1 accounts for 96.7 and 98 % of the variance in the signal for stroke and healthy subjects, respectively. Even though the percentage of the variance accounted for by PC1 is similar in both cases, the profile of the activation patterns are very different indicating that the percentage of variance accounted for by PC1 does not capture differences in activation patterns.

The loading scores for the data used in generating the PCs in Figure 16 are shown in Figure 17. The first twenty repetitions represent the “tuning” phase and the last twenty repetitions are for the “setting” phase of the exercise for the first and second sets, respectively. Variation in the values of the loading scores from one repetition to another could represent systematic fluctuations in the strength of muscle activation or in the relative contribution of one predominant pattern (e.g. PC1) compared to another (e.g. PC2). The loading scores appear to fluctuate more and in a more random fashion from repetition to repetition for the stroke subject than the healthy subject.
Figure 17: Loading scores calculated by two PCAs

A

B

Loading scores of individual repetitions for A) stroke subject (S4) and B) healthy age-matched subject (H7).
Next, the minimum number of PCs required to account for the 95% of the variance in each data set was determined for each of the 32 PCAs (Table 10). For all but one case, the minimum number of PCs was the same for both of the healthy subjects in a group. The exception was for subjects H5 and H6 in the case of the Haptic Knob exercises. For half of the stroke subjects, two PCs were needed to account for 95% of the variance in the pre-rehab data sets, whereas for the other half of the subjects PC1 alone accounted for 95% or more of the variance. In general, the minimum number of PCs required to explain 95% of the variance in the data set was not less for stroke subjects than healthy age-matched subjects, nor did the minimum number of PCs increase from pre- to post-rehab. Therefore, both hypotheses were proven wrong.
Table 10: Minimum number of PCs needed to account for 95% of the variance

<table>
<thead>
<tr>
<th>Haptic Knob</th>
<th>Subjects</th>
<th>HandCARE</th>
<th>Subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>S1</td>
<td>H1</td>
<td>H2</td>
</tr>
<tr>
<td>Pre-rehab</td>
<td>2</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td></td>
<td>S2</td>
<td>H3</td>
<td>H4</td>
</tr>
<tr>
<td>Pre-rehab</td>
<td>2</td>
<td>1</td>
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</tr>
<tr>
<td>Post-rehab</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>S3</td>
<td>H5</td>
<td>H6</td>
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<tr>
<td>Post-rehab</td>
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<tr>
<td></td>
<td>S4</td>
<td>H7</td>
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<td>1</td>
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</tr>
<tr>
<td>Post-rehab</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Minimum number of PCs needed to account for 95% of the variance in the RMS-EMG data set for Haptic Knob and Hand Care exercises. “S” and “H” indicate stroke and healthy subjects, respectively.

Because the hypotheses were proven to be wrong, it was decided to look for changes in the profile of the muscle activation patterns. The activation patterns of healthy subjects as represented by PC1 were first compared among exercises performed on both robots. Distinct differences in the PC1 profiles among exercises for healthy subjects would indicate whether or not similar differences could be expected for stroke subjects. PC1 for 7 of the 8 healthy subjects showed distinctly different profiles for the exercises performed with the two robots. PC1 profiles for a healthy subject are shown in Figure 18 for the Proprioception exercise with the Haptic Knob and the Somatosensory exercise with the HandCARE. In the Proprioception exercise, the subject predominantly used a finger extensor muscle (EDC) while in the Somatosensory exercise the
dominant muscle was a wrist flexor (FCU) and the activation of the abductor of the little finger muscle (ADM) was higher for this exercise. PC1 accounted for 98 and 99% of the variance in the signal for Proprioception and Somatosensory exercises, respectively.
Figure 18: Dissimilar PC1 profiles of a healthy subject

PC1 = 98%

PC1 of a healthy subject (H7) from PCAs for the A) Proprioception exercise and B) Somatosensory exercise.
For one healthy subject the PC1 profile was similar for Haptic Knob and HandCARE exercises (Figure 19). The subject performed the Pronation/Supination exercise with the Haptic Knob and Hangman and Somatosensory exercises with the HandCARE. However, PC2, which accounts for the second largest amount of the variance in the data set, had markedly different profiles for exercises performed with the two robots (Figure 20). The subject used FDS, BI and 1DI muscles for the exercise performed with the Haptic Knob while these muscles were silent during exercises performed with the HandCARE. ECR and ADM were the most active muscles for the exercises performed with the latter robot. Negative weighting coefficients represent inhibition of the corresponding muscle (only if the loading scores are positive).
Figure 19: Similar PC1 profiles of a healthy subject

A

PC1 = 95.8%

B

PC1 = 91.8%

PC1 of a healthy subject’s (H5) muscle activity from PCAs for one exercise performed with the A) Haptic Knob (Pronation/Supination) and B) two exercises performed with the HandCARE (Hangman and Somatosensory).
Figure 20: PC2 profile of a healthy subject

A

PC2 = 3.1%

B

PC2 = 5.9%

PC2 profiles for the PCAs corresponding to the PC1 profiles in Figure 19.
Similarly, PC1 profiles for stroke subjects based on the pre-rehab data sets were compared for exercises performed with both robots. All subjects demonstrated the ability to change their muscle activation patterns among different exercises as demonstrated by distinctly different PC1 profiles. PC1 profiles for one stroke subject are shown in Figure 21 for Haptic Knob and HandCARE exercises. The subject performed the Open/ Close exercise and the Pronation/ Supination exercise with the Haptic Knob and the Elastic exercise with the HandCARE. The PC1 profile shows the greatest activity in the BI and 1DI for the Haptic Knob exercises while 1DI activity decreases to almost half and the activity of finger flexors and extensor (FDS, EDC) muscles are relatively higher for the HandCARE exercises.
Figure 21: Dissimilar PC1 profiles of a stroke subject calculated from both robots

A

PC1 = 78.3%

B

PC1 = 94.6%

PC1 of a stroke subject's (S1) pre-rehab muscle activity from PCAs for two exercises performed with the A) Haptic Knob (Open/ close and Pronation/Supination) and B) one exercise performed with the HandCARE (Elastic).
The same process was carried out in comparing stroke subjects’ pre- and post-rehab activation patterns for the same exercises. All subjects showed distinct differences in PC1 profiles between pre- and post-rehab (with the exception of one subject). PC1 profiles for a stroke subject are shown in Figure 22 for the Haptic Knob exercises and in Figure 23 for the HandCARE exercises. The subject performed Open/ Close and Pronation/ Supination exercises with the Haptic Knob and Elastic exercise with the HandCARE. In the Haptic Knob exercises, the subject predominantly used BI and 1DI muscles in pre-rehab while BI activity was dramatically reduced post-rehab. In the Elastic exercise, the relative amount of BI activity was reduced post-rehab compared to pre-rehab while the relative activity of 1DI increased. Activity in EDC and FDS also became less prominent.
Figure 22: Dissimilar PC1 profiles of a stroke subject calculated from Haptic Knob exercises

A)

PC1 = 78.3%

B)

PC1 = 95.1%

PC1 of a stroke subject’s (S1) muscle activity from PCAs for the Open/ close and Pronation/Supination exercises performed with the Haptic Knob A) pre-rehab and B) in post-rehab.
Figure 23: Dissimilar PC1 profiles of a stroke subject calculated from HandCARE exercises

A

PC1 = 94.6%

B

PC1 = 90.2

PC1 of a stroke subject's (S1) muscle activity from PCAs for the Elastic exercise performed with the HandCARE A) pre-rehab and B) in-post rehab.
The PC1 profiles for the one subject whose patterns were similar for both the exercise performed with the Haptic Knob and the exercises performed with the HandCARE are shown in Figure 24 and Figure 25. However, the PC2 profile was very different pre- and post-rehab (Figure 26). One of the major differences in the PC2 profile post-rehab compared to pre-rehab was that the weighting coefficient for the PT muscle, which is a functional muscle for the Pronation/Supination exercise, changed from negative (inhibition) to positive (excitation), i.e. it added to the level of activation of this muscle in the PC1 profile rather than reducing the level of activation. The same is true of the extrinsic finger extensor muscle, EDC. On the other hand, the activity of ADM, which plays less of a functional role in the exercise, was inhibited by PC2 post-rehab. In the case of the exercises performed with the HandCARE (Figure 27) the PC2 profile shows a reduction in the weighting coefficients of all muscles except ADM post-rehab. These differences in the PC2 profiles indicate that the subject could somewhat modify her muscle activation for different exercises even though her predominant activation profile (PC1) did not change from pre- to post-rehab.
Figure 24: Similar PC1 profiles of a stroke subject calculated from Haptic Knob

A

PC1 = 94.8%

B

PC1 = 90.7%

PC1 of a stroke subject’s (S3) muscle activity from PCAs for the Pronation/Supination exercise performed with the Haptic Knob A) pre-rehab and B) in post-rehab.
Figure 25: Similar PC1 profiles of a stroke subject calculated from HandCARE

A

PC1 = 86.5%

B

PC1 = 91.1%

PC1 of a stroke subject’s (S3) muscle activity from PCAs for the Hangman and the Somatosensory exercises performed with the HandCARE A) pre-rehab and B) in post-rehab.
Figure 26: PC2 profiles of a stroke subject calculated from Haptic Knob

A

PC2 = 3.6%

B

PC2 = 7.5%

PC2 profiles for the PCAs corresponding to the PC1 profiles in Figure 24, A) pre-rehab and B) post-rehab.
Figure 27: PC2 profiles of a stroke subject calculated from HandCARE

A

PC2 = 8.9%

B

PC2 = 3.8%

PC2 profiles for the PCAs corresponding to the PC1 profiles in Figure 25, A) pre-rehab and B) post-rehab.
4.1.2 Comparison of amount of co-contraction

Raw EMG traces of representative subjects illustrate differences between muscle activation patterns of stroke and healthy age-matched subjects (Figure 28) and between pre- and post-rehab recording sessions (Figure 29). Figure 28 illustrates the EMG activity recorded from two antagonistic muscles (PT and BI) of a stroke (pre-rehab) and a healthy subject. Subjects performed five pronation and five supination movements. The healthy subject’s EMG signal shows a clear reciprocal activation pattern (Figure 28B). On the other hand, the stroke subject frequently shows co-activation of PT when BI is activated to initiate supination (Figure 28A). Thus, a much stronger BI contraction is required to perform the movement. PT activity during pronation is very weak.
Figure 28: Raw EMG activity recorded from a stroke and healthy subject

A

EMG activity recorded in pronator teres (PT) and biceps (BI) muscles from a A) stroke subject (S1) pre-rehab and B) healthy age-matched subject (H5).

Figure 29 illustrates EMG activity recorded from two antagonistic muscles (EDC and 1DI) of a stroke subject during pre- and post-rehab sessions. The EMG was recorded during the Open/Close exercise performed with the Haptic Knob. The exercise consisted of ten repetitions of passive opening (robot activated) and active closing movements of the hand. Subjects were instructed to not to resist the opening movement by keeping the hand relaxed during the opening phase of the exercise. Each burst of 1DI EMG activity corresponds to the onset of a closing movement. Pre-rehab EMG activity (Figure 29A) demonstrates tonic activation of the EDC muscle both during closing and
opening whereas post-rehab EDC activity appears stronger and a clear reciprocal activation pattern has been established (Figure 29B) indicating that the subject was able to overcome some of the flexor spasticity by activating the extensor muscle during the opening phase of the exercise.

Figure 29: Raw EMG activity recorded from a stroke subject

EMG activity recorded in extensor digitorum communis (EDC) and first dorsal interosseus (1DI) muscles from a stroke subject’s (S1) A) pre-rehab and B) post-rehab recording session

A Critical Binomial test was conducted to compare stroke subjects’ pre-rehab muscle co-contraction with that of healthy age-matched subjects. A total of 80 (10 × 2 × 4, total number of exercises × healthy subjects × muscle pairs) cases were classified, according to whether stroke subjects had a greater co-contraction index (CI) than healthy subjects. In order to achieve a 95% confidence level, 33 cases where CI was higher for stroke subjects than healthy
subjects were needed. There were 57 cases in which the CI was higher for stroke subjects, indicating that stroke subjects have significantly greater co-contraction than healthy subjects.

A second Critical Binomial test was conducted to determine whether co-contraction was reduced post-rehab compared to pre-rehab. A total of 40 cases were tested (10 × 4, total number of exercises × muscle pairs). Of the 40 cases, 20 represented lower CI post-rehab compared to CI pre-rehab. To achieve a 95% confidence level there should have been at least 25 cases in which CI of stroke subjects was lower post-rehab. The results indicate that the number of cases in which subjects had a decreased CI from pre- to post-rehab was not significant and could not be distinguished from chance occurrence.

4.1.3 Clinical assessment scores

Table 11 illustrates the scores of three clinical assessments for pre-, post-training and follow-up sessions. Two subjects showed 12% and 11% (subject 1 and subject 2, respectively) increase in the scores of Jebsen Test of Hand Functions. Both subjects’ scores decreased in the follow-up assessment session (one month after the end of the training). The other two subjects’ Jebsen Test of Hand Function test scores (subject 3 and subject 4) decreased in the post-training assessment session indicating no change occurred, in general, in the functions of post-stroke subjects. No subject demonstrated a considerable change in Purdue Pegboard and Action Research Arm test scores from pre- to post training and in follow-up sessions. No statistical test was conducted because of the small size of the subject group.
Table 11: Clinical assessment scores

<table>
<thead>
<tr>
<th>Subject</th>
<th>Jebsen Test of Hand Function (sec)</th>
<th>Purdue Pegboard Test (piece)</th>
<th>Action Research Arm Test (normal=57)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre-training</td>
<td>Post-training</td>
<td>Follow-up</td>
</tr>
<tr>
<td>1</td>
<td>233.1</td>
<td>261.7</td>
<td>172.8</td>
</tr>
<tr>
<td>2</td>
<td>192.3</td>
<td>215.4</td>
<td>199.1</td>
</tr>
<tr>
<td>3</td>
<td>196.2</td>
<td>184.1</td>
<td>159.1</td>
</tr>
<tr>
<td>4</td>
<td>184.37</td>
<td>177</td>
<td>176.75</td>
</tr>
</tbody>
</table>

Table 12 and Table 13 illustrate the outcomes of Jamar Grip Dynomometer test and Modified Ashworth scale, respectively. No subject showed a considerable change in grip strength from pre- to post-rehab and in follow-up assessment sessions. Two subjects who had spasticity in their wrist flexors (subject 1 and subject 2) showed reduced spasticity after training. One of these subjects (subject 1) had further decrease in spasticity at the follow-up assessment session.
Table 12: Outcomes of grip strength test

<table>
<thead>
<tr>
<th>Subject</th>
<th>Jamar Grip Dynamometer (kg±SD)</th>
<th>Pre-training</th>
<th>Post-training</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>19.6±1.5</td>
<td>19.6±2</td>
<td>20.3±1.5</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td>13.3±0.6</td>
<td>15±1</td>
<td>17.3±0.6</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td>11.7±2.3</td>
<td>13.3±0.6</td>
<td>13.7±0.6</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td>23±0</td>
<td>22±2.6</td>
<td>23±1</td>
</tr>
</tbody>
</table>

Table 13: Outcomes of spasticity measurement

<table>
<thead>
<tr>
<th>Subject</th>
<th>Wrist flexors</th>
<th>Finger flexors</th>
<th>Wrist flexors</th>
<th>Finger flexors</th>
<th>Wrist flexors</th>
<th>Finger flexors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1+</td>
<td>1+</td>
<td>1</td>
<td>1+</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Modified Ashworth Scale tests the degree of spasticity in five scales (scores=1, 1+, 2, 3, 4. 1 means no resistance to movement, 4 means movement cannot be completed due to high resistance).

Each subject had reported improved hand functions and ADL which they have observed after robot-assisted rehabilitation. These self-reported improvements were demonstrated in Table 14. One common activity that all subjects reported is more secure grasping and/or confidence when carrying objects.
# Table 14: Self-reported improved hand functions and ADL

<table>
<thead>
<tr>
<th>Subject 1</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>More flexibility of the right hand</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Better grasping of objects such as bottle of water</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved hand functions while performing ADL such as: opening/ closing a door knob, turning on/off light switch, opening a twist bottle lid, washing dishes/ utensils</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Typing with Index and Middle finger</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>More secure grasping of objects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bigger ROM of Thumb</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Able to involve Thumb for stronger grasp</td>
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<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject 2</th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>More secure grasping of objects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved speed of performance</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Stronger pinching</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Using right hand when eating</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Better control while carrying a full plate</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved ADL such as: brushing teeth, carrying a grocery bag, combing hair</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved little finger functions such as pinching</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject 3</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>More secure grasping of heavy objects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved hand functions while performing ADL such as: carrying a mug, pouring water from a pitcher, scrapping the dishes, using a knife, dialing phone numbers</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>More independency between the fingers</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Better fine control of movements</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Self-confidence while using the right hand</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Improved functions of Index finger such as pinching, using scissors, typing</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Subject 4</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Using right hand to start the car engine</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Better stabilization of objects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>More confidence for grasping objects</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>More participation of the right hand into tasks</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Better control while carrying a full plate</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CHAPTER 5: DISCUSSION

The objective of this study was to investigate the abnormalities in wrist and finger muscle activation patterns of post-stroke survivors compared to healthy controls as well as the changes in these patterns after robot-assisted hand rehabilitation. Robot-assisted rehabilitation of upper extremity is being used increasingly; however, the effects of therapies on hand and finger muscle function have not been quantified yet. We used Principal Component Analysis (PCA) to provide a measure of the ability of stroke survivors to vary their patterns of muscle activity before and after robot-assisted hand rehabilitation. We also estimated the amount of co-contraction of antagonistic muscles before and after rehabilitation. The outcomes from the two analyses were compared with healthy age-matched controls.

We assumed that with different exercises healthy subjects would use distinct muscle activation patterns that would result in different PCs accounting for a similar amount of variance in the activity. This assumption was based on the idea that there would normally be considerable flexibility in the neural control of hand and wrist muscles. On the other hand, muscle coordination in hemiplegic patients has been found to be very stereotyped (Hu et al., 2007). Therefore, it was assumed in this study that there would be less flexibility, i.e. less variety, in the activation patterns of stroke survivors than age-matched healthy subjects. However, the results proved that our hypothesis was wrong. According to the
findings of PCA, the number of principal components required to account for the 95% of the variance in stroke subjects’ muscle activation was not less than healthy subjects. What was surprising was that there was so little variety in the activation patterns of the healthy subjects. The results showed that in eleven out of sixteen cases, one PC was enough to account for the 95% of the variation in healthy subjects’ activity suggesting that one particular pattern of coordination was most common between muscles. Similarly, for stroke survivors four out of eight cases had one pattern which accounted for at least 95% of the variation in the muscle activation pre-rehab. Finally, all subjects (healthy and stroke) had at most two PCs to account for the greatest portion of the variety in the signal.

One reason might be that the exercises were similar in terms of the required muscle activation. Because the muscle activity was quantified in terms of RMS-EMG the PCA might not distinguish between co-activation and reciprocal activation of antagonistic muscles. For example, the same principal component could account for muscle activity in an exercise that required extensor muscle activation early and flexor activity later and for muscle activity in an exercise that required flexor activation early and extensor activation late. Exercises that had very similar muscle activation requirements (e.g. Open/Close and Proprioception exercises with the Haptic Knob required the use of the flexors and extensors of the hand which are commonly co-activated during grasping) also contributed to the inability of the PCA to identify more than one primary activation pattern.

By comparing PC profiles, we were able to show that the primary activation patterns (PC1) of both stroke (pre-rehab) and healthy subjects were
generally different for different exercises with the two robots. Furthermore, in seven out of eight cases, stroke subjects changed their PC1 profiles pre- to post-rehab. We found a variety of PC1 profiles, even though the number of components needed to account for 95% of the variance in the muscle activation did not necessarily differ. It is clear from this that the percentage variance accounted for by the PCs was not sensitive to changes in the profile of PC1 and therefore it did not capture changes in the activation patterns.

PC1 profiles of subjects (healthy and stroke (pre-rehab)) were different regardless of age group or robot used in training with the exception of two healthy subjects in the same age group who had similar PC1 profiles for different Haptic Knob exercises. Diverse PC1 profiles among subjects were obtained because each subject has a unique way of controlling his/her muscles. These different muscle activation patterns were not related to performance because outcomes were often similar, i.e. time taken to complete the exercise and/or scores. However, changes in PC1 profiles might also be explained by differences in the placement of electrodes from one recording session to another which would result in muscle activity being recorded from different motor units. The relative location of the electrodes to different muscles could also be a factor, in terms of the amount of cross-talk picked up in the recorded signal, although this is unlikely in the present study because the electrodes were placed by following a protocol which enabled us to be consistent among subjects and recording sessions. The quality and the amplitude of the signal for each muscle were checked using a real-time feedback display before the actual recording started.
PCA reduces the variety by having few PCs that account for the large amount of variety in the signal. Therefore, if the objective of the analysis is to detect the variety in the EMG signal, PCA is not the most beneficial analysis to use. On the other hand, it is reliable to detect the changes in the most common pattern of muscle activity and; therefore, it is appropriate to use in studies that compare the patterns of activities recorded at different time of the period (e.g. pre- and post-rehab).

In addition, PCA can be used for more than one purpose. For example, depending on how PCA was used, one can emphasize the changes in relative amplitude of the muscle activity compared to changes in temporal pattern of the muscle activity. However, the latter method is dependent on the duration of each repetition to be same; therefore, the data analysis could be difficult, if the recorded data set does not involve repetitions with same duration.

The amount of co-activation in wrist and hand muscles was measured before and after rehabilitation in order to observe the effect(s) of rehabilitation on stroke survivors’ ability to independently activate antagonistic muscles. The results showed that the amount of co-contraction in pairs of antagonistic muscles was higher for the majority of the stroke subjects’ than for healthy controls. This is in agreement with the findings of previous studies. Abnormal co-activation of flexor and extensor muscles on the hemiparetic side has been shown for fingers (Kamper and Rymer, 2001), wrist (Hammond et al., 1988), elbow and shoulder (Dewald et al., 1995; Gowland et al., 1992) and knee (Knutsson and Martensson, 1980). After two months of robot-assisted hand training, in half of the cases the
amount of co-contraction of wrist and finger muscles did not decrease. It has been previously shown that robot-assisted upper extremity rehabilitation can reduce the amount of abnormally high co-activation and improve functions (Hu et al., 2007). The results from the study of Hu et al. (2007) may differ from the present study for several reasons including the differences in 1) methodology, 2) therapy techniques, and 3) subject groups.

Hu et al. (2007) investigated the variation of muscle co-activation patterns during the course of robot-assisted rehabilitation of elbow flexors and extensors. Improvement in co-contraction indexes, which were quantified in terms of the amount of overlapping EMG activity for a muscle pair over the length of the signal, for eight muscle pairs was found to occur after the first half of the training. In this study, more intense therapy (1.5 hr therapy sessions, 3-5 times a week) was applied for a longer duration (20 sessions), and co-activation indexes were calculated from elbow and shoulder muscles compared to the present study in which wrist and hand muscle EMG was recorded and compared after the completion of a 16 session (1 hr, 2 times a week) rehabilitation program. Improvements in distal, i.e. hand and finger, muscle activity patterns and functions may take longer than more proximal muscles because impairments are more severe. Considering that the improvement in the co-activation of shoulder and elbow muscles occurred only after the first half of the training with more intense therapy, longer and more intense therapy sessions might be needed in order to reduce the amount of co-activation in hemiplegic hand muscles. Moreover, the effects of rehabilitation are reduced in chronic stages of stroke and
any delay may require more intense or longer duration therapy to achieve the same functional gains. This is especially important in hand rehabilitation. Previous studies have shown that only early initiation of rehabilitative therapy after stroke can improve fine digit control and reliance on somatosensory feedback in primates (Biernaskie et al., 2004). It is, therefore, necessary to have longer and more intense therapies and initiate these therapies early after the brain damage in order to gain the most benefit from hand rehabilitation. Other robot-assisted arm rehabilitation protocols that resulted in reduced motor impairment of the affected extremity, increased strength and/or functional recovery also involved greater intensity and/or longer duration therapy (Lum et al., 2002(1); Lum et al., 2002(2); Kahn et al., 2006, Volpe et al., 1999). These studies trained reaching movement of the arm and measured the function of the upper extremity by clinical assessments.

Another factor that may have contributed to co-contraction in the present study is fatigue related to MVC measurements. The MVC measurements consisted of maximum force exertion in sixteen functional movements repeated three times and were performed just before the exercise sessions which also involved resistive force training. Considering the size of the hand and finger muscles and the lack of strength of stroke survivors, the procedure might have resulted in fatigue and increased spasticity of muscles. Even though a resting period was given, it might have not been enough to overcome the spasticity. The protocol for MVC recording could be modified to reduce the chance of muscle fatigue by providing subjects with a longer resting period between the MVC
measurement and EMG recording, applying muscle stretching before and between measurements, and minimizing the number of required functional movements.

Hu et al. (2007) calculated the amount of overlapping activity of a muscle pair by considering the phasing of antagonistic muscle activation. For example, if the two antagonistic muscles were reciprocally active, then the overlapping EMG activity (i.e. amount of co-contraction) would be less than if they were synchronously active. In the present study, the duration of antagonistic muscle activity was not considered which could explain the difference between the results of two studies.

The nature of the training task was also different in the two studies. An active-assistive exercise was used in the study of Hu et al. (2007) in which assistive torque was provided during the extension movement in proportion to the recorded EMG. Stroke survivors mostly suffer from flexor spasticity meaning that extension is a more difficult movement. When performing robot assisted extension, subjects may generate less co-contraction of spastic flexors and weak extensors compared to active extension training which requires more muscle activation. In the present study, more than one type of training was sometimes performed in the same session including active, active-assistive and active-resistive training. Co-contraction values were pooled for the analysis for three kinds of exercises. It could be that particular exercises had better effects on reducing co-contraction. However, since there were a small number of subjects
and not all subjects performed all three types of training this study did not attempt to relate the amount of co-contraction with particular kinds of training.

The severity of post-stroke impairment may also affect the outcomes of rehabilitation. More severely impaired patients were shown to benefit more from active-assisted movement therapies because of a potential increase in motivation, benefits from proprioceptive stimulation and benefits to soft tissue provided by the mechanical assistance (Lum et al., 2002). Therapies involving active (unassisted) and repetitive movements may be more effective with less severely affected patients because active practice, which is critical to the recovery process in motor cortex (Nudo et al., 1996), can be facilitated. The levels of the impaired function were considered when designing individual rehabilitation programs in the present study. More severely disabled functions were trained with the help of assistive force and less severe impairments were assigned to active or active-resistive exercises. However, our study involved a wide range of impairment levels and stroke types. This may have contributed to variability in our results. Because of the limited number of subjects, the potential relationship between the changes in activation patterns and types of training or severity of impairment was not investigated.

Even though no significant changes were observed in the co-activation patterns of stroke survivors in the present study, all subjects progressed in duration and intensity of the exercises from the first session to the last session of the program. Noticeable positive changes were observed in the performance of subjects during and after the rehabilitation. All subjects were able to perform
exercises with an increased difficulty level such as higher resistive force/torque applied by the robot and/or required precision with the progression of the rehabilitation. They reported less effort while training and compensated for the changes in the exercises. Exercises performed by two of the subjects were changed during the rehabilitation after reaching a plateau with a high difficulty level. The time required to complete each exercise set was greatly reduced by the end of the rehabilitation with improved performance. In addition, subjects reported improved hand function and better performance of ADL in their self-reported evaluation of hand function.

Amelioration of abnormal co-contraction patterns is shown to be associated either with a reduction in the level of activity of muscles or with reduced duration in the co-contraction phase of the muscle pairs, i.e. greater reciprocal activation (Hu et al., 2007). Even though we have not investigated changes in the duration of co-contraction in the present study, improved performance and the self-reported improvements in hand function after rehabilitation may have been the consequence of better reciprocal activation of antagonistic muscles rather than the result of changes in the amplitude of the activation. Furthermore, stroke survivors can adapt their EMG pattern to the task mechanics in a similar way with healthy controls, even though their coordination pattern remains impaired (e.g. compensation mechanisms) (Kautz et al. 2005). Better performance could result from non-neural factors such as increased endurance, increased strength or more efficient use of abnormal compensatory mechanisms rather than improvement in control by the central nervous system
(Kautz et al. 2005). Moreover, abnormal activation patterns are not necessarily the primary cause of movement impairment. Rather, weakness in the agonist muscles (Tang and Rymer, 1981) or weakness and spasticity together, i.e. loss of reciprocal motor neuron control (Sahrmann and Norton, 1977; Hammond et al., 1988) resulting from hemiplegia, were suggested to be directly related with impaired function. Any positive changes in these factors could contribute to the improved functions. However, based on the results of the Jamar Dynamometer test and Modified Ashworth Scale in the present study, no significant increase in grasping strength or decrease in spasticity of wrist flexor muscles was observed after rehabilitation. Therefore, better use of compensatory mechanisms or increased endurance is more likely to explain the improved hand functions after robot-assisted hand rehabilitation. This is in agreement with findings of the study of Fischer et al. (2007) in which the task performances of chronic stroke subjects improved after a rehabilitation program involving repetitive practice with finger extension assistance; even though, no significant change in grip strength and spasticity was observed. The question of whether there are changes in the timing of EMG activity in addition to changes in the amplitude would have to be addressed in the future studies.

In general, the assessment scores did not show any change in function from pre- to post-rehab, consistent with the results of EMG analysis. However, this study did not involve enough subjects to test the significance of assessment scores before and after rehabilitation or the correlation between better performance and the assessment scores. More subjects are needed before
definite conclusions can be reached about the effects of the robot-assisted hand rehabilitation on hand functions.

A study based on a larger number of subjects (n=9), which was conducted to train hand and wrist functions with Haptic Knob subsequent to our feasibility study, showed a positive correlation between improved performance of the exercise and functional assessment scores after training (Lambercy et al. 2009). The subjects had homogeneous improvement of hand and arm function and decreased spasticity in flexor muscles of all parts of the upper extremity (shoulder, elbow, wrist and thumb flexors) after 6 weeks (3 × week, 1 hr sessions) training with the Haptic Knob (Lambercy et al. 2009). Improvements obtained with the training were maintained 6 weeks after the end of the therapy.

There are several limitations in this study. Because our sample size was limited to four post-stroke survivors, it might be that our analysis was not able to detect a significant difference or change in the muscle activity patterns after rehabilitation. A wide range in the time post-stroke (2 to 20 years), age (56 to 83) and severity of impairments likely contributed to a high variability in our results. Another limitation that needs to be addressed in future studies is that the clinical assessments in this study were not conducted by a blinded therapist. However, because the assessment outcomes were consistent with EMG measures, it can be said that there was not any bias in the assessment scores.

In terms of the safety of the robots, no subject recorded any discomfort or pain during or after testing suggesting that these robotic devices were safe to use in hand rehabilitation of both stroke survivors and healthy individuals.
CHAPTER 6: CONCLUSIONS

- Healthy individuals’ muscle activation patterns did not have more variety compared to muscle activity recorded from stroke survivors suggesting that impairments in hand function of post-stroke hemiparetic subjects is not due to lack of flexibility in muscle activation, but rather because they may not be able to produce the right coordination patterns.

- There was no significant change in the percentage of the variation in muscle activation accounted for by PC1 from pre- to post-rehab because it was not sensitive to changes in the muscle activation profile.

- Both stroke (pre-rehab) and healthy subjects generated different principal (PC1) muscle activation profiles for different exercises and PC1 profiles of stroke subjects (with the exception of one subject) also changed from pre- to post-rehab.

- Based on the co-activation measurements, the amount of co-contraction in the majority of the stroke subjects’ antagonistic muscle pairs was higher than in healthy controls.

- After two months of robot-assisted hand training, the amount of co-contraction of wrist and finger muscles did not decrease in half of the cases.
• All subjects showed improvement in their performance during and after rehabilitation and reported improved hand functions of ADL in their self-reported hand interviews.

• There was no change in the assessment scores from pre- to post-rehab consistent with the results of EMG measurement. However, because of the small number of subjects, the correlation between the improved performance and the clinical assessment scores could not be tested in this study.

• No subject recorded discomfort or pain during or after robot-assistant hand rehabilitation suggesting that robotic devices were safe to use in hand rehabilitation of both stroke survivors and healthy individuals.
APPENDIX A. CONSENT FORM FOR STROKE SUBJECTS

Form 2- Informed Consent By Participants In a Research Study

The University and those conducting this research study subscribe to the ethical conduct of research and to the protection at all times of the interests, comfort, and safety of participants. This research is being conducted under permission of the Simon Fraser Research Ethics Board. The chief concern of the Board is for the health, safety and psychological well-being of research participants.

Should you wish to obtain information about your rights as a participant in research, or about the responsibilities of researchers, or if you have any questions, concerns or complaints about the manner in which you were treated in this study, please contact the Director, Office of Research Ethics by email at hweinber@sfu.ca or phone at 604-268-6593.

Your signature on this form will signify that you have received a document which describes the procedures, whether there are possible risks, and benefits of this research study, that you have received an adequate opportunity to consider the information in the documents describing the study, and that you voluntarily agree to participate in the study.

Any information that is obtained during this study will be kept confidential to the full extent permitted by the law. Knowledge of your identity is not required.
You will not be required to write your name or any other identifying information on research materials. Materials will be maintained in a secure location.

Title: Pilot testing for virtual reality rehabilitation of arm and hand function after stroke

Investigator Name: Theodore Milner

Investigator Department: Kinesiology

Having been asked to participate in the research study named above, I certify that I have read the procedures specified in the Study Information Document describing the study. I understand the procedures to be used in this study and the personal risks to me in taking part in the study as described below:

Risks to the participant, third parties or society:

None

Procedures:

The subject will be required to complete a questionnaire related to hand function to determine their level of impairment. The subject will then perform a set of exercises using 3 different computer controlled mechanical devices. The exercises are designed to test strength and dexterity in using the hand.
Benefits of study to the development of new knowledge:

This study will aid in the development of a new robotic tool for rehabilitation of hand function in stroke patients. This tool will have the ability to assess impairment and rate of improvement of the subject’s hand function. The robot will integrate rehabilitation of the arm and hand to provide lasting improvements in acute or chronic stroke impairment patients. The rehabilitation will enable the stroke patient to increase their independence and has potential for at home use.

I understand that I may withdraw my participation at any time. I also understand that I may register any complaint with the Director of the Office of Research Ethics.

Director, Office of Research Ethics
8888 University Drive
Simon Fraser University
Burnaby, British Columbia
Canada V5A 1S6
+1 604 291 3447
email: dore@sfu.ca

I may obtain copies of the results of this study, upon its completion by contacting:
I have been informed that the research will be confidential.

I understand the risks and contributions of my participation in this study and agree to participate:

The participant and witness shall fill in this area. Please print legibly

- Participant Last Name:

- Participant First Name:

- Participant Contact Information:

- Participant Signature:

- Witness (if required by the Office of Research Ethics):

- Date (use format MM/DD/YYYY):
APPENDIX B. CONSENT FORM FOR HEALTHY SUBJECTS

Form 2- Informed Consent By Participants In a Research Study

The University and those conducting this research study subscribe to the ethical conduct of research and to the protection at all times of the interests, comfort, and safety of participants. This research is being conducted under permission of the Simon Fraser Research Ethics Board. The chief concern of the Board is for the health, safety and psychological well-being of research participants.

Should you wish to obtain information about your rights as a participant in research, or about the responsibilities of researchers, or if you have any questions, concerns or complaints about the manner in which you were treated in this study, please contact the Director, Office of Research Ethics by email at hweinber@sfu.ca or phone at 604-268-6593.

Your signature on this form will signify that you have received a document which describes the procedures, whether there are possible risks, and benefits of this research study, that you have received an adequate opportunity to consider the information in the documents describing the study, and that you voluntarily agree to participate in the study.

Any information that is obtained during this study will be kept confidential to the full extent permitted by the law. Knowledge of your identity is not required.
You will not be required to write your name or any other identifying information on research materials. Materials will be maintained in a secure location.

Title: Pilot testing for virtual reality rehabilitation of arm and hand function after stroke

Investigator Name: Theodore Milner

Investigator Department: Kinesiology

Having been asked to participate in the research study named above, I certify that I have read the procedures specified in the Study Information Document describing the study. I understand the procedures to be used in this study and the personal risks to me in taking part in the study as described below:

Risks to the participant, third parties or society:

None

Procedures:

The subject will be required to use a grip dynamometer to determine their grasping force. The subject will then perform a set of exercises using 2 different computer controlled mechanical devices. The exercises are designed to test strength and dexterity in using the hand.
Benefits of study to the development of new knowledge:

This study will aid in the development of a new robotic tool for rehabilitation of hand function in stroke patients. This tool will have the ability to assess impairment and rate of improvement of the subject’s hand function. The robot will integrate rehabilitation of the arm and hand to provide lasting improvements in acute or chronic stroke impairment patients. The rehabilitation will enable the stroke patient to increase their independence and has potential for at home use.

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The participant and witness shall fill in this area. Please print legibly

- Participant Last Name:
- Participant First Name:
- Participant Contact Information:
- Participant Signature:
- Witness (if required by the Office of Research Ethics):
- Date (use format MM/DD/YYYY):
FOR CONTACT IN REFERENCE TO THIS REVIEW
Application Number: 38144
Dr. H. Weinberg          B. Ralph, Ethics Officer
Director, Office of Research Ethics
Voice: (778) 782-6593     Voice: (778) 782-3447
Fax: (778) 782-6785       email: bralph@sfu.ca
Mobile: (778) 999-7251    email: hal_weinber@sfu.ca
Reference Ethics Policy 20.01: http://www.sfu.ca/policies/research/r20-01revised.htm

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<td>Investigator First Name</td>
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Grant Information
Submitted To Agency For Review | Yes |
Approved Subject To Ethics Approval | Yes |
Reviewed By Any Other Agency | Yes |
Title Of Grant | Virtual reality rehabilitation of arm and hand function after stroke |
Granting Agency | National University of Singapore Academic Research Fund |

APPROVED
By Hal Weinberg at 9:52 am, Mar 07, 2007

Page: 1
FOR CONTACT IN REFERENCE TO THIS REVIEW

Application Number: 38144

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e-mail: bralph@sfu.ca

Reference Ethics Policy 20.01: http://www.sfu.ca/policies/research/r20-01revised.htm

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<tr>
<td>Date Granting Agency Approval Began</td>
<td>September 1, 2005</td>
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<td>Date Grant Ends</td>
<td>August 31, 2008</td>
</tr>
<tr>
<td>Amendment Date</td>
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</table>
APPENDIX D. MODIFIED HAND FUNCTION INTERVIEW

Exclusion Questions

1. Which hand is affected? ................................................................. right / left
   a. Is this the dominant or non-dominant hand?
   .................................................................

2. What type of stroke do you have? (e.g. ischemic, hemorrhagic stroke)
   .................................................................

3. Is your arm also affected? ............................................................. right / left

4. Have you more than one stroke? ............................................... yes / no
   a. If no, did this stroke occur more than 6 months ago? ............. yes / no
   b. What was the date of the stroke? .............................................

5. Do you have any of the following:
   a. Other neurological diseases ................................................. yes / no
      (e.g. polio, Parkinson’s, multiple sclerosis, etc)
      If yes, list neurological disease (s) ........................................
   b. Medical problems ............................................................ yes / no
      If yes, list medical problem(s)..............................................
   c. Shoulder pain (any more than minor pain) ............................. yes / no
   d. Muscle wasting (Decrease in size of muscles) ....................... yes / no
   e. Perceptual or visual deficits ................................................ yes / no
      If yes list deficit(s)............................................................
   f. Neurological Disorders
      Apraxia (An inability to make purposeful movements) ............ yes / no
   g. Cognitive/ affective dysfunction ......................................... yes / no
      (e.g. Agnosia, Alexia, Astereognosia, etc)
      .................................................................
Pre-rehabilitation Interview:

Age:………………………..     Sex:…………………   Subject Number:……………
Date:………………………..  Examiner:………………………………………………

For the following activities of daily living, indicate the level of difficulty of this task by ranking the difficulty between 1 and 7. 1 being easy and 7 being difficult.

Activities:

<table>
<thead>
<tr>
<th>Activity</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening a door knob</td>
<td>easy</td>
</tr>
<tr>
<td>Opening / Closing a zipper</td>
<td>easy</td>
</tr>
<tr>
<td>Do-up / Undo a shirt button</td>
<td>easy</td>
</tr>
<tr>
<td>Using a key to open a lock</td>
<td>easy</td>
</tr>
<tr>
<td>Using scissors to cut paper</td>
<td>easy</td>
</tr>
<tr>
<td>Typing</td>
<td>easy</td>
</tr>
<tr>
<td>Handwriting</td>
<td>easy</td>
</tr>
</tbody>
</table>

Please comment if rating was four or greater about what aspect of the task is most difficult and why it was difficult. Explain if there was a difficulty in using your arm or hand during this task as well.

………………………………………………………………………………………………………
………………………………………………………………………………………………………
………………………………………………………………………………………………………
………………………………………………………………………………………………………

6. Is there particular digit/digits that you have the most difficulty using?........yes/ no
   a. If yes, which digit do you have the most difficulty with?
   b. What do you have difficulties doing with this digit? Demonstrate if necessary.

7. Do you experience weakness in your hand or arm when performing a particular task?.................................................................yes/ no
   a. If so, describe the type of task that causes this.
   b. What specific part of this task makes it causes the weakness?
   c. Do you feel the weakness in a specific area? Can you point to where this is? (Identify muscle group or area).
   d. What type of activity produces this weakness? E.g. repetitive, heavy, type of motion?

8. Do you experience any pain when performing any tasks with your hand/ arm?.................................................................yes/ no
   a. If so, which specific part of this task causes the pain?
   b. Do you feel the pain in a specific area? Can you point to where this is? (Identify muscle group or area).
   c. What types of work produces this pain? E.g. repetitive, heavy, type of motion?
9. Have you noticed an improvement in the function of your arm or hand in the time since your stroke?.................................................................yes/ no
   a. If so, how has it improved?

10. Have you continued to notice improvement in function of your arm or hand in recent months/ is it still improving?.........................................................yes/ no
    a. If so, how has it improved?

11. Do you participate in any physical activity/ sports? ......................... yes/ no
    a. If yes, what do you participate in?
    b. Did you participate in physical activity/ sports in the past?.............yes/ no
       If so, what did you participate in?

12. What activities would you most like to be able to do that you are no longer able to participate in?

13. Do you do any exercises with a therapist to regain hand/ arm function?.....yes/ no
    a. If yes, describe the exercises that you do?

14. Do you do any exercises on your own to regain hand/ arm function?....... yes/ no
    a. If yes, describe the exercises that you do?

15. Do you have any questions?
   ..........................................................................................................................
Post-rehabilitation Interview

1. For the following activities of daily living, indicate the level of difficulty of this task by ranking the difficulty between 1 and 7. 1 being easy and 7 being difficult.

Activities:

<table>
<thead>
<tr>
<th>Activity</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening a door knob</td>
<td>easy</td>
</tr>
<tr>
<td>Opening / Closing a zipper</td>
<td>easy</td>
</tr>
<tr>
<td>Do-up / Undo a shirt button</td>
<td>easy</td>
</tr>
<tr>
<td>Using a key to open a lock</td>
<td>easy</td>
</tr>
<tr>
<td>Using scissors to cut paper</td>
<td>easy</td>
</tr>
<tr>
<td>Typing</td>
<td>easy</td>
</tr>
<tr>
<td>Handwriting</td>
<td>easy</td>
</tr>
</tbody>
</table>

Please comment if rating was four or greater about what aspect of the task is most difficult and why it was difficult. Explain if there was a difficulty in using your arm or hand during this task as well.

…………………………………………………………………………………………………
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…………………………………………………………………………………………………

2. Are there other activities of daily living that you have noticed a change in performance in two months?

3. Was there particular digit/digits that you had the most difficulty using? ......yes / no
   a. If yes, which digit did you have the most difficulty with?
   b. What difficulties did you have doing with this digit? Demonstrate if necessary.
   c. Have you noticed an improvement in function of this digit since your participation into the study?

4. Have you continued to notice improvement in function of your hand since your participation into the study? If so, how has it improved?
**Re-call Interview**

1. For the following activities of daily living, indicate the level of difficulty of this task by ranking the difficulty between 1 and 7. 1 being easy and 7 being difficult.

**Activities:**

<table>
<thead>
<tr>
<th>Activity</th>
<th>Rating (1-7)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opening a door knob</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
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<tr>
<td>Opening / Closing a zipper</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
<tr>
<td>Do-up / Undo a shirt button</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
<tr>
<td>Using a key to open a lock</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
<tr>
<td>Using scissors to cut paper</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
<tr>
<td>Typing</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
<tr>
<td>Handwriting</td>
<td>easy 1 2 3 4 5 6 7 difficult</td>
</tr>
</tbody>
</table>

Please comment if rating was four or greater about what aspect of the task is most difficult and why it was difficult. Explain if there was a difficulty in using your arm or hand during this task as well.

…………………………………………………………………………………………………
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2. Have you continued to notice improvement in function of your arm or hand in recent months/ is it still improving?

........................................................................................................................................
APPENDIX E. ELECTRODE PLACEMENT PROTOCOL

The subjects are seated beside a table with the forearm resting on the table. All placements are described based on a right arm.

<table>
<thead>
<tr>
<th>Muscles</th>
<th>Electrode Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECR</td>
<td>1. The forearm is positioned in a full pronation. The lateral epicondyle and the biceps tendon are palpated with the middle finger and the thumb respectively. The first muscle that can be felt with index finger is ECR. Then the subject is asked to extend his wrist to make sure the palpated muscle is ECR. The electrode placement is aligned with the direction of muscle fibers towards the thumb.</td>
</tr>
<tr>
<td></td>
<td>2. The forearm is positioned in semi-pronation. The subject is asked to flex the elbow so that the brachioradialis muscle could be palpated. Then examiner places his left index finger on this muscle with the middle finger lined up beside it. And the electrode is placed on the line just beside the middle finger. To ensure that the palpated muscle is ECR and not extensor digitorum communis, the subject is asked to wriggle his/her fingers continuously so that to locate the ECR.</td>
</tr>
<tr>
<td>EDC</td>
<td>The forearm is positioned in a full supination. The forearm is grasped at function of upper and middle third with the thumb and the middle finger on radius and ulna respectively. These two points were bisected with the index finger and the electrode is placed at the tip of the index finger. To make sure the palpated muscle is right, the subject is asked to wriggle the fingers continuously or extend all fingers at the MCP joints.</td>
</tr>
<tr>
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</tr>
<tr>
<td><strong>FCU</strong></td>
<td>The forearm is placed in a full supination with the elbow flexed on the table. The experimenter places her left middle finger along the ulnar bone with her index finger lines up beside it. The placement of the electrode is right on the right side of the experimenter’s index finger and a perpendicular line that is one-third the length of the forearm from proximal to distal. To make sure that the experimenter is on the right muscle, the subject is asked to flex and ulnar deviate his hand against to a resistance.</td>
</tr>
<tr>
<td><strong>FDS</strong></td>
<td>The forearm is placed in a full supination. FCU is palpated with the middle finger and the index finger is beside it. The electrode is placed on the midway of the palmar surface of the forearm and medial to the index finger.</td>
</tr>
</tbody>
</table>
| **PT** | 1- Subject’s forearm is positioned in a full supination. The gap between the flexor and the extensor muscle groups are palpated on the palmar surface by placing the middle and the index fingers horizontally from the biceps tendon. The electrode was placed at the medial side of the middle finger in the direction of medial epicondyle.  
2- The forearm is positioned in a full supination. The examiner finds the midpoint of a line connecting to the medial epicondyle (ME) and biceps tendon (BT), and draw a line at two finger distance from the midline through the hand by placing her index and middle fingers to the distal of ME (do not place the fingers on the midpoint). When the index finger is pointing in the direction of BT and ME, the electrode is placed in the direction of where the middle finger is pointing. The alignment of the electrode is the same with the alignment of the muscle which lies towards the thumb. |
<p>| <strong>BI</strong> | Subject is asked to flex his forearm in a full supination. Once the biceps is palpated, the electrode is placed on the belly of the muscle. |
| <strong>1DI</strong> | The examiner draws a transmetacarpal line perpendicular to the long axis of the hand at the level of the first metacarpal bone on the dorsal side of the hand. The electrode is placed just radial to the second metacarpal. 1DI radially deviates the index finger. |</p>
<table>
<thead>
<tr>
<th></th>
<th>Description</th>
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<tr>
<td>APB</td>
<td>The electrode is placed on the midpoint of a line drawn between the volar aspect of the first MCP joint and the carpometacarpal joint. Direction of the electrode is aligned with the thumb.</td>
</tr>
<tr>
<td>ADM</td>
<td>The electrode is placed at the midpoint of a line drawn between the ulnar aspects of the fifth MCP joint and the ulnar aspect of the pisiform.</td>
</tr>
</tbody>
</table>
APPENDIX F. MVC MEASUREMENT PROTOCOL

Subjects were seated beside a table with the forearm resting on the table (except for the measurement of elbow flexion). A computer screen was placed in front of the subject. A real time force feedback, as a bar corresponding to the applied force, was shown on the computer screen. Three MVC trials were performed for sixteen different movements. A calibration of the force transducer was performed before the positioning of the transducer for each trial. The transducer was always placed on the distal part of the measured joint. Subjects were asked to apply their maximal contraction force against the resistive force applied by the experimenter. The amount of the resistive force was adjusted with the maximal force applied by the subject to allow the isometric force.

<table>
<thead>
<tr>
<th>Movement</th>
<th>Position of Extremity &amp; Transducer Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion at elbow joint</td>
<td>90° elbow flexion with forearm is in full supination. The arm is supported by the elbow for anti-abduction of the shoulder. Transducer is placed just above the wrist joint.</td>
</tr>
<tr>
<td>Pronation of forearm</td>
<td>Forearm is in semi-supination, resting on the table. The wrist is supported for anti-flexion of elbow. Transducer is placed on the thenar muscles.</td>
</tr>
<tr>
<td>Extension at wrist</td>
<td>Forearm is in full supination. Wrist is aligned by the edge of the table with hand is hanging down from the table. The transducer is placed on the dorsal side of the hand on the MC bones. The resistive force is applied at the 10° wrist flexion.</td>
</tr>
<tr>
<td>Flexion at wrist</td>
<td>Forearm is in full pronation. Same wrist alignment with the table as above. The transducer is placed on the palmar side of the hand in the palm. The resistive force is applied at the 0° wrist extension.</td>
</tr>
<tr>
<td>Activity</td>
<td>Description</td>
</tr>
<tr>
<td>----------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Radial deviation of wrist</strong></td>
<td>Forearm is in full pronation, resting on the table. Wrist is supported for anti-flexion of the elbow. The transducer is placed vertically on the thenar side of the hand.</td>
</tr>
<tr>
<td><strong>Ulnar deviation of wrist</strong></td>
<td>Same positioning with above. The transducer is placed vertically to the hypothenar side of the hand.</td>
</tr>
<tr>
<td><strong>Finger flexion at MCP joint</strong></td>
<td>Forearm is in full supination. The wrist is supported for anti-flexion. The transducer is placed on the palmar side of the hand on the four fingers allowing flexion only at the MCP joint.</td>
</tr>
<tr>
<td><strong>Finger extension at MCP joint</strong></td>
<td>Forearm is in full pronation. The MCP joints of four fingers were aligned with the edge of the table. The transducer is placed on the dorsal side of the hand on the four fingers, allowing extension only at the MCP joint. The resistive force is applied approximately at the full MCP flexion.</td>
</tr>
<tr>
<td><strong>Individual finger flexion at MCP joint</strong></td>
<td></td>
</tr>
<tr>
<td>Index finger</td>
<td>The index finger is fully extended. The transducer is placed between the table and the index finger. Subjects applied pressing force on the transducer without bending the finger.</td>
</tr>
<tr>
<td>Middle finger</td>
<td>Same as above. Performed with the middle finger.</td>
</tr>
<tr>
<td>Ring finger</td>
<td>Same as above. Performed with the ring finger.</td>
</tr>
<tr>
<td>Little finger</td>
<td>Same as above. Performed with the little finger.</td>
</tr>
<tr>
<td>Abduction of thumb</td>
<td>Forearm is in full pronation. The transducer is placed vertically, lateral to the thumb, below the MCP joint.</td>
</tr>
<tr>
<td>Opposition of thumb</td>
<td>Forearm is in full supination. The transducer is hold by the experimenter between the little finger and thumb, inside the palm. The opposition force is applied by the thumb, pressing on the transducer.</td>
</tr>
<tr>
<td>Abduction of little finger</td>
<td>Forearm is in full pronation. The transducer is placed vertically, lateral to the little finger, below the MCP joint.</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Abduction of index finger</td>
<td>Forearm is in full pronation. The transducer is placed vertically, lateral to the index finger, Below the MCP joint.</td>
</tr>
</tbody>
</table>
Ada L, Canning GC, Low Sheau-Ling Stroke patients have selective muscle weakness in shortened range. Brain 2003; 126:724±731


Hankey GJ. Stroke treatment and prevention. Cambridge University Press. 2005


Twitchell ET. The restoration of motor function following hemiplegia in man 1951.


